"INCIDENCE AND RISK FACTORS OF ACUTE KIDNEY INJURY IN CONGENITAL HEART DISEASE CHILDREN POST CARDIAC SURGERY"

BY

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Dr DNYANESH D K M.D., L.L.B
Professor & Head
Department of Pediatrics
J N Medical College
KAHER
Belagavi, Karnataka

Professor & Head
Department of Pediatrics
KLE University's
J.N. Medical College, Belagavi

Date: 01/07/24
Place: JNMC, Belagavi

Dr N S MAHANTASHETTI M.D.
Principal
J N Medical College
KAHER
Belagavi, Karnataka

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

Date: 1/7/2024
Place: JNMC, Belagavi

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Date:

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Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)
Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 25-06-2024

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BM0121013
Postgraduate Student,
2021-22 Batch,
Department of Paediatrics
J. N. Medical College, Belagavi.

ETHICAL CLERANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 100 **Date: 27/09/2022**

To.

REG NO. : BM0121013
PG Student in Paediatrics,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"INCIDENCE AND RISK FACTORS OF ACUTE KIDNEY INJURY IN CONGENITAL HEART DISEASE CHILDREN POST CARDIAC SURGERY..",** is ethical and justifiable.

The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVATIONS:

ABBREVATION	EXPANSION OF ABBREVATION
AKIN	Acute kidney injury network
AUC	Area under curve
CPB	Cardiopulmonary bypass
eCCl	Estimated creatinine clearance
eGFR	Estimated glomerular filtration rate
KDIGO	Kidney Diseases Improving Global Outcomes
RIFLE	Risk, injury, failure, loss, end stage kidney disease
UO	Urine output

ABSTRACT:

Background

Understanding of the epidemiology of Acute kidney injury with the modifiable and non-modifiable risk factors, recognising the illness earlier, assessing the incidence and severity of acute kidney injury will prevent the adverse outcomes in the children undergoing cardiac surgery for their congenital heart diseases. The definition of paediatric AKI was standardized based on upgrading in serum creatinine levels or decreased in urine output . The introduction of Risk , injury , failure , loss , end stage kidney disease criteria & Modified version proposed by Acute Kidney Injury Network (AKIN) have increased the hypothetical of early prediction of AKI. Renal angina index (RAI), which is derived from variations in renal function, used to risk stratify critically ill children who are at high risk of AKI. It is believed that the Renal Angina Index may be used to identify early indicators of AKI. The RAI is an idea that seems appealing, but it hasn't been proven to work or validated in non-Western populations like Asian nations.

Objectives:

Primary objective :

To compare AKIN criteria and Renal Angina Index (RAI) for early prediction of severe acute kidney injury in congenital heart disease patients undergoing cardiac surgery.

SECONDARY OBJECTIVE :

To ascertain the risk factors of acute kidney injury in these children.

Methodology:

This is a Cross sectional study, among 52 children with congenital heart disease patients undergoing cardiac surgery, in KAHERs Jawaharlal Nehru Medical College, Belagavi, Karnataka. All children aged 1month to 12years (post cardiac surgery) admitted during the study period of 1 year (2023 January to 2023 December) with congenital heart disease undergoing surgery were included. After obtaining the approval from ethical committee, written informed consent from each of children's parent/guardian were obtained. After obtaining detailed history, physical examination was done. Infants, children who have been diagnosed with acute kidney injury in congenital heart disease post operatively were assessed with AKIN criteria and Renal Angina Index (RAI) within 24 hours to 72 hours of surgery.

Results:

Among the subjects, 25 (48.08%) had AKI by Renal Angina index. Among the subjects, according to AKIN criteria, 50 (96.15%) had Stage 1, 1 (1.92%) had Stage 2 and 1 (1.92%) had Stage 3. 92% of the AKI by RA index had Stage 1 and 8% had stage 2 and 3. All of the subjects with RA index less than 8 had stage 1. The association between Acute Kidney Injury by RA index and AKIN was not statistically significant. Baseline variables such as Age, gender and body surface area, Pre-op parameters like urea, creatinine and GFR and diuretics, Intra-op characteristics like bypass time and aortic cross clamp time and post operative eGFR of the subjects had no statistically significant difference between the AKIN staging groups and RA index groups (except preop creatinine).

Conclusion:

Children undergoing cardiac surgery for congenital heart disease are at risk of developing acute kidney injury . Renal Angina Index and AKIN Criteria are useful in early prediction of Acute Kidney Injury in post op cardiac patients and therefore help in timely management**.** In future further more studies should be conducted regarding post op cardiac patient in order to prevent morbidities due to AKI.

Keywords: Congenital heart disease, CHD, Children, Acute kidney injury, cardiac surgery-associated acute kidney injury, CSA-AKI, Renal angina index, RAI,

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INTRODUCTION

Congenital heart disease (CHD) is the most common congenital anomaly, occurring with an incidence of 0.8 % to 1.2% of live births. Congenital heart disease is most frequent inborn cause of neonatal loss (1) After medical stabilization of acute heart failure symptoms or cyanosis, the majority of children require surgical or transcatheter repair. The exceptions to this rule include small valve malfunctions or particular ventricular septal defects that are likely to shrink or close over time. (2)

Nowadays, nearly 90% of children with congenital heart disease survive into adulthood due to the advancements in cardiac surgery. Early pioneering surgeons accepted a great risk associated with these procedures, but now the modern surgical practice places a strong emphasis on consistency and safety. (3)

"5-33% of Pediatrics patients suffers from AKI following cardiac surgery (depending on acute kidney injury definition used in study) with associated mortality of 20-79%.(4,5) Acute kidney damage (AKI) related to cardiac surgery (CS-AKI) is becoming more common and is linked to considerable morbidity and mortality. It is pivotal to identify people who are at risk and to make an early diagnosis.(6)

Previous studies data divulge raised serum creatinine levels in cyanotic heart disease, children with less than one year of age, extended cardiopulmonary bypass time, and postoperative lesser cardiac output syndrome as risk factors for acute kidney injury following Paediatric cardiac surgeries. The risk factors can be classified into three categories: preoperative (lower age, lower weight and RACHS-1), intraoperative (Use of CPB, Blood transfusion and intraoperative hypotension) and postoperative (Sepsis, use of nephrotoxic drugs and low cardiac output syndrome). (5)

"The national institute of health" and "American society of nephrology", recently called for developing a more efficient tools for diagnosis for acute kidney injury.

Three procedures are used to identify high-risk individuals in order to prevent cardiac surgery-associated acute kidney injury (CSA-AKI): biomarker analysis, clinical risk factor assessment, and detection of preoperative, intraoperative, and postoperative risk factors. Additionally, following the treatment guidelines provided by Kidney Disease: Improving Global Outcomes (KDIGO) may lower the risk of AKI. (7–9)

Renal angina index (RAI), which is derived from variations in renal function, used to risk stratify critically ill children who are at high risk of AKI. It is believed that the RAI may be used as a biomarker to identify early indicators of persistent AKI. When the RAI for adults was first developed, it involved a definition that was more consistent, albeit more complex, than it was for Pediatrics intensive care unit patients. The RAI is an idea that seems appealing, but it hasn't been proven to work or validated in non-Western populations like Asian nations. (10–12)

Rationale of the study:

Understanding of the epidemiology of acute kidney injury with the modifiable and non-modifiable risk factors, recognizing the illness earlier, assessing the incidence and severity of acute kidney injury will prevent the short term and long term adverse outcomes in the children undergoing cardiac surgery for their congenital heart diseases. (13–16)

Numerous studies have been done on this topic in developed countries. There are only very few studies in developing countries, especially India. Since this is the case, the purpose of this study is to find out the prevalence of acute renal damage and the factors that put children with congenital heart disease at risk for developing it after undergoing cardiac surgery.

AIM AND OBJECTIVES

AIM:

- To compare AKIN criteria and Renal Angina Index (RAI) for early prediction of severe acute kidney injury in congenital heart disease patients undergoing cardiac surgery.

OBJECTIVES:

Primary Objectives:

- To compare AKIN criteria and Renal Angina Index (RAI) for early prediction of severe acute kidney injury in congenital heart disease patients undergoing cardiac surgery.

Secondary Objectives:

- To ascertain the risk factors of acute kidney injury in these children.

REVIEW OF LITERATURE

This study's review of the relevant literature on “incidence rate and risk factors of acute renal damage among children with congenital heart disease undergoing post cardiac surgery”, is described in the below headings:

a. Congenital heart disease.
    - Prevalence
    - Aetiology
    - Pathophysiology
    - Classification
    - Clinical features
    - Investigations
b. Cardiac surgery in Congenital heart disease.
c. Acute Kidney Injury.
    - Definition
    - Risk factors
    - Pathogenesis
    - Management
    - Outcomes
    - Prevention
d. Criteria for diagnosis and staging of AKI:
e. Renal Angina Index (RAI)
f. Studies done on the similar topic.

a. Congenital heart disease:

Prevalence:

Congenital heart disease (CHD) (Synonym(s): Congenital heart defects, Cardiac malformations) is the most common congenital anomaly, occurring with an incidence of 0.8% to 1.2% of live births. Also it is the most frequent inborn cause of neonatal loss . (1)

With a cumulative prevalence rate of 48.4 per 10,000 livebirths, muscular and peri membranous ventricular septal defects are the most frequent congenital heart illnesses detected in infancy, followed by secundum atrial septal abnormalities. Tetralogy of Fallot is the most common cyanotic congenital cardiac condition, occurring in twice as many new-borns (4.7 vs. 2.3/10 000), as transposition of the major arteries. Bicuspid aortic valves are the most prevalent congenital abnormalities overall, with prevalence estimates ranging from 0.5% to 2.0%. (17–19)

Aetiology:

Congenital cardiac disease is influenced by both hereditary and environmental factors.

Environmental factors:

Typical environmental triggers include

- Maternal disease (diabetes, rubella, systemic lupus erythematosus),
- Mother's exposure to teratogenic substances (antiseizure medications, lithium, Valproic acid, Vitamin D, Warfarin, Phenyl alanine, Ethanol, Hydantoin, isotretinoin). (20)

Genetic factors:

The mother’s age is a known risk factor for a number of genetic problems like cardiac abnormalities including Down syndrome or other complications. A number of

chromosomal abnormalities are strongly associated with congenital heart illness to a significant degree, e.g.

i. Trisomy 21 (Down's syndrome),
ii. Trisomy 18,
iii. Trisomy 13,
iv. Monosomy X (Turner's syndrome).

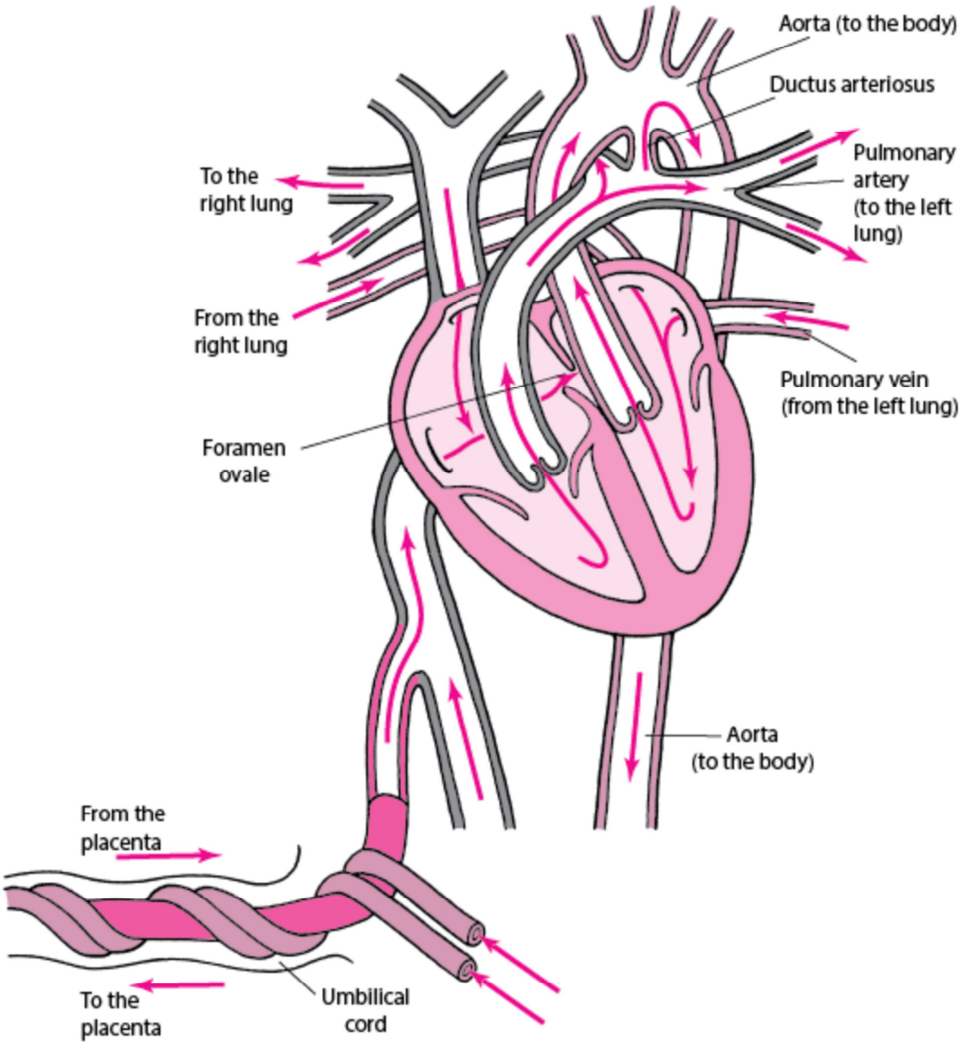
On the other hand, these abnormalities are only present in 5–6% of patients who have congenital heart illness.

It is possible for congenital syndromes to be caused by sub chromosomal deletions (also known as microdeletions), sub chromosomal duplications, or single-gene alterations. These syndromes can affect several organs in addition to the heart. A genetic aetiology cannot be identified in around 72 percent of the patients who have been diagnosed with congenital heart illness. (17,21,22)

Pathophysiology:

In the foetus first blood gets oxygenated in placenta then enters into the right side of the heart. In view of minimal requirement of ventilation of lungs in fetal period, the pulmonary artery only needs to carry a small volume of blood through it. The majority of blood from the heart's right side escapes the lungs by going through the Ductus arteriosus and foramen ovale. These two structures normally shut down soon after birth. Significant alterations to the foetal circulation take place after the baby has taken its first few breaths. These alterations lead to an increase in the flow of blood through the lungs and the closing of the foramen ovale from a functional standpoint. (23) The following figure represents the normal foetal circulation . (20)

Figure 1.Normal foetal circulation



Classification:

Congenital heart anomalies are classified into Cyanotic and Acyanotic (left-to-right shunts or obstructive lesions) heart anomalies. The physiologic consequences of congenital heart anomalies varies to a greater extent. Child can present with incidental heart murmurs alone or can have symptoms varying from mild to severe variety in form of cyanosis, heart failures, or sudden collapse of the circulation. The following list represents the categories of Congenital Heart Anomalies into Cyanotic and

Acyanotic (shunts of left-to-right side or obstructive valvular lesion) heart anomalies, with approximate decreasing order of frequency of individual diseases.

I. Cyanotic diseases:
    i. Tetralogy of Fallot (TOF)
    ii. Pulmonary atresia
    iii. Persistent truncus arteriosus (PDA)
    iv. Transposition of the great arteries (TGA)
    v. Tricuspid atresia
    vi. Total anomalous pulmonary venous return

II. Acyanotic diseases:

a) Left-to-right shunt
    i. Ventricular septal defects (VSD)
    ii. Atrial septal defects (ASD)
    iii. Patent ductus arteriosus (PDA)
    iv. Atrioventricular septal defects

b) Obstructive valvular lesions
    i. Pulmonary valve stenosis
    ii. Aortic stenosis
    iii. Coarctation of Aorta
    iv. This condition, known as hypoplastic left heart syndrome, frequently presents itself with cyanosis, which may be of a less severe type. (18,20,24)

Clinical Features:

Patients of congenital heart disease can present with different symptoms, but most commonly they are seen with features mentioned below:

i. Murmurs. (There is a continuous murmur caused by patent ductus arteriosus, and the majority of left to right shunts and obstructive lesions are responsible for producing systolic murmurs.)

ii. Cyanosis. (bluish discolouration of lips and tongue with or without nailbeds, due to high levels of deoxygenated haemoglobin [>5g/dl] and decreased blood oxygen saturation levels [oxygen saturation level <85%])

iii. Heart failure (Tachycardia and Dyspnoea)

iv. Diminished or nonpalpable pulses.

v. Other physical examination abnormalities may include circulatory shock, poor perfusion, Syncope, chest pain, abnormal 2nd heart sound (S2—single or widely split), systolic click, gallop, or abnormally slow, fast, or irregular rhythm.(20)

Investigations:

i. Screening by pulse oximetry

ii. Cardiac physical examination

iii. Chest x-ray and ECG

iv. Echocardiography (diagnostic especially among neonates)

v. Heart magnetic resonance imaging (MRI) or computed tomography (CT) angiography, as well as cardiac catheterization with angiocardiography. (Sometimes therapeutic also). (25)

b. Interventions in Congenital heart disease:

Medical stabilization of heart failure (eg , with drugs such as diuretics, angiotensin-converting enzyme [ACE] inhibitors, beta-blockers, spironolactone, digoxin, salt restriction, oxygen therapy, prostaglandin E1). (26)

Surgical repairs or transcatheter interventions:

The underlying issue must usually be corrected for definitive therapy to proceed. After medical stabilization of acute heart failure symptoms or cyanosis, the majority of children require surgical or transcatheter repair. The exceptions to this rule include small valve malfunctions or particular ventricular septal defects that are likely to shrink or close over time. Procedures for trans-catheters include,

i. for palliation of neonates with severe cyanosis with transposition of the great arteries -**Balloon atrial septostomy**
ii. **Balloon dilation** - For the severe stenosis of aortic valve and stenosis of pulmonary valves.
iii. **Transcatheter placement of pulmonary valve**.
iv. **Transcatheter closing of cardiac shunt** (most commonly with the ASD and PDA)
v. **Balloon dilation with or without stenting of vascular stenoses**, often with pulmonary artery stenosis. (20)

The following table represents the Eponyms for Surgical Procedures for the treatment of Congenitally Malformed Hearts,(27,28)

