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Research Article

# PSEUDOCHOLINESTERASE AS A BIOCHEMICAL MARKER IN HYPOTHYROIDISM

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**ABSTRACT**

**Context**: In a population-based study done in Cochin on 971 adult subjects, the prevalence of hypothyroidism was found to be 3.9%. In childhood, hypothyroidism can occur. In a clinic-based study from Mumbai, out of 800 children with thyroid disease, 79% had hypothyroidism. There is a delay in the diagnosis of hypothyroidism in the country. This delay is attributable to the lack of awareness about the illness and lack of facilities available for testing of this illness. There are no studies relating to pseudocholinesterase activity in hypothyroidism in the Indian subcontinent and its use as a biochemical marker in hypothyroidism in India.

**Aims**: To assess the serum Pseudocholinesterase activity in hypothyroidism.

To assess the correlation between Serum Pseudocholinesterase and Thyroid function test.

To assess pseudocholinesterase as a diagnostic marker in Hypothyroidism.

**Settings and Design**: This study was conducted in R.L. Jalappa Hospital during the years 2011-2012. A comparative case-control study was done on 48 hypothyroid patients attending outpatient clinic and those admitted in the medical wards.

**Methods and Material**: Newly detected and old cases of hypothyroidism were taken into the study. They were made into two groups respectively. A separate control group of 54 euthyroid patients were taken with age and sex matched.

Statistical analysis used: Descriptive and inferential statistical analysis has been carried out in the present study. Analysis of variance has been used to find the significance of study parameters between three or more groups of patients. Chi-square/ Fisher Exact test has been used. ROC curve analysis is performed to prove the Pseudocholinesterase as diagnostic marker.

**Results**: Patients who were newly diagnosed with hypothyroidism had a mean pseudocholinesterase level of 3170.19U/l. Hypothyroid patients on treatment showed a mean pseudocholinesterase level of 6320.32 U/l. Serum Pseudocholinesterase showed a negative correlation (-0.374) with TSH in the hypothyroid patients on treatment. Pseudocholinesterase levels in hypothyroids on treatment are more close to controls with P=0.189.

**Conclusions**: Serum pseudocholinesterase activity is decreased by about 31.6 % in hypothyroidism. Pseudocholinesterase was found to be a good biochemical marker for detecting new cases of hypothyroidism with a sensitivity of 100% and specificity of 96.2%. Serum Pseudocholinesterase levels may be used as a biochemical marker for diagnosis and prognosis in hypothyroidism.

**Keywords**: Pseudocholinesterase, Biochemical marker, Thyroid, TSH, Hypothyroidism, India, Kolar, Karnataka.

**Key Messages:** Pseudocholinesterase was found to be a diagnostic marker in detecting new cases of hypothyroidism with high sensitivity and high specificity. Pseudocholinesterase levels in hypothyroids on treatment are more close to controls with P=0.189 indicating this as a prognostic factor. There is also a negative correlation (r value -0.374) between pseudocholinesterase and TSH in hypothyroids on treatment.

## INTRODUCTION

Hypothyroidism results from reduced effects of thyroid hormone on tissues. Hypothyroidism is more common in women with a total prevalence of 1% to 2%, [1] and increases with age (approximately 10% in adults greater than 65 years). In the U.S. population, prevalence of biochemical hypothyroidism is 4.6%, but clinically evident hypothyroidism is present in 0.3%.[2] Congenital hypothyroidism is among the most common congenital diseases, with an incidence of 1/4000 newborns. [3]

In childhood, hypothyroidism can occur. In a clinic-based study from Mumbai, out of 800 children with thyroid disease, 79% had hypothyroidism. Thyroid dysgenesis, dyshormonogenesis, and thyroiditis were the common causes of hypothyroidism in these children. There is a delay in the diagnosis of hypothyroidism in the country. This delay is attributable to the lack of awareness about the illness and lack of facilities available for testing of this illness.[3]

In a population-based study done in Cochin on 971 adult subjects, the prevalence of hypothyroidism was found to be 3.9%.[4] The prevalence of subclinical hypothyroidism was also high in this study, the value being 9.4%. In women, the prevalence was higher, at 11.4%, when compared with men, in whom the prevalence was 6.2%. The prevalence of subclinical hypothyroidism increased with age.[5] There are no studies relating to pseudocholinesterase activity in hypothyroidism in the Indian subcontinent and its use as a biochemical marker in hypothyroidism in India.

Pseudocholinesterase is synthesized in liver and its serum activity is influenced by liver disease. Pseudocholinesterase level in serum is a useful test of liver function.[6] Only liver and gonads display the marked sex difference in enzyme content characteristic of the serum. The gonads are not necessary to the synthesis since castrates respond to administration of estrogen by an elevation of liver and serum cholinesterases. In both sexes a relatively constant enzyme threshold in the liver, above which level the esterase is liberated into the serum, constitutes a simpler explanation of the known facts than does a differential concentrating mechanism from serum to liver.[7] The liver produces serum albumins [7], and serum cholinesterase has been associated with the albumin fraction of serum proteins [7]. In keeping with the last statement are the facts that liver damage lowers serum albumins [7] and liver and serum cholinesterases [7]; liver diseases lower both serum albumins and cholinesterase in humans [7]; the "alarm reaction" lowers serum albumin concentrations [7] and serum choline esterase [7] ; estrogens elevate serum albumin levels [7] and serum non-specific cholinesterase [7]. The slight sex difference in liver specific cholinesterase content (mecholyl hydrolysis) is not statistically significant (P > 0.1).

