OUTCOME OF GRAFT FUNCTION WITH REFERENCE TO COLD ISCHAEMIA TIME IN CADAVER RENAL TRANSPLANT

Dissertation submitted to

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BRANCH-IV

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Declaration

I solemnly declare that this dissertation "**Outcome of graft function with reference to cold ischaemia time in cadaver renal transplant**" was prepared by me in the Department of Urology, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of **Prof.R.RADHAKRISHNAN** MCh (Uro), Professor &HOD and **Prof.P.GOVINDARAJAN** MCh (Uro), Professor, Dept. of Urology, Government Stanley Medical College, Chennai between 2008 and 2011.

This dissertation is submitted to the TamilNadu Dr. MGR Medical University, Chennai in partial fulfillment of the University requirements for the award of degree of MCh Genitourinary surgery

Place : Chennai

Date :

CERTIFICATE

This is to certify that this dissertation entitled "**Outcome of graft function with reference to cold ischaemia time in cadaver renal transplant**" is a bonafide record of the research work done by Dr.M.Seral Kannan for the award of MCh Genitourinary surgery under the guidance and supervision of **Prof.R.RADHAKRISHNAN** MCh (Uro), Professor &HOD and **Prof.P.GOVINDARAJAN** MCh (Uro), Professor, Dept. of Urology, Government Stanley Medical College, Chennai between 2008 and 2011. I also certify that this dissertation is the result of the independent work done by candidate.

Dr.R.Radhakrishnan MCh Uro
Professor and Head
Department of Urology
Govt Stanley Medical College
Chennai – 600 001.

Dr.P.Govindarajan MCh Uro
Professor,
Department of Urology
Govt Stanley Medical College
Chennai – 600 001.

Dean
Govt Stanley Medical College
and Hospital
Chennai – 600 001.

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Introduction **1 -3**

Magnitude of the problem

The Solution-Treatment strategies

INTRODUCTION

Magnitude of Burden

The population of India exceeds one billion and is projected to become the major reservoir of chronic diseases like diabetes and hypertension. With 25–40% of these subjects likely to develop ESRD the burden will rise. However, in the absence of any registry, data on incidence of ESRD in India do not exist.

A figure of 100 per million population (pmp) per year is often cited, based on estimates from rest of the world, tertiary care center data, and collective experience of experienced Nephrologists[39,40,41] Further, it has been estimated that less than 10% of all Indian ESRD patients receive any meaningful renal replacement therapy (RRT).

The Solution- Treatment Strategies

Treatment option for End stage renal Disease-Stage5 (ESRD-stage5) patients are Haemodialysis, Peritoneal dialysis and Renal Transplantation.

Many studies proved that the kidney transplantation is distinctly superior and it is associated with reduced mortality and morbidity compared to haemodialysis or peritoneal dialysis[3 – 16].

- Renal donors for transplant are of three types:

- live-related
- live-unrelated and
- cadaver

- Most ESRD don't have a suitable live-related donor for transplantation because of nuclear families, working members in the family and the increased prevalence of diabetes mellitus and hypertension among general population. The only option for them will be cadaver donors

- The outcome of cadaver transplant is inferior to live-related and even live-unrelated transplants

- However 70% reduction in the overall mortality rate than when maintaining them on hemodialysis

Most of the data for assessing transplant outcome is from United States Renal Database System (USRDS)[1], Collaborative Transplant Study (CTS)[2] and Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Aim of the Study *4*

- To study the **Outcome Of Graft Function With Reference To Cold Ischaemia Time In Cadaver Renal Transplant** Prospectively

REVIEW OF THE LITERATURE 5 - 20

ESRD

Factors affecting Graft Function

Cold Ischaemia time and its Impact

Stages of ESRD[17]

Stage	Description	GFR (ml/min/1.73m^2)
1	Kidney damage with normal or GFR	>90
2	Mild reduction in GFR	60-89
3	Moderate reduction in GFR	30-59
4	Severe reduction in GFR	15-29
5	Kidney failure	<15 or dialysis

Factors Affecting Renal Allograft Survival and Function

Prospective studies and analyses of registry data have shown that many factors are associated with renal allograft survival and function. These can be considered as

(1) Donor

(2) Recipient, or

(3) donor-recipient

Donor-Recipient Factors

Delayed Graft Function

Defined as failure of the renal allograft to function immediately post-transplantation, with the need for one or more dialysis sessions within a specified period, usually one week.

DGF is associated with poorer graft survival, poorer graft function, and higher risk of patient death[10], in part because of the association of DGF with higher rates of acute rejection. Rejection may be more common because ischemia-reperfusion injury increases the immunogenicity of the graft.

Most studies have also demonstrated that, even in the absence of documented acute rejection, DGF is associated with poorer long term graft function and survival[11].

Risk factors for DGF are:

- Donor age (>40 years)
- Cold ischemia time (>12 hrs)
- Recipient race
- PRA (>50%)
- HLA mismatch
- Duration of dialysis

HLA Matching

Better HLA matched deceased donor allografts have better survival[19] and function. So, many countries operate national or international sharing systems for zero-mismatched renal allografts, even though this prolongs cold ischemia time. The better outcomes are presumably related to fewer immunologic failures. However the benefits of HLA matching are diminishing, probably because of more effective immunosuppression[19].

Center Effect

Outcomes have varied widely among transplantation centers. This reflects normal statistical variance as well as center expertise. Thus, between centers comparisons are difficult.

Donor Factors

The quality of the kidney immediately prior to transplantation has a major impact on long term graft function and the risk of developing chronic allograft nephropathy.

Donor Source: Deceased versus Living Donor

Most important predictors of short and long term graft outcomes. In general, living donor grafts are superior to deceased donor grafts. The better healthy living donors, the absence of brain death, the general benefits of elective

as opposed to semi emergency surgery, avoidance of ischemia-reperfusion injury, high nephron mass and probably the effects of a shorter waiting time. Better compliance by the recipient in view of the relationship e.g., spouse, a care giver may also play a role.

Donor Age

Deceased donor and living donor allografts from those aged older than 50 years, and particularly older than 65 years, have poorer outcomes[1]. Grafts from older donors have fewer functioning nephrons because of the aging process and donor-related conditions such as hypertension and atherosclerosis.

Donor Sex

Grafts from deceased females' donors have slightly poorer survival, particularly in male recipients[1,21]. This probably reflects "nephron under dosing", as females have smaller renal mass than males. However, differences in the antigenicity of female grafts may also be a factor[20].

Donor Nephron Mass

An imbalance between the metabolic/excretory demands of the recipient and the functional transplant mass has been postulated to play a role in the development of chronic allograft nephropathy. "Nephron under dosing",

exacerbated by perioperative ischemic damage and postoperative nephrotoxic drugs, might lead to nephron overwork and eventual failure.

Cytomegalovirus Status of Donor and Recipient

Small but definite effect of donor and recipient cytomegalovirus (CMV) serologic status on renal allograft and recipient survival[1]. Donor negative-recipient negative-pairing have the best outcomes, whereas donor positive-recipient negative pairing have the worst. CMV probably affects graft outcomes via overt infection, but subclinical effects on immune function may also be important.

Recipient Factors

Recipient Age

In general, graft survival and function rates are poorer in those at the extremes of age: younger than 17 and older than 65 years[1]. In the young, technical causes of graft loss such as vessel thrombosis are relatively more common. Acute rejection is also common.

The elderly (those older than 65 years) have significant comorbid disease, particularly cardiovascular disease and type 2 diabetes mellitus. Nevertheless, age per se is not a contraindication to transplantation: among elderly patients carefully screened and deemed fit for the procedure, long term outcomes are

clearly better with transplantation than dialysis[3]. Conversely, acute rejection may be less common.

Recipient Gender

Association of recipient gender with transplantation outcomes have yielded differing results. In the CTS database[2], female recipients had slightly better allograft survival than male recipients of deceased donor kidneys or HLA identical kidney[21]. Females tend to be more sensitized because of pregnancy and possible because of more blood transfusions related to menstruation.

Recipient Sensitization: before or after Transplantation

Patients who are broadly sensitized (e.g., panel reactive antibody [PRA] status >50%) at the time of transplantation generally have poorer early and late graft survival compared to nonsensitized recipients. This is mainly related to an increased incidence of complications in the early post-transplantation period such as DGF and acute rejection. The principal reasons for sensitization are previous transplants, pregnancy, and previous blood transfusions. Highly sensitized patients are often given more intensive immunosuppression to reduce the risk of rejection, but this also exposes them to risk of infection and malignancy.

Presence of donor specific and nondonor specific HLA antibodies are associated with inferior graft survival[22].

Recipient HCV Antibody and HBsAg

Recipients who are hepatitis C virus (HCV) antibody positive at the time of transplantation have poorer allograft survival and poorer survival[1,24]. Higher mortality rates appear to be related to infection and worsening liver disease[24]. Nevertheless, transplantation of selected HCV positive patients confers a survival benefit as opposed to remaining on the dialysis[27].

The adverse effects of hepatitis B virus (HBV) surface antigen positivity on post-transplantation outcomes are much less pronounced.