Figure 2. Eponyms for Surgical Procedures for the treatment of Congenitally Malformed Hearts

Eponym	Description of procedure	Cardiovascular anomalies
Blalock-Hanlon shunt	Partial atrial septectomy (posterosuperior region)	Complete TGA with intact ventricular septum
Blalock-Taussig shunt	Subclavian-to-pulmonary artery (classic: end-to-side anastomosis; modified: interposed synthetic graft)	Conditions with decreased pulmonary blood flow (tetralogy of Fallot, PA-VSD, and DORV or DILV with PS)
Damus-Kaye-Stansel procedure	Proximal PT to ascending aorta (end-to-side anastomosis); conduit from RV to distal PT; VSD closure	Complete TGA without PS and with or without VSD
Glenn anastomosis	SVC to RPA (end-to-side); ligation of SVC at RA; ligation of proximal RPA (bidirectional Glenn: no ligation of RPA)	Tricuspid atresia, or DILV with PS
Fontan procedure (modified)	Anastomosis of SVC, RA, or RV to RPA or LPA; may include intra-atrial conduit from IVC to SVC	Hearts with single functional ventricle (tricuspid atresia, DILV, etc.)
Jatene procedure	Transection and switching of great arteries and coronary arteries	Complete TGA, and DORV with subpulmonary VSD
Konno procedure	Outlet (infundibular) septostomy, with patch enlargement of LV and RV outflow tracts, and aortic valve replacement	Tunnel subaortic stenosis, and severe hypertrophic cardiomyopathy
Mee procedure	Ascending aorta to MPA (side-to-side anastomosis)	Same as for Blalock-Taussig shunt
Mustard procedure	Resection of atrial septum; intra-atrial baffle directing cavai blood to LV, and pulmonary venous blood to RV	Complete TGA
Norwood procedure	Stage 1 (atrial septectomy; PDA ligation; PT transection; aortic incision; reconstruction of aorta with allograft; aorta-PT shunt). Stage 2 (modified Fontan operation)	Aortic atresia (hypoplastic left heart syndrome)
Potts shunt	Descending thoracic aorta to LPA (side-to-side anastomosis)	Same as for Blalock-Taussig shunt
Rastelli procedure	VSD closure directing LV blood to aorta; conduit from RV to distal PT; ligation of proximal PT	PA-VSD, PTA, complete TGA with VSD and PS, and DORV with PS
Ross procedure	Excision of aortic valve; excision of pulmonary valve and insertion into aortic position; insertion of prosthetic pulmonary valve	Severe aortic valve stenosis; tunnel subaortic stenosis
Senning procedure	Use of atrial septum to fashion intra-atrial baffle, similar to Mustard procedure	Complete TGA
Waterston shunt	Ascending aorta to RPA (side-to-side anastomosis)	Same as for Blalock-Taussig shunt

DILV, double inlet left ventricle; DORV, double outlet right ventricle; IVC, inferior vena cava; LPA, left pulmonary artery; LV, left ventricle; PA-VSD, pulmonary atresia with a ventricular septal defect; PDA, patent ductal artery; PS, pulmonary stenosis; PT, pulmonary trunk; PTA, persistent truncal artery; RA, right atrium; RPA, right pulmonary artery; RV, right ventricle; SVC, superior vena cava; TGA, transposition of the great arteries; VSD, ventricular septal defect.

On August 8, 1938, at the Children's Hospital in Boston, Dr. Gross successfully ligated a patent ductus arteriosus, marking the beginning of paediatric heart surgery. Aside from Gibbon's first successful closure of an atrial septal defect in Philadelphia with an artificial heart-lung machine, the origins of open-heart surgery for congenital malformation repair can be traced to University of Minnesota Department of Surgery personnel in the 1950s and 1960s.(29)

Nowadays, nearly 90% of children with congenital heart disease survive into adulthood due to the advancements in cardiac surgery. Early pioneering surgeons accepted a great risk associated with these procedures, but now the modern surgical practice places a strong emphasis on consistency and safety.(3)

c. Acute Kidney Injury:

Acute kidney damage (AKI) is a common side effect of paediatric heart surgery that affects 40–60% of high-risk kids. It is becoming more well acknowledged as a serious health issue for young patients.(30,31) Even in individuals with very slight changes in creatinine, AKI is linked to longer periods of mechanical breathing, inotropic support, and ICU admission, as well as higher mortality. The fluid overload caused by AKI is, additionally, related with death and worse clinical outcomes. Increased awareness of this issue has prompted researchers to investigate the possibility of developing more accurate diagnostics, potential therapies, the long-term significance of acute kidney injury (AKI), and the extent of AKI within nonsurgical cardiac cohorts. (32–34)

Definition of AKI:

The Kidney Disease: Improving Global Outcomes (KDIGO) criteria can be used to diagnose and stage cardiac surgery-associated acute kidney injury (CSA-AKI).

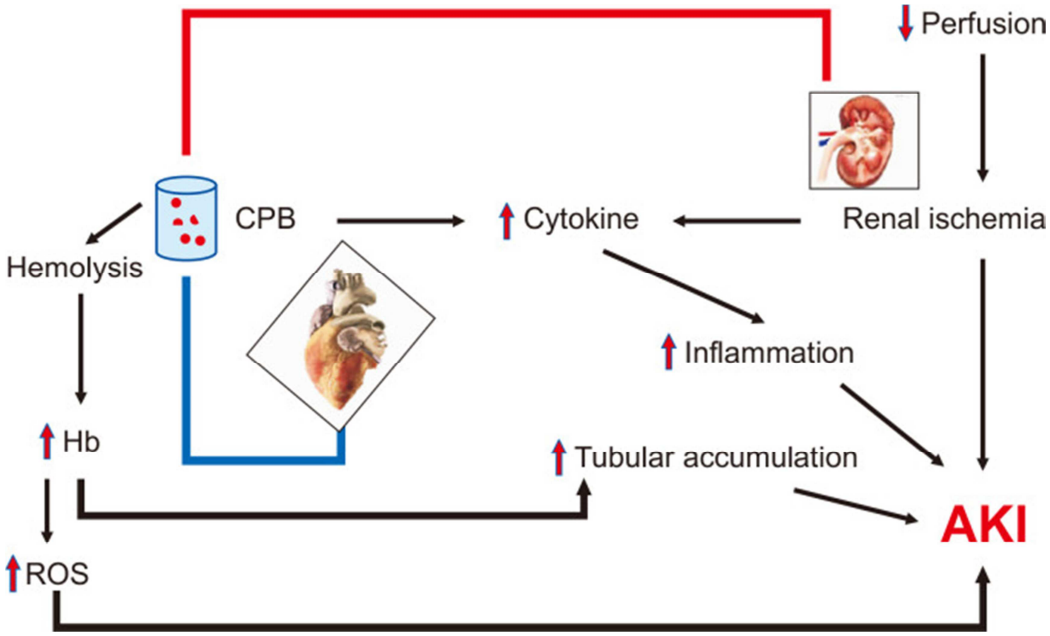
a. A raise in serum creatinine level by ≥ 0.3mg/dl (≥26.5μmol/l) within 48 hours.
b. The presence of a rise in blood creatinine levels that is at least 1.5–1.9 times higher than the baseline levels, and is either known or suspected to have occurred within the previous seven days.
c. Urine output less than 0.5ml/kg/hr for 6hr.(35)

Pathogenesis:

Following cardiac surgery, Acute Kidney injury (AKI) can primarily result from either renal ischemia ,reperfusion injury , inflammation , cardiopulmonary bypass (CPB) induced hemolysis. Additionally these can lead to repair and fibrosis causing irreversible damage to kidneys. Below is pictorial representation of the

Pathophysiology of AKI among children with congenital heart disease undergoing surgery, (CPB- Cardio Pulmonary Bypass, ROS- Reactive Oxygen Species)(5)

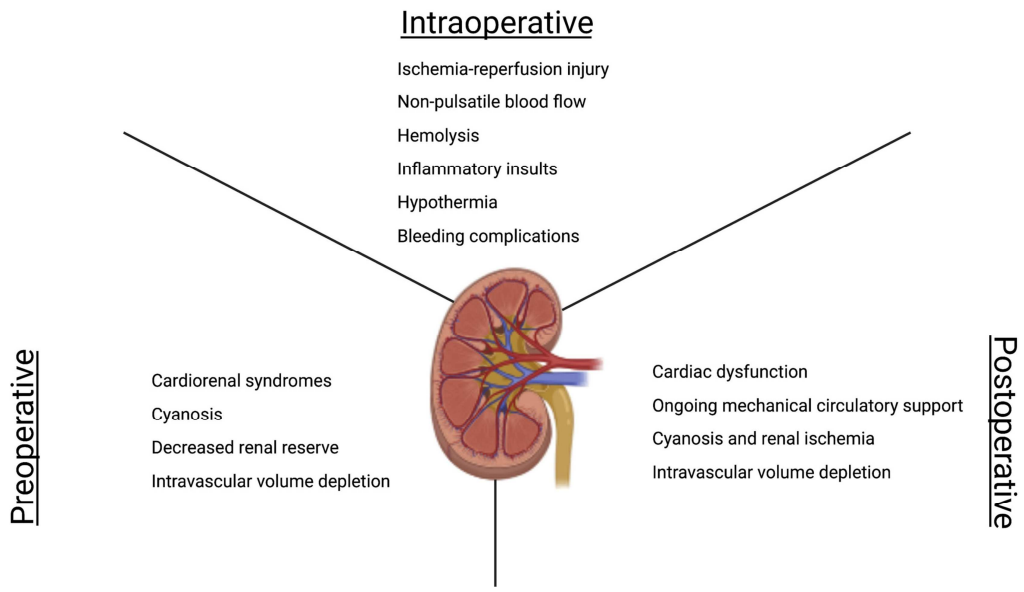
Figure 3. Pathophysiology of AKI among children with CHD undergoing surgery



Risk factors for AKI:

In children who are undergoing heart surgery, acute kidney injury (AKI) is frequently caused by a combination of factors, and can be classified into three categories: preoperative (lower age, lower weight and RACHS-1), intraoperative (Use of CPB, Blood transfusion and intraoperative hypotension) and postoperative (Sepsis, use of nephrotoxic drugs and low cardiac output syndrome). (5) The following figure represents the Risk factors for cardiac surgery-associated AKI. (36)

Figure 4.Risk factors for cardiac surgery-associated AKI



The following table represents the Risk factors and Biomarkers for Acute Kidney injury after Paediatric Cardiac Surgery,

Table 1. Risk factors and Biomarkers for Acute Kidney injury after Paediatric Cardiac Surgery

Risk factors	**Biomarkers**
Age	Serum creatinine
Functional single ventricle	Serum cystatin C
Higher baseline serum creatinine	Serum NGAL
Higher risk adjustment for congenital	Urine NGAL
heart surgery 1 category	Interleukin 6
CPB use	Interleukin 18
CPB duration	Kidney injury
Small kidneys (by preoperative ultrasonography)	molecule 1
Preoperative aminoglycoside exposure	Liver fatty acid
Selective cerebral perfusion	binding protein
Prolonged ventilation	Homovanillic acid
Low cardiac output syndrome	sulfate
Sepsis	
Platelet count <80,000/mm^3	

NGAL: Neurophilgelatinase associated lipocalcin, CPB:Cardiopulmonary bypass

Management:

The following table represents the Medical management of acute kidney injury post-cardiac surgery, (5)

Table 2.Pharmacological management of AKI post-cardiac surgery

Treatment	Mechanism	Action in kidney
Furosemide	Na^+, K^+, $2Cl^-$ symporter (NKCC2) antagonist	Diuresis
Fenoldopam	D1 receptor antagonist	Renal vasodilation and increased blood flow
Theophylline & aminophylline (xanthine derivatives)	Inhibit adenosine-induced vasoconstriction	Increased renal blood flow, diuresis

The following table represents the Treatment of AKI injury post-cardiac procedure with various types of dialysis,(5)

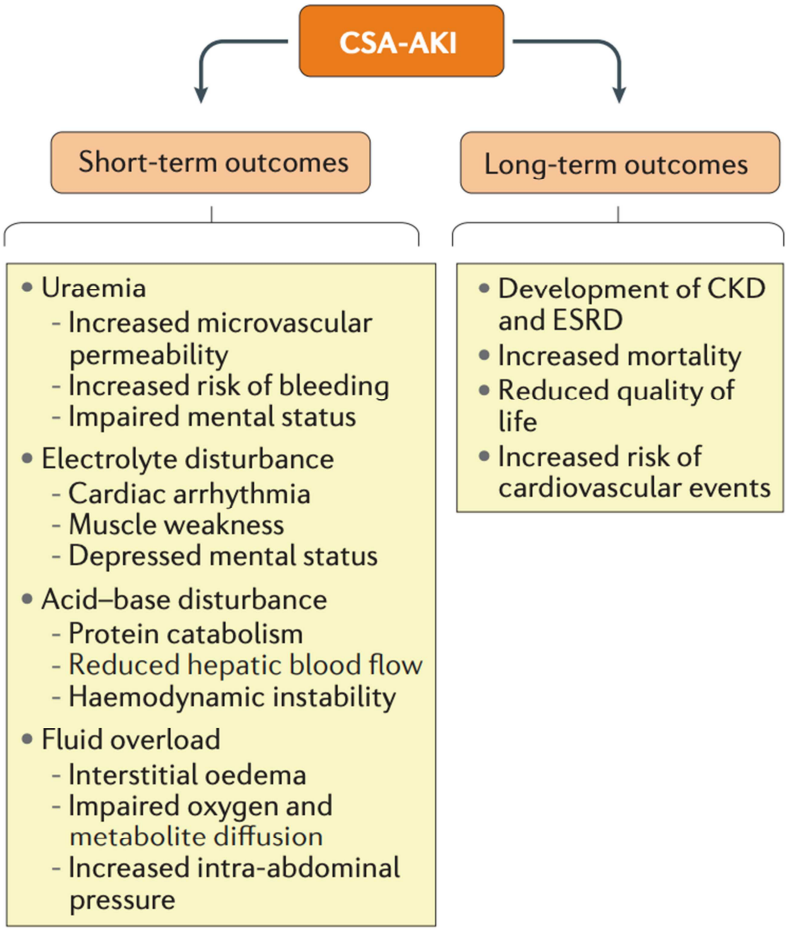
Table 3. Treatment of AKI post-cardiac procedure with various types of dialysis

Dialytic modality	Indication	Benefit
Peritoneal dialysis (PD)	Fluid overload, mild-moderate AKI	Ease of access, decreased time to negative fluid balance
Intermittent hemodialysis (IHD)	Fluid overload, AKI	Rapid fluid & solute removal
Continuous renal replacement therapy (CRRT)	Critically-ill patients, severe AKI, fluid overload	Avoidance of major fluid shifts; controlled removal of fluid
Continuous venovenous hemodiafiltration (CVVHDF)	Severe AKI & fluid overload	Highly-efficient filtration method to remove both solutes & fluids

Outcomes:

Acute kidney damage (AKI) following cardiac surgery can have either long-term or short-term effects, with the former happening within 90 days of the procedure. The following figure represents the effects of AKI on overall patient outcomes, (35)

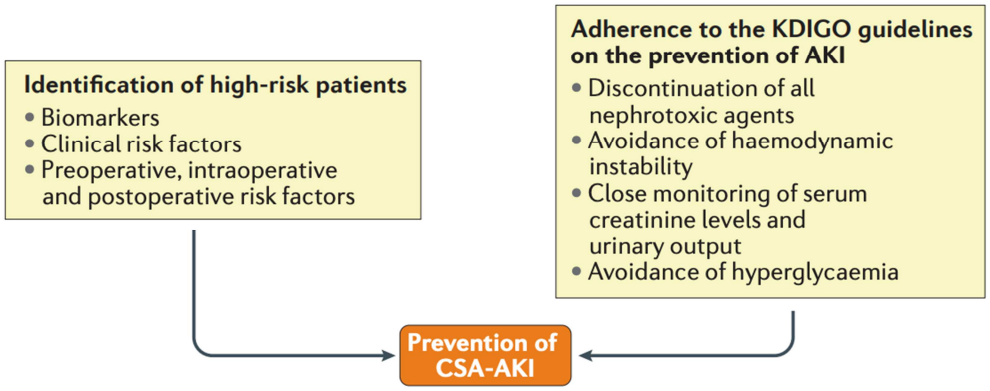
Figure 5.Effects of AKI on overall patient outcomes



Prevention:

Three procedures are used to identify high-risk individuals in order to prevent cardiac surgery-associated acute kidney injury (CSA-AKI): biomarker analysis, clinical risk factor assessment, in addition to the recognition of risk factors from preoperative, intraoperative, and postoperative stages. Additionally, following the treatment guidelines provided by Kidney Disease: Improving Global Outcomes (KDIGO) may lower the risk of AKI. The following figure represents the Preventive strategies for cardiac surgery-associated AKI, (35)

Figure 6.Preventive strategies for cardiac surgery-associated AKI



d. Criteria for diagnosis and staging of AKI:

Over ten years ago, the RIFLE (Risk, Injury, Failure, Loss, and End-Stage Renal Disease) criteria were developed by the Acute Dialysis Quality Initiative Group in response to the lack of a uniform definition of acute kidney injury (AKI). Numerous changes have taken place since then. Children were added to the RIFLE criterion (paediatric RIFLE, or pRIFLE). The Acute Kidney Injury Network (AKIN) criteria came next, which broadened the diagnosis of AKI to encompass individuals with a serum creatinine increase of less than 0.3 mg/dl during a 48-hour period. The RIFLE, pRIFLE, and AKIN classification systems were most recently combined into the Kidney Disease Improving Global Outcomes (KDIGO) classification system. (37–40) Acute kidney injury is concluded in the table that follows, which includes definitions as well as diagnostic criteria. (41)

Table 4.Definitions and criteria for diagnosis for AKI.