Two papers have demonstrated that the amount of non-specific cholinesterase in rat serum is controlled at least in part by sex hormones. Estrogen elevates the serum enzyme level and testosterone depresses it, while progesterone exerts no noticeable effect except indirectly through estrogen.[7]

Hypothyroidism can lead to low levels of SHBG (sex hormone binding globulin) which in turn can lead to higher concentrations of

free testosterone and increased testosterone throughout the body.[8] For both sexes, hyperthyroidism was associated with significant elevations of the mean total testosterone and sex hormone-binding globulin (SHBG) levels and significant depressions of the mean percentage and concentration of non-SHBG-bound testosterone and the mean percentage of free testosterone. For women, the mean free testosterone concentration was significantly lower during hyperthyroidism than during euthyroidism.[9]

In a study on Pseudocholinesterase activity in thyroid disease, 12 patients with myxoedema had a 30 % decrease in mean activity for pseudocholinesterase level than that found in random normal's using either substrate. Even when myxoedema is not associated with a subnormal esterase activity, a rise in activity does occur when the patient becomes euthyroid. In this study, treatment restored the esterase level to the average mean activity of random normal as the patient became euthyroid. There is no evidence in the study that any of the pseudocholinesterase variants modified the conclusions.[10]

Decreased levels of Serum cholesterol and beta-lipoproteins occurring in hyperthyroidism were found to be accompanied by an enhanced activity of pseudocholinesterase, while in patients with myxedema, the pathologically increased levels of serum cholesterol and beta-lipoproteins were associated with diminished serum pseudocholinesterase activity. The percentage of the pre-beta fraction was found to be increased in both hypo- and hyperthyroid patients, but mechanisms leading to this change are probably different in the two pathological conditions. The behaviour of cholesterol and pseudocholinesterase activity and especially the ratio between these paramameters might be used for the diagnosis of thyroid disease and for the control of therapeutic efficiency.[11]

Sex-hormone binding globulin, ferritin or LDL cholesterol have been used as endpoints in clinical studies of the responsivity of the liver to thyroid hormone in patients with thyroid hormone resistance.[12]

The objectives of this study are to assess the serum Pseudocholinesterase activity in hypothyroidism, to assess the correlation between Serum Pseudocholinesterase and Thyroid function test and to assess pseudocholinesterase as a diagnostic marker in Hypothyroidism.

**Subjects and Methods**

This study was a comparative case control study conducted in the R.L. Jalappa Hospital and research centre during the years 2011-2012. The study was done on 48 hypothyroid patients attending outpatient clinic and those admitted in the medical wards.

The study group consisted of patients above the age of 18 years. All freshly detected and old cases of hypothyroidism, irrespective of duration of hypothyroidism and type of treatment receiving were taken into the study. They were made into two groups respectively. A control group of 54 euthyroid individuals without any previous thyroid diseases were taken with both age and sex matched. The exclusion criteria were all cases of organophosphorus poisoning, cardiac failure, uremia, acute hepatitis, cirrhosis of liver, severe anemia (<7.0g/dl)[13], vysya community, third trimester of pregnancy, previously diagnosed cases of malignancies including head, neck, lung, cervix and colon.

History, physical examination, Thyroid function test, Pseudocholinesterase level, standard 12 lead ECG, hemoglobin, blood urea, AST/ALT, serum bilirubin, ALP and stool occult blood were done for all patients.

An immunometric immunoassay technique is used for TSH estimation. A competitive immunoassay technique is used for T4 and T3 estimation. Pseudocholinesterase estimation was done by the method of reflectance spectrophotometry. Cholinesterase hydrolyzes butyryl-thiocholine to thiocholine. The liberated thiocholine reduces potassium hexacyanoferrate III to potassium hexacyanoferrate II. The rate of change in reflection density is proportional to the cholinesterase activity in the sample. The rate of colour loss is monitored by reflectance spectrophotometry.

**Statistical Methods**

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients , Student t test ( two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis) on metric parameters. Levene's test for homogeneity of variance has been performed to assess the homogeneity of variance. Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. ROC curve analysis is performed to prove the Pseudocholinesterase (U/L) as diagnostic marker. [14-18]

Statistical software: The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data. Microsoft word and Excel have been used to generate graphs, tables.

**RESULTS**

Among the 48 hypothyroid patients studied, age distribution showed 35.4% (n=17) were between the age of 31-40 years (table 1 and Figure 1). Gender distribution showed 6.2% of hypothyroid patients were males and 93.8% were females when both groups were considered together (Table 2). 33.36% of patients on treatment were found to be on more than 50 micrograms of thyroxine (Table 3) and 54.5% were on a duration of treatment for 1-5 years. (Table 4 and figure 2). Comparison of study parameters including TSH and T4 among the study groups were found to be statistically significant (Table 5 and figure 3). Thyroid parameters among each group were separately analysed and shown in Table 6 and figure 4. The Mean Pseudocholinesterase (U/L) is significantly less in newly detected hypothyroidism cases with P <0.001** with a mean activity of 3170.19±1430.53 U/L (Table 7).

The most common symptoms in among hypothyroid patients both newly detected and on treatment were tiredness & weakness (75%), dry skin (50%) and hair loss (52%) (Table 8 and figure 5). The most common signs among hypothyroid patients both newly detected and on treatment were dry coarse skin (64.5%), alopecia (33.3%) and puffy face, hands and feet (25%) (Table 9 and figure 6).

Comparison of study parameters including TSH, T4, T3 and pseudocholinesterase among the study groups were found to be statistically significant (<0.001**, <0.001**, <0.001** respectively) (Table 10 and figure 7).

Pseudocholinesterase (U/L) is diagnostic marker in detecting new cases of hypothyroidism with Area under ROC curve is 0.999, indicating good marker with high sensitivity (100%) and high specificity (96.2%). (Table 11 and Graph 1).Pseudocholinesterase (U/L) is significantly less in cases not on treatment with F=78, P<0.001**, However the cases on treatment are more close to controls with P=0.189 indicating this as a good prognostic factor (Table 12).

In addition there is convincing evidence from our study showing that there is a negative correlation **(r value** -0.374) between pseudocholinesterase and TSH in hypothyroid patients on treatment (Table 13 and Figure 8).

**DISCUSSION**

Amidst few studies on Pseudocholinesterase activity in Hypothyroidism, the present comparative case control study tried to determine the same in hypothyroid patients attending a tertiary care teaching hospital in kolar.

In the present study 26 newly detected hypothyroid patients had a 53 % decrease in mean activity for pseudocholinesterase level than that found in random normal's. When all hypothyroid patients were

considered, a 31.6% decrease in mean activity for pseudocholinesterase level was found. As compared to a previous study by Thompson J.C et al, 12 patients with myxoedema had a 30 % decrease in mean activity for pseudocholinesterase level than that found in random normal's. The study also showed that even when myxoedema is not associated with a subnormal esterase activity, a rise in activity does occur when the patient becomes euthyroid. Thompson J.C et al found that treatment restores the esterase level to the average mean activity of random normal as the patient becomes euthyroid. There was no evidence in the present investigation that any of the pseudocholinesterase variants modify these conclusions. In our study we found that treatment with thyroxine supplementation restored the esterase level approximately near to the average mean activity of random normal as a patient became euthyroid.

