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Assessment Of Thyroxine Status In Patients With Chronic Kidney Disease

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ABSTRACT

Thyroid problems are common in patients with chronic kidney disease (CKD), which is becoming an increasingly important issue in public health across the world. This prospective research aimed to examine the relationship between thyroid dysfunction and the severity of renal diseases in 100 persons with CKD. Among the individuals surveyed, 60% had low T3 syndrome and 45% low T4 syndrome. Fifteen individuals also had primary hypothyroidism. The research discovered that when the glomerular filtration rate fell, there were significant variations in the frequency of individuals identified with low T3 syndrome. There was no change to the normal range in TSH levels throughout the stages of renal disease. Irrespective of the severity of their chronic illness or malnutrition, thyroid dysfunction was seen in most individuals with chronic renal impairment. As CKD promotes protein conservation, its low T3 state may actually be beneficial. Low T3 syndrome is more common in patients with advanced renal failure.

Key Words: Thyroid dysfunction; chronic kidney disease; low T_3 syndrome.

I.INTRODUCTION

A variety of pathophysiologic mechanisms linked to aberrant kidney function and a steady decrease in glomerular filtration rate (GFR)1,2 make up chronic kidney disease (CKD).

An irreversible decline in renal function that often occurs over a period of years is known as chronic renal failure (CRF). It starts off as a biochemical anomaly but progresses to a loss of kidney function in excretion, metabolism, and endocrine regulation. On top of that, it triggers the uremia-related clinical symptoms and indicators. "End stage renal failure" is a condition in which dialysis or kidney transplantation is likely to result in death.

The incidence of end-stage renal disease (ESRD) has risen sharply over the last 20 years, and the relative incidence of CRF aetiologies has shifted, as well.

It may be difficult to differentiate between thyroid dysfunction and chronic kidney disease (CKD) due to the multiple symptoms that patients have as a result of the kidney disease itself. Patients with chronic kidney disease (CKD) can have symptoms such as dry skin, cold sensitivity, asthenia, hyporeflexia, and oedema even in the absence of thyroid problems. Contrarily, even with extreme hypothyroidism, the traditional clinical symptoms and indications may be mild or nonexistent. Therefore, a simple clinical evaluation cannot rule out thyroid dysfunction in CKD patients.

Kidney development and growth, as well as the preservation of water and electrolyte balance, are reliant on thyroid hormones (TH). It is well-known that the kidney to body weight ratio6 decreases in hypothyroidism and rises in hyperthyroidism. Conversely, TH metabolism and disposal are facilitated by the kidneys.

Hyperthyroidism impacts RBF, glomerular filtration rate (GFR), tubular function, electrolyte balance, and kidney structure. Returning CKD patients to euthyroidism improves GFR, normalises RPF, and is a predictor of prognosis.

The majority of kidney problems caused by hypothyroidism may be reversed with the use of thyroxine administration.

The evaluation of kidney function is affected by hypothyroidism and hyperthyroidism, both of which are associated with clinically significant changes in renal function.

Extensive research on thyroid function in uremic individuals has shown contradictory findings. Some researchers have found hyperthyroidism, hypothyroidism, and a euthyroid condition in their samples.

It is suggested to do a prospective clinical and biochemical investigation to identify the connection of thyroid hormone with the severity of renal disease among patients with undialysed CKD, in light of

inconsistent findings of thyroid function tests in prior studies of patients with CKD.

OBJECTIVES

1. In order to measure how often thyroid dysfunction occurs in chronic renal disease patients.
2. For the purpose of researching the link between thyroid malfunction and the severity of kidney disorders.

II. LITERATURE REVIEW

Thyroid hormones

Upon both sides of the trachea, under the larynx, is where you'll find the thyroid gland. At 15–20 grammes in adulthood, the thyroid gland is among the heaviest endocrine glands.

Two main hormones, thyroxine (T4) and triiodothyronine (T3), are secreted by the gland. When the hormone is not secreted at all, the basal metabolic rate drops 40 to 50% below normal, and when it is secreted excessively, it rises 60 to 100% over normal. Secreted by the anterior pituitary gland, thyroid-stimulating hormone (TSH) is the primary regulator of thyroid production. Another crucial hormone for calcium metabolism, calcitonin, is secreted by the thyroid gland..

Synthesis and Secretion of the Thyroid Hormones

Approximately 93% of the thyroid gland's metabolically active hormones are thyroxine (T4), with just 7% being triiodothyronine (T3). Both T4 and T3 serve crucial physiological roles since almost all T4 is transformed to T3 in tissues. Although their effects are similar, the two hormones' rates of action and potencies are different. Even while T3 is almost four times as powerful as T4, it is far less abundant in the blood and doesn't stay there for nearly as long.

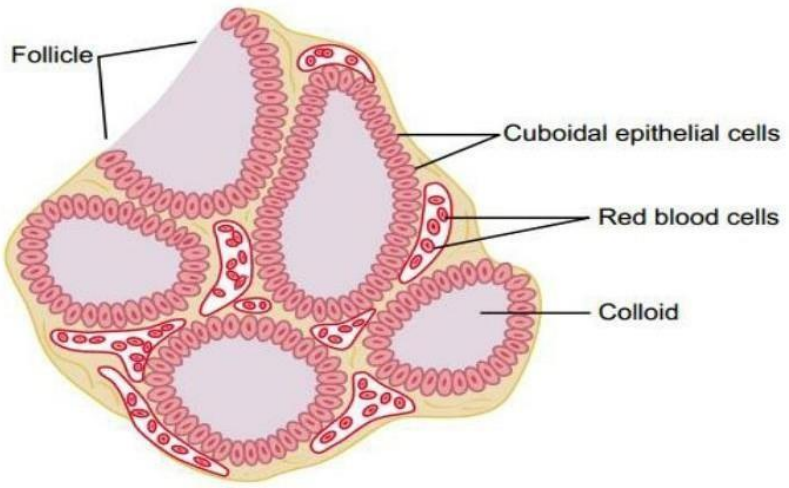


Fig-1 Microscopic appearance of the thyroid gland, showing secretion of thyroglobulin into the follicles.

Multiple closed follicles, each measuring 100–300 micrometres in diameter and filled with colloid, make up the thyroid gland. The cuboidal epithelial cells that border the follicles secrete into the follicle interior. The primary component of colloid, which houses the thyroid hormones, is thyroglobulin, a glycoprotein.

To serve its purpose, the secretion that has entered the follicles must be absorbed back into the bloodstream via the follicular epithelium. The thyroid gland receives around five times its weight in blood flow each minute.

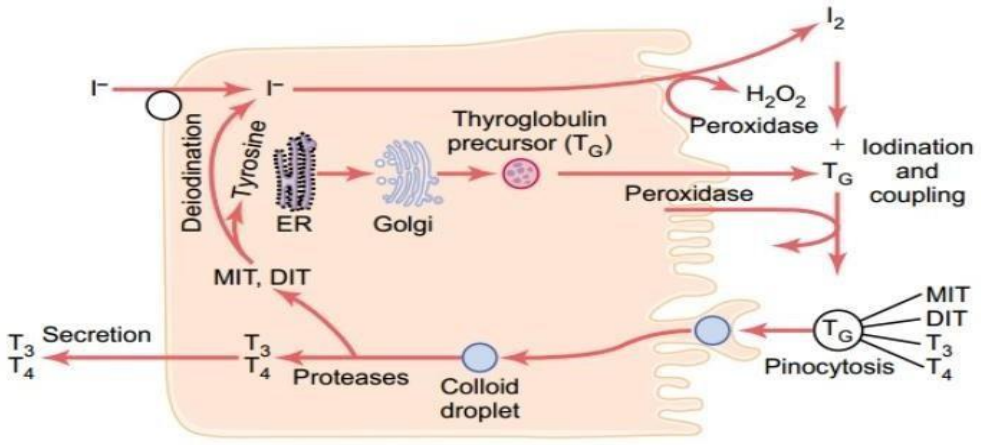


Fig-2 Mechanism for iodine transport, T4, T3 formation and its release.