Definition and stages	Criteria – serum creatinine	Criteria – urine output
pRIFLE		
Stage 1 (Risk)	eGFR decreased by 25%	Less than 0.5 ml/kg/h for 8 h
Stage 2 (Injury)	eGFR decreased by 50%	Less than 0.5 ml/kg/h for 16 h
Stage 3 (Failure)	eGFR decreased by 75%	Less than 0.3 ml/kg/h for 24 h or anuria for 12 h
Stage 4 (Loss)	Persistent failure more than 4 weeks	
Stage 5 (End-stage)	ESRD, persistent failure more than 3 months	
AKIN		
Stage 1	Increase in SCr by at least 50% or an absolute increase in SCr by 0.3 mg/dl	Less than 0.5 ml/kg/h for 6 h
Stage 2	Increase in SCr by at least 100%	Less than 0.5 ml/kg/h for 12 h
Stage 3	Increase in SCr by at least 200%	Less than 0.3 ml/kg/h for 24 h or anuria for 12 h
KDIGO		
Stage 1	Increase in SCr by at least 50% or absolute increase in SCr by 0.3 mg/dl	Less than 0.5 ml/kg/h for 6–12 h
Stage 2	Increase in SCr by at least 100%	Less than 0.5 ml/kg/h for more than 12 h
Stage 3	Increase in SCr by at least 200% or eGFR less than 35 ml/min/1.73 m^2 (if age less than 18 years)	Less than 0.3 ml/kg/h for at least 24 h or anuria for at least 12 h

AKIN, Acute Kidney Injury Network; eGFR, estimated glomerular filtration rate (estimated using the Schwartz method); ESRD, end-stage renal disease; KDIGO, Kidney Diseases Improving Global Outcomes; pRIFLE, pediatric RIFLE; SCr, serum creatinine.

e. Renal Angina Index (RAI):

The following table represents the calculation of the Renal Angina Index,(42)

Table 5.Calculation of the Renal Angina Index

Elevated Cr	score
<0.1 mg/dl	1
≥0.1 mg/dl	2
≥0.3 mg/dl	4
≥0.4 mg/dl	8

×

Condition	score
ICU admission	1
DM	3
Vasopressor or Ventilator	5

= **RA index**

f. Studies done on the similar topic:

Studies on RA Index:

Simon Li et al, from USA, did a prospective multicentre study, among 311 children aged 1 month to 18 years. They conducted research on the prevalence of acute kidney injury, as well as its risk factors, severity, and outcomes. (≥50% raised serum creatinine from the preoperative value) among children undergoing cardiac surgery. They observed that 130 patients (42%) out of 311 children experienced acute renal injury within 3 days following surgery. Prolonged mechanical ventilation and an

extended hospital stay were independently linked to the development of acute renal dysfunction. Age and cardiopulmonary bypass duration were found to be independently linked to the risk of acute renal dysfunction.(43)

Rajit K Basu et al, from Atlanta, USA, did a prospective observational study, among 1590 patients in intensive care units from 32 centres in 9 countries. They studied the accuracy and usefulness of renal angina index (RAI) for prediction of severe acute kidney injury among critically ill children. They observed that the Severe AKI was present in 247 (18.9) and 121 (42.3) among the renal angina present and absent patients respectively. Improved early diagnosis of severe AKI may lead to better patient outcomes related to Acute Kidney Injury. Renal angina risk assessment increased the accuracy of predicting severe AKI in critically sick children and young adults when compared to isolated, context-free changes in Serum Creatinine . (44)

Bilal Aoun et al, from Lebanon, did a retrospective chart review, among 150 children and infants who were undergoing heart surgery. They studied prevalence of acute renal injury, as well as its severity, risk factors, and outcomes among these children. They observed that Fourteen patients (9.3%) developed AKI using the AKIN criteria. They observed that AKI was more likely to occur in children with anaemia, hyper lactic acidaemia, and presence of cyanotic heart disease. (45)

Sidharth Kumar Sethi et al, from Gurgaon, India, did a study, among 208 patients. They conducted research on the prevalence of acute renal injury, as well as its severity, risk factors, and outcomes, among children who were undergoing heart surgery. They observed that the twenty patients - (9.6%) had (AKI-I 15 patients; AKI-II 1 patient and AKI-III 4 patients) (14 of them were infants) had acute renal damage (AKI). Age less than one year and young children cardiopulmonary bypass duration,

extended need for a ventilator, pump malfunction, sepsis, and haematological issues were found to be independent risk factors for acute kidney injury of any severity. (46)

Georgios Kourelis et al, from Greece, did a Observational Cohort study, in a single tertiary centre among 362 patients. They conducted research on the prevalence of acute renal injury, as well as its severity, risk factors, and outcomes, among children who were undergoing heart surgery. They observed that Seventy (19.3%) patients had AKI, and the in-hospital mortality rate for this group was 21.4%. Younger age, lower weight, longer cardiopulmonary bypass time, prior mechanical ventilation, and diagnostic category were all associated with postoperative acute kidney injury (AKI that occurred after surgery). Extended periods of mechanical breathing, intensive care unit stays, and hospital stays were linked to AKI.(47)

Scott I. Aydin et al, from New York, USA, did a review, among 458 children aged less than 18 years. They conducted research on the prevalence of acute renal injury, as well as its severity, risk factors, and outcomes, among children who were undergoing heart surgery. They observed that the 234 individuals (51%) had AKI. The development of AKI was linked to younger age, greater RACHS-1 (risk-adjusted classification for congenital heart surgery) category, and longer cardiopulmonary bypass time. AKI was linked to greater preoperative blood urea nitrogen and lower preoperative serum creatinine levels.(48)

In Portugal, **Francisco Ribeiro-Mourao** and colleagues conducted a study in which they hypothesised that the renal angina index (RAI) could serve as an early prediction aid for the development of acute kidney injury (AKI) in 593 patients. When it came to predicting the onset of acute kidney injury (AKI) on day 1, RAI was better indicator than serum creatinine rise and KIDGO AKI STAGING . In order to foresee issues and make it possible to take preventative actions, it is utilized. RAI generated a

sensitivity value of 87.5% and a specificity value of 88.1% when it came to the identification of renal injury that was present in all different stages. (with all-stage AKI having Area under curve of 0.878 for RAI). Its performance increased for severe AKI (with Area under curve of 0.93). (49)

In a Study by **Ryo Matsuura et al**, from Japan, studied the usefulness and accuracy of Renal angina index to identify high risk persistent AKI, in Japanese hospitals from 2012-2014. AKI was present in 12.8% patients. The concept of RAI (renal angina) has come into use to highlight the characteristics of renal injury which is used to increase the suspicious of acute coronary syndrome. (42)

Studies on AKIN criteria:

Fiona A I Duthie et al, from Edinburgh, UK, studied the usefulness of the additive Euro-SCORE, RIFLE score and AKIN staging scores for the prediction and diagnosis of presence of acute kidney injury after cardiac surgery among 4,651 patients. According to the AKIN criteria, 12.4% of the population under study experienced Acute Kidney Injury (AKI), compared to 6.5% based on the RIFLE criteria. Acute kidney injury (AKI) following heart surgery is linked to prolonged hospital stay and elevated mortality rates.(50)

Anthony J Bastin et al, from. London, UK, studied the usefulness of the KDIGO, RIFLE score and AKIN staging scores for the prediction and diagnosis of presence of acute kidney injury after cardiac surgery among 1881 patients. The prevalence of AKI, as determined by the AKIN and RIFLE criteria, was 25.9% and 24.9%, respectively. The AKIN criteria yielded a considerably greater area under the ROC curve for hospital mortality compared to the RIFLE criteria (0.86 vs 0.78, P = .0009). (51)

Batoul Khoundabi et al, from Tehran, Iran, did a retrospective study, and studied the effects of some acute kidney injury risk factors on acute kidney injury from 300 cardiac-operated patients and also to investigate changes in urine output as a predictor of acute kidney injury using joint modelling. Acute kidney injury, as defined by the AKIN criteria, was observed in 38.0% of the patients. Females, infected patients, and individuals with low diastolic blood pressure saw a notable reduction in urine production more frequently.(52)

RESEARCH QUESTION OR HYPOTHESIS

1.1 RESEARCH QUESTION:

What are the incidence and risk factors of AKI among children with CHD undergoing post cardiac surgery?

What is the role of Renal Angina Index in predicting the AKI among children with Congenital Heart Disease (CHD) undergoing post cardiac surgery?

1.2 NULL HYPOTHESIS:

There is no relationship between the risk factors related and the incidence of AKI among children with CHD undergoing post cardiac surgery.

There is no relationship between the Renal Angina Index and the incidence of AKI among children with congenital heart disease undergoing post cardiac surgery.

1.3 ALTERNATE HYPOTHESIS:

Relationship exists between the risk factors related and the incidence of acute kidney injury among children with congenital heart disease undergoing post cardiac surgery.

SThere is a relationship between the Renal Angina Index and the incidence of AKI among children with congenital heart disease undergoing post cardiac surgery.

METHODOLOGY

Study population:

52 patients with CHD post cardiac surgery, in Department of CVTS Dr Prabhakar kore Hospital and MRC, Belagavi, Karnataka.

Study Design:

The aims of the investigation were taken into consideration when selecting the cross-sectional study design. Studies that are cross-sectional are conducted either at a single moment in time or over a very short period of time. They are widely utilized in the process of determining the frequency with which a specific outcome occurs in a particular group. These studies also give information about individual factors like exposure to risk factors. also we can get details of outcome . These studies serves as a snapshot , at a particular point of time ,for outcome as well as its associated characteristics. Therefore, it was determined that this particular study design was suitable for the current investigation.

Study Period:

Total Study period – 1 year (2023 January to 2023 December).

PHASE	TIME PERIOD	Activities done
1	**Jan 2023 - March 2023**	1.Identification of the problem. 2.Review of literature 3.Protocol preparation 4.Study document development consent forms 5.Submission of protocol. 6.Ethical clearance
2	**March 2023 – August 2023**	1.Recruitment of subjects. 2.Data collection 3.Follow up of subjects.
3	**August 2023 - Dec 2023**	1.Analysis of collected data 2.Discussion and report writing. 3.Submitting the final report.

Study setting:

KAHERs Jawaharlal Nehru Medical College, Belagavi, Karnataka.

Sampling Procedure:

Sampling is defined as the process of selecting a number of subjects from all the subjects available in a particular group or universe . A conclusion based on sample results may be attributed only to the population sampled.

In this study we contemplate Congenital Heart Disease children post cardiac surgery aged from 1month to 12years admitted in Department of ITU at Dr Prabhakar Kore Hospital & MRC Belagavi till we meet the sample size.

Inclusion Criteria:

- ✓ All children aged 1month to 12years (post cardiac surgery)

Exclusion criteria:

- ☒ Chronic kidney disease.
- ☒ Acquired heart disease.

Sample Size:

Sample size is calculated as below :

$$n = \frac{p(100-p)Z^2}{E\,2}$$

where n is the total size of the sample needed,

E is the percentage maximum error that is required,

Z is the value that corresponds to the level of confidence that is necessary, and

p is the percentage occurrence of a state or condition (also known as the proportion or prevalence).

The incidence of acute kidney injury in children after cardiac surgery was reported to be 9.6- 42%. Considering that about 15% children will have AKI in CHD children post cardiac surgery, at 95% confidence level and 10% maximum error, the sample size is given by,

$$n = \frac{15\ x\ (100-15) x\ 1.96^2}{(10)2}$$

$$n = 48.9804 = 49$$

Hence, 49 is minimal sample size for our study . As sample size increases, accuracy of result also increases.

METHOD OF DATA COLLECTION :

The steps used for data collection were follows:

- ⊕ After obtaining the approval from ethical committee, informed written consent from each of children's parent/guardian were obtained. Cases are those children with congenital cardiac diseases following post cardiac surgery in the Department of CTVS at Dr Prabhakar Kore Hospital & MRC Belagavi .For the diagnosis of AKI post cardiac , all patients met the criteria set forth by KIDGO.
- ⊕ Subjects are recruited according to the inclusion and exclusion criteria.
- ⊕ A detailed history with physical examination was taken.
- ⊕ Children aged between 1month to 12years where included in study design , assessing there pre op ,intra op , post op parameters where considered.
- ⊕ Antenatal and , postnatal detected cardiac disease , pre op RFT levels , pre op eGFR pre op Diuretics , pre op mechanical ventilation parameters were considered.
- ⊕ Intraoperative Aortic cross clamp timing and bypass time were considered.
- ⊕ Post op RFT levels within 72hours is collected, post op eGFR , post op urine output on Day 1,Day 2 and Day 3,post op mechanical ventilation duration , post op 2D Echo Post of diuretics , Anti pyretics and Anti biotics where considered.
- ⊕ Renal Angina Index (RAI) has two Scores :
- ⊕ **Risk strata** :

  ICU Admission , Solid organ or stem cell Transplantation ,Mechanical Ventilation Vasoactive support.

Injury strata :

SCr/Baseline , % FO Accumulation

AKIN criteria – Has stage 1 , stage 2, stage 3 according to Serum creatinine and urine output done within 12hours to 24hours.

- ⊕ As per the KIDGO - AKIN criteria and RAI index children have been evaluated for early prediction of AKI within 24 hours to 72hours following surgery.

Ethical Consideration:

Institutional Ethical Committee approval, from KAHERs Jawaharlal Nehru Medical College, Belagavi, Karnataka, was obtained prior the commencement of the study. Informed written consent was acquired.

Funding Source: None of the Sources Declared

The absence of any declared conflicts of interest.

Study STEPS:

The steps used for data collection were as follows:

- ⊕ After obtaining the approval from ethical committee, written informed consent from each of children's parent/guardian were obtained.
- ⊕ Subjects are recruited according to the inclusion and exclusion criteria.
- ⊕ A detailed history with physical examination was taken.
- ⊕ Infants, children who have been diagnosed with acute kidney injury in congenital heart disease post operatively were assessed with AKIN criteria and Renal Angina Index (RAI) within 24 hours to 72 hours of surgery.

Budget:

Self. (No added investigation or intervention)

Personnel: Nil

Investigations:	Rs. 20,000
Printing and copying supplies :	Rs. 20,000
Data Collection and Transport:	Rs. 10,000
Meeting and Other Expenses:	Rs. 10,000
Total Cost:	Rs. 60,000

Statistical analysis:

SPSS software version 21 was used to perform the analysis after the data was entered into a Microsoft Excel sheet.

I. Descriptives:

1. Continuous variables like Age, Body surface area, Urea Creatinine values, pre -op and post op renal functions, intra op bypass time, Aortic cross clamp time, Urine output, etc., are expressed in mean, SD, median, mode, Range, minimum and maximum values. When it is deemed necessary, histograms are utilized.

2. Categorical variables like, gender, Timing of detection of CHD, diagnosis, Pre-op diuretic usage, Usage of post op inotropes, NSAIDS, Antibiotics, type of surgery, RISK criteria scores, Renal Angina Index categories, AKIN stages, etc., are expressed in frequency and percentage. When it is deemed appropriate, pie charts and bar diagrams are utilized.

II. Inferentials:

1. Renal Angina Index (RA index) was categorized with the cut off of 8 signifying more than 8 is suggestive of Acute Kidney Injury.
2. The association between RA index and AKIN staging was done using Chi square test.
3. When a Categorical Variable like gender and pre-op diuretics given were associated with RA index and AKIN staging, chi-square test was used for testing the significance. In situations where more than twenty percent of the cell values have an expected cell value that is lower than five, Fisher's exact test is utilized.
4. When a numerical variable like age and other pre-op and post-op parameters were associated with RA index and AKIN staging, the variables were represented by mean (± standard deviation) in tables and the significance of the difference between the means is tested by Student 't' test.
5. p-values less than 0.05 were considered statistically significant.

RESULTS

Findings of the study is being described under the following headings:

I. Age (years)

II. Gender

III. Body Surface Area

IV. Postnatal detection of CHD

V. Diagnosis

VI. Pre-op Renal functions

VII. Pre-op diuretics

VIII. Intra-op Bypass time

IX. Aortic cross clamp time

X. Post-op Renal functions

XI. Urine output

XII. Post-op Ventilation (days)

XIII. Post-op Inotropes

XIV. NSAIDS

XV. Antibiotics

XVI. RA index

XVII. AKI by RA index

XVIII. AKIN

XIX. RA index with AKIN

XX. Pre-op Characteristics with AKIN staging

XXI. Post-op Characteristics with AKIN staging

XXII. Pre-op Characteristics with RA index

XXIII. Post-op Characteristics with RA index

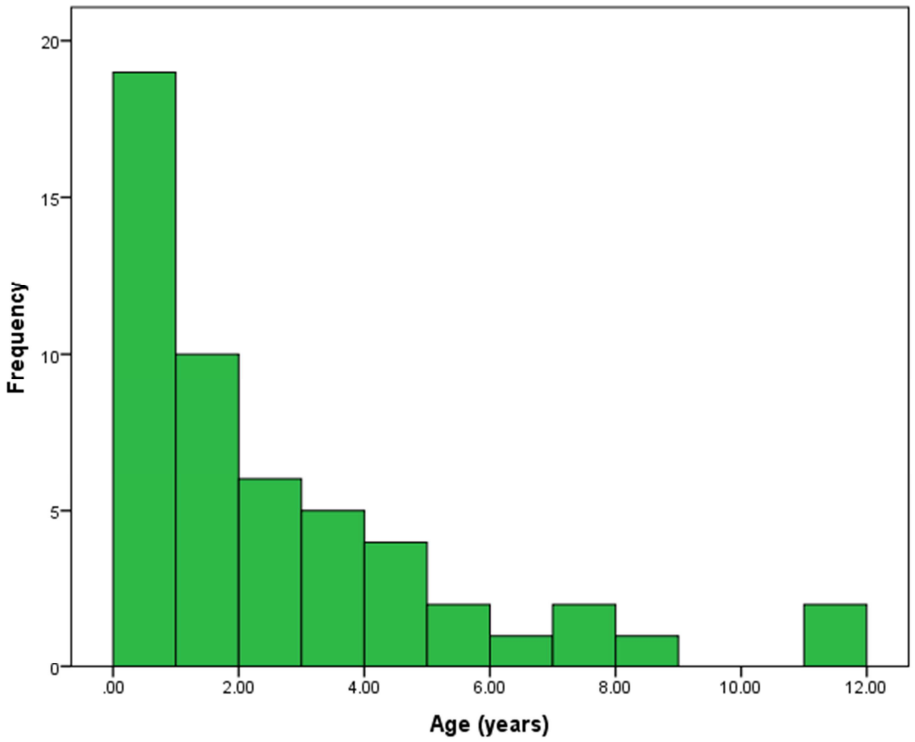
I. Age (years)

The mean Age (years) was 2.42 (± 2.6) ranging from 0.08 to 12 years.