Vlaicu R et al showed that decreased levels of Serum cholesterol and beta-lipoproteins occurring in hyperthyroidism were found to be accompanied by an enchanced activity of pseudocholinesterase, while in patients with myxedema, the pathologically increased levels of serum cholesterol and beta-lipoproteins were associated with diminished serum pseudocholinesterase activity. The behaviour of cholesterol and pseudocholinesterase activity and especially the ratio between these paramameters might be used for the diagnosis of thyroid disease and for the control of therapeutic efficiency. In the present study we estimated pseudocholinesterase as diagnostic and prognostic marker for hypothyroid cases. Pseudocholinesterase was found to be a diagnostic marker in detecting new cases of hypothyroidism with Area under ROC curve is 0.999, indicating a good marker with high sensitivity (100%) and high specificity (96.2%). Pseudocholinesterase is significantly less in cases not on treatment with F=78, P<0.001**, however the cases on treatment are more close to controls with P=0.189 indicating this as good prognostic factor.

In addition there is convincing evidence from our study showing that there is a negative correlation **(r value** -0.374) between pseudocholinesterase and TSH in hypothyroid patients on treatment.

**Table 1: Age distribution of patients studied**

| Age in years | Newly detected hypothyroid patients | | Hypothyroid patients on treatment | | Controls | |
|---|---|---|---|---|---|---|
| | No | % | No | % | No | % |
| 19-20 | 0 | 0.0 | 0 | 0.0 | 4 | 7.4 |
| 21-30 | 6 | 23.0 | 7 | 31.81 | 7 | 12.9 |
| 31-40 | 9 | 34.61 | 8 | 36.36 | 16 | 29.6 |
| 41-50 | 4 | 15.38 | 3 | 13.63 | 14 | 25.9 |
| 51-60 | 2 | 7.69 | 1 | 4.54 | 6 | 11.1 |
| >60 | 5 | 19.23 | 3 | 13.63 | 7 | 12.9 |
| Total | 26 | 100.0 | 22 | 100.0 | 54 | 100.0 |
| **Mean ±SD** | **42.92±16.39** | | **39.36±13.72** | | **43.29±14.16** | |

**Samples are age matched with P = 0.623**

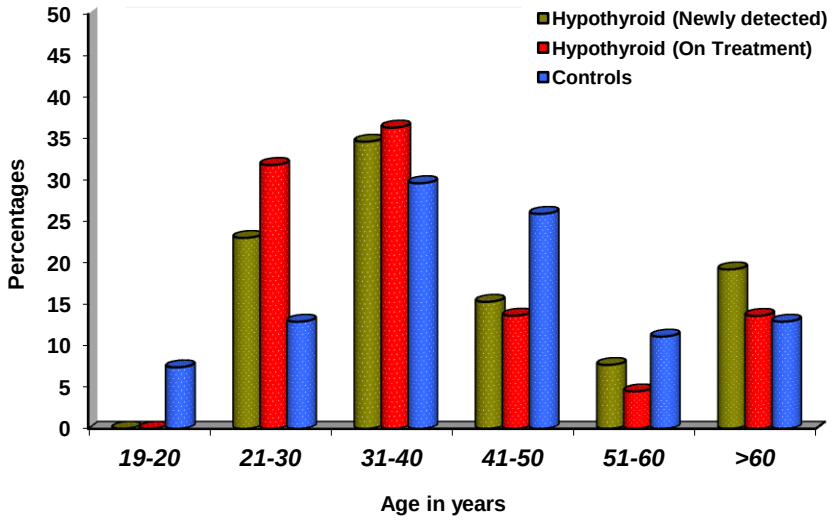



**Figure 1: Age distribution of patients studied.**

**Table 2: Gender distribution of patients studied.**

| Gender | Hypothyroid (Newly detected) | | Hypothyroid (on treatment) | | Controls | |
|---|---|---|---|---|---|---|
| | No | % | No | % | No | % |
| Male | 2 | 7.69 | 1 | 4.54 | 4 | 7.4 |
| Female | 24 | 92.3 | 21 | 95.45 | 50 | 92.6 |
| Total | 26 | 100.0 | 22 | 100.0 | 54 | 100.0 |

**Samples are gender matched with p=0.451**

**Table 3: Treatment with Thyroxine supplementation**

| Treatment (Thyroxine in micrograms) | Number of patients | % |
|---|---|---|
| <50 | 14 | 63.6 |
| >50 | 8 | 33.36 |
| Total | 22 | 100.0 |

**Table 4: On Treatment with thyroxine (years)**

| On Treatment (years) | Number of patients | % |
|---|---|---|
| < 1 year | 7 | 31.81 |
| 1-5 years | 12 | 54.54 |
| >5 years | 3 | 13.63 |
| Total | 22 | 100.0 |