Through a process known as "iodide trapping," the basal membrane of a thyroid cell actively pumps iodide into the cell from the bloodstream after it has been converted to iodine in the digestive tract. At its most active, the iodide pump may increase the concentration of iodide in the gland by as much as 250 times, while at rest it can increase it by 30 times.

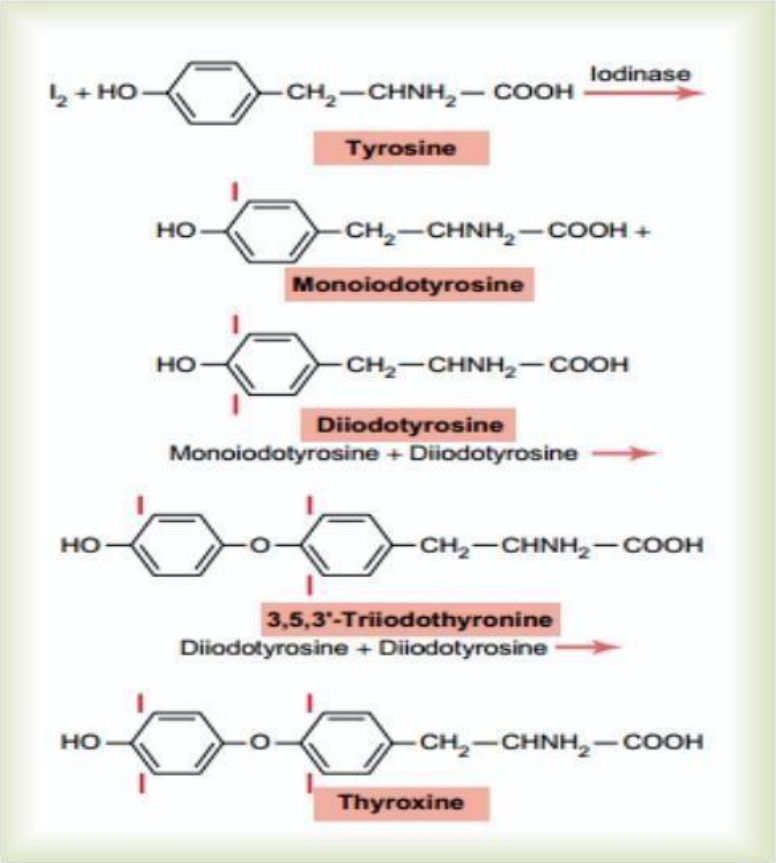


Fig-3 Triiodothyronine and Thyroxine formation

Diiodotyrosine (DIT) and Mono iodotyrosine (MIT) are formed when tyrosine and the trapped iodide are combined. T3 is formed by coupling MIT and DIT, whereas T4 is formed by coupling two DIT molecules. "Thyroid peroxidase" is responsible for catalysing oxidation, iodination, and coupling processes. Thyroglobulin binds the thyroid hormone that is so generated until it is secreted.

There are two ways it travels after being released into the bloodstream. In the first, known as "bound form," T3 and T4 are physically attached to plasma proteins that the liver produces. Thyroid binding globulin, prealbumin, and albumin are its primary binding partners. T3 is mostly bound to albumin, whereas T4 is mainly attached to thyroid binding globulin. There is also the free T3 and T4 variant. Bound form 6 and these free forms are in perfect harmony with each other.

In the peripheral, 5' Deiodenase converts one third of T4 to T3, while 5 deiodenase converts 45 percent to rT3. Their metabolism continues to produce diiodothyronines. Thyroid glands only create about 13% of T3, while the rest 87% are derived from T4.

Regulation of thyroid hormone levels

Thyroid stimulating hormone (TSH), also known as thyrotropin, regulates the release of thyroid hormones T3 and T4 and is released by the anterior pituitary gland. It has a molecular weight of 28000. It secretes itself in a pulsatile fashion, with nighttime being the highest secretion time. Tharotropin-releasing hormone (TRH) triggers the release of TSH. A negative feedback loop involving free T3 and T4 regulates the secretion of both TRH and TSH.

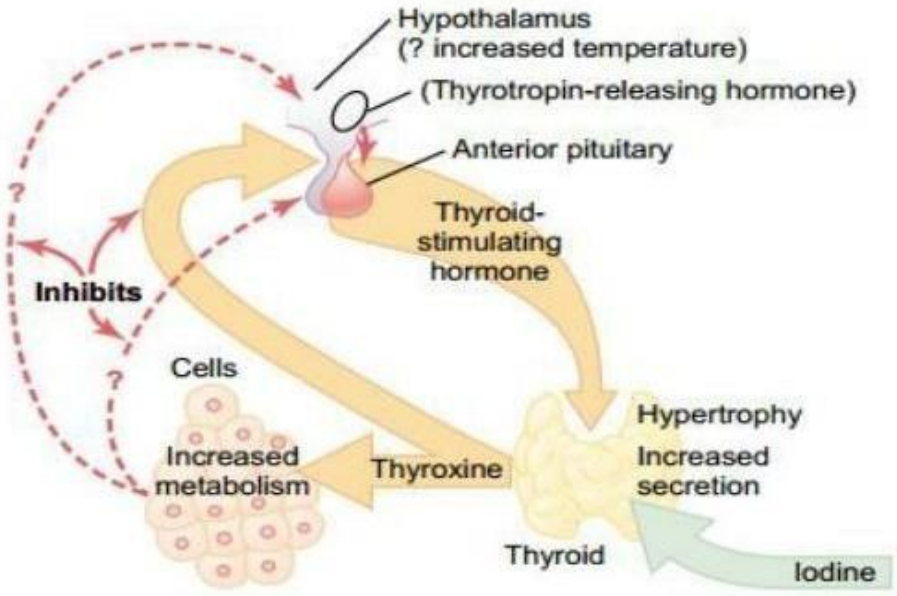


Fig-4 Control of Thyroid hormone

NON THYROIDAL ILLNESS

Changes in blood thyroid hormone, most noticeably to the T3 level, may occur in a broad range of disorders that are not thyroid-related. Under these circumstances, thyroid gland intrinsic disorders go undetected. "Low T3 syndrome," "Sick euthyroid syndrome," "Non thyroid illness syndrome," and "Thyroid hormone adaptation syndrome" were some of the names given to it.

At the outset of a non-thyroid disease state, reverse T3 (rT3) levels rise with total and free T3 (FT3) blood levels.

"Low T3, T4 State" refers to the decline in serum T4 levels that occurs as the disease advances. Free T4 (FT4) stays about the same or even goes down a little, even when total T4 drops. This condition is distinguished from primary hypothyroidism because, despite the decreased T3 and T4 levels, the blood TSH level stays normal or lower. However, a number of investigations have shown that TSH levels may rise somewhat in non-thyroidal illnesses even when hypothyroidism is not present.

