# “IODINE NUTRITIONAL STATUS IN CHILDREN WITH ACQUIRED HYPOTHYROIDISM”

Dissertation submitted for

**M.D., DEGREE EXAMINATION**

**BRANCH VII PAEDIATRIC MEDICINE**

**THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY**

**CHENNAI**


**INSTITUTE OF CHILD HEALTH AND HOSPITAL FOR CHILDREN**
**MADRAS MEDICAL COLLEGE**
**CHENNAI**

**APRIL 2015**

# CERTIFICATE

This is to certify that the dissertation titled **"IODINE NUTRITIONAL STATUS IN CHILDREN WITH ACQUIRED HYPOTHYROIDISM"** submitted by **DR.M.DIVYA** to the Faculty of Paediatrics, **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI** in partial fulfilment of the requirements for the award of **M.D., DEGREE (PAEDIATRICS)** is a bonafide research work carried out by him under our direct supervision and guidance.

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# DECLARATION

I **M.DIVYA** solemnly declare that the dissertation titled **"Iodine Nutritional Status in Children with Acquired Hypothyroidism"** has been prepared by me.

This is submitted to the TamilNadu **DR.M.G.R Medical University,** in partial fulfilment of the rules and regulations for the M.D Degree examination in paediatrics.

**DR.M.DIVYA**

Place : Chennai

Date :

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My sincere thanks to **PROF. DR. R.VIMALA M.D**., Dean, Madras Medical College, for allowing me to do this dissertation, utilizing the institutional facilities.

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# CONTENTS

# INSTITUTIONAL ETHICS COMMITTEE
# MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
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## CERTIFICATE OF APPROVAL

To
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Madras Medical College,
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Dear Dr. M. DIVYA,

The Institutional Ethics Committee has considered your request and approved your study titled **"Iodine Nutritional Status in Children with Acquired Hypothyroidism " No. 26082014.**

The following members of Ethics Committee were present in the meeting held on **05.08.2014** conducted at Madras Medical College, Chennai-3.

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| 9. Thiru S.Rameshkumar, Administrative Officer | : Lay Person |
| 10.Thiru S.Govindasamy, B.A., B.L., | : Lawyer |
| 11.Tmt.Arnold Saulina, M.A., MSW., | : Social Scientist |

We approve the proposal to be conducted in its presented form.
The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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INTRODUCTION

Thyroxine and triiodothyronine profoundly increase the metabolic rate of the body. Complete lack of thyroxine can cause the metabolic rate of the body to fall by 40 to 50 % below normal. Thyroxine and triiodothyronine increase the consumption of oxygen, stimulates the synthesis of proteins thereby promoting growth of the body, CNS development and affect the metabolism of both sugars and monosaccharides.

Anatomy:

The thyroid gland is an endocrine organ which is situated in front of the neck between the cricoid cartilage and the sternum. The gland has two lobes connected by a narrow median isthmus. No significant difference in thyroid gland volume has been observed between males and females from 8 months to 15 years. The blood supply to the gland comes from paired superior and inferior thyroid arteries, originates from external carotid artery and an inconstant artery, the thyroidea ima. The blood is drained via the venous plexus in the fibrous capsule. Thyroid has a rich supply of lymphatics. These draining superior portions of the gland and the isthmus collect in to internal jugular lymph nodes and those draining the inferior portions collect into pre and para tracheal lymph nodes.

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# ABBREVIATIONS

| | | |
|---|---|---|
| TSH | - | Thyroid stimulating hormone |
| T4 | - | Thyroxine |
| IDD | - | Iodine deficiency disorders |
| TPO | - | Thyroid peroxidase |
| TG Antibodies | - | Thyroglobulin antibodies |
| WHO | - | World health organization |

# ABSTRACT

**“Iodine nutritional status in children with acquired hypothyroidism”**

Hypothyroidism causes significant growth and mental retardation. The existing Universal Salt Iodization program has led to the reduction in Iodine Deficiency Disorders in endemic population groups. Inspite of this, prevalence of goiter is 13%, childhood hypothyroidism is 3% and Subclinical hypothyroidism is 10% in our country. Experience from southeast Asian countries also shows increase in detection of Juvenile autoimmune thyroiditis in post –iodization assessment. Urinary iodine is the prime indicator of a person’s iodine nutritional status. Hence this study is done to assess the iodine nutritional status in children with acquired hypothyroidism and healthy school children.

**Objectives**

**Primary Objective**

To find out the iodine nutritional status in children with acquired hypothyroidism of age 2-12 years attending tertiary care centre

**Secondary Objective**

To assess changes in iodine nutritional status in autoimmune thyroiditis

**Study Method**

Descriptive study

**Study Population**

61 cases of acquired hypothyroidism and 102 healthy children from 2 different schools in Chennai.