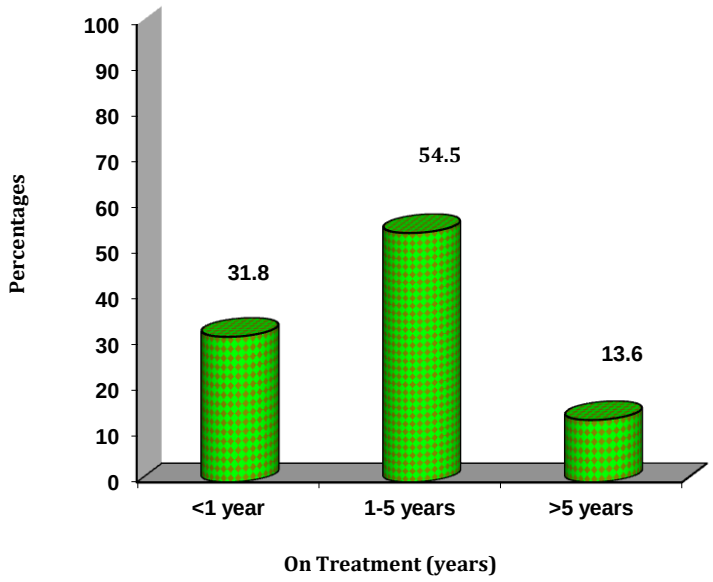



**Figure 2: On Treatment with thyroxine (years).**

**Table 5: Comparison of study parameters among three groups studied**

| Variables | Hypothyroid (Newly detected) | Hypothyroid (On Treatment) | Controls | P value |
|---|---|---|---|---|
| TSH(mcIU/ml) | 52.29±8.50 | 9.65±2.90 | 2.24±1.44 | <0.001** |
| T4 (mcg/dl) | 5.35±0.67 | 8.57±0.57 | 8.86±0.28 | <0.001** |
| T3(ng/ml) | 0.90±0.11 | 1.81±0.39 | 1.15±0.05 | <0.001** |

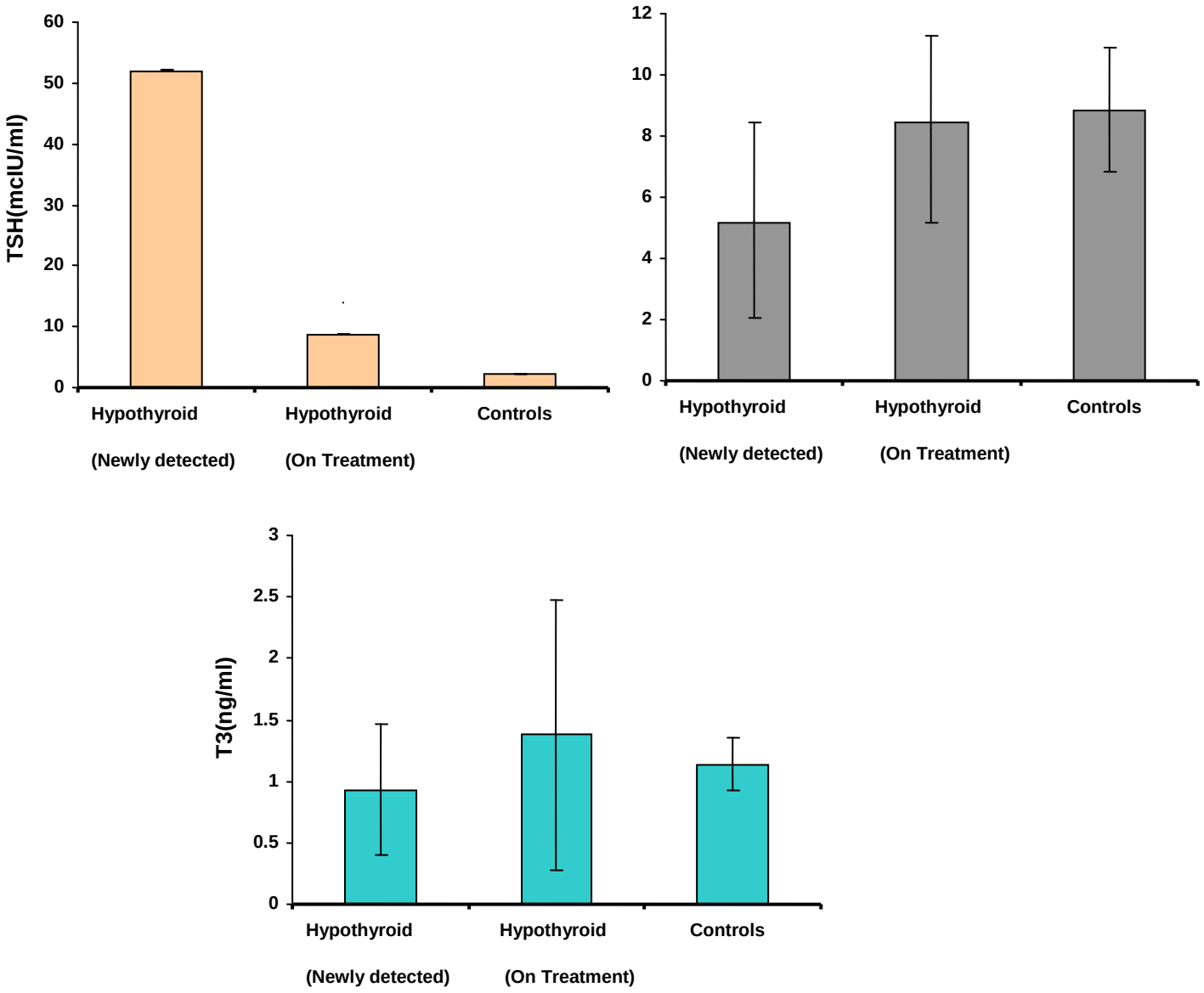



**Figure 3: Comparison of study parameters among three groups studied.**

**Table 6: Comparison of Thyroid parameters**

| Thyroid parameters | Hypothyroid (Newly detected) | | Hypothyroid (On Treatment) | | Controls | |
|---|---|---|---|---|---|---|
| | No | % | No | % | No | % |
| TSH (mcIU/ml) | **(n=26)** | | **(n=22)** | | **(n=54)** | |
| • <0.5 | 0 | 0.0 | 0 | 0.0 | 2 | 3.7 |
| • 0.5-4.7 | 0 | 0.0 | 6 | 27.27 | 48 | 88.9 |
| • >4.7 | 26 | 100.0 | 16 | 72.7 | 4 | 7.4 |
| T4 (mcg/dl) | **(n=26)** | | **(n=16)** | | **(n=54)** | |
| • <5.4 | 15 | 57.69 | 1 | 6.25 | 3 | 5.5 |
| • 5.4-11.7 | 11 | 42.30 | 14 | 87.5 | 47 | 87.0 |
| • >11.7 | 0 | 0.0 | 1 | 6.25 | 4 | 7.4 |
| T3(ng/ml) | **(n=23)** | | **(n=14)** | | **(n=54)** | |
| • <2.3 | 22 | 95.65 | 11 | 78.57 | 54 | 100.0 |
| • 2.3-4.2 | 1 | 4.34 | 2 | 14.28 | 0 | 0.0 |
| • >4.2 | 0 | 0.0 | 1 | 7.14 | 0 | 0.0 |

