DISSERTATION

A STUDY ON ACUTE KIDNEY INJURY IN POSTOPERATIVE PATIENTS AT GOVERNMENT STANLEY HOSPITAL, CHENNAI .

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032.

In partial fulfillment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001.
APRIL 2015

CERTIFICATE BY INSTITUTION

This is to certify that **Dr.MANIKANDAN.V,** Post - Graduate Student (MAY 2012 TO APRIL 2015) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**A STUDY ON ACUTE KIDNEY INJURY IN POSTOPERATIVE PATIENTS AT GOVERNMENT STANLEY HOSPITAL, CHENNAI.**" under my guidance and supervision in partial fulfillment of the regulations laiddown by the TamilnaduDr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2015.

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DECLARATION

I **Dr.MANIKANDAN.V** declare that I carried out this work on "**A STUDY ON ACUTE KIDNEY INJURY IN POSTOPERATIVE PATIENTS AT GOVERNMENT STANLEY HOSPITAL, CHENNAI** " at the Nephrology wards and Surgical wards of Government Stanley Hospital during the period February 2014 to september 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The TamilnaduDr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Branch 1Degree examination in General Medicine.

Dr.MANIKANDAN.V

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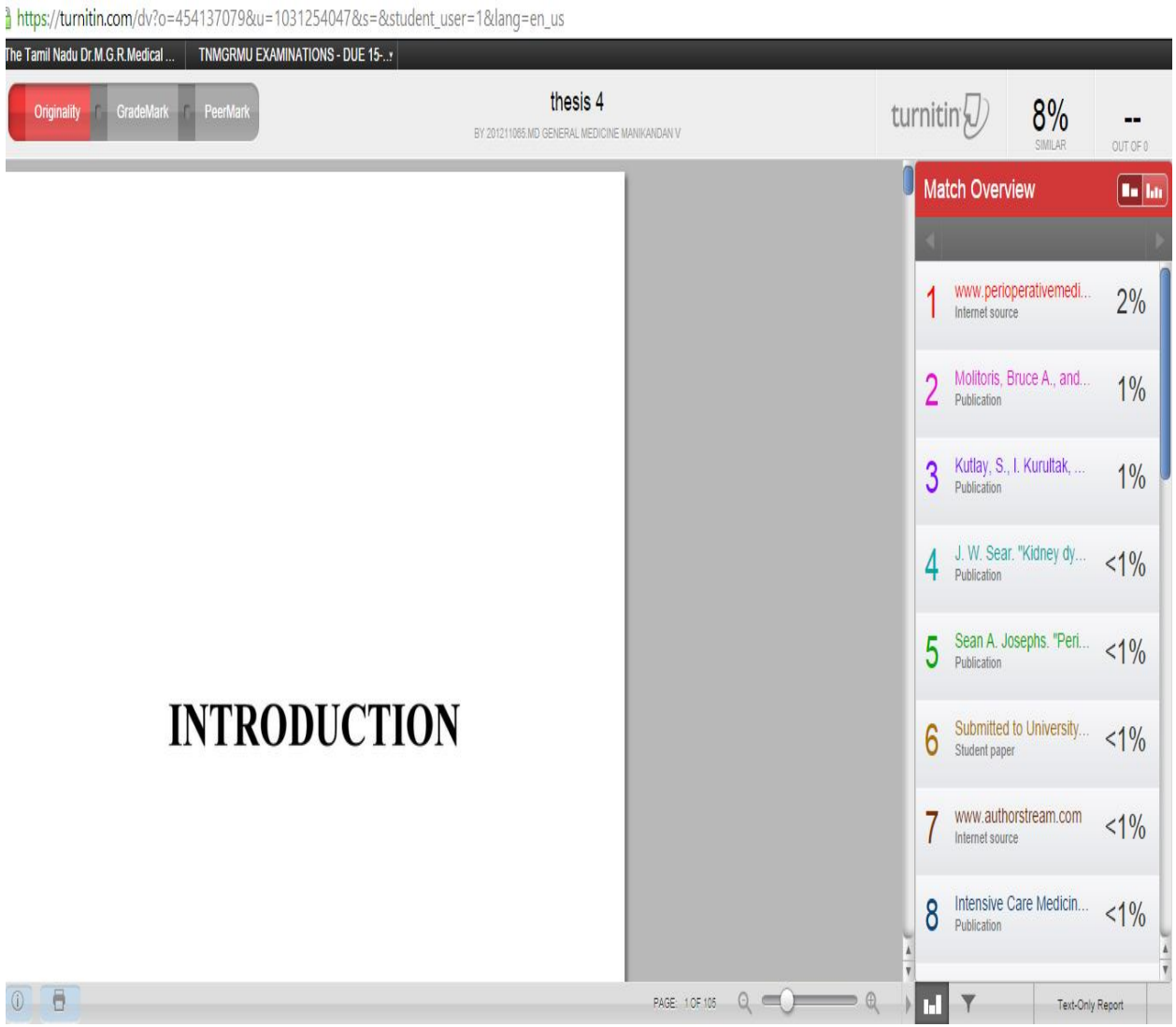
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ABBREVIATIONS

AKI	acute kidney inury
RFT	renal function test
RRT	renal replacement therapy
ICU	intensive care unit
PCT	proximal convoluted tubule
DCT	distal convoluted tubule.
CPB	cardio pulmonary bypass
GFR	glomerular filtration rate.
CKD	chronic kidney disease

A STUDY ON ACUTE KIDNEY INJURY IN POST OPERATIVE PATIENTS IN GOVERNMENT STANLEY HOSPITAL,CHENNAI.

ABSTRACT:

OBJECTIVE:

To assess the risk factors causing AKI in post operative patients.

To assess the incidence of AKI in post operative patients.

METHODOLOGY:

Patients in postoperative period with acute kidney injury in stanley medical college hospital,during the period of february to september 2014fulfilling the inclusion and exclusion criteria were included in the study and 60 patients had AKI fulfilling the criteria. Stastical analysis were done using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010. Continuous variables were analysed with Unpaired t test and categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$.

RESULTS:

The study was a prospective study and 60 patients had AKI during the time period in postoperative period. The incidence of AKI was 1.48%.

Intraoperative hypotension was found to be a significant risk factor.

Conclusion:

1)The risk of developing AKI in patients undergoing surgery were evaluated over a period of 6 months.

2) It was found that patients undergoing cardiac surgery were found to have high risk followed by vascular and other surgeries.

3) The incidence of AKI is 1.48%

4) When patients were classified according to AKIN criteria of >0.3 mg/dl rise of creatinine all the above patients were found to have AKI, but when classified according to RIFLE criteria of >50% rise around 31 patients were found to have AKI.

5) From this study patients undergoing cardiac surgery were found to be at higher risk,and intra operative hypotension was a significant risk factor.

6) The serum creatinine is not a true and early marker of acute kidney injury and there is delayed rise.

7) Most of the patients recovered from AKI after 7 days of surgery.

KEY WORDS:

AKI,CARDIAC SURGERY,GFR,RIFLE CRITERIA.

INTRODUCTION

ACUTE KIDNEY INJURY

Acute kidney injury previously known as acute renal failure is described by sudden impairment of kidney function which results in accumulation of nitrogenous and other waste products.

AKI in post operative period is common after major surgeries.
AKI requiring dialysis occurs in 1% of cardiac and vascular procedures.
AKI occurs in post operative patients due to various factors. It is commonly seen after vascular, cardiac and hepatobiliary surgeries.
AKI in the post operative period is influenced by many factors like age, Sex, type of surgery, duration of surgery, fluid balance, hemodynamic instability, nephrotoxic agents.

The latest classification of AKI based on AKIN and RIFLE criteria. In 2004 ADQI formulated the RIFLE CRITERIA, risk, injury, failure, loss and end stage disease.Cardiac surgeries commonly associated are valve replacement surgeries and bypass surgeries.vascular surgeries which involves aortic cross clamping have higher risk.

Even a minor increase in serum creatinine is related with increased mortality and cost.AKI occurs in a hospital under multiple settings in sepsis,ICU, burns, pancreatitis,post operative patients,patients receiving nephrotoxic drugs for longer duration,and in emergency procedures.

There are various outcomes for AKI in postoperative patients ranging from conservative management to patients requiring renal replacement therapy.AKI is assessed by the rise in serum creatinine or decrease in urine output.AKI might occur on the same day of post operative period to several days or weeks following the procedure due to gradual decline in renal function.

REVIEW OF LITERATURE

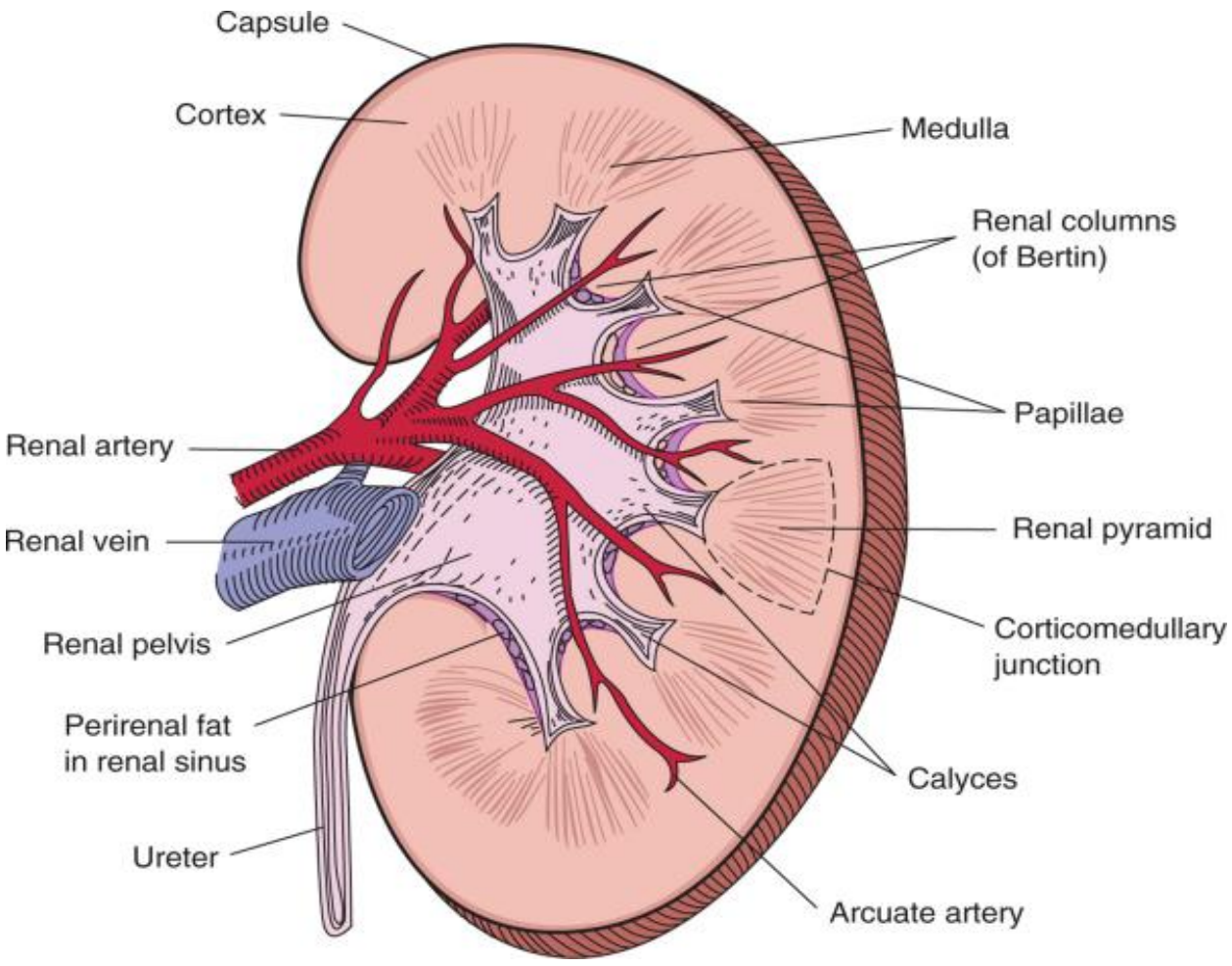
Kidney is a highly organized structure in the body. Nearly 30 varieties of cell types form the nephrons and capillaries in the kidney. This complex structure enables kidneys to perform different physiologic functions.

EMBRYOLOGY OF KIDNEYS:

- Development of kidneys starts at 4^{th} week
- It develops from intermediate mesoderm
- primordial components ----pronephros ,mesonephros,metanephros
- pronephros :appear as solid cell groups in cervical region ,which then regresses
- mesonephros : forms glomerulus bowman's capsule, which opens into mesonephric duct
- except mesonephric duct rest of these structures regresses
- mesonephric duct forms internal genitalia in males , an out growth from it forms ureteric bud.
- metanephros forms excretory part of definitive kidney upto DCT
- ureteric bud forms the rest, from collecting tubules upto trigone of the bladder

- interaction between metanephros and ureteric bud initiates development of kidney
- growth factors :metanephros ----WT1,GDNF,HGF

  ureteric bud-----FGF2,BMP7.



ANATOMY OF THE KIDNEYS

There are two kidneys in our body and they are like a bean shaped organ. They are present in the abdomen at the lumbar region at the level of T12-L3. Blood to the kidneys reach through aorta. The size is 11*6*3 cm. the right kidney is lower than the left kidney,the left kidney is more medial.

Each kidney consists of

1) cortex (renal arches ,renal columns)

2) medulla (pyramids,papillae,major calyx,minor calyx)

3) renal sinus (pelvis,renal vessels and lymphatics)

Blood supply : abdominal aorta at the level of L2

renal veins drains into IVC

VASCULAR ANATOMY:

- Aorta
- renal artery
- segmental artery
- inter lobar artery
- arcuate artery
- inter lobular artery
- afferent arteriole

- glomerular capillary
- efferent arteriole
- peritubular capillary
- Inter lobular vein
- arcuate vein
- inter lobar vein
- segmental vein
- renal vein
- ivc

Urine produced from the kidneys in the collecting tubules reaches the bladder through two tubes called as ureters.The bladder varies in its amount and it can hold around 500 to 750ml. Nephrons are the functional unit of the kidneys . The nephron is made up of 2 parts, one is glomerulus which are formed by a group of capillaries which filters the blood and the collecting tubules which connects the glomerulus.

The tuft of the glomerulus is formed by a capillary network made by two arterioles, an afferent arteriole and ends in an efferent arteriole. The bowman's capsule holds the glomerular tuft and is spherical in shape and is continous with the proximal tubule. Both the glomerulus and the bowman's capsule consist of epithelial cell lining. The tuft of the capillary is responsible for the ultrafiltration which occurs as the blood flows across it. The filtrate passes through the proximal tubule. The juxta-glomerular apparatus lies in between the afferent arteriole and the efferent arteriole and is bound by the arterioles, distal convoluted tubule and the cells of lacis which lies among the two arterioles.

There are another group of cells called the mesangial cells which form the mesangial matrix. Their function is thought be supportive and they also have a phagocytic function and they are also called as third reticuloendothelial system. These mesangial cells can contract and are mainly represented in the pathogenesis of nephropathy due to diabetes. Factors or agents that cause greater contraction of mesangial cells lead to sclerosis of the mesangium. The mesangial cells and matrix are surrounded by capillary loops. The capillary wall is made up of 3 layers namely

1. epithelial cells,
2. the glomerular basement membrane and
3. endothelial cells.

The word “proliferative glomerulonephritis” indicates multiplication of various types of cells inside the glomerulus and therefore various types of proliferative glomerulonephritis (GN). In this type of Crescenteric Glomerulonephritis,evidence shows that macrophages which are present in the circulation and which are normally located in the glomeruli achieve access inside the tuft of the glomerulus and convert themselves into special type of cells called epithelial cells and it is the multiplication and development of these newly formed cells of epithelium that creates the crescents in Crescenteric Glomerulonephritis. The basement membrane of the glomerulus (GBM) is formed by 3 layers : the lamina densa which is the dense zone present in the centre,and the lamina rara interna which is present inside and externa which is present outside.

The thickness of glomerular basement membrane is around 80 nm thick. The detailed electron microscopic studies identify that the “filtration barrier” is made up of an inner layer of endothelium which is fenestrated, the glomerular basement membrane which is the middle layer and the epithelial foot process which is interdigitated which is present in the outer layer.

The glomerular basement membrane consists of pores which cannot be visualized and is made up of a gel which is highly hydrated which consists of a collagen-alike and glycoproteins which are not-collagenous. The so called non-collagenase component of the glycoprotein is enriched in contents such as hydroxyproline, the galactose, the mannose and the sialic acid. The epithelium consists of interdigitated processes which is surrounded by glycoproteins that consists of high concentration of of sialic acid. The sialic acid consists of negative charge which separate the foot processes. The basement membrane is predominantly made up of negative charges because of deposition of glycosaminoglycans and sialic acid) which constitutes a filtration barrier.

It provides explanation for the excretion of particles which are negatively charged is lesser than those of particles which are neutral of same size as the particles which are charged negatively are repelled away at the basement membrane. The glomerular basement membrane attracts positively charged particles.

Therefore particles of positive charge have a greater amount of clearance than particles which are made up of negative charge . The nephrotoxicity is due to antibodies to the negatively charged particles but not due to the antibodies to the neutrally charged collagenous glycoproteins. So it is clear

that not alone the size of the molecule but also charge of particles which is responsible for a particle's excretion during glomerular filtration. The glomerular permselectivity is affected when the charge barrier is lost and it leads to proteinuria.

FUNCTIONS OF THE KIDNEYS:

The amount of urine excreted by kidney per day is about 1.5 to 2.5 litres of urine. It also helps to maintain the salt and water balance. Most of the salt which is filtered is reabsorbed by the tubules. This helps to maintain correct sodium balance in the body. The tubules also helps in reabsorbption of dissolved substances like amino acids and glucose.The kidney also plays an important role in maintaining the acid base balance and potassium homeostasis.The kidney also helps to excrete nitrogenous waste products like through urea.The kidney is produces many hormones like Renin . Renin is inactive and it exerts its function on angio tensin I to form angio tensin 2 that produces constriction of arteries.

The kidney also helps in the formation of active form of vitamin D through 1,25 hydroxylase which helps in the regularization of calcium and phosphorus. Erythropoietin, is another hormone synthesized in kidney which is required for the production of red cells. Patients with chronic kidney disease are

pale because they are deficient in erythropoietin. Prostaglandins, is also produced which manages the flow of blood and blood pressure.Thus kidneys work to excrete all the metabolic waste of our body. The kidneys control the rate of excretion of these substances which helps to achieve the "milieu interieur". The amount of the urine produced is dependent on the RBF(renal blood flow)and the GFR(glomerular filtration rate).

RENAL PHYSIOLOGY:

Kidneys receive approximately 20% of cardiac output. Blood reaches nephron through afferent arteriole which divides into glomerular capillaries and finally unites to form efferent arteriole which continues into second capillary network surrounding the tubules. Thus there are two capillary beds arranged in series. The distal capillaries unite to form small vein which finally drains into renal vein.

The hydrostatic pressure is the driving force for glomerular filtration. Plasma proteins determine the oncotic pressure and it increases towards the efferent arteriole. The filtration fraction is determined by the ratio of glomerular filtration rate to renal plasma flow.Glomerular filtration rate is a product of average filtration rate of each single nephron, multiplied by the number of nephrons in both kidneys.

The normal level of glomerular filtration rate(GFR) is approximately 130 ml/min per 1.73 sq.m for men and 120 ml/min per 1.73 sq m for women. As age advances GFR gradually declines,approximately 0.75 ml/min/year after the age of 40 years. The level of GFR does not indicate the loss of nephrons and GFR may remain within normal range inspite of substantial kidney damage.

RENAL BLOOD FLOW:

In a normal healthy individual, the renal system will receive about 20% of the cardiac output,and the amount of oxygen delivered is around in excess of 80 ml/min/100g tissue. The flow of blood inside the kidney is variable, renal cortex receives in excess of 90% of blood flow**.** Also, consumption of oxygen does not go above more than 10% of total body's consumption, so there is a very little A-V oxygen content difference about one and half ml oxygen per hundred ml blood. This suggests that kidneys have a adequate oxygen reserve.but the problem is that the kidneys are highly susceptible to tissue hypoperfusion, with acute kidney injury being a very common side effect of hypotension.

This phenomenon of paradox is due to the ability of renal medulla to work at oxygen tensions of 2-3 kPa. The cause for such a decreased tension of oxygen arises because of the increased demand for oxygen from tubular absorption of Nacl. Though the cortex large amount of blood it uses only approximately eighteen percent of oxygen delivered to it. But , renal medulla recieves only a low amount of blood but utilizes more oxygen.Oxygenation of the medulla is tightly regulated by various control mechanisms, which helps to coordinate intra regional oxygen supply and demand. When these regulations fail it results in the outer medullary region vulnerable to many episodes of hypoxic insult, which can cause acute tubular necrosis (ATN) mainly the proximal s3 segment and the thick ascending limb.

