ORAL AND PERIODONTAL FINDING IN PATIENTS SUFFERING FROM RENAL FAILURE

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CERTIFICATE

This is to certify that this dissertation titled **"ORAL AND PERIODONTAL FINDING IN PATIENTS SUFFERING FROM RENAL FAILURE"** is a bonafide record of work done by **DR. S.SHANMUGAPRIYA** under our guidance and to our satisfaction, during her postgraduate study period of 2011-2014.

This study is submitted to **THE TAMILNADU DR. MGR MEDICAL UNIVERSITY** in partial fulfillment for the award of the degree of **MASTER OF DENTAL SURGERY - PERIODONTICS**, **BRANCH II**. It has not been submitted (partial or full) for the award of any degree or diploma.

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TITLE OF DISSERTATION	***ORAL AND PERIODONTAL FINDING IN PATIENTS SUFFERING FROM RENAL FAILURE***
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"A good teacher can inspire hope, ignite the imagination and instill a love of learning"

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ABSTRACT

BACKGROUND: The oral health status of chronic renal failure (CRF) patients undergoing treatment is complex due to other comorbid conditions. These patients appear to be predisposed to a variety of dental problems such as periodontal disease, narrowing of the pulp chamber, enamel abnormalities, premature tooth loss and xerostomia. Renal replacement therapy can affect periodontal tissues such as gingival overgrowth in immunosuppressed renal transplantation patients and increased levels of plaque accumulation, calculus formation, gingival inflammation, possible increase prevalence and severity of periodontal diseases in CRF patients. The presence of undiagnosed periodontitis may have significant effect on the medical management of CRF patient. Periodontitis has been found to contribute to systemic inflammatory burden including the elevation of C-reactive protein (CRP) in the general population dental care as well as primary preventive measures seems to be neglected in these patients.

AIM: This study compared the periodontal and dental health status of patients on renal transplant patients (RT), hemodialysis patients (HD) and pre dialysis patients (PD) with healthy controls (C).

METHODS: This case control, prospective, parallel design study was conducted at the Department of Periodontics, Sri Ramakrishna Dental College & Hospital, Coimbatore and at Sri Ramakrishna Hospital, Coimbatore Kidney Center, Coimbatore. This study includes 25 renal transplant patients, 25 hemodialysis patients and 25 pre dialysis patients compared with 20 healthy controls (C) of age ranging more than 35 years. The clinical parameters were assessed using Plaque Index (PlI), Gingival Index (GI), probing pocket depth (PPD), clinical attachment level (CAL), gingival bleeding index (GBI) and gingival overgrowth (GO). The statistical analysis was performed using one-way analysis

of variance (ANOVA), Tukey Kramer analysis, inferential statistics, Pearson correlation analysis and Post Hoc test was applied.

RESULTS: In this study, difference between the groups in Plaque Index (PlI), Gingival Index (GI), probing pocket depth (PPD), clinical attachment level (CAL), gingival bleeding index (GBI) and gingival overgrowth (GO) was statistical significant difference ($p<0.01$). Clinical changes show all independent variables increased in renal transplant group when compared to dialysis and pre dialysis patients. All variables were comparably low in control group. The mean value of clinical parameters although insignificant, CAL was statistically significant. So, there was a direct correlation between CRF and periodontal disease.

CONCLUSION: CRF patients are characterized by gingival and periodontal diseases. The dental community's awareness of the implication of poor oral health within this population should be elevated and daily oral health maintenance should be reinforced.

KEY WORDS:

Chronic periodontitis; gingival inflammation; periodontitis; renal transplant; dialysis; pre dialysis.

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LIST OF ABBREVIATIONS

ACR	:	Albumin to creatinine ratio
ANOVA	:	Analysis of variance
APD	:	Automated peritoneal dialysis
ARF	:	Acute renal failure
ARIC	:	Atherosclerosis Risk in Communities
AV	:	Arteriovenous
BOP	:	Bleeding on probing
BUN	:	Blood urea nitrogen
C	:	Control group
CAL	:	Clinical attachment level
CAPD	:	Continuous ambulatory peritoneal dialysis
CKD	:	Chronic kidney disease
CP	:	Chronic periodontitis
CPITN	:	Community Periodontal Index of Treatment Needs
CRF	:	Chronic renal failure
CTI	:	Connective tissue inflammation
CVD	:	Cardiovascular disease
D	:	Dialysis
DMF	:	Decayed missing filled index
EC	:	Ethical Committee
ESRD	:	End-stage renal disease
GBI	:	Gingival bleeding index
GCP	:	Generalized chronic periodontitis
GFR	:	Glomerular filtration rate
GI	:	Gingival Index
GO	:	Gingival overgrowth

GT	:	Gingival thickness
HD	:	Hemodialysis
HPT	:	Hyperparathyroidism
IRB	:	Institutional Review Board
IRMA	:	Immunoradiometric assay
K/DOQI	:	Kidney Disease Quality Outcome Initiative
LPA	:	Loss of periodontal attachment
MDRD	:	Modification of diet in renal disease
PCR	:	Polymerase chain reaction
PD	:	Predialysis
PDI	:	Periodontal destruction index
PlI	:	Plaque Index
PPD	:	Probing pocket depth
PT	:	Periodontal treatment
QOL	:	Quality of life
RT	:	Renal transplant
SAA	:	Serum amyloid A
S-OC	:	serum osteocalcin
U-DPD	:	Urinary deoxypyridinoline

INTRODUCTION

Chronic kidney disease (CKD) also known as chronic renal disease is a progressive loss in renal function over a period of months or years. It is caused by damage of the functional unit of the kidney, the nephron. Diseases which cause the destruction of nephrons are diabetes mellitus, pyelonephritis, glomerulonephritis, nephrosclerosis, polycystic kidney disease and vascular collagen disease.[1] CKD is defined by Kidney Disease Quality Outcome Initiative (K/DOQI) as kidney damage or glomerular filtration rate (GFR) < 60 ml/min/1.73m^2 for 3months or more, irrespective of cause (**Levey et al. 2005**). The normal adult GFR is approximately 100-120 ml/min/1.73m^2 body surface area.[2]

It is a worldwide major public health problem with its fast-growing number of patients with end stage renal disease and enhance in mortality and morbidity. The incidence of chronic renal failure (CRF) and end-stage renal disease (ESRD) is rising constantly because the repair of the damaged parenchymal tissues is rare (**Berthoux et al. 1999**). The end result of a reduced renal function is uremia, which is a metabolic state of toxicity, affecting many organs like gastrointestinal, cardiovascular and neuromuscular. The treatment of CRF includes dietary changes, correction of systemic complications and dialysis or renal transplant.

In the last 3-4 decade, improvement in dialysis and renal transplant has reduced morbidity and mortality among patients with ESRD. Because of increased life span, increasing number of such patients will focus on dental treatment. The incidence of a variety of soft and hard tissue conditions such as gingival inflammation,

gingival overgrowth, periodontal disease, enamel hypoplasia, pulp obliteration and osseous changes of the jaw seems greater among chronic renal failure patients.[3]

Chronic Periodontitis is an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss.[4] The primary etiological agent is predominantly gram negative anaerobic facultative bacteria within the subgingival biofilm, the majority of periodontal tissue destruction is caused by an inappropriate host response to those microorganisms and their products. The pathogenesis of periodontitis elicits the production of cytokines, prostaglandins and in some cases, acute phase reagents, such as C-reactive protein. Chronic inflammatory condition leading to the formation of periodontal pockets, destruction of deep collagenous structures of the periodontium and alveolar bone, excessive mobility of the teeth and their premature loss.[5]

Periodontal destruction is evident in children and adolescent predialysis CRF patients and periodontitis is further exacerbated during maintenance dialysis therapy. Moreover, the factors predisposing to periodontal disease and accelerating its progression are widespread in CRF. They encompass hyposalivation and xerostomia, impaired immunity and wound healing, alveolar bone destruction due to renal osteodystrophy, bleeding diathesis, diabetes mellitus, malnutrition and a state of general disability impairing oral hygiene.[6]

With this background, the present study is designed to find the influence of chronic renal disease on oral and periodontal condition.

AIMS & OBJECTIVES

AIM OF THE STUDY:

The aim of the study was to gain an insight of oral and periodontal finding in patients suffering from chronic renal failure.

OBJECTIVE OF THE STUDY:

- To evaluate the oral and periodontal status of chronic renal failure patients undergoing predialysis (PD), dialysis (D) and renal transplant (RT).
- Compare the relationship between oral and periodontal variables among chronic renal failure (CRF) cases and non renal disease controls.
- To assess gingival overgrowth in renal transplant patients.

REVIEW OF LITERATURE

With normal day-to-day variations in the intake of food and water, preservation of the internal environment requires the excretion of waste products from intestines, lungs, and skin contribute to the excretory capacity and balances the quantities ingested. The greatest responsibility for solute and water excretion is borne by the kidneys.

ANATOMY AND PHYSIOLOGY OF KIDNEY

The human kidneys are bean shaped organs located in the retroperitoneum at the level of the waist. Each adult kidney weighs approximately 160 g and measures 10 to 15 cm in length. Coronal sectioning of the kidney reveals two distinct regions: an outer region, or cortex, and an inner region known as the medulla. Structures that are located at the corticomedullary junction extend into the kidney hilum and are called papillae. Each papilla is enclosed by a minor calyx that collectively communicates with the major calyces to form the renal pelvis. The renal pelvis collects urine flowing from the papillae and passes it to the bladder via the ureter.[7]

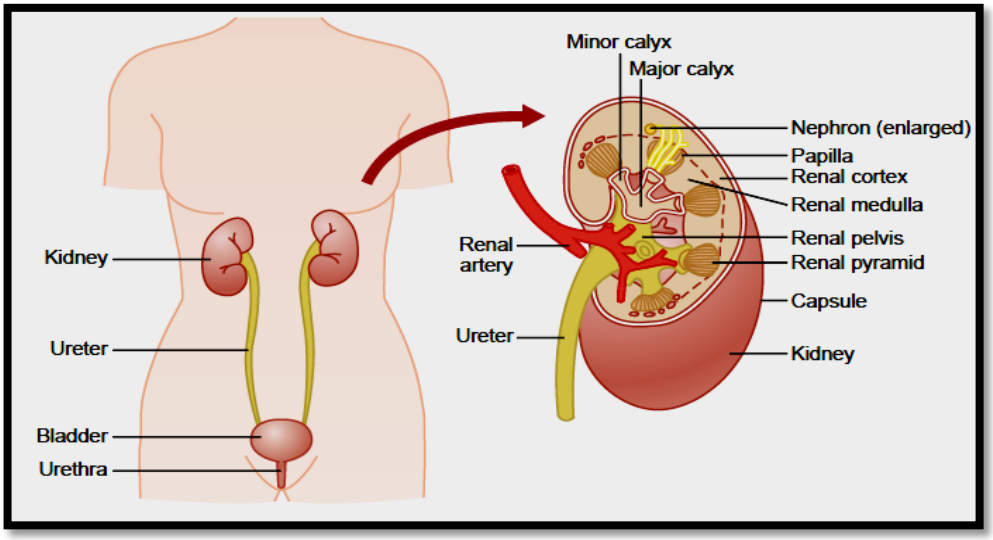


Fig. 1. Anatomy of the Kidney

Kidneys are one of the important organs of the body which are constituted from segmented nephrons, blood vessels, and filtering capillaries. Vascular flow to the kidneys is provided by the renal artery, which branches directly from the aorta. This artery subdivides into segmental branches to perfuse the upper, middle, and lower regions of the kidney. Further subdivisions account for the arteriole-capillary-venous network or vas recta. The venous drainage of the kidney is provided by a series of veins leading to the renal vein and ultimately to the inferior vena cava.[2]

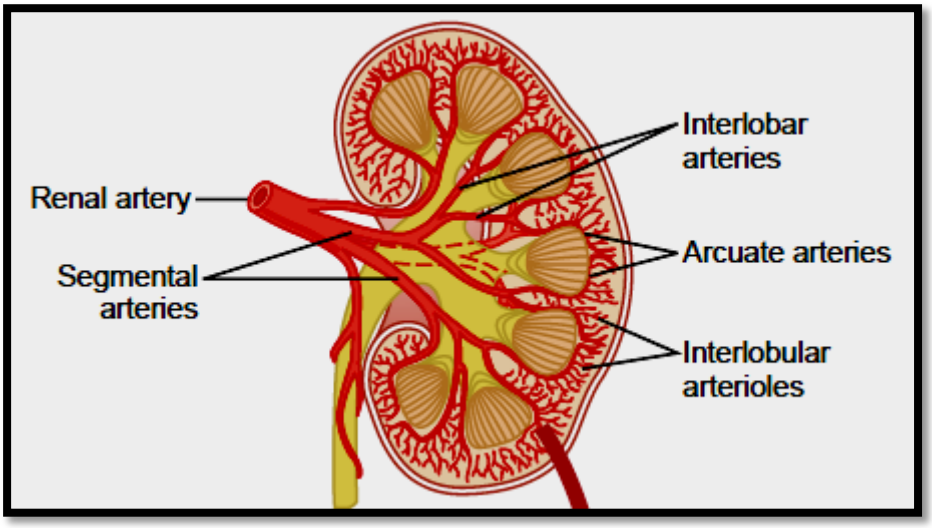


Fig. 2. Vascular supply to the Kidney

The nephrons are the functional unit of kidney. Each nephron is structured by afferent arteriole, bunch of glomerular capillaries named glomerulus enters into the dilated, blind end of the nephrons (Bowman's capsule) which filter large amounts of fluid from the blood and long tubule that converts the fluid to urine on the way to collecting duct, pelvis of the kidney efferent arteriole, proximal and distal tubules. The anatomy of the fenestrated endothelial cells in the glomerular capillary and epithelial cells in tubular systems are developed to filter the renal blood. Afferent

arteriole conducts blood to nephron and then it enters into a glomerular capillary for filtration of most amounts of fluid. The ratio of glomerular filtration rate (GFR) to renal plasma flow determines the filtration fraction.[8]

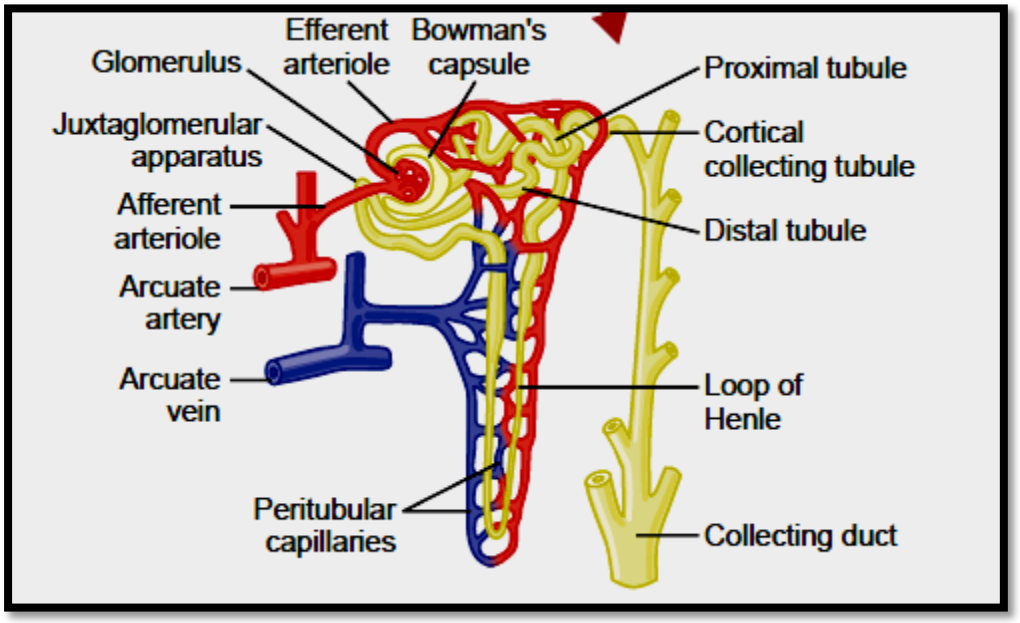


Fig. 4. Structure of the nephron

FUNCTION OF KIDNEY

The kidneys are vital organs for maintaining a stable internal environment (homeostasis). The kidneys have many functions, including regulating the acid-base and fluid-electrolyte balances of the body by filtering blood, selectively reabsorbing water and electrolytes, and excreting urine. In addition, the kidneys excrete metabolic waste products, including urea, creatinine and uric acid as well as foreign chemicals. Apart from these regulatory and excretory functions, the kidneys have a vital endocrine function, secreting renin, the active form of vitamin D and erythropoietin. These hormones are important in maintaining blood pressure, calcium metabolism

and the synthesis of erythrocytes, respectively. The most significant function is filtering plasma and eliminating substances and materials that are not needed for body. Indeed, the substances contain urea due to amino acids metabolism, creatinine from muscle creatine, uric acid from nucleic acids and products due to hemoglobin breakdown like bilirubin and metabolites of the hormones. Eventually, kidneys should remove these waste materials from the filtrate and blood and excrete approximately 1.5 to 2.5 L of urine per day.[8]

Under normal physiologic conditions, the kidneys serve several functions:[8]

- Non excretory functions
    - Degradation of polypeptide hormones
        - Insulin
        - Glucagon
        - Parathormone
        - Prolactin
        - Growth hormone
        - Antidiuretic hormone
        - Gastrin
        - Vasoactive intestinal polypeptide
    - Synthesis and activation of hormones
        - Erythropoietin (stimulates erythrocyte production by bone marrow)
        - Prostaglandins (vasodilators that act locally to prevent renal ischemia)

- Renin (important in regulation of blood pressure)
- 1,25-Dihydroxyvitamin D3 (final hydroxylation of vitamin D to its most potent form)

- Excretory functions
  - Excretion of nitrogenous end products of protein metabolism (eg. creatinine, uric acid, urea)
  - Maintenance of ECF volume and blood pressure by altering Na^+ excretion
  - Maintenance of plasma electrolyte concentration within normal range
  - Maintenance of plasma osmolality by altering water excretion
  - Maintenance of plasma pH by eliminating excess H^+ and regenerating HCO_3
  - Provision of route of excretion for most drugs

CHRONIC KIDNEY DISEASE

Chronic kidney disease (CKD) is a generalized term for a variety of chronic conditions that result in compromised kidney functions.[9] In a healthy body, the acid-base balance is maintained via buffers, breathing, and the amounts of acid or alkaline wastes in the urine this is because the daily load of endogenous acid is excreted into the urine with buffering compounds such as phosphates. When the kidneys are no longer able to maintain normal homeostasis it results in uremia caused by renal failure, retention of excretory products, and interference with endocrine and metabolic functions. As the GFR progressively decreases, the tubular excretory capacity for positive hydrogen (H^+) ions is overwhelmed because renal ammonia production

becomes inadequate. As the kidney deteriorates, metabolically derived acids accumulate results in acidosis leading to an increase in the anion gap.

