Distinctiveness : Aurangabad

Organ and Tissue Transplant Programme

Government Recognitions for Organ Transplantation

Government of Maharashtra

सत्यमेव जयते
महाराष्ट्र शासन

Office of the Appropriate Authority

Certificate of Renewal of Registration

No. DHS / THOA / MGMHOSPAUR/KIDNEY/ **F.No** 63 **/ D-20 / 20** 15

This is with reference to the application, dated 13/05/2015 from MAHATMA GANDHI MISSION MEDICAL COLLEGE & HOSPITAL AURANGABAD for renewal of certificate of registration for performing Organ Transplantation, under the Act.

After having considered the facilities and standards of the above-said hospital, the Appropriate Authority hereby renews the Certificate of Registration of the said hospital for the purpose of performing KIDNEY Organ Transplantation for a period of five years from the date of issue.

संचालक आरोग्य सेवा, महाराष्ट्र राज्य, पुणे
सत्यमेव जयते

Mumbai:

Date: 14/10/2015

Appropriate Authority
and
Director Health Services,
Maharashtra State, Mumbai

समुचित प्रा
तथ
संचालक आर
महाराष्ट्र राज्

Government of Maharashtra

सार्वजनिक आरोग्य विभाग

सत्यमेव जयते
महाराष्ट्र शासन

Certificate Of Registration

In exercise of the power conferred by Sub-Section (1) of section 24 of the Transplantation of Human Organ Act, 1994 (42 of 1994), the Appropriate Authority hereby certify that M.G.M. Medical College and Hospital located at _____ has been inspected by the Appropriate Authority & Certificate of Registration is granted for performing the organ transplantation of the following organs.

1. Kidney
2. —
3. —
4. —

This certificate of registration is valid for a period of 5 years from the date of issue.

Mumbai

Date 28/7/2010

Appropriate Authority
Director, Health Services,
Maharashtra State, Mumbai

आरोग्य सेवा संचालनालय
(महाराष्ट्र राज्य)
" आरोग्य भवन ", सेंट जॉर्जेस रुग्णालय आवार, पी.डिमेलो रोड, मुंबई- ४०० ००१

	दूरध्वनी	
कार्यालय	२२६२१०३१-३६	Website : http://maha-arogya.gov.in
संचालक (वैयक्तिक)	२२६२१००६	Email : dhs_2005@rediffmail.com
सहसंचालक (रुग्णालये-राज्यस्तर)	२२६११४७१	Email : miscell@rediffmail.com
सहसंचालक (प्राआकेंद्र-जिपस्तर)	२२६२०२४९	Fax No. 022-22621034 / 22620234 (DHS)
सहसंचालक (असंसर्गजन्य रोग)	२२६२११८६	022- 22679044(Hosp.)
सहसंचालक (खरेदी कक्ष)	२२६२६२८२	022-22622155(CAO)
सहसंचालक (अर्थ व आस्थापना)	२२६२६७५५	022-22703785(Control Room)
		022-22621047 (NCD)

क्र.संआसे/डायलेसीस/ प्रशिक्षण/कक्ष ३/२०१३
दिनांक - ४/०४/२०१३

प्रति,

१) विभाग प्रमुख,
नेफ्रॉलॉजी डिपार्टमेंट,
के.ई.एम. हॉस्पीटल मुंबई, बी.जे.एल.नायर रुग्णालय, जे.जे समूह रुग्णालय, बी.जे. वैद्यकिय महाविद्यालय पुणे, के.ई.एम रुग्णालय पुणे, सुपर स्पेशालिटी हॉस्पीटल नागपूर, (अंतर्गत जी.एम.सी.नागपूर), जी.एम.सी औरंगाबाद, एम.जी.एम औरंगाबाद, मिरज मेडिकल कॉलेज मिरज, वैद्यकिय अधिक्षक सुपर स्पेशालिटी हॉस्पीटल अमरावती व नाशिक

विषय:- राज्यातील जिल्हा रुग्णालये मधे सुरु करण्यात येणाऱ्या डायलेसीस युनिट मधील भिषक/वैद्यकिय अधिकारी /स्टॉफ नर्सेस/ डायलीसीस टेक्नीशियन यांना प्रशिक्षण देणेबाबत...

संदर्भ:- मा. संचालक डी.एम.ई.आर यांचे पत्र क्र.संवैशिवसं/संआसे/डायलेसीस/ तंत्रज्ञ/ प्रशिक्षण/४-३ दि.१५/२/२०१३.

उपरोक्त संदर्भाधिन विषयाव्दारे आपणास कळविण्यात येते की, राज्यातील सर्व जिल्हा रुग्णालये/ सामान्य रुग्णालये व उपजिल्हा रुग्णालय शेगांव व पंढरपूर/ नांदेड स्त्री रुग्णालय या ठिकाणी लवकरच डायलेसीस युनिटस सुरु करण्यात येणार आहेत. तत्पूर्वी या युनिट मधे कार्यरत होणाऱ्या सर्वांचे गुणवत्तापूर्ण प्रशिक्षण होणे गरजेचे आहे. या प्रशिक्षणाचा आराखडा ठरविणे तसेच प्रशिक्षणामध्ये समन्वयाच्या दृष्टीने नेफ्रॉलॉजी युनिट प्रमुख संबंधित हॉस्पीटलस यांची बैठक दिनांक १६/४/२०१३ रोजी दुपारी ३ वाजता आरोग्य सेवा संचालनालय, मुंबई (आठवा मजला,

DIRECTORATE OF HEALTH SERVICES.
(MAHARASHTRA STATE)
Arogya Bhavan, St.George's Hospital Compound, P.D'Mello Road, Mumbai-400 001.

	Tel.No.	
Office: Director(Personal) Jt.Director(Hospital) ADHS (HOTA)	22621031-36 22621006 22611471 22703861	Website : http://maha-arogya.gov.in Email : adhthoa@gmail.com Email : jdhs03@gmail.com Fax No. 022-22621034 / 22620234 (DHS) 022-22679044 (Hosp.) 022-22703861 (THAO)
		N0.DHS/THOA/ MGMHsop.Aurangabad /Liver Transp.Team/ D-20/ 17 Date- 27/07/2017

To,
Dr. R. B. Bohra
Dean
Mahatma Gandhi Mission Medical College & Hospital,
N-6, CIDCO
Aurangabad-431003.

Sub:- **Transplantation of Human Organ Act 1994**
Liver Transplant Team

Ref:- Your application dtd. 16/03/2017

With reference to your application, the **Liver Transplant Team** of specialists whose names have been sent to this office for the approval of the State Appropriate Authority under the provision of the Transplantation of Human Organs Act 1994, for the purpose of Liver Transplantations operations in your hospital, the State Appropriate Authority herewith grants recognition to the **Liver Transplant Team** of your hospital as shown as below. **This is valid for the period of five years from the date of issue.**

LIVER TRANSPLANT TEAM

Sr.No.	Designation	Name of Consultant
1	**Transplant Surgeon**	Dr. Ravi Mohanka, Transplant Surgeon Dr. Gaurav Chaubal, Transplant Surgeon Dr. Somnath Chattopadhyay, Transplant Surgeon Dr. Pravin Suryawanshi, Transplant Surgeon
2	**Transplant Physician**	Dr. Samir R. Shah, Gastroenterologist Dr. Akash Shukla, Gastroenterologist Dr. Parijat A. Gupte, Gastroenterologist Dr. Ashok Mohite, Gastroenterologist Dr. Vijay S. Gulwe, Gastroenterologist Dr. Sonali Bhattu, Gastroenterologist
3	**Transplant Anesthesiologist**	Dr. Sanhita Kulkarni, Anaesthesiologist Dr. Vasanthi Kelkar, Anaesthesiologist Dr. Balaji Asegaonkar, Anaesthesiologist Dr. Pramod Apsingekar, Anaesthesiologist Dr. Pramod Bhale, Anaesthesiologist

- If any doctor resigns the institute, then intimate immediately to the Appropriate Authority.

Govt. of Maharashtra

FORM 16

Certificate of Registration For Performing Organ / Tissue Transplantation / Retrieval And / Or Tissue Banking

[Refer rule 24(2)]

This is to certify that ..MGM MEDICAL COLLEGE &.....
HOSPITAL.....
Hospital/Tissue Bank located at...CIDCO, AURANGABAD.....
has been inspected and certificate of registration is granted for performing the organ/tissue retrieval/transplantation/banking of the following organ(s) /tissue(s) (mention the names) under the Transplantation of Human Organs Act, 1994 (42 of 1994):-

1. LIVER
2. .....
3. .....
4. .....

This certificate of registration is valid for a period of five years from the date of issue.

This permission is being given with the current facilities and staff shown in the present application form. Any reduction in the staff and/or facility must be brought to the notice of the undersigned.

संचालक आरोग्य सेवा, महाराष्ट्र राज्य, मुंबई

Place : MUMBAI

Date : 27/07/2017

Signature of Appropriate Authority

समुचित प्राधिकरण

Seal.....तथा.....

संचालक आरोग्य सेवा,

आरोग्य सेवा संचालनालय

(महाराष्ट्र राज्य)

आरोग्य भवन सेंट जॉर्जेस रुग्णालय आवार, पी.डिमेलो रोड, मुंबई- ४०० ००१

	दूरध्वनी	Website : http://maha-arogya.gov.in Email : adhsthoa@gmail.com Email : jdhs03@gmail.com Fax No. 022-22621034 / 22620234 (DHS) 022- 22679044(Hosp.) 022-22703861 (THAO)
कार्यालय	२२६२१०३१-३६	
संचालक (वैयक्तिक)	२२६२१००६	
सहसंचालक (रुग्णालये-राज्यस्तर)	२२६११४७१	
सहा.संचालक (माअप्र)	२२७०३८६१	
रजिस्टर ए.डी.		क्र.संआसे/माअप्र//एमजीएममेडिकलकॉलेज औरंगाबाद/ईडीसी/नोंदणी/कक्ष-२०/१५ दिनांकः- ०५/०८/२०१५

प्रति,
अधिष्ठाता एमजीएम मेडिकल
कॉलेज ॲन्ड हॉस्पीटल,
एन-६,सीडको, औरंगाबाद.

विषय - मानवी अवयव प्रत्यारोपण कायदा १९९४ अंतर्गत आयडोनेशन सेंटर म्हणून नोंदणी मिळणेबाबत.

संदर्भ - आपला प्रस्ताव दि. ६.१०.२०१४

उपरोक्त संदर्भिय विषयाच्या अनुषंगाने आपणास कळविण्यात येते की, आपण मागणी केल्यानुसार आपल्या रुग्णालयास आयडोनेशन सेंटर म्हणून नोंदणी नुतणीकरण प्रमाणपत्र देण्यात येत आहे. सदर प्रमाणपत्राची वैधता प्रमाणपत्र दिल्याच्या दिनांकापासून पुढील पाच वर्षासाठी राहील. नोंदणी प्रमाणपत्राच्या तारखेच्या ३ महिने अगोदर नूतणीकरणचा प्रस्ताव या संचालनालयास सादर करणे आवश्यक राहील याची नोंद घ्यावी.

सोबतः प्रमाणपत्र

5/8/15

समुचित प्राधिकरण तथा
संचालक आरोग्य सेवा
महाराष्ट्र राज्य मुंबई

Dr Mukhtar

प्रतः सहसंचालक आरोग्य सेवा (अंनिका) मुंबई यांना माहितीसाठी.

9-10-2015
3.40 PM.

Date of Issue - 5/8/2015
Date of Expiry Renewal - 5/8/2020
Date of Renewal - 5/4/2020.

Bhosale
5/10/15

Government of Maharashtra

सत्यमेव जयते
महाराष्ट्र शासन

Office of the Appropriate Authority

Certificate of Registration

No. DHS/ THOA /MGM MED COLL / EDC /F.No /D-20/2015

This is to certify that MAHATMA GANDHI MISSION, MEDICAL COLLEGE & Hospital located at CIDCO, AURANGABAD has been inspected by the Appropriate Authority and certificate of registration is granted for performing the organ transplantation of the following organs:-

1. EYE DONATION CENTER
2. ~~_____~~
3. ~~_____~~
4. ~~_____~~

This certificate of registration is valid for a period of five years from the date of issue .

Mumbai:

Date: 05/08/2015

संचालक, आरोग्य सेवा, महाराष्ट्र राज्य, मुंबई

Appropriate Authority
and
Director Health Services,
Maharashtra State, Mumbai

सक्षम प्राधिकारण
तथा
संचालक आरोग्य सेवा,
महाराष्ट्र राज्य, मुंबई

DIRECTORATE OF HEALTH SERVICES.
(MAHARASHTRA STATE)
Arogya Bhavan, St.George's Hospital Compound, P.D'Mello Road, Mumbai-400 001.

	Tel.No.	Website : http://maha-arogya.gov.in Email : adhsthoa@gmail.com Fax No. 022-22621034 / 22620234 (DHS) 022-22679044 (Hosp.) 022-22703861(THOA)
Office: Director(Personal) Jt.Director(Hospital) ADHS (THOA)	22621031-36 22621006 22611471 22703861	
		NO.DHS/THOA/MGM med College & Hosp,A'bad./Corneal TranpTeam /19 Date- 23 /01/2019

To,
Dean,
MGM Medical College & Hospital,
N-6, Cidco, Aurangbad-431003.,

Sub:- **Transplantation of Human Organ Act 1994 & Amendment 2011**
Cornea Transplant Team

Ref:- Your application dtd. 15/01/2019

With reference to your application, the **Cornea Transplant Team** of specialists whose names have been sent to this office for the approval of the State Appropriate Authority under the provision of the Transplantation of Human Organs Act 1994, for the purpose of Cornea Transplantations operations in your hospital, the State Appropriate Authority herewith grants recognition to the **Cornea Transplant Team** of your hospital as shown as below. **This is valid for the period of five years from the date of issue.**

CORNEA TRANSPLANT TEAM

Sr.No.	Designation	Name of Consultant
1	Transplant Surgeon	Dr. Sarika Gadekar, Ophthalmologist
2	Transplant Anesthesiologist	Dr. Vasanti Kelkar, Anesthesiologist Dr. Ajita Annachatre (Dunk), Anesthesiologist Dr. Anuradha Jogdand, Anesthesiologist

- If any doctor resigns the institute, then intimate immediately to the Appropriate Authority.
- If any new doctor is joining to your institute, then before joining the team, the institute has to take the permission on behalf of the doctor from Appropriate Authority, without which the newly joined doctor cannot work in the transplantation program.

Dr. Anupkumar Yadav
Commissioner (Health & Family welfare)
and
Director Health Services, Mumbai

DIRECTORATE OF HEALTH SERVICES.
(MAHARASHTRA STATE)

Arogya Bhavan, St.George's Hospital Compound, P.D'Mello Road, Mumbai-400 001.

	Tel.No.	Website : http://maha-arogya.gov.in
Office:	22621031-36	Email : adisthoa@gmail.com
Director(Personal)	22621006	Fax No. 022-22621034 / 22620234 (DHS)
Jt.Director(Hospital)	22611471	022-22679044 (Hosp.)
ADHS (THOA)	22703861	022-22703861(THOA)

No.DHS/THOA/ MGM med.College & Hosp.A'bad./ /Corneal Tranp.Reg/D-20/19
Date- 23 /04/2019

To,
Dean,
MGM Medical College & Hospital,
N-6, Cidco, Aurangbad-431003..

Sub:- **Transplantation of Human Organ Act 1994 & Amendment 2011**
Cornea Transplant Registration

Ref:- Your application dtd. 15/01/2019

With reference to your application, please find enclosed herewith the approval for following Committee.

1) Certificate of Registration for Cornea Transplantation.
2) Approval for following committees
   a) Cornea Transplant Team

You are instructed to affiliate your hospital with District Blindness Control Society & with Director Regional Organ & Tissue Transplant Organization (ROTTO) Mumbai & Director, National Organ & Tissue Transplant Organization (NOTTO) New Delhi for co-ordination of deceased (cadaver) donor organ transplant activities.

You should regularly submit monthly performance report in the prescribed format.

You are instructed to follow all the provisions in the Transplantation of Human Organs Act 1994 & Rules 1995, Transplantation of Human Organs (Amendment) Rules, 2008 and Transplantation of Human Organs (Amendments) Act, 2011 & Rules 2014.

Please acknowledge the same.

Dr. Anupkumar Yadav
Commissioner (Health & Family welfare)
and
Director Health Services, Mumbai

C.C.to: 1) Joint Director Health Services (NPCB) Mumbai.
2) Secretary, Regional Organ & Tissue Transplant Organization K.E.M. Hospital Parel Mumbai.
3) Director, National Organ & Tissue Transplant Organisation, 4th & 5th Floor, NIOP Bldg., Safdarjung Hospital, New Delhi-110029.

Government of Maharashtra

FORM 16

CERTIFICATE OF REGISTRATION FOR PERFORMING ORGAN/TISSUE TRANSPLANTATION/RETRIEVAL AND OR TISSUE BANKING

[Refer Rule No. 24(2)]

This is to certify that MAHATMA GANDHI MISSION MEDICAL COLLEGE & HOSPITAL Hospital/Tissue Bank located at N-6, CIDCO, AURANGABAD-431003 has been inspected and certificate of registration is granted for performing the organ/tissue retrieval/Transplantation/Banking of the following organ(s)/tissue(s) (mention the names) under the Transplantation of Human Organ Act, 1994(42 of 1994):-

1. CORNEA TRANSPLANT CENTRE

This certificate is valid for a period of five years from the date issue.

This permission is being given with the current facilities and staff shown in the present application form. Any reduction in the staff and /or facility must be brought to the notice of the undersigned.

संचालक आरोग्य सेवा, महाराष्ट्र राज्य, मुंबई

Place:- Mumbai

Date :- 23/04/2019

Signature of Appropriate Authority

Seal.....

समुचित प्रधिकरण
तथा
संचालक आरोग्य सेवा,
महाराष्ट्र राज्य, मुंबई

[नि.प्र.ध.अ./मुं.सा.वि./३म.

No. 4401

सत्यमेव जयते

नोंदणी प्रमाणपत्र

याद्वारे प्रमाणपत्र देण्यात येते की, खाली वर्णन केलेली सार्वजनिक विश्वस्तव्यवस्था ही आज, मुंबई सार्वजनिक विश्वस्तव्यवस्था अधिनियम, १९५० (सन १९५० चा मुंबई अधिनियम २९) या अन्वये Aurangabad Region, Aurangabad. येथील सार्वजनिक विश्वस्तव्यवस्था नोंदणी कार्यालयात योग्य रितीने नोंदण्यात आलेली आहे.

सार्वजनिक विश्वस्तव्यवस्थेचे नाव : Zonal Transplantation Coordination Center, Aurangabad.

सार्वजनिक विश्वस्तव्यवस्थांच्या नोंदणी पुस्तकातील क्रमांक : E - 1298 (A.bad)

Dr. Sudhir Gajanan Kulkarni यांस प्रमाणपत्र दिले.

आज दिनांक 12.04.2016 २०१- रोजी माझ्या सहीनिशी दिले.

Assistant Charity Commissioner, Aurangabad Region, Aurangabad

शिक्का :

सही 12/4/16

पदनाम

Organ donation Awareness Programs

Organ Donation Awareness Programs

1½ Mr. Nana Patekar & Mr. Makarand Anaspure pledge organ donation

26 jan. 2016 MGM Medical College & Hospital

लाखभर नागरिक अवयवदानास सज्ज

म. टा. प्रतिनिधी, औरंगाबाद

मागच्या दीड-पावणेदोन वर्षांपासून औरंगाबाद शहरात सुरू झालेल्या अवयवदान मोहिमेत आतापर्यंत १३ ब्रेन डेड रुग्णांनी हृदय, यकृत, मूत्रपिंड, बुब्बुळ आदी ४५ अवयवांचे दान केले आहेच; शिवाय तब्बल दीड लाखापेक्षा जास्त नागरिक अवयवदानासाठी इच्छुक असून, त्यांनी 'झेडटीसीसी'मार्फत, वेगवेगळ्या शासकीय-खासगी रुग्णालयांमार्फत अवयवदानाचे फॉर्म भरले आहेत. काहींनी 'नोटो'च्या वेबसाईटवर अवयवदानाची नोंद करुन अवयवदानाची इच्छा व्यक्त केली आहे. फॉर्म भरले म्हणजे अवयवदान झालेच, असे नसले तरी नागरिक अवयवदानासाठी इच्छुक आहेत आणि त्यांची अवयवदाची सुप्तेच्छा नातेवाईकांना सर्वश्रुत होऊन वेळ आल्यावर त्यांची अखेरची इच्छा पूर्ण होण्याची शक्यता नक्कीच वाढते, असेही मानले जात आहे.

मागच्या वर्षी म्हणजेच १५ जानेवारी २०१६ रोजी मराठवाडा-विदर्भ-खान्देशातील पहिले अवयवदान औरंगाबादेत झाले आणि त्या दिवसापासून अवयवदानाविषयी मोठ्या प्रमाणावर सकारात्मक वातावरण तयार होण्यास सुरुवात झाली. त्याच सुमारास स्थापन झालेल्या 'झोनल ट्रान्स्प्लान्ट कोऑर्डिनेशन कमिटी'मार्फतही (झेडटीसीसी) अवयवदानाचा प्रचार-प्रसार सुरू झाला. मराठवाडा-विदर्भ-खान्देशातील पहिला अवयवदाता राम मगर याच्या अवयवदानानंतर हॉटेल रामा इंटरनॅशनल येथे झालेल्या अवयवदात्याच्या कुटुंबियांच्या सत्कारावेळी अवयवदानाविषयी खास कार्यक्रम घेण्यात आला होता. त्यावेळी अवयवदानाविषयी इत्यंभूत माहिती देण्यात आली होती. त्यानंतरही वेळोवेळी विविध शाळा-महाविद्यालयांमधून तसेच वेगवेगळ्या कार्यक्रमांच्या निमित्ताने अवयवदानाविषयी जनजागृती करण्यात येत आहे. शासकीय स्तरावरही मागच्या वर्षापासून महाअवयवदान उपक्रम राबविण्यात येत आहे. अलीकडे प्रत्येक कार्यक्रमाअंतर्गत अवयवदानाचे फॉर्म वाटप मोठ्या प्रमाणावर होत आहे. त्याचा एकत्रित परिणाम म्हणून आतापर्यंत १३ ब्रेन डेड रुग्णांकडून सुमारे ४५ अवयवांचे दान झाले आहे.

मागच्या वर्षी कार्यक्रमानिमित्त आलेले प्रसिद्ध अभिनेते नाना पाटेकर व मकरंद अनासपुरे यांनीही अवयवदानाचे फॉर्म भरले.

वेगवेगळ्या कंपन्यांमध्ये जनजागृती

सद्यस्थितीत काही औद्योगिक कंपन्यांमध्ये अवयवदानाविषयी जनजागृती केली जात असून, अवयवदानाची शास्त्रशुद्ध माहिती देणे, शंकांचे निरसन करणे, अवयवदानाचे कार्ड वाटप करणे, फॉर्म भरुन घेणे किंवा 'नोटो'च्या वेबसाईटवर नोंद करण्यास प्रोत्साहन देणे, असे विविध उपक्रम हाती घेण्यात आले आहे, असे ट्रान्स्प्लान्ट को-ऑर्डिनेटर मनोज गाडेकर यांनी 'मटा'ला सांगितले.

एखाद्या व्यक्तीने अवयवदानाचा फॉर्म भरला म्हणजे संबंधित व्यक्तीने अवयवदानाची इच्छा प्रकट केली आहे आणि अशी इच्छा फॉर्मच्या किंवा 'डोनर कार्ड'च्या निमित्ताने संबंधितांच्या नातेवाईकांना स्पष्ट झाल्याशिवाय राहात नाही. त्यामुळे तशी वेळ आल्यास नातेवाईकांना अवयवदानाचे स्मरण राहू शकते किंवा अवयवदानाबाबत सुचविल्यास नातेवाईकांची तशी मानसिकता तयार होऊ शकते. त्यामुळे फॉर्म भरणे-भरुन घेण्यातून जनजागृती नक्कीच होत आहे.

– डॉ. सुधीर कुलकर्णी, अध्यक्ष, झेडटीसीसी

2. Wockhardt India Shendra Unit 16.09.2016

3. Clover Dell School MGM Campus, Aurangabad 19.08.2016

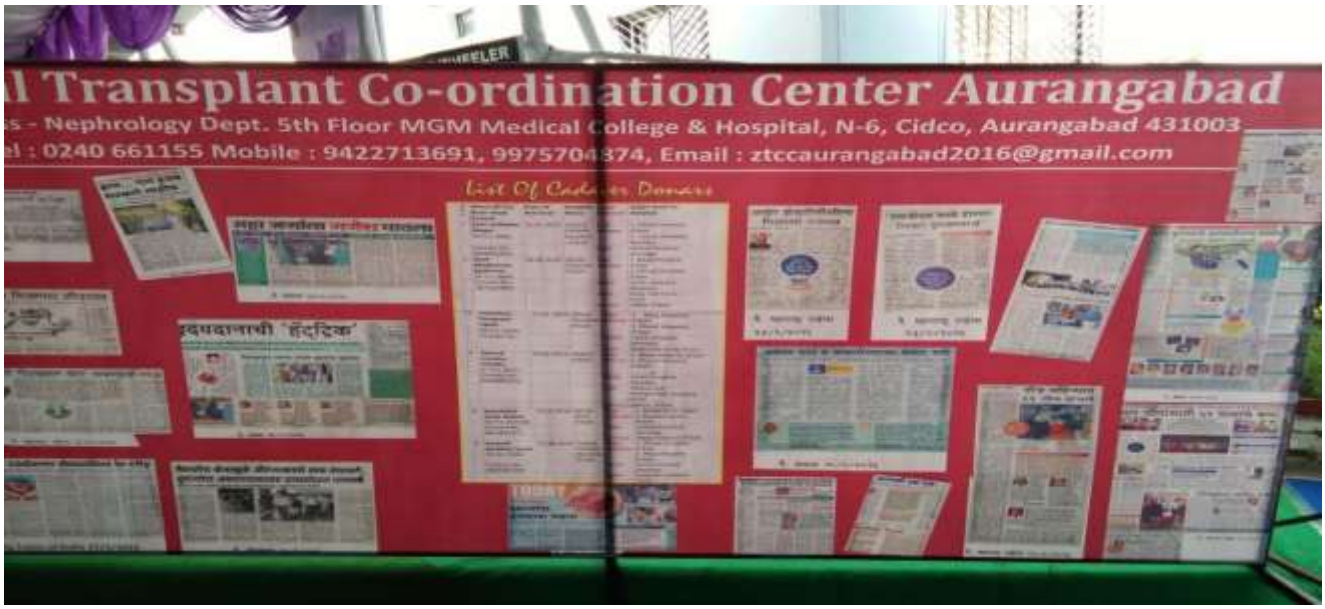
4. Rotary Club Youth Festival Aurangabad

23 Sept. 2016

5. Maharashtra Physician Conferences, Aurangabad

Organ Donation Awareness Stall 21, 22 &23/10/2016

6. Maharashtra Orthopedics Conference 2016, Organ Donation Awareness Stall . Nov. 2016



7. **World kidney day**
   09/03/2017 Rukmini hall MGM Hospital Aurangabad.