RENAL DAMAGE DUE TO HYPOXIA

Due to the difference in oxygen requirement of various parts of the kidney, the oxygen levels in the renal cortex is fifty mm mercury greater than that of the inner medulla. So renal medulla is very susceptiblee to hypoxic injury and hypoxic injury can be created by a forty to fifty percent decrease in renal blood flow..

The medullary injury is identified by the necrosis of the tubules which are situated far away from the blood vessels. The major factor which determines the

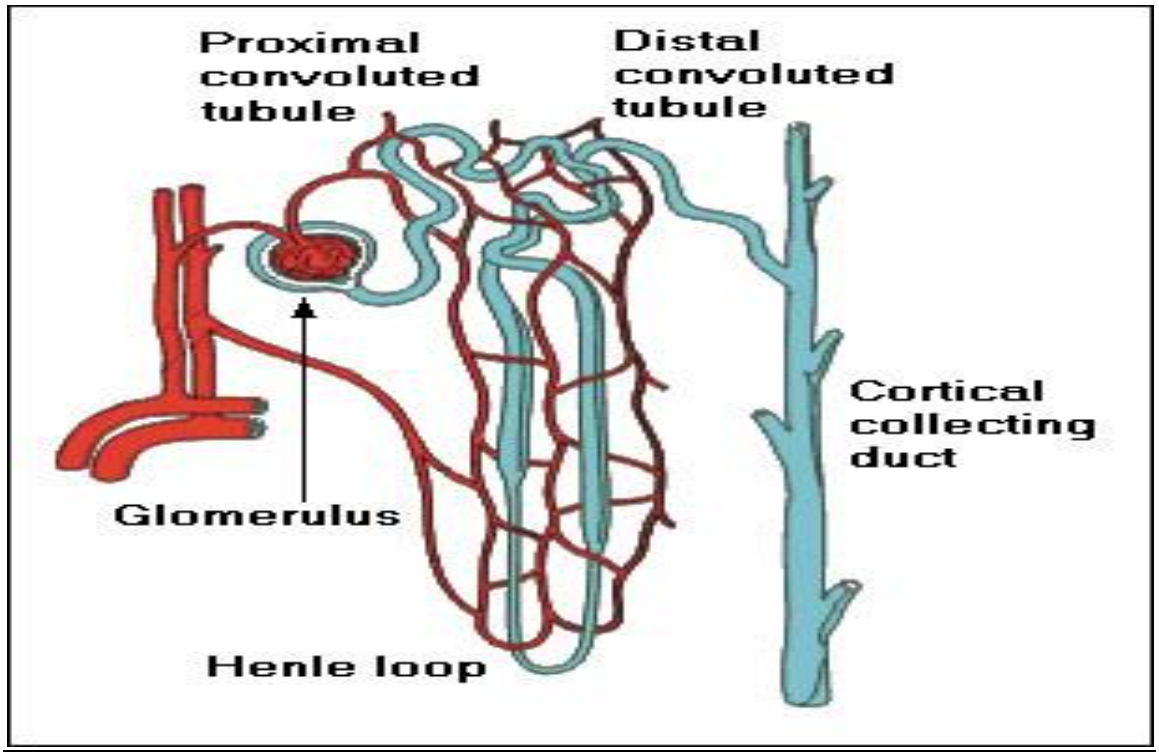
medullary oxygen requirement is the rate at which the salt and water reabsorption takes place.

The medullary flow of blood is affected by various number of molecules , that affects medullary blood flow, and cause ischemic injury. These include:

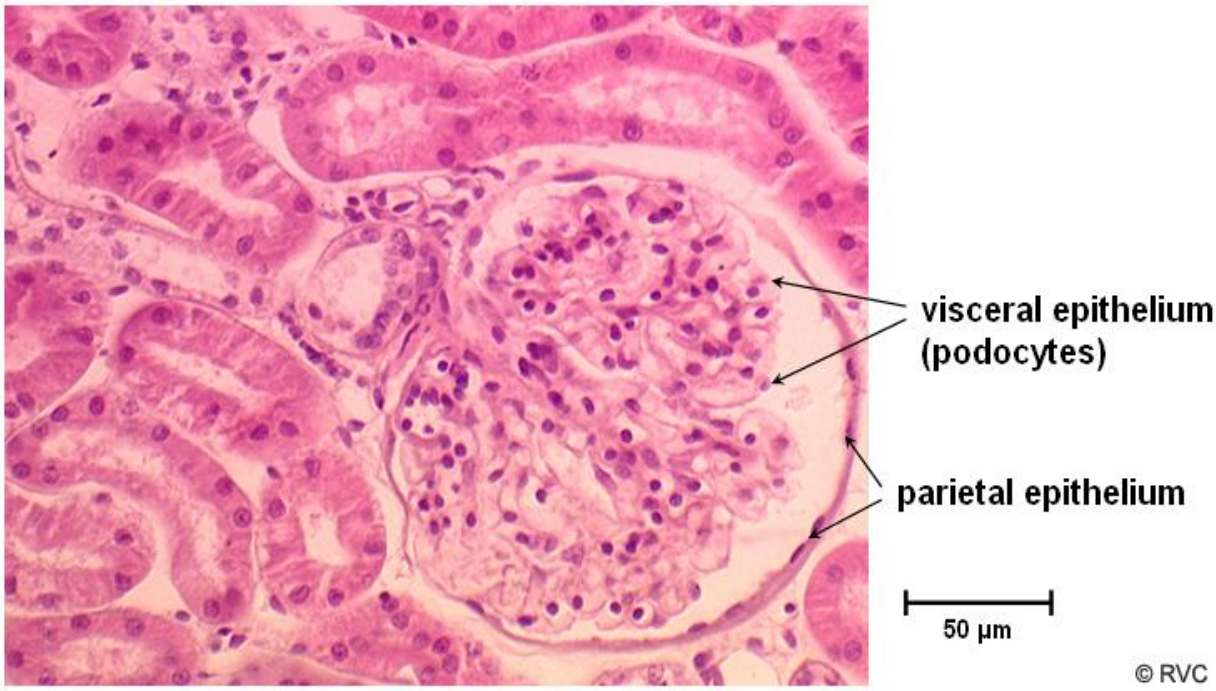
i. Vasodilators such as NO, prostaglandins that includes prostaglandin E_2, adenosine, dopamine.
ii. Vasoconstrictors: endothelin, angiotensin II, ADH (antidiuretic hormone or vasopressin).
iii. Tubulo-glomerular feedback: when the reabsorption of sodium is decreased, it leads to reflex mechanism of constriction of glomerular afferent arterioles,thus decreasing the filtration and tubular reabsorption of the solutes.

So the main factor predisposing renal medulla to hypoxia is associated with salt and water reabsorption, so the kidney can be prevented from such damage when there is volume of circulation is adequate. This helps in decreasing medullary oxygen consumption. So in a state of renal hypoxia multiple factors such as some drugs, renal hypertrophy, angiotensin II, calcium ions, myoglobin, hyperbilirubinaemia, and contrast media all worsen the damage.

NEPHRON



BOWMANS CAPSULE



EPIDEMIOLOGY OF AKI

A. Community-acquired AKI: on admission AKI is seen in 1% of inpatients. Mostly it is seen in patients with CKD. The majority is seen in patients with prerenal type around 2/3. The resulting mortality arising from community acquired AKI is around 15%.

B. Hospital-acquired AKI: AKI is common in hospitalized patients and is associated with mortality. The various causes causing AKI in hospitalized patients include ischemia, infection,drugs and contrast materials. Majority of causes for renal failure in ICU is due to ATN.

C. Prevention of AKI.AKI is due to numerous factors like decreased hydration,sepsis,drugs and radio contrast.attending to these details might help in reducing AKI.

Surgery is one of the leading causes of AKI in patients who are hospitalized. This has been time and again demonstrated in patients undergoing cardio thoracic surgery where it is identified up to 10 to 15% of patients who are subjected to bypass (CPB) are developing Acute kidney injury, witharound 1 to two percent requiring renal replacement therapy . The mortality and morbidity depends on the criteria used to categorise AKI. AKI is not only seen in cardiac surgeries but it is also seen in other surgeries but it has not been extensively studied.The incidence

was less than 1% in major non cardiac surgeries without preexisting kidney disease.

ACUTE KIDNEY INJURY(AKI):

In the postoperative period, clinicians try to give adequate facility for the individuals by going through systematic examination of all systems. It is very much essential for non emergency patients and for seriously ill patients. The perioperative period is a characterstic period for it is unique process in the body of the patient where important hemodynamic changes and events can be predicted depending on the previous examination of the patient and the type of surgery the patient underwent. This is a period when several protective therapy will be initiated for different patient groups if their risk can be considerably assesed.

Acute kidney injury is defined by rapid deterioration of GFR resulting in accumulation of metabolic waste products. Acute kidney injury is broadly classified into three types based on cause,

1) Prerenal AKI-due to hypoperfusion of kidneys with no damage to kidneys.
2) Intrinsic AKI-renal parenchyma is affected.
3) Post renal AKI-obstruction of urinary tract causing AKI.

The acute dialysis quality initiative(ADQI) formulated the RIFLE classification. It classifies AKI based on severity based on increase in

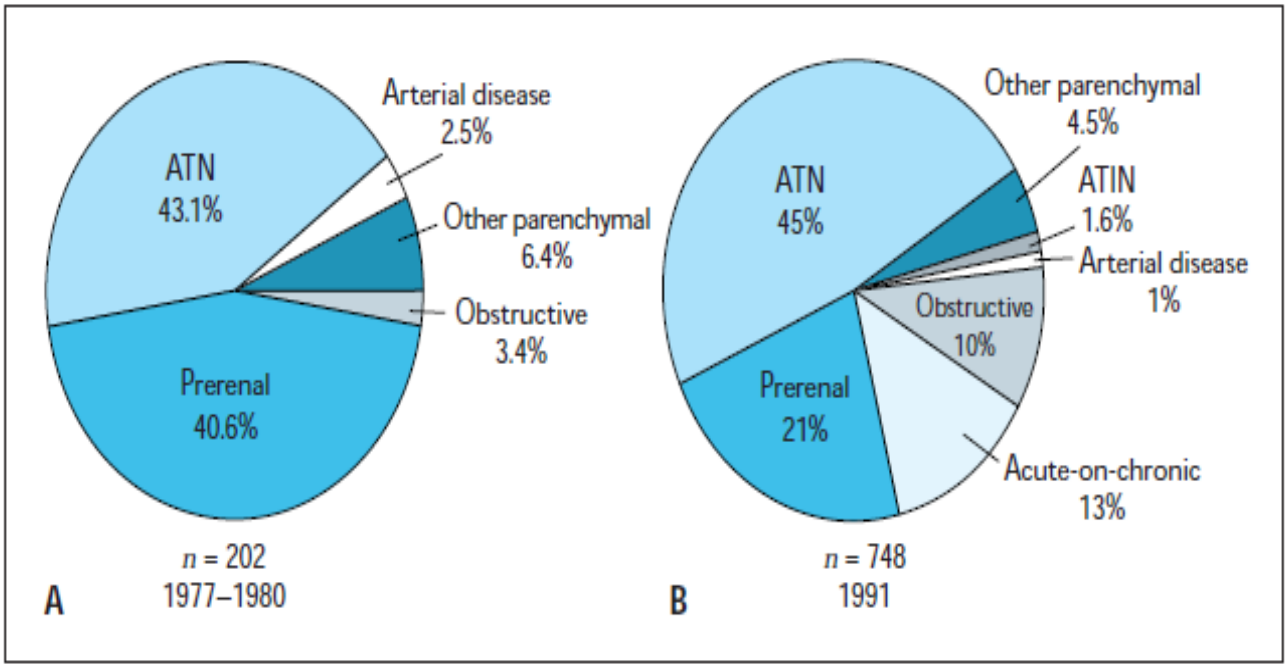
creatinine and decrease in urine output. It has been tested in multiple general and ICU populations. In RIFLE and AKIN classifications patients are categorized into the severe stage reached.AKI is associated with increased mortality in critical care settings. These patients are older,have comorbid illness,have associated sepsis. AKI is also associated with increased treatment costs. The RIFLE and AKIN classification are of great use in stratifying the patients into different grades and assessing their treatment options. Renal replacement therapy is required for loss and end stage.

The AKIN classification is upto 48 hours whereas RIFLE classification is upto 7 days.AKI is also associated with increased duration of hospital stay. The disadvantages of these criteria is there is poor correlation with GFR,there is lack of concordance between serum creatinine and urine output level. Both the criterias require basal level which is often unavailable and they use relative changes in creatinine. These classifications are not based on the causes of AKI.

The increased dependance on serum creatinine and urine output fails to identify the incipient stages of AKI which is most amenable to medical treatment. There are several criteterias for classifying AKI but most of them rely upon serum creatinine and urea levels. The

problem with relying on these levels is that they do not raise immediately and they take some time to raise and they are affected by various factors.

Represanatation of various causes of AKI.



Distinguishing AKI from CKD in acute settings will be tough. Lab findings such as increased phosphate,decreased albumin and hyperkalemia may be present in both conditions and can be misleading. They can be distinguished to some extent using

1. Old records: old records help to differentiate AKI from CKD. Elevated urea and creatinine and previous history of renal disease suggest that the disease may be chronic.

2. Shrunken kidneys with size less than 10 cm on ultrasound suggest that the disease may be chronic.

3. Anemia: anemia of normocytic normochromic type suggest that the disease is chronic kidney disease.anemia is usually present when GFR is less than 30 ml/min. If anemia is not present when GFR is less than 30 ml/min then the disease is most likely to be acute. There are some exceptions to this like in autosomal dominant polycystic kidney disease anemia may be absent in CKD and in TTP and HUS anemia may be present in AKI.

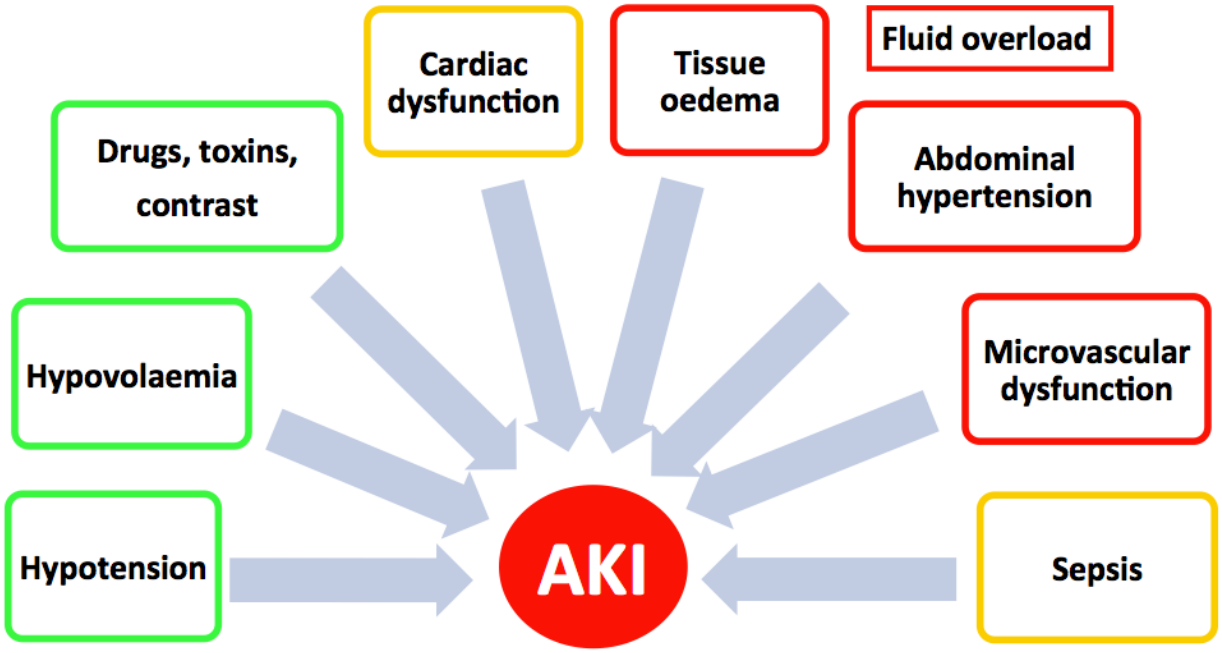
Thus it is difficult to identify AKI from CKD immediately in a clinical setting. The review of old records and the presence of shrunken kidney size and the presence of anemia favours the diagnosis of CKD over AKI.

RISK FACTORS FOR AKI IN POST OPERATIVE PATIENTS:

There are numerous risk factors for development of AKI in surgical procedures, which includes both patient and type of procedure done in both cardiac and non-cardiac surgery. But it is patient related factors which is more associated with development of AKI than the type of procedure done. The common risk factors are age of the patient,comorbid illnesses like systemic hypertension,diabetes mellitus ,heart failure,any vascular pathology and underlying CKD . out of these the most prominenet risk factor is a patient with an underlying kidney disease undergoing cardiac surgery. For each surgery risk factors remain specific which are commonly seen with postoperative acute kidney injury, including increased cardio pulmonary bypass time, double valve surgeries and graft (CABG) surgery, when the duration of aortic cross clamp is prolonged in vascular surgery and when there is a high intra-abdominal pressure in abdominal surgeries. Most of the times the reasons for acute kidney injury in postoperative patients is decreased renal perfusion and diminished renal reserve which can be identified prior to surgery and necessary steps be taken to reduce the development of AKI.

A study demenstorates a scoring system is developed for every patient,depending on various risks preoperatively, after that patients are classified into various classes. Class one in which patient has upto two risk

factors the chances for occurrence of AKI is 0.2%; whereas in Class five having more than 6 risk factors the risk of acute kidney injury is around 9%. Thus the development of AKI depends on various factors depending on the setup,patient and type of surgery done. So preoperative identification of risk factors is important to minimise the incidence of AKI.



There are various risk factors in the intraoperative period which are related with Acute kidney injury.These risks are tedious to be quantified,since they cannot be maintained precisely during the surgical procedure. In cardiothoracic surgery the various risk factors which occur through intraoperative period for kidney failure during postoperative period is the practice

of intra-aortic balloon pump, hypothermia causing cardiac arrest, decreased amount of urine during cardiopulmonary bypass (CPB), the requirement of ionotropes before cardiopulmonary bypass, number of transfusions of blood during surgery. The important contributing factor that is linked with acute kidney injury is the time for which the patient was exposed to cardio pulmonary bypass circuit. In patients who are subjected to on pump surgery, and if the duration of cardio pulmonary bypass is greater than two hours then there is a increased risk for acute kidney injury. The cause for AKI due to exposure to cardiopulmonary bypass system promotes a proinflammatory state which will affect the blood flow to the kidneys. Also there are many findings to suggest that decrease in pulsatile blood flow cause deleterious effects on perfusion of renal system,in spite of balanced preservation of mean arterial pressure. So because of these reasons doing off pump bypass surgery has gained curiousness as it appears to decrease the reduce the incidence of acute kidney injury.

There are multiple evaluations which have evaluated outcome of kidneys between patients who are subjected to on-pump and of-pump procedures. There was a group which evaluated the difference in outcomes between these patients. It was found that off pump surgery had a favourable outcome between the two and there was decrease in incidence of AKI.

The disadvantages were that because of different definitions of AKI and smaller sample sizes. So from these observations it appears that if there are high chances of postoperative acute kidney injury in a patient ,off-pump surgery should be considered as an option to decrease the AKI risk.

In non cardiothoracic surgery,only limited number of evaluations are available to assess the risk factors . In liver transplantation a method is developed to immediately identify the susceptible patients for acute kidney injury based on the risks. The suspected risk factors were amount of blood transfused , decreased blood pressure with mean b.p less than fifty mm of mercury, and increased lactate levels.Although the amount each of these factors contributes to risk is not known, its absence is comforting. Procedures which were not that penetrative offers a decreased risk for acute kidney injury.

In vascular procedures, the requirement of pressor support has been associated with acute kidney injury. Lot of problems involving the hemodynamics take place in vascular surgery involving aorta repairs which are open and when the duration of aortic clamping is prolonged and increased bleeding takes place.

In surgeries involving the thoracic region of aorta, endovascular procedures appear to have a beneficial effect compared to open procedures in reducing the incidence of acute kidney injury. Increased duration of surgery increased body mass and different positions in surgery can promote acute kidney injury. Thus in vascular procedures there appears a increased risk in open surgeries,prolonged bleeding and increased duration of clamping.

Postoperative evaluation of risk factors:

There are many events occurring in the post surgical period which affects kidney function. There are no clear cut evidence because of the lack of distinct relationship between non renal events and acute kidney injury. In cardio thoracic surgery, large postoperative loss of blood, large blood transfusion, postoperative infarction of the myocardium,and need for emergency resurgery are all factors for acute kidney injury. In patients undergoing cardiac transplantation, studies were done to understand the relation between starting of dialysis and the timing of

patients undergoing sepsis and other complications such as cardiac failure. Most of the cases of acute kidney injury who required dialysis were because of non renal complication.