Disorders of the kidneys can be classified into the following diseases or stages:[42]

1) Disorders of hydrogen ion concentration (pH) and electrolytes,
2) Acute renal failure (ARF),
3) Chronic renal failure (CRF),
4) End-stage renal failure or uremic syndrome.

Chronic Kidney disease has been defined according to the following criteria:[42]

1. Kidney damage for ≥ 3 months, as defined by structural or functional abnormalities of the kidney, with or without decreased Glomerular filtration rate (GFR), manifest by either, pathological abnormalities or markers of kidney damage, including abnormalities in composition of the blood or urine, or abnormalities in imaging tests.

2. GFR < 60 ml/ min/ 1.73 m^2 for ≥ 3 months, with or without kidney damage.

Table 1. Classification of kidney disease according to National Kidney Foundation According to National kidney foundation (2002) stages of chronic kidney disease

STAGE	DESCRIPTION	GRF (ml/ min/ 1.73 m^2)
1	**Kidney damage with normal or ↑ GFR**	**≥ 90**
2	**Kidney damage with mild ↓ GFR**	**60-89**
3	**Moderate ↓ GFR**	**30-59**
4	**Severe ↓ GFR**	**15-29**
5	**Kidney failure (ESRD)**	**< 15 (or dialysis)**

Stages 3 and 4 can also be characterized by uremia, but usually in lower levels than stage 5. Stage 5 is caused by any condition that could potentially destroy the nephrons. There are some diseases which cause the destruction of nephrons are diabetes mellitus, pyelonephritis, glomerulonephritis, nephrosclerosis, polycystic kidney disease and vascular collagen disease.[1]

EPIDEMIOLOGY OF CKD

Chronic kidney disease is a worldwide major public health problem with its fast-growing number of patients with end stage renal disease and enhance in mortality and morbidity. There are over one million dialysis patients worldwide, with an incidence of about a quarter of a million new patients each year. The incidence of patients with end-stage renal disease (ESRD) being treated by renal replacement therapy varies enormously depending on the level of affluence of the country.[11] The highly developed countries such as North America, Europe and Japan have the highest incident rates of treated end-stage renal disease, whereas the emerging countries have very low incident rates. The huge disparity in the prevalence of ESRD between the more and less developed countries probably due to the inadequacy of health care system, racial and ethnic differences. About 90% of treated ESRD patients come from more developed countries that can still afford the cost of renal replacement therapy.[12] In USA and Australia, the annual incidence of ESRD is significantly lower in whites than in African-Americans. The number of patients with ESRD probably underestimates the entire burden of the chronic kidney disease (CKD) because the number of subjects with early stage of the disease (stage 1 to 4) is likely to exceed by as much as fifty times those reaching ESRD.

In India, the prevalence of CKD was observed to be 17.2% with approximately 6% have CKD stage 3 or worse. It has been recently estimated that the age-adjusted incidence rate of ESRD to be 229 per million population (pmp), and >100,000 new patients who enter renal replacement programs annually.[13] It is a major health burdens in United State and 1,06,912 patients tolerated ESRD during 2005. The prevalence of end-2 stage renal disease is higher in Japan followed by Taiwan and USA. In USA, the rate of patients who need treatment is estimated to enhance up to 60% by 2010.[63] Premature death among people with CKD is three-fold higher than general population.

Coresh et al. (2005)[15] reported that 4.2% of individuals above 20 year-old had GFR 15 to 59 ml/min/1.73 m^2 in NHANESIII.

PATHOLOGY OF CKD

Type II Diabetic mellitus leads to diabetic nephropathy that is the most common cause of CKD and second cause being hypertensive nephropathy. The basis of nephrosclerosis caused by vascular disease is the same as which makes the coronary heart disease and cerebrovascular disease. Atherosclerotic vascular disease among the old patients is the manifestation of generalized vascular disease. The early stage of CKD is revealed by microalbuminuria and minor reduction in GFR which is a risk factor for CVD. The majority of this population does not show the last stages of CKD as they die due to cardiovascular and cerebrovascular diseases.[14]

Chronic inflammation has been proposed as one of the probable pathologies for CKD. Chronic systematic infection leads to elevated inflammatory markers level including C-reactive protein (CRP), interleukin-6, haptoglobin and fibrinogen.[16]Microalbuminuria is the early manifestation of renal damage due to

endothelial dysfunction before decrease in GFR which later leads to macroalbuminuria and more advanced kidney failure manifested by decreased GFR.[17]

INDICATORS OF CHRONIC KIDNEY DISEASE

Early stage of CKD manifested with microalbuminuria which later leads to macroalbuminuria. Albuminuria is indicated by urinary albumin-to-creatinine ratio (ACR) which is reported in mg/g. Microalbuminuria is defined as ACR > 30 mg/g to less than 300 mg/g and individuals with ACR ≥300mg/g are classified as having macroalbuminuria.

The recommendation of National Kidney Foundation and American Society of Nephrology is to use creatinine to estimate GFR (eGFR), within clinical settings. Among the young the normal range of GFR is 120-130 ml/min/1.73m² which decreases with age. CKD occurs with a GFR <60 ml/min/1.73m². The gold standard to determine the GFR in patients is to measure the clearance of insulin since it is not secreted by the renal tubules, but this remains an impractical test to perform clinically. Therefore, clinicians have relied on the measurement of serum creatinine as an indirect measure of the GFR and use creatinine-based formulae to calculate the GFR. There is a high degree of physiologic variability in GFR among normal individuals, thus making it difficult to define limits for normal GFR.[9]

Table 2. Normal range of laboratory test in chronic renal failure

Laboratory Test	Normal Range	Level in Symptomatic Renal Failure
Glomerular filtration rate	100–150 mL/min	< 6–10 mL/min
Creatinine clearance	85–125 mL/min (female)	10–50 mL/min (moderate failure)
	97–140 mL/min (male)	< 10 mL/min (severe failure)
Serum creatinine	0.6–1.20 mg/dL	> 5 mg/dL
Blood urea nitrogen	8–18 mg/dL	> 50 mg/dL
Serum calcium	8.5–10.5 mg/dL	Depressed
Serum phosphate	2.5–4.5 mg/dL	Elevated
Serum potassium	3.8–5.0 mEq/L	Elevated

EFFECT OF PERIODONTITIS ON CKD:

PATHOGENESIS*:*

Systemic inflammation is the proposed mechanism for the effect of periodontitis on the development of kidney disease. Cardiovascular diseases and CKD share same pathogenesis. Periodontal pathogens have the ability to adhere, invade and proliferate in coronary endothelial cells leading to atheroma formation and impaired vasculature relaxation. Host factors such as systemic diseases, genetic polymorphism or drug usage play a major role in the pathogenesis of periodontal disease by modifying the host response to periodontal infection or altering the susceptibility to infection by periodontal organisms. Chronic renal disease is associated with well-documented impairments in polymorphonuclear leucocyte (PMN) function. Both periodontitis and kidney diseases are associated with inflammatory markers such as C-reactive protein and chronic low level inflammation associated with periodontitis

may lead to endothelial dysfunction which plays a role in the pathogenesis of kidney disease.[18]The deleterious effects of systemic inflammation on kidney function could occur during the period of active periodontal infection and accumulate during the life time of the individual.[19]

ORAL MANIFESTATIONS OF CHRONIC RENAL FAILURE

Gingival Enlargement

Gingival diseases modified by medications are increasingly prevalent because of the increased use of drugs known to induce gingival enlargement (e.g immunosuppressive drugs such as cyclosporine, calcium channel blockers such as nifedipine, verapamil, diltiazem and sodiumvalproate). It creates difficulties in speech, mastication, tooth eruption, and aesthetic problems. Clinical and microscopic features of the enlargements caused by the different drugs are similar. The growth starts as a painless, beadlike enlargement. Gingival is mulberry shaped, firm, pale pink in color. It project from beneath the gingival margin, slowly increases in size 1-3 months after initiation and spontaneous disappears. The development and severity of gingival enlargement in response to medications are patient specific and may be influenced by uncontrolled plaque accumulation, as well as elevated hormonal levels.

(a) Immunosuppressants

i) Cyclosporine induced gingival enlargement

Cyclosporine is a potent immunosuppressive agent used to prevent organ transplant rejection and to treat several diseases of autoimmune origin. Clinical features of the enlargement starts as a painless, beadlike enlargement. Gingival is mulberry shaped, firm, pale pink in color. It project from beneath the gingival margin, slowly increases in size 1-3 months after initiation and spontaneous disappears.

Cyclosporine-induced gingival enlargement is more vascularized than phenytoin enlargement. Its mechanism of action is selectively and reversibly inhibits helper T cells, which play a role in cellular and humoral immune responses. Cyclosporine A is administered intravenously or by mouth and dosages greater than 500 mg/day to induce gingival overgrowth. Its occurrence varies from 25% to 70%. It affects children more frequently and its magnitude appears to be related more to the plasma concentration than patient's periodontal status. Gingival enlargement is greater in patients who are medicated with both cyclosporine and calcium channel blockers. In addition to gingival enlargement, cyclosporine induces other major side effects such as nephrotoxicity, hypertension, and hypertrichosis.

(ii) Tacrolimus

Tacrolimus is a macrolide molecule which has been shown to have major potential as an alternative immunosuppressant to cyclosporine. When cyclosporine is replaced by tacrolimus, it eliminates the upregulation of cyclosporine induced essential polypeptide growth factors which are the important mediator for gingival overgrowth.[20] Tacrolimus has been used effectively and is also nephrotoxic, but it results in much less severe hypertension, hypertrichosis, and gingival overgrowth. [21]

(b) Calcium Channel-blocker-induced Gingival Enlargement

Calcium channel blockers are prescribed to renal allograft recipients to reduce hypertension and cyclosporine-induced nephrotoxicity. They are nifedipine, amlodipine, diltiazem, verapamil, oxidipine, felodipine, and nitrendipine causing this gingival enlargement. They inhibit calcium ion influx across the cell membrane of heart and smooth muscle cells, blocking intracellular mobilization of calcium. This induces direct dilation of the coronary arteries and arterioles, improving oxygen

supply to the heart muscle. It also reduces hypertension by dilating the peripheral vasculature. Nifedipine is one of the most often used drug that induces gingival enlargement in 20% of patients.[22]The presence of dental plaque may predispose to nifedipine-induced gingival enlargement, but is not essential to its development. The dihydropyridine derivative, isradipine, can replace nifedipine in some cases and does not induce gingival overgrowth. Nifedipine is also used with cyclosporine in kidney transplant recipients, and the combined use of both drugs induces larger overgrowths.

Oral Hygiene and Periodontal Disease

Individuals receiving hemodialysis have poor oral hygiene, increased calculus and plaque deposits, premature tooth loss, localized suppurative osteomyelitis, secondary to periodontitis, attrition, recession, gingivitis and tooth mobility. 15% of 45 Virginian individuals receiving had a good standard of oral hygiene observed in individual receiving hemodialysis.[23]

Xerostomia

Xerostomia occurs in hemodialysis individuals due to restricted fluid intake, side effect of drug therapy and/or mouth-breathing, intake of alcohol containing mouth wash. Long-term xerostomia predispose to caries and gingival inflammation and give rise to difficulties with speech, denture retention, mastication, dysphagia, sore mouth, and dysgeusia infections such as candidosis and acute suppurative sialadenitis.[3]

Gingival bleeding

Dialysis patients have more bleeding on probing due to fibrinolysis defect at the level of plasminogen activation. The fibrinolysis defect in CRF deepens as renal function declines. The gingiva in individuals with CRF can be pale due to anemia.[24]

Oral Malodor/Bad Taste

Uremic patients have an ammonia-like oral odor also called as uremic odor occurs in one-third of individuals receiving hemodialysis and some patients complain of an unpleasant and/or metallic taste, or a sensation of an enlarged tongue.[25]

Mucosal Lesions

Individuals receiving dialysis have oral mucosal lesions, particularly white patches and/or ulceration, In particular, lichenplanus and oral hairy leukoplakia occurs as a consequence of the associated immunosuppression drugs. Uremic stomatitis may manifest as white, red, or grey pseudomembrane overlying painful erythema patches, while an ulcerative form is red with a 'pultaceous' covering. Oral mucosal macules and nodules also described in 14% of individuals receiving hemodialysis.[3]Oral hairy leukoplakia that occur secondary to drug-related immunosuppression although clinically and histopathologically similar, lesions lacking Epstein-Barr virus (EBV) have been observed with uremia. Other oral candidal lesions such as pseudomembranous (1.9%), erythematous (3.8%), and chronic atrophic candidosis (3.8%) also reported in immunosuppressed patients.

Dental Anomalies

Dental anomalies like delayed eruption of permanent teeth, enamel hypoplasia of the primary and permanent teeth, with or without brown discoloration can also occur, narrowing or calcification of the pulp chamber, dental caries observed in groups of patients with CRF.

Bone Lesions

Defects of calcium metabolism in CRF cause loss of hydroxylation of 1- hydroxycholecalciferol to active vitamin D (1,25- dihydroxycholecalciferol),

decreased hydrogen ion excretion (and resultant acidosis), hyperphosphatemia, hypocalcemia and resultant secondary hyperparathyroidism. Secondary hyperparathyroidism affects up to 92% of patients receiving hemodialysis. Hyperparathyroidism may present as a maxillary brown tumor, enlargement of the skeletal bases, or tooth mobility.

TREATMENT OF CHRONIC KIDNEY DISEASE

The treatment of CRF is often divided into

(1) Conservative therapy aimed at delaying progressive renal dysfunction(including dietary management)

(2) Renal replacement therapy (when conservative measures are no longer effective).

When the patient is diagnosed with CKD stage 5, the goals of treatment are to maintain quality of life, control disease progression and prevent further complications. At this stage, treatment focuses on dietary modification in an effort to decrease the retention of nitrogeneous waste products and control fluid and electrolyte imbalances. When conservative treatment fails and the number of functional nephrons is reduced to the point when the kidneys can no longer filter the blood and adequately remove nitrogen-containing compounds, either renal replacement therapy or renal transplantation is necessary.

Conservative therapy

An early symptom is hypertension, which untreated leads to hypertrophy in the glomerular and tubular structures which in turn cause increased filtration and rapid progression of the renal dysfunction. The disturbance causes retention of metabolites and alterations of the electrolyte and water balance. Increasing serum urea levels appear parallel to the uremic symptoms. Uremic toxicity partly occurs due to

malnutrition, acidosis, infections, inflammation, comorbidity, or psychological factors. Once the extent of renal impairment is established and reversible causes are excluded, medical management is devoted to the elimination of symptoms and the prevention of further deterioration. Initial conservative therapy is directed toward managing diet, fluid, electrolytes, and calcium-phosphate balance and toward the prevention and treatment of complications. Dietary regulation of protein (20 to 40 g/day) may improve acidosis, azotemia, and nausea. The restriction of protein reduces not only BUN levels but also potassium and phosphate intake and hydrogen ion production and also reduces the excretory load of the kidney, thereby reducing glomerular hyperfiltration, intraglomerular pressure, and secondary injury of nephrons. This restricted diet is often supplemented with multivitamins specific to the needs of the renal patient. Despite difficulties with hypertension, edema, and weight gain, salt and fluid excess and depletion must be avoided. Early renal insufficiency patients by restricting the intake of phosphate-containing foods and by supplementing the diet can prevent hyperphosphatemia.