Acute Rejection

Acute rejection has been consistently associated with an increased risk of graft loss. This is due to irreversible graft injury at the time of acute rejection and probably ongoing subclinical immunemediated injury. Acute rejection refractory to steroids, acute rejection where creatinine does not return near baseline, and late acute rejection (occurring after the first 6 months) are particularly associated with poorer graft and patient outcomes[17].

Recipient Immunosuppression

Tacrolimus was more effective than cyclosporine in preventing acute rejection and allograft loss but at the expense of higher rates of diabetes mellitus[29].

There is limited evidence that mycophenolate mofetil improves long-term graft survival both by preventing overt acute rejection and possible by other mechanisms. Significant level of cyclosporine and tacrolimus produces 30% increase in bioavailability of mycophenalate mofetil. Short term studies of sirolimus have shown contradictory results[30,31]

Although antilymphocyte antibody preparations (e.g., antithymocyte globulin or interleukin-2 receptor blockers) are often used, particularly in the setting of DGF, their effects on long term graft survival and function have not been well studied.

Recipient Compliance

Poor compliance with the immunosuppressive regimen is known to increase the risk of acute rejection, particularly late acute rejection, and chronic allograft dysfunction[33]

Obesity

Obesity is associated with more DGF, higher mortality (related to cardiovascular complications), and poorer graft survival[34]. The poorer long term graft survival probably reflects the effects of DGF, nephron overwork, and more difficult dosing of immunosuppressive drugs. Transplantation in patients with BMI >30 kg/m^2 provides a survival benefit over remaining on the waiting list (on dialysis) at least up to a BMI of 41 kg/m^2.

Recipient Hypertension: Angiotensin system

Greater the severity of post-transplantation hypertension is, the higher is the risk of graft loss[35]. Hypertension could also be secondary to graft damage. No prospective human studies of the effect of treating hypertension on allograft outcomes are available. However, control of hypertension is associated with improved allograft survival and function [36].

Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) slow the progression of both diabetic and nondiabetic proteinuric native kidney disease.

Recipient Dyslipidemia

Studies have suggested that hypercholesterolemia and/or hypertriglyceridemia are associated with poorer graft outcomes.

Recurrence of Primary Disease

Determining the incidence and prevalence of recurrent or de novo renal disease is difficult. The cumulative incidence of graft loss at 10 years was 8.4%[37]in glomerulonephritis. Recurrence is the most important cause of loss, after chronic rejection and death

Proteinuria

The degree of proteinuria correlates with poorer renal outcome in both native and transplant kidney disease. ACE inhibitors and ARBs has definite role in showing the progression of proteinuria in transplant renal disease.

Cold Ischaemia and Its Impact

HISTORY OF COLD PRESERVATION

The Early Days

The first recorded attempt at perfusion of an isolated organ occurred in 1849 by Loebel. In 1895, Langendorf devised a simple organ-perfusion technique.

In 1937, Lindbergh and Carrel, fascinated with hypothermic preservation, created a perfusion apparatus. In 1953, Lapchinsky from the Soviet Union started successfully transplanting limbs and kidneys preserved at 4°C.

In 1964, Belzer, while working with Najarian to develop a cadaver kidney transplant program at the University of California (San Francisco, CA), started to work on hypothermic perfusion techniques for the preservation of kidneys

Collin's Solution

However, it was Collins et al. who first developed a simple yet effective cold storage solution in 1969. The solution contained a high concentration of glucose and electrolytes, mostly of intracellular composition. The seminal observation of Collins et al. that storage at 4°C after a simple perfusion can extend the viability of cadaver kidneys changed the practice of transplantation from an emergency procedure to a semielective one.

University of Wisconsin Solution

Based on a series of attempts, Belzer and Southard in the early 1980s developed University of Wisconsin (UW) solution. A number of large-molecular-weight cell impermeants were added to reduce tissue swelling. Glucose was replaced with phosphate buffers. On theoretical grounds, adenosine for rapid ATP repletion and glutathione (GSH) and allopurinol for antioxidant property during the reperfusion phase were added

Cold Ischemia Time -Definition

Cold ischaemia time is the time between cold perfusion of cadaver kidney in the harvesting centre till clamp release after anastomosis in Cadaver renal transplants.

Cold Perfusion – Pros and Cons

Organs for transplantation require effective *ex vivo* preservation from the moment the organ is retrieved to the time of transplantation. Hypothermic preservation solutions have been developed to maintain tissue viability by reducing metabolic activity and the accumulation of toxic substances during the cold ischemic period. In clinical renal transplantation, prolonged cold storage has been demonstrated in many studies to be strongly associated with delayed graft function (DGF)[42-47]

Registry data suggest that >24 hours is particularly deleterious to the graft[1]. DGF results in complications in the immunosuppressive management of the transplant patient, prolonged hospitalization, and potentially detrimental effects to subsequent graft function and survival.

Pathological Changes during Ischaemia

There are at least four components to cold ischemic transplant injury:

(1) the coupled effect of ischemia and hypothermia during cold storage

(2) the coupled effect of reperfusion and rewarming after transplantation.

The effects of ischemia and reperfusion are widely studied, but the contribution of hypothermia and rewarming to them is difficult to separate and rarely studied

Effects of Ischaemia- cellular level

Ischemia favors the depletion of cellular adenosine nucleotides, alterations in membrane ATP-dependent ionic transporters, and the intracellular accumulation of Ca^{2+}, Na^{+}, and water.

The great swelling of endothelial and tubular epithelial cells due to ischemia not only increases the acidosis caused by anaerobic oxidation, but also alters cell permeability and favors the obstruction of capillary flow.

Outer medullary vascular congestion is a prominent feature of ischemic acute renal failure and transplanted kidneys damaged during cold preservation of the grafts[48] .

The outflow of blood from the medulla during reperfusion is blocked[49], limiting oxygenation of the tubule epithelial cells located in this region[50].

Reperfusion

The process and mechanism of reperfusion injury of cold ischemic organs are likely similar to the reperfusion injury of warm ischemic organs, although no

direct comparison studies are available. The reperfusion of ischemic tissues also increases the release of intracellular enzymes, the influx of intracellular Ca^{2+}. Free radicals and other reactive oxygen species that trigger T cell activation are produced after cold ischemia and rewarming during reperfusion.

The increase in intracellular Ca^{2+} activates membrane phospholipase A_2. The oxygen supplied by blood reperfusion generates free oxygen radicals, which react with lipid cellular membranes. The peroxidation of cell membrane lipids can disrupt the balance of vasoactive eicosanoid metabolism, leading to vasoconstriction due to excess thromboxane synthesis, and a decrease in the production of prostacyclin and prostaglandin I_2.

Endothelial Dysfunction , free radicals and cytokines

The release of proteases, inflammatory cytokines, chemoattractants, and growth factors such as fibrogenic growth factor TGF-ß is also associated with upregulation of adhesion molecules and activation of leukocytes, macrophages, and monocytes in postischemic reperfused kidneys

Endothelial cell dysfunction and activation of leukocytes contribute to the inflammatory process with the coordinated release of several cytokines and chemokines. Cold ischemic injury increases allograft immunogenicity, provoking acute and chronic rejections. Whether innate and adaptive immune responses play any key role in the process and whether the immune system within the allograft contributes to the injury process are of considerable interest[51,52]

Materials and Methods 21 - 30

Place

Period of Study

Criterias

Procedure and Data collection

State Organ Coordinator and Organ Sharing

Materials and Methods

Place

All cadaver renal transplants being carried out in Govt Stanley Medical College and Hospital are prospectively studied to assess the impact of variability in cold ischaemia on graft function in renal transplant recipient patients

Period of Study

October 2008 to April 2011

Inclusion criteria

All cadaver transplants first or second

Criteria for Taking Up For Cadaver Transplant:

- Patients with irreversible renal failure
- Patients in cadaver waiting list
- Dialysis dependent patients
- Patients under the age of 50 years
- Second Transplant patients
- ABO compatible patients

Exclusion criteria

Live related renal transplant

Live unrelated renal transplant

Non heart beating cadaver renal transplant

Cadavers of less than 6 years

Cadavers with creatinine of more than 2

Cadavers with hypertension and Diabetics

Design

Prospective study

Sample Size

40 cadaver transplants

Procedure and Data Collection

- Recipient's demographic factors like Age, Gender, Occupation, and Literacy were noted.
- Selection of recipients is based on their seniority in cadaver waiting list and cross match result.
- All recipients were maintained on Haemodialysis.
- All recipients were ABO compatible and cross-match negative and they are followed up regularly in our OP.

- Human Leukocyte Antigen (HLA) and Panel Reactive Antibody (PRA) were not done to any of our recipients.
- CMV status of the recipient was not checked routinely. However, if any suspicion of CMV infection like hepatitis, leucopenia, etc., the CMV status of the recipient was checked with pp65 antigen and treated with Vangancyclovir if they were positive.
- Graft function as assessed by serum creatinine within a week and first postop day urine output was the primary outcome analysed.
- There were no drop outs from follow-up.
- Donor kidneys were received from various hospitals in Tamil Nadu and from our own hospital.