NORMAL KIDNEY

Anatomy and physiology

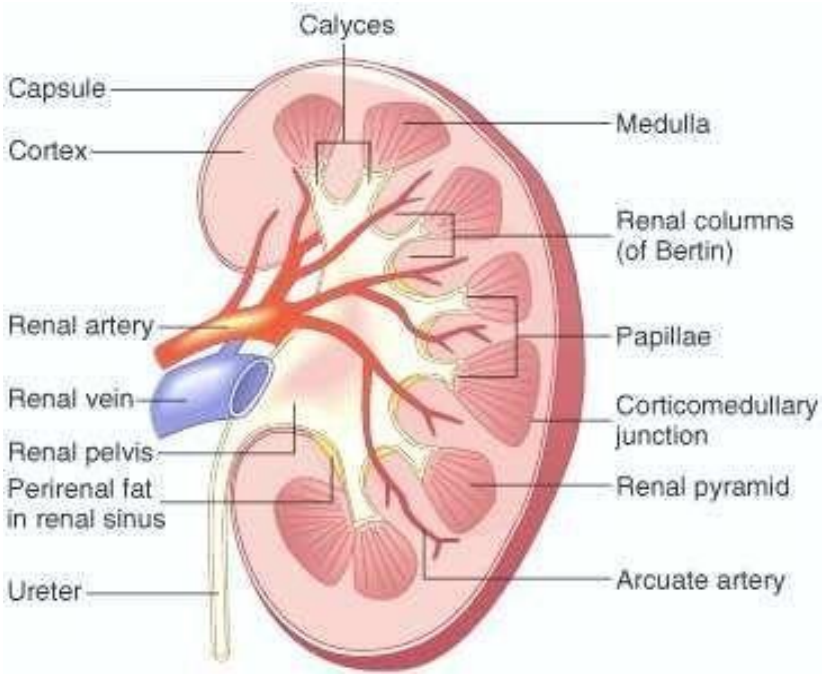


Fig-5 Anatomical Structure of a bisected Kidney

Each of the two sets of kidneys—weighing about 115 to 155 g in adult females and 125 to 170 g in adult males—are retroperitoneal organs located at the back of the abdomen, one on each side of the spinal column. Each kidney is about 11–12 cm in length and 5–7.5 cm in breadth. Approximately 2.5 to 3cm thick. Each kidney has an upper and lower pole. The higher pole is located opposite the 12th thoracic vertebra, while the lower pole is opposite the 3rd lumbar vertebra. The kidney volumes of a man range from 132 to 276 mL, whereas those of a female range from 87 to 223ml. Each kidney has a medial or concave surface called the hilum. The renal pelvis, veins and arteries, lymphatics, and a nerve plexus all go through this surface into the kidney's sinus. Within the kidneys is a delicate, robust, and fibrous covering.

Microscopic Anatomy:

Nephron

The nephron is the basic unit of the kidney's functioning system. There are around 2–2.5 million nephrons in one human kidney. The glomerulus, Bowman's capsule, proximal tubule, thin limbs, distal tubule, and connecting tubule are the fundamental components of a nephron.

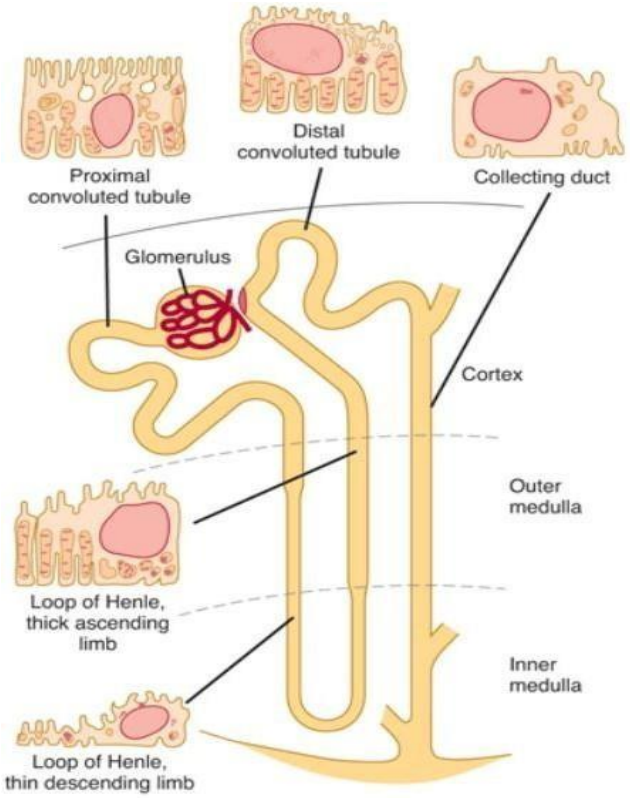


Fig-6 Structure of a Juxtamedullary Nephron

A tuft of capillaries invaginates the enlarged, blind end of the nephron (Bowman's capsule), forming the glomerulus. An afferent arteriole supplies blood to the capillaries, while a diminutive efferent arteriole carries waste products out of the body. It is from the glomerulus that the filtrate is produced.

In Bowman's capsules, the glomerular filtrate and blood are separated by two cellular layers. Podocytes with several pseudopodia that interdigitate to create filtration slits along the capillary wall compose the capillary endothelium and specialised epithelium of the capsule.

Contractile stellate cells, also known as mesangial cells, are involved in glomerular filtration control. In addition to taking up immunological complexes and secreting the extracellular matrix, mesangial cells have a role in the development and progression of glomerular disease.

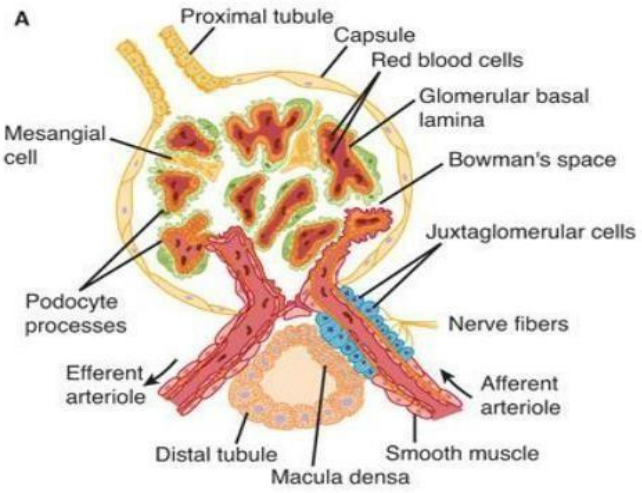


Fig-7 Glomerulus structure showing capillary loops

A diameter of 55 micrometres and a length of 15mm characterise the proximal convoluted tubule. The pars recta and pars convoluta make up the proximal region. After emptying into the loop of Henle, the twisted section flows into the straight section. The three parts that make up the loop of Henle are the descending limb, the thin segment, and the ascending limb.

With a length of about 5 mm, the distal convoluted tubule is made up of the pars recta, which means straight section. At its beginning, it is the rising continuation of the ascending limb of the Henle loop. About 20 mm long collecting ducts are formed when the distal tubules join together.

Blood vessels

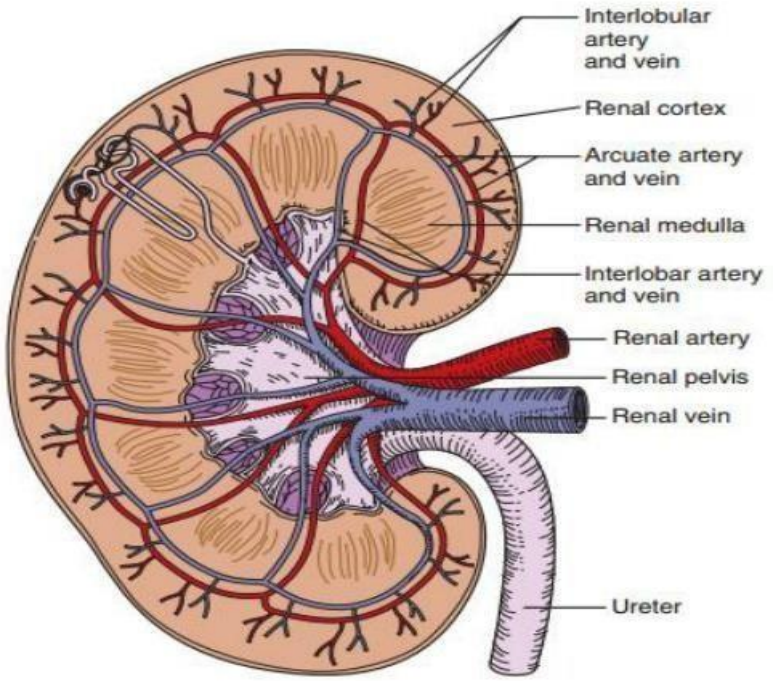


Fig-8 Organization of Renal vasculature

The renal arteries originate at or near the level of the top edge of the second lumbar vertebra and branch off of the abdominal aorta. Each of the five segmental arteries that branch from the renal artery before reaching the renal hilus eventually joins together to create a single vascular segment of the kidney.