Dialysis

The patient's blood is separated from the dialysis fluid (dialysate) by a membrane which allows water and toxins, but not blood cells, to pass out of the blood. There are two types of dialysis: hemodialysis and peritoneal dialysis (PD). Hemodialysis is the removal of nitrogenous and toxic products of metabolism from the blood by means of a hemodialyzer system. In peritoneal dialysis, the peritoneal membrane acts as the filter, whereas, in hemodialysis, the membrane is within the dialysis machine.[26] The dialysis system consists of a dialyzer, dialysate production unit, roller blood pump, heparin infusion pump and various devices to monitor the conductivity, temperature,

flow rate, and pressure of dialysate and to detect blood leaks and arterial and venous pressures Individuals commonly commence with peritoneal dialysis and may progress to hemodialysis, if renal function deteriorates further. In peritoneal dialysis, The Tenckhoff Silastic catheter has made peritoneal puncture for each dialysis unnecessary. The Tenckhoff catheter is a permanent intraperitoneal catheter that has two polyester felt cuffs into which tissue growth occurs. If used with a sterile technique, it permits virtually infection-free long-term access to the peritoneum. Most affected patients receive hemodialysis for up to 4 hrs, 2 or 3 times each week.[27] Disadvantage of hemodialysis, it increases the risk of viral transmission such as HIV and hepatitis B and C and is costly. There are three major types of vascular access for maintenance hemodialysis: primary arteriovenous (AV) fistula, synthetic AV graft, and double-lumen, cuffed tunneled catheters. Arteriovenous fistulae in the arm are required for regular vascular access via widebore needles. The fistula is usually fashioned from a native vein however, it is sometimes necessary to use animal or synthetic grafts if the local anatomy is unsuitable. Two of the benefits of peritoneal dialysis are that heparinization is unnecessary and that there is no risk of air embolism and blood leaks. It also allows a great deal of personal freedom; for this reason, it is often used as the primary therapy or as a temporary measure. These features, along with its simplicity, make peritoneal dialysis safe for patients who are at risk when hemodialysis is used. There are two types of peritoneal dialysis. Continuous ambulatory peritoneal dialysis (CAPD) requires 4 exchanges of approximately 2 liters throughout the day. An alternative method is automated peritoneal dialysis (APD), in which the dialysis fluid exchanges are carried out automatically by machine, during the hours of sleep (for 8-10 hrs).

Renal Transplantation

However, all the improvements in techniques of dialysis are in fact only temporary treatment forms until renal transplantation, which is the ideal mode of treatment. For chronic renal failure renal allografts may be cadaveric or from living donors, either related or non-related (although those from living relatives give rise to the best prognosis). Cadaveric organs are allocated on the basis of HLA tissue-typing, ABO compatibility, and the age and size of the donor and recipient. Although allograft failure causes rejection, adverse drug side-effects may also be a contributing factor.[26]Immunosuppressant therapy is required to minimize the risk of allograft rejection. Prednisolone, azathioprine, cyclosporin, and tacrolimus are the commonly used agents.

Schuller et al. (1973)[28]documented the periodontal status of allograft renal transplant individuals under immunosuppressive drug therapy mainly azathioprine (172.7mg) and cortisone [prednisone (24.5 mg)] for a duration of 4 to 60 months. Total number of patients examined in the study is 33 among them 26 male and 7 females who are selected from Toronto western hospital (Canada) compared with normal individuals without renal disease. Russell's periodontal index, oral hygiene index, prednisone and azathioprine dosage are assessed. Finally, no association exists between plaque and severity of periodontal disease. Low relationship exists between quantity of calculus and severity of periodontal disease. No statistically significant coefficients of correlation were obtained between the periodontal index and dosage of drugs, negative correlations for dosage and duration of prednisone.

Tollefsen et al. (1978)[29]compare the severity of gingival inflammation, in terms of the size of the cellular infiltrate in four categories of patients. First group is

healthy subjects (C), second group (CP) moderate accumulations of dental plaque, third group (UH) patients with uremia and in hemodialysis and fourth group (IS) had received renal allografts and was kept on an immunosuppressive regimen. Serum IgG, IgA,IgM are analysed from third group and fourth group. Gingival biopsies were obtained from each subject. The connective tissue inflammation (CTI) scores were compared between the groups. Inspite abundant local plaque accumulations, the UH group displayed essentially the same CTI scores as plaque-free controls (C), while the fourth group showed a significantly lower CTI score. The CTI scores of the CP group were significantly higher than those of the third group, fourth group and C groups.

Kardachi et al. (1978)[30]document the gingival condition of a group of patients who received renal transplants and were taking immunosuppressive drugs to combat rejection phenomena. 20 dentate patients who received kidney transplants at the Queen Elizabeth Hospital, Adelaide, were examined. They are compared with control group of subjects not undergoing any drug therapy. The group includes 14 males and 6 females with average age of 38.6 years. All members are receiving oral azathioprine and prednisone and at the time of the examination the average daily doses were 105 mg and 12 mg respectively for 1 to 10 years. 20 patients attending dental department [Royal Adelaide Hospital] selected as controls. This group includes 7 males and 13 females and their ages ranged from 16 to 73 years. Gingival Index (GI) and the Plaque Index (PI) are recorded. Finally the results suggest the mean PI of the renal transplant recipients slightly exceeded that of the control group, but the difference was not statistically significant. By contrast the mean GI of the transplant recipients was significantly lower than that of the control group.

Oshrain et al. (1979)[31] studied the host response of patients under immunosuppressant drug as well as corticosteroid therapy to periodontal treatment. Subjects included (a) 20 renal transplant patients, (b) 20 dialysis patients and (c) 20 healthy individuals. Plaque index (PI), gingival index (GI), pocket depth, gingival recession, gingival enlargement and periodontal destruction index (PDI) are recorded. The results suggest that all three groups exhibit the expected direct toxic effects of plaque antigens on periodontal tissues and hence GI and PDI, scores are similar. No significant differences among the three groups in plaque index, gingival index and periodontal destruction index. In the transplant and (to a lesser extent) dialysis groups, the degree of tissue response are dampened by the reduction in the numbers of immunocompetent cells and the subsequent paucity of complement generated components and lymphokines.

Been et al. (1982)[32] examined a prospective longitudinal study on periodontal status of 4 allograft renal transplant patients before and after 9 months. They also received immunosuppressive drugs as part of their therapy, beginning at the time of transplantation. 3 age matched hemodialysis patients and 6 patients free of systemic disease selected from eye clinic who were otherwise healthy selected as controls. Plaque index, pocket depths and gingival inflammation were assessed. The results suggest that the administration of the immunosuppressive drugs significantly reduces the level of gingival inflammation in the presence of high levels of bacterial plaque. No change in pocket depths either in the transplant patients or control. This study finally hypothesized that host inflammatory and immunological responsiveness to plaque bacteria is a primary factor in the pathogenesis of destructive periodontal disease in humans.

Nishikawa et al. (1991)[69] reported gingival hyperplasia in 2 patients treated with nifedipine for more than 3 months selected from Department of Periodontology Tokushima University, Tokushima, Japan. Clinical findings and hispathological features induced by nifedipine on gingival fibroblastic cells. In the first case, plaque control and surgical removal of the overgrowth of gingival tissues without discontinuing drug administration resulted in satisfactory progress, it suggests that the preexisting gingival inflammation was involved in the development of this periodontal disease. In the second case, change from nifedipine to thiazide resulted in spontaneous recovery, suggesting that the drug had induced the gingival hyperplasia. Study of these 2 cases suggests that both local inflammatory factors and long term administration of nifedipine were responsible for the gingival hyperplasia.

Yamalik et al. (1991)[70] reported the histological investigation of gingiva from patients with chronic renal failure (CRF) undergoing hemodialysis and renal transplant recipients (RTR) treated with immunosuppressive drugs and systemically healthy controls. Plaque index is similar in all groups, the gingival index was significantly less in RTR when compared to the other 2 groups. In light microscopic investigation the appearance of the connective tissue, mononuclear cell infiltration was similar in all of the groups. Number of inflammatory cells in patients with periodontitis was significantly higher than the other 2 groups. Desquamation like appearance in the superficial layers of the oral epithelium seen in patients with CRF. In electron microscopic investigation, fibroblasts and plasma cells with well-developed granular endoplasmic reticulum were found in connective tissue in RTR patients. In patients with CRF, epithelial cells presented swollen granular endoplasmic reticulum cisternae resembling vacuoles, indicating the presence of degeneration. It

was suggested that with the use of immunosuppressive drugs the response to bacterial plaque did not diminish completely.

Khocht et al. (1996)[33] compared periodontitis patient with chronic renal failure. Scaling and root planning along with apically positioned flap with osseous recontouring is done with 1-year follow up. The study reveals that periodontal infections in patients with depressed PMN function could still be managed successfully with standard periodontal treatment emphasizing plaque control. Conclusion of the study is periodontal management of CRF patients is to anticipate progression of renal disease and plan for continued periodontal care when the patient eventually reaches dialysis and/or transplantation stage. Patients on hemodialysis are systemically heparinized during treatment. To avoid potential bleeding problems, periodontal treatment should not be provided on the same day of dialysis treatment.

Westbrook et al. (1997)[34] evaluated the effect of nifedipine induced gingival hyperplasia (GH) of a switch to a dihydropyridine derivative with a low incidence of GH. 14 patients with nifedipine-induced GH were given a medical and periodontal examination. Probing depth (PD), gingival margin (GM), gingival thickness (GT), plaque index (PI), and gingival index (Gl) were assessed. Intraoral photographs, study models, and a gingival biopsy for histological examination were taken. Following baseline measures, patients were randomized to continued treatment with nifedipine or an equivalent dose of isradipine in a singleblind fashion. Biweekly periodontal parameters were taken for 8 weeks. At the end of 8 weeks, some patients elected to receive 4 weeks of open label isradipine therapy, with biweekly examination continuing through the open label phase. The isradipine treatment arm showed a mean decrease in PD. No other measured parameter (GM, GT, PI, GI) was significantly

changed, compared either to baseline or to the alternate treatment. Clinically, 60% of patients treated with isradipine exhibited a decrease in hyperplasia, while 66% of patients treated with nifedipine demonstrated an increase in hyperplasia. After 12 weeks, no patients treated with isradipine exhibited an increase in gingival overgrowth.

Naugle K et al. (1998)[23] determined the oral health status of individuals undergoing renal dialysis in southeastern Virginia. Three subgroups of the population were studied: 1) dialysis for less than a year; 2) dialysis for 1 to 3 years; and 3) dialysis for longer than 3 years. Three dental indices (Periodontal Disease Index; the Decayed, Missing, Filling Index; and the Simplified Oral Hygiene Index) were used to identify periodontal disease, dental caries activity, and oral hygiene status. Data were compiled and analyzed by using the parametric test, one way analysis of variance. Results suggested that 100% (n = 45) of the individuals undergoing renal dialysis presented with some form of periodontal disease. 45 individuals displayed severe gingivitis (28%) or early periodontitis (>36%). 64% of the sample displayed a high DMF Index, while 15% of the sample had excellent calculus score. Oral debris was present in 100% of the sample, resulting in a high Simplified Oral Hygiene Index score, suggesting an increased need for oral care instruction. Findings lead to the conclusion that the renal dialysis population is in need of comprehensive professional oral care and self-care instruction.

James et al. (2000)[71] examined short term effect of conversion of cyclosporine to tacrolimus in 4 renal transplant patients and management of gingival overgrowth induced by cyclosporine A. Gingival overgrowth scores, intraoaral photographs and alginate impression were taken prior to drug conversion and 6 to 9

months later. There was a reduction in overgrowth in the 4 renal transplant patients who switched from cyclosporine A to tacrolimus. One patient had complete regression. The author concluded that conversion of renal transplant patients with gingival overgrowth from cyclosporine A to tacrolimus may provide an effective management strategy for this clinical problem.

Thomas et al. (2001)[72] examined the medical and dental risk factors of renal transplant patients following administration of cyclosporine A and development of cyclosporine A induced gingival overgrowth. The 236 patients in this study comprised 152 males and 84 females with a mean age of 46 years and a mean duration of renal replacement therapy of 477 days. The patients medicated with both traditional oral cyclosporine A (*n* =220 individuals) and the new microemulsion form (*n*=229 individuals). Patients had either received cyclosporine A alone (*n*=45 individuals) or cyclosporine A and nifedipene (*n*=191 individuals. Gingival hyperplasia was present in 113 (48%) cases and the mean and maximum hyperplasia scores in affected individuals were 0.86 and 2.26. Pre and post transplant medical history and post-transplant renal function, i.e., serum creatinine levels, documented rejection episodes and glomerular filtration rates (GFR) gingival overgrowth was assessed. These data together with cyclosporineA serum levels and last-recorded dose of cyclosporineA, cyclosporineA microemulsion, nifedipene, azothioprine and prednisolone, were analysed. Results suggest that gingivitis and plaque were associated with hyperplasia. Duration of renal replacement therapy, age at transplantation, post transplant interval serum creatinine levels and documented rejection episodes were unrelated with the extent and severity of GH. GFR with last recorded doses of cyclosporineA and cyclosporine A microemulsion is significant.

Klassen et al. (2002)[3] reported a questionnaire and a noninvasive oral examination from hemodialysis and peritoneal dialysis patients registered in the dialysis program. 226 dialysis patients in central and northern Saskatchewan, 147 were interviewed and examined. Of these, 94 (64%) were dentate, and the same number had been on dialysis for a mean of more than 2 years; about a third were diabetic, almost all were hypertensive and all had non dental prosthetic devices or arteriovenous fistulae or both. 60 of the dentate patients were candidates for kidney transplantation. Most of the dentate patients reported brushing once or more daily, but they flossed infrequently or never. Dental visits were infrequent, less than every 5 years in 59 (63%) of the dentate patients. Findings in the dentate group included increased tooth mobility, fractures, erosion, attrition, recession, gingivitis and a high plaque index. A patient's dentist was contacted if the patient had seen him or her since starting dialysis (31 of the 94 dentate patients). Most (81%) of the dentists were aware that they were treating a dialysis patient. Medication records were incomplete for 29% of the patients, and only 2 (6%) of the patients had received antibiotic prophylaxis despite the fact that all had prosthetic devices or arteriovenous fistulae. Author concluded that the dental health of dialysis patients is poor and requires greater attention.

Frankenthal et al. (2002)[67] studied the effect of secondary HPT (hyperparathyroidism) on the periodontium of patients on hemodialysis. Experimental group consisted of 35 patients with secondary HPT, with chronic renal failure treated by hemodialysis (E group). Age and gender matched healthy control group (C group) are selected. Blood samples collected from E group, and the biologically active intact parathormone molecule, was assayed using two-site immunoradiometric assay

(IRMA). Clinical periodontal examination like Plaque index (PI), Gingival index (GI), Probing depth (PD) and Clinical attachment level (CAL) are recorded. A standardized panoramic X-ray was taken from all patients and computer-based linear measurements were used to assess alveolar bone loss. PI was also similar in the C and E group. GI is slightly greater in the C group compared to the E group. PD in the E group is almost identical to that of the C group. CAL in the E group did not differ from CAL in the C. It is concluded that secondary HPT does not have an appreciable effect on periodontal indices and radiographic bone height.

Wahadni et al. (2003)[35] examined periodontal disease and dental caries in individuals on renal dialysis in a Jordanian population. 47 individuals with a mean age of 42.9 years were examined for plaque deposits, gingivitis, periodontitis, and dental caries using the plaque index (Pl), gingival index (Gl), probing pocket depth (PPD), gingival recession and decayed, missing, or filling teeth (DMFT). Patients were categorized into three subgroups based on duration of renal dialysis as less than 1 year, 1 to 3 years and longer than 3 years. The results suggest no statistically significant differences in Pl, Gl, PPD, and gingival recession among the three subgroups examined. This study displayed an absence of gingival inflammation, DMFT index showed that there were statistically significant differences between subjects on renal dialysis for less than 1 year and subjects on dialysis for 1 to 3 years and more than 3 years.

Marakoglu et al. (2003)[1] evaluate the clinical response of 36 chronic renal failure patients receiving hemodialysis to existing microbial dental plaque. Gingival Index (GI) and Plaque Index (PlI) scores and probing depths (PD) were recorded. They are compared with 36 systemically healthy individuals matched with the patient

group based on age and extent of plaque accumulation. No statistically significant difference between the two groups. In hemodialysis patients, response is similar to existing bacterial plaque and their periodontal status when compared with control group. It is concluded that chronic renal failure does not seem to be an additional risk factor for more severe periodontal destruction.