- ❖ Donor's age ranged from 15 – 60 years without evidence of kidney disease or any infection.
- ❖ None of donors had diabetes mellitus or hypertension.
- ❖ All the donors had negative serology (HBV, HCV, HIV).
- ❖ All grafts were perfused with HTK solution (Custodial solution)
- ❖ <u>Custodial (HTK) solution (in mmol/L)</u>

• Sodium chloride	15.0
• Potassium chloride	9.0
• Potassium hydrogen 2-ketoglutarate	1.0
• Magnesium chloride	4.0
• Histidine Hcl	18.0
• Histidine	180.0
• Tryptophan	2.0
• Mannitol	30.0
• Calcium chloride	0.015

- ❖ They are stored in ice box with three bag technique during transportation
- ❖ Donor's age, sex, cause of death, graft side and abnormality and biochemical profile were noted.
- ❖ Transplant surgery was done by two teams of Urologists.
- ❖ Ethical Committee approval from Stanley Medical College, Chennai was obtained for this study.

State Organ Coordinator and Organ Sharing

All hospitals, approved for transplantation of human organs, and who are willing to participate in the arrangements for cadaver organ transplant program shall indicate their willingness to the Convenor, Cadaver transplant program, Tamil Nadu.

All participating hospitals will upload details of their waiting list of prospective cadaver organ recipients through an online form to a computer database that will be maintained by the Transplant Coordinator of the Government General Hospital, Chennai.

The database will maintain prioritization lists for

(i) each hospital

(ii) for all Government hospitals combined

(iii) for all private hospitals combined and

(iv) for Government plus private hospitals combined, based on preset criteria determined in this order.

The organ(s) of the brain dead patient shall be shared in the well discussed order, based on the respective prioritization list.

Deceased Donor Graft Allocation Policy

A separate cadaver waiting list for each blood group of potential recipients is maintained according to their date of induction into haemodialysis. This

seniority list is available online .Recipients with co–morbid conditions are temporarily deleted from the list and included again once they recover.

PROCEDURE

Pre operative treatment

All recipients were given haemodialysis pre operatively. They were started on immunosuppression prior to surgery as below.

Drugs	Day before Surgery 4 p.m.	0 POD (4 a.m.)
T.Tacrolimus	0.066 mg/kg	0.066 mg/kg
T.MMF	500 mg	500 mg
T.Prednisolone	0.5 mg/kg	0.5 mg/kg

Mode of preservataion and Transport

Nephrology team would go to organ harvesting centre and would wait for the organ to be handed over after cold perfusion. Government officials including traffic controllers provided green channel route for speedy delivery of organ to the transplant centre

Receipient selection and preparation

Prospective receipients of compatible blood groups from waiting list priority called for and blood investigations be carried out. They also underwent dialysis. Negative crossmatch with donor (Lymphocytic microcytotoxicity) patient were selected for transplant

Bench Dissection

Received donor cadaver kidney was dissected further in bench dissection by clearing excess perinephric fat and ligating gonadal vessel and renal vein tributaries.

Cadaver kidney was further perfused with HTK solution to preserve kidney from ischaemia and to avoid reperfusion injury.

Operative Technique

Under strict aseptic condition modified Gibson's incision was made over anterior abdomen. Abdomen was entered extraperitoneally. Receipient bed was prepared by dividing the inferior epigastric vessels and delineating both external and internal iliac vessels.

Anastomosis of the renal vessels to the iliac vessels was performed as follows.

Graft artery to internal iliac artery (end to end) or external iliac artery (end to side- and also in accessory artery anastomosis) using 6 ‘0’ prolene

Graft vein to external iliac vein – end to side using 6 ‘0’ prolene. Then clamps will be released.

Ureterovesical anastomosis was done using 4’0’ vicryl. DJ stent (5 F 16 cm) if any need was kept. DT will be kept. Abdomen was closed in layers.

During anastomosis of graft vessels, methyl prednisolone 1 g was given as I.V. infusion.

Post operative treatment

Fluids (half normal NS) were given according to their urine output. Immunosuppression was given as follows:

T.Tacrolimus 0.066 mg/kg bd (Target tacro level 10 – 12 ng/ml subsequently reduced to 5ng/ml by 6 months)

T.MMF 500 mg bd

T.Prednisolone 0.5 mg/kg od

Tacrolimus levels were assessed on POD-5 for all recipients. Doppler of graft vessels are assessed on POD-7. Recipients urinary Foley’s Catheter was removed when urine output less than3litres. Drainage tube was removed if

drainage fluid is less than 50 ml. DJ stent was removed on 4th post operative week.

After 10 days, recipients were discharged and they were seen as outpatient at intervals of twice weekly for one month than weekly once for two months, thereafter fortnightly for one year and monthly for one year life long. During each visit, patient's condition, renal function test and complete blood count were analyzed. Post operative drugs including immunosuppressants are given free of cost and all investigations are done at no cost.

Results **31 - 39**

Gender

Graft

Cold Ischaemia

Complications

Death of Receipient

Cause of Death -Cadaver

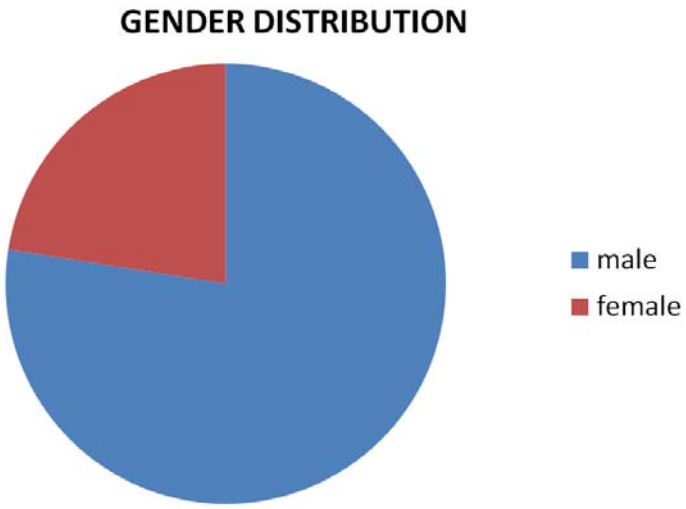
Results

40 patients received cadaver graft in our center from October 2008 to April 2011.

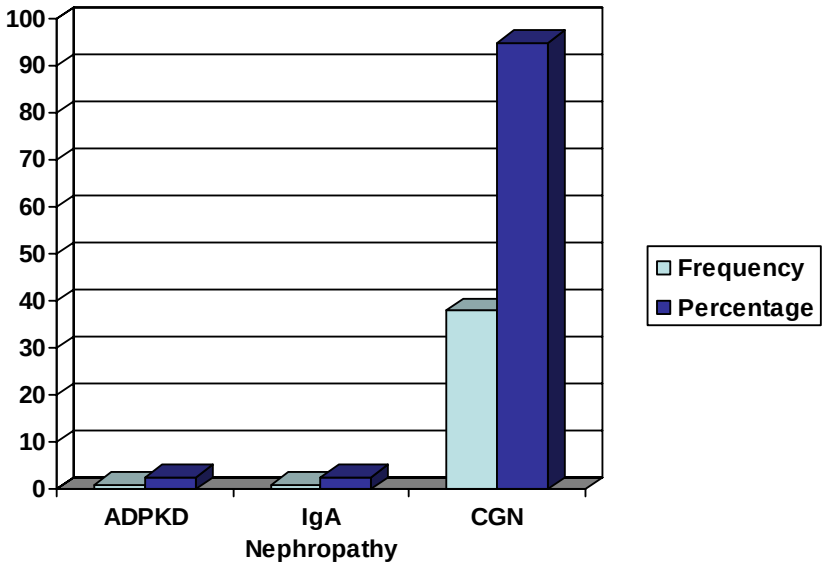
Mean age of the recipients was 33.75 years.

Among them males were 31 (77.5%) and

Female were 9 (22.5%).



Out of 40 patients one had ADPKD, one had biopsy proven IgA nephropathy and the remaining had contracted kidney for which native kidney biopsy was not done. The cause of chronic kidney disease for them is not known.