Table 6. Age (in years)

Age (in years)	
Mean value	2.42
Median value	1.50
SD	2.60
Range	10.92
Minimum value	0.083
Maximum value	11

Figure 1. Age (years)



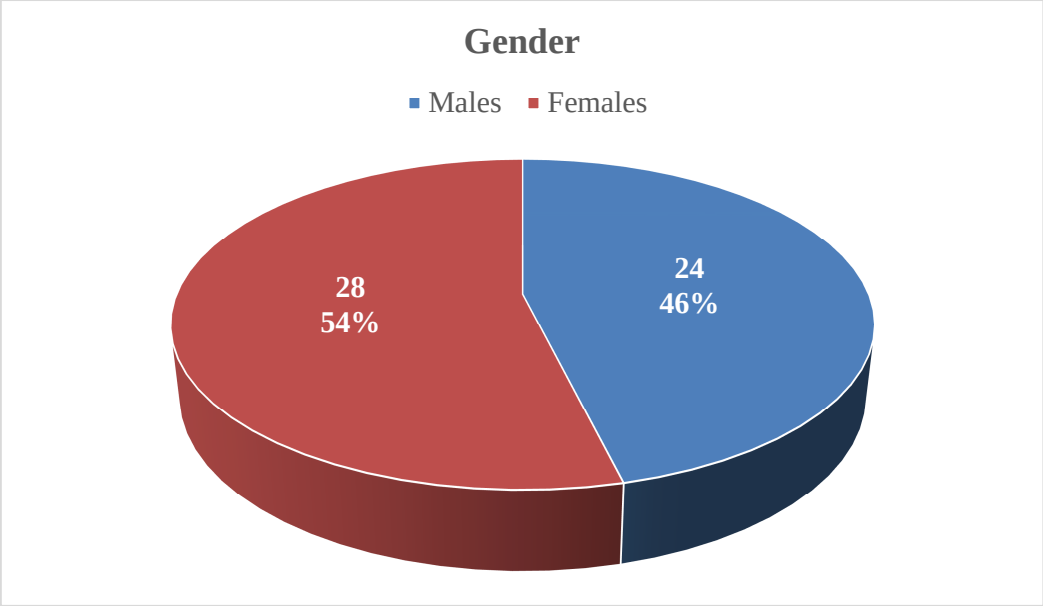
II. Gender

Among the subjects, 28 (53.85%) were Female children and 24 (46.15%) were Male children.

Table 7. Gender

SEX	Frequency	Percentage
Male	24	46.15
Female	28	53.85
Total	52	100.00

Figure 2. Gender



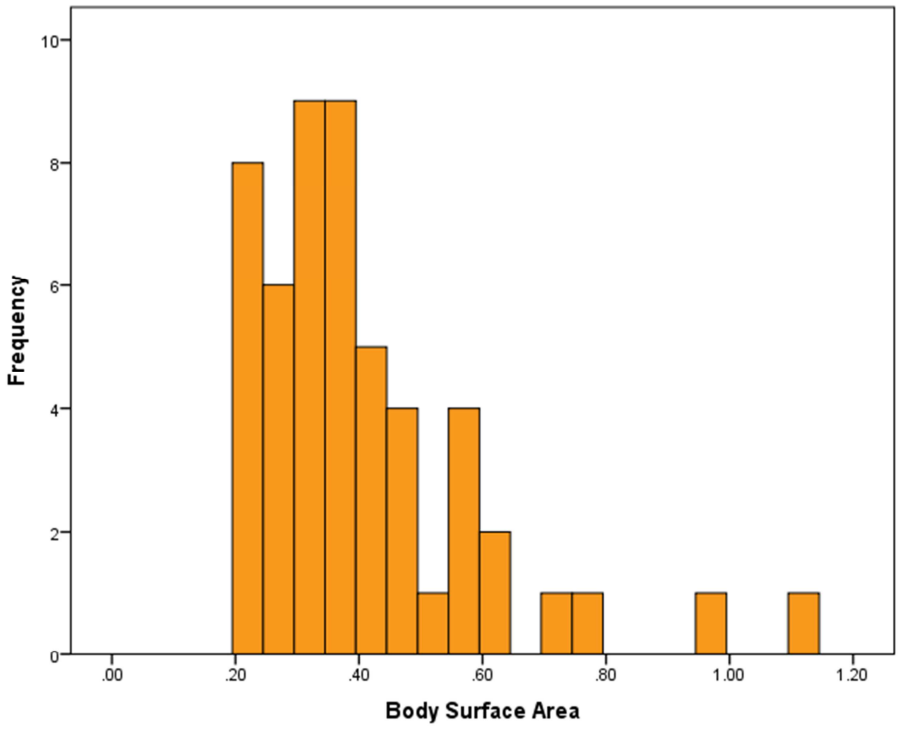
III. Body Surface Area

The mean Body Surface Area was 0.41 (± 0.18) ranging from 0.22 to 1.1 sq.m.

Table 8. Body Surface Area

Body Surface Area	
Mean	0.41
Median	0.36
Std. Deviation	0.18
Range	0.88
Minimum	0.22
Maximum	1.1

Figure 3. Body Surface Area



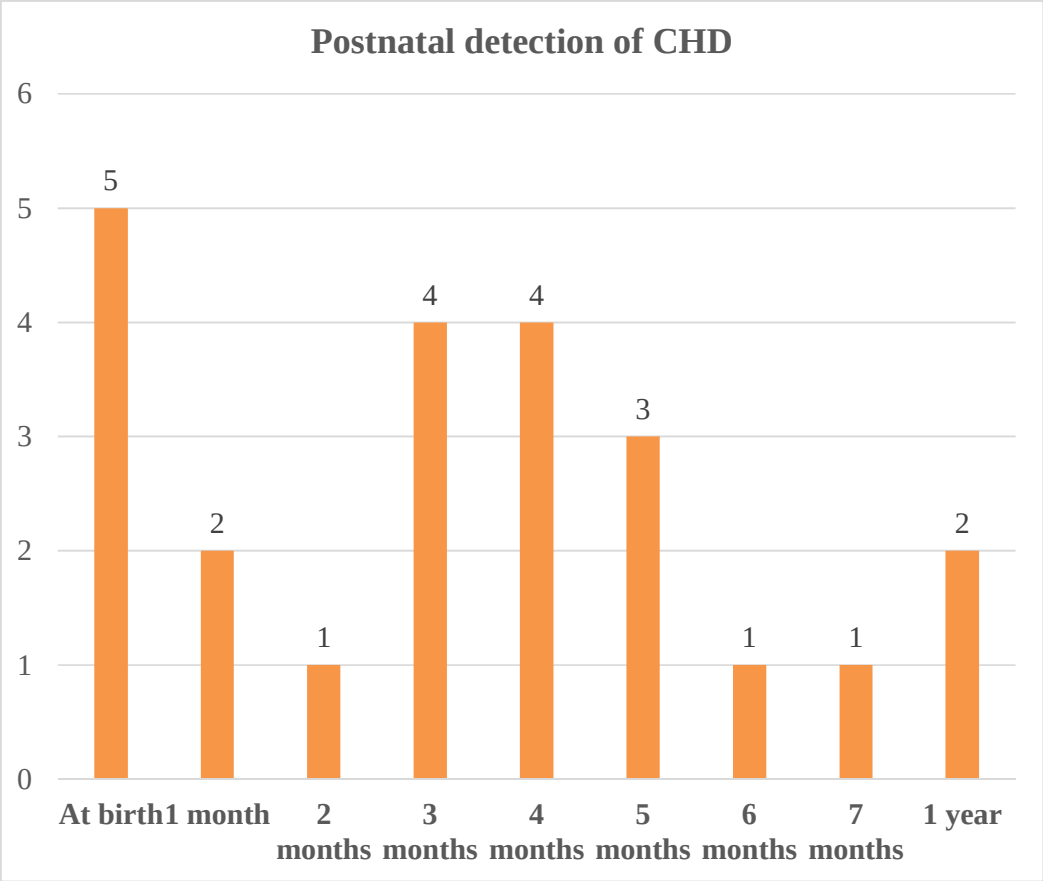
IV. Postnatal detection of CHD

Among the subjects, 29 (55.77%) had at No detection of CHD postnatally, 5 (9.62%) had at birth, 4 (7.69%) had at 3 months, 4 (7.69%) had at 3 months and 3 (5.77%) had at 5 months

Table 9. Postnatal detection of CHD

Postnatal detection of CHD	Frequency	Percent
No	29	55.77
At birth	5	9.62
1 month	2	3.85
2 months	1	1.92
3 months	4	7.69
4 months	4	7.69
5 months	3	5.77
6 months	1	1.92
7 months	1	1.92
1 year	2	3.85
Total	52	100.00

Figure 4. Postnatal detection of CHD



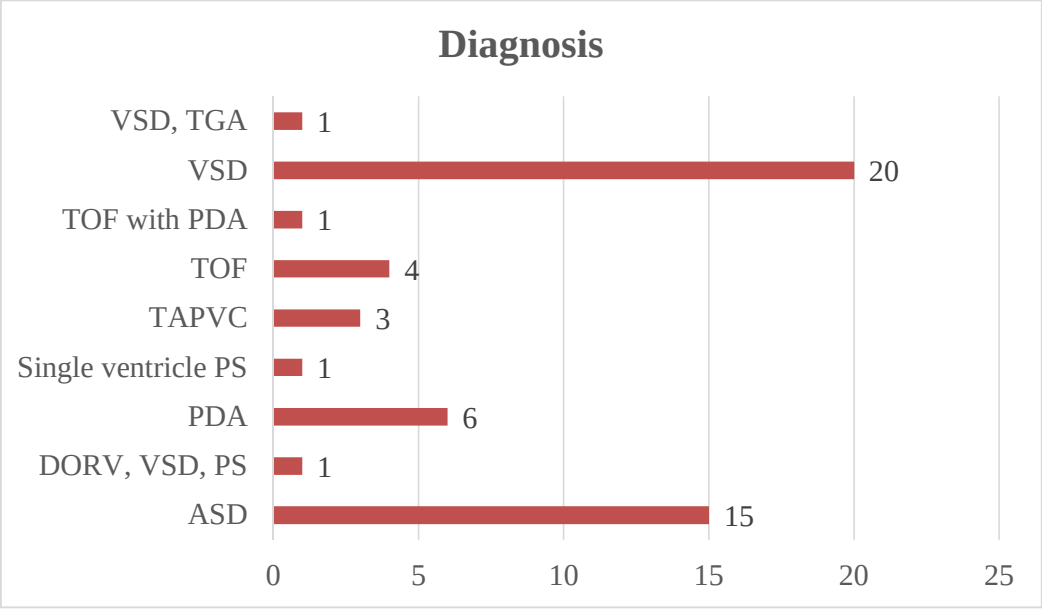
V. Diagnosis

Among the subjects, 20 (38.46%) had VSD, 15 (28.85%) had ASD, 6 (11.54%) had PDA, 4 (7.69%) had TOF and 3 (5.77%) had TAPVC

Table 10. Diagnosis

Diagnosis	Frequency	Percent
ASD	15	28.85
DORV, VSD, PS	1	1.92
PDA	6	11.54
Single ventricle PS	1	1.92
TAPVC	3	5.77
TOF	4	7.69
TOF with PDA	1	1.92
VSD	20	38.46
VSD, TGA	1	1.92
Total	52	100.00

Figure 5. Diagnosis



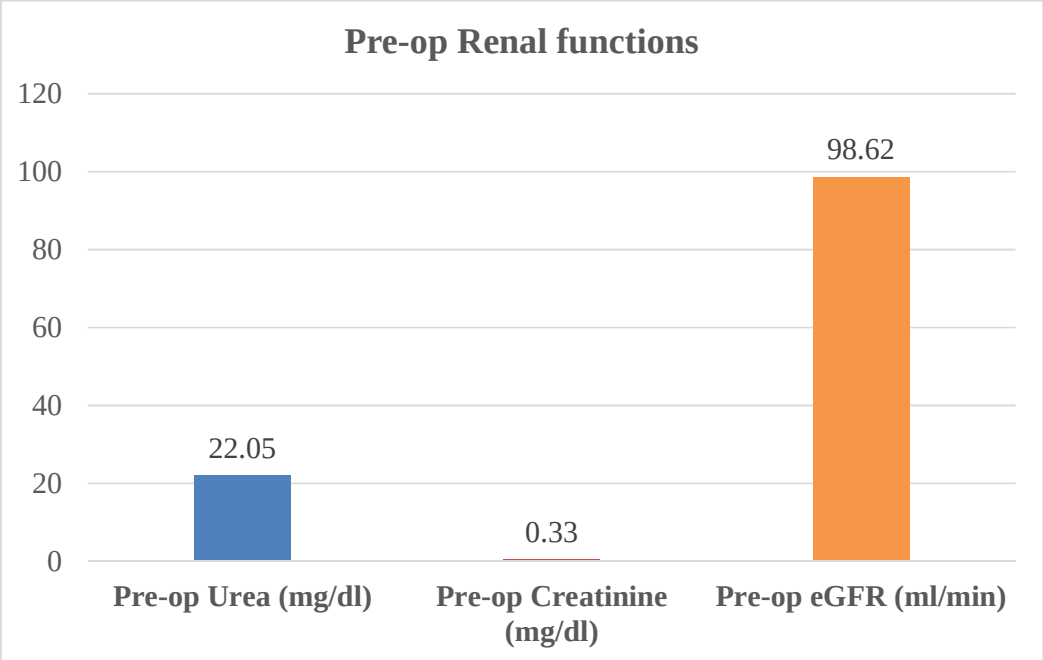
VI. Pre-op Renal functions

The mean Pre-op Urea (mg/dl) was 22.05 (± 8.22) ranging from 10 to 52 mg/dl. The mean Pre-op Creatinine (mg/dl) was 0.33 (± 0.11) ranging from 0.14 to 0.68 mg/dl. The mean Pre-op eGFR (ml/min) was 98.62 (± 30.56) ranging from 38 to 188.8 ml/min.

Table 11. Pre-op Renal functions

	N	Mean	S.D.	Minimum	Maximum
Pre-op Urea (mg/dl)	52	22.05	8.22	10.0	52.0
Pre-op Creatinine (mg/dl)	52	0.33	0.11	0.1	0.7
Pre-op eGFR (ml/min)	52	98.62	30.56	38.0	188.8

Figure 6. Pre-op Renal functions



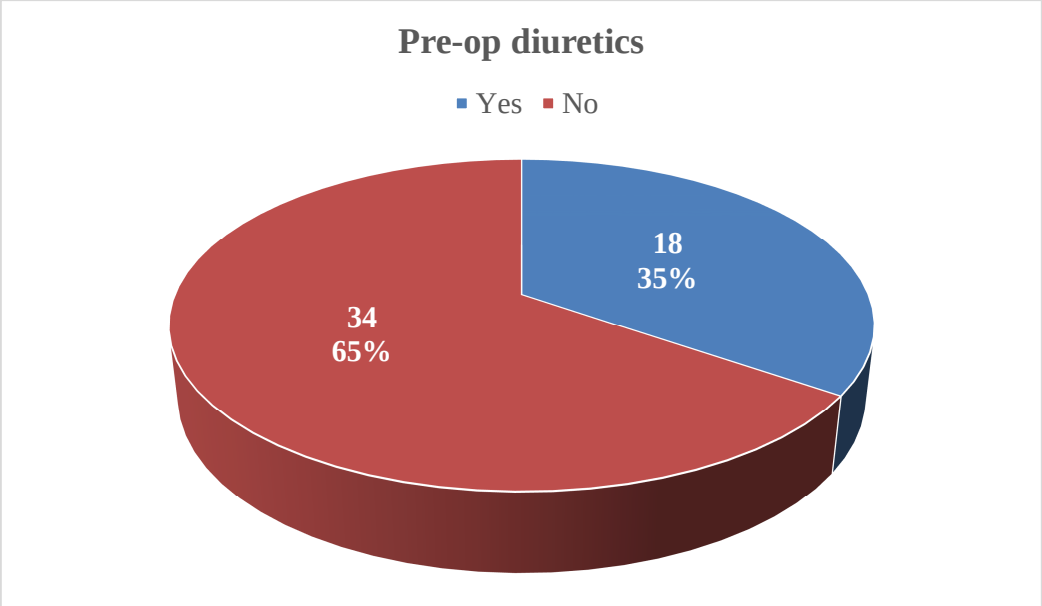
VII. Pre-op diuretics

Among the subjects, 18 (34.62%) had Pre-op diuretics.

***Table** 12. Pre-op diuretics*

Pre-op diuretics	Frequency	Percent
Yes	18	34.62
No	34	65.38
Total	52	100.00

Figure 7. Pre-op diuretics



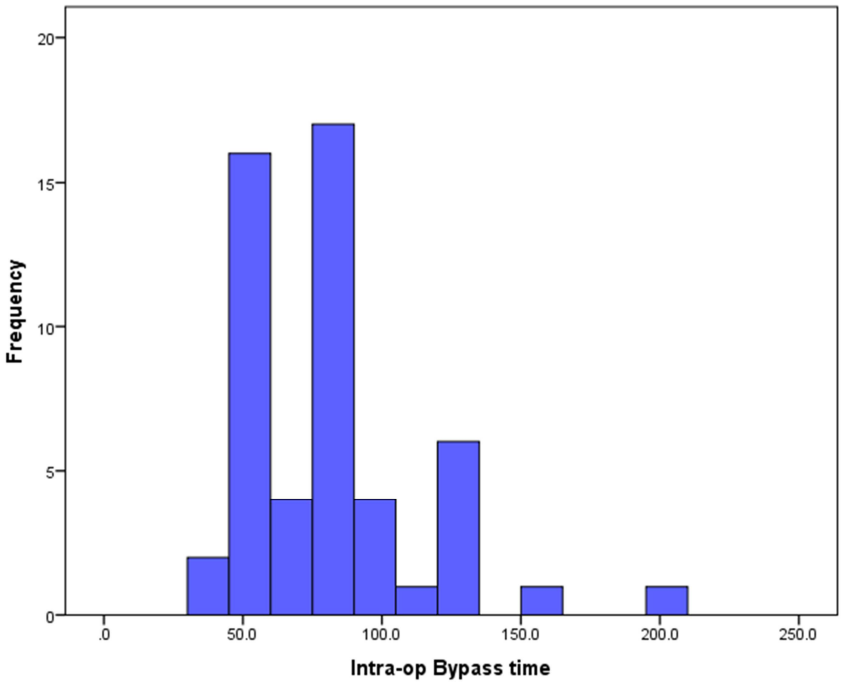
VIII. Intra-op Bypass time

The mean Intra-op Bypass time was 80.15 (± 32.84) ranging from 36 to 207 min.