In the renal sinus fat, each segmental artery ultimately branches out into at least one interlobar artery. At the foot of the neighbouring pyramid, the interlobar arteries branch out into many arcuate arteries that curve around and rest at the convex base of the pyramid. Interlobular arteries, which originate from the arcuate arteries and go up the cortex to the kidneys' surface, sit between the medullary rays. Afferent arterioles of the glomerulus and the capillaries and arterioles that feed the renal parenchyma originate in the interlobular arteries.

III. METHODOLOGY

Source of data

Patients hospitalised to the Khaja Banda Nawaz Teaching & General Hospital, Kalaburgi, who have chronic renal disease are affiliated with the Faculty of Medical Sciences at KBNU, Kalaburgi.

Data gathering techniques Subjects of the study:

This research looked at one hundred patients admitted to Khaja Banda Nawaz Teaching & General Hospital between March 2021 and August 2022 with a diagnosis of chronic renal disease. The basic random sampling approach is used to choose these samples. The study makes use of parametric and non-parametric tests, as well as statistical measures such as mean, standard deviation (SD), and correlations.

All patients were asked to provide their informed permission.

Enumeration Method: 100 The formula N=Z2 x P (1-P) / D2 was used to get the, with N being the sample size. The confidence level-corresponding statistic is Z. P represents the anticipated frequency and D the degree of accuracy.

Criteria for inclusion

Chronic renal disease patients. Patients with chronic kidney disease who are receiving conservative treatment.

IV. RESULTS

Table No.1: Age and gender wise distribution of patients

Age in years	Males		Females		Total	
	No.	%	No.	%	No.	%
≤ 40	12	19.0	14	37.8	26	26.0
41—50	25	39.7	10	27.0	35	35.0
51—60	8	12.7	4	10.8	12	12.0
61—70	16	25.4	7	18.9	23	23.0
> 70	2	3.2	2	5.5	4	4.0
Total	63	100.0	37	100.0	100	100.0
Mean ± SD	49.79 ± 12.48		47.24 ± 14.12		48.85 ± 13.10	
t-test, P-value	**t = 0.794, P = 0.429, NS**					

NS= not significant, S=significant, HS=highly significant

The research found that 35 patients, or 35.0% of the total, were between the ages of 41 and 50, 26 patients, or 26.0%, were between the ages of 40 and under, and 23 patients, or 23.0%, were between the ages of 61 and 70. Patients' average ages ranged from 48.85 to 49.79 and 47.24 years, respectively. With a range from 23 to 80 years old, patients were evaluated. The mean ages of the sexes do not vary significantly from one another (P>0.05).

Multiple bar diagram represents age wise distribution of patients

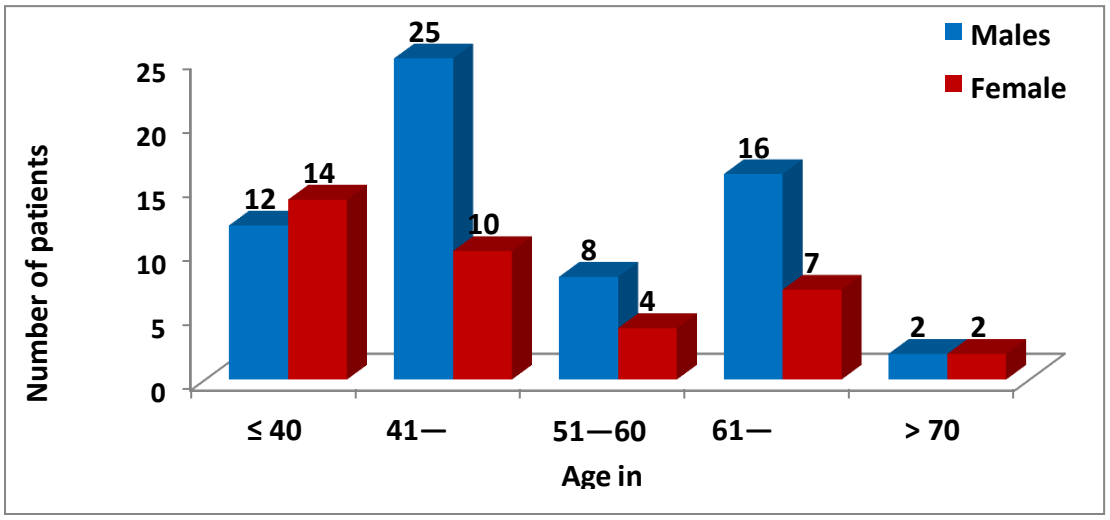


Table No.2: Gender wise distribution of patients

Gender	Number of patients	Percentage
Males	67	67.0
Females	33	33.0
Total	100	100.0

A total of 63(63.0%) male patients and 37(37.0%) female patients participated in the research. The ratio of males to females was 2.03:1.

Pie diagram represents gender wise distribution of cases

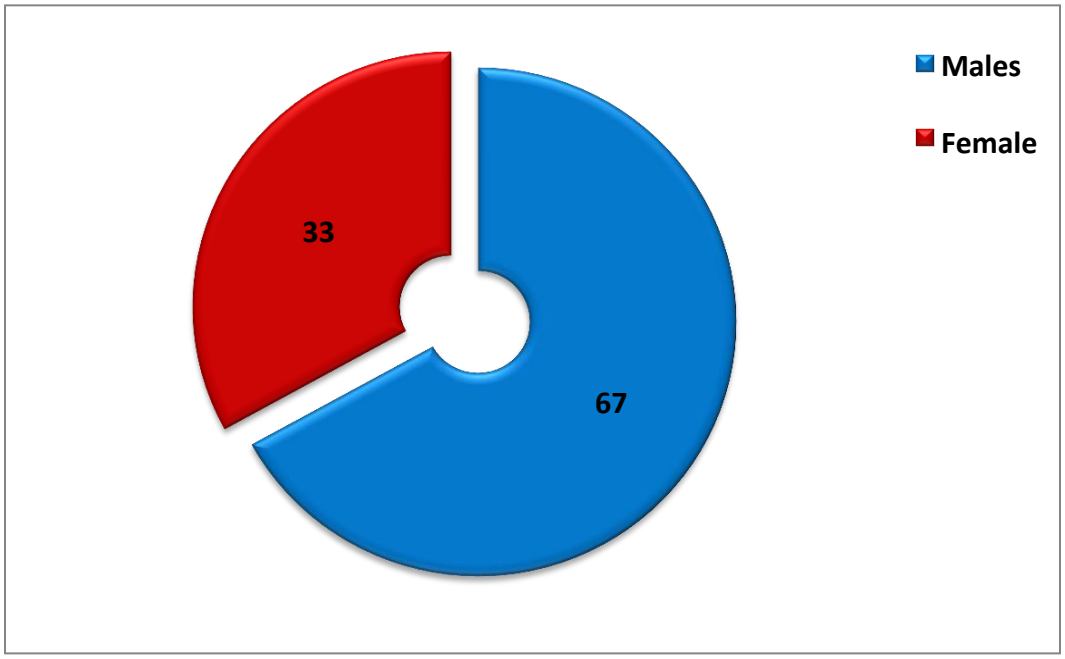


Table No.3: Duration symptoms wise distribution of patients

Duration in months	Number of patients	Percentage
≤ 5 Months	36	36.0
6—10 Months	49	49.0
> 10 Months	15	15.0
Total	100	100.0

Study observed that; out of 100 sample patients, 49 (49.0%) of patients duration of symptoms was 6—10 months and 36 (36.0%) of patients duration of symptoms was ≤5 years .