Acute kidney injury occurs in non cardiac surgeries during the postoperative period.In vascular surgery,the need for pressors and the need for prolonged postoperative mechanical ventilation have been associated with acute kidney injury. In liver transplantation, treatment of patients with dopamine for more than 6 days, liver graft failure, repeat surgery , and postoperative infection all were significantly related with acute kidney injury. Infections and sepsis were associated with more acute kidney injury and also AKI lead to further infections. The increased urea levels which occur due to acute kidney injury can also affect other organ systems.

Intra abdominal hypertension:

In abdominal surgeries increase in intra abdominal pressure affects the rnal blood flow.Normal intra-abdominal pressure < 7 mmHg.Normal abdominal perfusion pressure > 75 mmHg. When this pressure exceeds it leads to.

- Decreased RBF, increased venous pressures
- Impaired gut blood flow & gut translocation

- IAP > 20 + organ failure = compartment syndrome.

RISK FACTORS FOR DEVELOPMENT OF AKI

PATIENT RELATED	**SURGERY RELATED**
AGE	DURATION OF SURGERY
DIABETES,HYPERTENSION	INTRA OPERATIVE HYPOTENSION
UNDERLYING KIDNEY DISEASE	BLOOD TRANSFUSION
PERIPHERAL VASCULAR DISEASE	DRUGS
SEPSIS	TYPE OF SURGERY
ASCITES	
COPD,CAD WITH EF<40%,CEREBROVASCULAR DISEASE.	

STAGING CRITERIA

STAGE	INCREASE IN SERUM CREATININE	URINE OUTPUT	INCREASE IN SERUM CREATININE	AKIN STAGE
RISK	>50%	<0.5ML/KG/ HR FOR >6 HOURS	>0.3mg/dl Or >50%	Stage 1
INJURY	>100%	<0.5ML/KG/ HR FOR >12 HOURS	>100%	Stage 2
FAILURE	>200%	<0.5ML/KG/ HR FOR >24 HOURS OR ANURIA >12 HOURS	>200%	Stage 3
LOSS	Need for RRT For>4 week			
END STAGE	NEED FOR RRT >3 MONTHS.			

PARAMETERS FOR DIAGNOSIS OF ACUTE KIDNEY INJURY:

1)SERUM CREATININE

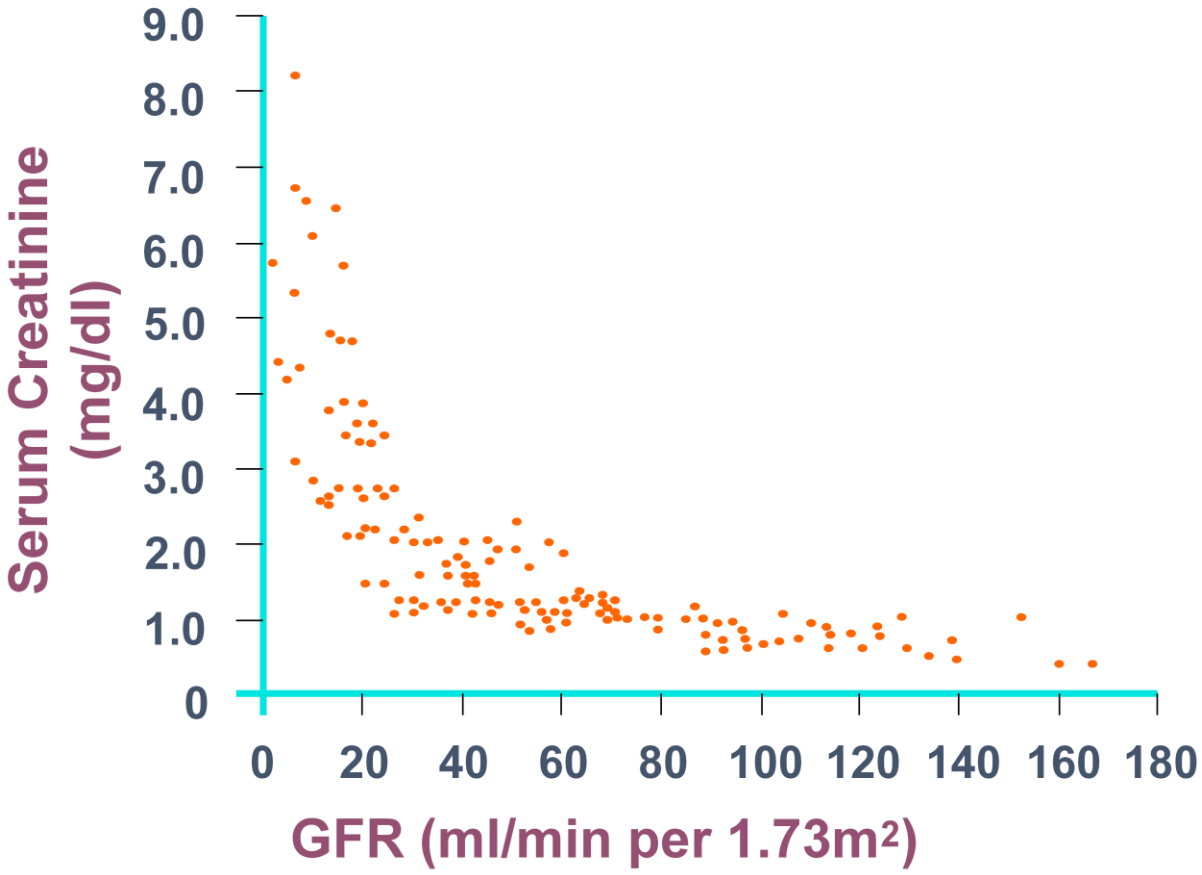
2)URINE OUTPUT

3)BLOOD UREA NITROGEN

4)BIOMARKERS

SERUM CREATININE:

Creatinine is mainly synthesized in liver and excreted mainly through kidneys.it is mainly removed glomerular filtration and also by proximal tubular secretion. The disadvantage of creatinine is that it is influenced by many factors.the factors affecting creatinine are age,sex,muscle mass,race,diet,drugs. The major determinants of creatinine is production,volume of distribution and elimination. The finding is that AKI occurs in a non steady state in which the three determinants of creatinine vary inconsistently. AKI is therefore not accurately determined by serum creatinine values. Urea levels can also be used but it also does not truly reflect GFR. Urea levels are also influenced by several factors like gastrointestinal bleeding, steroids and high protein diet.



Relationship between GFR and serum creatinine in AKI

URINE OUTPUT:

The urine output has to be monitored frequently to identify early insult to kidneys.several factors affect urine output besides kidney injury like volume of fluid given, diuretics administered. There are only few studies in which only urine output is taken as a criteria for AKI because of difficulties encountered in frequent monitoring of urine output. Comparison were made comparing studies using serum

creatinine and urine output and only urine output alone. The studies using only urine output showed lesser mortality associated with AKI.

BIOMARKERS:

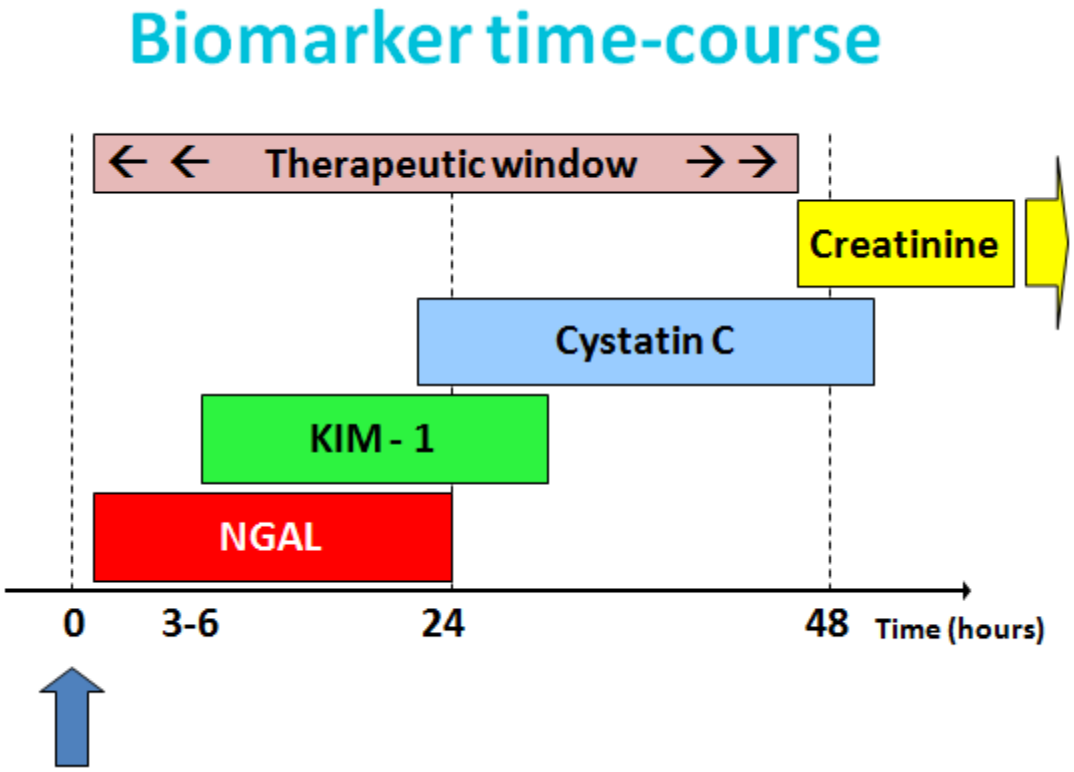
Search for serum biomarkers started for early identification of AKI since creatinine and urea had a delayed rise. The biomarkers are also useful in predicting the course and prognosis of the illness. But these biomarkers have not been extensively tested. The various biomarkers are

CYSTATIN-C,

NEUTROPHIL GELATINASE ASSOCIATED LIPOCALIN(NGAL), N-ACETYL-B-GLUCOSAMINIDASE,

KIDNEY INJURYMOLECULE-1,

INTERLUKIN-18.



CYSTATIN-C:

Cystatin-c is a 13-kD cysteine protease inhibitor protein which is produced in cell nucleus. Unlike creatinine, it does not undergo tubular filtration but instead glomerular filtration and it is not subject to any significant protein binding. Cystatin-c is also affected by various extra renal factors like thyroid disorder and steroids. It is helpful in measuring GFR. A study which was conducted using 72 patients who underwent cardio thoracic surgery showed no distinct association between acute kidney injury and plasma cystatin C although an recent, continous raise in urinary cystatin C was associated with acute kidney injury and the amount which was passed in urine was associated with the intensity of acute kidney injury.

From this it can be taken that cys-c in urine can be more useful. These studies have a susceptibility in that they have not been conducted in patients with chronic kidney disease who are at risk of undergoing acute kidney injury. These biomarkers are also altered by various factors, so it has to be time tested over various situations. It is difficult for a single biomarker to be able to predict the diagnosis of acute kidney injury. This means that we have to evaluate many biomarkers for assessing and identifying the early acute kidney injury.

NEUTROPHIL GELATINASE ASSOCIATED LIPOCALIN(NGAL):

It is a protien secreted by neutrophils and is bound to gelatinase. It is a 25kD protein and is elevated early after renal injury.NGAL has raised serious interest in past few years, mainly in Acute Kidney Injury following cardiac surgery. In patients who have adequate to normal kidney function, NGAL is not detectable in both urine and plasma, but still animal studies showed that NGAL is highly raised earlier after any ischemia to kidney. In further clinical studies, urinary NGAL is proven to be both sensitive and specific in suspecting postoperative acute kidney injury in young patients who are subjected to cardiac surgery. Also , plasma NGAL which is evaluated two hours following cardio pulmonary bypass was associated strongly with intensity and duration of AKI. In the adult group this result has been not so consistent.

Likewise, patients with raised plasma NGAL levels have also been shown to be raised in Acute Kidney Injury following CPB surgery, but it also has a low sensitivity causing limitation as an individual biomarker in the suspicion of acute kidney injury. This low sensitivity was because of many criterias depending on the value of creatinine for defining acute kidney injury. Acute kidney injury risk increases in patients who are subjected to prolonged duration of cardio pulmonary bypass. Though this increase in duration of CPB time has been shown to be a risk factor it also gives a possibity to suggest that NGAL (particularly plasma NGAL)might in fact represent the length of cardio pulmonary bypass causing inflammation. These studies do not include patients with acute kidney injury. An analysis further identified that in patients with underlying renal dysfunction urinary values were not reliable and it should be read with caution in patients in chronic kidney disease in patients having GFR less than sixty ml/min. thus this biomarker should be read with caution and is not of much use in CKD patients. In a study of patients undergoing cardio thoracic surgery NGAL raised within two hours of surgery. Urinary levels of NGAL was useful in trying to establish a difference between patients having intrinsic AKI and prerenal AKI.

N-ACETYL-D-GLUCOSAMINADASE:

It is a enzyme that is present in lysosomes in many cells in the human body. Its molecular weight is >130 kDa which helps to localize that anything found in urine is not due to glomerular filtration but due to tubular secretion.this urinary biomarker also helps to identify early AKI.

KIDNEY INJURY MOLECULE-1(KIM-1):

KIM-1 is present in the apical membrane of dilated tubules in acute and chronic injury. It cannot be detected in normal kidney function but elevated to higher levels in ischemic or toxic injury and is detected in urine. Research showed a distinct increase in kidney injury molecule protein expression which was shown at biopsy that was seen with elevated levels in urine,which could be detected prior to cast formation after a ischemic injury. After that, KIM-1has been demonstrated to be a very important marker for acute kidney injury in patients undergoing cardiothoracic surgery. When both of these markers were seen alongside there were cases of increased mortality and morbidity.

INTERLEUKIN-18:

It is also considered as a biomarker for acute kidney injury. IL-18 is a proinflammatory cytokine in proximal tubular injury and released into the urine. It reaches urine by glomerular filtration.

These biomarkers can be identified earlier than serum cretainine for example cystatin-c rises 1 to 2 days earlier than comparable rise in serum creatinine. But these biomarkers are poor in assessing the mode of treatment required like whether patient requires renal replacement therapy. These biomarkers are not only elevated when GFR is elevated but also in other conditions such as different patient characterstics,severity of illness and other reasons for decreased renal function.

PHYSIOLOGY OF REVERSIBILITY:

AKI has been classified into prerenal, intrinsic renal parenchymal disease and obstruction to renal flow. The problem arises in differentiating prerenal and intrinsic renal injuryin clinical setting. The concept of reversibility has been to approach the classification of AKI. Prerenal AKI has been classified as reversible form of renal dysfunction. There are three phases involved in this compensatory mechanism are amount of cardiac output that reaches the kidney,the filtration fraction and tubular reabsorption.

Prerenal AKI is the commonly form of acute kidney injury and constitutes upto 40% to 55% of all cases. It results from decreased blood supply to the kidney due

to a decreased effective volume. The causes for decreased blood supply include bleeding due to GI bleed,vomiting,diarrhea,renal loss and third space loss. The prerenal injury reverses rapidly if kidney blood supply is restored,because the kidney function is intact and there is no renal injury,but prolonged renal hypoperfusion results in acute tubular necrosis. Prerenal acute kidney injury and ischemic acute tubular necrosis are part of similar spectrum of renal injury. Prerenal acute kidney injury is divided into volume responsive and volume non responsive types. In volume nonresponsive forms further intravenous volume resuscitation is of no help in further treatment.

Hypovolemia results in decrease in mean arterial pressure which results in activation of baroreceptors which results in activation of several responses. These result in activation of sympathetic system and norepinephrine. vasoconstriction and water retention occurs. Angiotensin 2 activity is enhanced which is a highly potent vasoconstrictor which causes proximal tubular sodium reabsorption.

Renal sympathetic nerve activity is considerably enhanced in prerenal azotemia. The increased norepinephrine levels constrict the afferent arteriole and also changes the resistance of efferent arteriole.both angiotensin 2 and renal sympathetic nerves act simultaneously to help in salt conservation through sweat and result in increased thirst resulting in salt and water conservation to

maintain b.p. and preserve cardiac and brain perfusion. Autoregulation occurs till a mean systemic blood pressure of 75 to 80 mm hg is achieved. After this level glomerular filtration decreases drastically.

There is increased production of prostaglandins,kinins,kallikreins,nitric oxide which leads to vasodilatation. This is the reason why NSAIDS should not be given in hypoperfusion by inhibiting prostaglandins. Old age,renovascular disease,hypertensive and diabetic nephropathy all predispose to prerenal azotemia at even milder degrees of hypoperfusion.ACE inhibitors also should not be used in severe congestive heart failure and renal artery stenosis. Surgical procedures which normally would not cause renal injury will cause ischemic ATN in the presence of prerenal azotemia. Therefore it is essential to diagnose AKI initially and to treat it because it is a reversible condition and delay in treatment might lead to ischemic ATN or nephrotoxicity. Prerenal AKI is a potentially reversible condition because the intrinsic renal system is intact.

Decresed blood supply to the kidneys is the first risk in perioperative AKI, which leads to a decrease in blood flow to the medulla. The outer region of medulla has large oxygen demands upto 90% and is susceptible to both decreased perfusion and decreased oxygen demands not only in patients with CKD,instead it is similar in patients with normal renal function. This is important in acute kidney injury associated with Cardio pulmonary bypass surgery. Histologically, adequate

information is not available,which is mainly because renal biopsies are inavasive and are usually not done in patients in whom acute kidney injury. In some biopsies that was done in post-mortem there was a mismatch between the suspected thing and which showed in pathology report.

HOW TO EVALUATE A PATIENT HAVING AKI:

AKI should be evaluated in a systemic way to identify it early. Detailed history and a careful clinical examination helps in correct diagnosis.

The data evaluated should be noted down carefully for review for future evaluation. Vitals, weight of the patient, input output chart, old and new lab data, and the volume of fluid used and the drugs used should be collected and maintained. After the patient who was hospitalized for long time with a tedious course before going in to AKI, a detailed data sheet may help to identify the problem and help in initiating appropriate therapy.

Urine examination by dipstick and examination of urine routine must be done in patients who has AKI. Renal function tests like urea,creatinine,urine osmalility,sodium and urine protein must be measured.

Clinical signs and symptoms of a patient presenting with acute kidney injury is provided in the following:

PRERENAL AKI.

1. History. The history of following nature is indicative of prerenal AKI from true volume loss or hypovolemia: increased thirst, diminished fluid intake, history of fever, feeling of nausea, episodes of vomiting,history of diarrhea,patients having burns, and clinical features of pancreatitis. Prerenal AKI from decreased filling of arteries occurs usually in patients having congestive heart failure or hepatic failure. The suspicion of congestive heart failure(CHF) include patients who have had recent coronary artery disease, orthopnea, exertional dyspnea. Patients who have history of chronic alcoholism and jaundice is suggestive of cirrhosis.careful evaluation of drugs taken and abused previously must be noted down. The likely drugs are ACE inhibitors,cyclosporine,tacrolimus,non steroidal antiinflammatory drugs which affect intrarenal dynamics .

2. Physical examination. Signs of dehydration must be looked for which indicates that there is intravascular volume depletion.

a)decreased sweating especially in the axillary region.

b)decrease in body weight.

c)Orthostatic hypotension:A reduction in systolic pressure of greater than 20 mm Hg or a increase in pulse of greater than 10 beats per minute from supine to standing position.

d) tachycardia

e) Dryness of the mucousal areas.

f) delayed relaxation of skin after being pulled up.

g) in supine position JVP will not be visible.

b. Physical findings usually found in arterial underfilling states with an excess of ECF include:

- Rised jugular venous pressure,ascites,pitting pedal edema

CHF is identified by:

- Basal crepitations,S3 gallop.

Signs of hepatic failure include Jaundice,shrunken liver, Palmar erythema Spider nevi.

Urinary findings: the urinary findings will be the same in prerenal AKI regardless of the cause of prerenal AKI whether it is due to hypovolumia or arterial underfilling or drugs.

a. The urine dipstick will be negative for protein,blood and nitrate. The specific gravity will be greater than 1.020.

b. There might be presence of hyaline or granular casts.

Urine indices

DIAGNOSTIC INDEX	PRERENAL AKI	ACUTE TUBULAR NECROSIS
FENa(fractional excretion of sodium)	<1%	>1%
Urine sodium levels	<20	>40
Urine cr/plasma cr ratio	>40	<20
Urine urea nitrogen/plasma urea nitrogen ratio	>8	<3
Urine specific gravity	>1.018	1.010
Urine osmolality	>500	Around 300
Renal failure index	<1	>1
Urine sediments	Hyaline casts	Granular casts
Plasma blood urea nitrogen/creatinine ratio	>20	<10-15

Radiologic evaluation:

Radiologic evaluation helps to identify if there is any obstruction causing AKI. It also helps to assess the size of kidneys and to differentiate AKI from CKD.

RENAL BIOPSY:

Renal biopsy is not needed in prerenal AKI and is used in intrinsic AKI when the cause is not known. It is used when nephrotoxic injury and ischemic injury have been ruled out and when there is a specific diagnoses which may respond to therapy.