Souza et al. (2005)[36] evaluated the periodontal status of 30 patients and the amount of salivary IgA in chronic renal dialysis patients. Plaque, calculus and gingival indices are assessed. PSR and IgA in saliva are analysed. Results demonstrated plaque, calculus and gingival indexes were high in the studied group. Despite a greater accumulation of plaque, IgA in dialysis patients indicates 1/3rd of that population have serum IgA concentrations below normal. Patients presenting chronic kidney disease disclosed a tendency for greater bacterial plaque concentration, high formation of dental calculus suggesting the need for periodontal treatment.

Davidovich et al. (2005)[6] describe the prevalence and severity oral diseases, and their relation to type, duration and severity of the renal disease, and their treatment modalities in a relatively large population of children, adolescents and young adults suffering from chronic renal failure. Four renal failure groups: chronic renal disease (n=22), undergoing dialysis (n=22), after dialysis and transplant (n=21) and after transplant (n=32), and a healthy control (n=38) were examined. Caries, enamel hypoplasia, pulp obliteration, Plaque Index, gingival bleeding, recession, overgrowth and index, probing depths, attachment loss, renal treatments and their relations with the oral variables were analysed. The renal failure groups had higher gingival index (GI) and bleeding on probing, probing depths and attachment loss, and

less caries, hypoplasia and obliteration than the control. Plaque was higher in the dialysis and pre-dialysis (PD) groups. Overgrowth was evident after transplant. The PD group showed lower GI than other renal groups. Dialysis duration and end-stage renal failure significantly correlated with gingivitis, probing depth, attachment loss and enamel hypoplasia. Immuran correlated positively with probing depth, gingival recession and attachment loss. Normiten and Nifedipine had positive correlations with gingival overgrowth.

Kshirsagar et al. (2005)[66] examined the cross sectional study to find the association in a general population sample from the Dental component (D-ARIC) of the Atherosclerosis Risk in Communities (ARIC) study. The study included 5,537 middle-aged black and white men and women. Periodontitis patients were categorized as healthy/gingivitis, initial, and severe. 2,276 individuals had initial periodontitis, and 947 individuals had severe periodontal disease. 110 individuals (2%) had renal insufficiency. Healthy/gingivitis, initial and severe periodontal disease were associated with a GFR less than 60 mL/min/1.73 after adjustment for important risk factors for CVD and CKD. Initial and severe periodontitis were associated with an elevated serum creatinine level.

Bots CP et al. (2006)[37] made a cross-sectional study and compared the oral health status of chronic renal failure patients (CRF) on renal replacement therapy with a matched population. 42 dentate CRF patients aged 25-54 years were matched with control group of 808 dentate subjects. The oral health status is assessed using decayed missing filling (DMF) index, pocket depth, bleeding on probing and presence of calculus. In CRF patients, calculus is significantly higher than in controls. The self-

reported oral health questionnaire revealed increased TMJ problems, malodour in CRF patients than controls.

Boraswki et al. (2007)[41] compared the periodontal status of 3 groups of adult CKD patients: (i) undergoing maintenance HD (ii) treated with continuous ambulatory peritoneal dialysis (CAPD) and (iii) pre-dialysis CKD patients from north eastern Poland. 106 patients are enrolled among them 35 on HD with mean age of 56 years, 33 on CAPD with mean age 51 years and 38 pre-dialysis CKD stage 2–5 with mean age 51 years. 2 control groups comprised 26 generally healthy individuals with advanced periodontitis and 30 subjects from general population. Gingival Index (GI), papillary bleeding index (PBI), Plaque Index (PI), loss of clinical attachment level (CAL) and community periodontal index of treatment needs (CPITN) were examined. The PI values were higher in HD, CAPD and periodontitis patients than in general population subjects. The GI and PBI also were uniformly higher in HD patients than in CAPD were highest in HD patients comparably to patients with periodontitis, and exceeded the values found in CAPD, pre-dialysis CKD and general population subjects. CPITN is higher in periodontal disease patients and controls when compared with each of the renal failure.

Yoshihara et al. (2007)[38]investigated relationship between periodontal disease and chronic renal function in community dwelling elderly Japanese subjects. 145 subjects with mean age of 77 years participated in this study. A periodontal examination and urine was collected over 24 hours and morning blood samples were taken. Serum creatinine clearance, volume of creatinine, volume of urine per 24 hours was used as blood and urinary markers of kidney function. Biochemical parameters of bone turnover were measured: urinary deoxypyridinoline (U-DPD) as a bone

resorption marker and serum osteocalcin (S-OC) as a bone formation marker. The number of remaining teeth, smoking habit, gender, use of interdental brushes or dental floss, volume of urine per 24 hours, and creatinine clearance per 24 hours were independent variables in the first test. In addition, the number of remaining teeth, smoking habits, gender, use of interdental brushes or dental floss, U-DPD, and S-OC were independent variables in the second test. Creatinine clearance per 24 hours and S-OC were significantly associated with % ≥6-mm CAL per person. The % ≥6-mm CAL was significantly associated with renal function and bone metabolism markers.

Bayraktar et al. (2007)[39] compared the periodontal and dental health status of patients on hemodialysis (HD) with healthy controls (C). 76 HD patients and 61 controls were examined for plaque deposits, gingivitis, periodontitis, calculus accumulation and oral health status. There was no statistically significant difference in the measurement of probing pocket depths (PPD) in HD and C groups, but a highly significant difference was found for plaque index, gingival index (GI) and calculus surface index. There was a highly significant difference for GI and PPD scores between subgroups receiving HD for < 3 years or more. A positive correlation between time on dialysis and parameter of missing teeth, GI scores and measurement of PPD was found in the HD group. Decayed, missing and filled teeth index scores were higher in the controls than the HD group, with no statistical significance. The dental and periodontal health status of HD patients is comparable with healthy controls, but becomes worse with time on dialysis. Thus, oral health maintenance is of utmost importance in this patient group.

Shultis et al. (2007)[40] investigate the effect of periodontitis on development of overt nephropathy and end-stage renal disease (ESRD) in type 2 diabetes.

≥ 25 years individuals residing in the Gila River Indian Community with type 2 diabetes, one or more periodontal examination, estimated glomerular filtration rate ≥60 ml/min/1.73 m² and no macroalbuminuria (urinary albumin-to-creatinine ratio ≥300 mg/g) were identified. Of the 529 individuals, 107 (20%) had none/mild periodontitis, 200 (38%) had moderate periodontitis, 117 (22%) had severe periodontitis, and 105 (20%) were edentulous at baseline. During follow-up of up to 22 years, 193 individuals developed macroalbuminuria and 68 developed ESRD. After adjustment for age, sex, diabetes duration, BMI, and smoking in a proportional hazards model, the incidences of macroalbuminuria were 2.0, 2.1, and 2.6 times as high in individuals with moderate or severe periodontitis or those who were edentulous, respectively, compared with those with none/mild periodontitis. Incidences of ESRD in individuals with moderate or severe periodontitis or in those who were edentulous were 2.3, 3.5, and 4.9 times as high, respectively, compared with those with none/mild periodontitis.

Castillo et al. (2007)[42] in a cross-sectional study evaluated the periodontal status and oral microbiological patterns in population with end-stage renal disease (ESRD), undergoing haemodialysis (HD). This study involves 52 patients from the Nephrology Department and 52 matched control subjects. The subjects had a periodontal clinical examination, subgingival plaque samples were taken and analysed using a semiquantitative polymerase chain reaction (PCR) test to detect Porphyromas gingivalis, Tannerella forsythia, Prevotella intermedia, Prevotella nigrescens and Actinobacillus actinomycetemcomitans. Subgingival plaque and saliva samples were studied for Candida and Enterobacteriaceae. Outcome measures of 104 subjects had loss of periodontal attachment (LPA) ≥ 3 mm. Only 13 subjects (12.5%) presented

good periodontal health. No statistically significant differences were found between the HD patients and the control group regarding bleeding index, number of teeth or percentage of LPA ≥ 3 mm. HD patients presented a higher number of periodontopathic microorganisms than the matched control group, a prolonged duration of HD did not bear a statistically significant relationship with the percentage of sites with LPA ≥ 3 mm, specific microbiota or composition of biofilm.

Kshirsagar AV et al. (2007)[43] in a case control study, observed an association of severe periodontal disease and hypoalbuminemia in a group of patients who were receiving long term hemodialysis. The relationship between periodontitis and measures of systemic inflammation, serum albumin and C-reactive protein (CRP), were examined. 6 sites per tooth (up to 32 teeth per patient) were examined. A total of 154 patients mean age was 54.6 years completed the study. The average duration of dialysis was 4.0 years. 86 (54.6%) were men, and 89 (58.2%) were black. Common causes of end-stage kidney disease were hypertension (12.3%), diabetes (22.1%), glomerulonephritis (7.1%) and other (58.4%). 35 (23%) patients were periodontitis cases. Severe periodontitis was associated with low serum albumin compared with individuals without severe periodontitis disease after adjustment for age, gender, race, diabetes, hypertension, body mass index, smoking, study site, total cholesterol, serum calcium, serum phosphorus, and normalized protein catabolic rate. Increased levels of plaque have been reported for hemodialysis (HD) populations. There was no observed association of severe periodontitis with CRP.

Fisher et al. (2007)[44] conducted a cross-sectional, retrospective design study in 12,947 subjects (6922 female) with >18 years of age were enrolled in the study. Clinical (PPD, CAL and BOP) and microbiological periodontal examination and

laboratory tests were analysed. Chronic kidney disease prevalence was 3.6%, periodontal disease prevalence was 6.0%, and edentulism prevalence was 10.5%. Adults with periodontal disease and edentulous adults were twice as likely to have chronic kidney disease after simultaneously adjusting for other traditional and nontraditional risk factors.

Fisher et al. (2008)[45]suggested periodontal infection contributes to chronic kidney disease in United States population. 4,053 adults ±40 years of age were included. Clinical measures and serologic markers of periodontal infection were investigated. 9% of the study population had chronic kidney disease, 22% had high A. actinomycetemcomitans antibody titer, 24%had high P. gingivalis antibody titer, 9% had periodontal disease and 17% were edentulous. After simultaneously adjusting for recognized risk factors, adults with a high A. actinomycetemcomitans titer were less likely to have chronic kidney disease and adults with edentulism were more likely to have chronic kidney disease. These results support considering edentulism and low serum titer to A.actinomycetemcomitans as risk indicators for chronic kidney disease.

Joseph et al. (2009)[46] assessed the prevalence of periodontal disease among a group of patients with renal disease and compared their periodontal status to that of healthy controls. 77 patients with different forms of renal disease and 77 healthy controls were examined for oral hygiene status, gingival inflammation, probing pocket depth and clinical attachment loss. The subjects were grouped into three as no/mild, moderate and severe periodontitis. All periodontal parameters were significantly elevated in the case group as compared to controls ($p < 0.001$).

Nadeem et al. (2009)[47] investigate the association of periodontal disease with increased systemic inflammation reflected by CRP values, in patients with ESRD on

maintenance haemodialysis (HD) or peritoneal dialysis (PD). 80 patients with mean age of subjects was 50.3±9.06 years with a median time on dialysis therapy of 24 months who were in ESRD were included in the study. 42.5% subjects were male and 62% subjects reported their race as coloured. Plaque index (PlI) gingival index (GI), bleeding on probing (BoP), probing depths (PD) and clinical attachment loss (CAL) were assessed. 57.5% subjects were diagnosed to have periodontal disease, of these 52.2% had elevated CRP levels. 34 subjects with a healthy periodontium, only 29.4% had elevated CRP levels. The results of the study showed significantly elevated levels of CRP in ESRD patients with periodontitis.

A study by **Graziani et al. (2010)**[48] examined the effect of non-surgical periodontal treatment (PT) in subjects with generalized chronic periodontitis (GCP). 20 GCP systemically healthy subjects were treated with PT. Serum samples were collected at baseline, 1 day, 7, 30, 90 and 180 days after treatment. GFR was evaluated using cystatin C, a serum marker and modification of diet in renal disease (MDRD), an equation involving creatinine, urea and albumin. Serum markers of systemic inflammation such as C-reactive protein (CRP), D-dimer, serum amyloid A (SAA) and fibrinogen were also assessed. The cystatin C level decreased ignificantly from baseline to the end of the trial. Conversely, MDRD did not vary. Greater increases were noted for CRP and SAA within 24 h, while D-dimer and fibrinogen showed mild variations. The values of inflammatory markers were normalized after 30 days.

Artese et al. (2010)[49] hypothesised CKD predialysis patients with periodontitis would respond poorly to periodontal treatment owing to immunologic compromise. 21 predialysis patients (group 1) and 19 individuals without clinical

evidence of kidney disease (group 2) with chronic periodontitis were subjected to non-surgical periodontal treatment with no antibiotics. Clinical periodontal and systemic parameters were evaluated at baseline and 3 months after treatment. Both groups showed significant and similar post-treatment improvements in all periodontal parameters examined. Periodontal treatment had positive effect on the glomerular filtration rate of each individual. Results indicate that chronic periodontitis in predialysis kidney disease patients improved similarly in patients with chronic periodontitis without CKD after receiving non-surgical periodontal therapy. This study demonstrates that CKD predialysis patients show a good response to non-surgical periodontal treatment.

Iwasaki et al. (2011)[50] made an adjusted multivariate regression model (for serum creatinine level, mean GFR, sex, lower income, lower education, status of visits to a dentist, high alcohol consumption, smoking, proteinuria, hyperglycaemia, hypertension, hypercholesterolemia, hypertriglyceridemia, low HDL cholesterol level and obesity). Subjects were classified according to the amount of inflamed periodontal tissue. Results of the pooled analysis indicated a statistically significant risk of CKD for subjects with periodontitis.

Grubbs et al. (2011)[19]made a cross-sectional study analysis of 2001 to 2004 National Health and Nutrition Examination Survey data in U.S. population. These analyses included 6199 dentate adult participants (aged 21 to 75 years) with periodontal exams. The estimated prevalences of moderate/severe periodontal disease and CKD were 5.3% and 10.6%, respectively. Periodontal disease was associated with >2 fold higher risk of CKD that was moderately attenuated after adjustment for age, gender, race/ethnicity, tobacco use, hypertension, diabetes, educational

attainment, poverty index ratio, and dental care use. There were no statistically significant interactions between periodontal disease and race/ethnicity, educational attainment, or poverty status.

Anees et al. (2011)[51] conducted on patient on maintenance hemodialysis for more than 3 months at 3 dialysis centers of Lahore. 50 healthy individuals were included as controls from among the patients caregivers. 89 patients (71.2%) were men, 99 (79.2%) were married, 75 (60.0%) were older than 45 years, and 77 (61.6%) were on dialysis for more than 8 months. Patients on hemodialysis had a poorer QOL as compared to their caregivers in all domains except for domain 4 (environment). There was no difference in the QOL between the three dialysis centers of the study, except for domain 3 (social relationship) of the patients at Mayo Hospital (a public hospital), which was significantly better. Nondiabetic patients had a better QOL in domain 1 (physical health) as compared to diabetic patients. Duration of dialysis had a reverse correlation with the overall QOL.

Brotto et al. (2011)[52] made a cross sectional case control study and found the association between periodontitis and renal insufficiency by assaying kidney disease markers. Variables used to diagnose periodontitis were: (i) probing pocket depth (PPD), (ii) attachment loss (CAL), (iii) bleeding on probing (BOP) (iv) Plaque Index (PI) and (v) extent and severity index. Blood and urine were collected from 60 apparently healthy non-smokers (men and women), consisting of a test group of 30 subjects with periodontitis (age 46±6 yrs) and a control group of 30 healthy subjects (age 43±5 yrs). Kidney function markers (urea, creatinine, uric acid and albumin contents) were measured in the serum and urine. Also, the glomerular filtration rate was estimated from creatinine clearance, albumin: creatinine ratio in a 24–h sample of

urine. Results was found that the control group had a greater mean number of teeth than the test group and that the two groups also differed in PPD, CAL, BOP and PI, all these variables being higher in the test group. For the extent and severity index of both PPD and CAL, the test group had much higher medians of both extent and severity than the control group. With regard to kidney function, none of the markers revealed a significant difference between the control and test groups and all measured values fell within the reference intervals. It is proposed that severe periodontitis is not associated with any alteration in kidney function.

Vilela et al. (2011)[53] reported an interventional, non-randomized controlled trial to determine the impact of periodontal therapy on level of serum prohepcidin (the prohormone of hepcidin) and inflammatory markers in chronic kidney disease patients and correlates in patients with chronic periodontitis. This study included 56 chronic periodontitis patients, 36 with chronic kidney disease and 20 without systemic diseases and with normal renal function (control group). The inflammatory markers ultrasensitive C-reactive protein, interleukin-6, and prohepcidin were evaluated before and 3 months after periodontal treatment. Periodontal treatment resulted in significant reductions in ultrasensitive C-reactive protein, interleukin-6 and serum prohepcidin levels in both the groups.