- **Dr. Purushottam Bhapkar, IAS , MGM and Govt. Medical College and Hospital authority signing on Donor card.**

- **Felicitation of Donor**

8. **Mahaavayav Daan Abhiyan 2017**

Organ Donation Awareness Rally GMC Hospital Aurangabad. 29.08.2017

9. Indian Medical Associaton, Aurangabad IMA Hall 26.08.2017

10. Vasantrao Naik Maha Vidhyalaya, Aurangabad.

30/08/2017

अवयवदानामुळे दुसऱ्यांना जीवदान मिळू शकते : डॉ. गुंडरे

औरंगाबाद • डीबी स्टार

इतर देशांच्या तुलनेत आपल्या देशात अवयवदानाचे प्रमाण कमी आहे. आज अनेकांना अवयवदानाची गरज आहे. अवयवदानामुळे दुसऱ्यांना जीवदान मिळू शकते, असे प्रतिपादन एमआयटी हॉस्पिटलचे फिजिशियन डॉ. रोहन गुंडरे यांनी केले.

वसंतराव नाईक महाविद्यालयात महाअवयवदान मोहिमेनिमित्त आयोजित व्याख्यानात ते बोलत होते. अध्यक्षस्थानी प्रा. सुभाष चव्हाण, समाजशास्त्र विभागप्रमुख डॉ. डी. के. दराडे उपस्थित होते. डॉ. गुंडरे म्हणाले, नेत्र, त्वचा, हृदय, यकृत, मेंदू दान केल्याने गरजवंताला जीवदान मिळू शकते. जिवंत किंवा मृत व्यक्तीसुद्धा अवयवदान करू शकतात.' कार्यक्रम यशस्वितेसाठी राष्ट्रीय सेवा योजनेचे कार्यक्रम अधिकारी डॉ. महेश कुलथे, डॉ. सुनीता राठोड, प्रा. गजानन हणवते आदींनी परिश्रम घेतले.

वसंतराव नाईक महाविद्यालयात महाअवयवदान मोहिमेनिमित्त आयोजित विविध स्पर्धेतील विजेत्यांना बक्षीस वितरण करताना मान्यवर.

महाअवयवदानानिमित्ताने घेण्यात आलेल्या विविध स्पर्धेतील विजेत्यांना या वेळी पारितोषिके देण्यात आली. रांगोळी स्पर्धेत रिता धानोरकर, प्रगती नागुरे, पूजा बोरा तसेच निबंध स्पर्धेत प्रियंका आहेरराव, पूनम नागुरे, कमलाकर लांडगे यांना बक्षिसे मिळाली. चित्रकला स्पर्धेत दीपक ढाकणे, मयूरी कुलकर्णी, कोमल शेजवळ आणि पोस्टर्स स्पर्धेत आरती गावंडे, कोमल शेजवळ, ऐश्वर्या झिने यांना सन्मानित करण्यात आले.

11. Guruvarya Lahuji Salve Arogya Kendra, Aurangabad. 31/08/2017

12. IANCON Conference 2017 ZTCC Stall . 9&10 September 2017

13. Wockhardt Shendra Unit, Aurangabad 12/09/2017

14. Wockhardt Chikalthana MIDC Unit, Aurangabad 14/09/2017

15. Wockhardt Valuj MIDC Unit, Aurangabad 14/09/2017

16. New High School, Chowka, Dist. Aurangabad
23.09.2017

17. College of Social Work, Dr. B.A.M. Univercity Aurangabad.

25.09.2017

18. Sant Rohidas Arogya Kendra, Mukundwadi, Aurangabad 26.10.2017

19. Central Bank of India, Kranti Chowk, Aurangabad.

12.12.2017

20. Organ Donation (Stall) MAHA_AGRO State Level agriculture Exhibition, Aurangabad. 08.01.2018

21. Sant Savata Mali College, Phulambri. 31.01.2018

22. Nutan Mahavidyalaya, Jintur Road, Parbhani. Organ Donation (Stall) 13.02.2018 to 17.02.201

23.Health awareness Lecture, at. Jolly Board Ltd Chikalthana MIDC Aurangabad

16 May 2018

24.KIDNEYTHON (Kidney awareness marathon) Organized by United CIIGMA Hospital, Aurangabad. 04.03.2018

25.MGM,JNEC Campus Aryabhatta Hall, Aurangabad. World Kidney Day &World Women's Day, 8 March 2018 Organ Donation awareness (Stall).

26. Organ donation awareness campaign at the village of Vihamandava. 31.03.2018

27. (Marriage Ceremony) organ donation awareness Stall. Venue: - Ramchandra mangal Karyalaya, Aurangabad. 07.05.2018

लग्नसोहळ्यात अवयवदानाचा जागर

म. टा. प्रतिनिधी, औरंगाबाद

५० झाडांच्या संवर्धनाचा संकल्प

28. Sai Mandir, Waluj MIDC, Aurangabad. 07.06.2018

ORGAN DONATION AWARENESS PROGRAM 2016

Sr. No.	Date	Venue	Speaker/Guest	Audience	Remark
1	26.01.2016	MGM Medical College & Hospital,Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	50	In Presence of **Mr. Nana Patekar** & **Mr. Makarand Anaspure**
2	19.08.2016	Mahatma Gandhi Mission's Clover Dale School Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	60	
3	28.08.2017	GMC Hospital to Kranti Chowk Aurangabad	Organ Donation Awareness Rally	15000	Maha Avayavdaan Abhiyan 2016
4	31.08.2016	GMC Medical College & Hospital, Aurangabad	Felicitation program of Relatives of Cadaver Donor	100	Maha Avayavdaan Abhiyan 2016
5	16.09.2016	Wockhardt India, Shendra MIDC Unit, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	45	
6	24.09.2016	Sant Eknath Rangmandir, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad		Rotary Club Youth Festival Aurangabad
7	21, 22 & 23 Octo.2016	Jawaharlal Nehru Engineering College, Aurangabad	Organ Donation Awareness Stall	1000	Maharashtra Physician Conferences, Aurangabad
8	Nov. 2016	Jawaharlal Nehru Engineering College, Aurangabad	Organ Donation Awareness Stall	600	Maharashtra Orthopedics Conference 2016,

ORGAN DONATION AWARENESS PROGRAM 2017

Sr. No.	Date	Venue	Speaker /Guest	Audience	Remark
1	09.03.2017	Rukmini Hall MGM Hospital Aurangabad	**Dr. Purushottam Bhapkar, IAS**	200	Felicitation program of Relatives of Cadaver Donor Showed Phir Jindagi movie (Based on organ donation)
2	29.08.2017	GMC Hospital to Kranti Chowk Aurangabad	Organ Donation Awareness Rally	2700	Maha Avayavdaan Abhiyan 2017
3	26.08.2017	IMA Hall, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	35	Organised by Indian Medical Association, Aurangabad
4	30.08.2017	Vasantrao Naik College, Aurangabad	**Dr. Rohan Gundre,** Transplant Physician, MIT Hospital, Aurangabad **Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	210	
5	31.08.2017	Guruvarya Lahuji Salve Arogya Kendra, Aurangabad	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	80	participated slum areas womens & young girls

6	09 & 10 Sap. 2017	MGM Medical College & Hospital,Aurangabad	Organ Donation Awareness Stall	300	IANCON Conference 2017
7	12.09.2017	Wockhardt India, Shendra MIDC Unit, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	55	
8	14.09.2017	Wockhardt India, Chikalthana MIDC Unit, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	48	
9	15.09.2017	Wockhardt India, Valuj MIDC Unit, Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	40	
10	23.09.2017	New High School, Chowka, Tq. Phulambri,Dist. Aurangabad	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	90	
11	25.09.2017	College of Social Work, Dr. B.A.M. Univercity Aurangabad.	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	40	
12	26.10.2017	Sant Rohidas Arogya Kendra, Mukundwadi, Aurangabad	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	60	
13	12.12.2017	Central Bank of India, New Usmanpura, Aurangabad	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	40	Organ Donation camp organised by CBI Group.

ORGAN DONATION AWARENESS PROGRAM 2018

Sr. No.	Date	Venue	Speaker /Guest	Audience	Remark
1	08.01.2018	Ayodhya Nagri, Padampura, Ground, Aurangabad.	**Mr. Jayas Dhabale, Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	800	Organ Donation (Stall) MAHA_AGRO State Level agriculture Exhibition, Aurangabad.
2	31.01.2018	Sant Savata Mali College, Phulambri.	**Mr. Manoj Gadekar** Central Transplant Coordinator ZTCC Aurangabad	58	
3	13.02.1018 to 17.02.2018	Nutan Mahavidyalaya, Jintur Road, Parbhani.	**Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	6000	Organ Donation (Stall) MAHA AROGYA SHIBIR Nutan Mahavidyalaya, Jintur Road, Parbhani.
4	16.02.2018	Health awareness Lecture, at. Jolly Board Ltd Chikalthana MIDC Aurangabad	**Dr. Sudhir Kulkarni** Prof. & Head, Nephrology Dept. MGM. President, ZTCC Aurangabad	90	Topic: How to Keep Healthy & Organ Donation awareness
5	04.03.2018	United CIIGMA Hospital, Aurangabad.	**Mr. Manoj Gadekar, Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	1600	KIDNEYTHON (Kidney awareness marathon) Orgnised by United CIIGMA Hospital, Aurangabad.
6	08.03.2018	MGM,JNEC Campus Aryabhatta Hall, Aurangabad	**Mr. Manoj Gadekar, Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	300	World Kidney day&World Women's Day, 8 March 2018 Organ Donation awareness (Stall)

7	31.03.2018	Vihamandwa Tq. Paithan, Dist, Aurangabad.	**Mr. Manoj Gadekar, Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	95	31.03.2018 on the occasion of Hanuman Jayanti we conducted organ donation awareness campaign at the village of Vihamandva.
8	07.05.2018	Ramchandra mangal Karyalaya (Hall) Beed bypass , Aurangabad.	**Mr. Manoj Gadekar, Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	600	07.05.2018 on the occasion of Deshpande Family (Marriage Ceremony) we conducted organ donation awareness Stall. Venue:- Ramchandra mangal Karyalaya, Aurangabad.
9	07.06.2018	At. Sai Mandir, Waluj MIDC, Aurangabad.	**Mr. Jayas Dhabale** Central Transplant Coordinator ZTCC Aurangabad	1300	Organ Donation awareness (Stall)

ORGAN DONATION AWARENESS PROGRAM 2019

1	09.02.2019	SRM College of Social work Chandrapur/Sant Rohidas arogya jendta aurangabad	Dr. Sudhir Kulkarni, Manoj Gadekar	25	Organ Donation Awareness talk
2	03.02.2019	MASICON 2019/ MGM Medical college & Hospital Aurangabad	Dr. Sudhir Kulkarni, Manoj Gadhekar	400	Organ Donation Awareness talk / Cadaver donor's relatives felicitation and marathon for organ donation
3	21.02.2019	Sanvad setu pratishthan, Aurangabad	Manoj Gadhekar	27	Organ Donation Awareness talk
4	09.03.2019	Gappa katta self healp group, Nandanvan colony, Aurangabad	Manoj Gadekar	47	Organ Donation Awareness talk
5	14.03.2019	Shree rameshwar high school, Waghola, Dist. Aurangabad	Manoj Gadekar	60	Organ Donation Awareness talk / World Kidney day Human chain
6	14.03.2019	Waghola, Tq. Phulambri, Dist. Aurangabad	Manoj Gadekar	34	Organ Donation Awareness talk
8	01.04.2019	Helping Hands, Magal Papers, Supari Hanuman road, Aurangabad	Manoj Gadekar	16	Organ Donation Awareness talk

Publications

Case Report

Anesthesia for Combined Cesarean Section and Pheochromocytoma Resection

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Abstract

Pheochromocytoma (PCC) is a rare cause of hypertension during pregnancy [1:54000 pregnancies]. Fetomaternal morbidity and mortality is about 58% if the diagnosis is missed. Administration of anesthesia to patients with PCC is challenging. Associated pregnancy adds to the problems. This is a case report of a patient having PCC diagnosed at 26 weeks of gestation. With medical management pregnancy was continued till 34 weeks. She was posted for cesarean section and resection of PCC. Patient underwent surgery lasting for 7 h due to inferior vena cava tear and had stormy intra as well as postoperative course. Mother and baby had uneventful recovery due to continuous invasive monitoring and a good teamwork, despite limited anesthetic resources.

Keywords: *Cesarean section, hypertension, pheochromocytoma, pregnancy*

Introduction

Incidence of pheochromocytoma (PCC) during pregnancy is 1 out of 54,000.[1] Patient may be misdiagnosed as having preeclampsia, leading to increased fetomaternal mortality (58%).[2] This can be reduced by antenatal diagnosis and proper treatment. Anesthetic management of a parturient posted for a cesarean section and PCC resection in a limited resource setting is described.

Case Report

A 22-year-old fourth gravida, having PCC diagnosed at 26 weeks of gestation, was posted for elective cesarean section at 34 weeks. She was asymptomatic and had no pedal edema. Pulse rate was 84/min and blood pressure was 120/80 mm Hg. Her antihypertensive medication consisted of Nifedipine 10 mg TDS, Prazosin 1.5 mg QID, and Metoprolol 50 mg OD. Systemic examination did not reveal any abnormality. Hemoglobin was 9.5 gm%. Ultrasonography abdomen revealed right-sided suprarenal mass [7.6 cm × 5.4 cm]. Urinary vanillylmandelic acid was 30.4 mg (normal range: 2–7 mg) over 24 hours.[3]

There was no albuminuria. Electrolytes, blood sugar, uric acid, coagulation profile, liver, kidney and thyroid function tests, calcitonin levels, fundoscopy, ECG, two-dimensional Echo were within the normal limits.

Patient received oral 10 mg Diazepam and 50 mg Metoprolol at night. Last dose of Nifedipine (10 mg) and 1.5 mg Prazosin was administered 4 h before surgery. We planned epidural anesthesia for cesarean section followed by additional general anesthesia for tumor resection.[4]

Intravenous (IV) Ranitidine and Ondansetron were given. Preoperative Metoclopramide was avoided to prevent hypertensive crisis.[5]

Multiparamonitor was applied. Fetal heart surveillance (FHS) was 130/min. Internal jugular vein and radial artery cannulation was done after giving 50 µgm of Fentanyl and one mg of Midazolam. 500 ml of ringer lactate was administered for preloading. After negative aspiration, a test dose of 2 ml, 1.5% Lignocaine with Adrenaline[6] was administered epidurally, but she became drowsy and developed twitching of muscles with blood pressure of 170/130 mm Hg, 120/min pulse rate, and 90% SpO_2. Suspecting accidental intravascular injection of Lignocaine with Adrenaline, 100% oxygen was administered along with 50 mg of Thiopentone, 1 mg Phentolamine, and 5 mg of Labetalol. After 5 min, the patient was fully conscious and SpO_2 was 98% on air along with pulse rate 110/min, blood pressure 130/90 mm Hg,

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and there were no twitchings. FHS slowed down to 80/min. We planned for general anesthesia to avoid fetal distress. 100 mg Thiopentone, 50 mg Propofol, and 100 mg Suxamethonium were administered for induction and rapid sequence intubation. Oxygen, nitrous oxide, Halothane [0.5%], Vecuronium, and Fentanyl were used for maintenance. A baby (2 kg wt) with APGAR score 8 was delivered without applying fundal pressure and then shifted to neonatal intensive care unit (NICU) with APGAR score 10 (5 min) for monitoring. Patient received Oxytocin to control postpartum hemorrhage and blood transfusion was initiated.

Our aim to maintain mean arterial pressure (MAP) in the range of 70–100 mm Hg, heart rate 80–110/min, central venous pressure (CVP) 8–10 mm Hg was achieved by using phentolamine, metoprolol, and fluid administration. No additional drugs were required to maintain the parameters. Sodium nitroprusside (SNS) drip was added after delivery of baby.

There was a rent in inferior vena cava (IVC) during resection. IVC repair required 5 h and tumor resection another 2 h during which there were a lot of fluctuations in MAP (68–150 mm Hg), heart rate (80–150/min), and CVP (4–12 mm Hg).

Blood loss during this period was about 3000 ml. She required 3500 ml of crystalloids, 500 ml of hydroxy ethyl starch 6%, and 1800 ml of whole blood. Urine output and blood sugar level were within the normal limits.

After resection, CVP was 10 mm Hg but MAP was 40 mm of Hg for which infusion of noradrenalin (1 μg/kg/min) was started. At the end of surgery, while artificial ventilation was still continued, airway pressures progressively increased over 30 min (peak 28 cm and plateau pressure 24 cm) along with fall in SpO_2 90% (FiO_2 1). There were bilateral crepitations in chest. Pink, frothy secretions were seen through endotracheal tube. Blood pressure was 120/90 mm Hg, with pulse rate of 140/min and 14 mm Hg CVP. 60 mg Furosemide was administered and gradually 10 cm PEEP was instituted in stages. Noradrenalin was gradually tapered over 40 min. SpO_2 improved to 100% (FiO_2 0.5) and MAP was stable (60 mm Hg without vasopressor). Two packed cell volumes of blood were administered over 2 h as hemoglobin was 6 gm%. She developed hypoglycemia (50 mg%) during this period, which was promptly treated with 50 ml 50% dextrose and 100 ml/hour 5% dextrose maintenance drip. She was electively ventilated in ICU for 10 hours using Midazolam (1 mg/h) and Vecuron (1micrgm/kg/min) infusion. Postoperative analgesia was provided with 8 hourly IV 100 mg Tramadol and 75 mg diclofenac infusion as the epidural catheter was blocked due to blood clot. As her pulse rate (80–90/min) and MAP (70–80 mm Hg) were stable for next 4 h, she was extubated. Baby was shifted from NICU after 24 hours. Mother was discharged on 12th postoperative day.

Discussion

Goals of management of PCC during pregnancy are early diagnosis, control of blood pressure, and definitive surgery to reduce fetomaternal mortality (<5%).[7]

Hypertension in our patient was controlled with Prazosin, a selective α-1 antagonist. Its short duration helped in titration of dose,[8] without adverse effects on fetus.[9] Nifedipine helped to reduce vasospasm.[10] Terazosin and Doxazosin can also be used.[2] Phenoxybenzamine is commonly used but long duration of action predisposes to postoperative hypotension and needs monitoring of newborn.[11] Our patient had less than 20 mm orthostatic hypotension which assured adequate alpha blockade.[5] Tachycardia was treated with Metoprolol. Other beta-blockers[12] can be used after adequate alpha blockade.

Timing and mode of surgery remains controversial. PCC should be resected in second trimester or after delivery.[13] Cesarean section is preferred over vaginal delivery[14] and PCC should be resected simultaneously or after delivery.[12] Laparotomy is recommended for PCC resection,[12] after 24 weeks of gestation. So we planned cesarean section along with PCC resection.[15,16] If only cesarean section is performed, there can be postoperative hypertensive crisis. There are reports of PCC resection in postpartum period at interval as localization of tumor might be difficult during pregnancy.[17]

These patients may develop hypertension during shifting, induction, intubation, fundal pressure during cesarean section, tumor handling, and hypotension due to hemorrhage and following tumor resection.

Almost every possible anesthetic technique has been advocated by Hull.[17] Anesthetic technique does not have a major impact on surgical outcome.[6] Epidural, general anesthesia, or combined can be used for cesarean section and PCC resection.

We planned epidural for cesarean section to minimize chances of hypertensive crises.[4,18] Test dose of Lignocaine with Adrenalin can be administered as usual.[6] Three ml plain Bupivacaine can also be used. However, we had to switch to general anesthesia in anticipation of impending fetal distress as fetal heart rate decreased following accidental intravascular injection of Lignocaine with Adrenaline (140–80/min).

We added Propofol for induction to reduce intubation pressor response.[19] Rapid sequence intubation was done using Suxamethonium.[20] Fasciculation can compress tumor and stimulate autonomic ganglia.[6] But we had no other option at that time. As time was crucial, prior Magnesium sulfate (2 mg/kg over 10 min) could not be tried. Prior Phentolamine, Labetalol, and Propofol might have prevented hypertension after Suxamethonium in our patient. Rocuronium can be used but may cause hypertensive crises.[21]

Maintenance was done with Halothane 0.5%[22,23] but its usage in patients with PCC is controversial due to its arrhythmogenic property.[12] But that was the only volatile agent available with us 12 years back. All other volatile agents except desflurane can be used. Desflurane produces sympathetic stimulation.[5] Fentanyl was used for our patient. Remifentanyl is preferred due to its shorter half-life in neonates.

We have used phentolamine prior to delivery of baby. Although low-dose SNP infusion (<1 μg/kg/min)[12] is considered safe, keeping possibility of fetal cyanide toxicity, sodium nitroprusside was added later during tumor resection. Postoperative hypotension in our patient could be due to fall in catecholamine levels and due to blood loss. Sudden reductions in catecholamines also lead to hypoglycemia.[5] Pulmonary edema might be due to increased permeability of pulmonary vessels during pregnancy, IV fluids, and noradrenalin. Patient responded to addition of PEEP and furosemide. Gradual tapering of noradrenalin reduced systemic vascular resistance and preload.

PCC is a rare but treatable cause of hypertension during pregnancy leading to high fetomaternal morbidity and mortality. Multidisciplinary approach helps for better outcome. Anesthetic management is challenging and needs a good teamwork.

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Conflicts of interest

There are no conflicts of interest.

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Urinary Tract Infection in Chronic Kidney Disease

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Abstract: ***In a study of 120 CKD patients of nephrology unit in MGM Medical College, Aurangabad, a high incidence of urinary tract infection (U.T.I.) i.e. 35% was observed. E.coli was the predominating urinary pathogen. Presence of UTI is found to have associated with increased mortality in CKD patients indicating severe outcome in this study. However, the mortality is attributable only to the presence of UTI cannot be estimated due to presence of other confounding factors like diabetes mellitus and hemodialysis. Moreover, this study indicates the association of Diabetes, hemodialysis and advanced CKD stage as risk factors for U.T.I in CKD patients***

Keywords: UTI, CKD, Diabetes Mellitus, E.Coli, Hemodialysis

1. Introduction

Urinary tract infection is an infection of one or more structure in the urinary system. The urinary tract includes urethra, bladder, ureters, prostate and kidneys which is normally sterile and resistant to bacterial colonization. The body's defense mechanism against uti includes complete emptying of the bladder during urination, urine acidity, the vesicoureteral valve, and various immunologic and mucosal barriers. In cases of renal failure, there is a change in the composition of urine with oliguria, anuria, albuminuria and haematuria. The resultant changes in pH, osmolality and urinary urea definitely have their own effects in urinary infection.[5] The urinary tract is the most common site of nosocomial infection[1,2,3] and most of these infections follow instrumentation of urinary tract, mainly urinary catheterization and is a subsequent cause of significant morbidity, sepsis and death[4.] Most frequently bacteria from the urethral meatus ascend to the bladder between the catheter and urethral surfaces. Alternatively, bacteria may ascend within the urinary drainage systems following contamination of the drainage bag or catheter tubing junction. The presence of bacteria in the bladder constitutes a potential reservoir for multi resistant bacteria.[2,4]

The risk of acquiring a urinary tract infection depends on the method and duration of catheterization, and the quality of catheter care.[2,3]

Intensive care units are a meeting point between the most severely ill patients receiving aggressive therapy and the most resistant pathogens which are selected by the use of broad spectrum antimicrobial therapy.[5]

Epidemiological study suggests that the 3 most commonly seen infectious complications in the CKD population are: urinary tract infection, pneumonia, and sepsis.[1] However, there are few studies of patients with CKD and UTI[6] and the incidence of UTIs in patients with CKD is unclear.[7]

2. Aims & Objectives

1) To study the incidence of urinary tract infections in chronic kidney disease patients
2) To know the microorganisms responsible for urinary tract infections in chronic kidney disease patients
3) To know whether presence of urinary tract infections influences the outcome in chronic kidney disease patients

3. Material and Methods

A total of 120 CKD patients above 18 year old attending nephrology unit were included where as patient having obstructive uropathies (urethral strictures, renal calculi) or patients with indwelling catheter or patients who were on antibiotic therapy prior to hospitalization or on immunosuppressive therapy were excluded. In this study U.T.I was defined as asymptomatic[8] or symptomatic bateriuria[9, 11] with isolation of at least one micro organism in urine culture. A clean catch urine sample-midstream was collected in a wide mouth, leak proof container with straps on lids. Urine samples were sent to laboratory within 2-3 hours of collection. In case of any delay, urine specimens were preserved by using boric acid as preservative or refrigeration at 2 - 4°C up to a span of 24 to 72 hours [12,13]
In the present study diagnosis of U.T.I was established either by

a) Urine microscopy of clean catch urine in which presence of 10 or more white cells per cubic millimeter in a urine specimen, 3 or more white cells per high-power field of unspun urine[8] or leukocytes, leukocyte casts, and other cellular elements were observed directly under the microscope
b) All specimens were inoculated on Mac Conkey agar plate and was incubated at 35°C overnight and specific organism was isolated by inoculating colony on blood agar plates. The antibiotic sensitivity was based on Kirby Bauer method of antibiotic susceptibility
c) The ultrasound image shows a smaller kidney, thinning of the parenchyma and its hyperechogenicity (reflecting sclerosis and fibrosis) except in patients of diabetic

nephropathy in which both renal size and parenchymal thickness are preserved until end-stage renal failure.[17] Ultrasonogram can show evidence of pyelonephritis and cystitis as an additional clue for diagnosis of UTI and rule out obstructive uropathies

4. Results

Table 1: Incidence of UTI in CKD Patients:

UTI	*No. of patients*	*Percentage*
Positive	42	35.0
Negative	78	65.0
Total	120	100%

Out of 120 CKD patients, the total number of patients having urinary tract infection was 42. Hence, the incidence of UTI in CKD patients in this study was 35%.