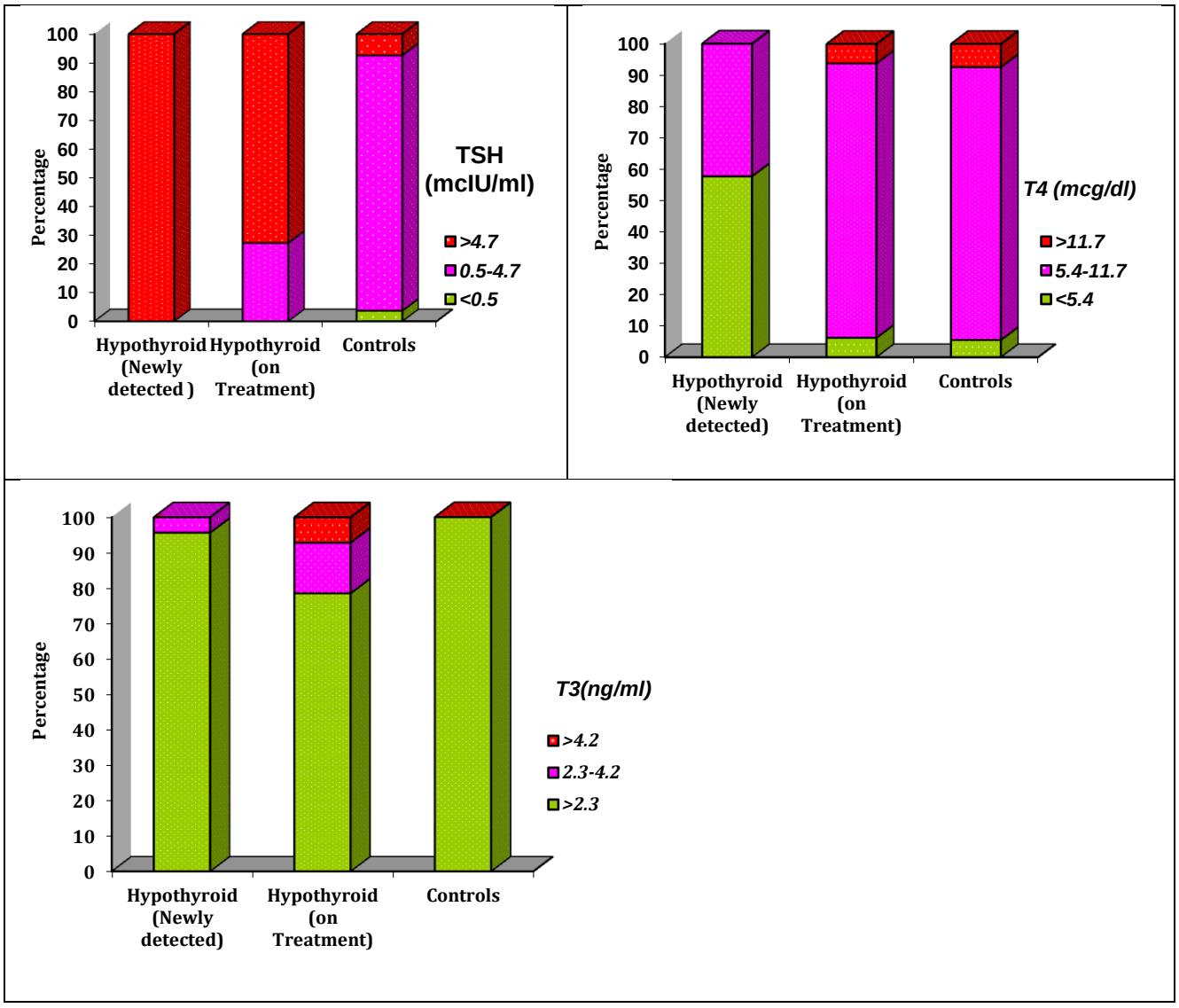



**Figure 4: Comparison of thyroid parameters in each study group.**

**Table 7: Pseudocholinesterase (U/L)**

| Pseudocholinesterase | Hypothyroid (Newly detected) | | Hypothyroid (On treatment) | | Controls | |
|---|---|---|---|---|---|---|
| | No | % | No | % | No | % |
| <1000 | 2 | 7.69 | 0 | 0.0 | 0 | 0.0 |
| 1000-2000 | 4 | 15.38 | 0 | 0.0 | 0 | 0.0 |
| 2000-4000 | 9 | 34.61 | 0 | 0.0 | 0 | 0.0 |
| 4000-6000 | 11 | 42.30 | 9 | 40.9 | 34 | 62.9 |
| 6000-8000 | 0 | 0.0 | 10 | 45.45 | 17 | 31.4 |
| >8000 | 0 | 0.0 | 3 | 13.63 | 3 | 5.6 |
| Total | 26 | 100.0 | 22 | 100.0 | 54 | 100.0 |
| **Mean ±SD** | **3170.19±1430.53** | | **6320.32±1553.25** | | **6753.20±1173.58** | |

**Mean Pseudocholinesterase (U/L) is significantly less in newly detected hypothyroidism cases with P <0.001****

**Table 8: Frequency of Symptoms among the two hypothyroid groups.**

| Symptoms | Hypothyroid (Newly detected ) (n=26) | | Hypothyroid (On Treatment) (n=22) | |
|---|---|---|---|---|
| | No | % | No | % |
| 1.Tiredness & weakness | 26 | 100.0 | 10 | 45.4 |
| 2.Dry skin | 20 | 76.92 | 4 | 18.18 |
| 3.Feeling cold | 14 | 53.84 | 2 | 9.09 |
| 4.Hair loss | 22 | 84.61 | 3 | 13.6 |
| 5.Difficulty in concentration & poor memory | 6 | 23.07 | 1 | 4.5 |
| 6.Constipation | 12 | 46.15 | 3 | 13.6 |
| 7.Weight gain with poor appetite | 11 | 42.30 | 2 | 9.09 |
| 8.Dyspnea | 1 | 3.8 | 0 | 0.0 |
| 9.Hoarse voice | 4 | 15.38 | 1 | 4.5 |
| 10.Menorrhagia | 5 | 19.23 | 0 | 0.0 |
| 11.Parasthesia | 4 | 15.38 | 0 | 0.0 |
| 12.Impaired hearing | - | - | - | - |