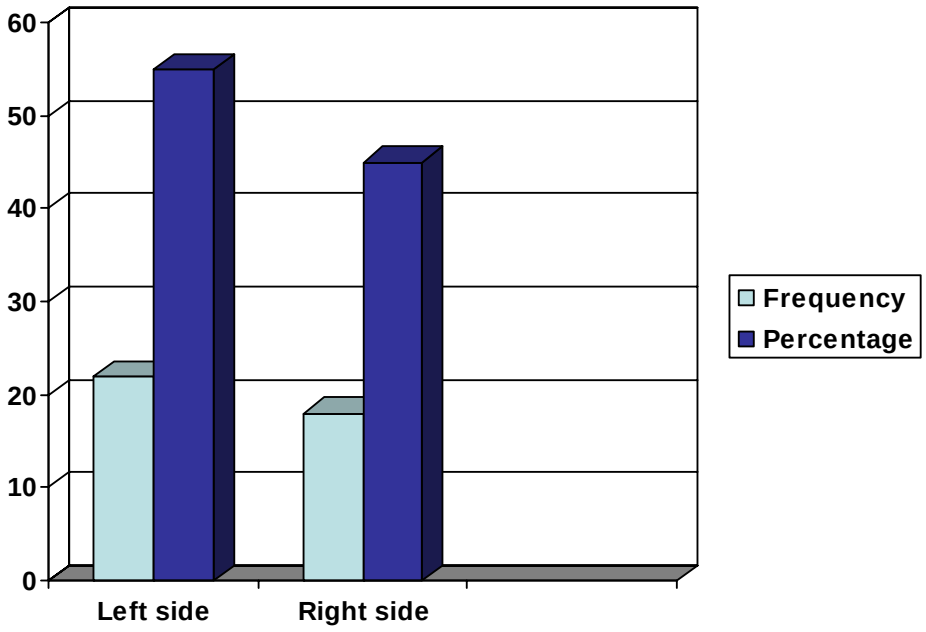
During the period of study, Diabetic patients were not included in the cadaver waiting list. All recipients were on antihypertensives. One of the recipients was Hepatitis B positive. One was Hepatitis C positive. None of the patients received induction therapy like ATG (Anti thymocyte globulin), Daclizumab or Basiliximab.

Only one recipient had second transplant and all other had first transplant. All the recipients received tacrolimus, mycophenolate mofetil and prednisolone

Graft side

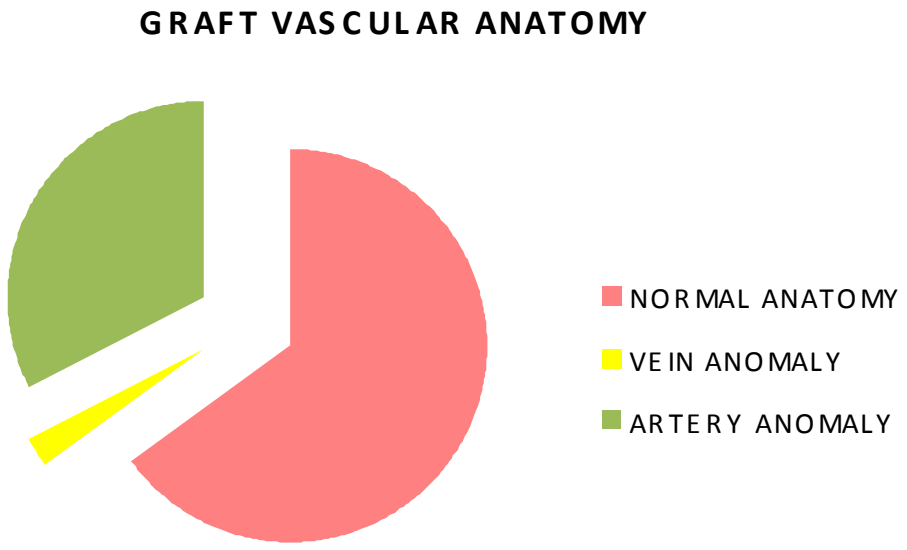
Left side graft : 22

Right side graft : 18



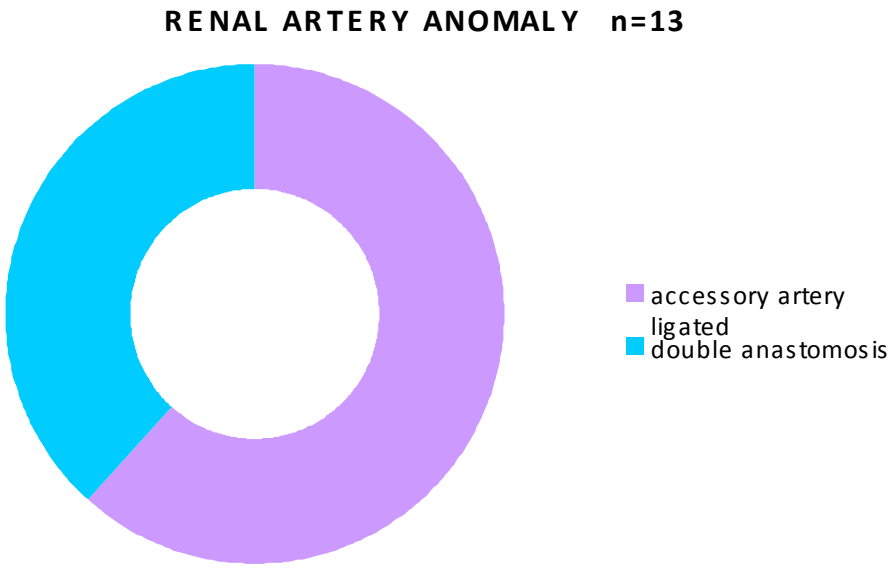
Graft

No anomaly	: 26
Vessel Anomaly	: 14
Renal vein anomaly	: 1
Renal artery anomaly	: 13