Table 13. Intra-op Bypass time

Intra-op Bypass time	
Mean	80.15
Median	77.50
Std. Deviation	32.84
Range	171
Minimum	36
Maximum	207

Figure 8. Intra-op Bypass time



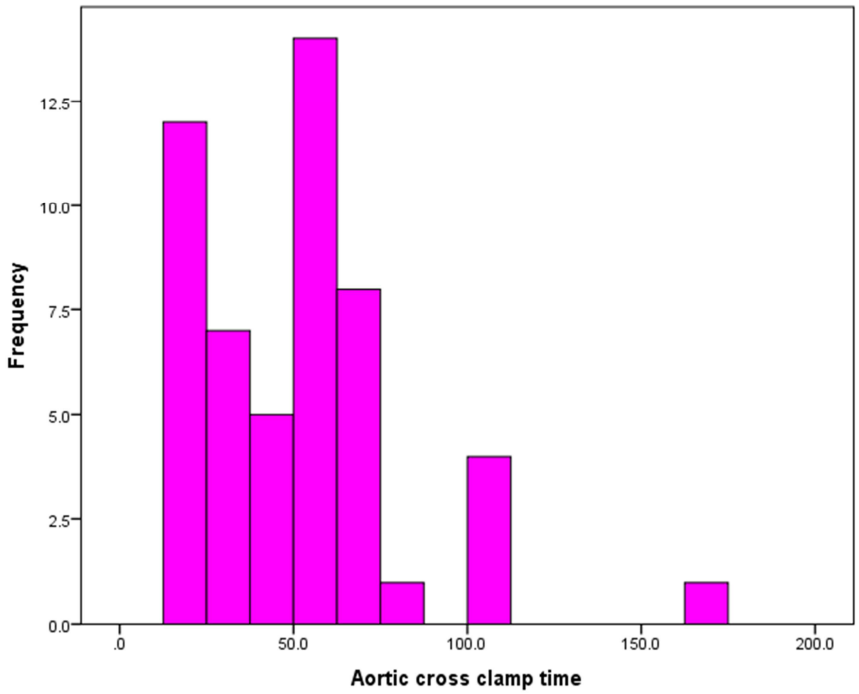
IX. Aortic cross clamp time

The mean Aortic cross clamp time was 52.19 (± 28.45) ranging from 18 to 165 min.

Table 14. Aortic cross clamp time

Aortic cross clamp time	
Mean	52.19
Median	54.00
Standard Deviation	28.45
Range	147
Minimum	18
Maximum	165

Figure 9. Aortic cross clamp time



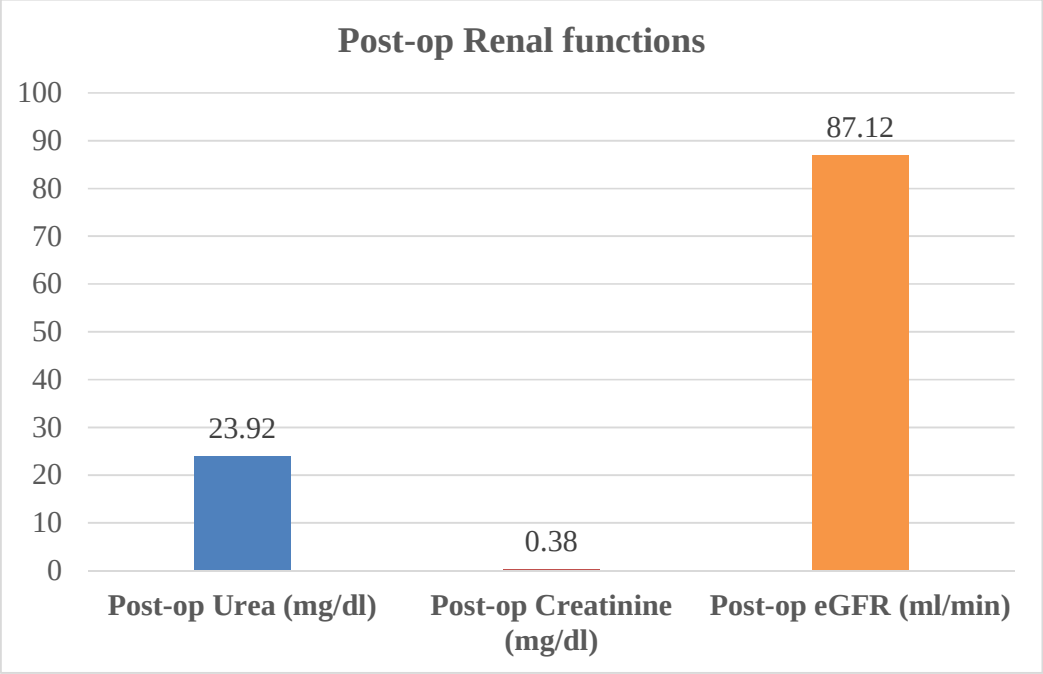
X. Post-op Renal functions

The mean Post-op Urea (mg/dl) was 23.92 (± 6.6) ranging from 16 to 41 mg/dl. The mean Post-op Creatinine (mg/dl) was 0.38 (± 0.19) ranging from 0 to 1.5 mg/dl. The mean Post-op eGFR (ml/min) was 87.12 (± 22.11) ranging from 37.5 to 132.1 ml/min.

Table15. Post-op Renal functions

	N	Mean	S.D.	Minimum	Maximum
Post-op Urea (mg/dl)	52	23.92	6.60	16.0	41.0
Post-op Creatinine (mg/dl)	52	0.38	0.19	0.0	1.5
Post-op eGFR (ml/min)	52	87.12	22.11	37.5	132.1

Figure 10. Post-op Renal functions



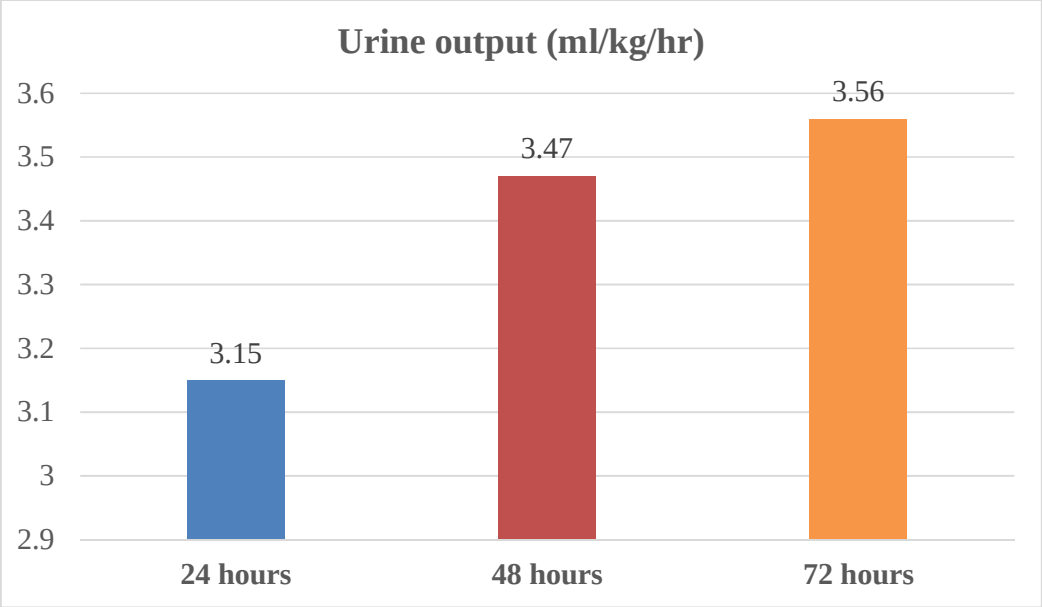
XI. Urine output

The mean 24hrs Urine output (ml/kg/hr) was 3.15 (± 1.29) ranging from 0.7 to 7 ml/kg/hr. The mean 48hrs Urine output (ml/kg/hr) was 3.47 (± 1.41) ranging from 0.7 to 7.4 ml/kg/hr. The mean 72hrs Urine output (ml/kg/hr) was 3.56 (± 1.56) ranging from 0.8 to 6.6 ml/kg/hr.

***Table** 16. Urine output*

	N	Mean	S.D.	Minimum	Maximum
24hrs Urine output (ml/kg/hr)	52	3.15	1.29	0.7	7.0
48hrs Urine output (ml/kg/hr)	52	3.47	1.41	0.7	7.4
72hrs Urine output (ml/kg/hr)	52	3.56	1.56	0.8	6.6

Figure 11. Urine output



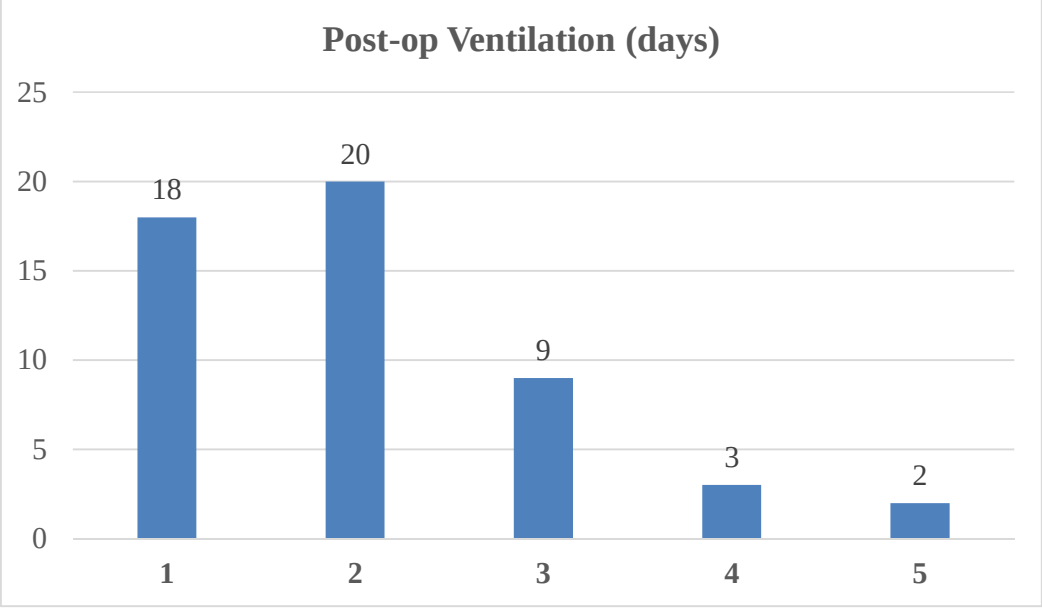
XII. Post-op Ventilation (days)

Among the subjects, 20 (38.46%) had 2 days, 18 (34.62%) had 1 days, 9 (17.31%) had 3 days, 3 (5.77%) had 4 days and 2 (3.85%) had 5 days of mechanical ventilation post operatively.

Table 17. Post-op Ventilation (days)

Post-op Ventilation (days)	Frequency	Percent
1	18	34.62
2	20	38.46
3	9	17.31
4	3	5.77
5	2	3.85
Total	52	100.00

Figure 12. Post-op Ventilation (days)



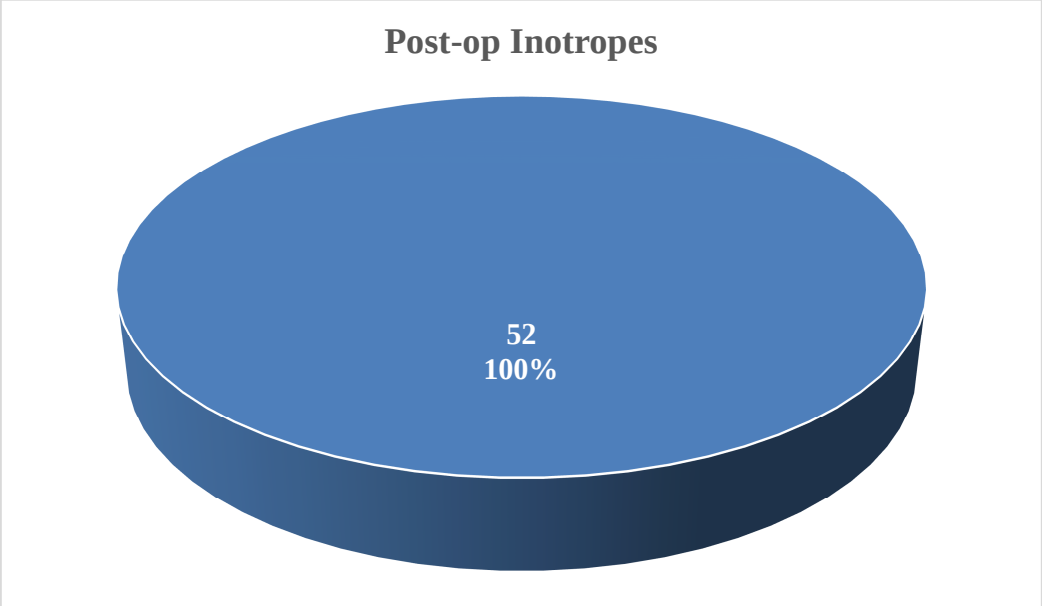
XIII. Post-op Inotropes

Among the subjects, 52 (100%) had Inotropes post operatively.

***Table** 18. Post-op Inotropes*

Post-op Inotropes	Frequency	Percent
yes	52	100.00

Figure 13. Post-op Inotropes



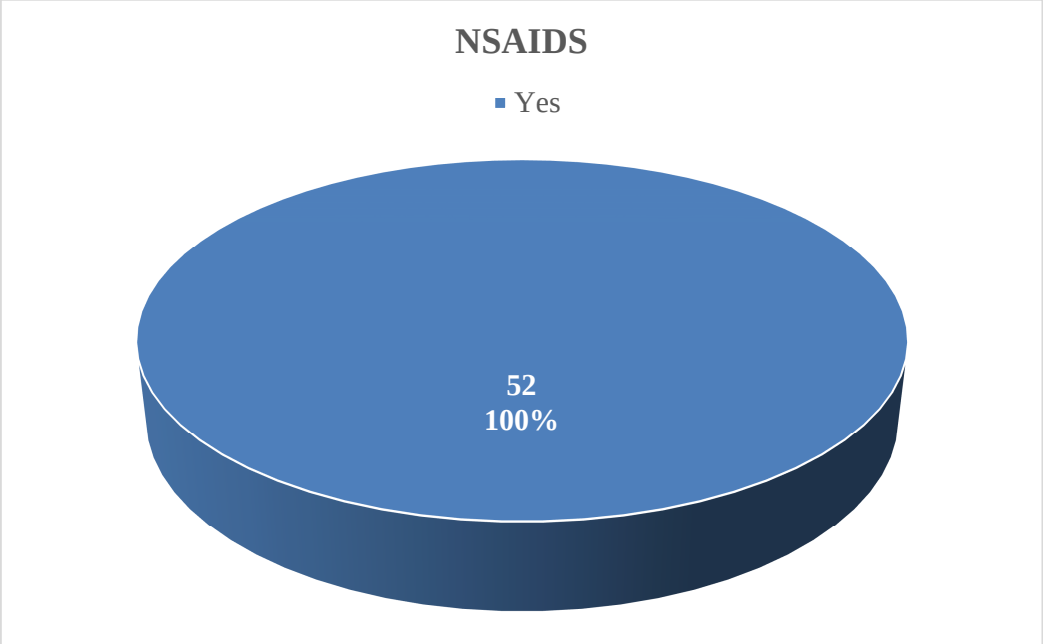
XIV. NSAIDS

Among the subjects, 52 (100%) had NSAIDS post operatively.

***Table** 19. NSAIDS*

NSAIDS	Frequency	Percent
Yes	52	100.00

Figure 14. NSAIDS



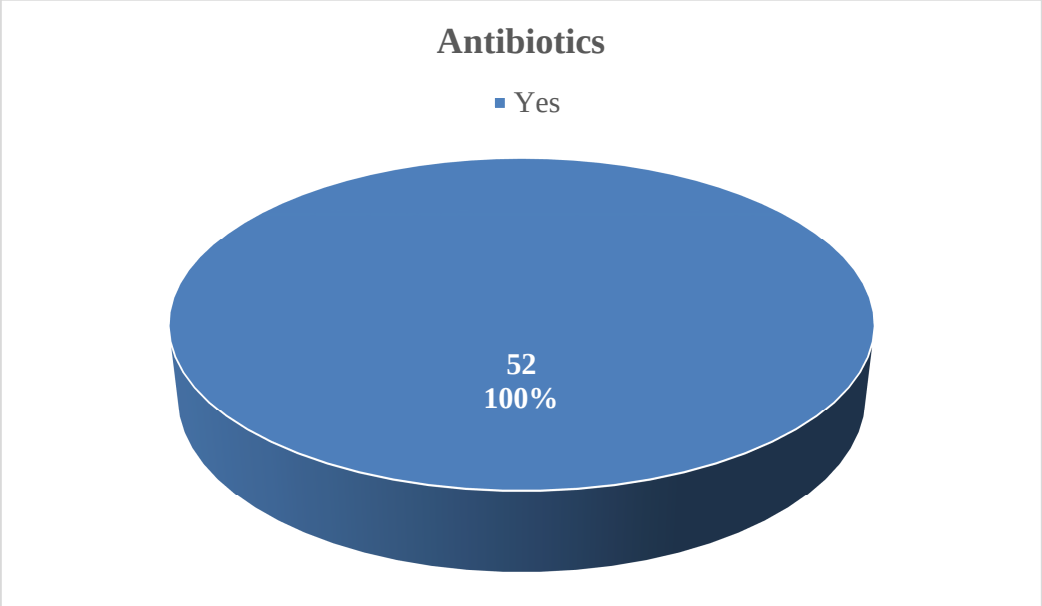
XV. Antibiotics

Among the subjects, 52 (100%) had Antibiotics post operatively.