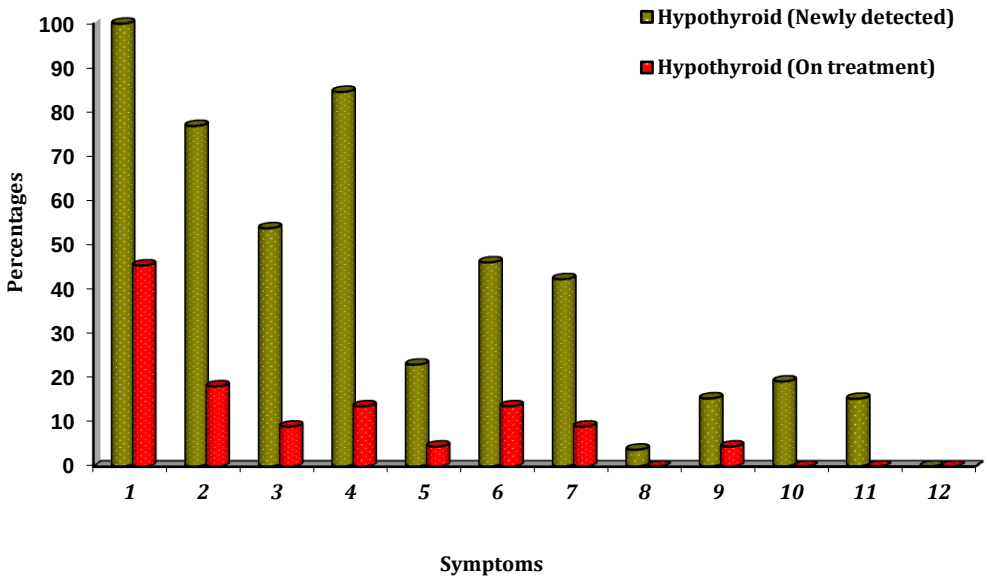



**Figure 5: Frequency of Symptoms among the two hypothyroid groups.**

**Table 9: Frequency of Signs among the two groups of hypothyroid patients studied.**

| Signs | Hypothyroid (Newly detected) (n=26) | | Hypothyroid (On Treatment) (n=22) | |
|---|---|---|---|---|
| | No | % | No | % |
| 1.Dry coarse skin | 26 | 100 | 5 | 22.7 |
| 2.Puffy face, hands & feet | 10 | 38.4 | 2 | 9.09 |
| 3.Diffuse alopecia | 14 | 53.8 | 2 | 9.09 |
| 4.Bradycardia | - | - | - | - |
| 5.Peripheral oedema | 3 | 11.5 | 0 | 0.0 |
| 6.Delayed tendon reflex relaxation | 11 | 42.3 | 1 | 4.5 |
| 7.Carpal tunnel syndrome | - | - | - | - |
| 8.Serous cavity effusions | - | - | - | - |

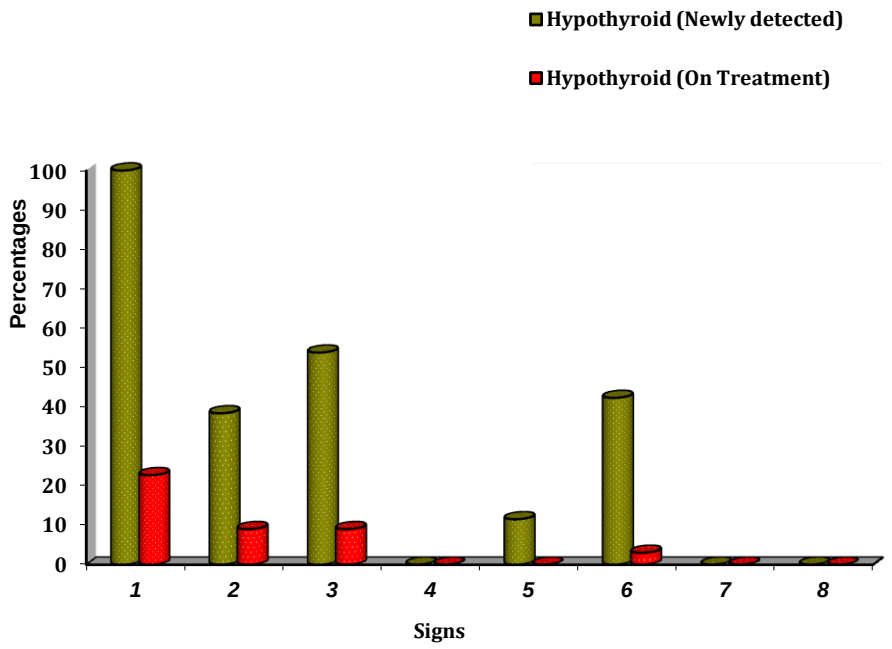



**Figure 6: Frequency of Signs among the two groups of hypothyroid patients studied.**

**Table 10: Comparison of study variables in cases and controls.**

| Variables | Cases (n=48) | Controls (n=54) | P value |
|---|---|---|---|
| Age in years | 41.50±15.01 | 43.29±14.16 | 0.535 |
| TSH (mcIU/ml) | 35.75±39.3 | 2.23±1.44 | <0.001** |
| T4 (mcg/dl) | 6.58±3.38 | 8.86±2.04 | <0.001** |
| T3(ng/ml) | 1.24±1.07 | 1.15±0.22 | 0.502 |
| Pseudocholinesterase (U/L) | 4614±2163.9 | 6753.20±1173.6 | <0.001** |