Management of Anomalous vessels

Anomalous upper pole artery ligated	:8
Double anastomosis	: 5

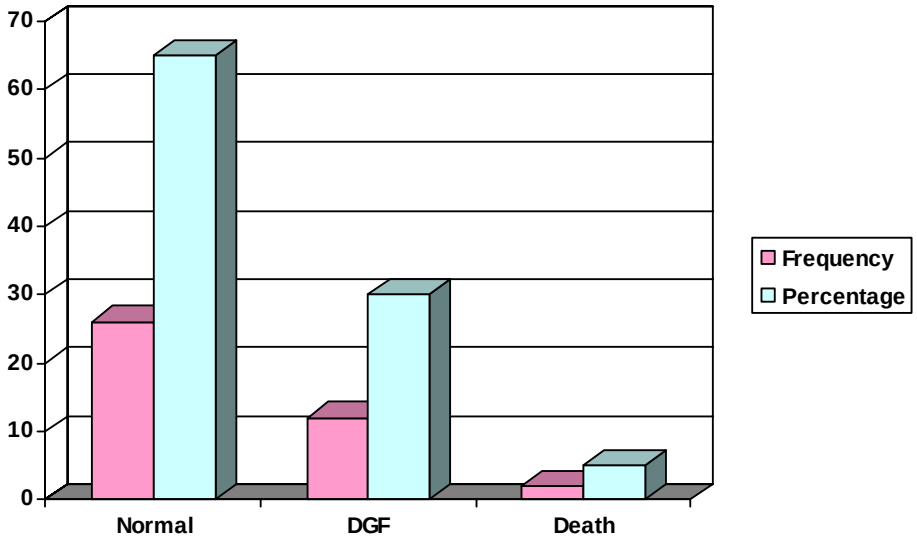


Graft Function

Normal : 26

DGF : 12

Death : 2

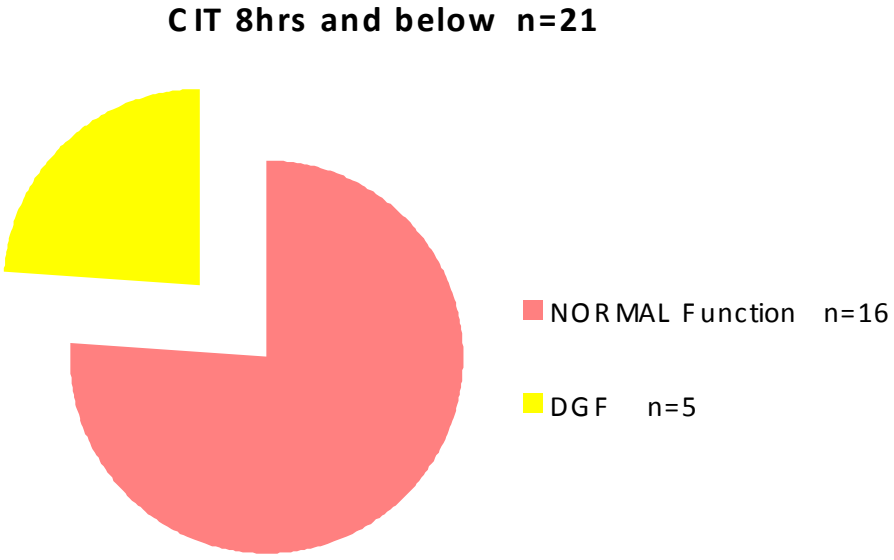


CIT and Graft Function

CIT at 8hrs and below : 21

Normal function : 16

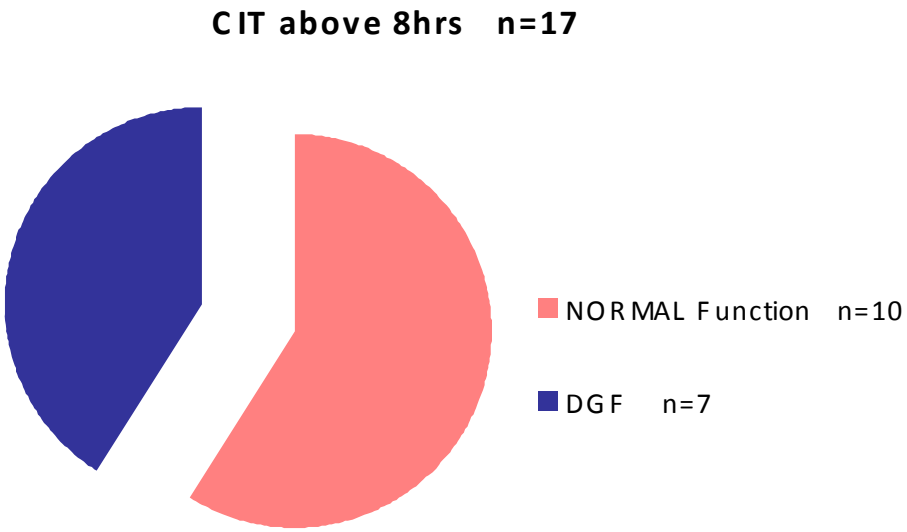
DGF : 5



CIT above 8 hrs : 17

Normal function : 10

DGF : 7

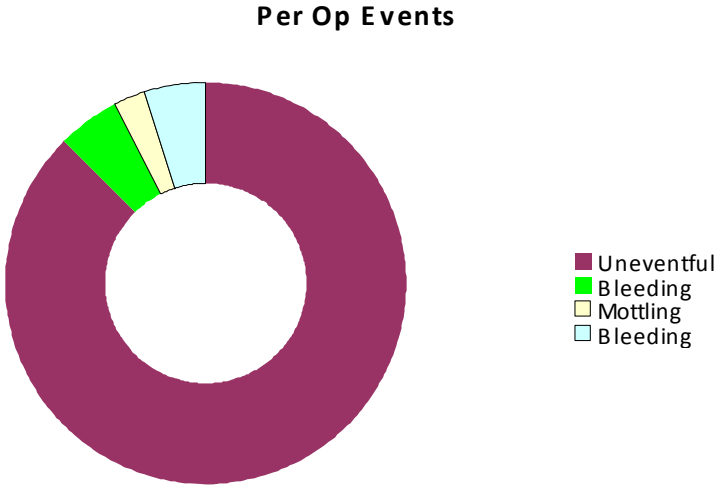


Per Op events

Uneventful : 35

Mottling due to rejection : 1

Bleeding : 2

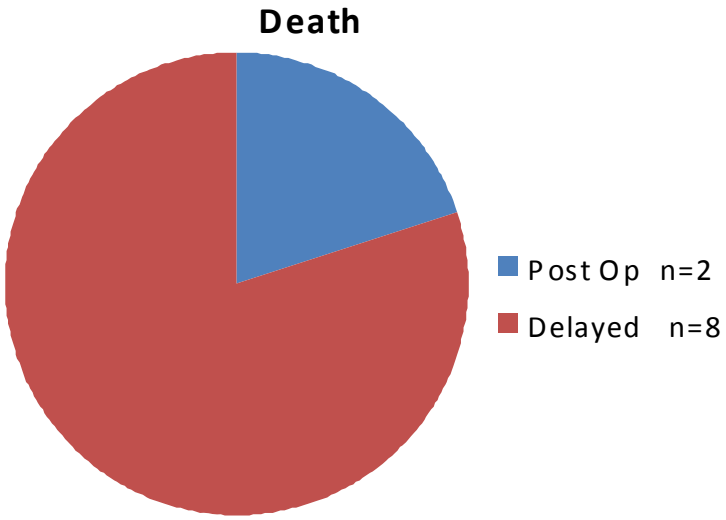


Post op Complications

Sepsis	: 2
Nephrectomy due rejection	: 1
Leg ischaemia	: 1
Pancreatitis	: 2
Persistent DT	: 2
Pneumonia	: 2
Haematuria	: 1
Fungal Sinusitis	: 1

Death n=10

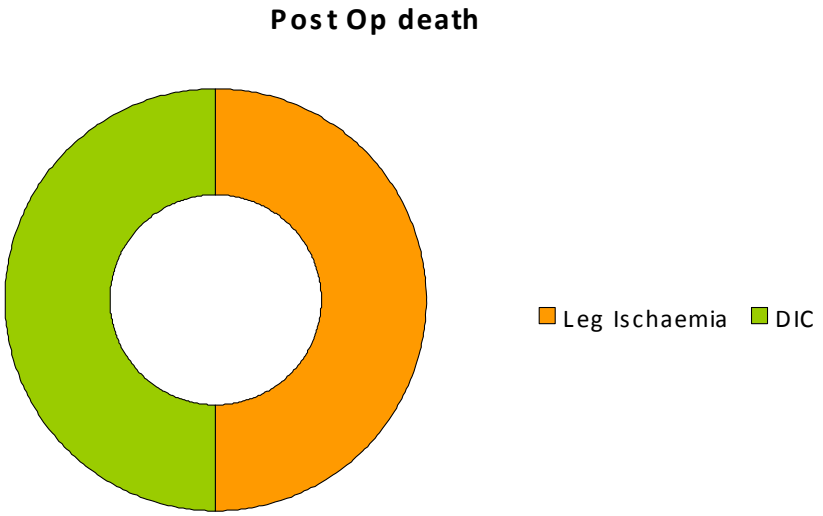
Post Op	: 2
Delayed	: 8



Post OP death – Cause

Leg ischaemia : 1

DIC : 1



Delayed death

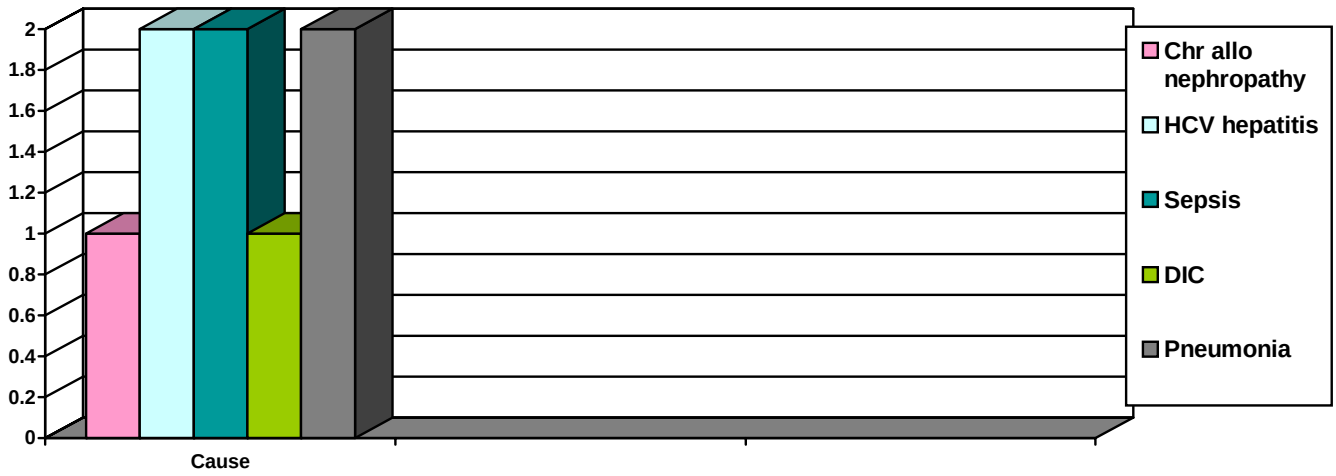
Chronic Allograft Nephropathy : 1

HCV hepatitis : 2

Pneumonia : 2

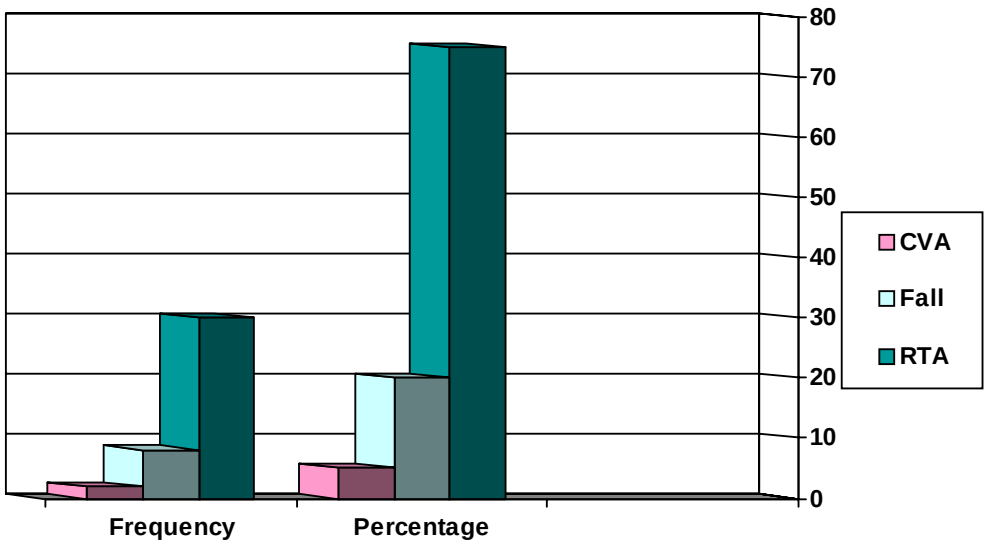
DIC : 1

Sepsis : 2



Cause of Death – Cadaver

CVA	: 2
Fall from Height	: 8
RTA	: 30



Discussion **40 -49**

Cold ischaemia Time- the cadaver specific

Newer Insights

Baseline Characteristics

General and Technical Factors

CIT on graft function – continuous trend

Harvesting centre (Transport) Vs CIT

DISCUSSION

Cold Ischaemia Time - the cadaver specific

Although there has been a substantial improvement in the acute survival of renal allografts, the chronic allograft loss, particularly those from cadaveric donors, continues to occur at an unacceptably higher rate[62] . The other parameter that has remained unchanged over the years is the CIT, which is again relevant to cadaveric kidneys.