Table 2: Gender wise distribution of UTI in CKD patients

Gender	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
Male	18	42.9	38	48.7	56	40.0	0.337	P=0.539 NS
Female	24	57.1	40	51.3	64	60.0		
Total	42	100	78	100	120	100		

Out of 120 CKD patients in this study, 56 (40%) were males and 64 (60%) were females. Among the 42 CKD patients having UTI, 18 (42.9%) were males and 24 (57.1%) were females. Among the 78 CKD patients not having UTI, 38 (48.7%) were males and 40 (51.3%) were females. The difference between the two groups was not statistically significant in this study.

Table 3: Age-Group wise distribution of UTI in CKD Patients:

Age-Group	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
19-30	03	7.1	06	7.7	06	5.0	6.68	P=0.246 NS
31-40	04	9.5	11	14.1	15	12.5		
41-50	05	11.9	20	25.6	27	22.5		
51-60	10	23.8	17	21.8	27	22.5		
61-70	16	38.1	22	28.2	38	31.7		
>70	04	9.5	02	2.6	07	5.8		
Total	42	100	78	100	120	100		

Out of 120 CKD patients, 38 (31.7%) were from age group 61-70 years followed by 27 (22.5%) each from age group 41-50 and 51-60 years. Among the 42 CKD patients having UTI, majority were from age group 61 – 70 years i.e. 16 (38.1%) followed by 51 – 60 years i.e. 10 (23.8%). Among the 78 CKD patients without UTI, majority were from age group 61 – 70 years i.e. 22 (28.2%) followed by 20 (25.6%) from 41-50 year age group.

Table 4: Association of Diabetics Mellitus with UTI in CKD Patients:

Diabetic status	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
Diabetic	26	61.9	34	43.6	60	50.0	3.69	P=0.041 S
Non diabetic	16	38.1	44	56.4	60	50.0		
Total	42	100	78	100	120	100		

Out of 120 CKD patients in this study, 60 (50%) were diabetic and 60 (50%) were non diabetic. Among the 42 CKD patients with UTI, 26 (61.9%) were diabetic and 16 (38.1%) were non diabetic. Among the 78 CKD patients not having UTI, 34 (43.6%) were diabetic and 44 (56.4%) were non diabetic. The statistical difference between two groups was significant indicating association between diabetes mellitus and UTI in CKD patients.

Table 5: CKD stage wise distribution of UTI:

CKD stage	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
G1	00	00	00	00	00	00	9.38	P=0.002 S
G2	03	7.1	07	8.9	10	8.3		
G3A	04	9.5	20	25.6	24	20.0		
G3B	06	14.3	20	25.6	26	21.7		
G4	10	23.8	14	17.9	24	20.0		
G5	19	45.3	17	21.8	36	30.0		
Total	42	100	78	100	120	100		

Out of 120 CKD patients, majority belonged to stage G5 i.e. 36 (30%). Among the 42 CKD patients having UTI, majority belonged to stage G5 i.e. 19 (45.3%) followed by G4 i.e. 10 (23.8%). Among the 78 CKD patients without UTI, majority belonged to CKD stage G3a and G3b with each being 20 (25.6%) of patients. The statistical difference between the two groups was significant indicating association of severity of CKD stage and UTI in this study.

Table 6: Association of Hemodialysis with UTI in CKD Patients:

Hemo – dialysis status	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
HD	29	69.0	31	39.7	60	50.0	9.38	P=0.002 S
Non HD	13	31.0	47	61.3	60	50.0		
Total	42	100	78	100	120	100		

Out of 120 CKD patients, 60 (50%) were on maintenance hemodialysis and 60 (50%) were managed conservatively. Among the 42 CKD patients having UTI, 29 (69%) were on maintenance hemodialysis and 13 (31%) were on conservative treatment. Among the 78 CKD patients without UTI, 31 (39.7%) were on hemodialysis and 47 (61.3%) were being managed conservatively. The statistical difference between two groups was significant indicating association between hemodialysis and UTI in CKD patients.

Table 7: Microorganisms isolated in UTI in CKD Patients:

Microorganisms isolated	UTI Positive	
	No	%
E.coli	24	57.1
S.aureus	06	14.3
Klebsiella spp.	08	19.1
Proteus spp.	03	7.1
Candida spp.	03	7.1
Acinetobacter spp.	1	2.4
Cons	03	7.1

The most common microorganism isolated from 120 CKD patients having UTI was E.coli in 24 (57.1%) patients followed by Klebsiella spp. in 8 (19.1%) patients. Other microorganisms were S.aureus in 6 (14.3%), Proteus spp. in

3 (7.1%), Coagulase negative staphylococci in 3 (7.1%), Candida spp. in 3 (7.1%) and Acinetobacter spp. in 1 (2.4%) patients in this study.

Table 9: Deaths in UTI with CKD Patients:

Death status	UTI				Total		Chi-square test	P-value
	Positive		Negative					
	No	%	No	%	No	%		
Dead	14	33.3	10	12.8	24	20.0	7.18	P=0.007 S
Alive	28	66.7	68	87.2	96	80.0		
Total	42	100	78	100	120	100		

Out of 120 CKD patients, 24 (20%) succumbed to death and 96 (80%) were salvaged. Among 42 CKD patients having UTI, 14 (33.3%) patients succumbed to death and 28 (66.7%) were salvaged. Among the 78 CKD patients without UTI, 10 (12.8) succumbed to death while 68 (87.2%) were salvaged. The statistical difference between the two groups is significant indicating association between severity of outcome and UTI in CKD patients.

5. Discussion

1) Incidence of UTI in CKD patients in this study was 35%. In a study conducted by Jadhav SK, et al., high incidence of urinary tract infection i.e. 57.5%, was observed in CKD patients.[5] This difference might be because of Exclusion of obstructive uropathies and catheterized patients in our study. In a cross sectional study conducted by Falah S Manhal, et al., the frequency of UTI in renal failure patients undergoing hemodialysis was found to be 37.5%.[18]
2) Among the 42 CKD patients having UTI, 18 (42.9%) were males and 24 (57.1%) were females. Among the 78 CKD patients not having UTI, 38 (48.7%) were males and 40 (51.3%) were females. The difference between the two groups was not statistically significant in this study. Similar results were observed in a study conducted by Chih-Yen HSIAO, et al. where, 52.2% were females and 47.8% were males out of all CKD patients with lower UTI.[19]
3) Among the 42 CKD patients having UTI, majority were from age group 61–70 years i.e. 16 (38.1%) followed by 51–60 years i.e. 10 (23.8%). Among the 78 CKD patients without UTI, majority were from age group 61 – 70 years i.e. 22 (28.2%). The statistical difference between the two groups was not significant in this study. Similar results were observed in a study conducted by Chih-Yen HSIAO et al. where, average ages of the upper and lower UTI patients with CKD were 59.21 ± 16.54 and 71.18 ± 14.77 years.[19] In a study by Zhang et al., they found a high prevalence (17.4%) of CKD among older adults 50 to 74 years from 9806 participants.[20] Another study done by Gauba C, et al., showed high incidence of urinary tract infections in CKD patients with older age group.[21]
4) Among the 42 CKD patients with UTI, 26 (61.9%) were diabetic and 16 (38.1%) were non diabetic. Among the 78 CKD patients not having UTI, 34 (43.6%) were diabetic and 44 (56.4%) were non diabetic. The statistical difference between two groups was significant indicating association between diabetes mellitus and UTI in this study. In a study conducted by Chih-Yen HSIAO et al., it was observed that 36.9% patients with lower UTI were found to have diabetes mellitus.[19] The difference might be attributable to the large number of diabetics in our study and high prevalence of diabetes mellitus in India.
5) Among the 42 CKD patients having UTI, majority belonged to stage G5 i.e. 19 (45.3%) followed by G4 i.e. 10 (23.8%). Among the 78 CKD patients without UTI, majority belonged to CKD stage G3a and G3b with each being 20 (25.6%) of patients. The statistical difference between the two groups was significant indicating association of severity of CKD stage and UTI in this study. A different study conducted by Gauba C et al. observed high incidence of UTI in patients with advanced CKD stage and low urine flow rates.[21] In a study conducted by Chih-Yen HSIAO et al. it was observed that patients belonging to CKD stage G4 and G5 were 13.4% and 8% respectively.[19] The difference might be due to geographic and genetic differences in Turkish and Indian population and large number CKD patients enrolled in our study belonged to CKD stage G4 & G5. Also all catheterized patients are excluded from our study.
6) Among the 42 CKD patients having UTI, 29 (69%) were on maintenance hemodialysis and 13 (31%) were on conservative treatment. Among the 78 CKD patients without UTI, 31(39.7%) were on hemodialysis and 47 (61.3%) were being managed conservatively. The statistical difference between two groups was significant indicating association between hemodialysis and UTI in this study. In a different study conducted by Jadhav SK, et al., out of 73 CKD patients undergoing hemodialysis 42 had UTI i.e. (57.5%).[5] In a study conducted by Falah S.Manhal et al., 37.5% of the CKD patients on maintenance hemodialysis had UTI.[18] The difference might be attributable to large number of ESRD patients requiring hemodialysis enrolled in our study.
7) The most common microorganism isolated from CKD patients having UTI was E.coli in 24 (57.1%) patients followed by Klebsiella spp. in 8 (19.1%) patients. Other microorganisms were S.aureus in 6 (14.3%), Proteus spp. in 3 (7.1%), Coagulase negative staphylococci in 3 (7.1%), Candida spp. in 3 (7.1%) and Acinetobacter spp. in 1 (2.4%) patients in this study. In a study conducted by FalahS. Manhal et al., (15%) patients had been infected with *E. coli*, (12.5%) patients with Klebsiella spp., and (2.5%) with Acinetobacter, α-hemolytic Streptococci, coagulase negative Staphylococci, and Proteus spp.[18] In a study done by HSIAO et al., Microorganisms isolated in upper and lower UTI were, E.coli (58.9% & 51.2%), Proteus (8.2% & 3%), Klebsiella (4.1% & 7.9%), Enterococcus (0% & 5.9%), Pseudomonas (2.7% & 6.9%) and staphylococcus (0% and 0.5%) respectively.[19] The minor difference may be attributable to exclusion of catheterized patients and obstructive uropathies in our study.
8) Among 42 CKD patients having UTI, 14 (33.3%) patients succumbed to death and 28 (66.7%) were salvaged. Among the 78 CKD patients without UTI, 10 (12.8) succumbed to death while 68 (87.2%) were salvaged. The statistical difference between the two groups is significant indicating association between severity of outcome and UTI in this study. A study done by Reinhard Funfstuck et al. observed that an acute infection can influence the course of a pre-existing renal

disease and enhance the development of renal failure in cases of existing damage of renal parenchyma or anatomical alteration of the urinary tract.[22]

6. Conclusion

This study was conducted on 120 CKD patients attending OPD and/or casualty of nephrology unit in MGM Medical College & Hospital, Aurangabad.

Incidence of urinary tract infection in chronic kidney disease patients is 35%.

E.coli was the most common microorganism isolated from urine cultures.

Presence of UTI is found to have associated with increased mortality in CKD patients indicating severe outcomes in this study. However, whether the mortality is attributable only to the presence of UTI cannot be estimated due to presence of other confounding factors such as diabetes mellitus and hemodialysis.

Moreover, this study indicates the association of Diabetes mellitus, hemodialysis and advanced CKD stage as risk factors for urinary tract infections in CKD patients.

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Case Report

Trimethoprim-induced Hyperkalemia in Renal Transplant Recipient

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Abstract

Trimethoprim-sulfamethoxazole (TMP-SMX) is an antimicrobial agent used in a variety of infections. Adverse reactions are more common in patients with AIDS but occasionally occur in immunocompetent patients. Renal toxicity is usually a hypersensitivity reaction to the sulfa component and manifests as interstitial nephritis or sulfa crystallization in the renal tubules. Reversible hyperkalemia is a rarely reported side effect of TMP-SMX therapy attributed to TMP inhibition of potassium secretion in the distal renal tubule in a manner similar to the potassium-sparing diuretic, amiloride. We report a case of hyperkalemia associated with TMP-SMX occurring in a 32-year-old renal transplant recipient with no other risk factors for hyperkalemia. He was treated with TMP-SMX (800 mg + 160 mg) two tablets QID for suspected pneumocystis jiroveci pneumonia. He developed severe hyperkalemia on day 9 posttherapy. Hyperkalemia reverted to normal with withdrawal of trimethoprim.

Keywords: Hyperkalemia, kidney transplant, pneumocystis jiroveci pneumonia, trimethoprim

Introduction

Trimethoprim-sulfamethoxazole (TMP-SMX) is an antibiotic of choice for pneumocystis jiroveci pneumonia (PJP). Adverse reactions that may be experienced include gastrointestinal upset and skin lesions.[1,2] Renal toxicity is usually a hypersensitivity reaction to the sulfa component and manifests as interstitial nephritis or sulfa crystallization in the renal tubules.[3,4] Reversible hyperkalemia is a rarely reported side effect of TMP-SMX therapy attributed to TMP inhibition of potassium secretion in the distal renal tubule in a manner similar to the potassium sparing diuretic, amiloride.[5] The occurrence of TMP-SMX-induced hyperkalemia has been reported in patients treated for upper respiratory infections,[6] PJP in non HIV patients,[7] PJP in acquired immunodeficiency syndrome[1] and as a standard dose prophylaxis in renal transplant recipients.[8] We present a case of life-threatening TMP-SMX-induced hyperkalemia in a renal transplant recipient.

Case Report

A 32-year-old male underwent second kidney transplant 2 years after failed graft due to biopsy-proven recurrence of immunoglobulin A nephropathy. In the present kidney transplant, the donor was a father-in-law with 5/7 mismatch. The donor's age was 65 years. He received antithymocyte globulin (2 mg/kg/day for 2 days) as induction therapy followed by standard triple immunosuppression. The patient was also given pneumocystis jiroveci prophylaxis with TMP-SMX, which was later omitted because of pancytopenia. Pancytopenia recovered after stopping TMP-SMX. The patient received cytomegalovirus (CMV) prophylaxis with valganciclovir 450 mg orally once a day. The patient was alright for 3-month posttransplant, with creatinine stable at 1.8 mg/dl. He presented with fever and cough for 7 days, for which he had received antibiotic (amoxicillin + clavulanate). In spite of antibiotic course, he developed sudden onset of breathlessness for which he was admitted. Chest X-ray on day 1 of admission showed bilateral opacities [Figure 1]. The patient's serum creatinine was 3.2 mg/dl when he developed PJP. His serum potassium was 3.8 meq/L at that time. ABG

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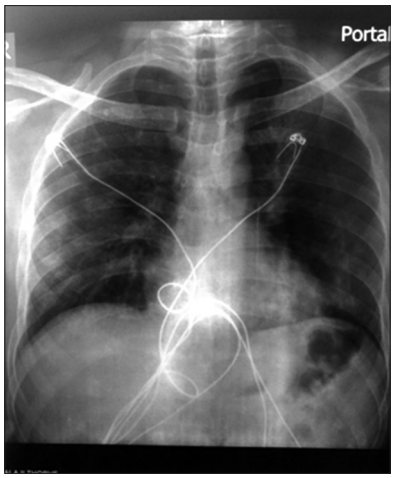

Figure 1: Chest X-ray: Day 1 of admission

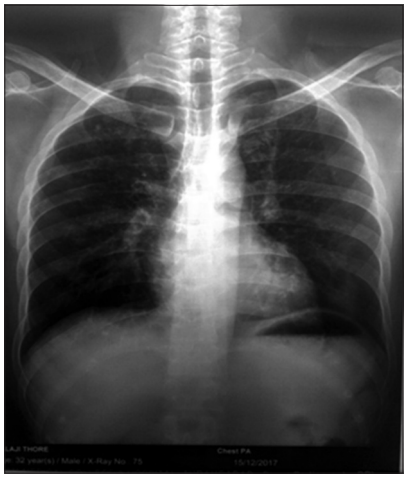

Figure 2: Chest X-ray: Day 14 of admission shows bilateral opacities; original photograph shows clearance

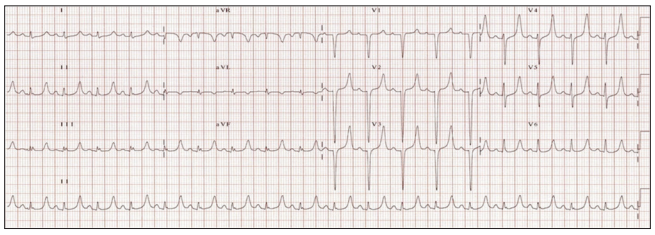

Figure 3: Electrocardiography: Signs of hyperkalemia

parameters were pH – 7.39, HCO3 – 13.2, PCO2 – 22.30, and PO2 – 79.70. Lactate level was not measured. His X-ray chest showed ground-glass opacities in both basilar areas. His serum lactate dehydrogenase levels were elevated to 890 mg/dl. CMV polymerase chain reaction (PCR) was negative. He did not produce sputum initially, and later, when sputum was available, it was sent for pneumocystis jiroveci PCR. The test was negative. He was started on TMP-SMX (two tablets QID). The dose of oral steroid was increased to 15 mg QID. The patient was given noninvasive ventilation as his oxygen saturation levels were low. The patient was maintained on oxygen therapy for 10 days. He showed gradual improvement in general condition. On the 9th day, his serum creatinine was 2.8 mg/dl, but he had serum potassium of 6.2 meq/L with electrocardiography evidence of hyperkalemia. Figure 2 the patient was not on any other confounding medications such as angiotensin receptor blockers/angiotensin-converting enzyme inhibitor which could contribute to hyperkalemia. His creatinine was 3.2 mg/dl at the time of admission, but it was improving gradually, so the possibility of hyperkalemia due to graft was not suspected. The possibility of trimethoprim-induced hyperkalemia was entertained, after ruling out tacrolimus as a cause of hyperkalemia. Tacrolimus levels were in normal range (9.76 ng/dl). The dose of TMP-SFX was reduced to two tablets BD. He was treated with intravenous calcium gluconate, glucose-insulin drip, and sodium bicarbonate. His serum potassium levels returned to normal on day 18. His serum creatinine had also come down to 2.5 mg/dl. The patient had symptomatic relief, and chest X-ray showed resolution of pneumonia. Figure 3 the patient was put on pneumocystis jiroveci prophylaxis once again with TMP-SMX double strength once a day. The dose of mycophenolate was reduced to 500 mg twice a day from 500 mg three times a day. Immunosuppression was restored to previous levels at discharge (tacrolimus 1.5 mg BD, mycophenolate 500 mg TDS, and oral steroid 20 mg BD).

Discussion

Our patient underwent second kidney transplantation (KT) with antithymocyte globulin as induction therapy. He did not receive pneumocystis jiroveci prophylaxis posttransplant completely due to thrombocytopenia. The donor was a father-in-law who was 65 years of age. The patient had reached serum creatinine of 1.8 mg/dl 15-day posttransplant. Three-month post-KT, his serum creatinine was 1.8 mg/dl. A British study by Akoh and Rana[9] showed that transplant patients who had donor above 60 years of age had mean serum creatinine of 2.0 mg/dl at the end of 3 months. Furthermore, our patient had good renal output, DSA was negative, graft rejection was not suspected, and renal biopsy was not performed during his initial 3 months after renal transplant. The patient developed PJP 3 months after transplant. The incidence of PJP in renal transplant recipient has been reported to be around 1%–6% in different studies.[10-12] The standard treatment for PJP is high-dose sulfamethoxazole-trimethoprim (TMP-SMX).[10] As bioavailability of co-trimoxazole is excellent, it can be used orally. Up to date, Thomas and Limper[13] recommended that co-trimoxazole has excellent bioavailability and oral administration is appropriate for all patients who have a functioning gastrointestinal tract. They also recommended the use of oral steroids in moderate-to-severe PJP. A study by Goto *et al*.[14] concluded that dose, duration, and timings of steroids had not been fully studied in transplant patients for treating PJP. Sometimes, hyperkalemia can be life threatening and levels can be as high as >6.5 mE/L, so it is mandatory to monitor potassium in patients receiving sulfamethoxazole + trimethoprim for PJP.

The mechanism of hyperkalemia with trimethoprim is blocking of potassium secretion from distal convoluted tubule.[3,4]

Conclusion

Potassium levels should be monitored when using high-dose SMX-TMP in patients of PJP. Reduction of dose and proper treatment of hyperkalemia will revert this complication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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CASE REPORT

Hyperhomocysteinemia with Anticoagulant-related Acute Kidney Injury

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ABSTRACT

A case of 40-year-old young woman with an extensive, acute thrombosis of left distal brachial artery following an elective laparoscopic cholecystectomy was reported. The patient underwent urgent surgical intervention for brachial artery thrombosis and was started on oral anticoagulant. Within a week, the patient presented with bleeding diathesis and acute renal insufficiency with sepsis. She was found to have markedly increased serum homocysteine level. No other thrombophilic factors could be found. On investigation, a genetic defect of homocysteine metabolism was found to be the underlying cause. The patient recovered completely on treatment with pyridoxine, cyanocobalamin, and folate.

Keywords: Acute kidney injury, Anticoagulant, Heterozygous methylenetetrahydrofolate reductase gene mutation, Thromboembolic event.

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INTRODUCTION

Thrombophilias, inherited or acquired, are conditions associated with hypercoagulable state and increased risk of arterial and venous thrombosis, which represent a significant cause of mortality and morbidity worldwide.[1] There may be interaction of genetic and environmental factors.[2] Investigating for thrombophilia requires an initial evaluation of classical prothrombotic risk factors, such as smoking, dyslipidemias, arterial hypertension, or diabetes mellitus. Extended profile of investigations is necessary in patients with arterial or venous thrombosis, which occurs repeatedly in unusual sites or at young age, also when family aggregation of thrombotic events is identified, as well as in women with recurrent idiopathic pregnancy loss. It must include a complete blood count and erythrocyte sedimentation rate, blood film examination, prothrombin time (PT) and activated partial thromboplastin time, factor V Leiden, antithrombin and fibrinogen levels, protein C and S, prothrombin gene mutations, homocysteinemia, methylenetetrahydrofolate reductase (MTHFR) gene mutations, and antiphospholipid antibodies.[3]

Mild to moderate hyperhomocysteinemia (HHC), meaning mildly to moderately increased plasma homocysteine (15–50 µmol/L), is uncommon in the general population. This condition is caused by either genetic factors (mutations of homocysteine metabolism enzymes) or acquired conditions, such as deficiencies in B vitamins, renal insufficiency, and some medications.[4] Two common mutations involving the MTHFR gene have been identified: C677T and A1298C.

CASE REPORT

The case of a 40-year-old nondiabetic and nonhypertensive female admitted with clinical picture of acute kidney injury (AKI) has been presented. Her medical history started 2 weeks prior to her admission, when she underwent an elective laparoscopic cholecystectomy. On the second postoperative day, she developed painful swelling of the left arm with clawing of the hand and ischemia of the fingers. Color Doppler study of left upper limb revealed acute thrombosis in left distal brachial, radial, ulnar, and median arteries with absent flow. She underwent urgent vascular intervention with left brachial artery embolectomy. She was heparinized and started on oral anticoagulant Acenocoumarol 3 mg twice a day. 1 week after embolectomy, she developed generalized swelling involving the face, arms and lower limbs, oliguria, hematuria, bleeding gums, hematemesis, shortness of breath, and fever.

On examination, she was febrile, conscious, oriented with pulse rate 124/minute, blood pressure 120/70 mm Hg, and relative risk 28/minute. She was pale, with facial and pedal edema. She had bleeding gums and left upper limb swelling. Her systemic examination was normal except bilateral fine crepitations on chest auscultation. Her investigations revealed hemoglobin (Hb) 2.9 gm/dL, total leukocyte count (TLC) 28,100/mm^3, platelets 600,000/mm^3, urea 124 mg/dL, creatinine 6.6 mg/dL, Na 118 mEq/L, K 4.4 mEq/L, PT 120 seconds, international

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normalized ratio (INR) 7.5, total serum proteins 5.2 gm/dL, serum albumin 1.8 gm/dL, Ca 9.5 mg/dL, PO_4 7.2 mg/dL, and uric acid 9.7 mg/dL. Urine analysis showed 4+ proteinuria, plenty of red blood cells (RBCs), and 30 to 40 pus cells/hpf. Liver function test, lipid profile, electrocardiogram, and two-dimensional echocardiogram were normal. Anticardiolipin antibodies and lupus anticoagulant tests were negative. Thrombophilia tests showed that protein C, protein S, and antithrombin III levels were within normal limits. Serum C3 complement was normal. Her homocysteine level was 43.08 µmol/L (normal 3.36 to 20.44 µmol/L in females).

The patient was negative for factor V Leiden and prothrombin gene mutation. However, she was found to have MTHFR gene polymorphism in the form of compound heterozygous for C677T and A1298C. Abdominal ultrasound found both normal-sized kidneys with increased echogenicity. Kidney biopsy was not performed due to risk of bleeding. She was treated conservatively with five packed cell volume (PCV) and eight fresh frozen plasma transfusions, intravenous vitamin K, injection Meropenem, injection Tranexamic acid, pyridoxine, cyanocobalamin, and folate supplements. Anticoagulants were discontinued. She did not need hemodialysis. By the end of 2 weeks, patient showed gradual improvement and was discharged with normal clinical and biochemical parameters. At the time of discharge, her laboratory tests showed PT 27.5 seconds, INR 2.29, normal renal functions, Hb 8.7 gm/dL, TLC 12,600/mm^3, PCV 26%, mean corpuscular volume 70 fL, mean corpuscular hemoglobin 18 pg, mean corpuscular hemoglobin concentration 26 gm/dL, and normal urinalysis.