Management:

The aim of management of acute kidney injury is to preserve the underlying renal function and as well as to avoid onset of complications like acidosis,electrolyte disturbances and volume overload and the need for dialysis.

The important things is avoidance of AKI which can be achieved by the following measures:

Preventive measures:

Fluid management:

Maintenance of adequate renal blood flow is the most necessary prophylactic measure, with majority of patients developing postoperative acute kidney injury having an episode of hypotension or increased blood loss in the intraoperative period. Adequate amount of fluids is therefore important and caution must be exercised as there can be complications arising from excessive fluid causing impaired wound healing and prolonged time of mechanical ventilation . There is accumulating support that maintenance of positive fluid balance in critical patients and in postoperative patients is leading to an raise in intra-abdominal pressure which further leads to deterioration of renal function . There will be development of hyperchloremia due to multiple infusions of 0.9% normal saline

which further leads to decrease in renal blood flow and function. Studies have not been able to establish a clear advantage of using restrictive versus liberal fluid therapy and the need for dialysis or poorer outcome in patients who have received liberal fluid therapy.A system of target based approach with attention to specific endpoints may be more relevant. The goal oriented approach is based on the use of intravenous fluids, blood transfusion and ionotropic supports to reach a specific endpoint . It is necessary to optimize the hemodynamics preop,introp and postoperatively which if balanced correctly leads to decreased morbidity and mortality. This is particularly important when there is lack of resources for preoperative regularization.

There has been a study evaluating the concept of bicarbonate infusion in cardiothoracic surgery. The study demonstrated a decreased incidence in acute kidney injury in patients who are on bicarbonate compared to patients receiving saline infusion. But this study failed to demonstrate any significant difference in need for renal replacement therapy in either sides. There were also no change in mortality rates. Further studies in this field are necessary and looks attractive. The use of colloid solutions as an alternative to albumin has been proposed but starch preparations is associated with AKI. After the initial volume replacement further losses of urinary and gastrointestinal losses should be corrected using hypotonic crystalloid solutions.

Effects of fluid overload:

It causes tissue edema. It affects multiple organs,the ffects of fluid overload on various organs are discussed below.

Brain: altered mental status and confusion.

HEART:arrhythmia,systolic and diastolic dysfunction.

LUNGS:impaired gas exchange and increased work of breathing.

LIVER: cholestasis.

KIDNEYS:decreased renal blood flow and glomerular filtration and venous congestion.

TISSUE:poor healing,pressure ulcers and infections.

Fluid overload worsens tissue perfusion by

Shedding of endothelial glycoclayx, triggered by hypervolaemia (ANP) & inflammation,Loss of vascular integrity causes leak and leucocyte and platelet adhesion causes microthrombi.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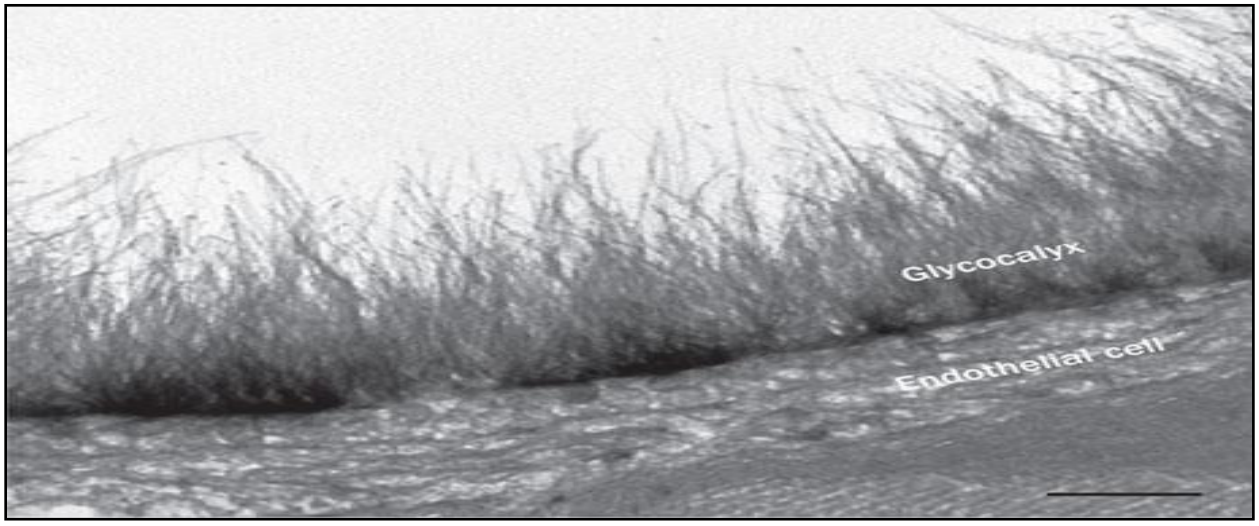

Glycocalyx
Endothelial cell

Avoidance of nephrotoxic agents :A various number of drugs routinely employed in the pre,intra and post op period have necessarily dangerous implications on kidney function. ACE and angiotensin receptor blockers and nonsteroidal inflammatory drugs are commonly used medications known to alter the mechanism of autoregulation. The decision to whether or not to continue the above said drugs in the perioperative period remains under doubt although studies have shown that ACE inhibitors provide no protection. Non steroidal inflammatory drugs can lead to interstitial nephritis and their potency to cause Acute kidney injury have led to the suggestion that it should be withheld in all patients with low volume and systemic inflammatory response syndrome irrespective of kidney function. Antibiotics can also cause acute kidney injury either directly, like aminoglycosides ,penicillins, quinolones and cephalosporins due to interstitial nephritis. So this suggests that monitoring of drug levels can help to reduce the complications. Intravenous contrast has an important role in causing AKI is well recognized and its use should be avoided where it is not necessary, or the minimum possible dose should be given and alternative newer iso-osmolar and low-osmolar non-ionic contrast, can be used which is less toxic. Surgery should be postponed in patients with acute kidney injury due to contrast. The role of N-acetylcysteine offering protection is controversial.

Hemodilution and transfusion in cardiac surgery:

In relation to cardiothoracic surgery, the concept of hemodilution and transfusion have been studied. There is a relation between blood transfusion and acute kidney injury. There is evidence to suggest that the level of hemoglobin in preoperative period has an role, related with a increased incidence of Acute kidney injury. There are studies to assess the role of erythropoietin in this setting but its effectiveness has to be studied further before advising in clinical setting.

Hemodilution occurs in cardiac surgeries during cardio pulmonary bypass,it decreases viscosity of the blood and improves microcirculatory flow in both decreased perfusion and hypothermia. It is associated with increase in acute kidney injury and, so recently lot of importance is attached to underline the importance of reducing hemodilution, with recommendations to maintain hematocrit greater than twenty one percent and hemoglobin greater than seven.

Pharmacological interventions:

There are numerous efforts to identify drug interventions in the treatment of acute kidney injury.Till recently there were no drugs that have shown any consistent benefit but there is now some evolving evidence in the cardiac surgery. The use of Dopamine has been studied and discussed over the years because of the assumption that increase in the kidney blood flow seen with dopamine is important

in the management of acute kidney injury. A meta-analysis which was done in 2001 failed to show any benefit in using dopamine for either the prevention or treatment of acute kidney injury.

Fenoldopam:

Fenoldopam is a dopamine agonist which till date has had varied results when used in the management of acute kidney injury. In cardiothoracic surgery, fenoldopam was identified as to persistently decrease the requirement for renal replacement therapy and morbidity and mortality, but it has a side effect of causing systemic hypotension. This side effect can be avoided with the use of infusions intrarenally, which in limited cases has been proven to be successful but larger trials are needed.

Diuretics:

The advantage of diuretics may improve urine volume in the phase of acute kidney injury, but evidence is not convincing to say that the use of diuretics will reduce the complications or the mortality or the need for renal replacement therapy. It is also shown that frusemide is not only non beneficial but also deleterious in causing increase in serum creatinine values in post operative patients.

Mannitol is added to the initial solution which is used in Cardio pulmonary bypass surgery. At first it was shown to provide some preventive role in children who are undergoing cardio pulmonary bypass surgery, but subsequent studies did not demonstrate this, with chances that mannitol is infact related with tubular injury when given along with dopamine.

Atrial natriuretic peptide (ANP):

Atrial natriuretric peptide is synthesized by the atrium in the heart in stimulation to dilatation of the atrium and it has the advantages of an endogenous diuretic which led to the subsequent evaluation of ANP as a therapy. Initaial randomized trials identified a advantage in select set of patients with decreased urine output which was not able to be reproduced in further studies and it was with associated with hypotension which complicated the therapy. A considerable decrease in the requirement for Renal replacement therapy was sometimes seen in post operative patients who underwent cardiac surgery who obtained minimal-dose infusions of recombinant human atrial natriuretic peeptide. The minimal dose therapy was related with a diminished occurence of hypotension. Besides cardiac surgery, there is no role for ANP in other surgeries.

Nesiritide : Nesiritide is a substance which is a peptide hormone by heart. Nesiritide has been shown to be advantageous in cardiac surgery and other abdominal and vascular surgery.it has shown to reduce mortality. In decompensated heart failure it has been shown to cause increased mortality.

Theophylline:

Theophylline, is a drug whose primary mode of action is as adenosine antagonist, by doing so it helps to maintain blood flow to the renal vessels by reducing the vasoconstriction of renal vessels. Several studies about theophylline have been studied but none were satisfactory and it was asvised to conduct a randomized trial on theophylline for further evaluation and to assess its benefit. In cardio thoracic surgeries theophylline was not helpful in reducing the occurrence of acute kidney injury.

N-acetylcysteine:

N-acetylcysteine has been studied in limited amount and it has not been shown to offer any benefit,but it does offer some help in contrast induced nephropathy.

Glycemic control:

One trial showed that strict blood sugar control and has shown improved benefits in critical care setting, with reduction in patients having AKI requiring renal replacement therapy. But further studies have not shown this benefit and was not able to prove this advantage of reduction in morbidity in patients having AKI. Recently, in patients undergoing heart surgery,the patients having increased blood sugar levels in perioperative period was related with bad outcomes, but fall in mean blood sugar levels did not cause any persistent benefit in outcome. Given these findings and pit falls, the phenomenon of strict blood sugar control requires further evaluation, with evolving of protocols that give importance on avoiding erratic variations in blood glucose.

SUPPORTIVE MANAGEMENT OF ACUTE KIDNEY INJURY:

Volume overload- salt restriction(less than1-1.5 g/day) and water(<1litre/day)

Hyponatremia- water restriction.

Hyperkalemia- restriction of potassium intake,stopping potassium diuretics,management of hyperkalemia.

Metabolic acidosis:restriction of protein intake.sodium bicarbonate,dialysis.

Hyperphosphatemia: restriction of dietary phosphate intake,phosphate binding agents.

Hypocalcemia-calcium carbonate

Hypermagnesemia-discontinuation of antacids.

Nutrition-restriction of dietary protein,oral nutrition preferred.

Drug dosage-adjustment of dosages for renal function.

Prophylactic RRT:

At present there is no strong evidence to prescribe the use of prophylactic renal replacement therapy in patients having high risk who are subjected to major surgery. The concept of dialyzing someone to prevent dialysis in future is not totally acceptable. A study analyzing patients to undergo prophylactic dialysis before undergoing surgery showed a decrese in incidence in patients going for AKI and patients requiring dialysis and reduced mortality. But this evidence is not seen

in clinical practice.Similarly, in patients who went in for contrast nephropathy, a small study identified that prophylactic dialysis was related with a decrease in complications like patients requiring dialysis but these findings are limited by the decrease in strict protocols. It requires more evidence before such an invasive strategy can be recommended.

Renal replacement therapy in acute kidney injury:

The main aim is to avoid acute kidney injury; when it occurs however, renal replacement therapy plays a main role in the management, with many patients requiring dialysis. After many years of studies and trials, it is not clear as to the correct timing for initiation of renal replacement therapy, but it is seen as a important factor which affects outcome in critically ill patients. Previous studies showed a improved outcome in patients receiving higher dose of dialysis or hemofiltration but this finding was not confirmed in subsequent findings

WHAT ARE THE INDICATIONS FOR REPLACEMENT THERAPY:

Hypervolumia which is not responding to diuretics.

Hyperkalemia which is not being controlled medically.

Metabolic acidosis which is persisting.

Uremic symptoms. Persistent oliguria.

Complications of acute kidney injury:

The AKI results in multiple disturbances in fluid,electrolyte and acid base balance of the body. It also affects the various functions of the body.

Disorders of potassium balance:

The rise in potassium called as hyperkalemia is a important and a dangerous complication of AKI. The potassium rises by 0.5 meq /day in patients who are anuric and is due to the potassium derived from the patients diet,drugs taken by the patient and the solutions containing potassium. It also may be complicated by coexisting metabolic acidosis and hyperglycemia and other hyper osmolar states that promotes potassium outside the cells and rises the serum potassium values. When potassium levels are high at the time of diagnosis of AKI conditions such as rhabdomyolysis or any destruction of RBC'S or tumour lysis must be suspected. Mild increase in potassium is usually asymptomatic,but higher levels are usually associated with ecg changes such as tall peaked T waves,prolongation of PR interval,flattening of P waves,and widening of the QRS complex. These ecg findings may be a precursor for an arrhythmia such as ventricular tachycardia and asystole. It also may cause neuromuscular abnormalities such as decreased

reflexes,flaccid paralysis and paralysis of the respiratory muscles. Low levels of potassium(hypokalemia) is usually not seen in AKI but may be seen in nonoiguric ATN caused by aminoglycosides, cisplatin or amphotericin B,because of impaired absorption of potassium due to epithelial cell injury because of these drugs.

Disturbances in acid base balance:

The normal daily food intake results in a 50 to 100 mmol/day of acids which is discarded by the kidneysto maintain the acid base balance. So when there is a renal injury there will a disturbance of acid base balance.AKI is usually accompanied by metabolic acidosis with increased anion gap. The acidosis may be severe when there is coexisting diabetes causing diabetic keto acidosis,underlying sepsis,lactic acidosis causing decreased tissue perfusion. There can be also metabolic alkalosis when there is a enthusiastic correction of acidosis by the bicarbonate or aspiration of gastric juices by continous ryles tube drainage. The disturbances of acid base balance is a very serious and potential threat due to AKI because it might lead to cardiac disturbances and might result in poor wound healing and they are associated with increased morbidity and mortality. Hyperkalemia also leads to metabolic acidosis which leads to dangerous condition where there is increased chances of cardiac arrhythmias.

DISTURBANCES OF MINERAL AND URIC ACID METABOLISM:

In AKI there is disturbance of phosphate balance,mild disturbance is usually seen but major disturbances in phosphate balance is usually seen in patients having sustained severe burns,hemolysis or tumour lysis or patients having tumour lysis. Hypocalcemia might occur due to decrease in 1,25 hydroxylase which is synthesized in kidneys.hypocalcemia leads to simple fasciculations to serious cardiac disturbances. Hypocalcemia is usually asymptomatic because it is counterbalanced by the acidosis on neuromuscular excitability. The manifestations of hypocalcemia include perioral paresthesias,muscle cramps, seizures, confusion and hallucinations. The ecg findings include prolongation of QT interval and nonspecific T wave changes on ecg. Trousseaus sign and chovstek sign are seen in hypocalcemia. These are useful indicators of latent tetany in patients having hypocalcemia.

Hypermagnesemia is seen in patients having AKI with oliguria and is due to impaired excretion of magnesium which is been absorbed by the body through antacids,laxatives. Hypomagnesemia is also seen with drugs such as cisplatin and amphotericin. It commonly occurs when there is injury to the thick ascending limb of loop of henle which is the important site for magnesium absorption. It is usually asymptomatic and is seen as irritability,seizures

arrhythmias. It is also one of the causes for resistant hypokalemia and hypocalcemia.

Uric acid is excreted by glomerular filtration and secreted by proximal tubular cells and some amount of uricemia is seen in AKI.

Cardiac complications due to volume overload:

Volume overload commonly occurs in AKI due to salt and water retention as a result of decreased renal function.it may present as hypertension,elevated JVP,pleural effusion,ascites,increased body weight and pulmonary edema. Hypervolumia might lead to hyponatremia causing altered sensorium,confusion,seizures. It might complicate leading to MI,cardiac arrhythmias.

Hematological complications:

Anemia might occur in AKI due to inhibition of erythropoiesis, increased bleeding,and reduced RBC survival time. Bleeding is common due to platelet dysfunction.

NUTRITIONAL AND GASTROINTESTINAL COMPLICATIONS:

There is increased protein breakdown and increased catabolism leading to malnutrition. Malnutrition leads to inability to eat,loss of apetite. Malnutrition also occurs due to nutrient losses in drainage fluids and dialysate. Gastrointestinal bleeding might occur in some cases of AKI. It is due to stress ulceration of gastric mucosa.

Infectious complications:

Infection is common in AKI and it complicates AKI and results in increased morbidity and mortality. The increased incidence of infection is due to defect in host immune responses and breaches in mucocutaneous barriers(central line,bladder catherisation).

SEQUALE OF ACUTE KIDNEY INJURY:

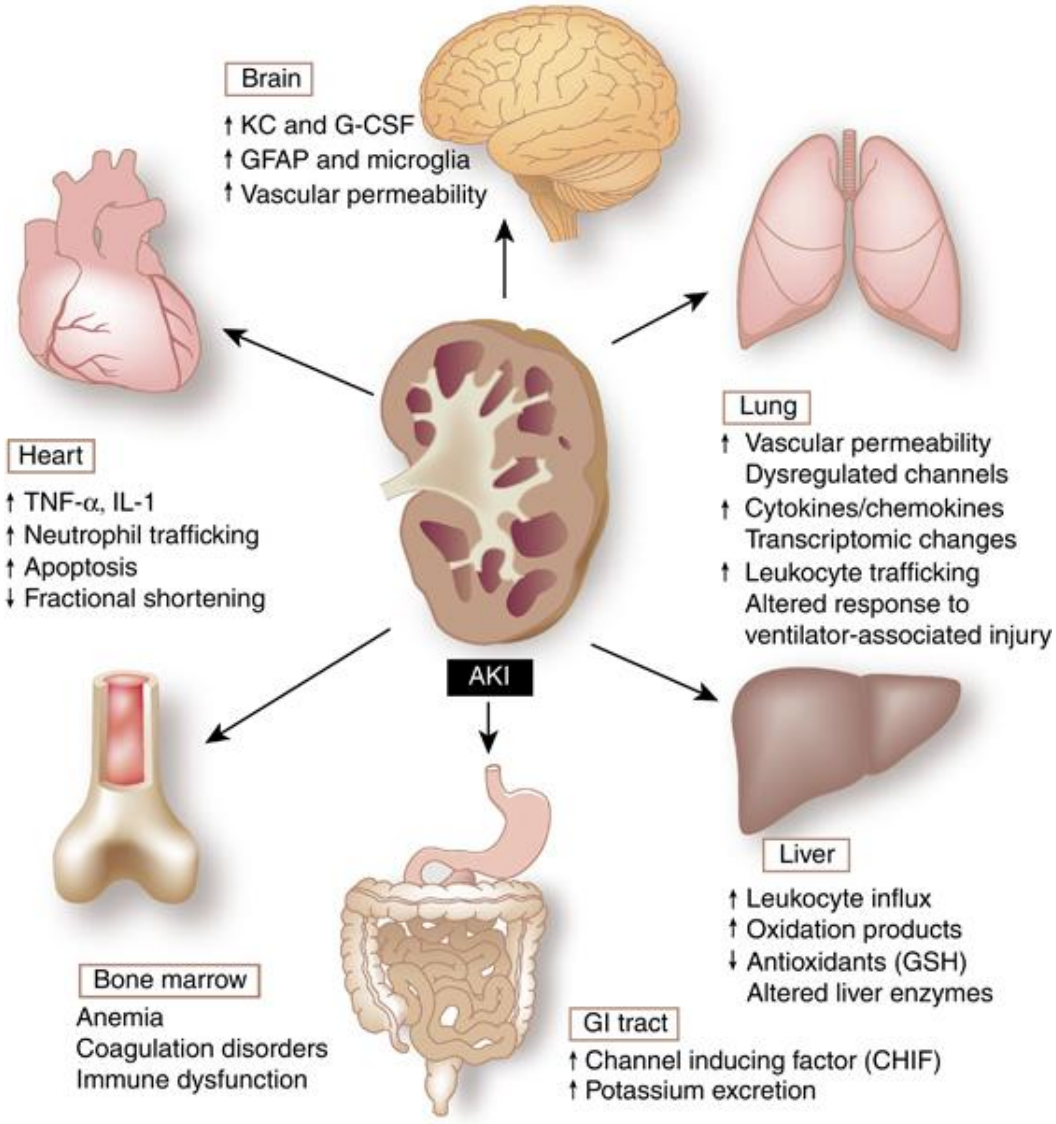
AKI can lead to uremic symptoms and uremia. Clinical signs of uremia includes nausea,vomiting,anorexia,lethargy,confusion,agitation,stupor,coma,asterixis,myoclonus,restless leg syndrome,seizures, focal neurological deficits. The toxin responsible for this has to be identified. Uremic syndrome can lead to cardiac tamponade,pericardial effusion,gastrointestinal complications like bleeding and hemorraghe.