***Table** 20. Antibiotics*

Antibiotics	Frequency	Percent
Yes	52	100.00

Figure 15. Antibiotics



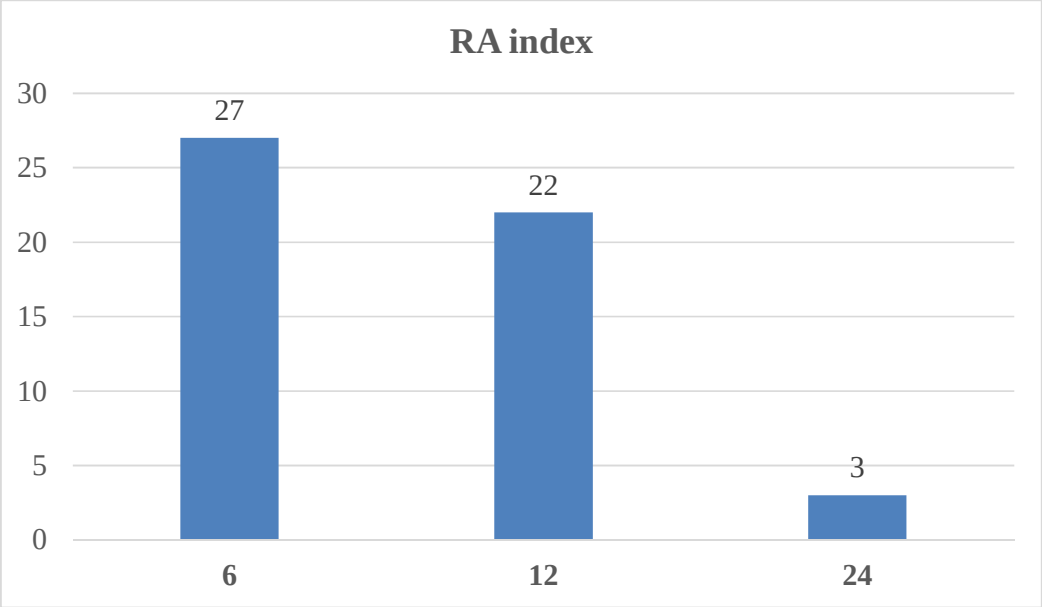
XVI. RA index

Among the subjects, 27 (51.92%) had RA index score of 6, 22 (42.31%) had score of 12 and 3 (5.77%) had score of 24 out of 40.

***Table** 21. RA index*

RA index (score / 40)	Frequency	Percent
6	27	51.92
12	22	42.31
24	3	5.77
Total	52	100.00

Figure 16. RA index



XVII. AKI by RA index

Among the subjects, 25 (48.08%) had AKI by RA index

Table 22. AKI by RA index

AKI by RA index	Frequency	Percent
Yes	25	48.08
No	27	51.92
Total	52	100.00

Figure 17. AKI by RA index

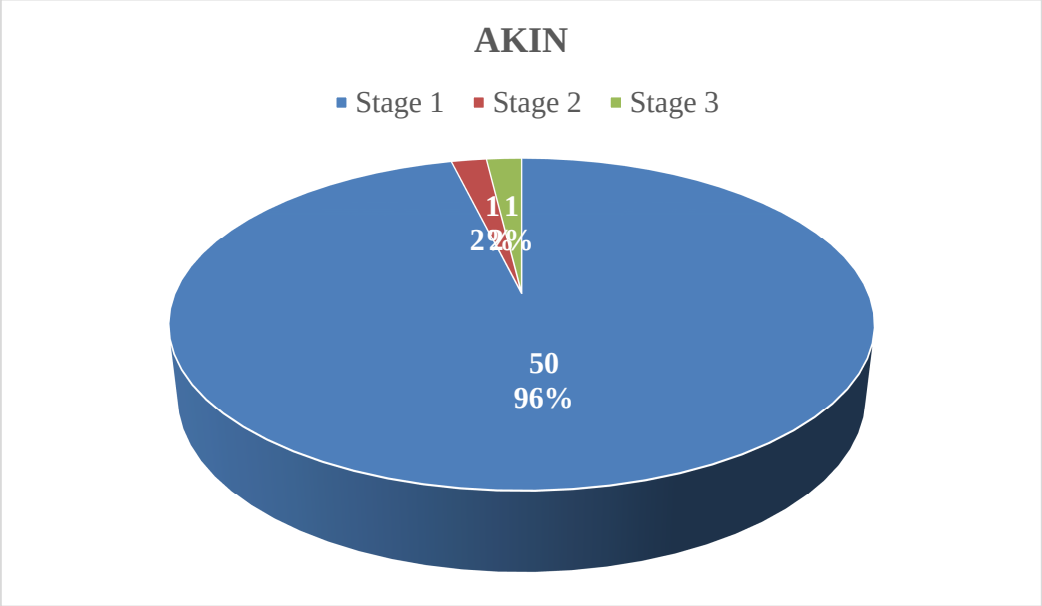
XVIII. AKIN

Among the subjects, 50 (96.15%) had Stage 1, 1 (1.92%) had Stage 2 and 1 (1.92%) had Stage 3.

***Table** 23. AKIN*

AKIN	Frequency	Percent
Stage 1	50	96.15
Stage 2	1	1.92
Stage 3	1	1.92
Total	52	100.00

Figure 18. AKIN



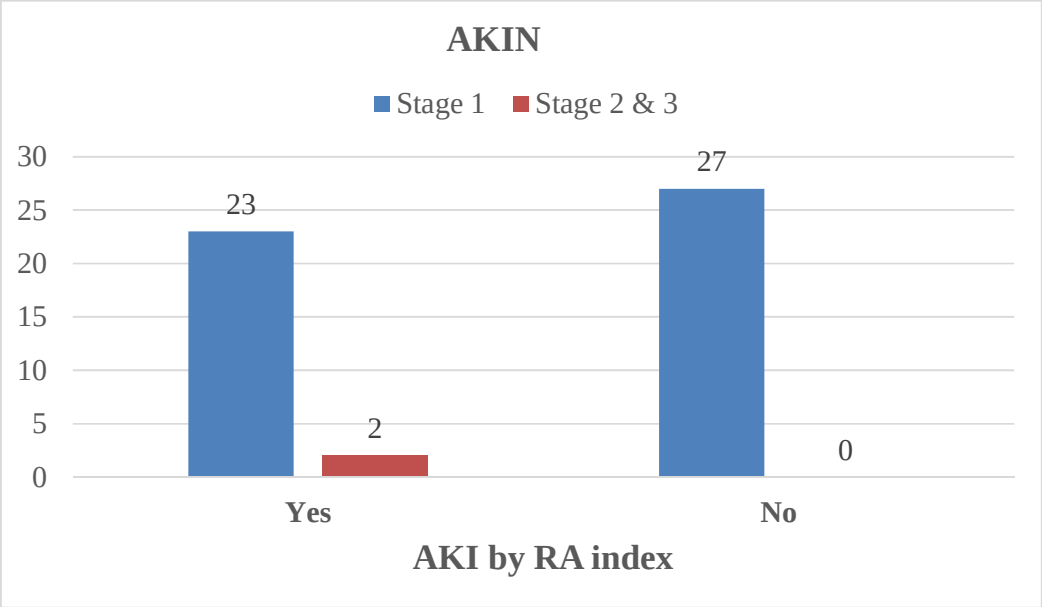
XIX. RA index with AKIN

92% of the AKI by RA index had Stage 1 and 8% had stage 2 and 3. All of the subjects with RA index less than 8 had stage 1. The association between AKI by RA index and AKIN was not statistically significant ($p > 0.05$) which signifies AKI by RA index and AKIN had no difference and correlates with each other.

Table 24. RA index with AKIN

AKI by RA index	AKIN		Total	Fisher exact p value
	Stage 1	Stage 2 & 3		
Yes	23 (92%)	2 (8%)	25 (100%)	0.227
No	27 (100%)	0 (0%)	27 (100%)	
Total	50 (96.15%)	2 (3.84%)	52 (100%)	

Figure 19. RA index with AKIN



XX. Pre-op Characteristics with AKIN staging

Age, gender and body surface area of the subjects had no statistically significant difference between the AKIN staging groups. Pre-op parameters like urea, creatinine and GFR and diuretics given also had no statistically significant difference between the groups and were comparable.

Table 25. Pre-op Characteristics with AKIN staging

CHARACTERISTIC	AKIN		p value by 't' test (# - Chi sq. test)
	Stage 1	Stage 2 & 3	
Age (years)	2.32 (± 2.53)	4.75 (± 4.6)	0.198
Gender (Males)	22 (91.7%)	2 (8.3%)	0.208
Body Surface Area	0.4 (± 0.17)	0.7 (± 0.42)	0.495
Pre-op diuretics	18 (100%)	0 (0%)	0.423
Pre-op Urea (mg/dl)	21.92 (± 7.96)	25.5 (± 17.68)	0.550
Pre-op Creatinine (mg/dl)	0.34 (± 0.11)	0.3 (± 0.08)	0.610
Pre-op eGFR (ml/min)	99.12 (± 29.51)	86.1 (± 68.02)	0.560

XXI. Post-op Characteristics with AKIN staging

The Intra-op characteristics like bypass time and aortic cross clamp time had no statistically significant difference between AKIN staging groups. Post-op urea and creatinine were significantly high among stage 2 & 3 subjects compared to stage 1 subjects. eGFR had no statistically significant difference between AKIN staging. Urine output was significantly higher in stage 1 subjects at 24 hours, 48 hours and 72 hours compared to stage 2 & 3 subjects.

Table 26. Post-op Characteristics with AKIN staging

CHARACTERISTIC	AKIN		p value by 't' test
	Stage 1	Stage 2 & 3	
Intra-op Bypass time	80.62 (± 33.14)	68.5 (± 30.41)	0.614
Aortic cross clamp time	52.6 (± 28.61)	42 (± 31.11)	0.610
Post-op Urea (mg/dl)	23.54 (± 6.26)	33.5 (± 10.61)	0.035
Post-op Creatinine (mg/dl)	0.35 (± 0.1)	1.04 (± 0.65)	0.001
Post-op eGFR (ml/min)	87.56 (± 21.04)	76.25 (± 54.8)	0.819
24hrs Urine output (ml/kg/hr)	3.2 (± 1.28)	1.7 (± 0.28)	0.009
48hrs Urine output (ml/kg/hr)	3.54 (± 1.4)	1.85 (± 0.21)	0.001
72hrs Urine output (ml/kg/hr)	3.65 (± 1.53)	1.3 (± 0)	0.036

XXII. Pre-op Characteristics with RA index

Age, gender and body surface area of the subjects had no statistically significant difference between the RA index groups. Pre-op parameters like urea, GFR and diuretics given also had no statistically significant difference between the groups and were comparable. Pre-op creatinine was slightly higher among subjects with RA index more than 8 which was statistically significant.

Table 27. Pre-op Characteristics with RA index

CHARACTERISTIC	**RA index**		**p value by 't' test (# - Chi sq. test)**
	> 8	**< 8**	
Age (years)	2.82 (± 3)	1.65 (± 1.37)	0.061
Gender (Males)	14 (58.3%)	10 (41.7%)	0.143#
Body Surface Area	0.42 (± 0.21)	0.39 (± 0.13)	0.569
Pre-op diuretics	14 (77.8%)	4 (22.2%)	0.100#
Pre-op Urea (mg/dl)	22.64 (± 8.73)	20.94 (± 7.25)	0.484
Pre-op Creatinine (mg/dl)	0.31 (± 0.08)	0.38 (± 0.14)	0.024
Pre-op eGFR (ml/min)	102.85 (± 31.59)	90.64 (± 27.61)	0.173

XXIII. Post-op Characteristics with RA index

The Intra-op characteristics like bypass time and aortic cross clamp time had no statistically significant difference between RA index groups. Post-op urea and creatinine, eGFR and urine output at various time intervals also did not have any significant difference between the RA index groups.

Table 28. Post-op Characteristics with RA index

CHARACTERISTIC	RA index		p value by 't' test
	> 8	< 8	
Intra-op Bypass time	70.97 (± 22.2)	97.5 (± 42.33)	0.051
Aortic cross clamp time	44.56 (± 20.26)	66.61 (± 35.98)	0.057
Post-op Urea (mg/dl)	24.4 (± 6.64)	23.02 (± 6.63)	0.479
Post-op Creatinine (mg/dl)	0.39 (± 0.22)	0.37 (± 0.09)	0.794
Post-op eGFR (ml/min)	88.56 (± 23.4)	84.41 (± 19.79)	0.525
24hrs Urine output (ml/kg/hr)	3.12 (± 1.3)	3.2 (± 1.3)	0.829
48hrs Urine output (ml/kg/hr)	3.56 (± 1.39)	3.31 (± 1.47)	0.549
72hrs Urine output (ml/kg/hr)	3.47 (± 1.44)	3.73 (± 1.81)	0.582

DISCUSSION

This cross-sectional study was conducted on 52 children with primary purpose of identifying the incidence of acute renal damage and its associated risk factors in children with congenital heart disease and are undergoing cardiac surgery. All children aged 1month to 12years (post cardiac surgery) admitted during the study period of 1 year (2023 January to 2023 December) with congenital heart disease undergoing surgery were included.

After obtaining the approval from ethical committee, written informed consent from each of children's parent/guardian were obtained. After obtaining detailed history, physical examination was done. Infants, children who have been diagnosed with acute kidney injury in congenital heart disease post operatively were assessed with AKIN criteria and Renal Angina Index (RAI) within 24 hours to 48 hours of surgery.

Baseline characteristics:

Age (years):

In this study, The mean Age (years) was 2.42 (± 2.6) ranging from 0.08 to 11 years. This is similar to study results from **Zaccaria Ricci et al,** The median age was 2.47 years (interquartile range [IQR], 0.38-9.85 years). (53) The mean Age (years) among those with AKIN stage I was 2.32 (± 2.53) which is not significantly lesser compared to 4.75 (± 4.6) in those without AKIN stage 2 & 3. Similar to our study results. Those with severe acute kidney injury were substantially older. ($p = 0.004$) in study by **Zaccaria Ricci et al.** (53)

On the contrary, **Murat Tanyildiz et al,** in their study, reported that Children younger than 11 months were more higher chance to develop AKI ($P < 0.005$). (54) **Sidharth Kumar Sethi et al**, observed that Age less than one year and young children were found to be independent risk factors for acute kidney injury of any severity. (46)

Gender:

In this study, 28 (53.85%) were Female children and 24 (46.15%) were Male children. 8.33% of the Males had AKIN stage 2 & 3, which is higher than 7.14% in Females but the association between Gender and AKI was not statistically significant ($p > 0.05$). **Sonia A EL-Saiedi et al** in their study observed among the 44 participants, 26 males (59.1%) and 18 (40.9%) females, but there was no significant relationship with the incidence of AKI, similar to our study. (55)

Diagnosis:

The survival rate for infants born with congenital heart disease (CHD) has significantly increased due to surgical procedures like PDA ligation, coarctation of the aorta repair, ASD repair, VSD repair, tetralogy of fallot repair, transposition of the great vessels repair, truncus arteriosus repair, tricuspid atresia repair, TAPVR correction, and hypoplastic left heart repair. (56,57) In this study, majority of the participants were undergoing surgery for 20 (38.46%) VSD, followed by 15 (28.85%) had ASD, 6 (11.54%) had PDA, 4 (7.69%) had TOF and 3 (5.77%) had TAPVC.

AKI:

In this study, 25 (48.08%) had AKI by RA index. Among the subjects, 50 (96.15%) had Stage 1, 1 (1.92%) had Stage 2 and 1 (1.92%) had Stage 3 according to AKIN. In the study by **Simon Li et al**, 130 patients (42%) out of 311 children experienced acute renal injury within 3 days following surgery.(43) **Bilal Aoun et al**, observed that Fourteen patients (9.3%) developed AKI using the KDIGO criteria.(45) **Sidharth Kumar Sethi et al**, observed that the twenty patients - (9.6%) had (AKI-I 15 patients; AKI-II 1 patient and AKI-III 4 patients) (14 of them were infants) had acute renal damage (AKI). (46) **Georgios Kourelis et al**, observed that Seventy (19.3%) patients had AKI, and the in-hospital mortality rate for this group was 21.4%.(47) Cardiorenal syndrome, or "interorgan "cross talk," may be mediated by cytokines and chemokines and result in left ventricular failure when AKI is present.(58)

Pre operative findings:

Pre-op Renal functions:

This study showed average Pre-op Urea (mg/dl)levels as 22.05 (± 8.22) ranging from 10 to 52 mg/dl. The mean Pre-op Urea (mg/dl) was among the stage I was 21.92 (± 7.96) and among the stage 2 and 3 were 25.5 (± 17.68) and these differences were not statistically significant. The mean Pre-op Creatinine (mg/dl) was 0.33 (± 0.11) ranging from 0.14 to 0.68 mg/dl. The mean Pre-op Creatinine (mg/dl) was among the stage I was 0.34 (± 0.11) and among the stage 2 and 3 were 0.3 (± 0.08) and these differences were not statistically significant. The mean Pre-op eGFR (ml/min) was 98.62 (± 30.56) ranging from 38 to 188.8 ml/min. The mean Pre-op

eGFR (ml/min) was among the stage I was 99.12 (± 29.51) and among the stage 2 and 3 were 86.1 (± 68.02) and these differences were not statistically significant. **Scott I. Aydin et al**, observed that AKI was linked to greater preoperative blood urea nitrogen and lower preoperative serum creatinine levels.(48) In this study, 18 (34.62%) had Pre-op diuretics.

Intraoperative findings:

Intra-op Bypass time:

The mean Intra-op Bypass time was 80.15 (± 32.84) ranging from 36 to 207 min. The mean Intra-op Bypass time (min) among those with AKIN stage I was 80.62 (± 33.14) which is higher by 12.12 but statistically insignificant as compared to 68.5 (± 30.41) in those with AKIN stage 2 &3. In the study by **Simon Li et al**, cardiopulmonary bypass duration were found to be independently linked to the risk of acute renal damage.(43)

Sidharth Kumar Sethi et al, observed that cardiopulmonary bypass duration was found to be independent risk factors for acute kidney injury of any severity. (46) **Georgios Kourelis et al**, observed that long time for performing cardiopulmonary bypass, and preoperative mechanical ventilation, were linked to AKI.(47)

In this study, The mean Aortic cross clamp time was 52.19 (± 28.45) ranging from 18 to 165 min. The mean Aortic cross clamp time (min) among those with AKIN stage I was 52.6 (± 28.61) which is higher by 10.6 but statistically insignificant as compared to 42 (± 31.11) in those with AKIN stage 2 &3.