Brito et al. (2012)[54] made a cross sectional study and determine the extent and severity of periodontitis in chronic kidney disease patients undergoing the following three different treatment modalities: predialysis, continuous ambulatory peritoneal dialysis (CAPD), and hemodialysis (HD) and to compare the findings with those from systemically healthy individuals. A total of 198 individuals among them 40 CAPD patients, 40 HD patients, 51 predialysis patients and 67 healthy individuals were

examined. The periodontal examination included plaque index, probing pocket depth, clinical attachment loss and bleeding on probing. Patients with at least 4 sites with clinical attachment loss ≥ 6 mm were considered to have severe chronic periodontitis, and those with > 30% of sites with clinical attachment loss ≥ 4 mm were considered to have generalized chronic periodontitis. Predialysis and HD patients had significantly more sites with clinical attachment loss ≥ 6 mm than healthy individuals. The CAPD patients had generalized chronic periodontitis similar to healthy subjects. There were significantly more cases of severe chronic periodontitis in predialysis and HD patients.

Manjunath et al. (2013)[55] studied the influence of periodontal health on renal dialysis patients. 234 patients undergoing renal dialysis among them 60 % male and 40 % female were included in the study. Periodontal disease status was measured by CPITN (Community Periodontal Index of Treatment Needs). Other parameters includes age, sex occupation, family history, history of other systemic diseases, medication taken, number of dialysis undergone, type of dialysis, history of renal transplant, personal history included oral hygiene status and loss of teeth was assessed. The author concluded that the prevalence of moderate to severe gingivitis and moderate periodontitis are seen in renal dialysis of patients. The population needs comprehensive oral and periodontal care.

MATERIALS & METHODS

STUDY DESIGN AND PATIENT SELECTION

This study was designed as a multicenter, cross sectional, prospective, parallel design study conducted over a period of 3 months. Test group were selected in the Department of Nephrology, Sri Ramakrishna Hospital and Coimbatore Kidney Center, Coimbatore and controls obtain from Department of Periodontology, Sri Ramakrishna Dental College and Hospital, Coimbatore. The ethical clearance was obtained from the institutional ethical committee. The study included 75 cases of chronic renal failure patients compared with 20 healthy individuals who had no systemic disease.

STUDY ETHICS AND SAFETY:

This clinical study followed the principles in the Declaration of Helsinki. Approval from the institutional Review Board (IRB) and Ethical Committee (EC) of Sri Ramakrishna Dental College and Hospital was obtained prior to implementation. The subjects enrolled in this study had to satisfy the following criteria,

CRITERIA OF INCLUSION:

Test group with chronic renal failure patients:

- Undergoing dialysis
- Predialysis
- Renal transplant patients

Control group:

- Patients received treatment in dental hospital

- No systemic disease

STUDY GROUPS:

After considering the inclusion and exclusion criterias, 75 patients in the test group were further divided into three groups based on their treatment.

1. Group-I comprised of 25 patients who were on predialysis.
2. Group-II comprised of 25 patients who were on dialysis.
3. Group-III consists of 25 patients who underwent renal transplant.

20 patients were included in the control group.

ARMAMENTARIUM:

DIAGNOSTIC INSTRUMENTS:

1. Dental mouth mirror
2. William's periodontal probe
3. Dental Explorer
4. Tweezer
5. Sterile cotton pellets
6. Surgical mask & Head cap
7. A pair of examination gloves

CLINICAL EXAMINATION:

On every examination, the following clinical parameters were scored:

i) **Plaque Index (PlI):** Full mouth plaque score was recorded by using Plaque Index given by Silness and Loe (1964).[56]

ii) **Gingival index (GI):** Full mouth gingival score was recorded by using Gingival Index given by Loe and Silness (1963).[57]

iii) **Gingival bleeding index (GBI):** Full mouth gingival bleeding score was recorded by using Gingival bleeding index given by Muhlemann & Son (1971).[58]

iv) **Periodontal pocket depth**:

Conventional probing depth: The probing pocket depth was assessed in each tooth from the gingival margin to the base of the sulcus using Williams periodontal probe at six specific sites.[59]

1. Distofacial line angle to the midline of distal surface
2. Facial surface
3. Mesiofacial line angle to the midline of mesial surface
4. Distolingual line angle to the midline of distal surface
5. Lingual surface
6. Mesiolingual line angle to the midline of mesial surface

v) **Clinical attachment level:** From the cemento enamel junction (CEJ) to the base of the pocket in all the 6 sites using Williams periodontal probe as mentioned for probing depth.[60]

1. Gingival margin located on the anatomic crown- subtracting the depth of pocket from the gingival margin to cementoenamel junction

2. Gingival margin coincides with cementoenamel junction the loss of attachment equals to the pocket depth

3. Gingival margin located apical to cementoenamel junction the distance between the cementoenamel junction and gingival margin should be added to the pocket depth

vi) Gingival overgrowth: (GO)

Gingival overgrowth is scored dichotomously, as either present or absent. For each tooth, a score of 1 was given, if GO was present at the clinical examination, and if GO was absent the score was 0. The average GO score per subject was calculated based on the number of sites exhibiting GO and the total number of sites evaluated.[61]

vii) Radiographic assessment: The radiographic evidence of bone loss was determined if the distance from CEJ to the alveolar crest was > 2mm. [62]

DEPARTMENT OF PERIODONTICS AND ORAL IMPLANTOLOGY

ORAL AND PERIODONTAL STATUS IN PATIENTS SUFFERING FROM RENAL FAILURE

PROFORMA

FORM I - SCREENING PROFORMA

NAME:

AGE: SEX:

POSTAL ADDRESS:

TELEPHONE NUMBER:

OCCUPATION:

CRITERIA OF INCLUSION:

SELECTION OF CHRONIC RENAL FAILURE PATIENTS :

- Number of patients undergoing dialysis : 25
- Number of predialysis patients : 25
- Number of renal transplant patients : 25
- Age of the patient : >35 years

SELECTION OF CONTROLS :

- Number of subjects : 20
- Age group > 35 years
- No systemic disease

FORM II- HISTORY PROFORMA

Chief complaint with duration:

	Absent	**Present**
1. Bleeding gums	☐	☐
2. Bad breath	☐	☐
3. Pain in gums	☐	☐
4. Swollen gums	☐	☐
5. Pus discharge from gums	☐	☐
6. Mobility	☐	☐
7. Hypersensitivity	☐	☐

8. Any other complaint's (Specify):

PERSONAL HISTORY:

1. Brushing habit:

2. Smoking: Yes ☐ No ☐

3. Drug history :

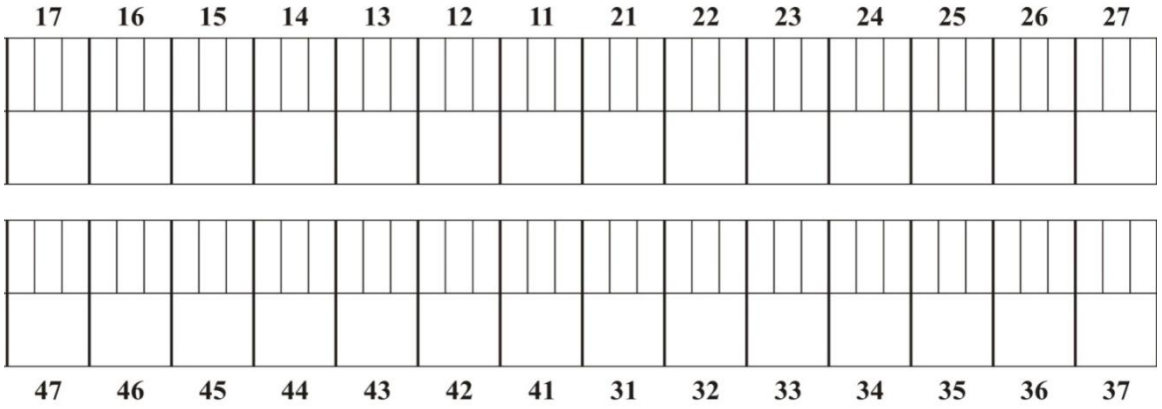
Dosage :

Duration :

FORM III- CLINICAL ASSESSMENT

DATE: _____

1. **PLAQUE INDEX (SILNESS AND LOE 1964):**



Calculation : $\frac{\text{Sum of score of each teeth}}{\text{Total number of teeth examined}}$

Inference: ----------

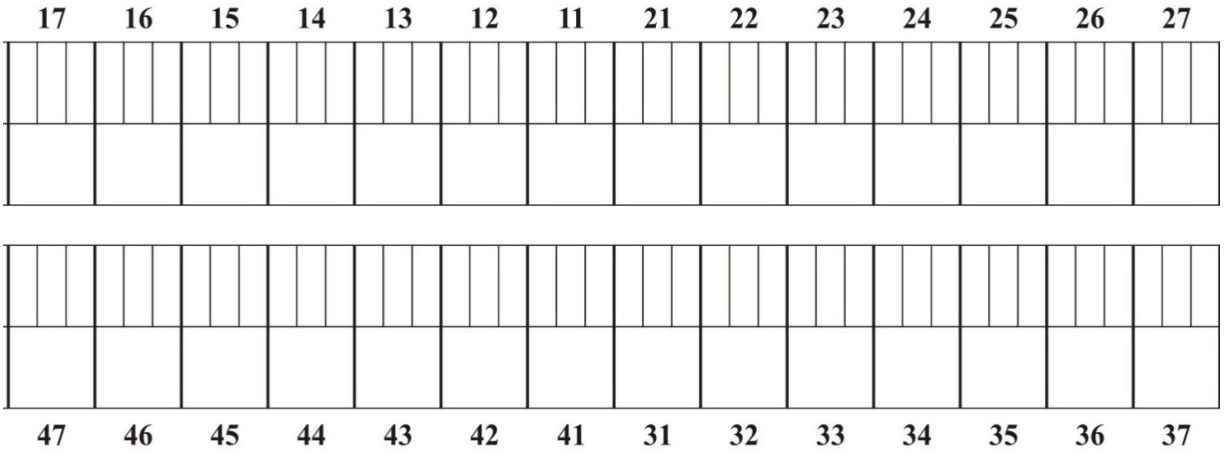
Excellent: 0

Good: 0.1 – 0.9

Fair: 1.0 – 1.9

Poor: 2.0-3.0

2. GINGIVAL INDEX: (LOE & SILNESS 1963)



Calculation : $\frac{\text{Sum of score of each teeth}}{\text{Total number of teeth examined}}$

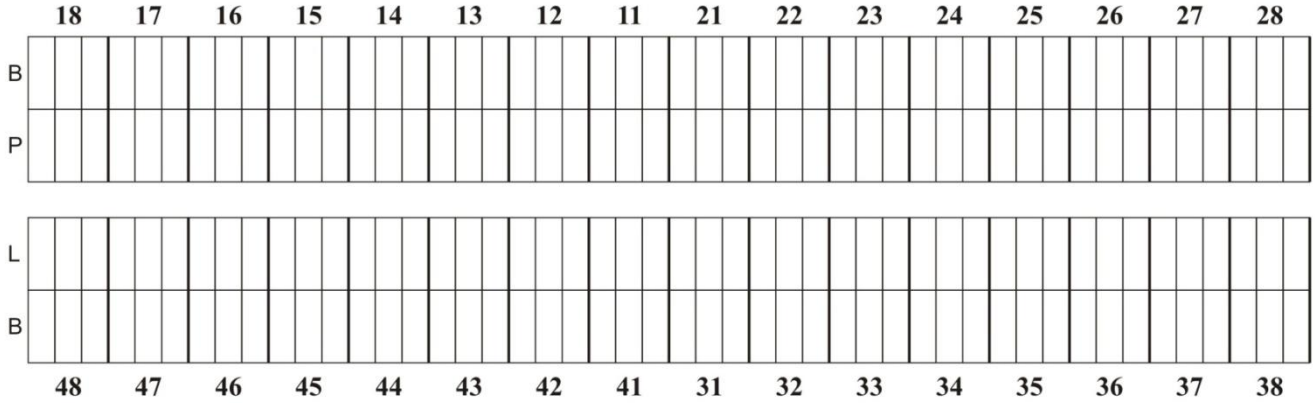
Inference : ---------- Mild gingivitis: 0.1 - 1.0

Moderate gingivitis: 1.1 – 2.0

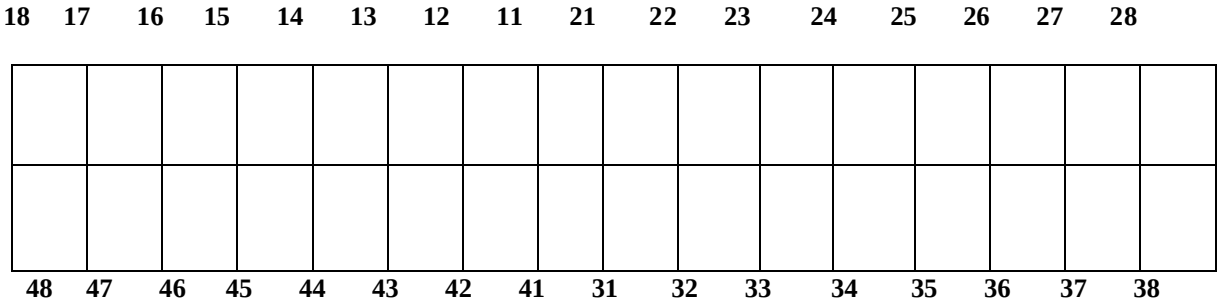
Severe gingivitis: 2.1 – 3.0

3. PERIODONTAL STATUS

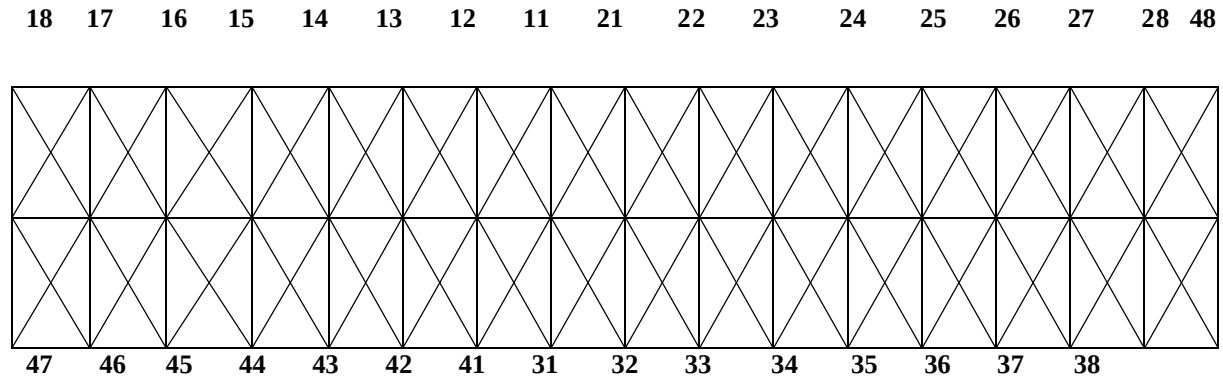
a) PROBING DEPTH (CONVENTIONAL PROBING METHOD)



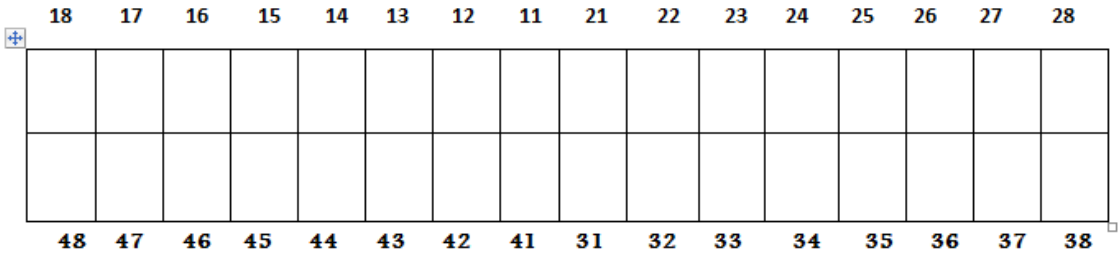
b) CLINICAL ATTACHMENT LEVEL



c) GINGIVAL BLEEDING INDEX (GBI) (MUHLEMANN & SON 1971)



d) GINGIVAL OVERGROWTH



e) LABORATORY DATA IN THE CRF SUBGROUPS

Creatinine	**:**	**mg/dL**
Blood urea	**:**	**mg/dL**
Calcium	**:**	**mg/dL**
Albumin	**:**	**g/dL**
Phosphate	**:**	**mEq/dL**
Alkaline phosphatase	**:**	**IU/L**
Uric acid	**:**	**mg/dL**
Hemoglobin	**:**	**g/dL**
Bicarbonate	**:**	**mEq/dL**
Potassium	**:**	**mEq/dL**
Sodium	**:**	**mEq/dL**

f) RADIOGRAPHIC ANALYSIS :

FORM V

CONSENT FORM

CERTIFICATE BY INVESTIGATOR

I certify that I have disclosed all details about the study in the terms easily understood by the patient.