COMPLICATIONS IN RECOVERY PHASE OF AKI:

Vigorous dieresis complicates the recovery phase of AKI,and results in depletion of intravascular volume that results in depletion of volume and causes in decline in renal function. This phenomenon is due to osmotic dieresis due to retaine urea and waste products and to excrete the retained salt and solutes.

Hypokalemia,hypomagnesemia and hypocalcemia occur during the recovery phase of AKI.

EFFECT OF AKI ON OTHER ORGAN SYSTEMS:



Outcome of ACUTE KIDNEY INJURY:

Acute kidney injury has three possible outcomes,

- patients get back to baseline renal function (recovery may take long periods in elderly patients)
- acute kidney injury leads on to chronic kidney disease in patients who had normal kidneys before.
- Rapid progression of disease in patients having pre-existing chronic kidney disease, fast progression of upto fivefold increased risk for end stage disease.

It is not clear whether patients who recovered from AKI subsequently progress to chronic kidney disease at a later stage.

D IALYSIS-TYPES:

Hemodialysis:

Hemofiltration (high convective transport of substances)

Hemodialysis (high transport rates of LMW substances by diffusion)

Hemodiafiltration (both)

Peritoneal dialysis

CAPD – Continuous Ambulatory Peritoneal Dialysis

APD – Automated Peritoneal Dialysis

Hemodialysis relies on the principles of solute diffusion across a semipermeable membrane. Movement of metabolic waste products takes place down a concentration gradient from the circulation into the dialysate. Movement is via

both diffusive clearance (dialysis) and convective clearance (ultrafiltration).The removal of small solutes occurs primarily by diffusion while larger components are effectively removed by convection.

There are three essential components to hemodialysis:

- The dialyzer,
- The composition and delivery of the dialysate, and
- The blood delivery system

The HD procedure is targeted at removing both low- and high-molecular-weight solutes.Fractional removal of urea nitrogen and its derivations are considered to be the standard methods by which "adequacy of dialysis" is measured.For the majority of patients with ESRD, between 9 and 12 h of dialysis are required each week, usually divided into three equal sessions. Several studies have suggested that longer hemodialysis session lengths and more frequent cycles may be beneficial.

COMPLICATIONS OF HEMODIALYSIS:

- ⦿ Hypotension
- ⦿ Muscle cramps
- ⦿ Dialysis disequilibrium syndrome

- Restless leg syndrome
- Arrhythmia and angina
- hypoglycemia
- Hemorrhage
- Blood membrane interaction
- Intradialytic hemolysis

PERITONEAL DIALYSIS:

- CAPD
- Dry day APD
- CCPD
- APD with two Day Dwells
- APD with short day Dwells
- TIDAL PD with no day dwell

COMPLICATIONS OF PERITONEAL DIALYSIS:

Peritonitis

- Gram positive 71% (MC-CONS, Staph aureus)
- Gram negative 24%(MC-E.coli)

Mechanical complications

- Hernias
- Diaphragmatic tear – hydrothorax
- Gastric reflux

Metabolic complications

- Hyperglycemia
- Hyperlipidemia
- Hypoalbuminemia
- Hypokalemia>hyperkalemia
- hyponatremia

AIMS AND OBJECTIVES

1)To assess the incidence of acute kidney injury in postoperative patients.

2)To analyze the various risk factors for acute kidney injury in post operative patients in Stanley hospital.

MATERIALS AND METHODS

PLACE OF STUDY:

DEPARTMENT OF NEPHROLOGY,

DEPARTMENT OF SURGERY,

STANLEY MEDICAL COLLEGE CHENNAI.

DURATION OF STUDY:FEBRUARY 2014 TO SEPTEMBER 2014

STUDY DESIGN:

PROSPECTIVE AND OBSERVATIONAL STUDY

SOURCE OF DATA:

Patients in postoperative period with acute kidney injury in stanley medical college hospital,during the period of february to september 2014fulfilling the inclusion and exclusion criteria..

SAMPLE SIZE:60 patients.

INCLUSION CRITERIA:

patients in postoperative period after major cardiac, vascular,hepatobiliary and other surgeries with acute kidney injury.

EXCLUSION CRITERIA:

patients having chronic kidney disease.

METHODOLOGY:

Patients undergoing major surgeries like cardiac surgeries,vascularsurgeries,hepatobiliary surgeries and other major surgeries in STANLEY MEDICAL COLLEGE HOSPITAL for a duration of 6 months are included in the study.

All pateientspre operativeRFT AND URINE OUTPUT AND POST OPERATIVE RFT AFTER 48 HOURS and upto 7 days are followed up.

Patients comorbid conditions,and the type of surgery,drugs used ,intra operative hypotension and blood transfusions are taken into account.

AKI is assessed based on AKIN and RIFLE criteria.

The patients requiring dialysis and the recovery of patients is assessed.

Data will be collected using a pretested proforma meeting the objectives of the study,and necessary investigations will be done and the nature of study will be explained to the patient and informed consent be obtained.

The analysis of data will be done using appropriate statistitical methods.

Study Groups

Treatment Groups	Name of Group	Study	Number of Subjects
Group A	AKI -	Acute Kidney Injury absent in post-operative patients based on increased serum creatinine post operatively	29
Group B	AKI +	Acute Kidney Injury present in post-operative patients based on increased serum creatinine post operatively	31

RIFLE criteria and AKIN criteria were used to classify patients developing AKI. When the criteria of 0.3 mg/dl rise of creatinine was used all patients were seen to Acute kidney injury,but when > 50% rise was applied the above patients were Classified into AKI- and AKI+.

Statistics

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with Unpaired t test and categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010.

Sample Size Calculation

Sample size was determined on the basis of a pilot study in which the presence of acute kidney injury in post-operative patients was measured at 4%. We calculated a minimum sample size of 59 patients was required, assuming a type 1 error (two-tailed) of 0.05 and a margin of error of 10%. Therefore, the final sample selected was n=60.

$$n = \frac{t^2 \times p(1-p)}{m^2}$$

Description:

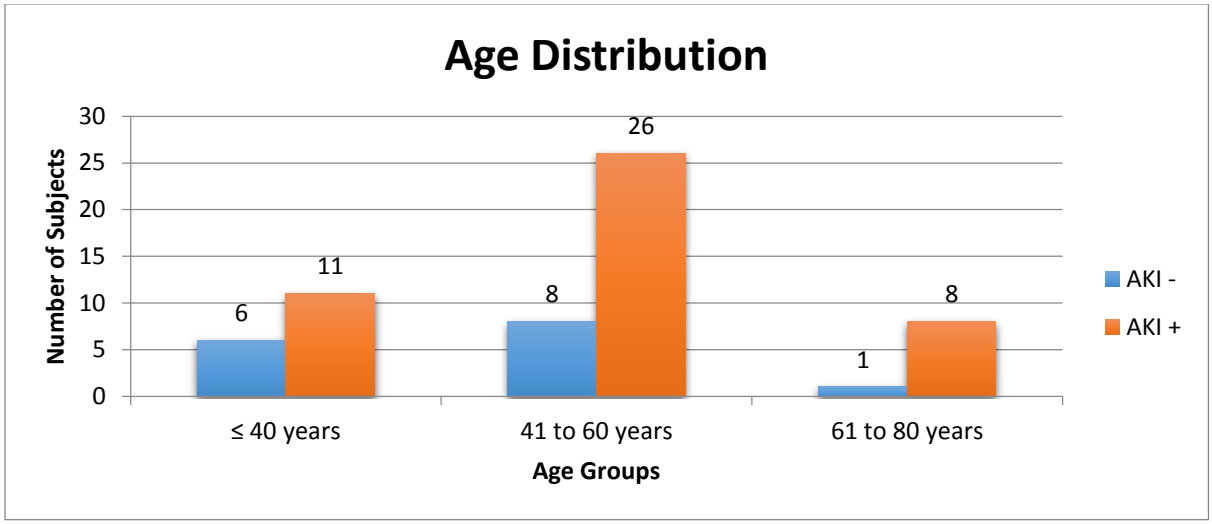
n = required sample size
t = confidence level at 95% (standard value of 1.96)
p = estimated prevalence of malnutrition in the project area
m = margin of error at 10% (standard value of 0.05)

$$n = \frac{(1.96)^2 \times 0.04(1-0.96)}{(0.05)^2}$$

$$n = \frac{3.8146 \times 0.0384}{0.0025}$$

$$= 59$$

Age

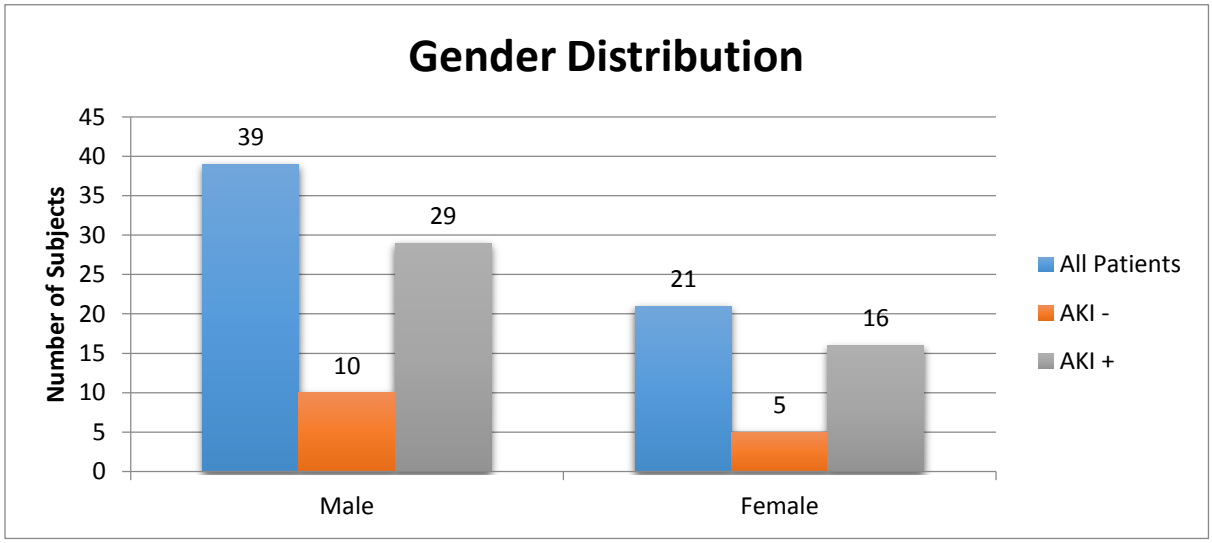


Age Distribution	All Patients	%	AKI -	%	AKI +	%
≤ 40 years	17	28.33	6	40.00	11	24.44
41 to 60 years	34	56.67	8	53.33	26	57.78
61 to 80 years	9	15.00	1	6.67	8	17.78
Total	60	100	15	100	45	100

Age Distribution	AKI -	AKI +
N	15	45
MEAN	42.87	48.13
SD	10.13	12.84
P value	0.114450633	
Unpaired t test		

By conventional criteria the association between the study groups and age is considered to be not statistically significant since p > 0.05

Gender

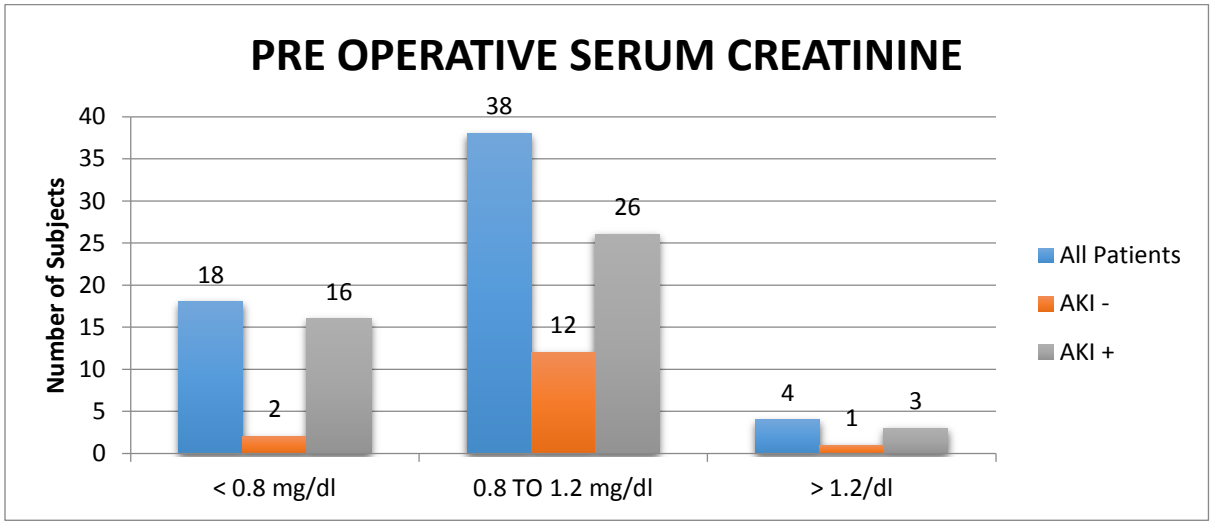


Gender Distribution	All Patients	%	AKI -	%	AKI +	%
Male	39	65.00	10	66.67	29	64.44
Female	21	35.00	5	33.33	16	35.56
Total	60	100	15	100	45	100
Chi-square value		0.660				
Degrees of freedom		1				
P value		0.935				
Chi Squared Test						

By conventional criteria the association between the study groups and gender is considered to be not statistically significant since p > 0.05.

Since age and gender is not statistically significant, it means that there is no difference between the groups. In other words the groups contain subjects with the same basic demographic characteristics.

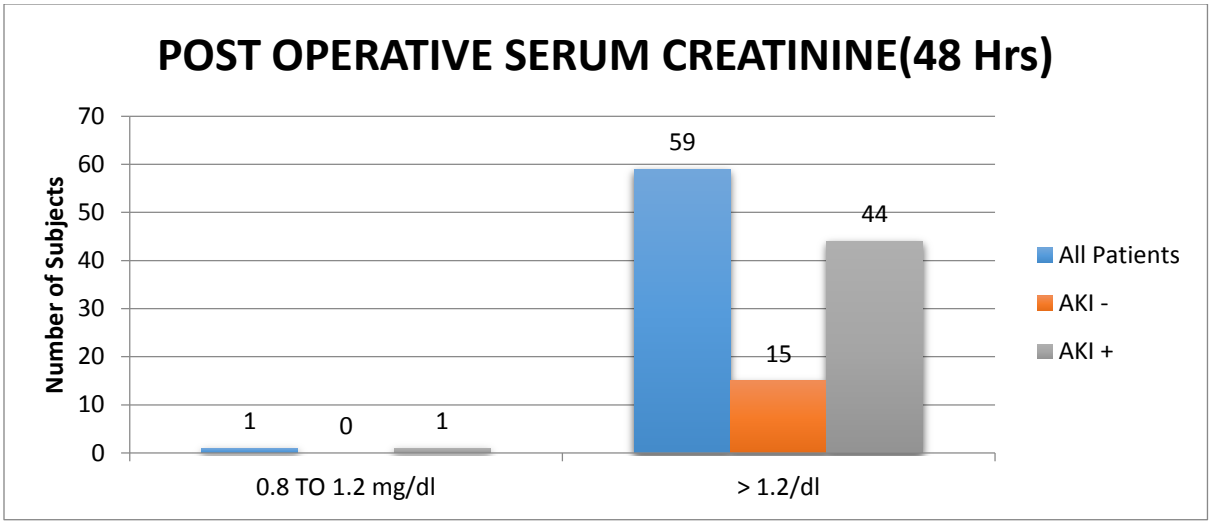
Serum Creatinine



PRE OP SERUM CREATININE	All Patients	%	AKI -	%	AKI +	%
< 0.8 mg/dl	18	30.00	2	13.33	16	35.56
0.8 TO 1.2 mg/dl	38	63.33	12	80.00	26	57.78
> 1.2/dl	4	6.67	1	6.67	3	6.67
Total	60	100	15	100	45	100

PRE OP SERUM CREATININE	AKI -	AKI +
N	15	45
MEAN	0.86	0.85
SD	0.19	0.33
P value	0.872576421	
Unpaired t test		

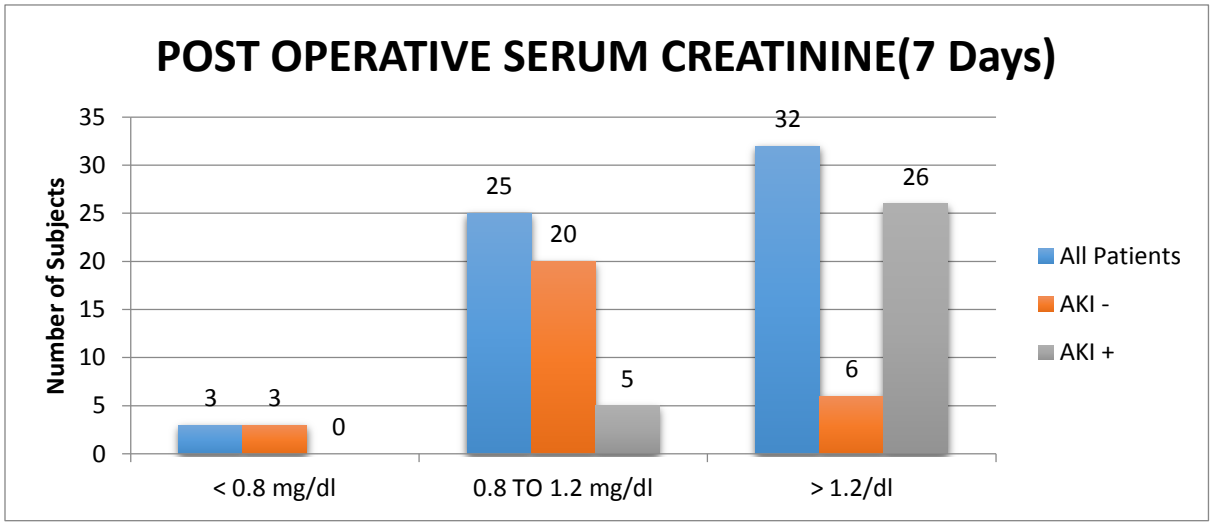
By conventional criteria the association between the study groups and pre-operative serum creatinine levels is considered to be not statistically significant since $p > 0.05$



POST OP CREATININE (48 HRS)	All Patients	%	AKI -	%	AKI +	%
< 0.8 mg/dl	0	0.00	0	0.00	0	0.00
0.8 TO 1.2 mg/dl	1	1.67	0	0.00	1	2.22
> 1.2/dl	59	98.33	15	100.00	44	97.78
Total	60	100	15	100	45	100

POST OP CREATININE(48 HRS)	AKI -	AKI +
N	15	45
MEAN	1.67	1.79
SD	0.33	0.45
P value	0.0282724462	
Unpaired t test		

By conventional criteria the association between the study groups and post-operative serum creatinine levels(48 hrs) is considered to be statistically significant since $p < 0.05$



POST OP CREATININE (7 DAYS)	All Patients	%	AKI -	%	AKI +	%
< 0.8 mg/dl	3	5.00	2	13.33	1	2.22
0.8 TO 1.2 mg/dl	25	41.67	12	80.00	13	28.89
> 1.2/dl	32	53.33	1	6.67	31	68.89
Total	60	100	15	100	45	100

POST OP CREATININE(7 DAYS)		AKI -	AKI +
N		15	45
MEAN		1.04	1.89
SD		0.18	1.04
P value	0.000003		
Unpaired t test			

By conventional criteria the association between the study groups and post-operative-serum creatinine levels(7 days) among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the occurrence of acute kidney injury among post operative patients post-operative-serum creatinine levels(7 days)in AKI - group is predominantly less when compared to AKI + group. It is statistically significant with a p-value of 0.0000 according to unpaired t test.