DISCUSSION

The mechanism by which MTHFR gene mutations produce prothrombotic states is represented by elevated levels of plasma homocysteine due to decreased enzymatic activity of MTHFR that participates in regulating homocysteine metabolism, and a mutation of MTHFR may be a marker for possible elevated homocysteine levels. At present, HHC is considered to represent a risk factor for deep vein thrombosis and a common risk factor for recurrent venous thrombosis.[5] Heterozygotic status for two polymorphisms of the MTHFR gene, the C677T and A1298C, was found in our case. There are studies which suggest supplementation with folic acid; vitamin B6 and B12 may help in lowering the homocysteine concentrations, and even in reversing endothelial dysfunction regardless of the underlying cause of HHC.[5]

Acute kidney injury resulting from glomerular hemorrhage has been described in patients with glomerular lesions in the absence[6-8] and presence[9,10] of coagulopathy (INR 6–9 range). A biopsy study in patients who developed otherwise unexplained AKI in association with anticoagulant overdose revealed the predominant lesion of distal tubular injury and obstruction with RBCs and RBC casts.[11] The glomeruli show little or no abnormalities by light, immunofluorescence, or electron microscopy.[11] The recognition of a characteristic histologic lesion that was associated with the clinical presentation of otherwise unexplained AKI in the setting of overanticoagulation led to the term "anticoagulant-related nephropathy."

The pathogenesis event appears to be glomerular hemorrhage[12,13] resulting in the formation of obstructing RBC casts within renal tubules, which is the most conspicuous histologic feature of anticoagulant-related nephropathy.[11] The diagnosis of anticoagulant-related nephropathy should be suspected among patients who present with AKI in the setting of excessive anticoagulation. A definitive diagnosis is made by renal biopsy. However, biopsies are usually not performed, at least initially, among patients who are anticoagulated because the risk of bleeding is high.

Among patients who develop AKI and are on anticoagulant therapy, a presumptive diagnosis of anticoagulant-related nephropathy may be made if a severe warfarin coagulopathy is present and if other causes of AKI have been excluded by clinical features and serologic tests. Restoration of a therapeutic INR may limit the extent of AKI and chronic kidney injury that results from glomerular hemorrhage. The patient was discharged from the hospital with folic acid, vitamin B6, and vitamin B12 supplements. The peculiarities of the present case were the thrombotic events and the extensive arterial thrombosis in a young patient with HHC due to two heterozygotic mutations in the MTHFR gene. Anticoagulant nephrotoxicity presented as AKI, which was successfully treated conservatively.

CONCLUSION

In patients with unexplained arterial or venous thrombosis, it is appropriate to investigate for the possible coexistence of multiple predisposing factors for thrombosis, including measurement of the serum homocysteine level, in addition to investigations for mutations of the MTHFR, the prothrombin, and the factor V genes.

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Study of Intradialytic Hypertension: A Single Centre Analysis

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ABSTRACT

In India around 20,000 people are dependent upon hemodialysis. The greatest burden of morbidity and mortality for hemodialysis patients are cardiovascular diseases (CVDs) including fluctuations in blood pressure, as CVDs account for approximately 50% of all deaths. Intradialytic hypertension (IDH) is one such complication responsible for increased morbidity and mortality in chronic kidney disease (CKD) patients undergoing hemodialysis. In India, there is limited data available in the literature for the incidence of IDH in CKD patients on hemodialysis. In this observational study, we evaluated the incidence of IDH in Indian CKD patients undergoing hemodialysis. We found a higher incidence of IDH (34.51%) in our cohort than in Western studies. These patients were further evaluated for the association of IDH with contributory factors and patient outcomes after one year of follow-up. This analysis yielded a novel finding of a higher incidence of IDH in patients with lower creatinine, which needs to be confirmed with multicenter trials.

KEY WORDS: Intradialytic hypertension; Chronic kidney disease (CKD); Hemodialysis.

ABBREVIATIONS: CVDs: Cardiovascular diseases; IDH: Intradialytic hypertension; CKD: Chronic Kidney Disease; ESA: Erythropoietin-stimulating agent;

INTRODUCTION

In India around 20,000 people are dependent upon hemodialysis.[1] The greatest burden of morbidity and mortality for hemodialysis patients are cardiovascular diseases (CVDs), which accounts for approximately 50% of all deaths.[2] Fluctuations in blood pressure (BP) is one of the most common complication that occurs in patients taking hemodialysis. A recent study from South Africa showed that intradialytic hypertension (IDH) may affect as many as 28% of the dialysis population.[3] The only Indian study, reported from south India in 2016, looked at the incidence of IDH on the Indian hemodialysis population but did not study the factors responsible for IDH and the impact of IDH on mortality.[4] The aim of this study was to evaluate the incidence of IDH, to study factors responsible for IDH and effect of IDH on mortality in patients attending regular dialysis sessions at a single dialysis unit.

MATERIALS AND METHODS

The present study was conducted on 142 patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis at the dialysis centre of MGM Medical College & Hospital, Aurangabad, Maharashtra, India. This was a prospective observational cohort study over a period of 2.5 years from January 2013 to June 2015. All CKD patients over 18 years of age were included in the study. Patients of acute kidney injury were excluded. Primary end point was the development of IDH by patients undergoing regular hemodialysis. Secondary end points were potential biological markers associated with IDH and mortality in all patients. IDH was defined as an increase in systolic blood pressure of more than 10 mmHg during hemodialysis more than

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two hemodialysis sessions.[5] Using this definition, patients were stratified into IDH & Non-IDH categories for analysis.

Potential associated factors which were studied include age, sex, known case of hypertension (HTN) or diabetes mellitus, serum creatinine level, IV erythropoietin use, oedema, serum albumin level, and number of anti-hypertensive drugs. All patients were observed for one year to determine the mortality rate.

Dialysis prescription used for patients included in our study was as follows:

- Dialyser- Nipro elision 13 M synthetic polynephron
- Time- 4 hours
- Blood flow rate- 200-300 ml/hour
- Dialysate flow rate- 500 ml/hour
- Ultra filtration rate- as per weight gain
- Dialysate composition- Na^{++} 135-145 meq/L, K^{+} 0-4 meq/L, Ca^{++} 2.5-3.5 meq/L, Mg^{++} 0.5-0.75 meq/L, Cl 98-124 meq/L, Acetate 2-4meq/L, HCO_3 30-40 meq/L, Dextrose 11 g, pH 7.1-7.3
- Dialysate temperature- 36 ºC to 37 ºC
- Anticoagulation- Heparin 2000 IU at start of hemodialysis & 1000 IU per hour

Dialysis prescription was modified for IDH patients in the form of ultrafiltration rate and time of hemodialysis session. Patients who had IDH were treated either with injectable metoprolol 5 mg or injectable labetalol 20 mg with dose modified as per need. Effect of these drugs on IDH and on outcome was not studied in our study. The study was conducted in accordance with the ethical principles set out by the declaration of Helsinki and approval for the study was granted by the Human Research Ethics Committee of MGM University of Health Sciences (Registration Number- ECR/581/Inst/2013).

Statistical Analysis

SPSS software version 20 was used for the analysis of this data. Chi-square test was applied to check significant association between different groups and outcome of different attributes. *p*-value was checked at 5% level of significance.

RESULTS

Out of 142 patients, 49(34.51%) patients were found to have IDH (Figure 1). Among them 33(67.34%) were male, 30(61.22%) were above 60 years of age, 39(79.59%) were hypertensive, 10(20.41%) were diabetic, 34(69.38%) patients were receiving IV erythropoietin, 33(67.35%) were oedematous, 26(53.06%) had a serum albumin less than 2.5 mg/dL. 21(53.85%) patients

Graph 1: Incidence of IDH.

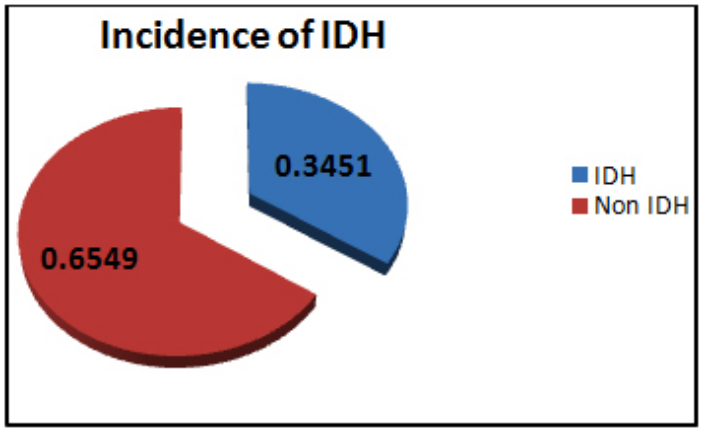

Table 1: Results Showing Non-modifiable Factors Responsible for IDH.

Factor		Results: IDH	Results: NON IDH	*p*-value
Age	>60 years	30 (61.22%)	60 (64.52%)	*p*=0.699
	<60 years	19 (38.78%)	33 (35.48%)	NS
Sex	Male	33 (67.34%)	70 (75.27%)	*p*=0.315 NS
	Female	16 (32.66%)	23 (24.73%)	

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Table 2: Results Showing Modifiable Factors Responsible for IDH.

Factor		Result		
		IDH	Non-IDH	*p*-value
Hypertension	HTN	39 (79.59%)	69 (74.19%)	*p*=0.474
	Non-HTN	10 (20.41%)	24 (25.81%)	NS
Diabetes mellitus	Diabetic	10 (20.41%)	16 (32.66%)	*p*=0.639
	Non-Diabetic	39 (79.59%)	77 (82.80%)	NS
Serum creatinine	<10 mg/dL	42 (85.71%)	61 (65.60%)	*p*=0.003
	>10mg/dL	7 (14.29%)	32 (34.40%)	S
Erythropoietin IV	EPO	34 (69.38%)	41 (44.09%)	*p*=0.004
	Non-EPO	15 (30.62%)	52 (55.91%)	S
Oedema	Oedematous	33 (67.35%)	30 (32.26%)	*p*<0.0001
	Non-Oedematous	16 (32.65%)	63 (67.74%)	S
Serum albumin	<2.5 mg/dL	26 (53.06%)	30 (32.26%)	*p*=0.016
	>2.5 mg/dL	23 (46.94%)	63 (67.74%)	S
No. of antihypertensive drugs	<2 drugs	21 (53.85%)	49 (71.01%)	*p*=0.073
	>2 drugs	18 (46.15%)	20 (28.99%)	NS

IDH Intradialytic hypertension; HTN Hypertension.

were taking less than two anti-hypertensive drugs. 19(38.77%) patients died within 1 year of study initiation. Novel finding was 42(85.71%) patients among IDH population had serum creatinine less 10 mg/dL A detailed description of all factors is listed in Tables 1 and 2.

DISCUSSION

The present study is a prospective observational study of the incidence of IDH in CKD patients undergoing maintenance hemodialysis. A total of 142 CKD patients were studied over a period of 2.5 years. Out of 142 patients, 49 were found to have IDH with incidence of 34.51%, which was greater than the incidence found in western studies by Stidley et al[6], Mees D[7], in which it was found to be 5-15%. A recent study from South Africa showed that IDH may affect as many as 28% of the dialysis population.[3] None of these studies focused in the Indian patient population. One study from South India by Darimireddi SK et al[4] found the incidence of IDH to be 49% in 100 patients studied. This supports our finding that the incidence of IDH is higher in Indian population.

Out of 142 patients in our study, 103 were males and 39 were females. 90 patients were above 60 years of age. In this study, no relation was found between the incidence of IDH and the age or sex of the patient. No previous studies demonstrated a statistically significant difference among males and females. According to a study by Inrig et al[5] on 32,295 patients, incidence of IDH was found more amongst the elderly. Similarly, in our study we found that IDH occurred more frequently in the elderly but it was not statistically significant.

No significant relationship was found between the incidence of IDH and previous HTN or diabetes in CKD patients. Although, no study examined the burden of IDH in known hypertensive patients, removal of anti-hypertensive medications during hemodialysis is one of the proposed mechanisms for IDH.[8] No comparable data regarding the relationship of diabetes mellitus and IDH was found in previous studies.

A significant inverse relationship was found between the incidence of IDH and serum creatinine level, with a higher incidence of IDH in patients with lower serum creatinine levels (Figure 2). In a study previously published by Inrig et al[9] patients who experienced IDH were thinner, had lower muscle mass (lower serum creatinine) and were more likely to experience a rise in blood pressure (BP) with minimal volume excess. Similarly, in our study occurrence of IDH among patients with lower creatinine value was statistically significant with greater incidence of IDH in lower creatinine group. Majority of our study population were frail, mostly due to malnutrition and the disease itself. Acute intradialytic changes in endothelial cell function have been proposed as etiologies for the increase in vascular resistance, although it is unclear if endothelin-1 or some other vasoconstrictive peptide is responsible. Chou et al[10] demonstrated imbalances in endothelial cell-derived mediators after dialysis in the IDH patients. Specifically, there were higher levels of the vasoconstrictor endothelin-1 (ET-1) and smaller ratios of the vasodilator nitric oxide to ET-1. Karakelleoglu et al[11] showed that the patients with malnutrition have higher endothelin-1 levels. Recent study by Park et al[12] indicate that lower creatinine levels in patients undergoing hemodialysis are associated with lower muscle mass, malnutrition, and mortality. These findings sup-

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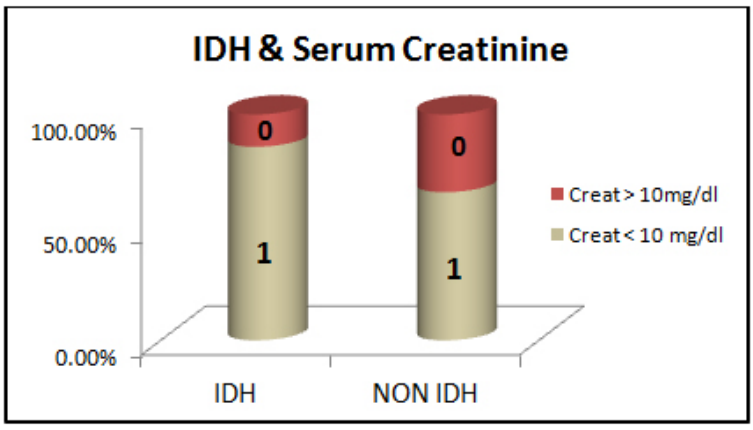
Graph 2: IDH and Serum Creatinine Co-Relation.



port that lower creatinine level which is a marker for malnutrition and is responsible for IDH through ET-1 mediated IDH.

A study by Sarkar SR et al[13] found an increased incidence of IDH among patients receiving IV erythropoietin therapy. A study by Abraham et al[14] found that 21% of patients had clinically important increases in BP during treatment of anaemia with erythropoietin. In a small investigation of the acute effects of erythropoietin-stimulating agent (ESA) in hemodialysis patients within 30 minutes following intravenous ESAs, there was a significant increase in ET-1 and a concomitant rise in mean arterial pressure (MAP) which was not demonstrated in patients given subcutaneous ESA or placebo.[12] In addition, 53% (10/19) of patients given intravenous ESA, had an increase in MAP>10 mmHg in the intradialytic period. Thus, if ESA is given intravenously, prior to the end of hemodialysis, it is possible that this may contribute to development of IDH in susceptible patients. It is possible that vasoconstriction, arising due to improved tissue oxygenation may result in IDH in some patients. In our study, incidence of IDH was higher in patients receiving IV erythropoietin during hemodialysis sessions, which was found to be statistically significant. This result was similar to studies done by Abraham et al[14] and Buckner et al.[15]

A significant relationship was also found between incidence of IDH and presence of oedema, with greater incidence of IDH in oedematous patients secondary to volume overload. Similar findings were shown in studies by Inrig et al[9] and Agarwal et al.[16] It was also observed that BP may paradoxically rise with ultrafiltration, when patients are volume overloaded. In a study by Inrig et al[9] it was found that incidence of IDH was higher in patients having low serum albumin levels. Similar results were found in our study. The mechanism of IDH may be due to reduced blood viscosity causing high cardiac output and increased peripheral vascular resistance.

In the Inrig et al[9] study, it was found that IDH incidence was greater in hypertensive patients who were prescribed greater number of anti-hypertensive drugs compared to those who were given standard regimen. In our study, incidence of IDH was more with patients receiving more than two anti-hypertensive drugs than patients receiving less than two anti-hypertensive drugs but it was statistically not significant. It is studied in literature that there is an association between dialysate to serum sodium gradients and BP increase during dialysis in patients with IDH, although it is unclear if this is related to endothelial cell activity or acute osmolar changes. In addition to probing the dry weight of patients with intradialytic hypertension, other management strategies include lowering dialysate sodium and changing anti-hypertensives to include carvedilol or other poorly dialyzed anti-hypertensives will help to reduce IDH.[17] All patients in our study were prescribed similar dialysis prescription to remove this confounding factor, and all patients of IDH were prescribed non-dialyzable anti-hypertensives for treatment of IDH. Except angiotensin receptor blocker (ARB) and angiotensin-converting enzyme (ACE) inhibitors, most of other drugs are dialyzed and hence incidence of IDH is more when more drugs are prescribed.

A significant relationship was found between incidence of IDH and survival of CKD patients. Patients having IDH had a more frequent occurrence of deaths at one year (Figure 3). Inrig et al[18] in a cohort of 438 prevalent hemodialysis patients, demonstrated that systolic BP elevations of more than 10 mmHg with hemodialysis are associated with a 20% increased odds of death or hospitalization at 6 months. Inrig et al[9] also demonstrated that increasing systolic BP in incident hemodialysis patients was associated with poorer 2-year survival.

CONCLUSION

Incidence of IDH in our study was 34.51% which was higher than what was found in the African study.[3] Only one Indian study showed incidence of 49% in 100 patients studied.[4] This suggests incidence of IDH is more in Indian population. Prognosis of CKD patients was worse among IDH group as 38.77% patients died within 1 year. We found a statistically significant relation between incidence of IDH and serum creatinine level, use of IV erythropoietin, oedema, and serum albumin level. However, no

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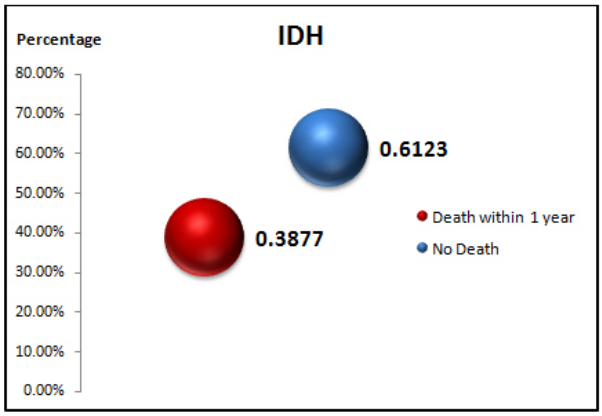
Graph 3: Results Showing Death within 1 Year of IDH.



significant relation was found between incidence of IDH and age of the patient, sex of the patient, hypertension, diabetes mellitus and number of anti-hypertensive drugs.

IDH is preventable if we control modifiable risk factors such as avoiding use of IV erythropoietin, reducing interdialytic weight gain, correction of serum albumin. This will reduce cardiovascular morbidity and mortality in CKD patients on maintenance hemodialysis. This is the first study in India which showed factors responsible for IDH and impact of IDH on mortality in Indian population. The other Indian study[4] has studied only incidence of IDH without studying factors responsible for IDH and its impact on mortality. No studies in literature till date evaluated the role of previous hypertension or diabetes with incidence of IDH. There was no association found between IDH and hypertensive or diabetic status of patient in our study. Higher incidence of IDH in patients with lower creatinine is a novel finding which needs to be confirmed with multicenter trials. Thus, IDH is an important cardiovascular event in Indian hemodialysis population which contributes to increased mortality in patients on hemodialysis although its exact pathogenesis is not known. Measures should be taken to reduce its incidence in hemodialysis patients.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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Case Report

Fusarium Peritonitis an Uncommon Complication in a Patient on Continuous Ambulatory Peritoneal Dialysis - A Case Report

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ABSTRACT

Fungal Peritonitis is a serious complication of treatment with peritoneal dialysis, with high rates of morbidity and mortality. In majority of the cases cause of fungal peritonitis is *Candida* species, with *Candida albicans* predominating. Infections by *Fusarium* species can be superficial or limited to single organs in otherwise healthy patients. In contrast, disseminated fusariosis affects the immunocompromised host. Fusarium infection is uncommon cause of peritonitis among patients on Continuous Ambulatory Peritoneal Dialysis [CAPD]. Here, we report a case of peritonitis due to *fusarium* species in a patient on Continuous Ambulatory Peritoneal Dialysis. Fusarium infection in patients on CAPD can be life threatening

Key words: *Fusarium;* Fungal peritonitis, Continuous Ambulatory Peritoneal Dialysis [CAPD].

INTRODUCTION

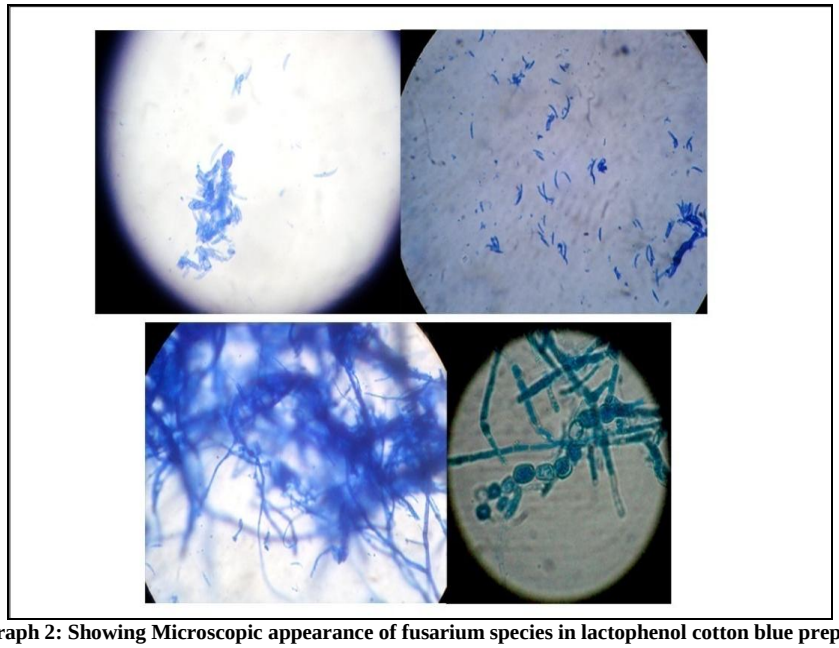
Peritonitis is the main complication of continuous ambulatory peritoneal dialysis. Fungal Peritonitis accounts for 1 - 16 % episodes in various studies. [1-3] Patients with previous bacterial peritonitis and antibiotic usage are at greater risk of developing fungal peritonitis. [3] Predominant cause of Fungal Peritonitis is Candida species. [2,4,5] The genus Fusarium is a common soil saprophyte and important plant pathogen that causes a broad spectrum of human disease, including mycotoxicosis, and infections which can be superficial, locally invasive or disseminated. [6] Fusariosis is an invasive mold infection associated with *Fusarium* species, most commonly *F. solani*. The skin and respiratory tract are the primary portals of entry. Localized skin infections may occur at sites of trauma in immunocompetent hosts. [7] Fusarium infection in immunocompromised patients has been reported in various studies. [8,9] Fusarium infection is uncommon cause of peritonitis among patients on CAPD. [4] This report presents the first known case of *Fusarium* peritonitis in a patient on CAPD in MGM Medical College, Aurangabad.

CASE REPORT

A 27 year young female patient had been on CAPD for one & half years. She presented in Medicine OPD of our Institute with H/o fever, vomiting, cough, pain in epigastric region & decreased urine output four days prior to hospitalization in 14 March 2013. She was a known case of chronic kidney disease with hypertension with hypothyroidism. She was on treatment for hypertension and hypothyroidism for last one year duration.

The diagnosis of peritonitis was based on clinical manifestations. She had not documented any episode of peritonitis prior to admission in our Institute. She was non diabetic. In the hospital she received inj metronidazole 400 mg TDS for 5 days, inj ceftazidime 1 gm after each cycle of peritoneal dialysis, inj Vancomycin 1 gm every 48 hrs, followed by Tab metronidazole 400 mg TDS, tab rifaximin 400 mg TDS & Tab fluconazole 150 mg once a day and vancomycin powder locally QID prior to availability of fungal culture report. Analysis of CAPD fluid was carried out, which showed total leucocyte count of 100 cells per mm. [3] It showed predominant [80%] polymorphs. The percentage of lymphocytes in CAPD fluid was 20%.

Photograph 1: showing growth of fusarium species on Sabraoud's dextrose agar.



Photograph 2: Showing Microscopic appearance of fusarium species in lactophenol cotton blue preparation

We received CAPD fluid of this patient for Gram stain, routine bacterial culture & fungal culture in Microbiology department. No organism could be detected on gram staining of the specimen. Routine culture was negative for growth of bacterial pathogen. Direct microscopic examination of CAPD fluid revealed no fungal element but culture of CAPD fluid on Sabraoud's agar without cycloheximide yielded growth which was identified on the basis of their macroscopic [photograph1] and microscopic appearance. Microscopic examination of colony showed presence of sickle shaped multicelled microconidia having 3 - 5 septae typical of fusarium species [photograph 2].

The patient was discharged with treatment advised which included inj fluconazole 200 mg on alternate day, inj vancomycin 1 gm after 48 hrs intra

peritoneally, in addition to other supportive treatment. She was advised to continue CAPD and come for follow up after 7 days. While on treatment, patient died due to sepsis in May 2013.