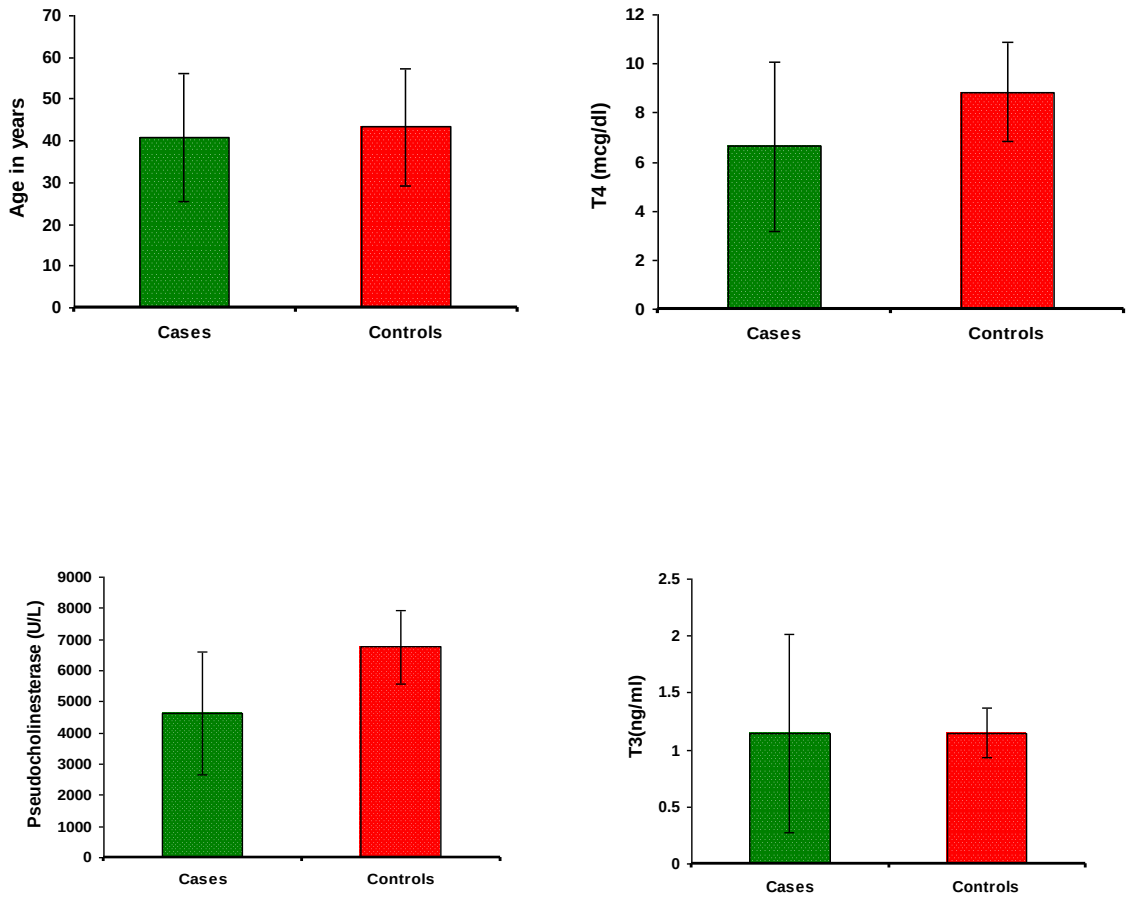
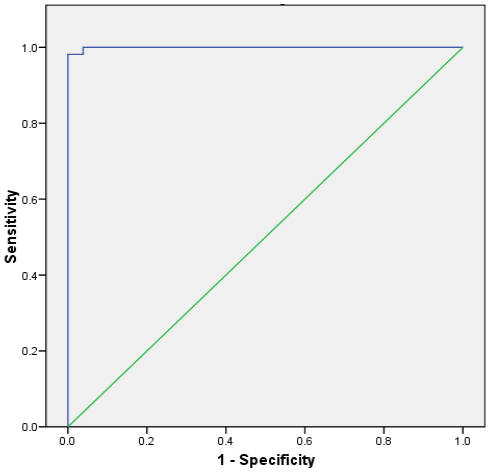



**Figure 7: Comparison of study variables in cases and controls.**

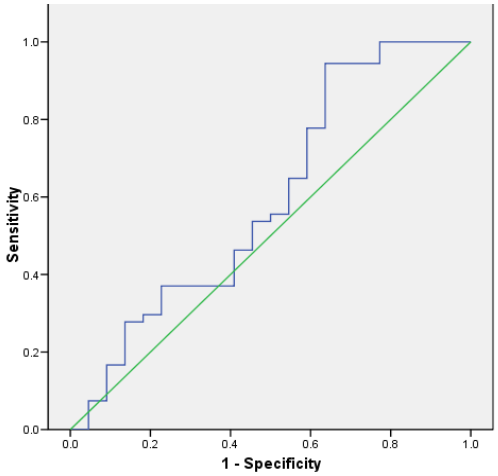
**Table 11: ROC curve analysis to predict Pseudocholinesterase (U/L) as a diagnostic marker for hypothyroid cases.**

| | Sensitivity | Specificity | 95%CI | AUC | P value |
|---|---|---|---|---|---|
| Pseudocholinesterase (U/L) (Hypothyroid Newly Detected Vs Control) | 100.00 | 96.2% | 0.987-1.00 | 0.999 | <0.001** |
| Pseudocholinesterase (U/L) (Hypothyroid On Treatment Vs Control) | 55.6 | 50.0 | 0.44-0.79 | 0.596 | 0.192 |

**ROC curve analysis for Newly detected Hypothyroid cases**




**ROC curve analysis for Hypothyroid cases on Treatment**




**Graph 1: ROC curve analysis to predict Pseudocholinesterase (U/L) as a diagnostic marker for hypothyroid cases.**

**Pseudocholinesterase is a diagnostic marker for hypothyroid patients who were newly detected with Area under ROC curve of 0.999, indicating a good marker with high sensitivity (100%) and high specificity (96.2%). (Table 11 and Graph 1).Pseudocholinesterase (U/L) is significantly less in cases not on treatment with F=78, P<0.001**, However the cases on treatment are more close to controls with P=0.189 indicating this as a prognostic factor (Table 12).**