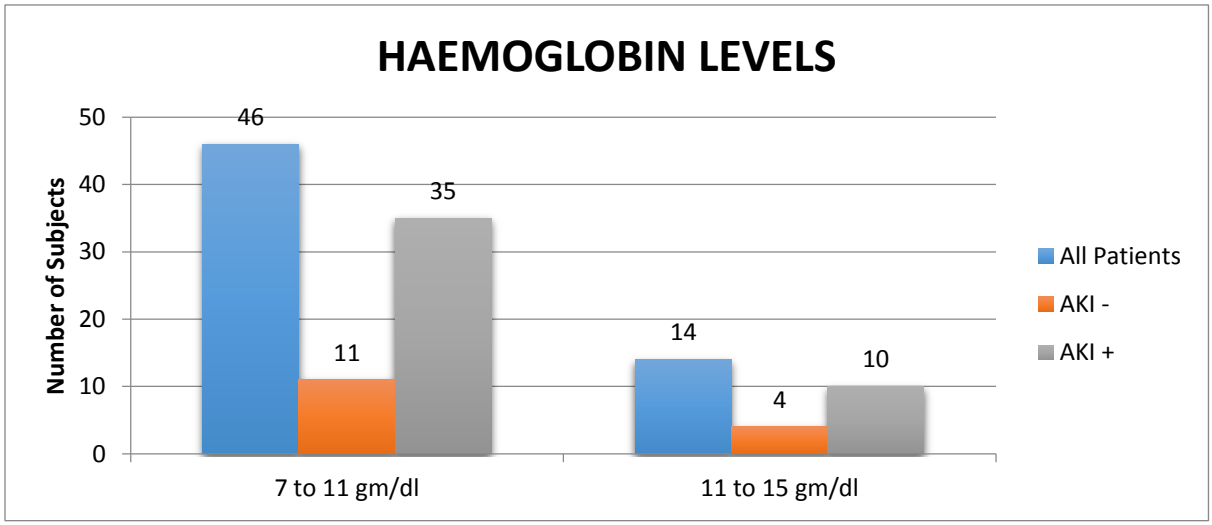
Clinical Significance

The , the percentage of post-operative-serum creatinine levels(7 days)is meaningfully less in AKI – group than AKI + group by 1.81 times with a difference of 0.85mg/dl between the groups. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in post-operative-serum creatinine levels in AKI + group. So patients developing acute kidney injury are more lkely to have highly elevated post-operative-serum creatinine levels.

HB

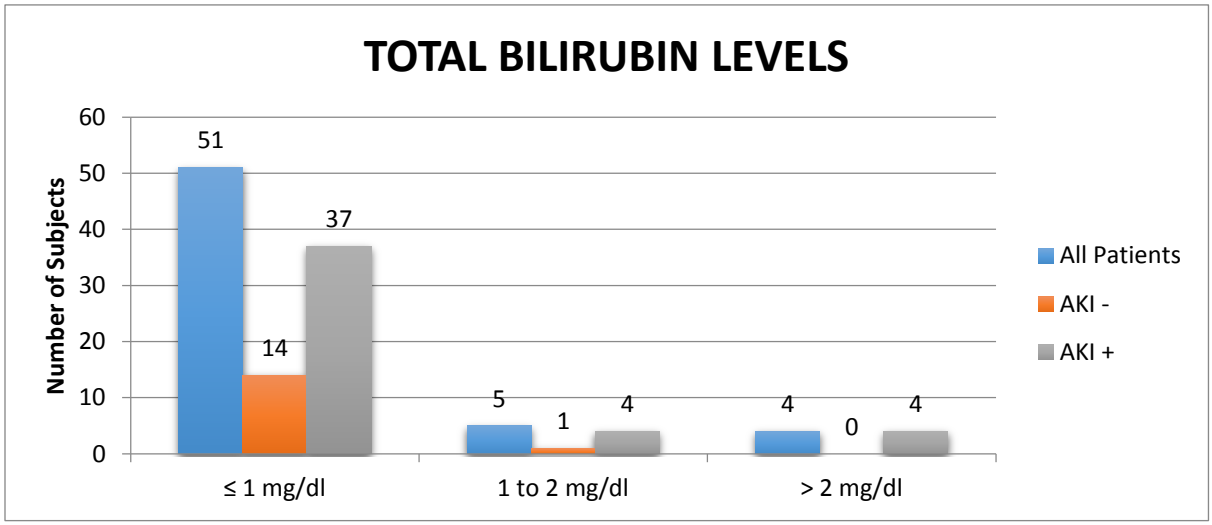


HB	All Patients	%	AKI -	%	AKI +	%
< 7 gm/dl	0	0.00	0	0.00	0	0.00
7 to 11 gm/dl	46	76.67	11	73.33	35	77.78
11 to 15 gm/dl	14	23.33	4	26.67	10	22.22
Total	60	100	15	100	45	100

HB	AKI -	AKI +
N	15	45
MEAN	10.65	10.50
SD	1.29	1.09
P value	0.692246404	
Unpaired t test		

By conventional criteria the association between the study groups and haemoglobin levels is considered to be not statistically significant since $p > 0.05$

Bilirubin

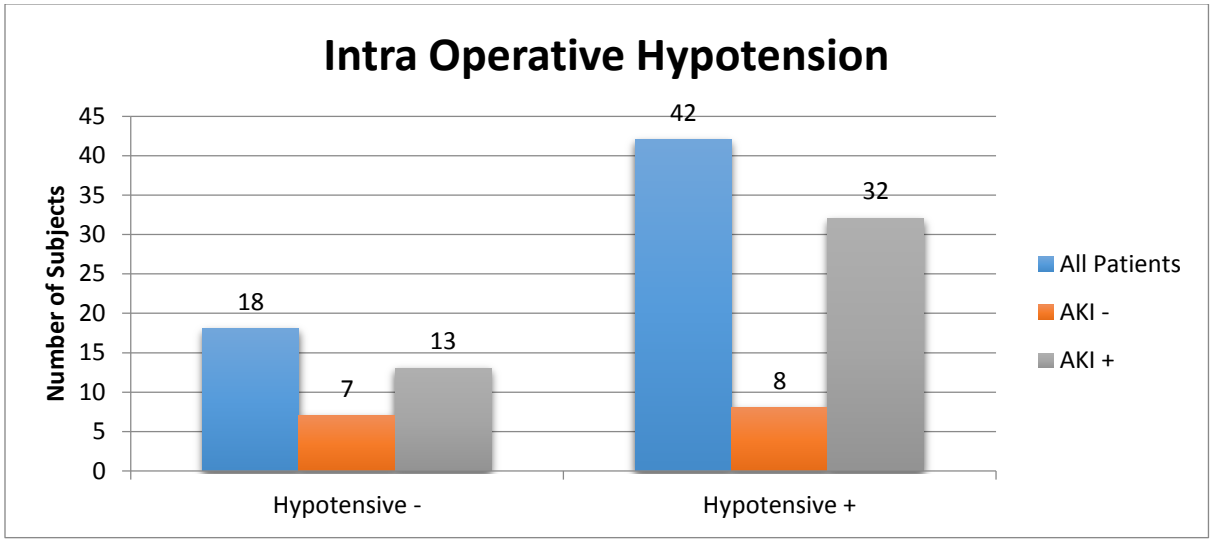


BILIRUBIN	All Patients	%	AKI -	%	AKI +	%
≤ 1 mg/dl	51	85.00	14	93.33	37	82.22
1 to 2 mg/dl	5	8.33	1	6.67	4	8.89
> 2 mg/dl	4	6.67	0	0.00	4	8.89
Total	60	100	15	100	45	100

BILIRUBIN	AKI -	AKI +
N	15	45
MEAN	0.82	1.35
SD	0.11	2.16
P value	0.110137241	
Unpaired t test		

By conventional criteria the association between the study groups and serum bilirubin levels is considered to be not statistically significant since $p > 0.05$.

Intra Operative Hypotension



PRE OP HYPOTENSION	All Patients	%	AKI -	%	AKI +	%
Hypotensive -	18	30.00	7	46.67	13	28.89
Hypotensive +	42	70.00	8	53.33	32	71.11
Total	60	100	15	100	45	100
Chi-square value		5.37				
Degrees of freedom		1				
P value		0.0464				
Chi Squared Test						

By conventional criteria the association between the study groups and intra operative hypotension among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the occurrence of acute kidney injury among post operative patients, intra operative hypotension episodes in AKI - group is predominantly less when compared to AKI + group. It is statistically significant with a p-value of 0.0464 according to chi squared test.

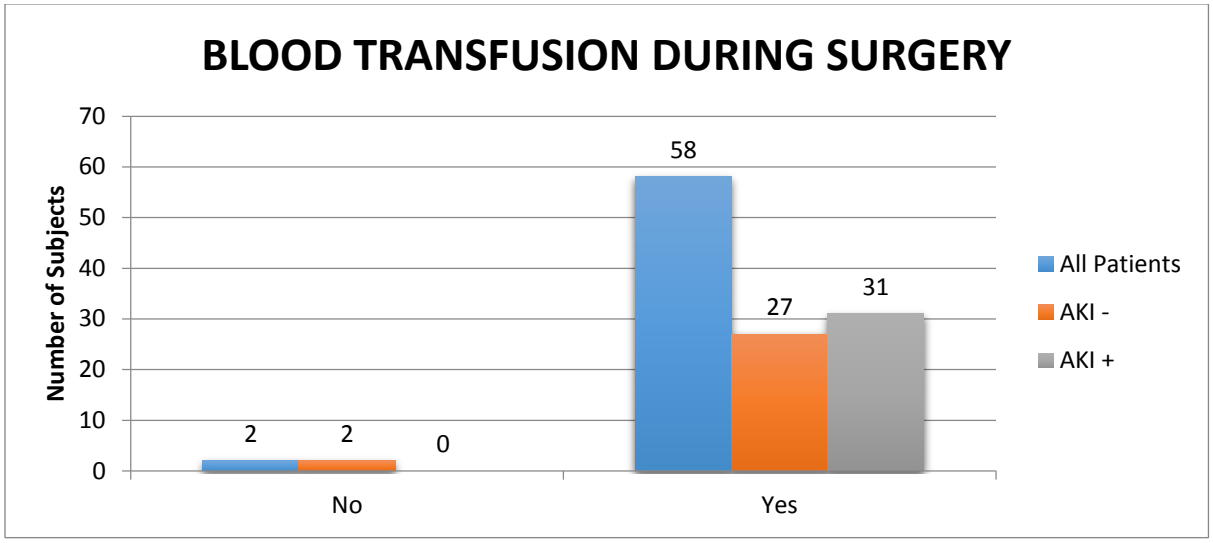
Clinical Significance

The , the percentage of intra operative hypotension episodes is meaningfully less in AKI – group than AKI + group by 1.33 times with a difference of 17.78 percentage points between the groups. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in intra operative hypotension in AKI + group. So patients developing intra operative hypotension episodes are more prone to develop acute kidney injury.

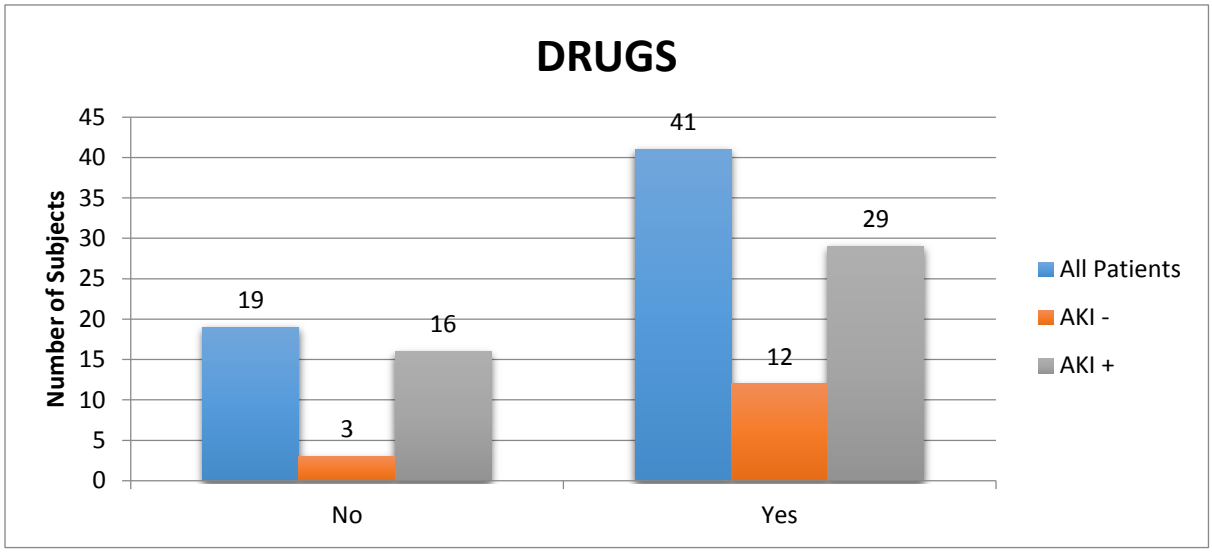
Blood



BLOOD TRANSFUSION DURING SURGERY	All Patients	%	AKI -	%	AKI +	%
No	2	3.33	2	13.33	0	0.00
Yes	58	96.67	13	86.67	45	100.00
Total	60	100	15	100	45	100
Chi-square value		2.21				
Degrees of freedom		1				
P value		0.137				
Chi Squared Test						

By conventional criteria the association between the study groups and blood transfusion during surgery is considered to be not statistically significant since $p > 0.05$

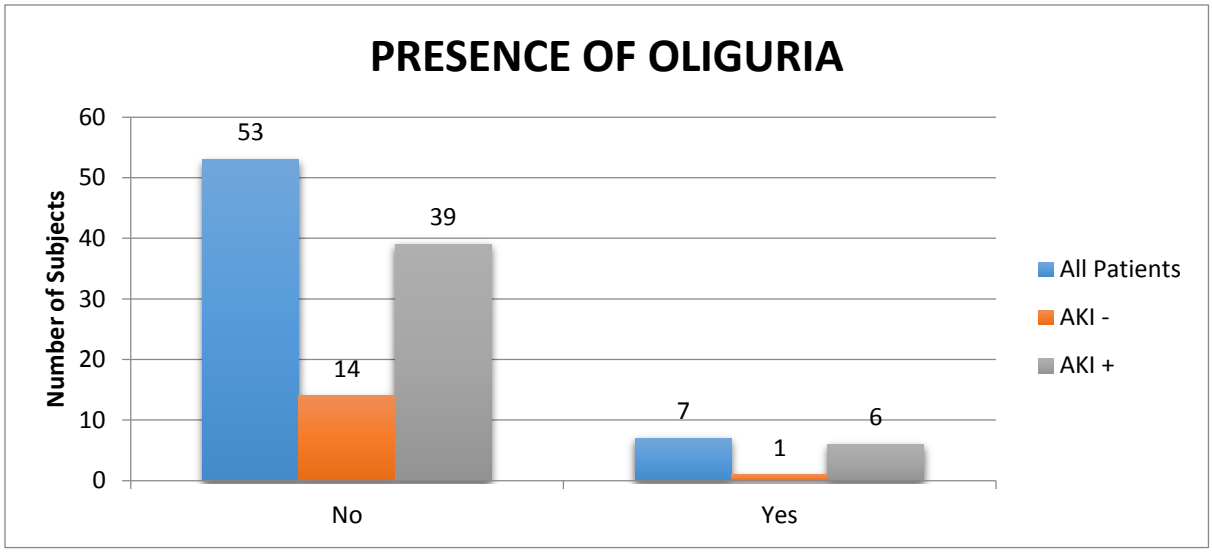
Drugs



DRUGS INTAKE	All Patients	%	AKI -	%	AKI +	%
No	19	31.67	3	20.00	16	35.56
Yes	41	68.33	12	80.00	29	64.44
Total	60	100	15	100	45	100
Chi-square value		0.206				
Degrees of freedom		1				
P value		0.650				
Chi Squared Test						

By conventional criteria the association between the study groups and drug intake during and after surgery is considered to be not statistically significant since $p > 0.05$

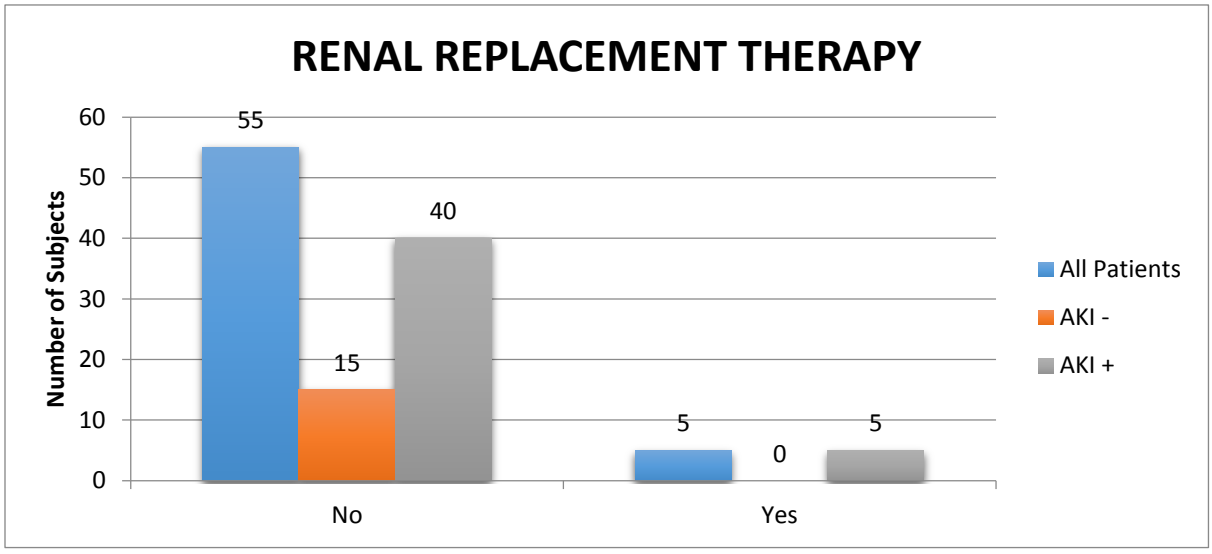
Oliguria



PRESENCE OF OLIGURIA	All Patients	%	AKI -	%	AKI +	%
No	53	88.33	14	93.33	39	86.67
Yes	7	11.67	1	6.67	6	13.33
Total	60	100	15	100	45	100
Chi-square value		3.68				
Degrees of freedom		1				
P value		0.055				
Chi Squared Test						

By conventional criteria the association between the study groups and onset of oliguria after surgery is considered to be not statistically significant since $p > 0.05$

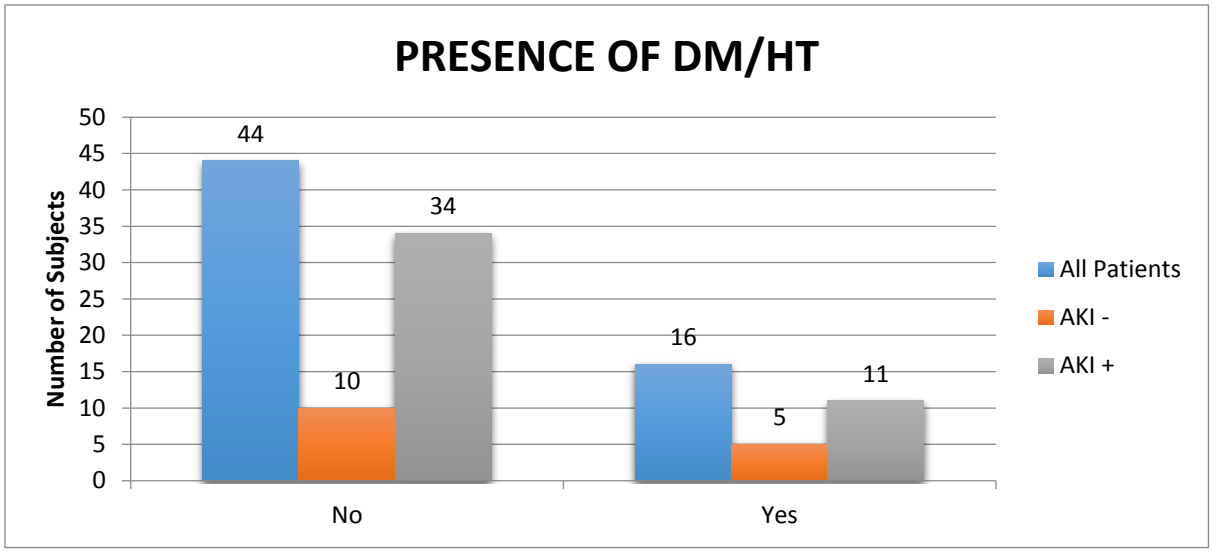
RRT



RENAL REPLACEMENT THERAPY	All Patients	%	AKI -	%	AKI +	%
No	55	91.67	15	100.00	40	88.89
Yes	5	8.33	0	0.00	5	11.11
Total	60	100	15	100	45	100
Chi-square value		1.75				
Degrees of freedom		1				
P value		0.185				
Chi Squared Test						

By conventional criteria the association between the study groups and RRT after surgery is considered to be not statistically significant since $p > 0.05$