DISCUSSION

Peritoneal dialysis has been shown to be practical, safe & cost effective alternative to chronic haemodialysis. Bacterial peritonitis is most commonly encountered in these patients. The definition of CAPD peritonitis includes at least two of the following criteria: symptoms or signs (or both) of peritonitis, a cloudy dialysate (effluent) and a positive culture (and / or Gram stain of the dialysate). [10] The criteria for diagnosis of fungal peritonitis do not differ from those of bacterial peritonitis. The isolation of fungal organism on gram stain and or culture is diagnostic of fungal peritonitis. [4] Patients with previous bacterial peritonitis and antibiotic usage are at greater risk of developing fungal peritonitis. [3] Various studies report that fungal peritonitis accounts for 1-16 % episodes of peritonitis in patients on peritoneal dialysis. [1-3] *Fusarium* species are commonly found as saprophytes on organic debris & in soil. [11] *Fusarium* species cause a broad spectrum of infections in humans, including superficial, locally invasive, and disseminated infections. The clinical form of fusariosis depends largely on the immune status of the host and the portal of entry of the infection. [8] The principal portal of entry for Fusarium *spp*. is the airways, followed by the skin at site of tissue breakdown and possibly the mucosal membranes. [8] The duration of peritoneal dialysis treatment before the diagnosis of fungal peritonitis in our patient is also similar to the range reported by other studies [1,12] In our study Gram staining of the fluid revealed no organisms this finding is in concordance with study by Joseph et al [13] The organism has a propensity to attach to foreign bodies such as intravascular and intraperitoneal catheters. Therefore, successful treatment of infections caused by *Fusarium* may require catheter removal in addition to systemic antifungal therapy. [1,12,13] Prasad et al in their study reported that abdominal pain, abdominal pain with fever, and catheter in situ are the most commonly noted risk factors for mortality. [4,14] Fungal peritonitis, though uncommon, has great morbidity and is more difficult to treat successfully than bacterial peritonitis. In present study, the patient died of sepsis.

CONCLUSION

Fungal agents cause significant morbidity and mortality in patients with CAPD peritonitis and are usually more difficult to treat. Fusarium infection in patients on CAPD can be life threatening Fungal infections may be clinically suspected on the basis of clinical and laboratory findings, which should lead to prompt therapy.

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Role of Iron Deficiency Anemia in Patients with Chronic Kidney Disease

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Abstract:
Background: *Chronic kidney disease (CKD) is a worldwide health problem. CKD is a progressive condition and ultimately end up with kidney failure. A normocytic, normochromic anemia is observed in CKD. The primary cause in patients with CKD is insufficient production of erythropoietin (EPO) by the diseased kidneys. Anemia is both a complication of CKD as a part of uremic syndrome and a risk factor which influences the adverse outcomes of CKD, So evaluation and management of anemia is important to prevent the progress of CKD and for the general well being of the patient. As the renal function worsens, there is a progressive increase in the percentage of CKD patients with anemia.*
Aims and Objectives*: To determine the prevalence of iron deficiency anemia in patient with chronic kidney disease and study the effect of iron deficiency anemia on survival of CKD patients.*
Results: *In present study the overall prevalence of iron deficiency anemia in chronic kidney disease was 42.63% whereas in males, the prevalence of iron deficiency anemia was 44.4% which was less than female patients (55.6%). The observed values of iron deficiency anemia in CKD in relation to age group, hypertension and type of iron therapy have been found satistically nonsignificant, however iron deficiency anemia outcome with sex group, stage of CKD, diabetes and dialysis therapy were found to be satistically significant.*
Conclusion: *Iron deficiency anemia is common in CKD patients (42.63%). Functional Iron deficiency is seen in 39.03%. Iron deficiency is related to stage of CKD, Sex, Diabetes mellitus, erythropoietin therapy and dialysis therapy. There was no relation of Iron deficiency anemia with age, hypertension, and type of iron therapy. However mortality was not related to iron deficiency in CKD patients.*
Keywords: *Chronic kidney disease, Iron deficiency anemia, Hemodialysis, Erythropoietin.*

I. Introduction

Chronic kidney disease is a significant cause of morbidity and mortality world wide[1]. In India, there is a rising incidence and prevalence of kidney failure, with poor outcomes and high cost. The hallmark of CKD is structural and functional damage of the glomeruli of the kidney. The most important outcomes of this kidney damage are loss of kidney function and cardiovascular disease leading to premature death. In CKD, Erythropoietin (EPO)is produced in the peritubular cells of the kidney and is the major hormone involved in the production of red blood cells (erythropoiesis) when erythrocyte cells are produced. Anaemia starves the body of Oxygen and causes decreased capacity, cognitive impairment, and diminished quality of life[2]. Management of anemia in chronic kidney disease involves use of EPO. However EPO is effective only when iron is available in sufficient quantity. Hence evaluation of iron status in patients with chronic kidney disease is very vital. This study is under taken to study haematological profile of iron deficiency anemia in chronic kidney disease.

Defining CKD[3] as Kidney damage for 3 months or more as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifests by either : a) Pathological abnormalities detected by histopathological studies; or b) Markers of kidney damage, including abnormalities in the composition of the blood or urine , or abnormalities in imaging tests. GFR less than 60ml/min/1.73 m^2 for 3 months or more, with or without kidney damage.

Fishbane et al. found high rates of iron deficiency in adult men (57.8 to 58.8%) and women (69.9 to 72.8%) with CKD stages 3-5 in the NHANES 3 and 1999-2004 survey[4].

The prevalence of anemia in stage 3-5 CKD patients aged >64 in 2007-2010 NHANES survey was 24.4%. The prevalence of CKD in the SEEK-India cohort was observed to be 17.2% with 6% have CKD stage 3 or worse. Prevalence of CKD stages 1, 2, 3, 4 and 5 was 7%, 4.3%, 4.3%, 0.8% and 0.8% respectively[5].

National kidney foundation kidney dialysis outcomes quality initiative
(NKF K/DOQI 2012) DEFINES IRON DEFICIENCY ANEMIA[6, 7] :
1. **Absolute Iron Deficiency:** Ferritin <100 ng/ml and TSAT <20%.
2. **Functional Iron Defeciency:** Ferritin > 100 ng/ml and TSAT <20%.

Iron deficiency is common in patients with CRF. 25-37.5% patients of CRF have iron deficiency[8]. The cause's are : Decreased iron absorption as a part of uremic syndrome, loss of RBCs and Fe due to bleeding tendencies in uremic syndrome, dialysis related loss of RBCs and Fe, blood loss due to frequent blood tests.

Serum ferritin reflects body stores of iron[9]. A level of <100 ng/ml indicates absolute iron deficiency in CKD. But this is not very sensitive; it can reflect depleted stores only when the depletion is very low. Serum ferritin values < 30ng/ml indicate severe iron deficiency and are highly predictive of absent iron stores in bone marrow. It is not very specific as serum ferritin is also an acute phase reactant and elevated in conditions such as hyperthyroidism, inflammation/infection, hepatocellular disease, malignancies, alcohol consumption and oral contraceptives[10]. Transferrin saturation reflects the amount of available iron for erythropoiesis. In iron deficiency, elevated transferrin levels maintain the circulating iron pool despite the marked decrement in Tsat. A level of <20% in CKD indicates absolute iron deficiency. Transferrin saturation is decreased only when serum ferritin is decreased in absolute iron deficiency.

The ESRD National Cooperative Anemia Project, a personal communication, J. Wish, April;1997 - 60% of patients were found to be iron deficient. Recent analysis of the National Health and Nutrition Examination Survey 4 suggests that up to 50% of patients with CKD stages 2-5 have absolute or relative (functional) iron deficiency. In CKD, both absolute and relative iron deficiency are common[11].

Aims and Objectives :
- To determine the prevalence of iron deficiency in patient with chronic kidney disease.
- To study the effect of iron deficiency anemia on survival of CKD patients.

II. Material And Methods

It is an open non randomized prospective, cross sectional observational study to determine the prevalence of iron deficiency anemia with CKD and the effect of IDA on survival of CKD patients. The data obtained was studied on 9 parameters and chi-square test was applied and P value was calculated to the attributes to test their significance at 5 % level of significance.
No. Of cases: 190

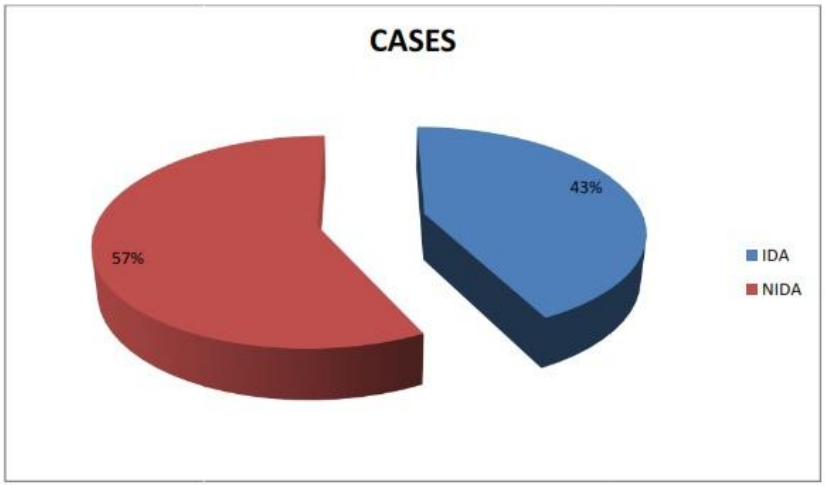
Case definition: Patient was defined as having Iron deficiency anemia in chronic kidney disease if there was: Serum ferritin >100ng/ml and transferring saturation <20%.

Inclusion criteria: All cases diagnosed as chronic kidney disease.

Exclusion criteria: Cases of acute kidney injury, bleeding diathesis, acute bleeding (urological or gastro enterological).
Methodology : The patients under study evaluated as per protocol.
1- **History :** Age, sex, history of hypertension / diabetes mellitus, EPO therapy/ Iron therapy, Hemodialysis.

2. Laboratory investigations :
a) Hemoglobin
b) Serum creatinine
c) Iron studies (serum iron, serum ferritin, TIBC, Transferrin saturation)

Results: Pia chart : showing prevalence of iron deficiency anemia (IDA)



Out of 190 patients, 81(43%) patients were found to have iron deficiency anemia whereas 109(57%) patients were NON-IDA.

Table 1 : Association of different parameters (satistically significant) with iron deficiency anemia (IDA) :

PARAMETERS	IDA	NIDA	TOTAL	CHI-SQUARE	P- VALUE
SEX : MALE	36(44.4%)	73(66.9%)	109	9.6427	0.001 S
: FEMALE	45(55.6%)	36(33.1%)	81	S (>3.84)	(<0.05)
DIABETES	30(37%)	23(21.1%)	53	5.8676	0.015 S
NON-DIABETIC	51(63%)	86(78.9%)	137	S	
EPO THERAPY	55(67.9%)	56(51.4%)	111	5.224	0.022 S
NON-EPO	26(32.1%)	53(48.6%)	79	S	
HEMODIALYSIS	70(86.4%)	78(71.6%)	148	5.9593	0.014 S
NON-HD	11(13.6%)	31(28.4%)	42	S	

The observed values of iron deficiency anemia in CKD in relation to sex group, diabetes and erythropoietin therapy and hemodialysis therapy have been found satistically significant.

Table 2: Association of different parameters (satistically nonsignificant) with iron deficiency anemia (IDA)

PARAMETERS	IDA	NIDA	TOTAL	CHI- SQUARE	P- VALUE
AGE: 21-40	20(24.69%)	26(23.85%)	46	0.0373	0.9981 S
: 41-60	30(37.03%)	40(36.69%)	70	NS	(>0.05)
: > 60	31(38.28%)	41(39.46%)	74	(<3.84)	
HYPERTENSION	54(66.7%)	83(76.1%)	137	2.0763	0.14 NS
NON-HTN	27(33.3%)	26(23.9%)	53	NS	
IRON : ORAL	10(12.34%)	20(18.34%)	30		
: I. V.	27(33.33%)	43(39.445)	70	2.7376	0.6026 NS
NOT ON IRON	44(54.32%0	46(42.22%)	90	NS	
CKD STAGE : 4	8(10.1%)	6(5.7%)	14	1.2485	0.2616 NS
: 5	71(89.9%)	99(94.3%)	170	NS	
DIED	27(33.3%)	36(33%)	63	0.002	0.9612 NS
ALIVE	54(66.7%)	73(67%)	127	NS	

The observed values of iron deficiency anemia in CKD in relation to age group, hypertension and type of iron therapy, CKD staging and prognosis have been found satistically non-significant.

III. Discussion

This study was undertaken in view of prevalence of iron deficiency anemia in chronic kidney disease patients. In the present study, prevalence of iron deficiency anemia, factors responsible for it and its effect on survival of chronic kidney disease patients was studied. Total 190 patients diagnosed as chronic kidney disease were studied. Out of 190 patients, 81 (42.63%) patients were found to have iron deficiency anemia (IDA). The observed values of iron deficiency anemia in CKD in relation to age group, hypertension and type of iron therapy have been found satistically non significant, however iron deficiency anemia outcome with sex group, stage of CKD, diabetes and dialysis therapy were found to be satistically significant.

According to a study by Bowling CB, Inker LA et al[12] , Prevalence of iron deficiency anemia was found more in elderly age group. In this study, contradictory results are found probably because of more young patients developing CKD in Indian population.

According to a study by Singh et al[5] . Prevalence of iron deficiency anemia is higher in males than females. In this study, prevalence of iron deficiency anemia is higher in females than males.

In a a study previously published by New JP, Aung T, Baker PG et al[13] and Fishbane et al[14]. Prevalence of iron deficiency anemia was found to be higher in patients with Diabetes Mellitus. In this study, Prevalence of iron deficiency anemia is found to be higher in patients with Diabetes Mellitus, which is similar to previous studies. According to a study by James B. Post et al[15] and Saul Nurko et al[16] , there was increased prevalence of iron deficiency anemia among the patients receiving erythropoietin. In the present study, there is a direct relation between erythropoietin therapy and IDA i.e. prevalence of IDA is higher in patients receiving erythropoietin therapy, which is similar to previous studies.

According to a study by Melissa E. Stauffer, Tao Fan et al[17] , Prevalence of anemia increased with stage of CKD, from 8.4% at stage 1 to 53.4% at stage 5. In the present study, prevalence of iron deficiency anemia is increased with stage of CKD, which is similar to previous studies.

In a study previously published by Allen R. Nissenson and Jur Strobes et al[18] , 60% of patients were found to be iron deficient on hemodialysis. In our study, There is a direct relation between Hemodialysis and IDA i.e. prevalence of IDA is higher in patients on Hemodialysis, which is similar to previous studies.

IV. Conclusion

Iron deficiency anemia is common in CKD patients (42.63%). Functional Iron deficiency is seen in 39.03%. Iron deficiency is related to stage of CKD, Sex, Diabetes mellitus, erythropoietin therapy and dialysis therapy. There was no relation of Iron deficiency anemia with age, hypertension, and type of iron therapy. However mortality was not related to iron deficiency in CKD patients.

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Study of Clinical Profile and Prognostic Factors of Acute Kidney Injury (AKI) In Tertiary Referral Centre in Marathwada

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Abstract: *Mortality in acute kidney injury (AKI) remains impressively high despite technical and medical advances made following the advent of dialysis. Previous studies of prognosis in AKI have analyzed the influence of demographic factors, severity of AKI, nature of diseases causing AKI, coexisting diseases, treatment received and complications. Several other factors also affect prognosis of AKI viz. oliguria, a rise in serum creatinine greater than 3 mg%, older debilitated patients, multiorgan failure, associated comorbid conditions, need of dialysis, suspected or proven sepsis. Hence cases of AKI were studied at tertiary health centre in Marathwada region of Maharashtra to study the clinical profile and factors affecting the prognosis of acute kidney injury.*

Keywords: *Acute Kidney Injury (AKI), Dialysis, Oliguria.*

I. Introduction

The kidney is a highly vascular organ. The kidneys are important organs of our body, which deals with the excretion of the waste products of protein catabolism from the body and maintenance of water and electrolyte balance. The portion of the total cardiac output that passes through the kidneys, called as renal fraction is about 20%. Kidneys are prone to develop ischemic injury whenever blood supply to them is decreased. They are also susceptible to nephrotoxic injury by virtue of their rich blood supply and the ability to concentrate toxins in the medullary interstitium and renal epithelial cells.

Acute kidney injury (AKI), previously known as acute renal failure (ARF), is a syndrome characterized by rapid decline of glomerular filtration rate (hours to weeks), retention of nitrogenous waste products and perturbation of extracellular fluid volume and electrolyte and acid base homeostasis[1].

The term failure reflects only part of the spectrum of damage to the kidney that occurs clinically. In most cases of damage, the reduction in kidney function is modest. Nevertheless, this modest change has been documented to be associated with negative effects on outcome, albeit not nearly as ominous as seen with large decreases in kidney function associated with frank kidney failure that often requires acute dialysis therapies. Furthermore, the term renal is not well understood in the general population and this makes communication with patients and family more challenging; hence "kidney" has replaced "renal". Hence the name acute renal failure was changed to "acute kidney injury."[1]

Urine output is generally reduced to < 400 ml/day called as oliguric AKI but some patients continue to pass > 400 ml of urine per day, called as non-oliguric AKI. In AKI nitrogenous waste products accumulate in the body and renal mechanisms responsible for maintaining water and electrolyte balance is disturbed. In addition the renal contribution to the control of acid base balance is deficient; hydrogen ions accumulate in the body producing metabolic acidosis. Loss of excretory function leads to hyperkalemia and oedema, with or without pulmonary oedema, if dietary intake of potassium and water is not restricted.

AKI is defined as any of the following[2]:

1. Increase in Serum Creatinine by 0.3 mg/dl within 48 hours; or
2. Increase in Serum Creatinine to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
3. Urine volume < 0.5 ml/kg/h for 6 hours.

It is one of the most common clinical syndrome encountered in the clinical practice. Most AKI is reversible, the kidneys being relatively unique among major organs in its ability to recover in function.

Acute Kidney Injury is sub classified into three categories, viz prerenal, intrinsic renal and post renal failure. Prerenal failure the most common form and is characterized by renal hypoperfusion without compromising to integrity of renal parenchyma. Intrinsic renal failure is produced by disorders that directly involve renal parenchyma. These are sepsis, ischemic, various nephrotoxins and diseases of glomeruli. Post renal failure is produced by urinary tract obstruction[1].

As there are no specific therapies for treatment of ischemic and nephrotoxic AKI and mortality is so high, prevention is of paramount importance. Early identification of patients at risk with prompt elimination of potential insults is the golden rule. Aggressive restoration of intravascular volume has been shown to reduce the incidence of ARF dramatically in volume depleted states. Injudicious use of nephrotoxic drugs like aminoglycosides and NSAIDs should be avoided especially in elderly and in combination with diuretics. Dose modification should be done when renal failure has already developed. Maintenance of volume status to optimum during operative and post operative periods is very important.

Sepsis is by far the commonest cause of death in AKI, so all steps should be taken to avoid or limit it. Many a times, doctors are responsible for development of AKI in hospitals, with a little more awareness about the precipitating factors, avoidance of the injudicious use of nephrotoxic drugs, proper maintenance of intake and output records in post operative cases and a watchful readiness to act promptly if unavoidable circumstances arises, can prevent and minimize the number of AKI cases. Factors that predispose AKI are renal insult, myeloma, diabetes mellitus, proteinuria, previous cardiac or renal insufficiency, diuretic use, volume depletion, advanced age.[3]

Mortality rate among patients of AKI approximates 50 percent and mortality rate vary greatly, depending on the cause of AKI[4].

Several factors affect prognosis of AKI viz oliguria, a rise in serum creatinine greater than 3 mg%, older debilitated patients, multiorgan failure, associated comorbid conditions, need of dialysis, suspected or proven sepsis[5]. Hence cases of AKI were studied at Tertiary Health Centre in Marathwada region of Maharashtra MGM Medical College and Hospital, Aurangabad to study the clinical profile and factors affecting the prognosis of acute kidney injury.

Observations

Table – 1 Age And Gender Distribution

Age Group	Male	Female	Total
0-20	6(4.16)	5(3.47)	11(7.63)
21-40	21(14.58)	20(13.88)	41(28.47)
41-60	36(25)	16(11.11)	52(36.11)
≥ 61	25(17.36)	15(10.41)	38(26.38)
Total	88(61.11)	56(38.88)	144(100)

Figures in ( ) indicates percentage.

Graph – 1 Age & Gender Distribution

Male preponderance was seen in all age groups.

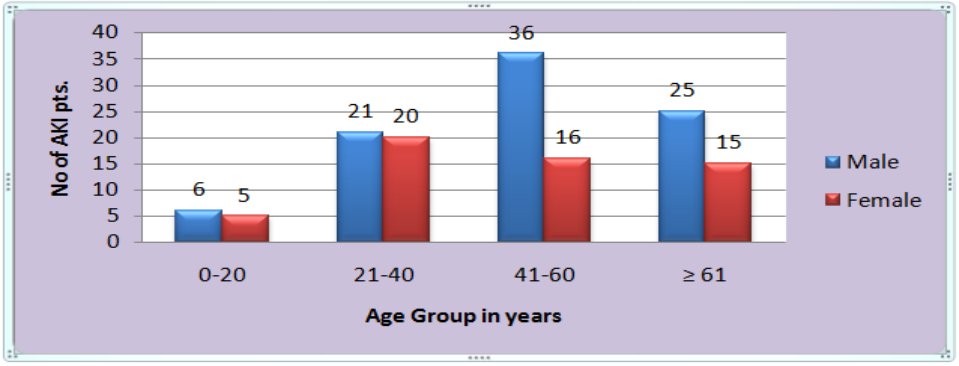
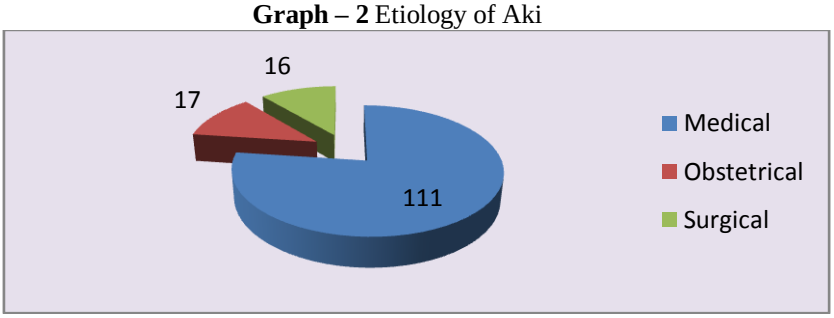


Table 2 – Etiology Of Aki

Etiology of AKI	No. Of pts.	Percentage (%)
Medical AKI	111	77.08
Obstetric AKI	17	11.80
Surgical AKI	16	11.11
Total	144	100

Graph – 2 Etiology of Aki



The commonest cause of AKI was Medical (77.08%), followed by Obstetrical (11.80%) and Surgical causes (11.11%).

Table - 3 Clinical And Laboratory Data In 144 Patients Of Aki

Parameters	Mean ± SD, Ratio
Age (years)	48 ± 18.24
Sex (M: F)	88:56 (1.57:1)
Oliguria/Nonliguria	61/83
Hypotension	41 (28.47%)
Bleeding tendency	11 (7.63%)
Hyperkalemia	40 (27.77%)
Peak Blood Urea (mg %)	131.50 ± 69.94
Peak serum Creatinine (mg %)	4.96 ± 3.23
Mortality	29.16 %

Out of 144 patients of AKI studied, male to female ratio was 1.57:1. Mean age was 48 ± 18.24. Overall mortality was 29.16 %.

Table – 4 Causes Of Oliguric V/S Nonoliguric Aki

Causes	Oliguric patients (n=61)	Non-oliguric patients (n=83)
Surgical	11(18.03)	5(6.02)
Obstetrical	5(8.19)	12(14.45)
Medical	45(73.77)	66(79.51)
Medical causes		
Sepsis	16(35.55)	20(30.30)
Acute Gastroenteritis	6(13.33)	12(18.18)
Acute Pancreatitis	7(15.55)	10(15.15)
ContrastInduced Nephropathy	5(11.11)	5(7.57)
Hepatorenal Syndrome	6(13.33)	3(4.54)
Malaria	2(4.44)	3(4.54)
Dengue Fever	1(2.22)	4(6.06)
Snake Bite	2(4.44)	1(1.51)
Leptospirosis	0(0)	2(3.03)
HUS	0(0)	1(1.51)

Figures in ( ) indicates percentage.

Non-oliguric AKI was more common than Oliguric AKI. Non-oliguric AKI was seen in all etiologies.

Table - 5 Age And Medical Etiology Of Aki

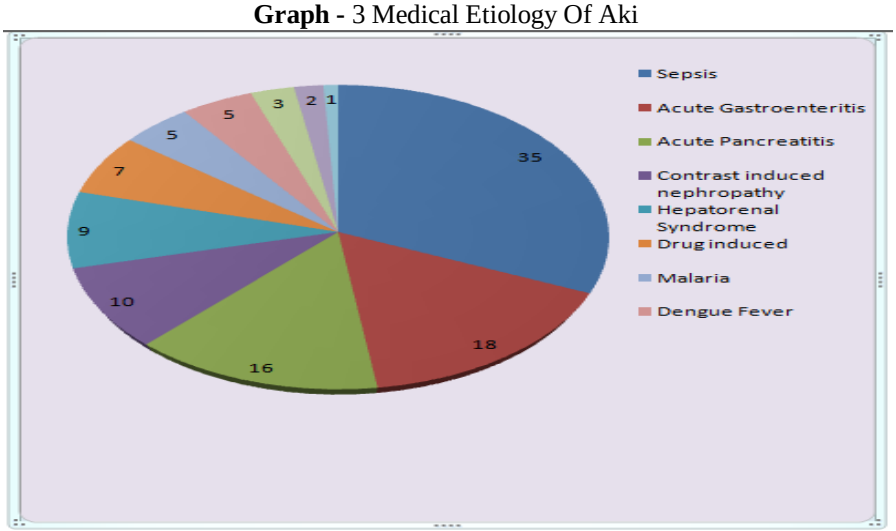
Age	0-20	21-40	41-60	≥ 61	Total	Percentage (%)
Sepsis	0(0)	6(17.14)	12(34.28)	17(48.57)	35(31.53)	31.53
Acute Gastroenteritis	2(11.11)	3(16.66)	8(44.44)	5(27.77)	18(16.21)	16.21
Acute Pancreatitis	0(0)	10(62.5)	6(37.5)	0(0)	16(14.41)	14.41
Contrast Induced Nephropathy	0(0)	1(10)	4(40)	5(50)	10(9)	9
Hepatorenal Syndrome	1(11.11)	0(0)	4(44.44)	4(44.44)	9(8.10)	8.10
Drug Induced	1(14.28)	0(0)	5(71.42)	1(14.28)	7(6.30)	6.30
Malaria	1(20)	3(60)	1(20)	0(0)	5(4.50)	4.50
Dengue Fever	0(0)	2(40)	1(20)	2(40)	5(4.50)	4.50
Snake Bite	1(33.33)	1(33.33)	1(33.33)	0(0)	3(2.70)	2.70

Leptospirosis	0(0)	0(0)	2(100)	0(0)	2(1.80)	1.80
HUS	1(100)	0(0)	0(0)	0(0)	1(0.69)	0.69
Total	7(6.30)	26(23.42)	44(39.63)	34(30.63)	111(100)	100

Figures in ( ) indicates percentage.