Post operative findings:

Post-op Renal functions:

The current bench mark criteria for AKI identification is serum levels of creatinine (SCr); however, due to its dependence on nonrenal variables such as age, gender, muscle mass, fluid balance, and drugs that may change creatinine tubular excretion, SCr is not the best biomarker. (59,60) Significant renal damage is seen in even minor increases in SCr (0.3 mg/dl), which are linked to unpropitious patient outcomes. (61)

This study showed the average Post-op Urea (mg/dl)level as 23.92 (± 6.6) ranging from 16 to 41 mg/dl. The mean Post-op Urea (mg/dl) among those with AKIN stage 1 was 23.54 (± 6.26) which is lesser by 9.96 which is statistically significant compared to 33.5 (± 10.61) in those without AKIN stage 2 & 3. The average Post-op Creatinine (mg/dl) was 0.38 (± 0.19) ranging from 0 to 1.5 mg/dl. The mean Post-op Creatinine (mg/dl) among those with AKIN stage 1 was 0.35 (± 0.1) which is significantly lesser by 0.69 compared to 1.04 (± 0.65) in those without AKIN stage 2 & 3. The mean Post-operative eGFR (ml/min) was 87.12 (± 22.11) ranging from 37.5 to 132.1 ml/min. The mean Post-op eGFR (ml/min) among those with AKIN stage 1 was 87.56 (± 21.04) which is high by 11.31 but statistically insignificant compared to 76.25 (± 54.8) in those without AKIN stage 2 & 3.

Urine output:

Reduced urinary output (oliguria), a quantitative indicator of urine production, and elevated serum creatinine levels, a measure of kidney excretory function, indicate acute kidney injury (AKI), which is defined as an abrupt loss of kidney function

lasting no more than seven days. Serum creatinine and urine output level are diagnostic markers that indicate renal function decline rather than damage. People who experience transient volume depletion episodes can meet the diagnostic criteria for AKI even in the absence of damage indicators. (62–64)

In this study, the mean 24hrs Urine output (ml/kg/hr) was 3.15 (± 1.29) ranging from 0.7 to 7 ml/kg/hr. The mean 48hrs Urine output (ml/kg/hr) was 3.47 (± 1.41) ranging from 0.7 to 7.4 ml/kg/hr. The mean 72hrs Urine output (ml/kg/hr) was 3.56 (± 1.56) ranging from 0.8 to 6.6 ml/kg/hr. Urine output was significantly higher in stage 1 subjects at 24 hours, 48 hours and 72 hours compared to stage 2 & 3 subjects.

Post-op Ventilation (days):

In this study, 20 (38.46%) had 2 days, 18 (34.62%) had 1 days, 9 (17.31%) had 3 days, 3 (5.77%) had 4 days and 2 (3.85%) had 5 days of mechanical ventilation post operatively. In a study by **Simon Li et al**, Prolonged mechanical ventilation and an extended hospital stay were independently linked to the development of acute renal damage.(43) **Sidharth Kumar Sethi et al**, observed that, extended need for a ventilator was found to be independent risk factors for acute kidney injury of any severity. (46)

RA index:

Among the subjects, 27 (51.92%) had RA index score of 6, 22 (42.31%) had score of 12 and 3 (5.77%) had score of 24 out of 40. Among the subjects, 25 (48.08%) had AKI by RA index. **Rajit K Basu et al**, did the Renal angina risk assessment and found that it increased the accuracy of predicting severe AKI in critically sick

children and young adults when compared to isolated, context-free changes in Serum creatinine. (44)

In this study, 92% of the AKI by RA index had Stage 1 and 8% had stage 2 and 3. All of the subjects with RA index less than 8 had stage 1. The association between AKI by RA index and AKIN was not statistically significant ($p > 0.05$) which signifies AKI by RA index and AKIN had no difference and correlates with each other.

In a Study by **Francisco Ribeiro-Mourao et al**, RAI generated a sensitivity value of 87.5% and a specificity value of 88.1% when it came to the identification of renal injury that was present in all different stages. (with all-stage AKI having Area under curve of 0.878 for RAI). Its performance increased for severe AKI (with Area under curve of 0.93).(49)

Christina Zulu et al, observed that when a child is hospitalized to a paediatric intensive care unit with limited resources, the renal angina index reliably predicts a low risk of developing severe acute kidney injury. With sensitivity of 55.6%, specificity of 85.6%, PPV of 40.0%, NPV of 91.8%, and an AUC of 0.77, a positive Day 0 RAI predicted Day 3 sAKI. (65)

In this study, we considered the RA index as a screening test with cutoff > 8 for predicting AKI, whereas **Katja M Gist et al**, observed that With a score >8, sensitivity of 63%, specificity of 73%, and negative predictive value of 83%, the sensitivity analysis for the fulfilment of renal angina was performed. (66)

Suma Sundarraju et al, in their study, concluded that RAI is linked to the length of hospital stay and mechanical ventilation, and it can be used to predict the

development of following severe AKI on days 3 and 7. Compared to RAI ≥8, a higher RAI threshold (≥12 or ≥20) is more discriminating.(67)

In this study, the baseline parameters such as Age, gender and body surface area of the subjects had no statistically significant difference between the RA index groups. Pre-op parameters like urea, GFR and diuretics given also had no statistically significant difference between the groups and were comparable. Pre-op creatinine was slightly higher among subjects with RA index more than 8 which was statistically significant.

In this study, Intra-op characteristics like bypass time and aortic cross clamp time had no statistically significant difference between RA index groups. Similar to our study, **Hatice Işil Dayi et al**, did not observe any significant relationship between cardio-pulmonary bypass and aortic cross-clamping times with renal function and the incidence of post-operative atrial fibrillation. (68)

On the contrary, study by **Fabiano Gonçalves Jucá et al**, observed that the time difference between cardio-pulmonary bypass and cross-clamping times (TDC-C) is significantly associated with the increased incidence of complications especially kidney failure (odds ratio of 1.90, 95% confidence interval: 1.32-2.74), following CABG. In this study, Post-op urea and creatinine, eGFR and urine output at various time intervals also did not had any significant difference between the RA index groups.

CONCLUSION

Children undergoing cardiac surgery for congenital heart disease are at risk of developing acute kidney injury. Renal Angina Index and AKIN Criteria are useful in predicting Acute Kidney Injury in post op cardiac patients.

These indices help in early diagnosis of AKI and therefore help in timely management**.**

LIMITATIONS

In this study, we did not study the role of Confounding factors on the development and prognosis of the AKI. Matching, stratification, adjusted analysis occur less meaningful with the small sample size.

This was study conducted as a hospital based study in a tertiary care setting affiliated with the teaching institution, when the standards of care are comparatively high. This would affect the results positively compared to other health care settings.

In our study various other biomarkers which could add to the validity of the early prediction of the development of AKI (or the risk factor adjusted RAI) were not assessed.

In this study, we did not get adequate number to study the mortality and prognosis, owing to the smaller sample size acquired in the study period.

STRENGTHS

Given that the lead investigator was the one who largely gathered the data, it is reasonable to assume that the interobserver bias and information bias will be quite low in the study.

The study attempted to explore the usefulness of the RAI, in early identification and producing the better prognosis among the children undergoing cardiac surgeries for congenital heart diseases.

RECOMMENDATIONS

Renal Angina Index is simple to calculate with the elevated creatine level and condition of the patient. In our study, RAI cut-off of >8, is 100% sensitive and 37.5% specific. RAI would be more specific if increase the cut-off values.

Further studies can focus on incorporation of biomarkers or the risk factor into RAI, which would add up to the validity of early prediction of the development of AKI and its prognosis.

Further studies with increased sample size and more positive cases for AKI, done also in other settings such as primary and secondary care will represent the true usefulness of RAI for early prediction of the development of AKI and its prognosis.

SUMMARY OF RESULTS

The study was conducted over one year from (2023 – 2024) at KLEs Dr Prabhakar Kore Hospital .All the samples of congenital heart disease post cardiac surgery children aged 1month to 12years was collected within 24hours to 48hours from Department of CVTS ,Dr Prabhakar Kore Hospital and Medical Research centre.

Results were recorded in tabular and graphical forms Mean , Median , SD and ranges were accounted for quantitative data. The association between RA index and AKIN criteria was done using chi square test .The variables were represented by mean (± standard deviation) in tables and the significance difference between the mean is tested by student 't' test. The confidence interval was set to be at ninty five % limit , with level of significance to be at $p < 0.05$.

The study included 52 post op cardiac patients from whom blood sample was taken and various parameters was evaluated. Renal Angina Index and AKIN criteria was calculated for all patients.

- The mean Age (years) was 2.42 (± 2.6) ranging from 0.08 to 11 years.
- Among the subjects, 28 (53.85%) were Female children and 24 (46.15%) were Male children.
- Among the subjects, 20 (38.46%) had VSD, 15 (28.85%) had ASD, 6 (11.54%) had PDA, 4 (7.69%) had TOF and 3 (5.77%) had TAPVC.
- **RA index:** Among 52 patients**,** 27 (51.92%) had Renal angina index score of 6, 22 (42.31%) had score of 12 and 3 (5.77%) had score of 24 out of 40. Among the subjects, 25 (48.08%) had AKI by Renal Angina Index. Pre-op

creatinine was slightly higher among subjects with RA index more than 8 which was statistically significant.

- According to AKIN criteria -50 (96.15%) had Stage 1 ,1 (1.92%) had stage 2 and 1 (1.92%) had stage 3.
- **RA index with AKIN:** 92% of the AKI by RAI had Stage 1 injury according to akin criteria and 8% had stage 2 and 3 of akin criteria.
- Post-op urea and creatinine were significantly high among stage 2 & 3 subjects compared to stage 1 subjects. Urine output was significantly higher in stage 1 subjects at 24 hours, 48 hours and 72 hours compared to stage 2 & 3 subjects.

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ANNEXURE-I

INFORMED CONSENT FORM

I am making a voluntary decision to participate in the study ***" INCIDENCE AND RISK FACTORS OF ACUTE KIDNEY INJURY IN CONGENITAL HEART DISEASE CHILDREN POST CARDIAC SURGERY"*** My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

PATIENT INFORMATION SHEET

" INCIDENCE AND RISK FACTORS OF ACUTE KIDNEY INJURY IN CONGENITAL HEART DISEASE CHILDREN POST CARDIAC SURGERY"

Principle Investigator: **Co-Investigator**:

with congenital heart disease post cardiac surgery.

Introduction : Acute kidney injury is common after paediatric cardiac surgery occurring in 5-33% with an associated mortality of 20-79%.In children ,increasing AKI severity is independently associated with incremental increases in mortality. Current management guidelines for patients with Acute kidney injury is based on mitigation of established acute kidney injury. Proactive approach aimed at prevention of injury requires the ability to reliably identify patients at risk for acute kidney injury. A broader appreciation and recognition of acute kidney injury risk factors is a corner stone of international directives to reduce the global acute kidney injury burden.

Explanation of procedure: A detailed history with physical examination is taken children who have been diagnosed with acute kidney injury in congenital heart disease post operative will be assessed with AKIN CRITERIA and RENAL ANGINA INDEX (RAI) within 24hours to 48hours of surgery.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups.

However, your identity will never be revealed.

Questions:

If you have any question or complaints with regard to your right as study participant you may contact Dr Roopa M Bellad, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

ANNEXURE II – PROFORMA

PROFORMA **DATE:**

PATIENT NAME:

AGE:

SEX:

IP NO:

PH NO:

WEIGHT(kg): HEIGHT(cm): BSA(m^2):

ADDRESS:

SURGERY:

H/O OF RENAL DISEASE:

PRE-OPERATIVE:

1.SERUM CREATININE eGFR-

2.2D ECHO

a) SINGLE VENTRICLE
b) VENTRICULAR DYSFUNCTION

3. INOTROPIC THERAPHY YES NO

4.NEPHROTOXIC DRUG EXPOSURE YES NO

(NSAIDs,Gentamycin,Aminoglycoside)

5. MECHANICAL VENTILATION YES NO

INTRA-OPERATIVE

1.CARDIOPULMONARY BYPASS YES NO

2.BYPASS TIME

3.AORTIC CROSS CLAMP TIME DURING CPB

POST-OPERATIVE:

1. SERUM CREATININE (72HRS)-

2. INPUT (ml) OUTPUT (ml) U/O (ml/kg/hr) FO%

DAY 1

DAY 2

DAY 3

3. MECHANICAL VENTILATION (duration)

4. INOTROPE SUPPORT (duration)

5. POST OPERATIVE NSAIDS

6. POST OPERATIVE ANTIBIOTICS

7.DURATION OF ICU STAY

8.POST OP 2D ECHO

9.AKIN CRITERIA

10.RAI INDEX

ANNEXURE IV – MASTER CHART

s.no	Name	Age	Gender	Bsa	Antenatal	post natal	Diagnosis	Cardiac suregery	Pre -op						Intra -op		Post op											
									Rft	eGFR	Nephrotoxic drug	Inotropes	diuretics	Ventilation	CPB TIME	Aortic cross clamp time	Rft	24hrs u/0	48hrs u/0	72hrs u/0	ventilation	2d echo	inotropes	NSAIDS	Antibiotic	AKIN	RAI (Risk x injury)
1	pratik bevur	8years	male	0.9	not detected	detected 5months	Acyanotic CHD ,single ventricle PS	separated Bi directed Azygous ligation	urea-13	pre-184.4																	
								PA banding fonton completion procedure creat-0.35	creat-0.30	post-36.17 no	no	no	yes	no	138min	117min	urea-41	1.5ml/kg/hr	1.7ml/kg/hr	1.3ml/kg/hr	7days	post right BDGS + PAB	yes	yes	yes	stage 3	24/40
																	cret-1.53					Atrial septectomy large VSD .						
2	spandana dattatray	10months	female	0.34	not detected	not detected	large perimembranous muscular VSD ,large	sauvage pathch closure of vsd	urea-52	pre -79.2																	
							ASD (L-R shunt)		cret-0.37	post-73.3	no	no	yes	no	88min	58min	urea-	4.1ml/kg/hr	5.3ml/kg/hr	4.6ml/kg/hr	4days	post VSD patch closure	yes	yes	yes	stage 1	6/40
																	creat					VSD patch in situ					
3	Aryan jambagi	7months	male	0.3	not detected	not detected	Large perimembranous VSD ,severe PAH	sauvage patch closure	urea-17	PRE-88.1	no	no	yes	no	78min	56min	urea -20	3.8ml/kg/hr	3.4ml/kg/hr	2.7ml/kg/hr	5days	vsd patch is seen in situ	yes	yes	yes	stage 1	6/40
									cret-0.30	Post-70.5							cret-0.38					normal ventricular function					
4	srushti suresh	11years	female	1.1	Not	CHD detected	Large secundum ASD (L-R shunt)	pericardial patch closure of ASD	urea-24	pre-126.5	no	no	yes	no	50min	20min	urea-30	1.5ml/kg/hr	2.1ml/kg/hr	1.6ml/kg/hr	1day	post ASD patch closure	yes	yes	yes	stage 1	12/40
					detected		moderate PAH ,sinus rhythm		cret-0.46	post-105.8							cret-0.55					ASD patch insitu					
5	pradhnya	1.5years	female	0.4	not	not detected	secundum ASD (L-Rshunt)	closure of ASD	urea-35	pre-54.4												ASD patch seen					
					detected		moderate PAH		cret-0.66	post-70.4	no	no	no	no	36min	21min	urea-38	3.6ml/kg/hr	0.7ml/kg/hr	6.1ml/kg/hr	1day	normal LV function	yes	Yes	yes	stage 1	6/40
																	cret-0.51										
6	Aryan chandaragi	2years	male	0.28	not detected	CHD detected	TOF ,pulmonary atresia	TOF with pulmonary atresia	urea-24	pre-40	no	no	no	no	47min	20min	urea-26	1.9ml/kg/hr	2.0ml/kg/hr	1.3ml/kg/hr	1day	TOF with PA					
						nicu admitted	constricting PDA	large perimembranous VSD (L-R)	cret-0.68	post-46.9							cret-0.58					large perimembraneous VSD	yes	yes	yes	stage 1	6/40
7	naveed	2years	male	0.47	fetal cardiac	not detected	perimembraneous VSD (L-Rshunt)	sauvage patch closure	urea-21	pre-93.4	no	no	no	no	88min	55min	urea-28	2.0ml/kg/hr	4.4ml/kg/hr	2.8ml/kg/hr	1day	VSD patch in situ					
					anomaly		sinus rhythm		cret-0.38	post-88.7							cret-0.40					normal ventricular function					
8	shourya yallappa	2years	male	0.38	not detected	not detected	Lar (L-R shunt)ge OS-ASD	pericardial patch closure	urea-14	pre-54.0	no	no	no	no	46min	20min	urea-21	3.1ml/kg/hr	4.2ml/kg/hr	4.1ml/kg/hr	1day	ASD patch insitu	yes	yes	yes	stage 1	6/40
							mild PAH	of ASD	cret-0.55	post-59.4							cret-0.50					minimal pericardial effusion					
9	Husman sandramani	11months	male	0.36	not detected	detected 4months	perimembraneous VSD (L-R shunt)	sauvage patch closure	urea-10	pre-110.1	no	no	yes	no	77min	55min	urea-16	3.1ml/kg/hr	3.3ml/kg/hr	0.8ml/kg/hr	4days	post ASD patch closure	yes	yes	yes	stage 1	6/40