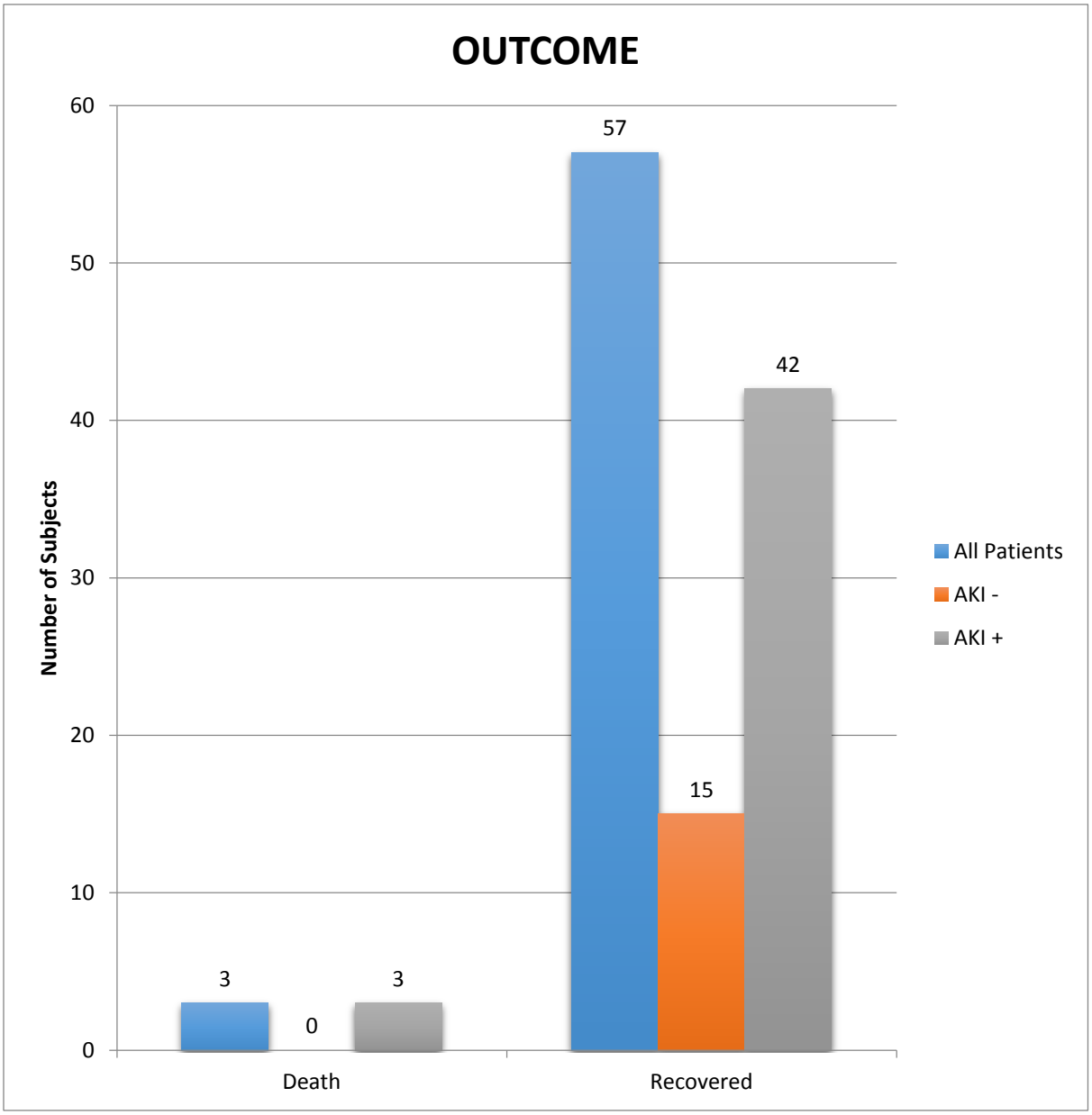
DM/HT



PRESENCE OF DM/HT	All Patients	%	AKI -	%	AKI +	%
No	44	73.33	10	66.67	34	75.56
Yes	16	26.67	5	33.33	11	24.44
Total	60	100	15	100	45	100
Chi-square value		0.243				
Degrees of freedom		1				
P value		0.876				
Chi Squared Test						

By conventional criteria the association between the study groups and presence of DM/HT is considered to be not statistically significant since $p > 0.05$

Outcome


OUTCOME
Number of Subjects
60
50
40
30
20
10
0
57
42
15
3
0
3
Death
Recovered
All Patients
AKI -
AKI +

OUTCOME	All Patients	%	AKI -	%	AKI +	%
Death	3	5.00	0	0.00	3	6.67
Recovered	57	95.00	15	100.00	42	93.33
Total	60	100	15	100	45	100
Chi-square value		2.95				
Degrees of freedom		1				
P value		0.086				
Chi Squared Test						

By conventional criteria the association between the study groups and outcome after acute kidney injury is considered to be not statistically significant since $p > 0.05$

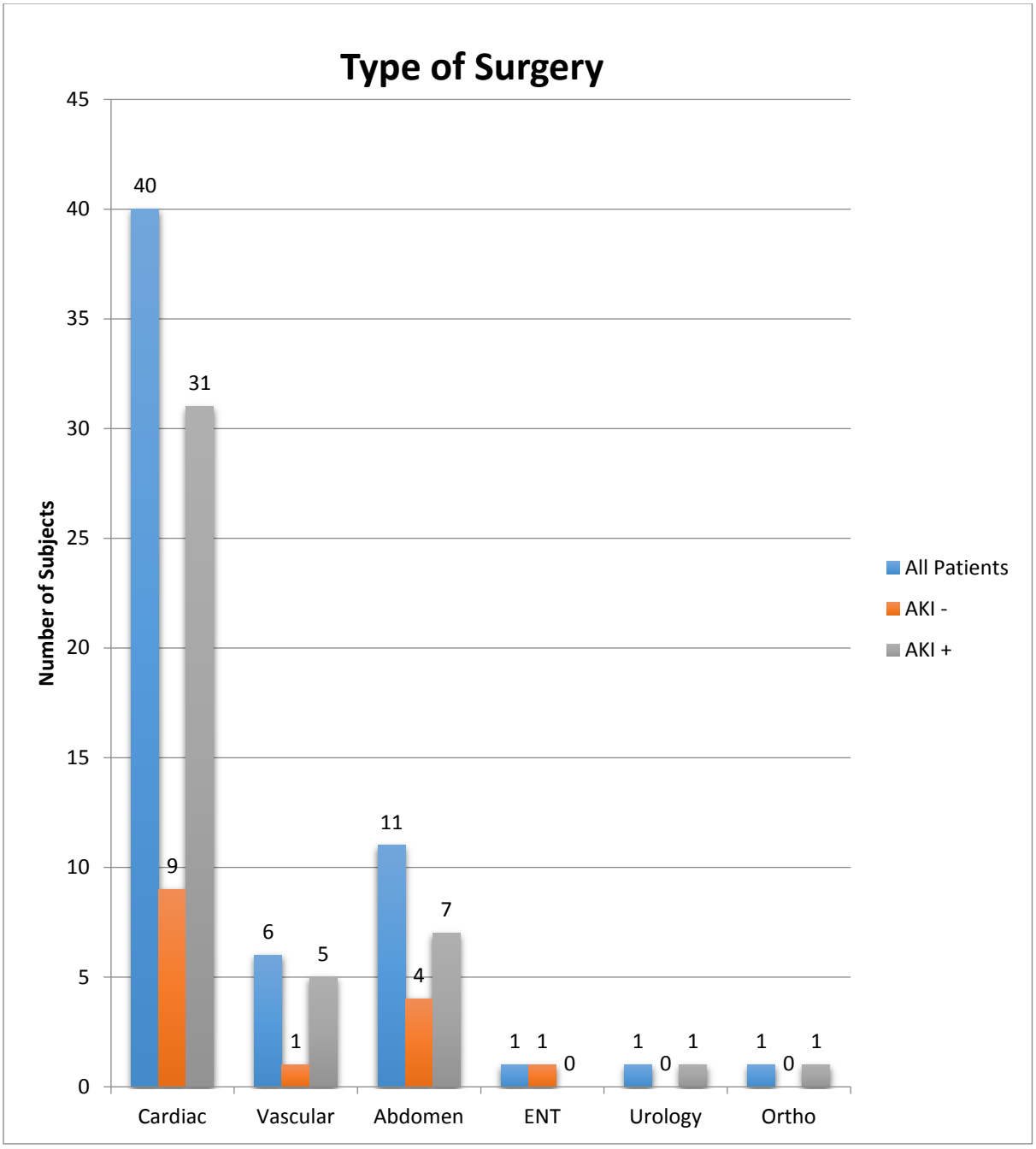
Total number of major surgeries done from February 2014 to September 2014.

4031 major surgeries done from February 2014 to September 2014.

Number of patients developed acute kidney injury-60

INCIDENCE OF AKI : 1.48%

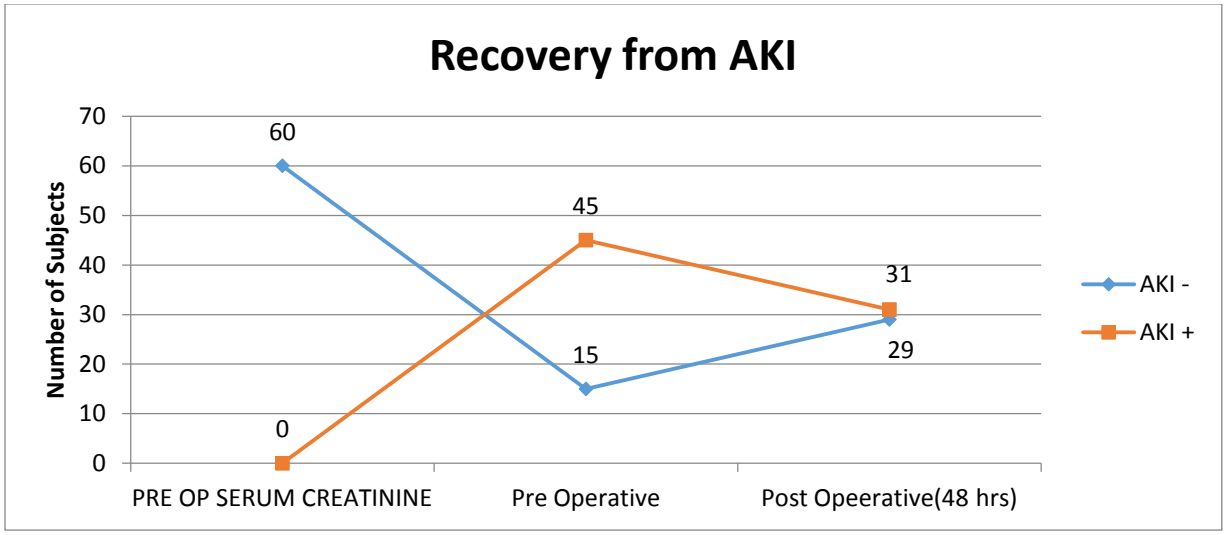
Type of Surgery



Type of Surgeries	All Patients	%	AKI -	%	AKI +	%
Cardiac	40	66.67	9	60.00	31	68.89
Vascular	6	10.00	1	6.67	5	11.11
Abdomen	11	18.33	4	26.67	7	15.56
ENT	1	1.67	1	6.67	0	0.00
Urology	1	1.67	0	0.00	1	2.22
Ortho	1	1.67	0	0.00	1	2.22
Total	60	100	15	100	45	100
Chi-square value		2.05				
Degrees of freedom		6				
P value		0.2221				
Chi Squared Test						

By conventional criteria the association between the study groups and type of surgery is considered to be not statistically significant since $p > 0.05$

Recovery from AKI



Recovery from AKI	PRE OP SERUM CREATININE	%	Pre Operative	%	Post Opeerative(48 hrs)	%
AKI -	60	100	15	25	29	48
AKI +	0	0	45	75	31	52
Total	60	100	60	100	60	100
Chi-square value		3.85				
Degrees of freedom		2				
P value		0.03485				
Chi Squared Test						

By conventional criteria the association between the study groups and recovery from AKI among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the occurrence of acute kidney injury among post operative patients, 42 subjects had AKI at the end of 48 hours and 31 subjects had AKI at the end of 7 days. It is statistically significant with a p-value of 0.03485 according to chi squared test.

Clinical Significance

The , the percentage of AKI patients at the end of 48hours after surgery is meaningfully more (75%) when compared to the percentage of [atients with AKI at the end of 7 days (52%). The recovery from AKI between both time periods is 1.45 times with a difference of 23 percentage points. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in Recovery of patients at the end of 7 days who develop AKI post operatively.

CITATIONS:

1) 2005 American Society of Nephrology

Acute Kidney Injury, Mortality, Length of Stay, and Costs in post operative Patients

Glenn M. Chertow, Elisabeth Burdick, Melissa Honour, Joseph V. Bonventre and David W. Bates.

A study was done in hospitalized post operative patients on 19,982 adults. It was found that large increase in creatinine was rare(>2), moderate increase was common(>0.5 mg/dl). It was associated with increased mortality and costs.

2) Independent association between acute renal failure and mortality following cardiac surgery.

Chertow GM, Levy EM, Hammermeister KE, Grover F, Daley J

The American Journal of Medicine [1998, 104(4):343-348].

42,773 patients undergoing valvular heart surgery or coronary artery bypass surgery were studied and the number of patients going in for acute kidney injury and requiring dialysis were analyzed.

Acute kidney injury occurred in 460 (1.1%) patients. The increased morbidity was around 63.7% in these patients, compared with 4.3% in patients without

this complication. The study concluded that AKI was independently associated with mortality and morbidity following cardiac surgery and early intervention was needed urgently in this aspect.

3) Costs and outcomes of acute kidney injury (AKI) following cardiac surgery

Nephrology Dialysis Transplantationndt.oxfordjournals.org

Joseph F. Dasta1, Sandra L. Kane-Gill2, Amy J. Durtschi3, Dev S. Pathak4 and John A. Kellum5.

3741 patients were studied and classified according to RIFLE criteria and of that 6.9% had acute kidney injury and of that , 3.7% were RIFLE-R, 1.9% RIFLE-I, 1.3% were RIFLE-F. The cost of treatment and the length of stay and the requirement of dialysis were high in these patints.

4) Predictors of postoperative acute renal failure after noncardiac surgery in patients with previously normal renal function.

Kheterpal S[1], Tremper KK, Englesbe MJ, O'Reilly M, Shanks AM, Fetterman DM, Rosenberg AL, Swartz RD.

The study was conducted on adult patients undergoing non cardiac surgery with preoperative renal function of creatinine clearance greater than 80 ml/min and patients were assessed for postoperative acute kidney injury with creatinine clearance less than 50 ml/min for 7 days.65,043 cases were studied,121 patients

developed acute kidney injury (0.8%), and 14 required renal replacement therapy (0.1%). The risk factors found were high risk patients,emergencysurgery,peripheral vascular disease and comorbid illness. It also found that use of diuretics and vasopressors are also associated with acut kidney injury. It was found that in all these patients AKI was associated with increased morbidity and costs.

5) Determinants of postoperative acute kidney injury

Fernando José Abelha, **MiguelaBotelho**, **Vera Fernandes** and **Henrique Barros.**

1597 post op patients were studied in post anaesthesia care unit out of which 1166

Met the criteria for AKI. It found that AKI was associated with independent risk of

Increased morbidity and mortality. The risk factors identified were emergency
Surgery, Coronary artery disease,high risk surgery and congestive heart failure.

6).Bellomo R, Ronco C, Kellum JA, et al, and Acute Dialysis Quality Initiative workgroup. Acute renal failure-definition, outcome measures, animal models, fluidtherapy and information technology needs: the Second International ConsensusConference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care. 2004;8:R204–R212 found that AKI was associated with increased morbidity and mortality in post operative patients.

Conclusion :

1)The risk of developing AKI in patients undergoing surgery were evaluated over a period of 6 months.

2) It was found that patients undergoing cardiac surgery were found to have high risk followed by vascular and other surgeries.

3) The incidence of AKI is 1.48%

4) When patients were classified according to AKIN criteria of >0.3 mg/dl rise of creatinine all the above patients were found to have AKI, but when classified according to RIFLE criteria of >50% rise around 31 patients were found to have AKI.

5) From this study patients undergoing cardiac surgery were found to be at higher risk,and intra operative hypotension was a significant risk factor.

6) The serum creatinine is not a true and early marker of acute kidney injury and there is delayed rise.

7) Most of the patients recovered from AKI after 7 days of surgery.

ANNEXURES

BIBLIOGRAPHY

1. Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery.Circulation. 1999;100:1043–1049.

2. Boersma E, Poldermans D, Bax JJ, et al, and Decrease Study Group (Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography).Predictors of cardiac events after major vascular surgery: role of clinical characteristics,dobutamine echocardiography, and beta-blocker therapy. JAMA. 2001;285:1865–1873.

3. Poldermans D, Boersma E, Bax JJ, et al. The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography Study Group. N Engl J Med. 999;341:1789-1794.

4. Grines CL, Bonow RO, Casey DE Jr, et al, American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, American Dental Association, and American College of Physicians. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from

the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association,
with representation from the American College of Physicians. Circulation. 2007;115:813–818.

5. Rabbitts JA, Nuttall GA, Brown MJ, et al. Cardiac risk of noncardiac surgery after percutaneous coronary intervention with drug-eluting stents. Anesthesiology. 2008;109:596–604.

6. Bellomo R, Ronco C, Kellum JA, et al, and Acute Dialysis Quality Initiative workgroup. Acute renal failure-definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care. 2004;8:R204–R212.

7. Mehta RL, Kellum JA, Shah SV, et al, and Acute Kidney Injury Network. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11:R31.

8. Zanardo G, Michielon P, Paccagnella A, et al. Acute renal failure in the patient undergoing cardiac operation. Prevalence, mortality rate, and main risk factors.J Thorac Cardiovasc Surg. 1994;107:1489–1495.

9. Mangano CM, Diamondstone LS, Ramsay JG, et al. Renal dysfunction after

myocardial revascularization: risk factors, adverse outcomes, and hospital resource utilization. The Multicenter Study of Perioperative Ischemia Research Group. Ann Intern Med. 1998;128:194–203.

10. Yeboah ED, Petrie A, Pead JL. Acute renal failure and open heart surgery. Br Med J.1972;1:415–418.

11. Thakar CV, Arrigain S, Worley S, et al. A clinical score to predict acute renal failure after cardiac surgery. J Am Soc Nephrol. 2005;16:162–168.

12. Chertow GM, Lazarus JM, Christiansen CL, et al. Preoperative renal risk stratification. Circulation. 1997;95:878–884.

13. Thakar CV, Kharat V, Blanck S, et al. Acute kidney injury after gastric bypass surgery.Clin J Am Soc Nephrol. 2007;2:426–430.

14. Kheterpal S, Tremper KK, Englesbe MJ, et al. Predictors of postoperative acute renal failure after noncardiac surgery in patients with previously normal renal function.Anesthesiology. 2007;107:892–902.

15. Gordon AC, Pryn S, Collin J, et al. Outcome in patients who require renal support after surgery for ruptured abdominal aortic aneurysm. Br J Surg. 1994;81:836–838.

16. Barratt J, Parajasingam R, Sayers RD, et al. Outcome of acute renal failure following surgical repair of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg.2000;20:163–168.

17. Boyle JM, Moualla S, Arrigain S, et al. Risks and outcomes of acute kidney injury requiring dialysis after cardiac transplantation. Am J Kidney Dis. 2006;48:787–796.

18. Cabezuelo JB, Ramirez P, Rios A, et al. Risk factors of acute renal failure after liver transplantation. Kidney Int. 2006;69:1073–1080.

19. Yalavarthy R, Edelstein CL, Teitelbaum I. Acute renal failure and chronic kidney disease following liver transplantation. Hemodial Int. 2007;11(suppl 3):S7–S12.

20. Nuno J, Cuervas-Mons V, Vicente E, et al. Renal failure after liver transplantation:analysis of risk factors in 139 liver transplant recipients. Transplant Proc. 1995;27:2319–2320.

21. McCauley J, Van Thiel DH, Starzl TE, et al. Acute and chronic renal failure in liver transplantation. Nephron. 1990;55:121–128.

22. Bilbao I, Charco R, Balsells J, et al. Risk factors for acute renal failure requiring dialysis after liver transplantation. Clin Transplant. 1998;12:123–129.

23. Ishitani M, Wilkowski M, Stevenson W, et al. Outcome of patients requiring hemodialysis after liver transplantation. Transplant Proc. 1993;25:1762–1763.

24. Rimola A, Gavaler JS, Schade RR, et al. Effects of renal impairment on liver transplantation. Gastroenterology. 1987;93:148–156.

25. Chertow GM, Levy EM, Hammermeister KE, et al. Independent association between acute renal failure and mortality following cardiac surgery. Am J Med. 1998;104:343–348.

26. Thakar CV, Liangos O, Yared JP, et al. ARF after open-heart surgery: Influence of gender and race. Am J Kidney Dis. 2003;41:742–751.

27. Star RA. Treatment of acute renal failure. Kidney Int. 1998;54:1817–1831.

28. Berisa F, Beaman M, Adu D, et al. Prognostic factors in acute renal failure following aortic aneurysm surgery. Q J Med. 1990;76:689–698.

29. Braams R, Vossen V, Lisman BA, et al. Outcome in patients requiring renal replacement therapy after surgery for ruptured and non-ruptured aneurysm of the abdominal aorta. Eur J Vasc Endovasc Surg. 1999;18:323–327.

30. Townsend DR, Bagshaw SM, Jacka MJ, et al. Intraoperative renal support during liver transplantation. Liver Transpl. 2009;15:73–78.