AKI secondary to sepsis and contrast induced nephropathy were common in age group more than 61 years. AKI secondary to acute pancreatitis was more common in age group 21-40.

Graph - 3 Medical Etiology Of Aki



Sepsis was the commonest cause of medical AKI (31.53%). Other causes of medical AKI were as follows: Acute Gastroenteritis (16.21%), Acute pancreatitis (14.41%), Contrast induced nephropathy (9%), Hepatorenal syndrome (8.10%), Drug induced (6.30%), Malaria (4.50%), Dengue fever (4.50%), Snake bite (2.70%), Leptospirosis (1.80%), HUS (0.69%).

Table-6 Age And Etiology Of Aki

Age	Medical	Obstetric	Surgical	Total
0-20	7 (4.86)	4(2.77)	0(0)	11(7.63)
21-40	26(18.05)	13(9.02)	2(1.38)	41(28.47)
41-60	44(30.55)	0(0)	8(5.55)	52(36.11)
≥ 61	34(23.61)	0(0)	6(4.16)	40(27.77)
Total	111(77.08)	17(11.80)	16(11.11)	144(100)

Figures in ( ) indicates percentage.

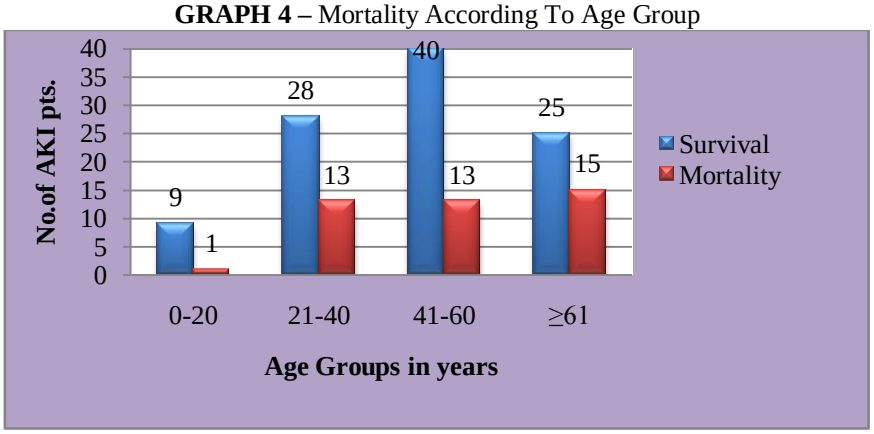
AKI due to medical and surgical causes were more common in patients with age more than 41 years. AKI due to obstetric causes was more common in age group 21-40 years.

Table – 7 Relation Of Age Distribution To Mortality

Age (yrs)	Survival	Mortality	Total patients	Percentage Mortality
0-20	9(90)	1(10)	10(6.94)	10
21-40	28(68.29)	13(31.70)	41(28.47)	31.70
41-60	40(75.47)	13(24.52)	53(36.80)	24.52
≥ 61	25(62.5)	15(37.5)	40(27.77)	37.5
Total	102(70.83)	42(29.16)	144(100)	29.16

(Chi square=2.79) $p > 0.05$ (non-significant)

GRAPH 4 – Mortality According To Age Group



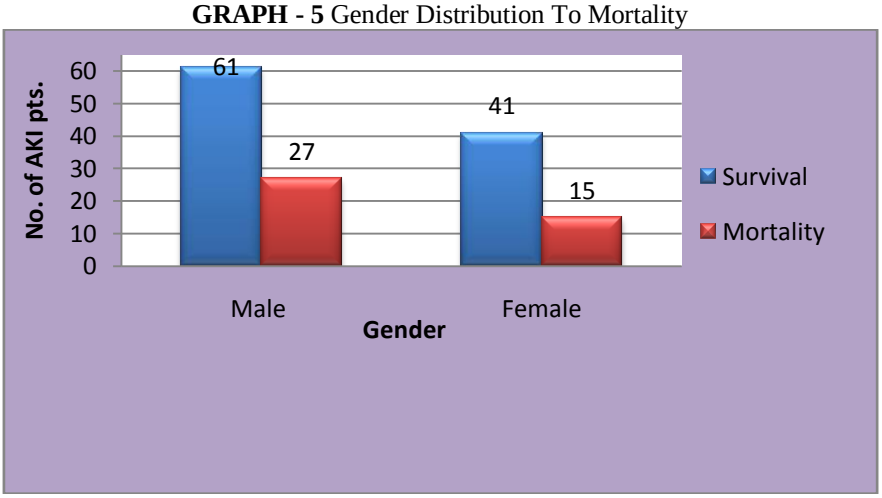
Although the mortality was maximum (37.5%) in age group more than 61years as compared to other age groups, chi square test showed no difference in mortality among various age groups. Overall mortality was 29.16 %.

TABLE – 8: Relation Of Gender Distribution To Mortality

Gender	Survival	Mortality	Total patient	Percentage Mortality
Male	61(69.31)	27(30.68)	88(61.11)	30.68
Female	41(73.21)	15(26.78)	56(38.88)	26.78
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =0.2517) p > 0.05 (non-significant)

GRAPH - 5 Gender Distribution To Mortality



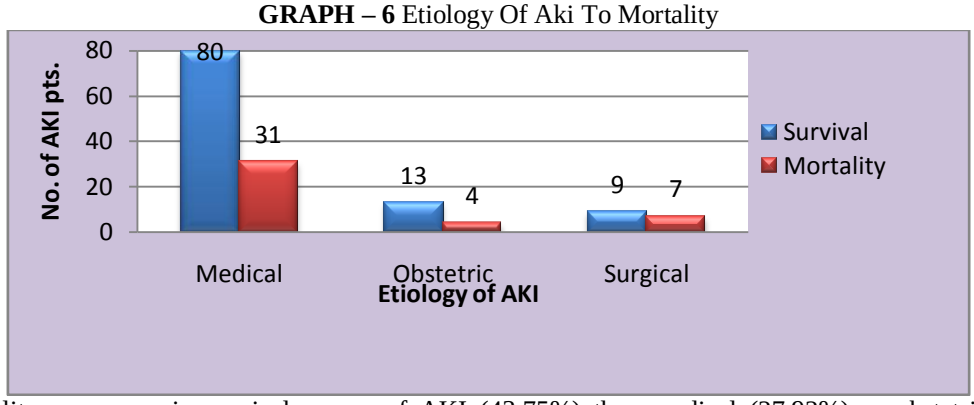
Although mortality was more in males (30.68%) than females (26.78%) but statistically there was no significant difference in mortality.

TABLE – 9: Relation Of Etiology Of Aki To Mortality

Etiology	Survival	Mortality	Total Patients	Percentage Mortality
Medical	80(72.07)	31(27.92)	111(77.08)	27.92
Obstetric	13(76.47)	4(23.52)	17(11.80)	23.52
Surgical	9(56.25)	7(43.75)	16(11.11)	43.75
Total	102(70.83)	42(29.16)	144(100)	29.16

Figure in ( ) indicates percentage (Chi square =1.995) p > 0.05 (Non Significant)

GRAPH – 6 Etiology Of Aki To Mortality



The mortality was more in surgical causes of AKI (43.75%) than medical (27.92%) or obstetrical causes (23.52%) of AKI, but the difference in mortality among these three groups was statistically not significant.

TABLE – 10: Relation Of Medical Causes Of Aki To Mortality

Etiology	Survival	Mortality	Total	Percentage Mortality
Sepsis	25(71.42)	10(28.57)	35(31.53)	28.57
Acute Gastroenteritis	17(94.44)	1(5.55)	18(16.21)	5.55
Acute Pancreatitis	8(50)	8(50)	16(14.41)	50
Contrast Induced Nephropathy	5(50)	5(50)	10(9)	50
Hepatorenal Syndrome	6(66.66)	3(33.33)	9(8.10)	33.33
Drug Induced AKI	6(85.71)	1(14.28)	7(6.30)	14.28
Malaria	4(80)	1(20)	5(4.50)	20
Dengue fever	3(60)	2(40)	5(4.50)	40
Snakebite	3(100)	0(0)	3(2.70)	0
Leptospirosis	2(100)	0(0)	2(1.80)	0
HUS	1(100)	0(0)	1(0.90)	0
Total	80(72.07)	31(27.92)	111(100)	27.92

Among medical causes of AKI highest mortality was seen in AKI secondary to Acute Pancreatitis (50%) and Contrast induced nephropathy (50%) followed by Hepatorenal syndrome (33.33%). Mortality in sepsis induced AKI was 29.57%. Overall mortality in medical causes of AKI was 27.92%. High mortality in contrast induced nephropathy was due to associated multiple comorbid conditions were present in 70% of contrast induced nephropathy cases.

TABLE – 11: Relation Of Obstetric Causes Of Aki To Mortality

Etiology	Survival	Mortality	Total Patients	Percentage Mortality
Puerperal sepsis	6(66.66)	3(33.33)	9(52.94)	33.33
Eclampsia	4(100)	0(0)	4(23.52)	0
PPH	1(50)	1(50)	2(11.76)	50
Abortion	2(100)	0(0)	2(11.76)	0
Total	13(76.47)	4(23.52)	17(100)	23.52

Figures in ( ) indicates percentage. (Chi square =3.1207) $p > 0.05$ (non-significant)

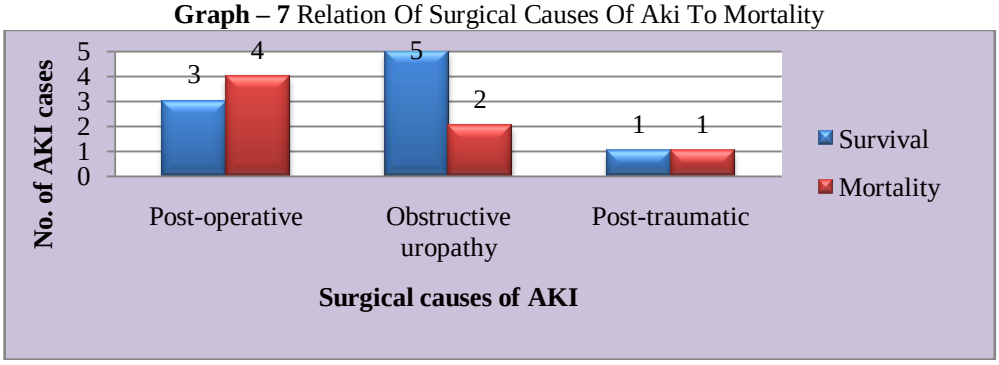
Puerperal sepsis was the major cause of Obstetric AKI (52.94%). Among obstetric causes maximum mortality was seen in PPH (50%). But the difference in mortality among various causes of obstetric AKI was statistically non-significant. Overall mortality in obstetric AKI was 23.52%.

TABLE – 12: Relation Of Surgical Causes Of Aki To Mortality

Etiology	Survival	Mortality	Total	Percentage Mortality
Postoperative	3(42.85)	4(57.14)	7(43.75)	57.14
Obstructive Uropathy	5(71.42)	2(28.57)	7(43.75)	28.57
Post-traumatic	1(50)	1(50)	2(12.5)	50
Total	9(56.25)	7(43.75)	16(100)	43.75

Figures in ( ) indicates percentage (Chi square =2.47) $p > 0.05$ (non-significant)

Graph – 7 Relation Of Surgical Causes Of Aki To Mortality



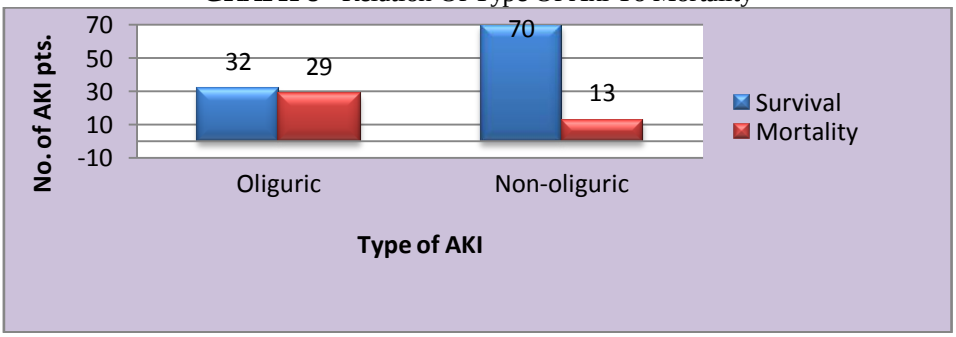
Mortality was more in post-operative cause of surgical AKI (57.14%) than other causes of surgical AKI, but it was statistically non-significant. The overall mortality in surgical group of AKI was 43.75%.

TABLE – 13: Relation Of Type Of Aki To Mortality

Type of AKI	Survival	Mortality	Total Patients	Percentage Mortality
Oliguric	32(52.45)	29(47.54)	61(42.36)	47.54
Non-oliguric	70(84.33)	13(15.66)	83(57.63)	15.66
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =17.28) $p < 0.001$ (Significant)

GRAPH 8 - Relation Of Type Of Aki To Mortality



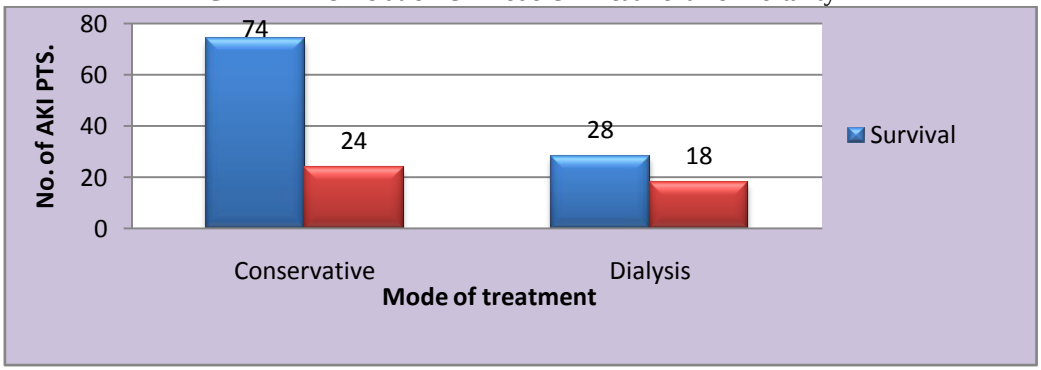
Non-oliguric AKI (57.63%) was more common than oliguric AKI (42.36%). Mortality was more in oliguric AKI (47.54%) than in non-oliguric AKI (15.66%). This difference in mortality among oliguric and non-oliguric AKI was statistically significant.

TABLE – 14 Relation Of Mode Of Treatment To Mortality

Mode of treatment	Survival	Mortality	Total	Percentage Mortality
Conservative	74(75.51)	24(24.49)	98(68.05)	24.49
Dialysis	28(60.87)	18(39.13)	46(31.94)	39.13
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =3.24) $p < 0.05$(Significant)

GRAPH – 9 Relation Of Mode Of Treatment To Mortality



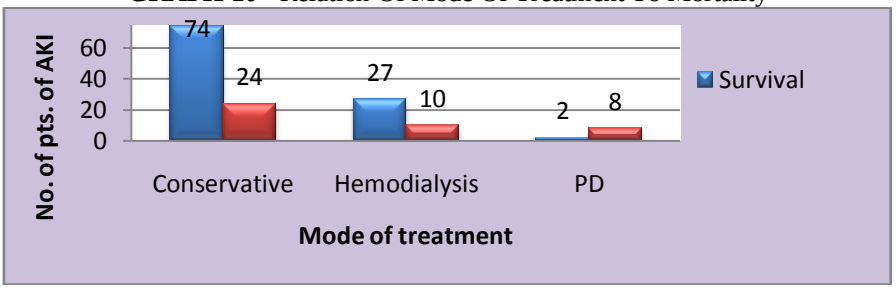
Mortality in patients who needed dialysis was 39.13% while mortality in patients who were treated conservatively was 24.49%. This difference in mortality was statistically significant. Thus mortality was more in patients who needed dialysis.

TABLE – 15: Relation Of Mode Of Treatment To Mortality

Mode of treatment	Survival	Mortality	Total Patients	Percentage Mortality
Conservative	74(75.51)	24(24.49)	98(68.05)	24.49
Hemodialysis	27(72.97)	10(27.02)	37(25.69)	27.02
Peritoneal Dialysis	2(20)	8(80)	10(6.94)	80
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =13.66) $p < 0.001$ (Significant)

GRAPH 10 - Relation Of Mode Of Treatment To Mortality



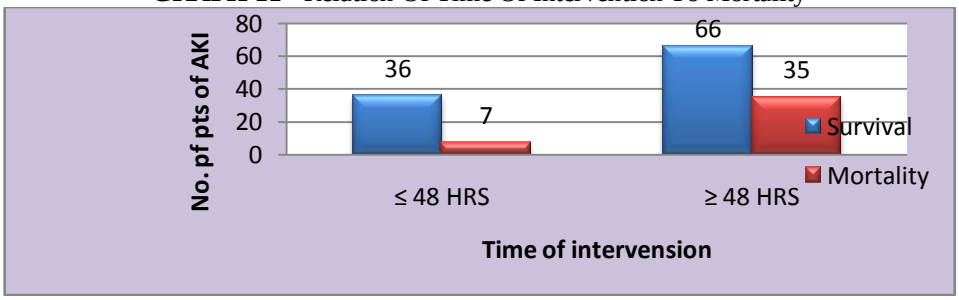
Majority of patients were treated conservatively (68.05%). Mortality was more in patients treated with peritoneal dialysis (80%) than in patients treated conservatively (24.49%) and with hemodialysis (27.02%). This different in mortality with different modes of treatment was statistically significant. This was because in our institute peritoneal dialysis was considered in patients who were hemodynamically not suitable for hemodialysis.

TABLE – 16 Relation Of Time Of Intervention To Mortality

Time	Survival	Mortality	Total Patients	Percentage Mortality
Within 48 hrs	36(83.72)	7(16.27)	43(29.86)	16.27
After 48 hrs	66(65.34)	35(34.65)	101(70.13)	34.65
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =4.94) $p < 0.05$ (Significant)

GRAPH 11 - Relation Of Time Of Intervention To Mortality



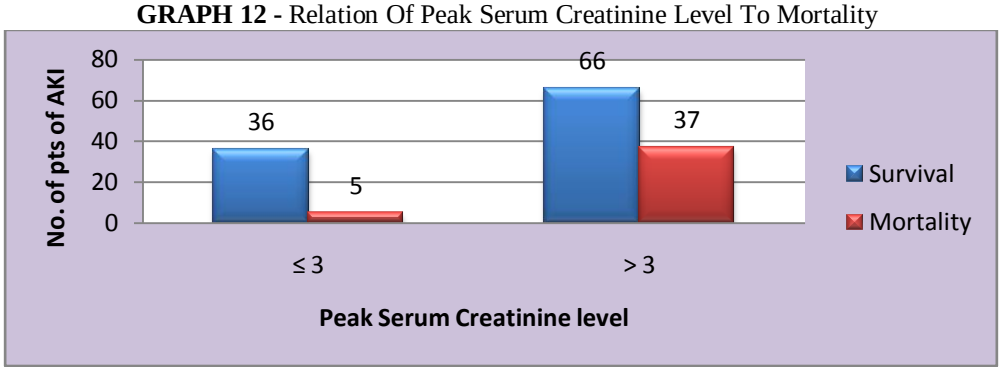
Majority of patients were treated after 48 hrs of diagnosis of AKI. Mortality was more in patients treated after 48 hours of diagnosis of AKI (34.65%) as compared with patients treated within 48 hours of diagnosis of AKI (16.27%). This difference in mortality due to time of intervention was statistically significant.

TABLE – 17: Relation Of Peak Serum Creatinine Level To Mortality

Peak Serum Creatinine (mg %)	Survival	Mortality	Total Patients	Percentage Mortality
< 3	36(87.80)	5(12.19)	41(28.47)	12.19
≥ 3	66(64.07)	37(35.92)	103(71.52)	35.92
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =7.98) $p < 0.05$ (Significant)

GRAPH 12 - Relation Of Peak Serum Creatinine Level To Mortality



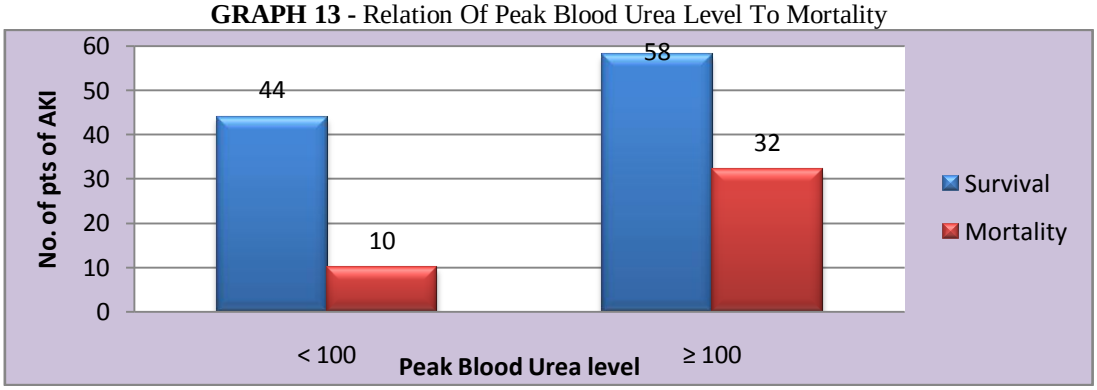
Majority of patients had peak serum creatinine more than 3 mg % (71.52 %). Mortality was more in patients who had peak serum creatinine more than 3 mg % (35.92%) than in patients who had peak serum creatinine less than 3 mg% (12.19%). This difference in mortality was statistically significant.

TABLE – 18: Relation Of Peak Blood Urea Level To Mortality

Peak Blood Urea level (mg %)	Survival	Mortality	Total Patients	Percentage Mortality
< 100	44(81.48)	10(18.51)	54(37.5)	18.51
≥ 100	58(64.44)	32(35.55)	90(62.5)	35.55
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =4.74) $p < 0.05$ (Significant)

GRAPH 13 - Relation Of Peak Blood Urea Level To Mortality



Majority of patients had peak blood urea level more than 100 mg % (62.5 %). Mortality was more in patients who had peak blood urea level more than 100 mg % (35.55%) as compared to patients who had peak blood urea level less than 100 mg% (18.51%). This difference in mortality was statistically significant.

TABLE – 19: Relation Of Serum Sodium Level To Mortality

Serum Sodium (meq/L)	Survival	Mortality	Total Patients	Percentage Mortality
< 136	57(75)	19(25)	76(52.77)	25
≥ 136	45(66.17)	23(33.82)	68(47.22)	33.82
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =1.35) $p > 0.05$ (Non Significant)
Hyponatremia was seen in 76 patients (52.77 %). There was no difference in AKI mortality among patients with or without hyponatremia.

TABLE – 20: Relation Of Serum Potassium Level To Mortality

Serum Potassium level (meq/L)	Survival	Mortality	Total Patients	Percentage Mortality
≤ 5.0	75(72.81)	29(28.15)	103(71.52)	28.15
>5.0	27(67.5)	13(31.70)	40(27.77)	31.70
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage (Chi square =0.4924) $p > 0.05$ (Non Significant)
Hyperkalemia was seen in 40 patients (27.77 %). There was no difference in mortality in patients with or without hyperkalemia.

TABLE – 21: Relation Of Serum Bilirubin Level To Mortality

Serum Bilirubin level (meq/L)	Survival	Mortality	Total Patients	Percentage Mortality
< 2	73(73)	27(27)	100(69.44)	27
≥ 2	29(65.90)	15(34.09)	44(30.55)	34.09
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =0.7431) p > 0.05 (Non Significant)

Serum bilirubin more than 2 mg % was seen in 44 patients (30.55 %). Hyperbilirubinemia had statistically no significant effect on mortality.

TABLE – 22 : Relation Of Platelet Level To Mortality

Serum Platelet level (/mm3)	Survival	Mortality	Total Patients	Percentage Mortality
< 50,000	14(60.87)	9(39.13)	23(15.97)	39.13
≥ 50,000	88(72.72)	33(27.27)	121(84.02)	27.27
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi Square=0.7844) p > 0.05 (Non Significant)

Platelet count less than 50,000 was seen in 23 patients (15.97 %). There was no statistically significant difference in mortality in AKI patients with or without thrombocytopenia.

TABLE – 23: Relation Of Total Leucocyte Count To Mortality

Total Leucocyte Count(/mm3)	Survival	Mortality	Total Patients	Percentage Mortality
< 11000	30(73.17)	11(26.82)	41(28.47)	26.82
≥ 11000	72(69.90)	31(30.09)	103(71.52)	30.09
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =0.1501) p > 0.05 (Non Significant)

Leucocytosis (TLC≥ 11,000 / cmm) was seen in 103 patients (71.52 %). Mortality was more in patients with leucocytosis (30.09%) as compared with patients without leucocytosis (26.82%), but the difference in mortality was statistically insignificant.

TABLE – 24: Relation Of Bleeding Tendency To Mortality

Bleeding Tendency	Survival	Mortality	Total Patients	Percentage Mortality
Present	5(45.45)	6(54.54)	11(7.63)	54.54
Absent	97(72.93)	36(27.06)	133()	27.06
Total	102(70,83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =3.73) p < 0.05 (Significant)

Bleeding tendency was seen in 11 patients (7.63 %). Mortality was more in patients who had bleeding tendency (54.54%) as compared with patients without bleeding tendency (27.06%). This difference in mortality was statistically significant.

TABLE – 25: Relation Of Hypotension To Aki Mortality

Hypotension	Survival	Mortality	Total Patients	Percentage Mortality
Present	13(31.70)	28(68.29)	41(28.47)	68.29
Absent	89(86.40)	14(13.59)	103(71.52)	13.59
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =42.49) p < 0.001 (Significant)

Hypotension was seen in 41 patients (28.47 %). Mortality was more in patients who had hypotension (68.29%) than in patients without hypotension (13.59%). This difference in the mortality was statistically significant.