Simple bar diagram represents duration symptoms wise distributionof patients

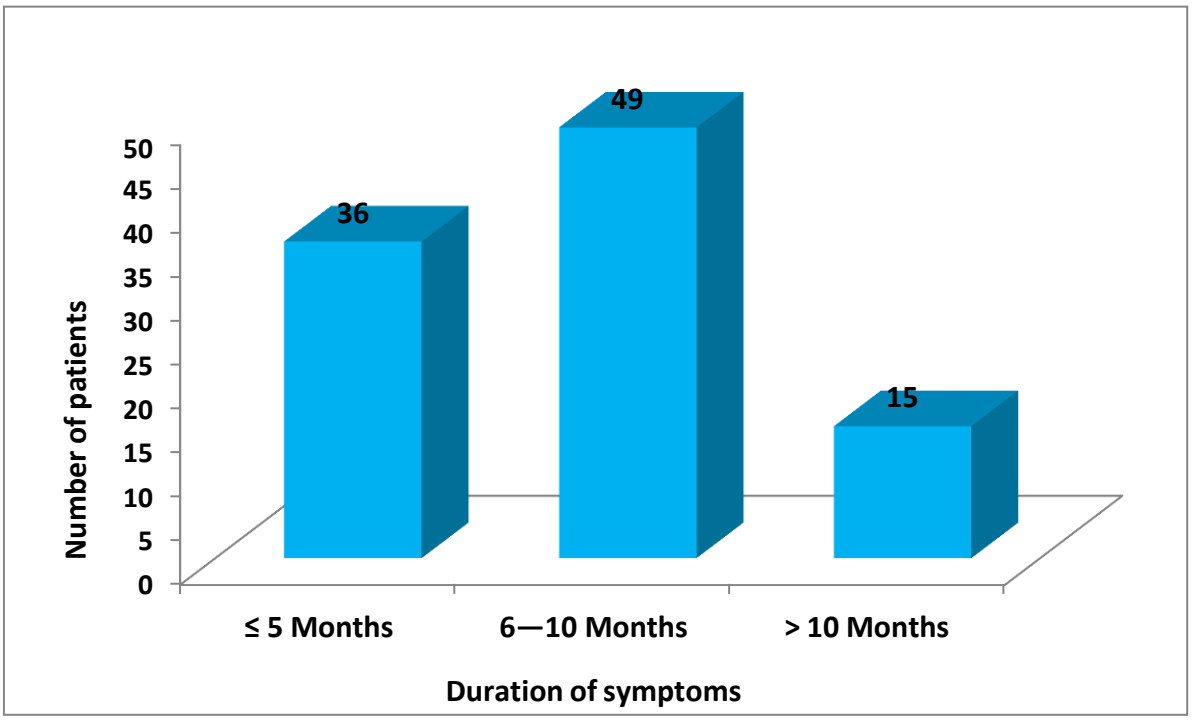


Table No.4: Descriptive statistics of CKD Patients

Variables	No. of patients	Minimum	Maximum	Mean	SD
Age	100	23	80	48.85	13.10
Urea	100	24	186	111.09	41.83
Creatinine	100	0.9	16.4	8.95	5.58
CCI (ml/min)	100	6	46	20.75	13.69
T3	100	0.01	2.0	0.64	0.48
T4	100	0.4	8.5	5.31	2.63
TSH	100	0.7	27	7.66	7.93

The parameters measured in the research were as follows: mean urea level of 111.09, mean creatinine level of 8.95 (with a range of 0.9 to 16.4), and mean creatinine clearance of 20.75 (with a range of 6 to 46). Thyroid function test results showed an average blood T3 of 0.64 (with a range of 0.01-2.00), an average serum T4 of 5.31 (with a range of 0.4-8.5), and an average serum TSH of 7.66 (with a range of 0.7-27.0).

Table No.5: Distribution of low T3 and T4 among various levels of TSH

TSH level µIU/ml	Total no. of Patients	No. of Patients with Low T3	No. of Patients with Low T4
≤ 7	77	41 (53.2%)	26 (33.7%)
7.1—20	8	4 (50.0%)	4 (50.0%)
> 20	15	15 (100.0%)	15 (100.0%)
Total	100	60 (60.0%)	45 (45.0%)
χ2 –Test value, P-value and Significance	**χ2 = 1.231, P = 0.372, NS**		

NS= not significant, S=significant, HS=highly significant

When compared to TSH levels, low T3 and T4 readings do not vary statistically significantly (P>0.05).

Multiple bars shows distribution of low T3 and T4 among various levels of TSH

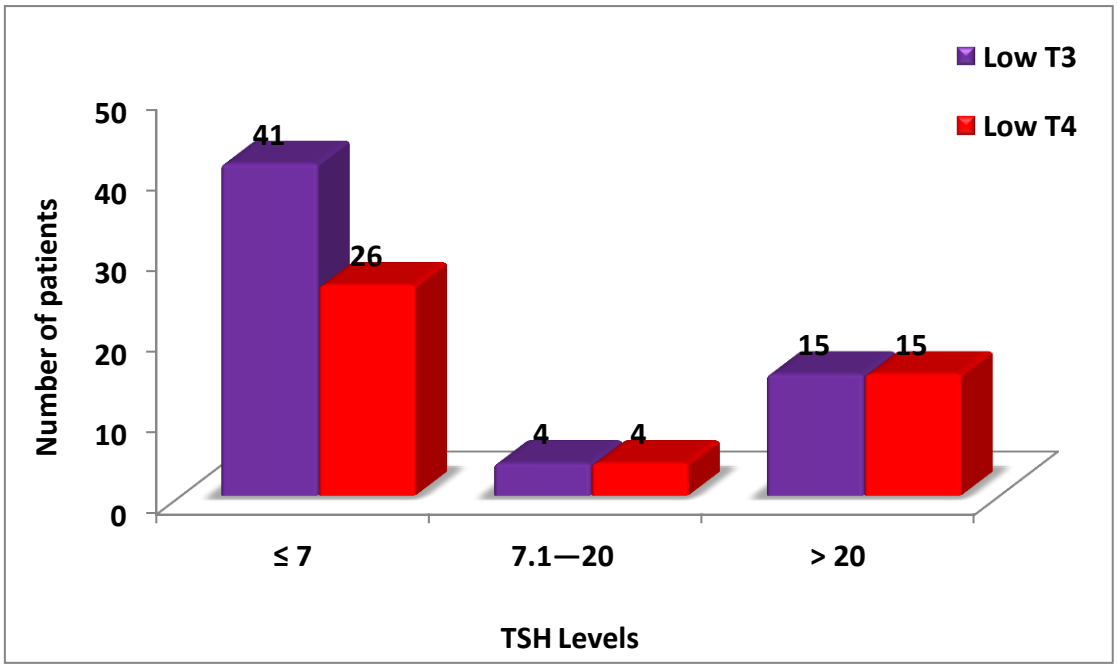


Table No.6: Analysis of thyroid dysfunction in this study

Thyroid dysfunction	Number of patients	Percentage
Low T3 Syndrome	60	60.0
Low T4 Syndrome	45	45.0
Hypothyroidism	15	15.0

According to the study, 60 patients (60.0%) with chronic kidney disease had low T3, 45 patients (45.0%) had low T4, and 15 patients (15.0%) had hypothyroidism.