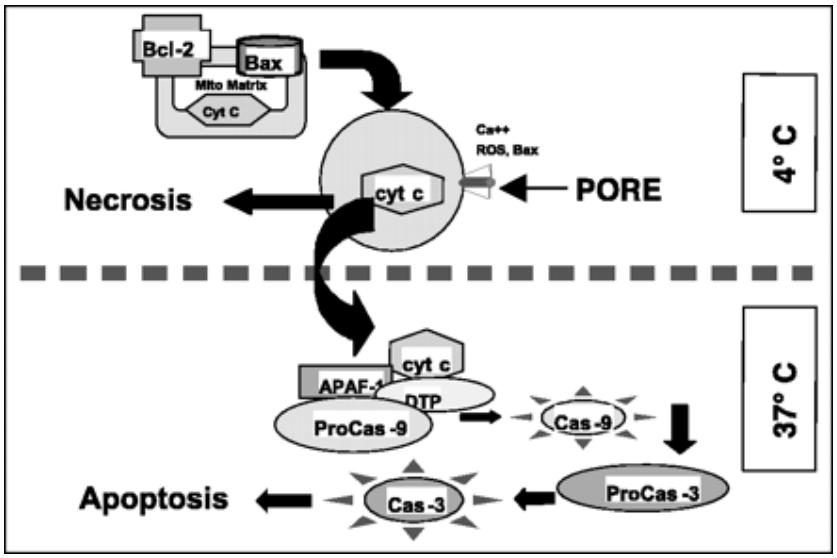
Donor factors such as brain death and CIT are unique to cadaveric donors, and their influence may account for much of the survival difference There is mounting evidence from experimental and clinical studies that the level of injury to organs from cadaver donors may be influenced by events occurring in the intensive care unit (ICU)[55] and around the time of brain death[56] , and that these may affect subsequent transplant outcome.

Having been exposed to factors related to the dying process other influences will be added to the donor organ which will impact on the final outcome of transplantation. These will be related to the retrieval process itself and the subsequent period of cold ischaemia before reperfusion.

Finally, recipient factors will become active on reperfusion and for the lifetime of the graft. It is this blending of multiple donor and recipient factors that generates the final outcome of the transplant process.

NEWER INSIGHTS INTO COLD ISCHEMIC INJURY MECHANISMS

Although cold ischemic injury, as in warm ischemic injury, is considered to be due to massive cell necrosis, several recent studies suggest that the apoptotic form of cell death does occur, but only after the reperfusion of transplanted organs. Currently, suppressing apoptosis in the acute injury setting is considered beneficial.



Permeability transition pore (PTP)

Cold (*top*) via calcium and free radicals opens permeability transition pore (PTP), causing marked mitochondrial swelling, which, in turn, triggers key

apoptotic events and sets the stage for apoptosis during rewarming. Bcl-2 family of proteins, Bax, Bcl-X_L, and Bcl-2, are particularly abundant at the junction between inner and outer mitochondrial membranes, which is the site where PTPs are formed and the site of membrane disruption during cold storage-rewarming. Bcl-2 counteracts the proapoptotic activity of the pore-forming Bax protein.

Ratio of Bcl-2 to Bax

Normally, the ratio of Bcl-2 to Bax is maintained in favor of Bcl-2, but during cold storage it is shifted toward Bax. Mitochondrial leakage of cytochrome *c* and other proapoptotic proteins such as Apaf-1 leads to the formation of apoptosome complexes composed of cytochrome *c* (cyt c), Apaf-l, ATP, and procaspase-9. Formation of the complex makes Apaf-I more competent at binding procaspase-9 and recruiting other caspases-1, -2, -3, and -4 through its recruitment domain (CARD), triggering the caspase cascade, the latter occurring during the rewarming phase after cold storage[53,54]

BASELINE CHARACTERISTICS OF RECIPIENTS

No	Characteristics	Frequency	Percentage
1	**Gender**		
	Male	**31**	**77.5**
	Female	**9**	**22.5**
	HT	**40**	**100**
2	**Blood Group**		
	O positive	**14**	**35**
	B positive	**15**	**37.5**
	A positive	**5**	**12.5**
	AB positive	**4**	**10**
	B Negative	**1**	**2.5**
	A Negative	**1**	**2.5**
3	**NKD**		
	ADPKD	**1**	**2.5**
	Unknown cause	**38**	**92.5**
	IgA Nephropathy	**1**	**2.5**
5	**Graft side**		
	Left	**22**	**55**
	Right	**18**	**45**
6	**Anomaly**	**14**	**35**

7	**I/II Transplant**		
	I	**39**	**97.5**
	II	**1**	**2.5**
8	**Immunosuppression**		
	Tacro + MMF+ Prednisolone	**40**	**100**

General Factors

- A single-centre study permits the use of data in a fairly homogeneous set and provides a useful complement to multi-centre studies.
- In the major part of statistical analysis two post op deaths were excluded.
- Like most studies, males predominated in the renal receipient group

- Analyses of the datas were carried out by using EPI-INFO software.
- Females in the receipient group were less in number (22.5%). So could not stratify the receipient into male and female group
- Similarly the numbers in individual blood groups were minimal. So graft function vs blood group could not be carried out.
- Age of the patient and tacro level did not appear to influence the graft function statistically.

Technical Factors

- Post-transplant serum creatinine as a marker of graft function is limited, as it varies by age, sex, race, and body weight. So first
  post -op day urine output is also taken to assess renal function.
- In cadaveric allografts, DGF occurs in 20% to 50% of patients. DGF in this study (12/38) is well within this limit despite all logistical problems and financial strains of carrying the major surgery in the Government setup.

- The average CIT reported in the UNOS registry over the years has more or less remained unchanged around 20 hours. In the present study it is 8.3 hrs

Cold Ischaemia Time-Chi Square Test

CIT: 8hrs

Relative risk : 1.96(range 0.6 -3.5)

P value : 0.14

CIT: 9hrs

Relative risk: 2.4(range 1-6)

CIT on graft function – continuous trend

CIT divided into 8 hrs, 8-10 hrs, more than 10 hrs and linear trend analysis of cold ischaemia time on graft function showed as cold ischaemia time increases the risk of delayed graft function increases

Odds ratio : 2.9

P value : 0.05

- CIT was the most significant risk factor for the development of DGF and its effect appears to be continuous. This observation is supported by other investigators[57].

- Other studies have suggested that there are significant time points after which the risk of DGF accelerates[58]. It is attractive to imagine a specific threshold CIT after which the risk of DGF is significantly increased. However, it is clear that each hour even at short CIT adds additional risk.
- Recent multi-centre studies have confirmed the importance of cold ischaemia time and donor age for graft survival. Su et al[59] show that the effect is significant for times over 37 h compared with baseline. However, they do not test for discontinuity and overall, their data appear consistent with a continuous effect of CIT. The Collaborative Transplant Study[60] suggests that there is 'little effect below 25 h'.

Harvesting centre (Transport) Vs CIT

- In US reduction in CIT was observed during the 10-year period with an overall reduction of 4.8 hour) with fewer kidneys being cold-stored over 30 hour in the second half (13% in 1996 to 2000 vs 25% in 1990 to 1995, P < .001)[61] .Initially kidneys were received from different corners of Tamilnadu by different transport .

Cadaver kidney from different places n =11.

Average CIT was 10hrs

- Now majority of cadaver kidneys are from Government General Hospital and from our institution Govt Stanley Hospital

  n=29

  Average cold ischaemia time was 7.76hrs

  Clear reduction in CIT

- Difficulty in deceased donor graft procurement, transport, delay in getting cross match results especially during odd hours and arranging theatre during odd hours are some factors accounting for variability in cold ischaemia time. Over a period of time these are bound to improve

Conclusion **50 - 52**

Strength and limitation of the present study

The Future

Conclusion

Strength and limitation of the present study

- The present study is limited as a result of failure to accurately assess renal core temperature and absence of continuous hypothermic machine perfusion which gives better and predictable perfusion than flushing the kidney with cold perfusion fluid and storing in hypothermic ice.

- Despite these limitations, present analysis is important as it clearly shows Cold ischaemia time is the most significant risk factor for the development of DGF and its effect appears to be continuous.