Dated: _____ **Signature: _____**

Name: _____

CONSENT BY SUBJECT

I have been informed to my satisfaction, by the attending physician, the purpose of the clinical trial and the nature of treatment and follow up including the laboratory investigation to be performed to monitor and safe guard my body functions.

Dated: _____ **Signature or thumb impression: _____**

FIGURES

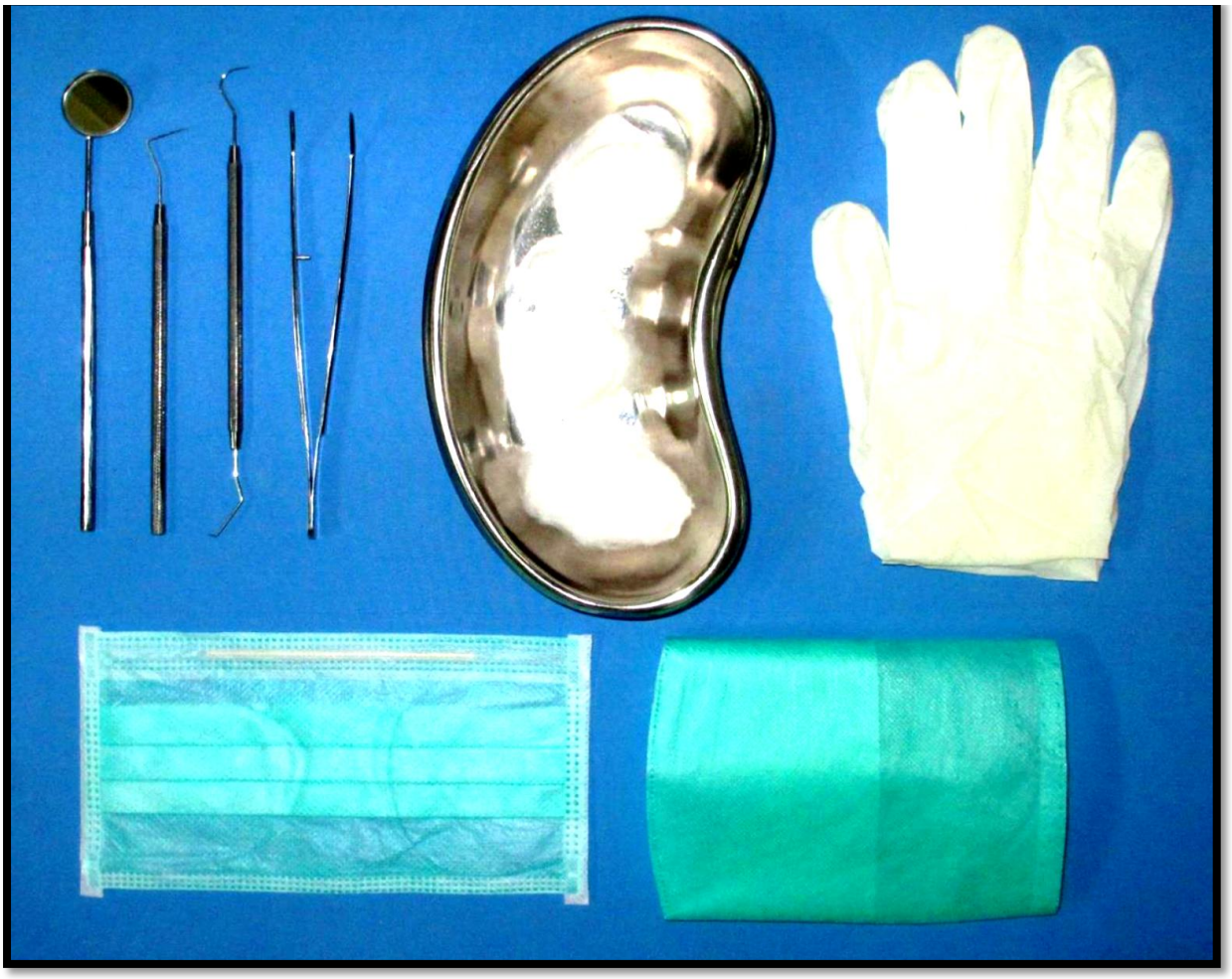


Fig. 4. Armamentarium for clinical examination

Fig. 5. Clinical examination

Fig. 6. Patient undergoing dialysis

Fig. 7. Dialysis machine

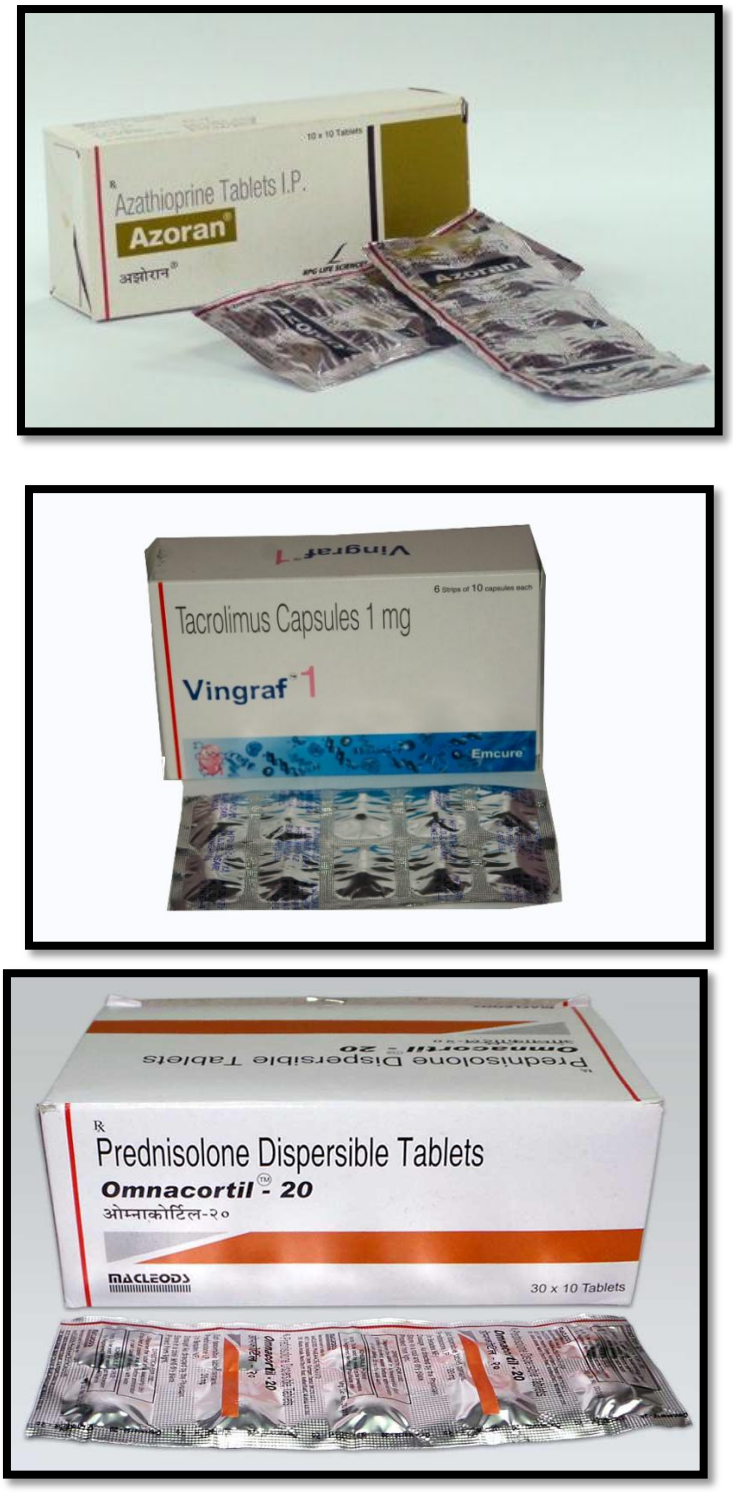


Fig. 8. Drug protocol for renal transplant patients

RESULTS

This study was a multicentered, cross sectional, prospective, parallel design study and purposive non probability sampling technique was applied. Statistical analysis used in this study is descriptive (frequency), inferential statistics like mean, standard deviation and ANOVA, to examine the significance of the differences between the clinical parameters among different groups. When a difference was found, a Tukey Kramer analysis for multiple regression analysis was utilized to elucidate which groups were statistically different from the others. The Pearson correlation analysis was utilized to examine the significance of the correlation between the variables. The statistical analysis was calculated by using analysis of variance (ANOVA) and Post Hoc test was applied for multiple comparisons. A p value=0.01 was chosen as level of significance. The observation of the study is given as follows.

Sample characteristics:

Description of age in study group:

Total number of individuals included in this study were 95 (CRF patients=75, Control=20), among them 68.4% were males (n=65) and 31.6% were females (n=30). In CRF groups, the total number of renal transplant (RT) patients were 25, among them 80% were males (n=20) and 20% were females (n=5). Total number of dialysis (D) patients were 25, among them 56% were males (n=14) and 44% were females (n=11). In predialysis group (PD), total number of patients were 25, among them 72% were males (n=18) and 28% were females (n=7). The total number patients in control group (C) were 20, among them 65%were males (n=13) and 35% were females (n=7)

[Table 1]. Association between the groups was given by Pearson Chi-Square which denotes **(p<0.01)**.The individuals age between the group was statistically significant.

Description of gingival overgrowth:

In this study, 52% (n=13) of renal transplant (RT) patients, 12% (n=3) dialysis (D) patients and 4% (n=1) predialysis (PD) patients showed significant amount of gingival overgrowth. No patients in the control group (C) (0%, n=0) had gingival overgrowth. Pearson Chi Square denotes the association within the groups was statistically significant value (**p<0.01**) as shown in [Table 2].

Description of clinical parameter in study groups:

The mean values among the CRF groups indicate that there was no significant difference in GBI, PPD, GI and PlI which was done using ANOVA analysis. The mean value of PlI in RT group was 1.7096, D group was 1.4032, PD group was 1.2872 and C group was 0.6075 which were statistically insignificant (p = 0.036). Similarly, the mean values of GI in RT, D, PD, C group was 1.8220, 1.5064, 1.3048, 0.5965 respectively were also statistically insignificant (p = 0.078). For PPD, the mean values in RT, D, PD, C group was 3.9708, 3.8084, 3.5016, 2.8250 respectively but these values were statistically insignificant (p = 0.306). The mean value of GBI in RT, D, PD, C was 2.9268, 2.4728, 2.3124, 1.1960 respectively but these values were statistically insignificant (p = 0.476). In contrast with other clinical parameters, the mean value of CAL in RT, D, PD, C is 2.3020, 1.6008, 0.4832, 0.0750 were statistically significant (**p = 0.000**). As shown in Table 3, the clinical parameters of the study group like PlI, GI, GBI, PPD were statistically insignificant and only statistically significant parameter was CAL.

Description of intergroup comparison in the study groups:

The mean difference of independent variable PlI was statistically significant between RT and PD (**p=0.019**), RT and C (**p=0.000**), D and C (**p=0.000**), PD and C (**p=0.000**) [Table 4, Fig 1] and statistically insignificant values between RT and D (p=0.142), PD and D (0.846).

The mean difference of independent variable GI was statistically significant between RT and PD (**p=0.001**), RT and C (**p=0.000**), D and C (**p=0.000**), PD and C (**p=0.000**) [Table 5, Fig 2] and statistically insignificant value between RT and D (p=0.073), D and PD (p=0.400).

The mean difference of independent variable PPD was statistically significant between RT and C (**p=0.000**), D and C (**p=0.000**), PD and C (**p=0.006**) [Table 6, Fig 3] and statistically insignificant values between RT and D (p=0.827), RT and PD (p=0.070), D and PD (p=0.373).

The mean difference of independent variable CAL was statistically significant between RT and PD (**p=0.000**), RT and C (**p=0.000**), D and C (**p=0.001**), D and PD (**p=0.011**) [Table 7, Fig 4] and statistically insignificant values between RT and D (p=0.203), PD and C (p=0.699).

The mean difference of independent variable GBI was statistically significant between RT and PD (**p=0.018**), RT and C (**p=0.000**), D and C (**p=0.000**), PD and C (**p=0.000**) [Table 8, Fig 5] and statistically insignificant value between RT and D (p=0.125), D and PD (p=0.861).

Overall, RT groups shows higher difference in clinical parameters when compared to D group, D group shows higher values when compared to PD group and PD shows higher value when compared to C group.

In this study, the mean values of PlI, GI, PPD and GBI was statistically insignificant though the mean difference between the groups were significant. Clinical changes show all independent variables increases in renal transplant group when compared to dialysis and pre dialysis patients. All variables were comparably low in control group. Clinical attachment level is the marker of periodontal disease. In this study, clinical attachment loss was more in renal transplant group when compared to other groups and it was statistically significant. So, there was correlation between CRF patients for developing gingivitis and periodontitis.

STUDY SAMPLE CHARACTERISTICS

Table 3. Description of age in study group:

	CRF TREATMENT GROUPS (n=75)				
Groups	**RT**	**D**	**PD**	**C**	**Total**
Number of subjects	25	25	25	20	95
Male (%)	20(80%)	14(56%)	18(72%)	13(65%)	65(68.4%)
Female (%)	5(20%)	11(44%)	7(28%)	7(35%)	30(31.6%)
p value §	**0.000***	**0.000***	**0.000***	**0.000***	**0.000***

*represent p value **(<0.01)** which is statistically significant

§ represent **Pearson Chi-Square**

Table 4. Description of Gingival Overgrowth

	CRF TREATMENT GROUPS (n=75)				
Groups	**RT**	**D**	**PD**	**C**	**Total**
Number of subjects (%)	25(100%)	25(100%)	25(100%)	20(100%)	95(100%)
Gingival overgrowth present (%)	13(52%)	3(12%)	1(4%)	0(0%)	17(17.9%)
Gingival overgrowth absent (%)	12(48%)	22(88%)	24(96%)	20(100%)	78(82.1%)
p value §	**0.000***	**0.000***	**0.000***	**0.000***	**0.000***

*represent p value **(<0.01)** which is statistically significant

§ represent **Pearson Chi-Square**

Table 3. Baseline Description of Study Groups (ANOVA)

Parameters	Groups	Mean	Std.dev	Minimum	Maximum	p Value ¶
PlI	**RT**	1.7096	0.61304	0.60	2.95	
	D	1.4032	0.38021	0.80	2.10	
	PD	1.2872	0.56015	0.20	2.40	
	C	0.6075	0.38872	0.10	1.40	0.036
GI	**RT**	1.8220	0.55359	0.80	2.70	
	D	1.5064	0.38397	0.90	2.35	
	PD	1.3048	0.50869	0.13	2.40	
	C	0.5965	0.29116	0.10	1.10	0.078
PPD	**RT**	3.9708	0.86901	1.40	5.30	
	D	3.8084	0.54516	3.00	5.30	
	PD	3.5016	0.51569	2.63	4.50	
	C	2.8250	0.69953	1.20	4.00	0.306
CAL	**RT**	2.3020	1.71837	0.00	5.70	
	D	1.6008	1.59464	0.00	5.10	
	PD	0.4832	0.65895	0.00	2.80	
	C	0.0750	0.15174	0.00	0.50	**0.000***
GBI	**RT**	2.9268	0.77163	1.50	4.40	
	D	2.4728	0.58966	1.70	4.40	
	PD	2.3124	0.86214	0.20	4.50	
	C	1.1960	0.61029	0.30	2.50	0.476

*represent p value **(<0.01)** which is statistically significant

¶ represent one way analysis by **ANOVA**

Table 4. Comparison of Plaque Index (PlI) between study groups (Tukey Test)

Independent variables	Groups (I)	Groups (J)	Mean difference (I –J)	Lower bound ∂	Upper bound ∂	p value ǂ
PlI	**RT**	**D** **PD** **C**	0.3064 **0.4224*** **1.1021***	- 0.0649 0.0511 0.7083	0.6777 0.7937 1.4959	0.142 **0.019*** **0.000***
	D	**RT** **PD** **C**	- 0.3064 0.1160 **0.7957***	- 0.6777 - 0.2553 0.4019	0.0649 0.4873 1.1895	0.142 0.846 **0.000***
	PD	**RT** **D** **C**	**-0.4224*** -0.1160 **0.6797***	- 0.7937 - 0.4873 0.2859	-0.0511 0.2553 1.0735	**0.019*** 0.846 **0.000***
	C	**RT** **D** **PD**	**-1.1061*** **-0.7957*** **-0.6797***	-1.4959 -1.1895 -1.0735	-0.7083 -0.4019 -0.2859	**0.000*** **0.000*** **0.000***