TABLE – 26: Relation Of Other Organ Involvement (Associated Comorbid Condition To Aki Mortality)

Other organ involvement	Survival	Mortality	Total Patients	Percentage Mortality
Present	36(53.73)	31(46.26)	67(46.52)	46.26
Respiratory	10(38.46)	16(61.53)	26(38.80)	61.53
Hepatic	22(62.85)	13(37.14)	35(52.23)	37.14
CNS	4(66.66)	2(33.33)	6(8.95)	33.33
Absent	66(85.71)	11(14.28)	77(53.47)	14.28
Total	102(70.83)	42(29.16)	144(100)	29.16

Figures in ( ) indicates percentage. (Chi square =17.73) $p < 0.001$ (Significant)
Associated comorbid conditions were seen in 67 patients (46.52%). Associated comorbid conditions like hepatic, respiratory failure or CNS involvement increased the mortality in AKI. This difference in mortality was statistically significant.

TABLE –27complications Related To Etiology Of Aki

Etiology	Hypotension	Bleeding Tendency	Hyperkalemia	Respiratory failure	CNS (Encephalopathy)
Medical					
Sepsis	13	2	9	9	2
Acute Gastroenteritis	0	0	6	0	3
Acute Pancreatitis	6	1	7	2	0
Contrast Induced Nephropathy	3	1	3	5	1
Hepatorenal Syndrome	4	2	1	0	0
Drug Induced	1	0	4	1	0
Malaria	1	0	0	1	0
Dengue Fever	1	1	1	1	0
Snake Bite	1	2	0	0	0
Leptospirosis	0	0	0	0	0
HUS	0	0	0	0	0
Obstetrical	5	2	6	2	0
Surgical	6	0	3	5	0
Total(144)	41(28.47%)	11(7.63%)	40(27.77%)	26(18.05%)	6(4.16%)

Hypotension (28.47%) was the commonest complication of AKI followed by hyperkalemia (27.77%).

TABLE – 28recovery Of Renal Function

Recovery	Total Patients	Percentage
Mortality	42	29.16
Incomplete recovery	6	4.16
Complete recovery	96	66.66

Most of the patients had complete recovery from AKI (66.66%). Incomplete recovery was observed in (6/144) 4.16% of patients.

II. Conclusion

Total 144 cases of AKI were studied at Tertiary Health Centre (MGM Medical College and Hospital, Aurangabad) of Marathwada region of Maharashtra, during the period from June 2011 to Nov 2013 to study clinical profile, etiology and prognostic factors of AKI.

Majority of cases were in the age group 41-60 years and male to female ratio was 1.57:1. Medical causes of AKI were more common than obstetrical and surgical. In medical causes sepsis was the most common cause. In medical causes, Acute Pancreatitis and Contrast induced nephropathy had the highest mortality (50%). High mortality in contrast induced nephropathy in present study was due to 70% patients of CI-AKI had associated multiple comorbid conditions, 80% patients of CI-AKI had baseline serum creatinine more than 3 mg% and 40% patients needed dialysis. The mortality was more in surgical AKI than medical and obstetric AKI, but the difference in mortality among surgical, medical and obstetrical AKI was statistically not significant. Sepsis and contrast induced nephropathy were more common in age more than 61 years. Hypotension, hyperkalemia, bleeding tendency and respiratory failure were the common complications observed. Non-oliguric AKI was more common than oliguric AKI. Non-oliguric AKI was seen in all etiologies. Mortality in oliguric AKI was more than nonoliguric AKI. Mean peak blood urea and Sr. creatinine level were 131.50 ± 69.94 mg% and 4.96 ± 3.23 mg% respectively. Overall mortality was 29.16%. Maximum mortality was seen in patients with age more than 61 years but there was no statistically significant difference in mortality among various age groups. Thus age was a weak determinant of mortality in AKI. Mortality was more in males than females however, this was not statistically significant. Thus gender was a weak determinant of mortality in AKI. In obstetrical AKI, Puerperal Sepsis was the commonest cause of AKI (52.94%). Overall mortality in obstetric cases was 17.64%. There was no significant statistical difference in mortality among obstetric causes of AKI. In surgical AKI, post-operative AKI and obstructive uropathy were the commonest causes of AKI (43.75%). Overall mortality in surgical causes was 43.75%. Among surgical causes maximum mortality was seen in postoperative causes of surgical AKI. But statistically there was no difference in mortality among various surgical causes of AKI. Most of the cases were treated conservatively (68.05%). The difference in mortality with various modalities of treatment i.e. conservative, peritoneal dialysis and hemodialysis was statistically significant. Mortality was more in patients who needed dialysis. Mortality was more in patients

treated with peritoneal dialysis than in patients treated conservatively and with hemodialysis. This was because in our institute peritoneal dialysis was considered in patients who were hemodynamically unstable for hemodialysis. Delay in the initiation of treatment was found to be important factor in deciding outcome of AKI. Patients who received treatment after 48 hours of onset of AKI had higher mortality than those who received treatment within 48 hours. Our institute is the tertiary referral centre. Patients are referred from various primary and secondary canters to our institute; therefore there was delay in the treatment of AKI. Peak serum creatinine ≥3 mg% and peak blood urea ≥100 mg% were associated with high mortality rate. In our institute patients come mainly from poor socioeconomic strata, therefore they were not affording for costly investigations like biomarkers of acute kidney injury. Hence peak serum creatinine and peak blood urea levels were still the primary investigations for prognostic indicators of AKI.Hypotension, Bleeding tendency were found to be significantly associated with high mortality. Presences of hyponatremia, hyperkalemia, increased serum bilirubin (≥2 mg %), low platelet count (≤50,000), leucocytosis were not found to be predictor of high mortality. Other organ involvement in the form of respiratory, liver, CNS and cardiac was associated with high mortality. Most of the cases had complete recovery from AKI. Thus the only factors which were statistically significant in deciding mortality were peak serum creatinine ≥ 3 mg%, peak blood urea ≥ 100 mg%, oliguric AKI, delay in initiation of treatment after onset of AKI (≥48 hours), bleeding tendency, hypotension, need of dialysis and associated other organ involvement. Thus most cases of the AKI are reversible, if the etiology is identified and treated early.

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A Study of Various Angioaccess in Haemodialysis Patients

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Abstract: *Contemporary societies are in the midst of an epidemic of chronic non communicable diseases including that of chronic kidney disease (CKD). India is no exception to this rule. Hemodialysis is still the main treatment given for patients of Chronic Kidney Disease, although renal transplantation is slowly changing the trends with better immunosuppression and better surgical techniques with wider acceptance of the masses. The provision of effective and timely hemodialysis requires a stable and reliable vascular access. Thus, vascular access is not only the obvious 'Achilles heel' of hemodialysis (HD) but it is also the quiet undercurrent of trends in patient outcomes. Data on the comparisons between various angioaccess are very limited in literature. So we undertook a study to compare the various angioaccess used for hemodialysis in patients of CKD.*
Key Word: *CKD- Chronic Kidney Disease, HD- Hemodialysis*

I. Introduction

Contemporary societies are in the midst of an epidemic of chronic non communicable diseases including that of chronic kidney disease (CKD). India is no exception to this rule. Many parts of India are undergoing rapid epidemiological transition as a consequence of economic and social changes.[1] The increase in non communicable diseases is real and not simply due to better diagnosis. This epidemiologic transition is partially attributable to better nutrition, control of infectious disease and gain in life expectancy. An untoward consequence of this extension of life is the emergence of chronic diseases as leading causes of death.[2]

Chronic kidney disease is a worldwide health problem. According to World Health organization (WHO) Global Burden of Disease project, diseases of the kidney and urinary tract contribute to global burden with approximately 850,000 deaths every year and 115,010,107 disability adjusted life years. CKD 12th leading cause of death and 17th cause of disability.[3] This global prevalence, however, may be grossly underestimated for a number of reasons. Patients with CKD are at high risk for cardiovascular disease (CVD) and cerebrovascular disease (CBVD), and they are more likely to die of CVD than to develop end-stage renal failure. Moreover, patients with CVD often develop CKD during the course of their disease, which may go unrecognized. Therefore, an unknown proportion of people whose death and disability attributed to CVD have kidney disease as well.[4]

Moreover, most epidemiological data (prevalence, incidence, patient demography, morbidity, and mortality) on CKD are derived from renal registries. However, most registries record data of patients who are at late stage of kidney disease. Much less is known about the prevalence of the earlier stages of the CKD. Indeed, it has been acknowledged that the majority of the individuals at early stages of CKD have gone undiagnosed and under treated.[4] The number of end stage of renal disease patients that need dialysis or renal transplantation increased in the world. The overall magnitude and pattern of chronic kidney disease (CKD) in India has been studied sporadically. The CKD Registry of India is a new initiative that has been started to document CKD and its course in our country.

Modern haemodialysis therapy started on 17 March 1943, when Willem Kolff, a young doctor in the small hospital of Kampen (The Netherlands), treated a 29-year-old housemaid suffering from malignant hypertension and 'contracted kidneys'. Kolff had constructed a 'rotating drum kidney' with the support of Mr Berk, the director of the local enamel factory. First, Kolff used only venipuncture needles to obtain blood from the femoral artery and to reinfuse it by puncturing a vein. Later, he performed surgical cut-down of the radial artery which caused severe bleeding during heparinization.

In the years that followed, substantial technical developments are linked to the names of Nils Alwall in Lund (Sweden) and John P. Merril in Boston (USA). In the 1950s, the technical devices were available for regular haemodialysis treatments, e.g. Kolff's so-called twin-coil kidney[5] but, the Achilles heel was a reliable access to the circulation for multiple use which did not yet exist. Vascular access is not only the obvious 'Achilles heel' of hemodialysis (HD) but it is also the quiet undercurrent of trends in patient outcomes.

It took another 16 years before Quinton and Scribner introduced the first permanent vascular access: the Scribner shunt.[6] This device consisted of 2 Teflon® tubes connecting the patient to the dialyser; one tube was inserted into a suitable peripheral artery and one into a suitable vein. After treatment, the circulatory access

was kept open by connecting the two tubes outside the body using a small U-shaped Silastic device over a stainless steel plate. The major disadvantages of Scribner shunts were high thrombosis and infection rates resulting in a limited shunt and hence patient life span.

In 1962, Cimino and Brescia reported on veno-venous access for HD which used a sphygmomanometer to dilate an accessible forearm vein and a blood pump, and in which blood was returned through another vein, usually in the ankle.[6] This experience led them to make one of the most important developments in HD - the arteriovenous fistula.[7] Even though this required a blood pump for dialysis, the blood access problem was solved and use of the shunt declined rapidly. In 1966, Brescia and Cimino solved the blood access problem with a surgically created arteriovenous fistula (AVF) between the radial artery and a vein.[7] This new vascular access was able to deliver flow rates of 250-300 mL/min for unlimited intervals. Results were satisfactory, 13 AVFs (87%) functioned without any complication and two failed before cannulation. Nowadays, the Brescia-Cimino (radio-cephalic) AVF is still the preferred type of vascular access.[8-9]

In 1961, Shaldon Higgs and Chiandussi introduced temporary HD catheters and these catheters continue to be the primary means of achieving acute hemodialysis access. The ready availability of the CVC as a vascular access (VA) for HD often makes them the access of choice, especially when urgent or emergent HD is required either at the time of initiation of renal replacement therapy or when a permanent access becomes dysfunctional.[10]

Central Venous Catheter remain an important method to obtain VA as a bridge to the placement and maturation of an arteriovenous fistula (AVF) or arteriovenous graft (AVG), pending renal transplantation, and as the sole access in many patients. The use of CVCs has several advantages in short term: It does not require the integrity of the peripheral blood vessels, a number of sites are available for immediate insertion, it can be used immediately and for prolonged periods, and it provides painless access.

A catheter conundrum' remains in existence where we hate catheters, but cannot live without them.[11] Thus, while advantageous in very short term, unavoidable and often necessary, CVC are a hazard in most other situations, especially if used for longer periods.

It has been convincingly argued that there is a disproportionately high use of Central venous catheters for dialysis in the US. According to the Dialysis Outcomes and Practice Patterns Study (DOPPS), CVC were the major type of vascular access for initiation of hemodialysis in the US in comparison to countries in Europe and in Japan.[12]

There is a lack of pre dialysis care by nephrologists in US and this seems to have an important correlation with the use of catheters as incident access. According to USRDS 2009 report, in year 2007, 43% of ESRD patients were not followed by a Nephrologist prior to the initiation of HD.[13] The early referral to nephrologist by primary care physicians, and an early referral to surgeon for fistula placement by nephrologists are the key interventions to improve incident CVC use. Preoperative vascular mapping can improve fistula placement rates, and perhaps the fistula maturation rates, which has the potential of reducing prevalent CVC rates.[14]

The provision of adequate hemodialysis is dependent on repeated and reliable access to central circulation. An ideal access delivers a flow rate adequate for the dialysis prescription, has a long use-life and has a low rate of complications (e.g. stenosis, thrombosis, aneurysms, limb ischemia, and infections). Although no current access type fulfils all of these criteria, the native arteriovenous (AV) fistula comes the closest to doing so.

For prevalent patients, it would be important to consider placing secondary AVF as a conscious strategy to reduce use of CVC in patients with failing primary access. It has been shown that a significant number of patients using CVC as their access have suitable veins for AVF creation.[15] Special attention should be paid to the patients using TDC on a 'permanent' basis, as a significant percentage of these patients tend to have suitable veins for AVF creation. Continual evaluation and patient education regarding their next access is extremely important, as it is a challenge for dialysis staff and nephrologists to convince patients to give up the "ease" of catheter use for the safety and long term benefits of an AVF or AVG.

Basic principles of using distal sites first and preferring autogenous fistulae over grafts are the mainstay of decision making. Among autogenous fistulae, direct fistulae, transpositions and translocations should be considered in that order with an aim of performing simpler and less-morbid procedures first. Lower limb and body wall sites should be considered after all upper limb options are exhausted. Use of non-dominant hand first holds true only when access opportunities are equal on both sides, otherwise hand with more suitable veins gets preference.[16]

Aims and Objectives

1. To study the percentages and frequencies of the various types of angioaccess used in our hospital.
2. To study the failure rates of various types of angioaccess in our hospital and associated co-morbidities.
3. To identify the prevalence of various complications of the angioaccess under consideration.

II. Materials And Methods

Study centre: Department of Nephrology and Renal Transplant Centre, Mahatma Gandhi Mission's Medical College & Hospital, Aurangabad.
Sample size: 211
Sampling technique: outpatient and inpatients visiting the hospital for dialysis
Duration of study: 2 ½ years

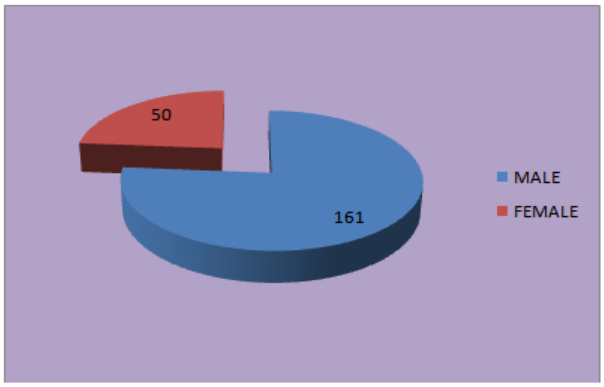
Study Design: observational prospective, single centre, non-randomized prospective study.
Inclusion criteria: Testing and evaluation was carried out on all OPD and IPD patients above the age of 18 years of chronic kidney disease and candidates on dialysis (maintenance or first time or emergency basis).
Exclusion criteria: All patients who were below the age of 13 years, those patients with acute renal failure and those with normal kidney function tests.

Methodology: Detailed history taking, regarding the condition: onset, duration, symptoms, first time diagnosed, personal history, co-morbid conditions, mode of angioaccess used, surgical measures undertaken such as AV Fistula creation.

- Assessment of patency and frequency of change of angioaccess and follow up along with complications.
- Assessment of serum creatinine at onset of dialysis.
- Assessment of eGFR was done by the Cockcroft-Gault equation.
- Chi square test was used to find any statistical significance amongst the groups under comparison.

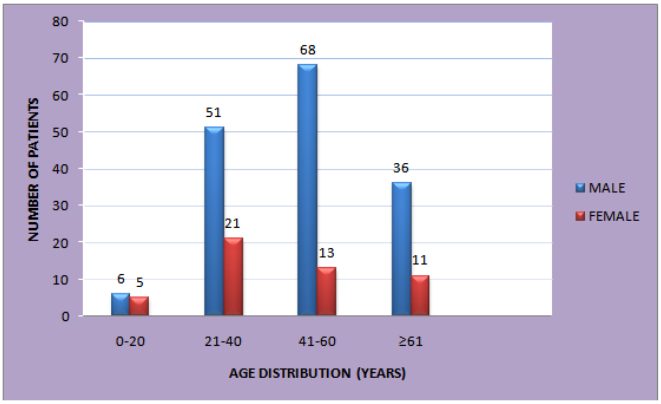
III. Observations

Graph 1 - Gender Distribution



In this study, total number of patients were 211. Of these, 161 (76.30%) participants were male and 50 (23.69%) were female. The Male : Female ratio was 161:50 = 3.22:1. Thus, a male preponderance was seen in our study.

Graph 2 - Age And Sex Distribution

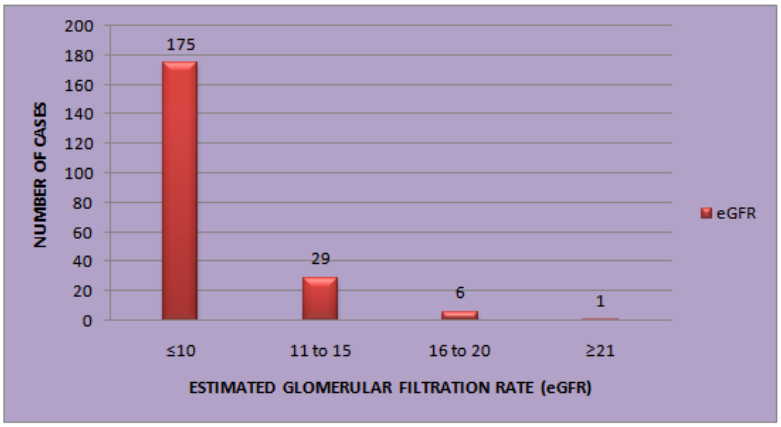


The mean age in the present study was 46.84 years with a range of 13-80 years. The maximum number of patients were from the 41-60 age group, i.e. 81/211(38.38%). The maximum number of male participants were from the 41-60 age group, i.e. 68/161 (42.23%). The maximum number of female patients were seen in the 21-40 age group, i.e. 21/50(42%). Male preponderance was seen as shown in the above graph.

Table No. 1 -Estimated Glomerular Filtration Rate (Egfr) In The Study Population

eGFR	NUMBER OF PATIENTS	PERCENTAGE %
≤10	175	82.93%
10-15	29	13.74%
15-20	6	2.84%
≥21	1	0.47%
TOTAL	**211**	

Graph-3 - Comparison Of Estimated Glomerular Filtration Rate (Egfr) In The Study Group

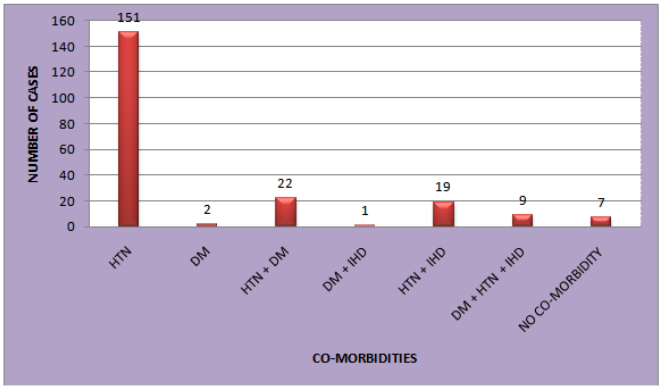


The maximum number of patients had eGFR ≤10, i.e 175 (82.93%) in the study population.

Table No.2 - Co-Morbidities Present In The Study Population

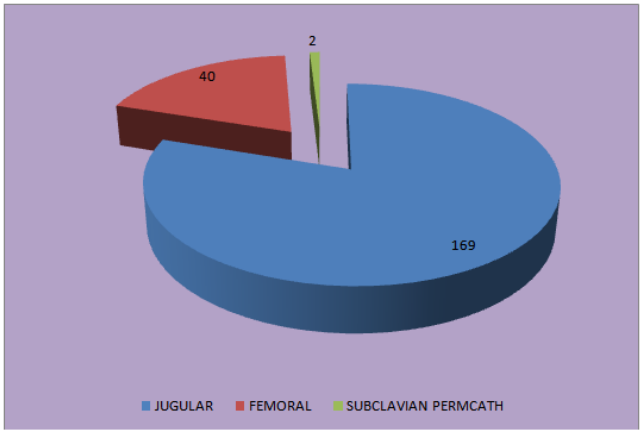
CO-MORBDITIY	NUMBER OF PATIENTS	PERCENTAGE %
HTN	151	71.56%
DM	2	0.94%
HTN + DM	22	10.42%
DM + IHD	1	0.47%
HTN + IHD	19	9.00%
DM + HTN + IHD	9	4.26%
NO COMORBIDITY	7	3.31%
TOTAL	**211**	**100%**

Graph 4 - Co-Morbidities Present In The Study Population



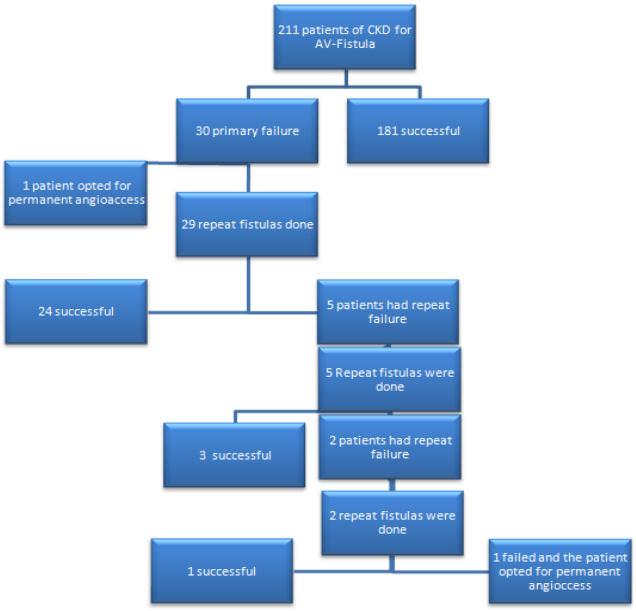
In the study population, 71.56% of the study population had hypertension as the single most common co-morbidity as compared to diabetes and ischemic heart disease.

Graph 5 - Type Of Artificial Angioaccess Used In Ckd Patients On Hemodialysis

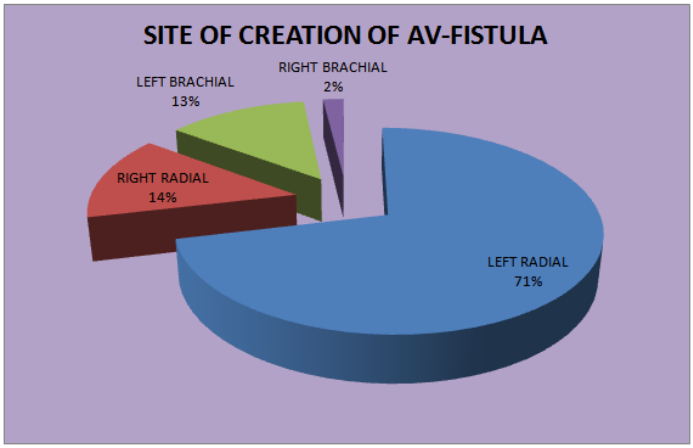


The total number of artificial angioaccess established was 211. In this study, the most commonly used angioaccess was the Internal Jugular 169 (80.09%) as compared to the femoral route 40 (18.95%) and subclavian permanent catheter 2(0.94%)

Flow-Chart



Graph 6 - Site Of Arterio-Venous Fistula Creation In Ckd Patients On Hemodialysis



The total number of AV-Fistulas created in 211 patients during the duration of the study was 248. Amongst these there were 39 documented primary failures and repeat fistulas were done. In one patient, after primary AV-fistula failure, the patient opted for permanent angioaccess. In a second patient, after 4 failed AV-fistulas, the patient opted for permanent artificial angioaccess. Thus only in 209 patients AV-fistula was used as the primary medium for CRRT. Out of 211 patients in whom AV-fistula was the access used for providing CRRT, 170 never had any primary failures. Thus there was a primary success rate of 80.56%(170/211). In this study, the preferred site for AV-fistula creation was the left radial; 149(71.29%) and the least preferred site was the right brachial with 4(1.9%).

Table No. 3 -Complications Of Artificial Angioaccess

COMPLICATION	JUGULAR (NO.)	FEMORAL (NO.)	TOTAL
INFECTION	14 (8.28%)	10 (25%)	24(11.48 %)
BLEEDING	17 (10.05%)	6 (15%)	23(11 %)
POOR FLOW	15 (8.87%)	8 (20%)	22(10.52%)

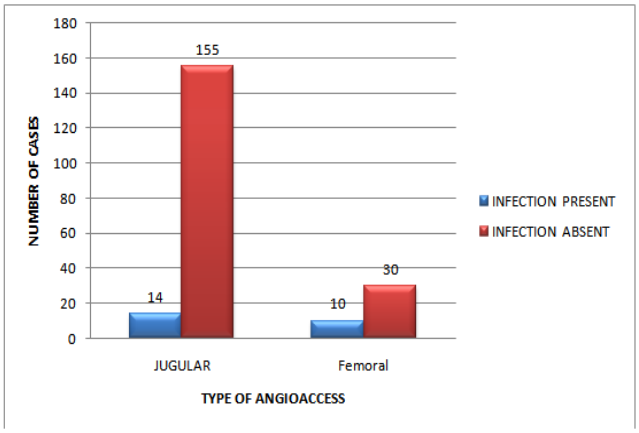
In the present study, infection was the commonest complication associated with use of artificial angioaccess in CKD patients. The incidence of complications were higher with femoral as compared with the internal jugular HD catheter.

Table No.4 - Relation Of Infection With Artificial Angioaccess.