31. Chawla SK, Najafi H, Ing TS, et al. Acute renal failure complicating ruptured abdominal aortic aneurysm. Arch Surg. 1975;110:521–526.

32. Olsen PS, Schroeder T, Perko M, et al. Renal failure after operation for abdominal aortic aneurysm. Ann Vasc Surg. 1990;4:580–583.

33. Conlon PJ, Stafford-Smith M, White WD, et al. Acute renal failure following cardiac surgery. Nephrol Dial Transplant. 1999;14:1158–1162.

34. Waikar SS, Liu KD, Chertow GM. Diagnosis, epidemiology and outcomes of acute kidney injury. Clin J Am Soc Nephrol. 2008;3:844–861.

35. Candela-Toha A, Elias-Martin E, Abraira V, et al. Predicting acute renal failure after cardiac surgery: external validation of two new clinical scores. Clin J Am Soc Nephrol.2008;3:1260–1265.Perioperative Acute Kidney Injury ’ 103 www.anesthesiaclinics.com

36. Wald R, Waikar SS, Liangos O, et al. Acute renal failure after endovascular versus open repair of abdominal aortic aneurysm. J Vasc Surg. 2006;43:460, 466; discussion 466.

37. Slogoff S, Reul GJ, Keats AS, et al. Role of perfusion pressure and flow in major organ dysfunction after cardiopulmonary bypass. Ann Thorac Surg. 1990;50:911–918.

38. Orime Y, Shiono M, Hata H, et al. Cytokine and endothelial damage in pulsatile and nonpulsatile cardiopulmonary bypass. Artif Organs. 1999;23:508–512.

39. Sezai A, Shiono M, Orime Y, et al. Major organ function under mechanical support:comparative studies of pulsatile and nonpulsatile circulation. Artif Organs. 1999;23:280–285.

40. Nigwekar SU, Kandula P, Hix JK, et al. Off-pump coronary artery bypass surgery and acute kidney injury: a meta-analysis of randomized and observational studies. Am J Kidney Dis. 2009. [Epub ahead of print]

41. Rueggeberg A, Boehm S, Napieralski F, et al. Development of a risk stratification model for predicting acute renal failure in orthotopic liver transplantation recipients.Anaesthesia. 2008;63:1174–1180.

42. Stouffer CW, Mansour MA, Ott MM, et al. Initial results of a thoracic aortic endovascular program: safer in high-risk patients. Ann Vasc Surg. 2009;23:478–484.

43. Bavaria JE, Appoo JJ, Makaroun MS, et al, and Gore TAG Investigators. Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: a multicenter comparative trial. J Thorac Cardiovasc Surg. 2007;133:369–377.

44. Glassman DT, Merriam WG, Trabulsi EJ, et al. Rhabdomyolysis after laparoscopic nephrectomy. JSLS. 2007;11:432–437.

45. Rehman J, Boglia J, Chughtai B, et al. High body mass index in muscular patients and flank position are risk factors for rhabdomyolysis: case report after laparoscopic live-donor nephrectomy. J Endourol. 2006;20:646–650.

46. Thakar CV, Yared JP, Worley S, et al. Renal dysfunction and serious infections after open-heart surgery. Kidney Int. 2003;64:239–246.

47. Kheterpal S, Khodaparast O, Shanks A, et al. Chronic angiotensin-converting enzyme inhibitor or angiotensin receptor blocker therapy combined with diuretic therapy is associated with increased episodes of hypotension in noncardiac surgery.J Cardiothorac Vasc Anesth. 2008;22:180–186.

48. Malinowska-Zaprzalka M, Wojewodzka M, Dryl D, et al. Hemodynamic effect of propofol in enalapril-treated hypertensive patients during induction of general anesthesia. Pharmacol Rep. 2005;57:675–678.

49. Reich DL, Hossain S, Krol M, et al. Predictors of hypotension after induction of general anesthesia. Anesth Analg. 2005;101:622–628.

50. Nigwekar SU, Kandula P. N-acetylcysteine in cardiovascular-surgery-associated renal failure: a meta-analysis. Ann Thorac Surg. 2009;87:139–147.

51. Hersey P, Poullis M. Does the administration of mannitol prevent renal failure in open abdominal aortic aneurysm surgery? Interact Cardiovasc Thorac Surg. 2008;7: 906–909.

52. Schoenwald PK. Intraoperative management of renal function in the surgical patient at risk. Focus on aortic surgery. Anesthesiol Clin North America. 2000;18:719–737.

53. Mahesh B, Yim B, Robson D, et al. Does furosemide prevent renal dysfunction in high-risk cardiac surgical patients? Results of a double-blinded prospective randomised trial. Eur J Cardiothorac Surg. 2008;33:370–376.

54. Lassnigg A, Donner E, Grubhofer G, et al. Lack of renoprotective effects of dopamine and furosemide during cardiac surgery. J Am Soc Nephrol. 2000;11:97–104.

55. Sirivella S, Gielchinsky I, Parsonnet V. Mannitol, furosemide, and dopamine infusion in postoperative renal failure complicating cardiac surgery. Ann Thorac Surg.2000;69:501–506.104 ’ Josephs and Thakar

www.anesthesiaclinics.com

56. Rivers E, Nguyen B, Havstad S, et al, and Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock.N Engl J Med. 2001;345:1368–1377.

57. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med. 2001;345:1359–1367.

58. Finfer S, Chittock DR, Su SY, et al, Nice-Sugar Study Investigators. Intensive versus conventional glucose control in critically ill patients. N Engl J Med. 2009;360:1283–1297.

59. Wiedemann HP, Wheeler AP, Bernard GR, et al, National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network.Comparison of two fluid-management strategies in acute lung injury. N Engl J Med.2006;354:2564–2575.

60. Wheeler AP, Bernard GR, Thompson BT, et al, National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network. Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. N Engl J Med. 2006;354:2213–2224.perioperative Acute Kidney Injury ’ 105.

PROFOMA:

NAME:

AGE:

SEX:

I.P.NO:

S.L.NO:

ADDRESS WITH CONTACT NUMBER:

DATE OF ADMISSION:

DATE OF SURGERY:

DATE OF DISCHARGE/DEATH:

DIAGNOSIS:

COMORBID CONDITIONS:

DIABETES

HYPERTENSION

CHRONIC KIDNEY DISEASE

CORONARY ARTERY DISEASE

THYROID DISORDER

PREOPEARATIVE

HB:

RFT:

UREA

CREATININE

URINE ROUTINE:

SERUM BILIRUBIN:

URINE OUTPUT:

SURGERY:

INTRAOPERATIVE HYPOTENSION:

BLOOD TRANSFUSION:

POST OPERATIVE

SERIAL RFT'S

1)RFT AFTER 48 HOURS.

UREA

CREATININE

2^{ND} RFT:

URINE OUTPUT:

DIALYSIS:

NEPHROTOXIC DRUGS:

OUTCOME:

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC: "A STUDY ON ACUTE KIDNEY INJURY IN POST-OPERATIVE PATIENTS AT GOVERNMENT STANLEY HOSPITAL,CHENNAI".

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.

I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:

Signature/Thumb impression of the Volunteer

Date:

Witnesses:
(Signature, Name & Address)

Date:
Name and signature of investigator:
Date:

அரசுஸ்டான்லிமருத்துவமனையில் அறுவை சிகிச்சைக்கு பின் நோயாளிகளுக்கு ஏற்படும் சிறுநீரக செயலிழப்பு, பற்றி ஒரு ஆய்வு.

ஆய்வாளர்:

வீ. மணிகண்டன்.

முதுநிலைபட்டமேற்படிப்புமாணவர்,

பொதுமருத்துவ பட்டபடிப்பு.

அரசு ஸ்டான்லி மருத்துவமனை.

வழிகாட்டி :

மரு. எட்வின் பெர்னாண்டோ

துறை தலைவர், சிறுநீரகவியல் துறை,

அரசு ஸ்டான்லி மருத்துவமனை.

<u>சுயஒப்புதல்படிவம்</u>

பெயர்: வயது: உள்ளிருப்புஎண்:

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களைக்கேட்கவும்,அதற்கான தகுந்தவிளக்கங்களைப்பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் ,எந்தகட்டத்திலும், எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளைப்பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்துகொண்டேன்.என்னைப்பற்றிய தகவல்கள் இரகசியமாகப்பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் .ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப எவ்விதமாகப்பயன்படுத்திக்கொள்ளவும், அதனைபிரசுரிக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்குகொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்துகொள்வதுடன், ஆய்வாளருக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன் .என் உடல்நலம் பாதிக்கப்பட்டாலோ அல்லது வழக்கத்திற்கு மாறானநோய்க்குறிதென்பட்டாலோ உடனே அதை தெரிவிப்பேன் என உறுதிகூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும், சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

நோயளியின் கையொப்பம்: ஆய்வாளர்கையொப்பம்:

அரசுஸ்டான்லிமருத்துவமனையில் அறுவை சிகிச்சைக்கு பின் நோயாளிகளுக்கு ஏற்படும் சிறுநீரக செயலிழப்பு, பற்றி ஒரு ஆய்வு.

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மரு. எட்வின் பெர்னாண்டோ

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<u>பங்கேற்பாளரின் தகவல் படிவம்</u>

நீங்கள் இந்த ஆய்வில் பங்கேற்க அழைக்கப்படுகிறீர்கள். இந்த ஆய்வில் பங்கேற்கும் முன், இதன் நோக்கத்தையும், முறைகளையும், இதனால் ஏற்படும் பின் விளைவுகளையும் நீங்கள் அறிந்து கொள்ள ஆய்வாளர் அளிக்கும் தகவல்:

தைரயுடு உள்ள நோயாளிகள் இந்த ஆய்வில் சேர்த்துக் கொள்ளப்படுவார்கள். உங்கள் நோயின்வரலாறும், உங்களின் முழு உடல் பரிசோதனையும் தெளிவாகவும் விரிவாகவும் பதிவு செய்யப்படும். தைராய்டு ஸ்கேன் மற்றும் இரத்தத்தில் தைராய்டு ஹார்மோன் அளவும் சோதனை செய்யப்படும்.

இந்த ஆய்வின் முடிவுகள் மருத்துவ காரணங்களுக்காகவும், மருத்துவ கல்விக்காகவும் பயன்படுத்தப்படும். இந்த ஆய்வு பற்றிய சந்தேகங்களுக்கு உரிய முறையில் விளக்கமளிக்கப்படும். தங்களைப் பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும்.

இந்த ஆய்வில் இருந்து எப்போது வேண்டுமானாலும் தாங்கள் எவ்வித முன்னறிவிப்பின்றியும், எவ்வித சட்ட சிக்கலும் இன்றி விலகிக் கொள்ளலாம்.

இந்த ஆய்வில் பங்கேற்குமாறு கேட்டுக் கொள்கிறேன்.

நன்றி,

ஆய்வாளர் கையொப்பம் நோயாளியின்கையொப்பம்

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A Study on acute Kidney Injury in Post-Operative Patients at Govt Stanley Medical College, Chennai.

Principal Investigator : Dr. V Manikandan

Designation : PG in MD (General Medicine)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 02.07.2014 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

S.L.NO	AGE	SEX	SURGERY	PRE OP RFT (UR,CR)	HB	T.B	URINE OUTPUT	HYPOTENSION	BLOOD	DRUGS	P.O RFT 48 HRS (UR,CR)	P.O RFT 7 DAYS(UR,CR)	OLIG
1	32	M	CARDIAC(LV CLOT)	18,0.7	10	0.8	NORMAL	YES	YES	YES	60,1.8	37,1.0	NO
2	62	M	CARDIAC(AVR)	27,0.8	9	1	NORMAL	YES	YES	YES	54,1.5	80,1.8	NO
3	45	F	CARDIAC(MVR)	33,0.8	9.5	0.7	NORMAL	YES	YES	YES	95,2.7	140,5.5	YES
4	65	M	VASCULAR(I.F.BYPASS)	26,0.7	10.4	0.9	NORMAL	NO	YES	NO	46,1.6	32,1.2	NO
5	58	M	VASCULAR(A.I.BYPAS)	18,0.8	9.6	0.7	NORMAL	YES	YES	NO	38,1.5	46,1.7	NO
6	44	M	CARDIAC(MVR)	18,0.9	11	0.8	NORMAL	YES	YES	NO	40,1.4	58,2.1	NO
7	49	F	ABDOMEN(LAPAROTOMY)	40,1.4	12.5	2.2	NORMAL	NO	YES	YES	76,2.3	108,3.5	NO
8	64	M	ABDOMEN(LAPAROTOMY)	30,1.0	9.4	1.2	NORMAL	NO	YES	NO	50,1.6	83,3.8	YES
9	35	M	CARDAIC(MVR)	26,0.8	10	0.7	NORMAL	YES	YES	YES	46,1.4	30,1.1	NO
10	50	M	CARDIAC(DVR)	19,0.5	10.6	0.7	NORMAL	YES	YES	YES	35,2.2	26,1.0	NO
11	28	M	ABDOMEN(BILIARY SURG)	26,0.8	9	1.6	NORMAL	NO	YES	NO	40,1.7	56,2.2	NO
12	50	M	CARDIAC(MVR+TVR)	20,0.8	12.5	0.8	NORMAL	YES	YES	YES	42,1.6	50,1.7	NO
13	26	M	CARDAIC(DVR)	12,0.5	11.8	0.6	NORMAL	YES	YES	YES	34,1.5	28,1.0	NO
14	48	M	CARDIAC(CABG)	30,1.0	10.6	1	NORMAL	YES	YES	YES	56,2.0	102,4.5	YES
15	46	M	CARDIAC(CABG)	30,0.7	14	1	NORMAL	YES	YES	YES	50,1.5	30,0.7	NO
16	44	M	CARDIAC(AVR)	24,0.8	11.2	0.8	NORMAL	YES	YES	YES	42,1.5	35,1.0	NO
17	52	M	CARDIAC(MVR)	32,0.9	12.3	0.8	NORMAL	YES	YES	YES	70,2.4	116,3.5	NO
18	52	M	CARDIAC(CABG)	20,0.6	10.9	0.7	NORMAL	YES	YES	YES	62,1.9	46,1.3	NO
19	70	F	CARDIAC(PCI)	20,0.8	10	0.8	NORMAL	YES	YES	YES	35,1.4	20,0.7	NO
20	45	M	CARDIAC(DVR)	20,0.9	11	0.8	NORMAL	YES	YES	YES	35,1.5	30,1.1	YES
21	65	M	CARDIAC(CABG)	18,0.6	10	0.7	NORMAL	YES	YES	YES	40,1.6	36,1.1	NO
22	57	M	CARDIAC(PCI)	20,0.8	10.6	0.9	NORMAL	YES	NO	YES	40,1.5	26,0.9	NO
23	29	M	CARDIAC(CABG*2)	28,0.9	12	0.8	NORMAL	YES	YES	YES	50,1.5	40,1.1	NO
24	35	M	ABDOMEN(SMV THROM)	26,0.8	12.2	0.8	NORMAL	NO	YES	NO	40,1.6	28,1.0	NO
25	53	F	CARDIAC(CABG)	37,0.9	10.1	0.9	NORMAL	YES	YES	YES	82,2.1	120,4.0	YES
26	45	M	VASCULAR(A.I.BYPAS)	23,0.5	10.2	0.8	NORMAL	NO	YES	NO	38,1.7	31,1.5	NO
27	37	F	CARDIAC(MVR)	27,0.8	9.8	0.7	NORMAL	YES	YES	YES	40,1.6	28,1.1	NO
28	45	F	CARDIAC(MVR)	20,0.7	10	0.8	NORMAL	YES	YES	YES	48,2.1	64,2.3	NO
29	17	F	CARDIAC(AVR)	26,0.8	11.2	0.9	NORMAL	YES	YES	YES	45,1.9	50,1.8	NO
30	67	M	VASCULAR(A.I.BYPAS)	28,0.9	10.6	0.7	NORMAL	NO	YES	NO	36,1.8	42,1.9	NO
31	40	F	CARDIAC(DVR)	18,0.9	9	0.8	NORMAL	YES	YES	YES	32,1.6	43,1.2	NO
32	45	F	ENT(CA CHEEK)	40,1.5	10.6	0.9	NORMAL	NO	NO	NO	50,2.2	40,1.4	NO
33	44	F	CARDIAC(MVR)	18,0.7	11.7	0.8	NORMAL	YES	YES	YES	30,1.3	40,1.5	NO
34	50	F	CARDIAC(MVR)	24,0.8	10.6	0.7	NORMAL	YES	YES	YES	48,2.1	70,1.9	YES

35	65	M	ABDOMEN(3 BYPASS)	28,0.6	8.7	14	NORMAL	YES	YES	NO	40,1.5	36,1.6	YES
36	42	M	ABDOMEN(OP.CHOLE)	26,0.8	10.2	1.1	NORMAL	NO	YES	NO	64,2.2	34,1.1	NO
37	42	M	CARDIAC(DVR)	28,0.9	10	0.7	NORMAL	YES	YES	YES	72,2.4	36,1.2	NO
38	47	F	CARDIAC(MVR)	20,0.6	10.6	0.8	NORMAL	YES	YES	YES	42,1.6	32,1.1	NO
39	44	F	CARDIAC(MVR)	24,0.8	9.2	0.7	NORMAL	YES	YES	YES	38,1.4	26,1.0	NO
40	35	F	CARDIAC(DVR)	22,0.6	10.6	0.7	NORMAL	YES	YES	YES	50,1.8	58,2.0	NO
41	56	F	ORTHO(AMPUTATION)	40,1.3	9.8	1.1	NORMAL	NO	YES	YES	64,2.8	42,2.0	NO
42	62	F	URO(CALCULI REMOVAL)	41,2.6	7.2	0.9	NORMAL	NO	YES	NO	60,3.5	70,3.2	NO
43	75	M	ABDOMEN(CHOLANGIOCA)	30,1.0	9	6	NORMAL	NO	YES	NO	63,1.5	42,1.2	NO
44	45	M	CARDIAC(CABG)	21,0.7	12	0.9	NORMAL	YES	YES	YES	40,1.5	28,0.9	NO
45	60	F	ABDOMEN(LAPAROTOMY)	34,0.9	11	1.2	NORMAL	NO	YES	NO	50,1.8	34,1.0	NO
46	60	F	CARDIAC(CABG*2)	26,0.8	10.7	0.9	NORMAL	YES	YES	YES	46,1.5	34,1.2	NO
47	40	F	CARDIAC(MVR)	20/0.8	10	0.7	NORMAL	YES	YES	YES	48,1.5	32,1.3	NO
48	50	M	VASCULAR(A.I.BYPAS)	26,0.7	11	0.8	NORMAL	NO	YES	NO	38,1.6	46,1.9	NO
49	46	M	CARDIAC(DVR)	28,0.6	10	0.7	NORMAL	YES	YES	YES	32,1.4	40,1.8	NO
50	24	M	CARDIAC(MVR)	19,0.6	9.6	0.7	NORMAL	YES	YES	YES	30,1.0	38,1.4	NO
51	50	M	CARDIAC(CABG)	28,0.9	10.4	0.6	NORMAL	YES	YES	YES	38,1.4	32,1.2	NO
52	40	M	ABDOMEN(IRR.HERNIA)	30,1.0	11	0.7	NORMAL	NO	YES	NO	40,1.9	36,1.3	NO
53	26	M	CARDIAC(MVR)	20,0.8	12	0.6	NORMAL	YES	YES	YES	40,2.1	50,2.6	NO
54	45	M	ABDOMEN(CA PANCREAS)	30,1.0	9	4.5	NORMAL	NO	YES	NO	40,2.0	39,1.6	NO
55	30	F	CARDIAC(MVR)	16,0.9	11	0.7	NORMAL	YES	YES	YES	40,1.5	38,1.3	NO
56	54	M	ABDOMEN(PERFORATION)	30,1.0	10.5	0.9	NORMAL	NO	YES	NO	60,2.2	38,1.2	NO
57	60	M	CARDIAC(CABG*2)	26,1.0	11.2	1	NORMAL	YES	YES	YES	38,1.6	32,1.4	NO
58	40	M	VASCULAR(A.I.BYPAS)	30,0.9	10.9	0.7	NORMAL	NO	YES	NO	51,1.3	26,1.0	NO
59	32	F	CARDIAC(MVR)	20,0.7	12	0.8	NORMAL	YES	YES	YES	42,1.7	25,0.7	NO
60	45	M	CARDIAC(DVR)	25,0.8	11	0.7	NORMAL	YES	YES	YES	45,1.4	33,1.2	NO

KEY TO MASTER CHART

ABBREVIATION:

PRE OP RFT :- PREOPERATIVE RENAL FUNCTION TEST

UR : - UREA

CR :- CREATININE

HB :- HEMOGLOBIN.

P.O RFT :- POST OPERATIVE RENAL FUNCTION TESTS.

RRT :-RENAL REPLACEMENT THERAPY.

DM/HT :-DIABETES MELLITUS,HYPERTENSION.

T.B :-TOTAL BILIRUBIN

Creatinine : 0.5-1.5 mg/dl.

Urea : 20-40 mg/dl.