									cret-0.27	post-95.9							cret-0.31					RVOT resection trans anular					
																						patch & monocusp valve					
10	kushi arunkumar	4years	female	0.46	not detected	detected 5months	DORV ,VSD,PS ,non routable VSD	Bi-directional glenn shunt	urea-18	pre-129	no	no	no	no	47min	20min	urea-20	5.1ml/kg/hr	5.6ml/kg/hr	2.7ml/kg/hr	3days	BD glenn shunt + MPA ligation	yes	yes	yes	stage 1	12/40
									cret-0.29	post-121.2							cret-0.31					normal functioning glenn shunt					
11	Tanu kumara	3years	female	0.23	not detected	detected 7months	perimembraneous VSDwith inlet extension	sauvage patch closure	urea-23	pre-115.6	no	no	yes	no	86min	59min	urea-16	2.1ml/kg/hr	1.9ml/kg/hr	6.5ml/kg/hr	1day	VSD patch insitu mild TR	yes	yes	yes	stage 1	6/40
							severe PAH ,sinus rhythm		cret-0.20	post-96.3							cret-0.24										
							TGA intact ivs ,large VSD ,regressed LV	stennings operation	urea-39	pre-74.1	no	no	yes	no	207min	165min	urea-39	2.8ml/kg/hr	1.6ml/kg/hr	2.1ml/kg/hr	2days	post rerouting of TAPVC	yes	yes	yes	stage 1	6/40
12	Arhan javid	9months	male	0.32	not detected	detected 4months			creat-0.39	post-70.5							cret-0.41					laminae flow noted in					
																						pulmonary venous					
13	samruddi mahesh	4years	female	0.61	not detected	not detected	sinus venous /ASD (L-R shunt)	pericardial patch closure	urea-15	pre-118	no	no	no	no	64min	44min	urea-17	1.2ml/kg/hr	1.3ml/kg/hr	1.4ml/kg/hr	3days	ASD patch insitu	yes	yes	yes	stage 1	6/40
								of ASD	cret-0.37	post-115.2							cret-0,38										
14	nagaraj	7years	male	0.76	not detected	not detected	moderate sized VSD (L-R shunt)	Trans PA sauvage	urea-23	pre-133	no	no	no	no	86min	60min	urea-24	2.3ml/kg/hr	3.2ml/kg/hr	3.5ml/kg/hr	1day	VSD patch insitu	yes	yes	yes	stage 1	6/40
							mild PAH ,CHF	patch closure of VSD	cret-0.36	post-126							cret-0.38										
15	sanvi shrinath	1.6years	female	0.4	not detected	not detected	PDA	PDA /double ligation	urea-23	pre-125.6	no	no	no	no	68min	54min	urea-20	4.5ml/kg/hr	4.3ml/kg/hr	4.6ml/kg/hr	3days	PDA ligation ,no	yes	yes	yes	stage 1	12/40
								of PDA	cret-0.23	post-85							cret-0.34					residual PDA					
16	Aryan teninalli	1.5years	male	0.4	nil	detedted day 1 of	VSD	VSD patch closure	urea-38	pre-134.2	no	no	no	no	47min	20min	urea-26	1.9ml/kg/hr	2ml/kg/hr	1.3ml/kg/hr	2days	VSD patch insitu	yes	yes	yes	stage 2	24/60
						life			cret-0.24	post-115.0							cret -0.46										
17	Trinayanni	9months	female	0.29	nil	detected 3months	large perimembranous VSD	sauvage patch closre VSD	urea-14	pre-140.4	no	no	yes	no	90min	69min	urea-18	4.7ml/kg/hr	4.6ml/kg/hr	5.5ml/kg/hr	1day	VSD patch insitu	yes	yes	yes	stage 1	6/40
									creat-0.20	post-104.0							cret-0.27										
18	Kiran mayappa	9months	male	0.4	nil	detected CHD	secundum ASD (L-R)	pericardial patch closure of ASD	urea-19	pre-109.1		no	yes	no	46min	23min	urea-20	2.2ml/kg/hr	2.4ml/kg/hr	2.5ml/kg/hr	1day	ASD patch insitu	yes	yes	yes	stage 1	6/40
						6months age	moderate PAH /CHF		cret-0.28	post-101.8							cret-0.30										
																	urea-18	3.3ml/kg/hr	3.3ml/kg/hr	4.5ml/kg/hr	2days	VSD patch insitu	yes	yes	yes	stage 1	12/40
19	Bhakti jinnappa	1.3years	female	0.37	nil	not detected	Large perimemnraneous VSD (L-R) shunt	sauvage patch closure of VSD	urea-13	pre-150		no	no	no	95min	70min	cret-0.27										

							moderate PAH		cret-0.22	post-122.3																	
20	Juned kulagod	2years	male	0.37	nil	not detected	ASD /PAPVC	pericardial patch closure of ASD	urea-14	pre-107		no	yes	no	86min	54min	urea-18	3.8ml/kg/hr	5.9ml/kg/hr	6.5ml/kg/hr	2days	post ASD patch closure	yes	yes	yes	stage 1	12/40
									cret-0.27	post-96							creat-0.30					ASD patch insitu					
21	swaranjali	11years	female	0.72	nil	not detected	TOF	Transunlar pericardial patch closure	urea-21	pre-139.2		no	yes	no	75min	60min	urea-38	2.9ml/kg/hr	3.4ml/kg/hr	3.9ml/kg/hr	1day	Normal ventricular function	yes			stage 1	24/40
									cret-0.23	post-108.2							cret-0.45					No PAH					
							secundum ASD (L-R)	pericardial patch closure of ASD	urea-17	pre-188.8		no	yes	no	81min	57min	urea-18	4.3ml/kg/hr	7.4ml/kg/hr	5.1ml/kg/hr	2days	VSD patch insitu	yes	yes	yes	stage 1	12/40
22	Anaya tashildar	6months	female	0.3	nil	not detected	moderate PAH/chf		cret-0.14	post-132.1							creat-0.20					large muscular VSD					
23	mahesh goud	1year	male	0.36	nil	CHD deted 3months	secundum ASD,moderate PAH	pericardial patch closure of ASD	urea-19	pre-103.2		no	yes	no	56min	37min	urea-20	2.7ml/kg/hr	3.1ml/kg/hr	3.3ml/kg/hr	2days	ASD patch insitu	yes	yes	yes	stage 1	6/40
						of age.			cret-0.28	post-96.3							cret-0.30										
24	Siddi banasode	7months	female	0.24	nil	not detected	secundum ASD (L-R)	ASD-pericardial patch closure	urea-39	pre-75.3		no	no	no	55min	30min	urea-20	1.4ml/kg/hr	2.7ml/kg/hr	3.7ml/kg/hr	1day	ASD patch Insitu	yes	yes	yes	stage 1	12/40
							moderate PAH	PDA ligation	cret-0.34	post-64.0							cret-0.40					no residual PDA					
25	Dhanya vaghesh	5months	female	0.24	nil	CHD 1month age	large perimembraneous VSD	sauvage patch closure	urea-26	pre-104.9		no	yes	no	70min	43min	urea-28	5ml/kg/hr	3.2ml/kg/hr	1.8ml/kg/hr	1day	VSD patch insitu	yes	yes	yes	stage 1	12/40
						detected		PDA ligation	cret-0.24	post-93.0							cret-0.27					no residual PDA					
26	nithya yuvraj	7months	female	0.25	nil	CHD detected	Large perimembraneous VSD	sauvage patch closure of VSD	urea-17	pre-95.8		no	yes	no	71min	54min	urea-20	7.0ml/kg/hr	5.8ml/kg/hr	6.4ml/kg/hr	2days	VSD patch in situ	yes	yes	yes	stage 1	12/40
						3months age		PDA ligation	cret-0.28	post-78.9							cret-0.34					small residual flow					
																						moderate MR					
27	shreenesh shrishail	3years	male	0.45	nil	not detected	Large ASD	pericardial patch closure of ASD	urea-20	pre-93.9		no	yes	no	70min	59min	urea-20	2.9ml/kg/hr	2.8ml/kg/hr	3.9ml/kg/hr	3day	ASD patch in situ	yes	yes	yes	stage 1	12/40
									cret-0.29	post-75							creat-0.36										
28	padmashree	6months	female	0.3	nil	not detected	TAPVC	intra cardiac TAPVC	urea-20	pre-58.6		no	no	no	133min	79min	urea-22	5.1ml/kg/hr	4.0ml/kg/hr	6.1ml/kh/hr	2days	post intra cardiac TAPVC repair	yes	yes	yes	stage 1	6/40
								Rerouting of TAPVC	cret-0.38	post-56.7							cret-0.40					No MR ,trivial TR.					
								Right lateral schumarker B/L technique														no effusion					
29	Labhansh parab	5months	male	0.38	nil	not detected	large perimembraneous VSD	Direct closure of VSD	urea-17	pre-55.06		no	yes	no	150min	105min	urea-20	4.1ml/kg/hr	5.4ml/kg/hr	6.1ml/kg/hr	1day	VSD patch in situ	yes	yes	yes	stage 1	6/40
							severe PAH/CHF		cret-0.60	post-67.4							cret-0.19					no residual flow					
																						normal ventricular function					
30	Trisha	3years	female	0.48	nil	not detected	secundum ASD (L-R) PDA	ASD /pericardial patch closure pf ASD	urea-25	pre-118		no	no	no	51min	20min	urea-30	3.1ml/kg/hr	3.5ml/kg/hr	3.7ml/kg/hr	1day	ASD insitu with PDA	yes	yes	yes	stage 1	12/40

							mild PAH		cret-0.28	post-91.7							cret-0.36					Ligation					
31	mayur patil	2.5years	male	0.42	nil	not detected	ASD with mild PS	pericordial patch closure of ASD	urea-20	pre-102.6		no	no	no	58min	28min	urea-24	0.7ml/kg/hr	3.2ml/kg/hr	3.4ml/kg/hr	2days	ASD patch insitu	yes	yes	yes	stage 1	6/40
									cret-0.37	post-94.9							cret-0.40										
32	nishanth	2months	male	0.24	nil	not detected	VSD	sauvage patch closure of VSD	Urea-28	pre-57		no	no	no	120min	72min	urea-30	4.8ml/kg/hr	3.5ml/kg/hr	4.3ml/kg/hr	2days	post VSD patch closure	yes	yes	yes	stage 1	6/40
									cret-0.42	post-58.4							cret-0.41					VSD patch insitu					
33	manasa manju	1.6years	female	0.36	nil	not detected	secundum ASD (L-R)	pericardial patch closure	urea-27	pre-92.6		no	no	no	41min	18min	urea-28	3.9ml/kg/hr	4.4ml/kg/hr	3.5ml/kg/hr	1day	post ASD closure	yes	yes	yes	stage 1	6/40
									cret-0.33	post-84.8							cret-0.36					ASD patch insitu					
34	vaishnavi	4years	female	0.52	nil	not detected	moderate size perimembraneous VSD	Direct closure of VSD	Urea-25	pre-101		no	no	no	86min	48min	urea-20.3	2.2ml/kg/hr	3.3ml/kg/hr	3.7ml/kg/hr	2days	post ASD closure	yes	yes	yes	stage 1	6/40
							with extreme fibrous tissue along margin		cret-0.38	post-96.0							cret-0.40					ASD patch insitu					
35	priya chougala	1year	female	0.31	nil	detected 5months	PDA	triple ligation of PDA	urea-16	pre-90.3		no	no	no	75min	36min	urea-20	2.6ml/kg/hr	3ml/kg/hr	3ml/kg/hr	1day	No residual PDA	yes	yes	yes	stage 1	12/40
						age			cret-0.32	post-74.5							cret-0.41										
36	nishanth	2months	male	0.24	nil	detected at birth	VSD	sauvage patch closure	urea-28.1	pre-57		no	no	no	120 min	72 min	urea-30	4.8ml/kg/hr	3.5ml/kg/hr	4.3ml/kg/hr	2days	post VSD patch closure	yes	yes	yes	stage 1	6/40
								of VSD	creat-0.42	post-58.4							cret-0.41					VSD patch insitu					
37	Rachit Janagouda	3years	male	0.57		detected 5months ageVSD	vsd	Direct closure of VSD	urea-25	Pre-106.5		no	no	no	92min	68min	urea-27	2.6ml/kg/hr	3.1ml/kg/hr	2.7ml/kg/hr	2days	VSD patch insitu	yes	yes	yes	stage 1	6/40
									crt-0.38	post-101.1							cret-0.40					post VSD patch status					
38	savita channadasar	2years	female	0.38		detected 8months	moderate sized perimembranous VSD	sauvage patch closure of VSD	urea-11.0	pre-99.12		no	no	no	88min	70min	urea-16	2.1ml/kg/hr	2.0ml/kg/hr	2.2ml/kg/hr	3days	VSD patch insitu	yes	yes	yes	stage 1	12/40
						age	with L-R shunt mild PAH		crt-0.30	post-87.4							cret-0.34										
39	uday	2months	male	0.35		not detected	PDA	double ligation of PDA	urea-28.1	pre-58.6		no	no	no	46min	23min	urea-28	2.4ml/kg/hr	2.5ml/kg/hr	2.4ml/kg/hr	2days	post PDA ligation	yes	yes	yes	stage 1	6/40
									cret-0.38	post-55.7							cret-0.40					no residual PDA					
																						dilated LA/LV					
																	urea-27	2.9ml/kg/hr	2.8ml/kg/hr	3.3ml/kg/hr	2days	post ASD patch status	yes	yes	yes	stage 1	6/40
40	bibi sakina	5years	female	0.58		detected 9months	large secundum /ASD (L-R)	pericardial patch closure	urea-23.0	pre-99.9		no	no	no	46min	34min	cret-0.40					ASD patch in situ					
						age	shunt mod PAH sinus rhythm	of ASD	cret-0.38	post-94.9																	

41	Srujan dundappa	3years	male	0.58		detected 12month	TOF	PBT shunt /ICR/Trananular	urea-18	pre-106		no	no	no	128min	102min	urea-21	2.4ml/kg/hr	3.6ml/kg/hr	2.6ml/kg/hr	3days	Transanular pericardial	yes	yes	yes	stage 1	12/40
								pericardial patch with	creat-0.36	post-96							cret-0.40					patch in situ					
								monocusp valve																				
42	Shravya adiveppa	1.5years	female	0.33		detected 5day	VSD	Sauvage patch closure of VSD	urea-20	pre-125.6		no	no	no	58min	33min	urea-20	4.0ml/kg/hr	4.1ml/kg/hr	4.3ml/kg/hr	1day	post VSD patch closure	yes	yes	yes	stage 1	12/40
						of life			cret-0.23	post-107.0							cret-0.27					VSD patch in situ					
43	Sidik Daval korabu	1year	male	0.34		detected 4months	Secundum ASD (L-R)	pericardial patch closure	urea-18	pre-108.0		no	no	no	56min	34min	urea-20-	2.9ml/kg/hr	3.3ml/kg/hr	3.5ml/kg/hr	2days	post ASD patch closure	yes	yes	yes	stage 1	12/40
						age	moderate PAH.		cret-0.26	post-87.7							cret-0.32					ASD patch insitu					
44	Apporva	1month + 5day	female	0.22		not detected	TAPVC with severe PAH	Re routing of TAPVC by shumaerous	urea-24	pre-56.9		no	no	no	88min	46min	urea-26	4ml/kg/hr	3.6ml/kg/hr	3.4ml/kg/hr	2days	post infracardia TAPVC repair	yes	yes	yes	stage 1	6/40
								right lateral approach	cret-0.37	post-55.4							cret-0.38					moderate PAH					
																							no effusion						
45	Swetha p naik	2months	female	0.27		not detected	PDA	Double ligation of PDA	Urea-25	pre-59.7		no	no	no	46min	23min	urea-36.5	2.7ml/kg/hr	5.5ml/kg/hr	3.3ml/kg/hr	5day	Post PDA ligation	yes	yes	yes	stage 1	12/40
									cret-0.38	post-47.4							cret-0.50					no residual PDA					
46	KUM Alina vannur	5years	female	0.62		4months detected	TOF + pulmonary	ICR with short valve sparing	urea-11.2	pre-125		no	no	no	132min	103min	urea-21	2.2ml/kg/hr	1.06ml/kg/hr	1.1ml/kg/hr	4days	no residual flow	yes	yes	yes	stage 1	12/40
							arteries PDA	pericardial patch	cret-0.31	post-107.8							cret-0.36					normal ventricular function					
47	Kalmesh basavaraj	1month +5day	male	0.24		not detected	supracardial TAPVC	Re routing of TAPVC by	urea-17	pre-95.3		no	no	no	113min	72min	urea-21	6.1ml/kg/hr	6.4ml/kg/hr	6.6ml/kg/hr	3day	post infracardiac TAPVC	yes	yes	yes	stage 1	12/40
								schumakier bi atrial incision	cret-0.28	post-81.3							cret-0.33					no effusion					
								technique																				
48	Keertana kuragund	7months	Female	0.28		detected 3months	VSD	VSD closure of savage patch	urea-18	pre-118.5		no	no	no	86min	55min	urea-18	3.7ml/kg/hr	5.1ml/kg/hr	5.3ml/kg/hr	3days	VSD patch intact	yes	yes	yes	stage 1	6/40
						age		very large perimembranous VSD	cret-0.23	post-113.5							cret-0.24					no significant residual VSD					
																							Mild TR						
49	B/O mamata pranati	6months	female	0.29		detected 1month	PDA	PDA ligation done	urea-37.4	pre-104.8		no	no	no	84min	54min	urea-37	3.1ml/kg/hr	3.7ml/kg/hr	4.1ml/kg/hr	2days	post PDA ligation	yes	YES	yes	stage 1	6/40
									cret-0.26	post-97.35							cret-0.28					no residual PDA					
50	Shivraj Bhima Bilage	8months	male	0.23		detected 2months	Perimembranous VSD (L-R) SHUNT	PTFE patch closure of VSD.	urea-23	pre-115.6		no	no	no	86min	59min	urea-16	1.8ml/kg/hr	1.9ml/kg/hr	3.5ml/kg/hr	3days	post VSD patch closure	yes	yes	yes	stage 1	12/40
							Severe PAH ,CHF sinus rhythm		creat-0.20	post-95.3							creat-0.24					VSD patch in situ					
																							no residual shunt						
51	Srujan Bante	4years	male	0.58		detected 1year	TOF	P.BT shunt/ICR with	urea-18	pre-106.0		no	no	no	128min	102min	urea-21	2.4ml/kg/hr	3.6ml/kg/hr	1.5ml/kg/hr	2days	TOF with perimembraneous VSDno	yes	yes	yes	stage 1	12/40
								transannular pericardial	creat-0.36	post-96							cret-0.40										
								patch with monocusp valve																				

52	B/O nayana	1years	female	0.31		detected 5months	PDA (L-R) shunt	Double ligation of PDA	urea-16	pre-90.3		no	no	no	75min	36min	urea-20	2.3ml/kg/hr	3ml/kg/hr	2.8ml/kg/hr	3days	post PDA ligation in situ	yes	yes	yes	stage 1	12/40
								Double clipping	cret-0.32	post-74.5							cret-0.41										