Simple bars represent analysis of thyroid dysfunction in this study

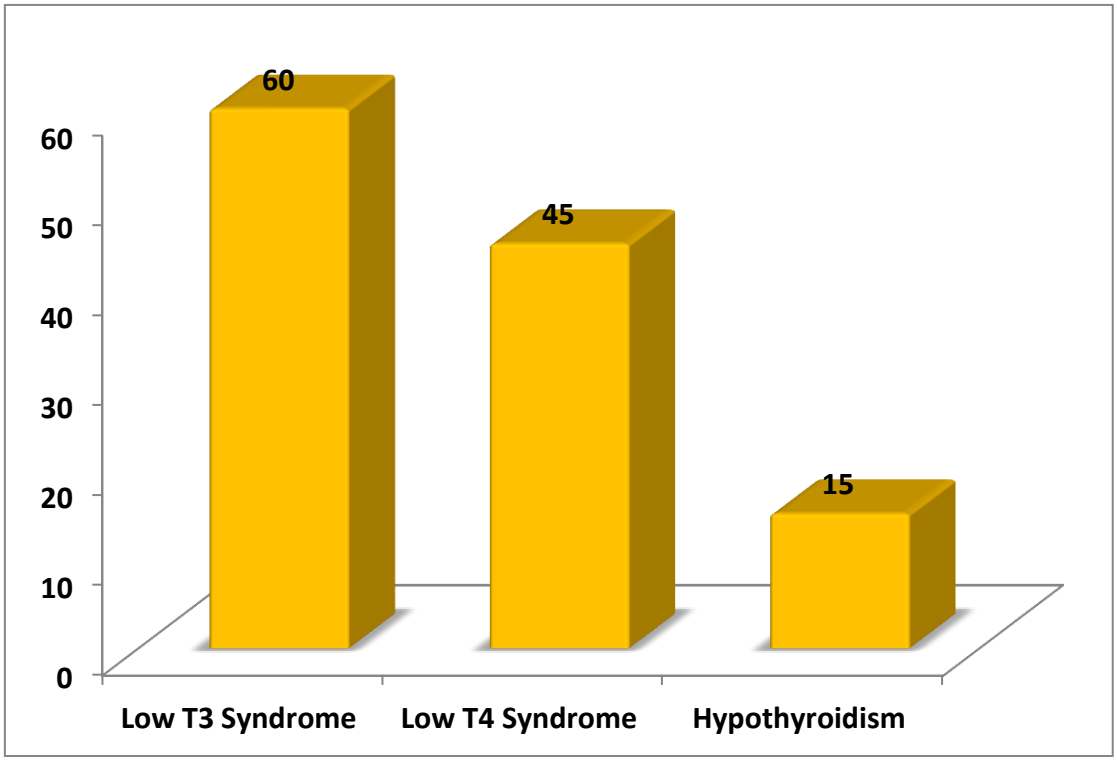


Table No.7: Comparison of serum T3, T4 and TSH

Thyroid dysfunction	**Normal Values**	**Low Values**	**High Values**
T3	40 (40.0%)	60 (60.0%)	0 (0.0%)
T4	65 (65.0%)	45 (45.0%)	0 (0.0%)
TSH	85 (85.0%)	0 (0.0%)	15 (15.0%)
$\chi 2$ –Test value, **P-value & Significance**	**$\chi 2$ = 23.872, P = 0.000, HS**		

NS= not significant, S=significant, **HS=highly significant**

Regarding Normal, low, and high levels, there was a statistically significant variation in the distribution of T3, T4, and TSH (P<0.001).

Multiple bar diagram represents comparison of serum T3, T4 and TSH

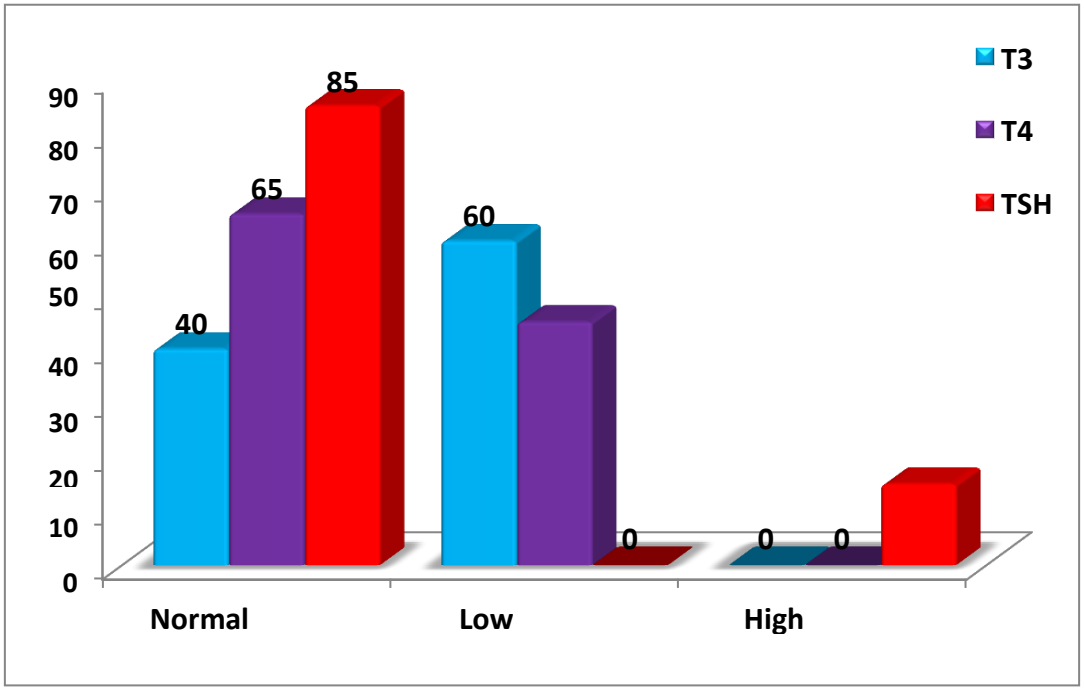


Table No.8: Age incidence/association of Low T3 syndrome

Age in years	Number of patients	Low T3	Low T4
		No. (%)	No. (%)
≤ 40	26	13 (50.0%)	11 (42.3%)
41—50	35	18 (51.4%)	17 (48.8%)
51—60	12	9 (75.0%)	6 (50.0%)
61—70	23	17 (74.0%)	7 (30.4%)
> 70	4	3 (75.0%)	2 (50.0%)
Total	100	60 (60.0%)	45 (45.0%)
Fisher exact test		P = 0.041, S	P = 0.572, NS

A statistically significant correlation between age and low T3 was found (P<0.05). Patients with T3 impairment were much more common as they became older. However, there is no statistically significant correlation between age and low T4. (P>0.05)