ANGIOACCESS	INFECTION PRESENT	INFECTION ABSENT	TOTAL
JUGULAR	14	155	169 (8.28%)
FEMORAL	10	30	40(25%)
TOTAL	**24**	**185**	**209(11.48%)**

Chi square = 8.89 $p < 0.01$ (Significant)

Graph 7 - Relation Of Infection With Angioaccess



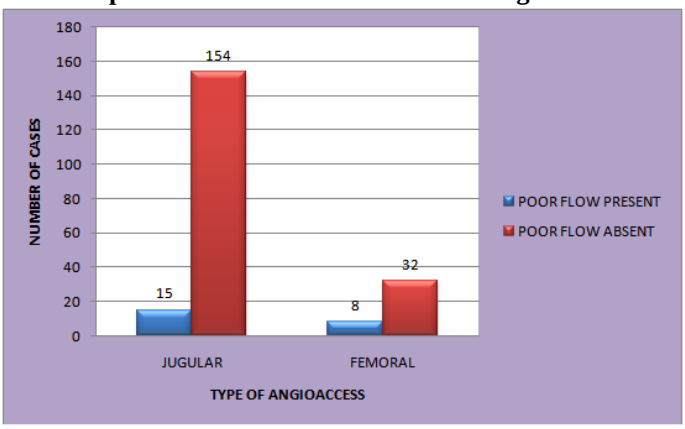
In this study, the total number of infections were 11.48% (24/209). It was noted that a significant higher rate of infection occurred amongst femoral HD catheters, i.e. 10/40 (25%), as compared to the jugular access 14/169 (8.28%). This increased percentage of infection in femoral angioaccess as compared to jugular angioaccess was statistically significant.

Table No.5 - Relation Of Poor Flow To Angioaccess

ARTIFICIAL ANGIOACCESS	POOR FLOW PRESENT (NO.)	POOR FLOW ABSENT (NO.)	TOTAL
JUGULAR	15	154	169 (8.87%)
FEMORAL	8	32	40 (20%)
TOTAL	**23**	**186**	**209(11%)**

Chi Square = 4.09 $p < 0.05$ (Significant)

Graph 8 - Relation Of Poor Flow To Angioaccess



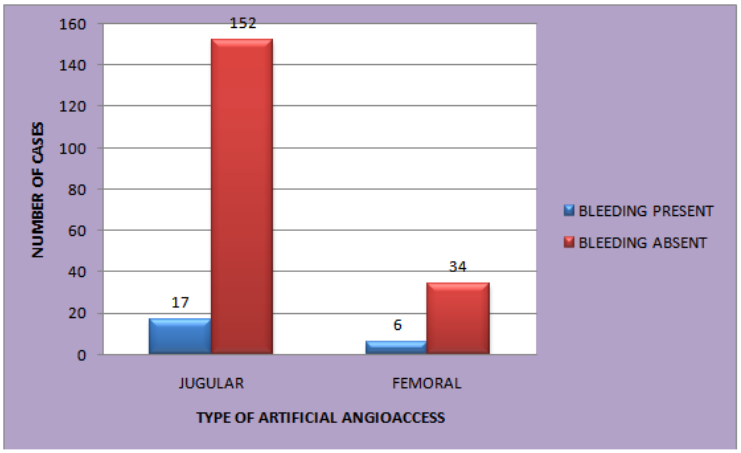
In the study, poor flow was recorded in 11.00% (23/209) of artificial angioaccess. A higher incidence of poor flow was associated with the femoral type, i.e. 8/40(20%) of artificial angioaccess as compared to the jugular 15/169 (8.87%), which was statistically significant.

Table No.6 - Relation Of Bleeding With Artificial Angioaccess

ANGIOACCESS	BLEEDING PRESENT (NO.)	BLEEDING ABSENT (NO.)	TOTAL
JUGULAR	17	152	169 (10.05%)
FEMORAL	6	34	40 (15%)
TOTAL	**23**	**186**	**209 (11 %)**

Chi Square= 0.806 p > 0.05 (not significant)

Graph 9 - Incidence Of Bleeding In Various Artificial Angioaccess



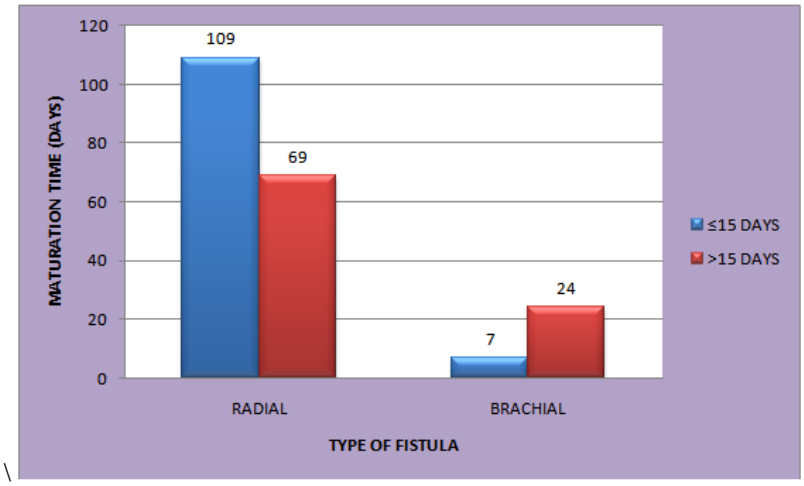
The overall incidence of bleeding was in 11.00% (23/209). Although the percentage of bleeding was more in femoral catheter (15%) as compared to jugular, (10.05%), it was not statistically significant.

Table No. 7 - Relation Of Fistula With Maturation

SITE OF AV-FISTULA	MATURATION ≤15 DAYS	MATURATION >15 DAYS	TOTAL
RADIAL	109	69	178 (38.76%)
BRACHIAL	7	24	31 (77.41%)
TOTAL	**116**	**93**	**209 (44.49%)**

Chi Square =15.9 p< 0.05 (Significant)

Graph 10 - Relation Of Fistula With Delayed Maturation



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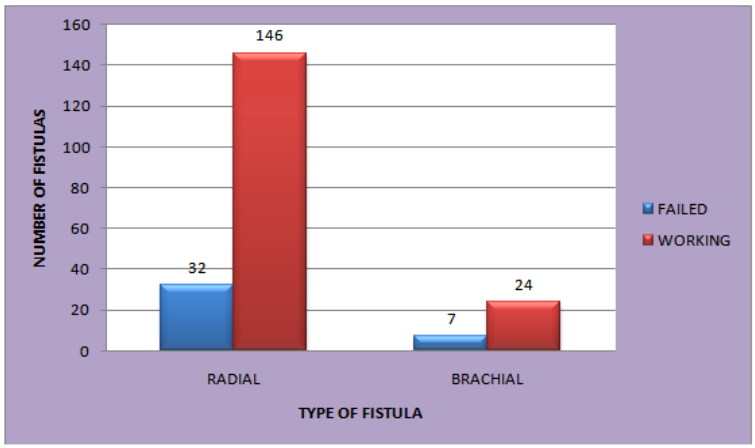
In the study, of 44.49% (93/209) of AV-Fistulas matured after 15 days. There was a higher significant incidence of delayed maturation (>15 days) with Brachial fistulas (77.41%) as compared to Radial fistulas (38.76%).

Table No.8 - Relation Of Site Of Fistula With Failure.

SITE OF AV-FISTULA	FISTULA FAILED ≥ 1 TIME	FISTULA NOT FAILED	TOTAL
RADIAL	32	146	178 (17.98%)
BRACHIAL	7	24	31(22.58%)
TOTAL	**39**	**170**	**209 (18.66%)**

Chi Square = 0.368 p>0.05 (Significant)

Graph 11 - Relation Of Site Of Fistula With Failure



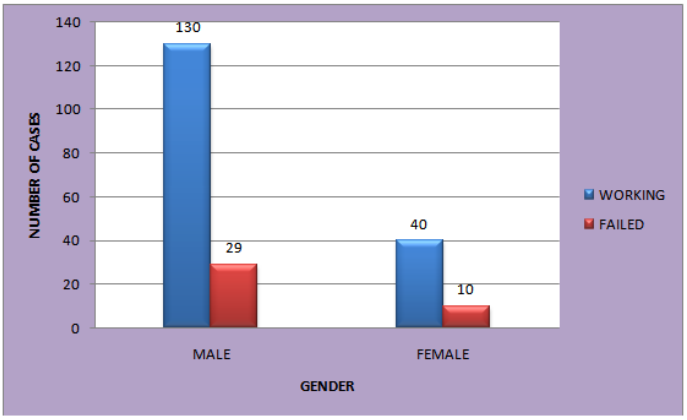
In the study, AV-Fistula failure was seen in 18.66% (39/209) of patients. Higher failure rates were noted with brachial fistulas, i.e. 7/31 (22.58%) as compared to radial AV-Fistulas32/178(17.98%), which was statistically significant.

Table No.9 - Fistula Failure Rate Comparison In Gender

GENDER	FISTULA FAILED (NO.)	FISTULA WORKING (NO.)	TOTAL (NO.)
MALE	29	130	159 (18.23%)
FEMALE	10	40	50 (20%)
TOTAL	**39**	**170**	**209 (18.66 %)**

Chi Square = 0.07 p > 0.05(not significant)

Graph 12 - Gender Comparison Of Av-Fistula Failure Rates



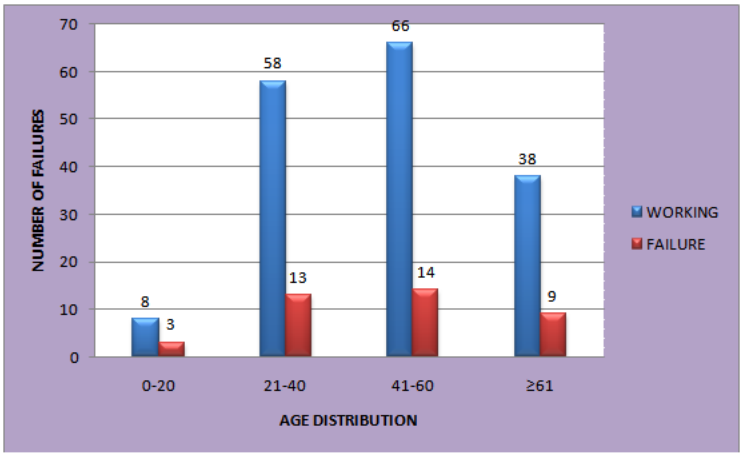
The AV-Fistula failure rate amongst males was 18.23% while that in females was 20%. Although the failure rate was more in females, there was no statistically significant difference in failure rates recorded between male and female gender.

Table No.10 - Relation Of Age Distribution To Failure Rates

AGE GROUP	FAILURE (NO.)	WORKING (NO.)	TOTAL
0-20	3	8	11 (27.27%)
21-40	13	58	71(18.31%)
41-60	14	66	80 (17.5%)
61-80	9	38	47 (19.14%)
TOTAL	**39**	**170**	**209 (18.66%)**

Chi Square = 0.59 p > 0.05 (not significant)

Graph 13 - Comparison of Av-Fistula Failure Rates In Various Age Groups



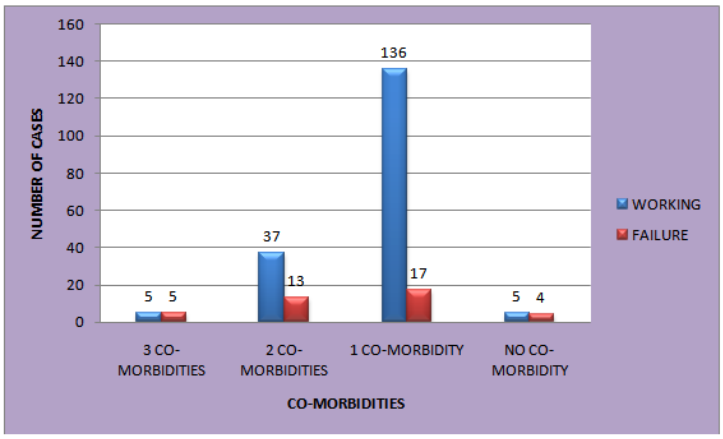
Although the failure rate was more in the age group of 0-20 years (27.27%) as compared to other age groups, it was not statistically significant.

Table No.11 - Relation Of Fistula Failure With Number Of Co-Morbidities

NUMBER OF CO-MORBIDITIES	FAILURE (NO.)	WORKING (NO.)	TOTAL
3 CO-MORBIDITIES	5	5	10(50%)
2 CO-MORBIDITIES	13	37	50(26%)
1 CO-MORBIDITY	17	136	153(11.11%)
NO CO-MORBIDITY	5	5	9(44.44%)
TOTAL	**39**	**183**	**222**

Chi Square = 18.65 p < 0.001(significant)

Graph 14 - Relation Of Number Of Co-Morbdities With Fistula Outcome

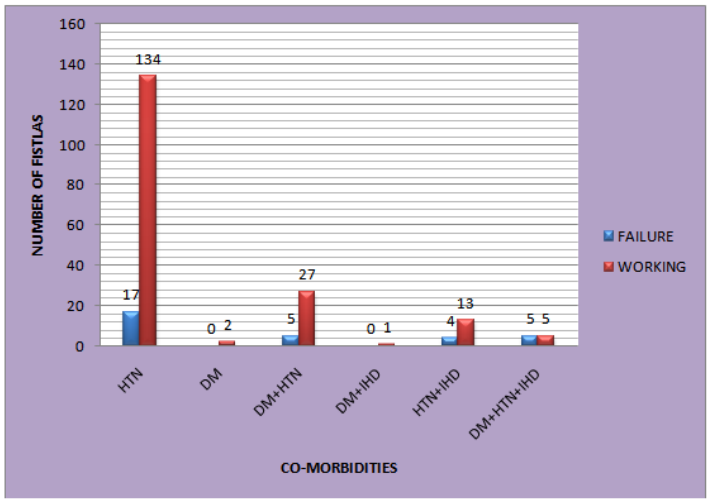


In the present study, the presence of 3 co-morbidities resulted in a greater incidence of failure 5/10 (50%) for AV-fistula. A significant incidence of failure in patients with no underlying co-morbidities (44.44%), but the number of subjects in that subset were small. The cause of failure was not evaluated.

Table No.12 - Relation Of Fistula Outcome With Co-Morbidities

CO-MORBIDITY	FISTULA FAILED (NO. OF CASES)	FISTULA WORKING (NO. OF CASES)	TOTAL
HTN	17	134	151 (11.25%)
DM	0	2	2 (0%)
HTN + DM	9	23	32 (28.12%)
DM + IHD	0	1	1(0%)
HTN + IHD	4	13	17 (23.52%)
DM + HTN + IHD	5	5	10 (50%)
NO CORMOBIDITY	4	5	9 (44.44%)
TOTAL	**39**	**183**	**222**

Graph 15 - Relation Of Co-Morbidities With Fistula Outcome



In the study, there was a significant higher incidence of failure in patients with co-existence diabetes, hypertension and ischemic heart disease - 5/10 (50%), as compared to each of these co-morbidities alone. Also noted were failure in 44% of patients with no co-morbidities, but the cause of failure was not further evaluated.

Table No.13 - Complications Of Av-Fistula

COMPLICATION	RADIAL (NO.)	BRACHIAL (NO.)	TOTAL
INFECTION	5 (2.80%)	2 (6.45%)	7 (3.34%)
BLEEDING	16 (8.98%)	1 (3.22%)	17 (8.13 %)
POOR FLOW	13 (7.30%)	4 (12.90%)	17 (8.13 %)

In the present study, poor flow was seen in 8.13% (17/209) fistulas. The complication of bleeding was similarly noted at 8.13%(17/209) and infection was less noted at 3.34%(7/209) of AV-Fistulas. There was greater incidence of infection (6.45% V/s 2.8%) and poor flow (12.90% V/s 7.30%) associated with brachial fistulas as compared to radial fistulas. The incidence of bleeding however was more with radial fistulas as compared to brachial fistulas (3.22% V/s 8.98%).

Table No.14 -Incidence Of Complications In Native Angioaccess (Av-Fistula) Vs Artificial Angioaccess

COMPLICATION	AV-FISTULA (NO.)	ARTIFICIAL ANGIOACCESS (NO.)	TOTAL
INFECTION	7(3.3%)	24 (11.48%)	31
BLEEDING	17(8.13%)	23(11.00%)	40
POOR FLOW	17(8.13%)	22(10.52%)	39

Complications of infection rate, poor flow and bleeding were higher amongst artificial angioaccess as compared to AV-fistulas (69 V/s 41).Thus, AV-Fistula is associated with lesser incidence of complications as compared to artificial angioaccess.

IV. Summary And Conclusions

The present study was done in Department of Nephrology and Renal Transplant Centre, Mahatma Gandhi Mission's Medical College & Hospital, Aurangabad in CKD patients who underwent AV-fistula creation and temporary angioaccess insertion and the associations with various risk factors.

- A male preponderance was seen in our study with the Male : Female Ratio 3.22:1
- The mean age in the study was 46.84 years with a range of 13-80 years.
- The maximum number of patients were from the 41-60 age group (38.38%). The maximum number of male participants were from the 41-60 age group (42.23%). The maximum number of female patients were seen in the 21-40 age group (42%).
- The maximum number of patients had eGFR < 10 (82.93%) in the study population.
- Hypertension was the most prevalent (71.56%) single co-morbidity in the study population.
- The most commonly used angioaccess was the Internal Jugular (80.09%).
- The primary success rate of AV-Fistula creation was 84.27%.
- The preferred site for AV-fistula creation was the left radial (71.29%) and the least preferred site was the right brachial (1.9%).
- Infection was the most commonly seen (11.48%) complication associated with the use of artificial angioaccess.
- A significant higher rate of infection was noted in femoral HD catheters (25%), when compared to the jugular access (8.28%).
- Poor flow was more commonly associated with the femoral HD catheters (20%) as compared to the jugular (8.87%).
- Although the percentage of bleeding was more in femoral catheter (15%) as compared to jugular (10.05%), but it was not statistically significant.
- A higher incidence of maturation (>15 days) was observed in our study with Brachial fistulas (77.41%) as compared to Radial fistulas (38.76%).
- There was equal incidence of bleeding and poor flow as complications of native angioaccess(8.13%).
- Infection was the least common complication seen in AV-Fistulas (3.34%).
- Higher failure rates were noted with brachial fistulas (22.58%) as compared to radial AV-Fistulas (17.98%).
- There was no statistically significant difference in failure rates recorded between male and female gender.
- The failure rate was highest in the 0-20 age group (27.27%), but this was not statistically significant.
- The presence of 3 or more co-morbidities resulted in a greater incidence of failure (50%)for AV-fistula.
- Although there was a significant incidence of failure in patients with no underlying co-morbidities (44.44%), the number of subjects in that subset were small. This was not statistically significant. The cause of failure was not further evaluated.

- The complications of bleeding and poor flow were more commonly seen (8.13%) as compared to infection (3.34%) among AV-fistulas.
- Bleeding however was seen more with radial fistulas (3.22% V/s 8.98%), whereas greater incidence of infection (6.45% V/s 2.8%) and poor flow (12.90% V/s 7.30%) was associated with brachial fistulas as compared to radial fistulas.
- The incidence of complications were more associated with artificial angioaccess as compared to native angioaccess. Thus, AV-Fistula was associated with lesser incidence of complications as compared to artificial angioaccess.

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Pleuroperitoneal fistula complicating peritoneal dialysis

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Abstract: 66 years female with ESRD, treated with CAPD started complaining of dyspnoea, dry cough and back ache within 2 days of commencing CAPD. Chest X-ray revealed massive hydrothorax on right side .Investigations eliminated infective, cardiac and primary respiratory causes .CAPD related hydrothorax was suggested by biochemistry and a pleuroperitoneal leak was confirmed by peritoneal scintigraphy using -99 m Tc -MAA (Macro aggregated albumin).Laparoscopic suturing of Pleuroperitoneal fistula was done and CAPD initiated successfully.

Key Words: CAPD, Pleuroperitoneal fistula, peritoneal scintigraphy, 99 m TC-MAA, laparoscopic repair of fistula

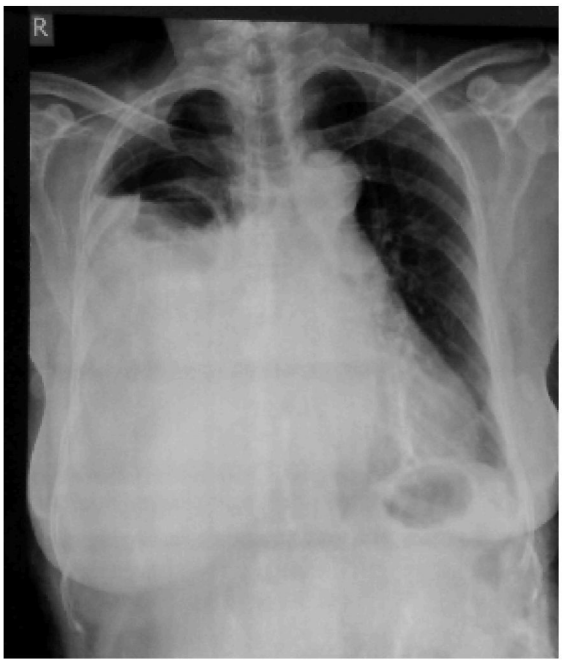
Introduction

PD-related hydrothorax was first reported in 1967 [1]. The reports of prevalence rates of hydrothorax vary, ranging from 1.6 to 10% of PD patients [2]. Usually, these patients present with sudden dyspnea, a decrease in ultrafiltration and chest pain. Some of them may remain asymptomatic or just complain of a dry cough [3]. An early and accurate diagnosis of pleural leakage is important. Here we report a case of pleural leakage which was diagnosed by scintigraphy and repaired laparoscopically.

Case History:

66 year female with ESRD secondary to hypertension was treated with CAPD. After 2 days she started complaining of shortness of breath, dry cough and back ache. On physical examination she was tachypneac and chest examination showed signs of fluid on right side. X-ray chest revealed right sided hydrothorax (Fig.1)

Fig 1 : X-ray chest showing right hydrothorax



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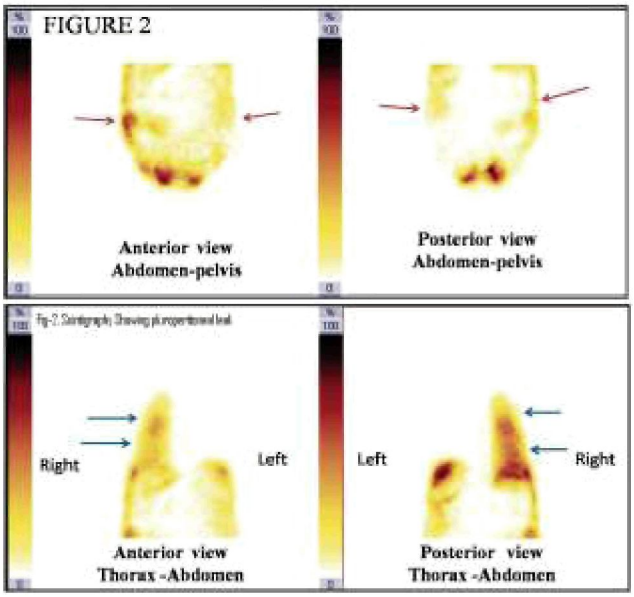
.Thoracocentesis was done and 1 L of clear fluid was removed. The fluid examination showed glucose concentration 278 mg %, much higher than serum glucose, which raised suspicion of leakage of peritoneal fluid in the pleural space. Report of pleural fluid showed protein-0.3mg/dl, WBC-60/cu mm, Polymorph-60%, lymphocytes-40% and ADA level was 11.0U/L

For confirmation of peritoneal leak peritoneal scintigraphy using radioisotope 99mTc-MAA was performed. 3mci of 99mTc was instilled with 300ml of dialysate in the peritoneal cavity via CAPD catheter under asepsis. Images of thorax and abdomen demonstrated abnormal tracer accumulation in right pleural cavity suggesting a pleuroperitoneal fistula (Fig.2). Decision to withhold PD was taken after the scan.

Fig 2 : Scintigraphy showing leakage of isotope on right side of chest



Conservative approach with small volume exchanges was tried but failed, so patient was considered for other form of renal replacement therapy. As patient was reluctant to continue on heamodialysis , minimal invasive surgery with Laparoscopic suturing of peritoneopleural fistula was performed .CAPD was continued after 4 days with ICD drain, which was removed after 7 days as the repeat chest X-ray confirmed no further leak .The patient is on CAPD for last two years.

Discussion:

Hydrothorax due to the migration of dialysis fluid from the abdominal space into the pleural space creates a serious complication of PD; it is normally not life-threatening. Hydrothorax frequently presents as shortness of breath or even dyspnea, Hydrothorax is almost always on the right side (4). Our patient also suffered from right pleural effusion.

The sensitivity of radionuclide scans (such as Tc-99m DTPA) to demonstrate peritoneo-pleural fistula is reported to be between 40 and 50% (4,5). Although the leakage location could not be located in this case, pleuroperitoneal communication was definitively diagnosed through scintigraphy.

Fig 3 : Laparoscopic picture showing pleuroperitoneal fistula.

There are a lot of ways of treating pleuroperitoneal communication. First of all, PD should be interrupted for a period of time. Resting the membrane for at least 4-6 weeks will allow the mesothelium to reconstitute itself over the defect, and the pleuroperitoneal communication may reseal. A temporary interruption of PD was associated with a 53% success rate in resuming PD (4). Furthermore, invasive procedures like pleurodesis and thoracotomy as well as video-assisted thoracic approaches can be applied if the conservative treatment is failing. Nearly 60% of patients with pleural defects resume maintenance PD after either conservative or interventional treatment (6) but the others have to permanently transfer to hemodialysis.

PD-related hydrothorax should be recognized and treated timely, although most of the diagnostic strategies that confirm it is unsatisfactory, CT peritoneography provide a non-invasive and useful diagnostic tool for pleuroperitoneal communication. Conservative treatment generally precedes invasive intervention. The discontinuation of PD for a few weeks often leads to the successful resolution of hydrothorax.

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Recognitions/Awards

Felicitation by Health Minister Of Odisha and VC Kalinga University(Dr.Samantha) 2018

(Retd) Justice Venkatachalaya felicitating Dr.Sudhir Kulkarni July 2013

Award from Vice President of India