- Sharing of cadaveric kidneys at national level improves tissue matching, but often lengthens the cold ischemia time (CIT).

The Future- Measures to improve renal allograft function

In future the following aspects could be evaluated and research would provide more possibilities

- Preemptive transplantation in live kidney transplantation.
- Increased donation from younger, previously healthy deceased donors.
- Preferential matching of younger deceased donors with younger recipients.

- Zero mismatching of HLA antigens
- Improved organ preservation
- Reduced cold ischemia time
- Nephron dosing (e.g. matching of donor recipient sex, body mass index)
- Calcineurin inhibitor sparing immunosuppressive protocols.
- Angiotensin converting enzyme inhibitors, angiotensin receptor blockers.
- Aggressive control of hyperlipidemia, hypertension.

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RECIPIENT PROFORMA

Name: **Age:** **Sex:** **Blood Group:**

Address: **Occupation:**

Income: **Social Status:** **Wt:** **Ht:** **BMI:**

Medical History: **DM** **Hypertension:**

Lab:

Urea:

Creatinine: **eGFR** **NKD**

Sodium **Potassium** **Bicarbonate** **Chloride**

LFT **Serum Billurubin(T)** **D** **Alb** **Glob** **SGOT** **SGPT** **SAP**

Urine Routine: **24hrs urine Protein** **Urine C/S**

HB **PCV** **Platelets** **TC** **PT** **aPTT**

INR

BT **CT** **FT4** **TSH**

Calcium **Phosphorus** **Uric acid**

Serology: **HBsAg** **Anti Hcv** **HIV** **CMV**

ECHO **Gynecology** **Dermatology**

Dental **ENT** **MGE**

Urology **Endoscopy** **Anesthesia**

Cross matching **USG**

Renal Biopsy **Chest X-ray** **ECG**

On MHD Duration: **Weekly:**

H/o Blood Transfusion

H/o access problem **Doppler iliac vessels:**

Date of Reg: **Date of Transplant:** **Waiting time:**

Intra op events:

Post op events:

Post Transplant Outcome

Immunosuppression

	POD 1	POD 3	POD 5	POD 7	POD 1 MON	POD 3 MON	POD 6 MON
Creatinine							
Urine output							

Discharge Cr:

Post Transplant Ultra sound / Doppler

Tacro level **HB** **PCV**
Platelets

Biopsy **TC**

Urine routine

24 hrs Urine Protein

DONOR PROFORMA

Name: **Age:** **Sex:**

Address: **Occupation:**

Blood Group:

Income: **Social Status:** **Wt:** **Ht:** **BMI:**

Cause of Brain death:

Medical History: **DM** **Hypertension:**

Urea:

Creatinine:

Sodium **Potassium** **Bicarbonate** **Chloride**

LFT **Serum Billurubin(T)** **D** **Alb** **Glob** **SGOT**
SGPT **SAP**

HB **PCV** **Platelets** **PT** **aPTT** **INR**

Serology: **Anti Hcv** **HBsAg** **HIV** **CMV**

Clinical:

BP: **Pulse:** **Ionotropic support:**

Urine Output:

Clamp Time: **CIT:** **Graft abnormality:**
Graft Side:

USG KUB

DOS: **Donor Nephrectomy:**
Date of Transplant:

MASTER CHART

Sl.No	Name	Age	Sex	Blood group	Cross Match	Date of Transplant	BMI	Cr- 1wk	Cr-1Mon	Cr- 6Mon	Native Kidney Disease	Hypertension	Diabetes Mellitus	on HD-mon	ECHO	Harvesting place	INTRA OP	POST OP	Tacro Level	Serology	Doppler	DONOR	Age	Sex	Blood group	Diabetes Mellitus	Hypertension	Graft side	CIT	Sl.No	Graft anamoly	Cause of Death	Graft function	Patient Status	Graft Biospy	Perfusion Sol	Immuno suppression	Tx - I/II	POD1 output	cause of death receipier
1	**Kondiaraj**	32	M	O+	5.10%	25.10.08	23.1	1.7	1.6	1.4	CGN	YES	NO	36	N	Lifeline	NIL	NIL	10	NEG	N	**Radhakrishnan**	49	M	O+	NO	NO	LEFT	10	1	3 RA/CUFI	RTA	N	DIED(18/1/10	CAN	HTK	T/M/P	I	1500	CAN,sepsis
2	**Balaraman**	32	M	B+	5.10%	21.11.08	22.2	1.6	1.2	1.1	CGN	YES	NO	12	LVH	coimbat	NIL	NIL	8	NEG	N	**Gnanaprakasem**	26	M	B+	NO	NO	LEFT	8	2	NIL	RTA	N	DIED(20/1/10	NIL	HTK	T/M/P	I	3400	HCV sepsis
3	**Bala krishnan**	31	M	B+	5.10%	14.1.09	20.2	8.7	1.4	1.3	CGN	YES	NO	6	LVH	apollo	acc ligated	HD-3	10	NEG	N	**Premkumar**	48	M	B+	NO	NO	LEFT	12	3	2RA	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	390	
4	**Lilly Theresa**	29	M	B+	5.10%	28.1.09	32.2	2	1.3	1.2	CGN	YES	NO	6	N	apollo	eeding,2RA liga	NIL	3	NEG	N	**Jeevarathinam,**	56	F	B+	NO	NO	LEFT	12	4	3RA	RTA	N	ALIVE	NIL	HTK	T/M/P	I	4000	
5	**SasiKumar**	29	M	A+	5.10%	4.2.09	18.7	1	1.2	--	CGN	YES	NO	28	N	Stanley	acc to EIA	NIL	16.3	NEG	N	**Suganya**	15	F	A+	NO	NO	LEFT	3	5	2RA	RTA	N	DIED(9/4/9)	NIL	HTK	T/M/P	I	21000	CMV pneumonia,sepsis
6	**Baskar**	38	M	AB+	5.10%	9.3.09	17.9	2.3	1.6	1.7	CGN	YES	NO	3	LVH	SRMC	NIL	ACC.HT	10	NEG	N	**Asha**	20	F	AB+	NO	NO	LEFT	10	6	NIL	RTA	N	DIED(15/6/9)	HUS	HTK	T/M/P	I	5750	tic microangiopathy, graft rejecti
7	**Dasan**	48	M	O+	5.10%	14.3.09	25.6	4.2	4	1.6	ADPKD	YES	NO	24	N	cmc	NIL	SEPSIS	2.9	HBV+	N	**Jeyanthi Reddy**	39	M	O+	NO	NO	LEFT	10	7	NIL	RTA	DGF	DIED(30/4/9)	NIL	HTK	T/M/P	I	5000	HCV sepsis(fungal)
8	**Sakthivel**	27	M	B+	5.10%	26.4.09	25.4	13.1	--	--	CGN	YES	NO	30	N	apollo	Mottling	Nephrectomy	3.1	HCV+	N	**Chandru**	27	M	B+	NO	NO	LEFT	10	8	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	330	
9	**Renuka**	34	M	B+	5.10%	11.5.09	22.2	1.4	1	0.9	IgAN	YES	NO	48	N	apollo	Venous leak	NIL	14.2	NEG	N	**Dharani**	19	F	B+	NO	NO	LEFT	7	9	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	2125	
10	**Xavier**	43	M	O+	5.10%	21.6.09	25.8	--	--	--	CGN	YES	NO	24	N	SRMC	EIA	Rt leg ischemia		NEG	N	**Sivaprakasam**	42	M	O+	NO	NO	LEFT	12	10	2 RA	RTA	DGF	DIED(22/6/9)	NIL	HTK	T/M/P	I	350	postop, ischaemic leg
11	**GopiKrishnan**	40	M	B+	5.10%	8.8.09	22.1	3.1	1.2	1.3	FSGS	YES	NO	3	EF40%	kamatchi	Hilum anas	Hypotension	18	NEG	N	**John rayan**	57	m	B+	NO	NO	LEFT	10	11	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	3000	
12	**Subramani**	48	M	O+	5.10%	15.10.09	19.5	0.9	0.9	0.8	CGN	YES	NO	48	N	cmc	NIL	NIL	8	NEG	N	**Vinoth Kumar**	28	M	O+	NO	NO	LEFT	11	12	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	14800	
13	**JayaKumar**	30	M	B-	5.10%	27.10.09	20.8	2.1	1.2	1.1	CGN	YES	NO	1	N	GH	NIL	Pancreatitis	11	NEG	N	**Iyyappan**	28	M	B+	NO	NO	RIGHT	10	13	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	6370	
14	**Eswaran**	31	M	B+	5.10%	13.11.09	28.1	7.3	5.6	--	CGN	YES	NO	12	N	GH	Hypotension	persistent DT	13	NEG	N	**Loganathan**	23	M	B+	NO	NO	RIGHT	12	14	2 RV	Fall from h	DGF	DIED (7/6/10)	ACR/ AHR	HTK	T/M/P	II	200	Nephrectomy,sepsis
15	**Prema**	35	F	A-	5.10%	4.12.09	31.2	7.4	1	1.2	CGN	YES	NO	24	LVH	GH	Hypotension	Ionotropes	3.8	NEG	N	**JaiAnand**	18	F	A+	NO	NO	LEFT	12	15	2 RA	Fall from h	DGF	ALIVE	NIL	HTK	T/M/P	I	800	
16	**Revathy**	24	F	O+	5.10%	12.12.09	17.6	1.2	1.3	1.1	CGN	YES	NO	12	LVH	GH	NIL	SEPSIS/ARDS	15	NEG	N	**Palanivel**	24	M	O+	NO	NO	RIGHT	11	16	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	17600	
17	**Devaraj**	46	M	O+	5.10%	16.12.09	18.1	1.3	1.2	1.1	CGN	YES	NO	12	LVH	GH	NIL	NIL	10.3	NEG	N	**chandran**	56	M	O+	NO	NO	LEFT	11	17	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	14000	
18	**Palani**	37	M	O+	5.10%	27.12.09	19.3	1.6	1.4	1.2	CGN	YES	NO	24	LVH	Stanley	NIL	NIL	15.2	NEG	N	**Jayabharthi**	15	F	O+	NO	NO	LEFT	3	18	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	14700	
19	**Elawarasan**	22	M	AB+	5.10%	20.2.10	17.4	1	0.9	--	CGN	YES	NO	24	N	GH	NIL	NIL	9	NEG	N	**Vijay**	12	M	AB+	NO	NO	LEFT	5	19	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	10400	
20	**Riyaz ali**	25	M	B+	5.10%	27.2.10	18.9	0.9	0.8	0.8	CGN	YES	NO	12	N	GH	ow p acc ligate	NIL	12	NEG	N	**Venkatasen**	29	M	O+	NO	NO	RIGHT	5.5	20	2 RA	RTA	N	ALIVE	NIL	HTK	T/M/P	I	9650	
21	**Dass Prakash**	31	M	B+	5.10%	19.3.10	26.6	1.6	1.2	1	CGN	YES	NO	6	N	GH	NIL	NIL	10.9	NEG	N	**Kuppan**	45	M	B+	NO	NO	RIGHT	10	21	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	10000	
22	**Rajan**	36	M	B+	5.10%	6.4.10	22.3	5.3	--	--	CGN	YES	NO	3	N	GH	NIL	Fungal sinusitis	9.2	NEG	N	**Malliga**	34	F	B+	NO	NO	RIGHT	8	22	NIL	RTA	DGF	DIED(16/4/10	NIL	HTK	T/M/P	I	300	Fungal sepsis
23	**Devi**	29	F	O+	5.10%	11.4.10	17.3	1.2	0.9	0.9	CGN	YES	NO	1	LVH	GH	NIL	eumonia,Stitch absc	9.3	NEG	N	**Lakshmi**	45	F	O+	NO	NO	RIGHT	9	23	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	5300	
24	**Nirmala**	34	F	AB+	5.10%	14.4.10	18.7	6.5	0.9	0.8	CGN	YES	NO	36	LVH	Stanley	NIL	ATN	9.8	NEG	N	**Rajadurai**	19	M	A+	NO	NO	LEFT	8	24	NIL	Fall from h	DGF	ALIVE	NIL	HTK	T/M/P	I	1500	
25	**Meramohiden**	35	M	O+	5.10%	18.6.10	17.3	1	1.4	1.5	CGN	YES	NO	6	EF30%	Stanley	NIL		3.5	NEG	N	**Rajarathinam**	24	M	O+	NO	NO	LEFT	6	25	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	20000	
26	**Kumar**	32	M	A+	5.10%	24.6.10	20.5	1.4	1.7	1.7	CGN	YES	NO	36	N	GH	ow p acc ligated		11.2	NEG	N	**Prabhakar**	22	M	A+	NO	NO	RIGHT	8	26	2 RA	Fall from h	N	ALIVE	NIL	HTK	T/M/P	I	6150	
27	**Kamaraj**	43	M	O+	5.10%	01.7.10	19.8	3.5	1.9	1.3	CGN	YES	NO	8	LVH	Stanley	NIL	left pneumonia	3.5	NEG	N	**Gaja**	50	M	O+	NO	NO	LEFT	5	27	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	300	
28	**Abdul rahim**	30	M	AB+	5.10%	04.7.10	16.3	1	1.1	0.9	CGN	YES	NO	24	LVH	GH	NIL		4.1	NEG	N	**Ravikumar**	29	M	AB+	NO	NO	RIGHT	7	28	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	12300	
29	**Nasir ali**	25	M	O+	5.10%	15.7.10	27.2	7	2	1.2	CGN	YES	NO	7	N	GH	NIL		15.3	NEG	N	**Ramesh**	22	M	O+	NO	NO	RIGHT	15	29	NIL	Fall from h	DGF	ALIVE	NIL	HTK	T/M/P	I	400	
30	**Amuthali**	20	F	B+	5.10%	22.7.10	16.2	1.1	1.2	1.2	CGN	YES	NO	9	EF48%	GH	acc ligated	persistent Dt ooze	5.1	NEG	N	**Kasirajan**	31	M	B+	NO	NO	RIGHT	6.5	30	2 RA	RTA	N	ALIVE	NIL	HTK	T/M/P	I	4650	
31	**Periasamy**	29	M	A+	5.10%	13.10.10	17.5	4	1.5	1.3	CGN	YES	NO	36	N	GH	acc art toEIA	d clot,scopy,evacua	5.9	NEG	N	**ThadaTherotti**	20	M	A+	NO	NO	RIGHT	6	31	2 RA	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	7650	
32	**Arumugam**	35	M	A+	5.10%	19.10.10	21.2	1.6	1.2	1.2	CGN	YES	NO	18	LVH	Stanley	NIL		5.6	NEG	clot+	**Mohan**	25	M	A+	NO	NO	RIGHT	8	32	NIL	CVA	N	ALIVE	NIL	HTK	T/M/P	I	7550	
33	**Narasingam**	23	M	B+	5.10%	30.10.10	17.4	6.8	1.2	1	CGN	YES	NO	42	N	GH	hain EIA, acc II	Pancreatitis	1	NEG	N	**Desingh**	32	M	B+	NO	NO	LEFT	4	33	2 RA	RTA	N	ALIVE	NIL	HTK	T/M/P	I	1725	
34	**Ramadoss**	57	M	B+	5.10%	17.11.10	19.6	6.2	1.5	1.4	ADPKD	YES	NO	11	N	GH	NIL	HD	9.6	NEG	N	**Jyothi**	60	F	B+	NO	NO	RIGHT	8	34	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I	1620	
35	**Prakasam**	40	M	B+	5.10%	6.12.10	19.2				CGN	YES	NO	6	N	Stanley	acc art toEIA		_	NEG			35	M	B+	NO	NO	LEFT	8	35	2 RA	CVA	_	DIED	ATN	HTK	T/M/P	I	_	DIC
36	**Suja**	27	F	O+	5.10%	28.12.10	18.7	1.8	1	1.2	CGN	YES	NO	12	N	GH	acc art toEIA		5.1	NEG	N	Kali	23	M	O+	NO	NO	RIGHT	8.5	36	2 RA	Fall from h	N	ALIVE	NIL	HTK	T/M/P	I	13000	
37	Balaji	32	M	O+	5.10%	25.01.11	16.8	1.2	1.2		CGN	YES	NO	8	N	GH	NIL		6.8	NEG	N	Zegan	20	M	O+	NO	NO	RIGHT	5	37	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	18050	
38	**poonkodi**	45	F	B+	5.10%	05.02.11	19.4	1.2	1.1		CGN	YES	NO	7	N	GH	ow p acc ligated		11	NEG	N	Arun	22	M	B+	NO	NO	LEFT	8	38	2 RA	Fall fror	N	ALIVE	NIL	HTK	T/M/P	I	9500	
39	**Sajeevan**	34	M	A+	5.10%	09.03.11	18.3	1.1	1.1		CGN	YES	NO	10	N	Stanley	NIL		9.1	NEG	N	Sivaprakasam	33	M	A+	NO	NO	RIGHT	5	39	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I	2520	
40	**Mather**	32	M	O+	5.10%	27.03.11	26.3	1.1	1.1		CGN	YES	NO	9	N	GH	NIL		5.8	NEG	N	Jegadish	29	M	O+	NO	NO	RIGHT	8	40	NIL	Fall fror	N	ALIVE	NIL	HTK	T/M/P	I	4330	

Cr.1 wk :
Cr.1 mon :
Cr.6 mon :
Intra OP :
Post OP :
Tacro level :
CIT :
Tx - I/II :
CGN :
ADPKD :
IgAN :
LVH :
RTA :
T/M/P :

Creatinine value at 1

Creatinine value at 1 month Post Operation

Creatinine value at 6 months Post Operation

Intra Operative events

Post operative events

Tacrolimus through level

Cold Ischemic Time

First or Second Transplant

Chronic Glomeulo Nephritis

Autonomal Dominant Polycyotic Kidney disease

IgA Nephropathy

Left Ventricular Hypertrophy

Road Traffic Accident

s,

Mycophen