Simple bar represents age wise association of Low T3 syndrome

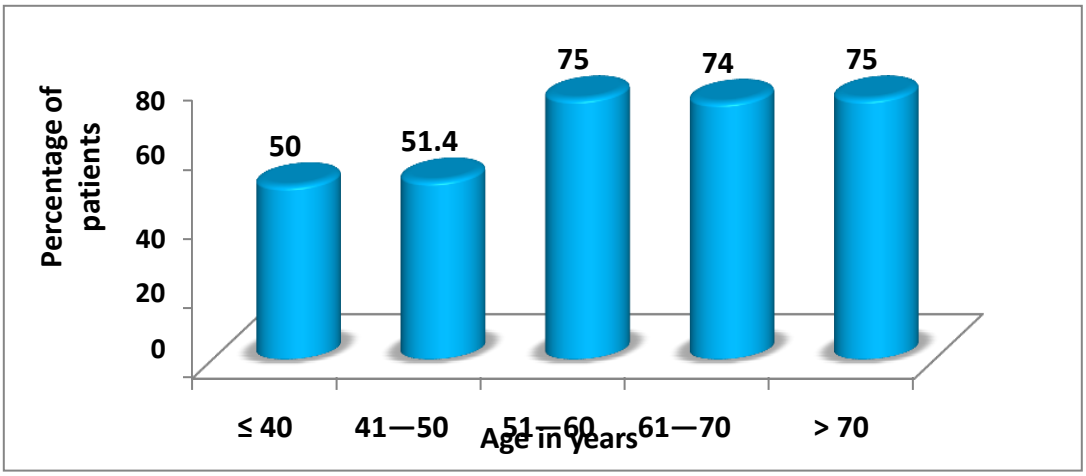
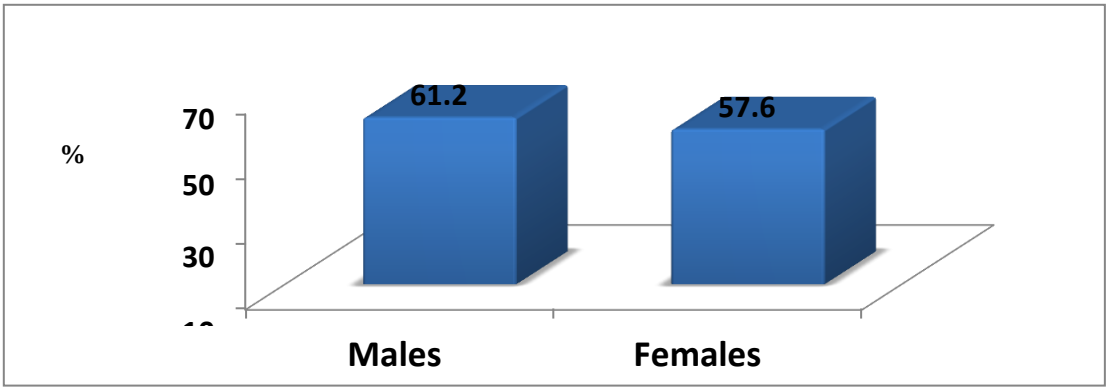


Table No.9: Gender incidence/association of Low T3 syndrome

Gender	Number of patients	Low T3	Percentage
		No. (%)	No. (%)
Males	67	41 (61.2%)	26 (38.8%)
Females	33	19 (57.6%)	19 (57.6%)
Total	100	60 (60.0%)	45.0 (45.0%)
χ2 –Test, P-value		**P = 0.691, NS**	**P = 0.215, NS**

There was no statistical significant association of low T3 and low T4 with respect togender (P>0.05).

Bar diagram represents gender wise association of Low T3 syndrome



Bar diagram represents gender wise association of Low T4 syndrome

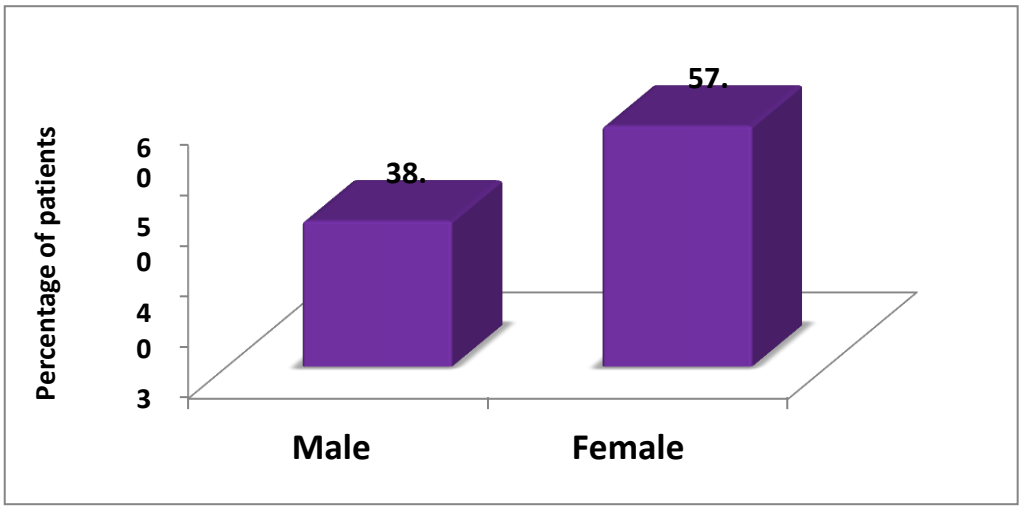


Table No.10: Analysis of hypothyroid symptoms in CKD

Variants	Number of patients with symptoms	Percentage
Low T3 syndrome (N = 60)	36	60.0
Hypothyroidism (N = 15)	15	100.0
CKD with Thyroid dysfunction (N = 35)	28	80.0
Total	79	79.0
$\chi 2$ –Test, P-value	**$\chi 2$ = 7.53 P = 0.036, S**	

According to the study, there was a notable disparity in the distribution of thyroid symptoms among CKD patients with regard to T3 variations, hypothyroidism, and CKD associated with thyroid dysfunction ($P<0.05$). Symptoms of thyroid dysfunction are seen in all people with hypothyroidism.

Table No.11: Distribution of thyroid dysfunction in this study among variouscreatinine clearance levels

Creatinine clearance (ml/mm)	No. of patients	Low T3 Syndrome	Low T4 Syndrome	Hypothyroidism
		No. (%)	No. (%)	No. (%)
<10	39	35 (89.7%)	26 (67.6%)	7 (17.9%)
10—20	8	6 (75.0%)	5 (62.5%)	2 (25.0%)
>20	53	19 (35.8%)	14 (26.4%)	6 (11.3%)
Total	100	60	45	15
χ2 –Test, P-value	--	P = 0.001, HS	P = 0.000, HS	P = 0.085, NS

Cleinine clearance was shown to be significantly associated with low T3 syndrome and T4 (P<0.001), according to the study. A higher percentage of individuals with low T3 and T4 syndrome had a creatinine clearance level below the recommended minimum. Despite this, hypothyroidism and creatinine clearance did not correlate statistically.

Considering the creatinine clearance of individuals with low T3 and T4 distributions (P>0.0%)

Bar diagram represent distribution of thyroid dysfunction in this study among various creatinine clearance levels