*represent p value **(<0.01)** which is statistically significant

ǂ **Tukey HSD test**

∂ represent **95% confidence interval.**

Table 5. Comparison of Gingival Index (GI) between study groups (Tukey Test)

Independent Variables	Groups (I)	Groups (J)	Mean difference (I –J)	Lower bound ∂	Upper bound ∂	p value ǂ
GI	**RT**	**D** **PD** **C**	0.3156 **0.5172*** **1.2255***	-0.0201 0.1815 0.8694	0.6513 0.8529 1.5816	0.073 **0.001*** **0.000***
	D	**RT** **PD** **C**	-0.3156 0.2016 **0.9099***	-0.6513 -0.1341 0.5538	0.0201 0.5373 1.2660	0.073 0.400 **0.000***
	PD	**RT** **D** **C**	**-0.5172*** -0.2016 **0.7083***	-0.8529 -0.5373 0.3522	-0.1815 0.1341 1.0644	**0.001*** 0.400 **0.000***
	C	**RT** **D** **PD**	**-1.2255*** **-0.9099*** **-0.7083***	-1.5816 -1.2660 -1.0644	-0.8694 -0.5538 -0.3522	**0.000*** **0.000*** **0.000***

*represent p value **(<0.01)** which is statistically significant

ǂ **Tukey HSD test**

∂ represent **95% confidence interval.**

Table 6. Comparison of Probing Plaque Depth (PPD) between study groups (Tukey Test)

Independent Variables	Groups (I)	Groups (J)	Mean difference (I –J)	Lower bound ∂	Upper bound ∂	p value ǂ
PPD	**RT**	**D**	0.1624	-0.3333	0.6581	0.827
		PD	0.4692	-0.0265	0.9649	0.070
		C	**1.1458***	0.6200	1.6716	**0.000***
	D	**RT**	-0.1624	-0.6581	0.3333	0.827
		PD	0.3068	-0.1889	0.8025	0.373
		C	**0.9834***	0.4576	1.5092	**0.000***
	PD	**RT**	-0.4692	-0.9649	0.0265	0.070
		D	0.3068	-0.8025	0.1889	0.373
		C	**0.6766***	0.1508	1.2024	**0.006***
	C	**RT**	**-1.1458***	-1.6716	-0.6200	**0.000***
		D	**-0.9834***	-1.5092	-0.4576	**0.000***
		PD	**-0.6766***	-1.2024	-0.1508	**0.006***

*represent p value **(<0.01)** which is statistically significant

ǂ **Tukey HSD test**

∂ represent **95% confidence interval.**

Table 7. Comparison of Clinical Attachment Level (CAL) between study groups (Tukey Test)

Independent variables	Groups (I)	Groups (J)	Mean difference (I –J)	Lower bound ∂	Upper bound ∂	p value ǂ
CAL	**RT**	**D** **PD** **C**	0.7012 **1.8188*** **2.2270***	-0.2259 0.8917 1.2436	1.6283 2.7459 3.2104	0.203 **0.000*** **0.000***
	D	**RT** **PD** **C**	-0.7012 **1.1176*** **1.5258***	-1.6283 0.1905 0.5424	0.2259 2.0447 2.5092	0.203 **0.011*** **0.001***
	PD	**RT** **D** **C**	**-1.8188*** **-1.1176*** 0.4082	-2.7459 -2.0447 -0.5752	-0.8917 -0.1905 1.3916	**0.000*** **0.011*** 0.699
	C	**RT** **D** **PD**	**-2.2270*** **-1.5258*** -0.4082	-3.2104 -2.5092 -1.3916	-1.2436 -0.5424 0.5752	**0.000*** **0.001*** 0.699

*represent p value **(<0.01)** which is statistically significant

ǂ **Tukey HSD test**

∂ represent **95% confidence interval.**

Table 8. Comparison of Gingival Bleeding Index (GBI) between study groups (Tukey Test)

Independent variables	Groups (I)	Groups (J)	Mean difference (I –J)	Lower bound ∂	Upper bound ∂	p value ǂ
GBI	**RT**	**D**	0.4540	-0.0811	0.9891	0.125
		PD	**0.6144***	0.0793	1.1495	**0.018***
		C	**1.7308***	1.1632	2.2984	**0.000***
	D	**RT**	-0.4540	-0.9891	0.0811	0.125
		PD	0.1604	-0.3747	0.6955	0.861
		C	**1.2768***	0.7092	1.8444	**0.000***
	PD	**RT**	**-0.6144***	-1.1495	-0.0793	**0.018***
		D	-0.1604	-0.6955	0.3747	0.861
		C	**1.1164***	0.5488	1.6840	**0.000***
	C	**RT**	**-1.7308***	-2.2984	-1.1632	**0.000***
		D	**-1.2768***	-1.8444	-0.7092	**0.000***
		PD	**-1.1164***	-1.6840	-0.5488	**0.000***

*represent p value **(<0.01)** which is statistically significant

ǂ **Tukey HSD test**

∂ represent **95% confidence interval**.

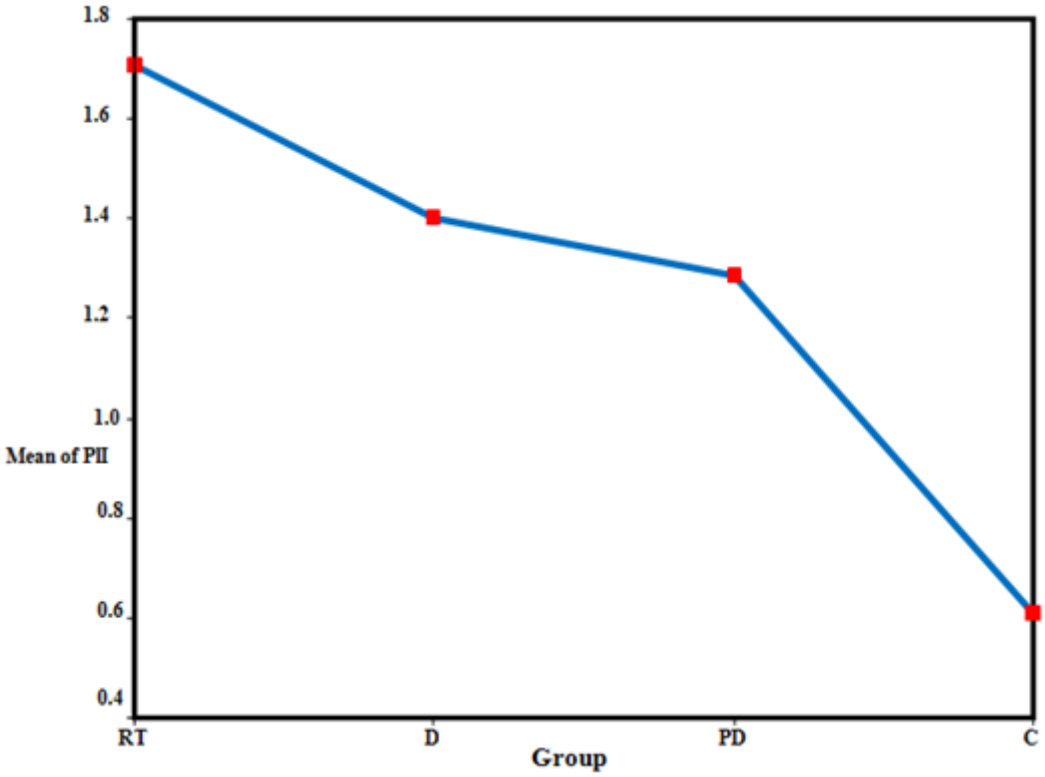


Fig 1: Comparison of Plaque Index (Pll) between study groups

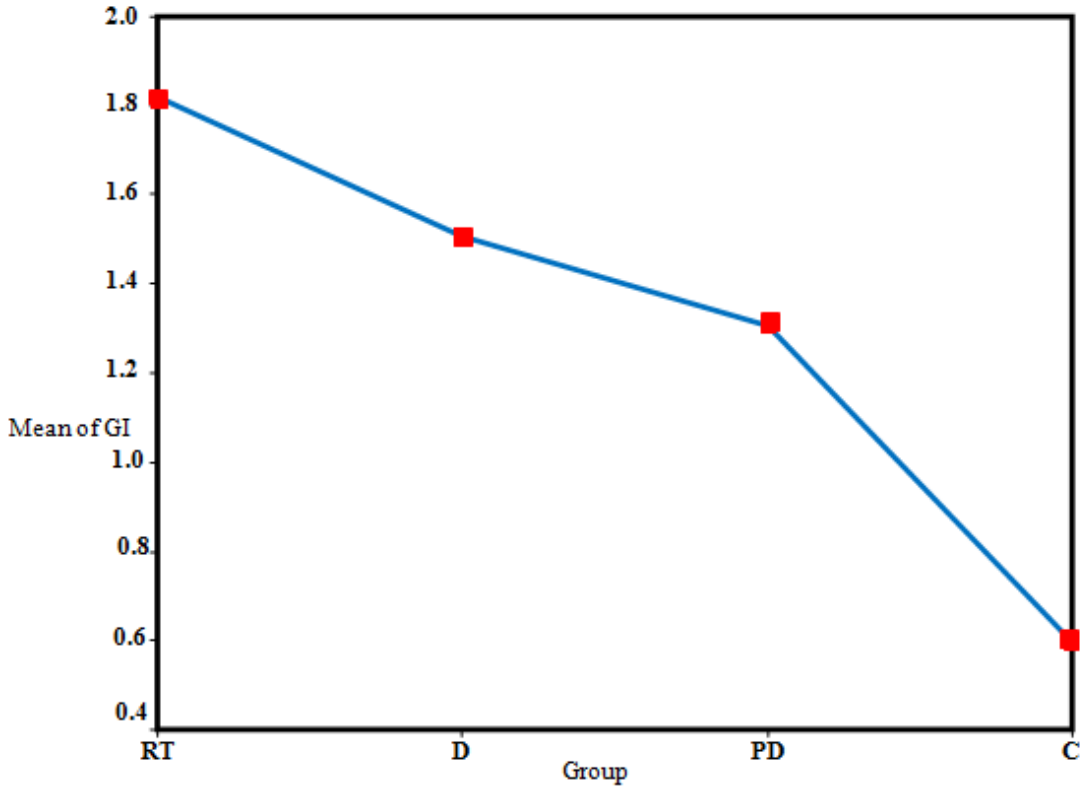


Fig 2: Comparison of Gingival Index (GI) between study groups

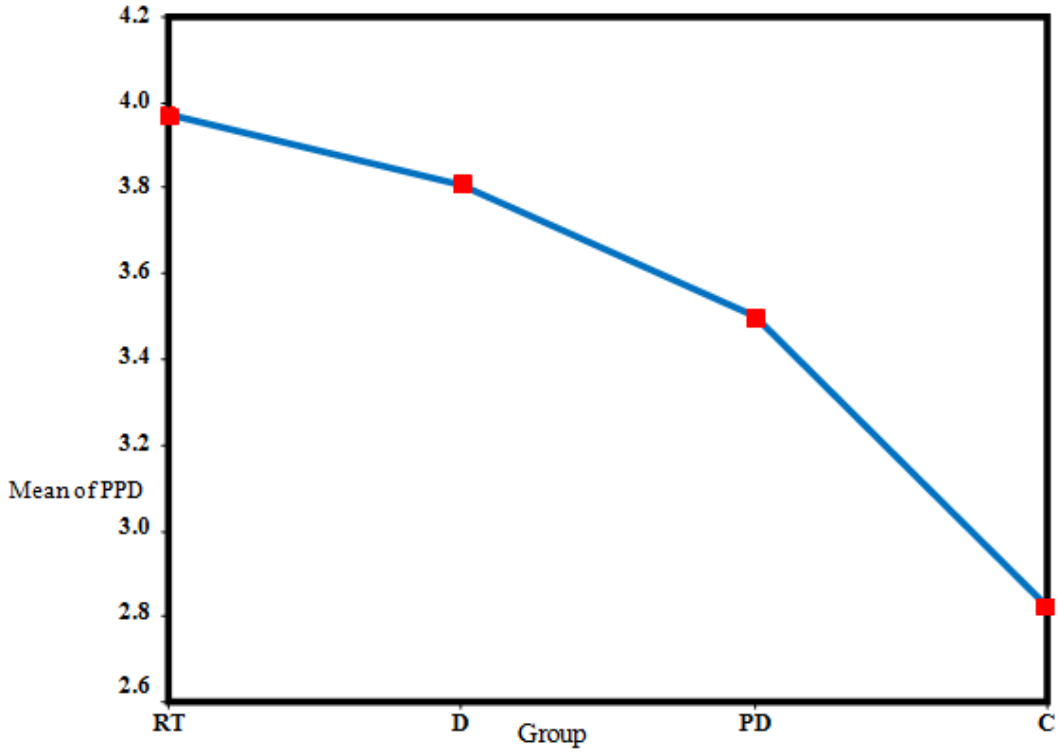


Fig 3: Comparison of Probing Pocket Depth (PPD) between study groups

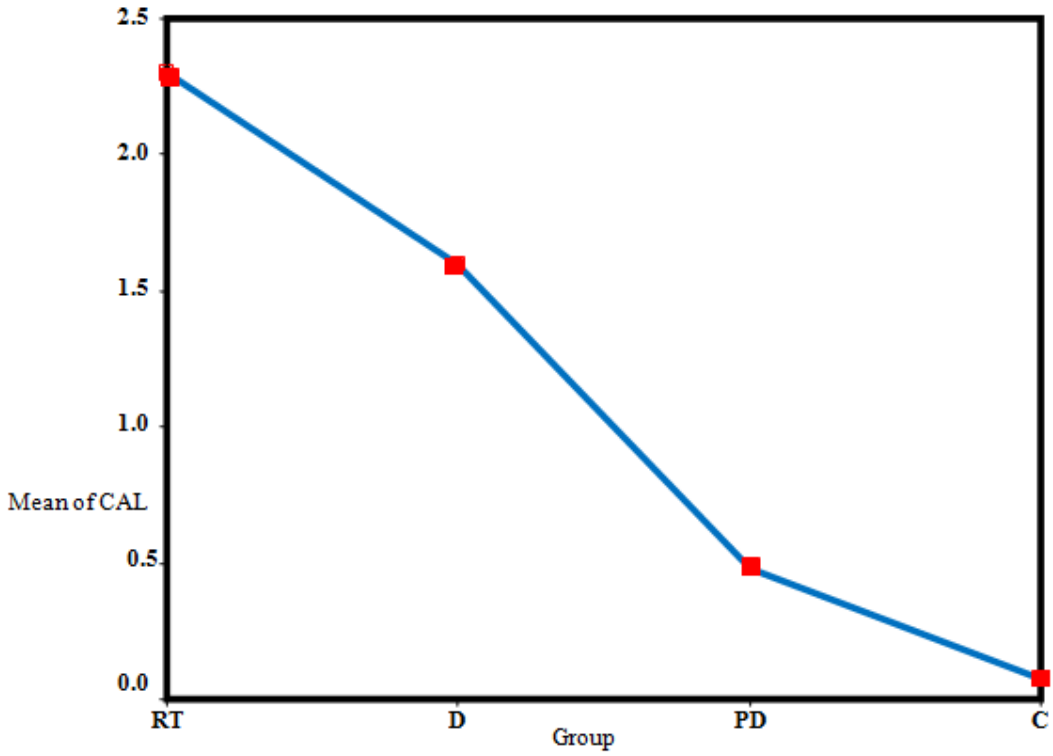


Fig 4: Comparison of Clinical Attachment Level (CAL) between study groups

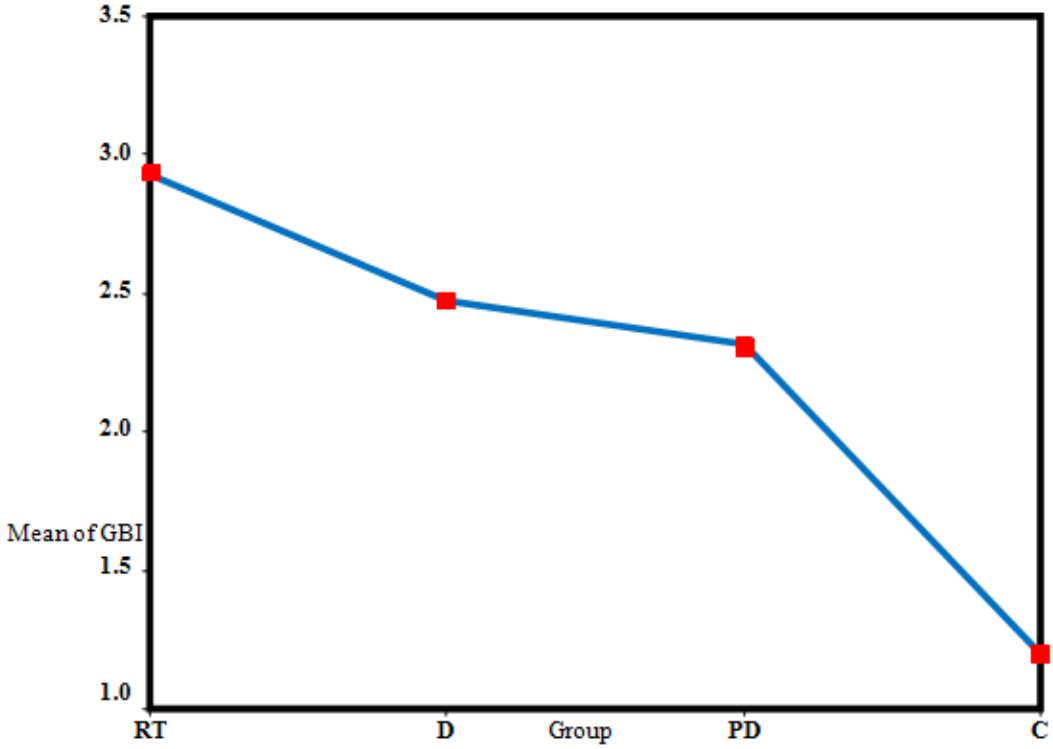


Fig 5: Comparison of Gingival Bleeding Index (GBI) between study groups

DISCUSSION

A state of immunodeficiency exists in CRF patients are due to altered immunity and protein malnutrition.[1] In CRF patients, various treatment modalities make them high risk for infection and bleeding abnormalities. **Kshirsagar et al. (2007)[43], Fisher et al. (2008)[45], Levey et al. (2011)[10]** suggested that, different forms of acute and chronic inflammatory processes can stimulate an inflammatory response in the kidneys, leading to CRF. Periodontal disease is a chronic infectious disease of microbial origin which results in destruction of the tissues housing the tooth and ultimately leads to tooth loss. Periodontitis could be considered a non-traditional risk for CRF due to the following factors: [45]

(1) Systemic inflammatory burden caused by periodontal inflammation (and its locally produced inflammatory mediators, such as IL-1, IL-6, PGE_2 and TNFα)

(2) Presence of bacteria and their products in the bloodstream

(3) Elevated levels of C-reactive protein, a mild acute-phase systemic inflammatory response which acts as a source of "permanent inflammation" that could contribute to CRF.