**Table 12: Evaluation of Pseudocholinesterase (U/L) as diagnostic marker for hypothyroid cases.**

| Pseudocholinesterase (U/L) | Hypothyroid (Newly detected) | Hypothyroid (On Treatment) | Controls |
|---|---|---|---|
| Min-max | 558.00-5113.0 | 4135.0-10381.00 | 5104.00-9625.00 |
| Mean ± SD | 3170±1430.53 | 6320.32±1553.25 | 6753.21±1173.58 |
| 95%CI | 2592.39-3748.00 | 5631.64-7008.99 | 6432.88-7073.53 |
| Inference | Pseudocholinesterase (U/L) is significantly less in cases not on treatment with F=78, P<0.001**, However the cases on treatment are more close to controls with P=0.189 indicating this as a good prognostic factor | | |

**Table 13: Pearson correlation of Pseudocholinesterase (U/L) with thyroid parameters.**

| | Hypothyroid (Newly detected) | | Hypothyroid (On Treatment) | |
|---|---|---|---|---|
| | r value | p value | r value | p value |
| Pseudocholinesterase (U/L) vs. TSH | 0.058 | 0.778 | -0.374 | 0.086 |
| Pseudocholinesterase (U/L) vs. T4 | -0.133 | 0.517 | 0.328 | 0.214 |
| Pseudocholinesterase (U/L) vs. T3 | 0.199 | 0.363 | -0.069 | 0.813 |

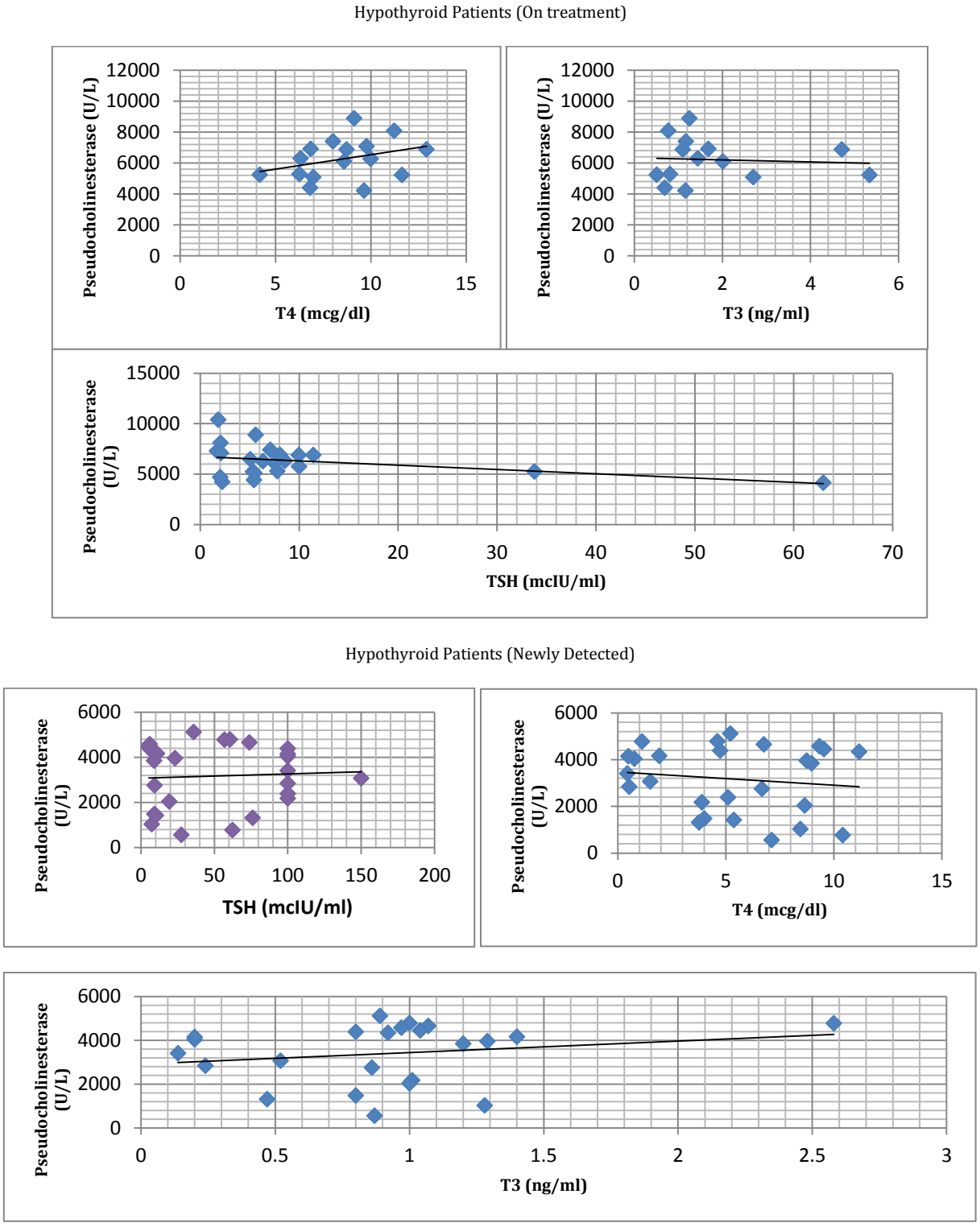



**Figure 8: Correlation of Pseudocholinesterase (U/L) with thyroid parameters.**

## CONCLUSION

Serum pseudocholinesterase activity is decreased by about 31.6 % in hypothyroidism. In this comparative case control study, the correlation between pseudocholinesterase and TSH activity in hypothyroidism was examined. There is convincing evidence from the present study showing a negative correlation between pseudocholinesterase and TSH in hypothyroid patients who are on treatment. Pseudocholinesterase was found to be a good biochemical marker for detecting new cases of hypothyroidism with a sensitivity of 100% and specificity of 96.2%.

## LIMITATION

However larger studies with generalised population are necessary to determine about the pseudocholinesterase activity in hypothyroidism, the correlation of pseudocholinesterse levels with thyroid function test and for the assessment of pseudocholinesterase as a diagnostic and prognostic marker in hypothyroidism. Given the

study location, our results may not be projected to the pediatric population.

**AUTHOR'S CONTRIBUTION**

Dr. Shiju K Sleeba and Dr.B.N Raghavendra Prasad conceived the idea and strategies for the study. Dr. Mukesh Bangera supervised the project. Dr.Shiju K Sleeba designed the study and wrote the paper. Data was validated by Dr.B.N Raghavendra Prasad. All authors discussed the results and implications and commented on the manuscript at all stages.

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