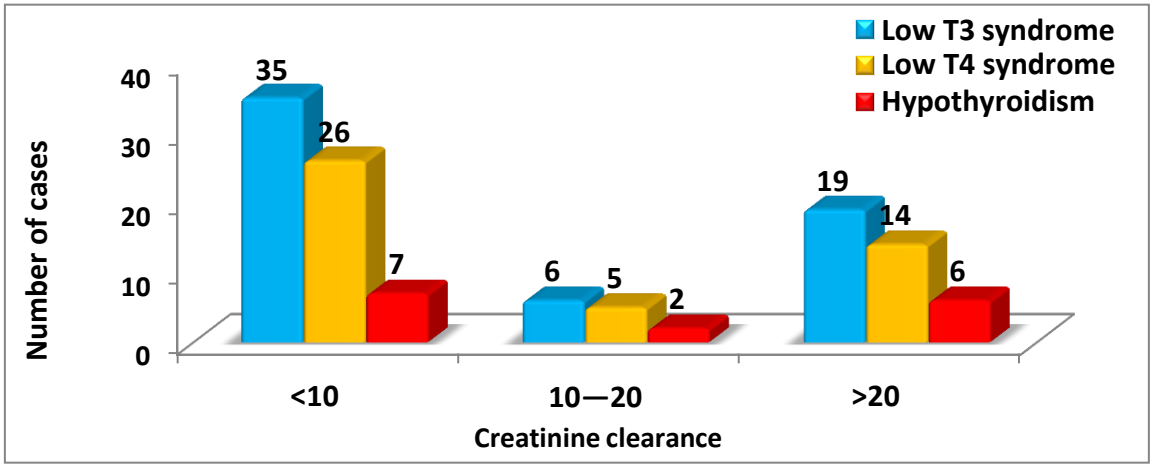
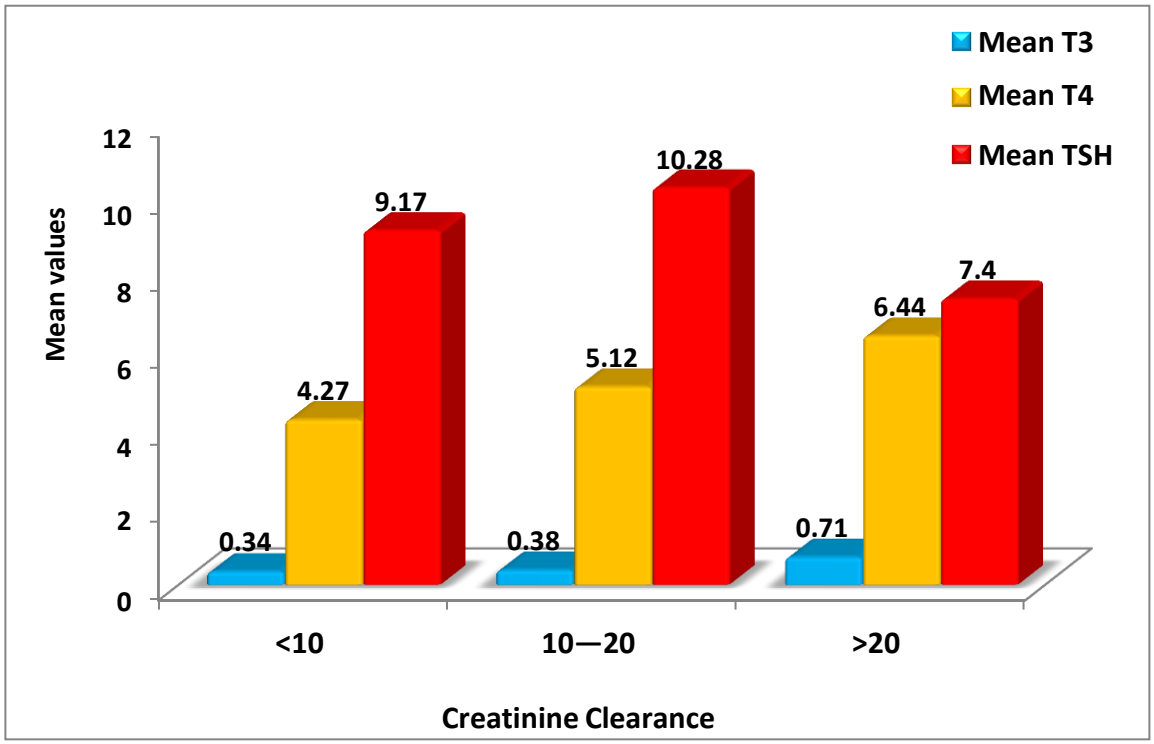


Table No.12: Correlation of thyroid hormones with severity of renal failure

Creatinine Clearance ml/mm	T3 (ng/dl)	T4 (µg/dl)	TSH (µIU/ml)
	Mean ± SD	Mean ± SD	Mean ± SD
<10	0.34 ± 021	4.27 ± 3.03	9.17 ± 10.79
10—20	0.38 ± 0.34	5.12 ± 2.55	10.28 ± 10.58
>20	0.69 ± 0.43	6.44 ± 1.54	7.40 ± 2.21
Total	0.64 ± 0.48	5.31 ± 2.63	7.66 ± 7.93
ANOVA Test, P-value & Significance	F = 13.58, P = 0.000 HS	F = 7.483, P = 0.000 HS	F = 1.89, P = 0.087NS

Correlation of thyroid hormones with severity of renal failure



DISCUSSION

Extensive research on thyroid function in uremic individuals has shown contradictory findings. Several researchers have documented cases of hyperthyroidism, hypothyroidism, and euthyroid states.

Dialysis alters thyroid parameters, which do not rely on the presence or absence of chronic renal disease. In individuals with renal failure, dialysis may alter the earlier thyroid hormone state. Numerous studies, including Ramirez's and Kayima et al.'s, have compared haemodialysis patients with chronic kidney disease

patients receiving conservative management.

Low T3 readings have been seen in many investigations of people with CKD. Several investigations have shown low levels of T3, including those by Ramirez et al., Hegedus et al., Beckett et al., Pon Ajil Singh et al., P Iglesias and JJ Diez, and many more. The degree of renal failure was shown to be linearly correlated with mean serum T3 and T4, according to the research by Ramirez and Spector et al.

Of the 100 CKD patients in our research, 67 were men and 33 were girls, and all of them were treated conservatively. Twelve patients were in the 51-60 age range, thirty-five were in the 41-50 age bracket, and twenty-six were less than 40 years old. The age range of the 23 patients was 61 to 70 years. four patients were 70 and above.

Fifty-three patients had glomerular filtration rates (GFRs) over 20 ml/min, eight patients had GFRs between 10 and 20 ml/min, and 39 patients had GFRs below 10 ml/min.
The proportion of patients with low T3 and T4 levels rises steadily as the GFR declines.

The mean TSH level in our research is within the normal range, excluding hypothyroidism. For the different ranges of GFR, the mean TSH levels are likewise within normal limits. However, there is no linear relationship between TSH level and the degree of renal failure. Rajagopalan B et al. and V. Bhavani et al. found the same thing. Because uraemic individuals' TSH responses to TRH were diminished, these investigations showed that there was an anomaly in the hypophyseal mechanism of TSH release.

Low T3 T4 levels and high TSH levels were seen in other investigations by Joseph et al. and Hardy et al., which may indicate that the pituitary thyroid axis is being maintained.

Ramirez et al. 138 found low T3, low T4, and normal or moderate increase of TSH, which is in line with our data. However, the exact role that these alterations play in the development of symptoms associated with Uraemic syndrome remains unknown. Several investigations have pointed to these abnormalities in the thyroid profile as an adaptive mechanism in the body.

Dialysis

A thyroid profile might be affected by haemodialysis and continuous ambulatory peritoneal dialysis apart from chronic kidney disease (CKD), as mentioned before. The thyroid profile may also be impacted by dialysis-related medications, such as heparin and furosemide.

Research on the effects of dialysis on chronic kidney disease (CKD) patients with thyroid dysfunction has been carried out by Kayima et al. 141 and Giordano et al. 49. In these trials, the thyroid profile did not improve significantly after many rounds of haemodialysis. However, with a TSH below normal, the majority of thyroid function measures recovered to normal in patients who had kidney transplant surgery.

Hypothyroidism

Hypothyroidism is common in chronic kidney disease (CKD), according to prior research by Quion verde et al. Patients with end-stage renal failure had an estimated 5%.

A comprehensive analysis conducted by Kaptein et al. revealed that individuals with chronic renal illness and dialysis were 2.5 times more likely to have primary hypothyroidism. It was estimated that hypothyroidism in CKD ranged from 0% to 9.5%. A titer of anti-thyroid antibodies was predicted to be present in 6.7% of CKD in the Kaptein research.

While 15% of patients in our research had hypothyroidism, we found no association between this condition and kidney failure severity. Hypothyroid and CKD individuals in our research had similar distributions of hypothyroidism symptoms. In CKD patients without hypothyroidism, symptoms of hypothyroidism were more prevalent than in those with hypothyroidism.

Therefore, a low blood T4 level and a very high TSH level (>20 µ IU/dl) are the key diagnostic criteria for hypothyroidism in chronic kidney disease. Hyperthyroidism was not seen in any of the participants analysed in this investigation.

V. CONCLUSION

For those suffering from chronic kidney disease It is possible to see changes in T3 and T4 levels in CKD as protective, encouraging protein conservation, as thyroid dysfunction is seen in most individuals with the disease. Sixty percent of patients and forty-five percent of controls in our research had low T3. Hypothyroidism is more common in those who have chronic renal disease. Low T3 and T4 syndrome is more common in people with more advanced chronic renal disease. With hypothyroidism excluded, 60% of patients had low T3 levels and 45% have low T4 levels.

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