With respect to the presence of bacteria in the bloodstream, circulating periodontal bacteria could lead to kidney endothelium damage.[25, 19] It seems biologically plausible, that such an association could constitute a source of systemic inflammatory burden.[45]The predictive role of inflammatory biomarkers creates a poor outcome in CRF patients.

This study was a multicentered, cross sectional, prospective, parallel design and purposive non probability sampling technique was applied. Statistical analysis

used in this study was descriptive (frequency), inferential statistics like mean, standard deviation and ANOVA, to examine the significance of the differences between the clinical parameters among different groups. When a difference was found in ANOVA test, a Tukey Kramer analysis for multiple regression analysis was utilized to elucidate which groups were statistically different from the others. The Pearson correlation analysis was utilized to examine the significance of the correlation between the variables. The statistical analysis was calculated by using analysis of variance (ANOVA) and Post Hoc test was applied for multiple comparisons. The clinical parameters assessed were PlI, GI, GO, GBI, PPD, CAL in patients with CRF under various treatment like renal transplant(RT), dialysis(D) and predialysis(PD) [Test group] and compared with systemically healthy subjects [Control].

The inference of the present study showed, the mean values of PlI, GI, PPD and GBI were statistically insignificant between the study groups. Test [renal transplant(RT), dialysis(D), predialysis(PD)] groups showed clinically increased oral parameters when compared with control group, which revealed considerably low values. The mean value of clinical attachment loss was statistically significant.

The present study showed mean value of PlI score in renal transplant(RT) group was 1.7096, dialysis(D) group was 1.4032, predialysis(PD) group was 1.2872 and control(C) group was 0.6075 and it was not statistically significant (p=0.036). This result was in accordance with the study by **Oshrain et al. (1979)[31],** who found mean value of PlI was statistically insignificant.

In the current study, the mean difference of independent variable PlI was statistically significant between renal transplant(RT) and predialysis(PD) (**p=0.019)** groups, renal transplant(RT) and control(C) groups (**p=0.000)**, dialysis(D) and control(C) groups (**p=0.000)**, predialysis(PD) and control(C) groups (**p=0.000)** and statistically insignificant values between renal transplant(RT) and dialysis(D) groups (p=0.142), predialysis(PD) and dialysis(D) groups (p=0.846).

In accordance with the present study, comparing the mean difference of plaque index in renal transplant(RT) and predialysis(PD) groups, renal transplant(RT) and control(C) groups, **Bastos et al. (2011)[64]** found a statistically significant difference. Comparing dialysis(D) and control(C) groups, **Sauza et al. (2005)[36], Castillo et al. (2007)[42], Bayraktar et al. (2007)[39], Brito et al. (2012)[54], Davidovich et al. (2005)[6]** found it to be statistically significant. Comparing predialysis(PD) and control(C) group, **Davidovich et al. (2005)[6]** found to be statistically significant. Comparing renal transplant(RT) and dialysis(D) groups, **Oshrain et al. (1979)[31]** found mean difference were statistically insignificant. According to **Davidovich et al. (2005)[6],** who found predialysis(PD) and dialysis(D) groups were statistically insignificant.

In contrast to the present study, **Davidovich et al. (2005)[6]** found statistically insignificant difference between renal transplant(RT) and predialysis(PD) groups. **Oshrain et al. (1979)[31], Kardachi et al. (1978)[30], Davidovich et al. (2005)[6]** found statistically insignificant between renal transplant(RT) and control(C) groups. **Frankenthal et al. (2002)[67], Marakoglu et al. (2003)[1], Bots et al. (2005)[37], Borawski et al. (2007)[41], Oshrain et al. (1979)[31]**found a statistically insignificant

difference between dialysis(D) and control(C) groups. **Borawski et al (2007)[41], Artese et al. (2011)[49], Bastos et al (2011)[64], Vilela et al. (2011)[53]** found statistically insignificant difference between predialysis(PD) and control(C) groups. **Davidovich et al. (2005)[6]** found statistically significant difference between renal transplant(RT) and dialysis(D) groups. The result of the present study highlighted PlI was higher in test group when compared to control group. To overcome increased levels of plaque accumulation, regular periodontal care, non surgical periodontal therapy and maintenance is indicated in CRF patients.

In the present study, the mean values of GI in renal transplant(RT), dialysis(D), predialysis(PD), control group(C) was 1.8220, 1.5064, 1.3048, 0.5965 respectively and these values were also statistically insignificant (p=0.078). This result was in accordance with the study by **Oshrain et al. (1979)[31],** who found mean value of GI was statistically insignificant.

The mean difference of independent variable GI was statistically significant between renal transplant and predialysis(PD) groups (**p=0.001**), renal transplant(RT) and control(C) groups (**p=0.000**), dialysis(D) and control(C) groups (**p=0.000**), predialysis(PD) and control(C) groups (**p=0.000**) and statistically insignificant value between renal transplant(RT) and dialysis (D)groups (p=0.073), dialysis(D) and predialysis (PD) groups (p=0.400).

In accordance with the present study, comparing the mean difference of gingival index, **Davidovich et al. (2005)[6]** found the renal transplant(RT) and predialysis groups(PD), renal transplant(RT) and control(C) groups, dialysis(D) and

control (C)groups, predialysis(PD) and control(C) groups were statistically significant. **Bayraktar et al. (2007)[39], Kardachi et al. (1978)[30]** stated statistically significant difference between renal transplant(RT) and control(C) groups. **Sauza et al. (2005)[36]** who compared dialysis(D) and control(C) group and found to be statistically significant. **Joseph et al. (2009)[46]** compared predialysis(PD) and control(C) groups and found to be statistically significant. **Davidovich et al. (2005)[6]** compared renal transplant(RT) and dialysis(D) groups were statistically insignificant.

In contrast to the present study, **Marakoglu et al. (2003)[1], Frankenthal et al. (2002)[67]** compared dialysis(D) and control(C) groups were statistically insignificant. **Davidovich et al. (2005)[6]**compared dialysis (D) and predialysis (PD) groups and found to be statistically significant. Gingival inflammation has been reported, due to plaque accumulation and poor oral hygiene. Increased plaque accumulation has the potential to increase the inflammatory component of gingival disease in patients with renal failure.

The mean difference of independent variable PPD was statistically significant between renal transplant(RT) and control(C) groups (**p=0.000**), dialysis(D) and control(C) groups (**p=0.000**), predialysis(PD) and control(C) groups (**p=0.006**) and statistically insignificant values between renal transplant(RT) and dialysis(D) groups (p=0.827), renal transplant(RT) and predialysis(PD) groups (p=0.070), dialysis(D) and predialysis(PD) groups (p=0.373).

In accordance to the present study, **Davidovich et al. (2005)[6],** who found the mean difference of PPD in renal transplant(RT) and control(C) groups, dialysis(D) and control(C) groups, predialysis(PD) and control(C) groups were statistically

significant. When comparing predialysis(PD) and control(C) groups, **Joseph et al. (2009)[46], Vilela et al. (2011)[53]** found that mean difference was found to be statistically significant.

In contrast to the present study, **Bayraktar et al. (2003)[39], Bots et al. (2005)[37] Marakoglu et al. (2003)[1], Frankenthal et al. (2002)[67]** examined dialysis(D) and control(C) groups and found it to be statistically insignificant. However, current study finding revealed periodontal pockets were significantly deeper in the CRF groups than in the control indicating a different mechanism of periodontal disease in CRF patients. Furthermore, the significant correlation between periodontal pockets and the duration of renal failure suggests that prolonged renal dysfunction has a direct effect on the progression of periodontal disease.

Among all the clinical parameters, the mean value of CAL was statistically significant (**p=0.000**) and the mean difference of independent variable CAL was statistically significant between renal transplant(RT) and predialysis(PD) groups (**p=0.000**), renal transplant(RT) and control(C) groups (**p=0.000**), dialysis(D) and control(C) groups (**p=0.001**), dialysis(D) and predialysis(PD) groups (**p=0.011**) and statistically insignificant values between renal transplant(RT) and dialysis(D) groups (p=0.203), predialysis(PD) and control(C) groups (p=0.699).

In accordance with the present study, **Bastos et al. (2011)[64]** who found statistically significant of attachment loss in predialysis(PD) and renal transplant(RT) groups. **Davidovich et al. (2005)[6], Bastos et al. (2011) [64]** found renal transplant(RT) and control(C) groups were statistically significant. **Parkar et al. (2012)[68], Borawski et al. (2007)[41], Brito et al. (2012)[54]** compared dialysis(D) and control(C) group and

found it to be statistically significant. When comparing predialysis(PD) and control(C) groups, **Bastos et al. (2011) [64], Artese et al. (2010)[49]** found the mean difference was statistically insignificant.

In contrast to the present study, **Vilela et al. (2011)[53], Brito et al. (2012)[54], Davidovich et al. (2005)[6]** compared predialysis(PD) and control(C) group and found to be statistically significant. **Castillo et al. (2007)[42], Joseph et al. (2009)[46]** found statistically insignificant mean difference between dialysis(D) and control(C) groups. Clinical attachment loss which denotes the presence of periodontitis, was higher in CRF patients compared to control subjects. These findings suggest that attachment loss may be influenced by the uraemic status and its duration.

The mean difference of independent variable, GBI, was statistically significant between renal transplant(RT) and predialysis(PD) groups (**p=0.018**), renal transplant(RT) and control(C) groups (**p=0.000**), dialysis(D) and control(C) groups (**p=0.000**), predialysis(PD) and control(C) groups (**p=0.000**) and statistically insignificant value between renal transplant(RT) and dialysis(D) groups (p=0.125), dialysis(D) and predialysis(PD) groups (p=0.861).

In accordance with the present study, **Davidovich et al. (2005)[6],** stated that BOP was statistically significant between renal transplant(RT) and control(C) groups, dialysis(D) and control(C) groups, predialysis(PD) and control(C) groups. **Brito et al. (2012)[54]** found statistically significant difference between predialysis(PD) and control(C) groups. **Borawski et al. (2007)[41]** found statistically significant difference between dialysis(D) and control(C) groups.

In contrast to the present study, **Artese et al. (2010)[49], Bostos et al. (2011) [64]**, **Vilela et al. (2011)[53]**found statistically insignificant between predialysis(PD) and control(C) groups. **Brito et al. (2012)[54], Castillo et al. (2007)[42]** found statistically insignificant gingival bleeding between dialysis(D) and control(C) groups. From this study it was revealed that gingival bleeding was more in test group when compared control(C) group. Bleeding on probing was examined separately from GI because of coagulation disturbances in CRF patients (impaired thrombocyte function and heparin administration during dialysis in transplanted patients). In the present study, bleeding sites prevalence was significantly higher in the CRF groups as compared to the controls(C) who had normal coagulation (the dialysis group showing the highest gingival bleeding prevalence). This finding may be related to more severe gingival inflammation and/or coagulation disturbances in CRF patients. However, one should take in consideration that the examination was performed 1 h after dialysis. Therefore, the patients were heparinized and the urea reduction was not enough to improve thrombocytes function).

In the present study, presence of gingival overgrowth was statistically significant in renal transplant patients and this was due to intake of calcium channel blockers. 52% (n=13) of renal transplant (RT) patients, 12% (n=3) dialysis(D) patients and 4% (n=1) predialysis(PD) patients showed significant amount of gingival overgrowth. No patients in the control group (C) (0%, n=0) had gingival overgrowth. This result was supported by **Davidovich et al. (2005)[6],** where he found the renal transplant group showed higher gingival overgrowth and slight increase in predialysis and dialysis groups and it was statistically significant. This study showed that gingival

overgrowth was prevalent in test group and completely absent in control(C) group. Extensive gingival overgrowth was found in transplanted patients due to the use of cyclosporine, and most likely also related to the use of calcium channel blockers and poor oral hygiene in the predialysis and dialysis patients.

According to present study, test group showed increase in plaque accumulation, gingival inflammation, loss of attachment, bleeding on probing and gingival overgrowth when compared to control(C) group and the mean difference between test and control(C) group was statistically significant. This indicates a direct association between the CRF patients and periodontitis. The issues of poor oral health status deserve dental awareness in CKF patients. Since, the periodontal status gets worsened in CRF patients, early periodontal examination and oral prophylaxis should be considered as an integral part of treating these patients.

SUMMARY& CONCLUSION

Bacterial plaque is the primary etiological factor for periodontal disease and along with that, the systemic factors/conditions of the host will also alter the prevalence, progression and severity of periodontal diseases. CRF patients present a complex clinical problem with multisystem involvement, including oral disease and some systemic conditions like anemia, increased liability to bleeding, cardiovascular disease, but with the use of well supervised diagnosis and treatment protocol, management of these individuals will be effective and safe. The CKD patient with periodontitis is medically complex and presents the dental practitioner with several challenges in the management of their periodontal condition. Accordingly, close communication between the dentist and nephrologist is essential to optimize periodontal management. Comprehensive clinical investigation of periodontal disease in CRF patients are needed to gain new insight about dental awareness and to improve their oral health.

This study evaluates the oral and periodontal status of chronic renal failure patients undergoing predialysis (PD), dialysis (D) and renal transplant (RT). The study includes 75 cases of chronic renal failure patients compared with 20 healthy individuals with age ranging more than 35years. The test group consists of three groups based on their treatment. Group-I comprised of 25 patients who were on predialysis, Group-II comprised of 25 patients who were on dialysis, Group-III consists of 25 patients who were underwent renal transplant. The control group consists of 20 patients were included in the control group without any systemic disease. Test group were selected in the Department of Nephrology, Sri Ramakrishna

Hospital, Coimbatore and Coimbatore Kidney Center, Coimbatore and controls obtain from Department of Periodontology, Sri Ramakrishna Dental College and Hospital, Coimbatore.

In this study, the clinical parameters PlI, GI, PPD, CAL, GBI, GO were assessed. The mean value of PlI, GI, PPD, GBI were statistically insignificant but CAL was statistically significant. When considering the mean difference between the groups, all clinical parameters were statistically significant. The finding of the study showed that the test group showed increase in plaque accumulation, gingival inflammation, bleeding on probing, loss of attachment and gingival overgrowth when compared to control group. This indicates CRF patients were more prone for progression of periodontal disease. Hence, there is a relationship that exists between CRF patients and periodontitis. Therefore, maintaining good oral health is of major importance since oral pathologies or infections could jeopardize the opportunity to receive a successful kidney transplant.

There is still a long way to discover the relationship between CRF and periodontal disease. Limitation of this study was its cross-sectional design and smaller sample size. Therefore, further longitudinal studies with larger sample size, comparison of pre and post periodontal treatment has to be assessed.

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