**Materials and Methods**

Children with acquired hypothyroidism of age 2-12 years attending a tertiary care center were evaluated with history, clinical examination, goiter grading, antithyroid antibody levels, urinary iodine levels, ultrasound and FNAC thyroid gland if goiter is present. Urinary iodine levels were assessed in 102 healthy schoolchildren. If the iodine levels were more than adequate, >200μg/l they were further evaluated with thyroid function tests, antiTPO antibody and Thyroglobulin antibody levels.

**Results**

The mean urinary iodine level in cases were 440.2 and 789.9 in healthy school children. Female : Male ratio was 2.7:1. Ultrasound is as sensitive as FNAC in diagnosing thyroiditis.

**Conclusion**

Autoimmune thyroiditis is the most common cause of acquired hypothyroidism. Iodine deficiency is no longer the cause of hypothyroidism. There is no correlation between urinary iodine levels and antithyroid antibody levels.

**Keywords:**

Urinary iodine levels, anti TPO antibodies, anti Thyroglobulin antibodies, autoimmune thyroiditis

# INTRODUCTION

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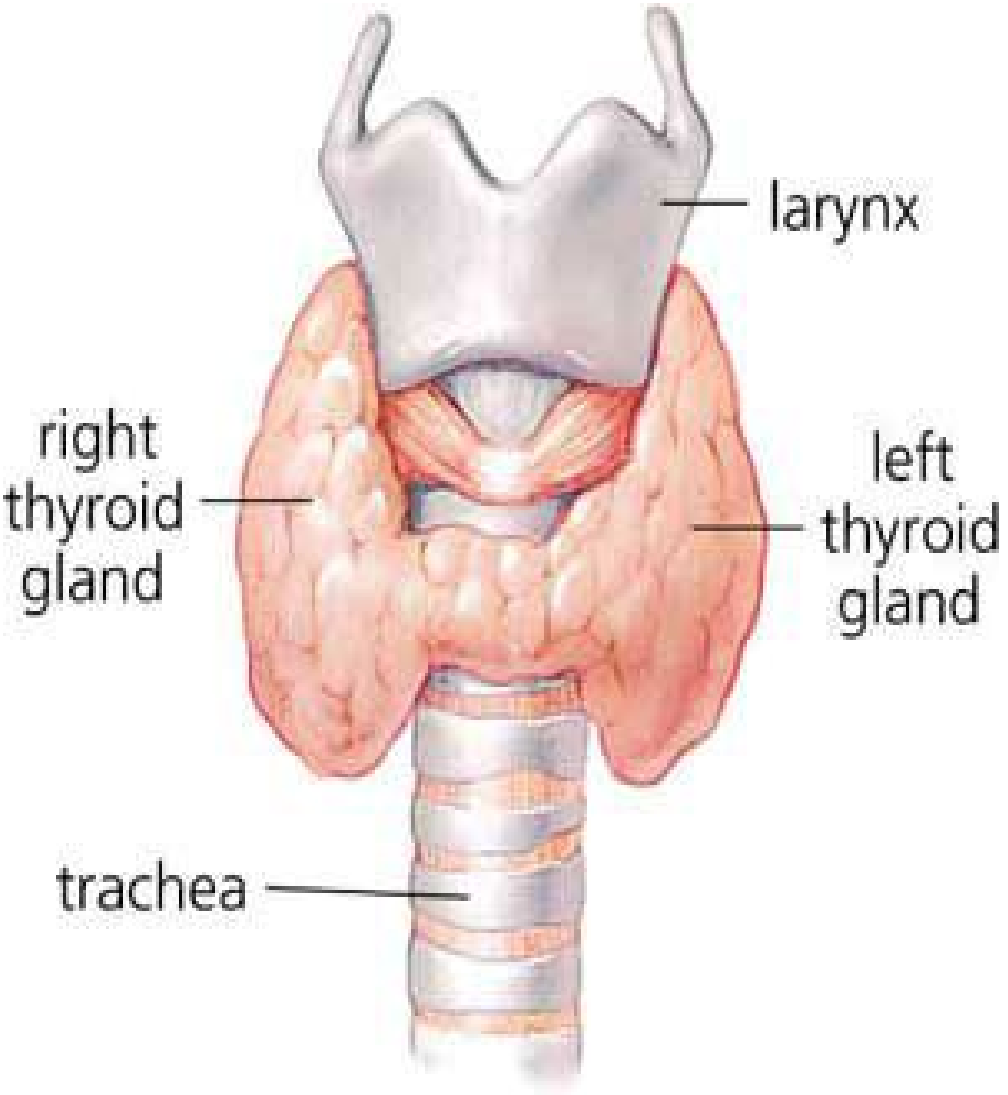
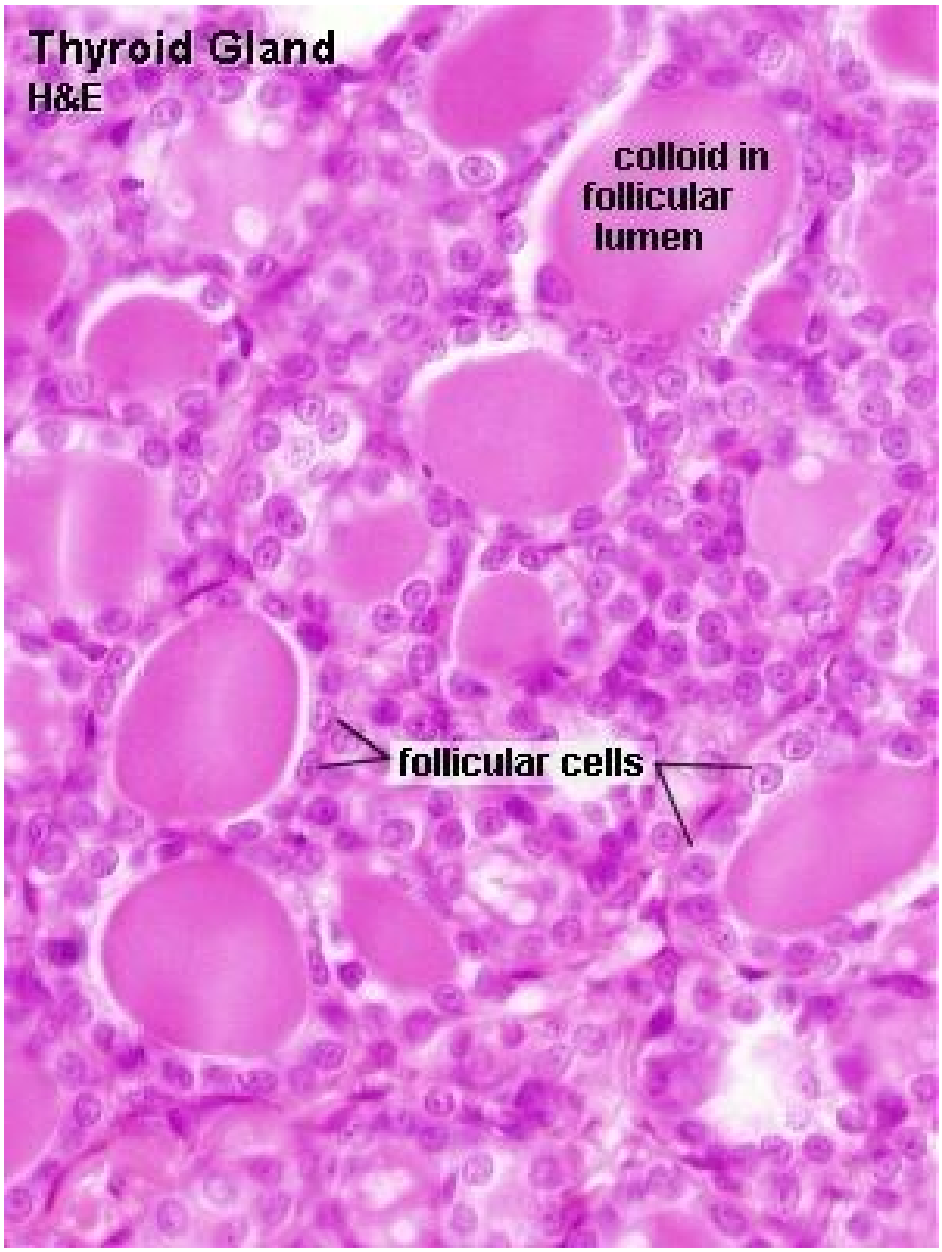
**Anatomy of Thyroid gland**



## Histology

The gland consists of two functionally and morphologically distinct endocrine systems. The principle component of the gland being follicles made up of the thyrocyte or follicular cell proper and the second minor endocrine component being parafollicular or C cells. In addition to the above two, there is a third anatomical structure of unknown physiologic significance constituting ultimobranchial remanants. The follicles are filled with colloid which contains thyroglobulin. Thyroid hormones are stored as thyroglobulin, a large protein with numerous residues of iodinated thyroid including biologically active T3 and T4. When the gland is activated by TSH to release thyroid hormones, the endocytosis of colloid and proteolysis of thyroglobulin by lysosomal enzymes occur.

**Histology of Thyroid gland**

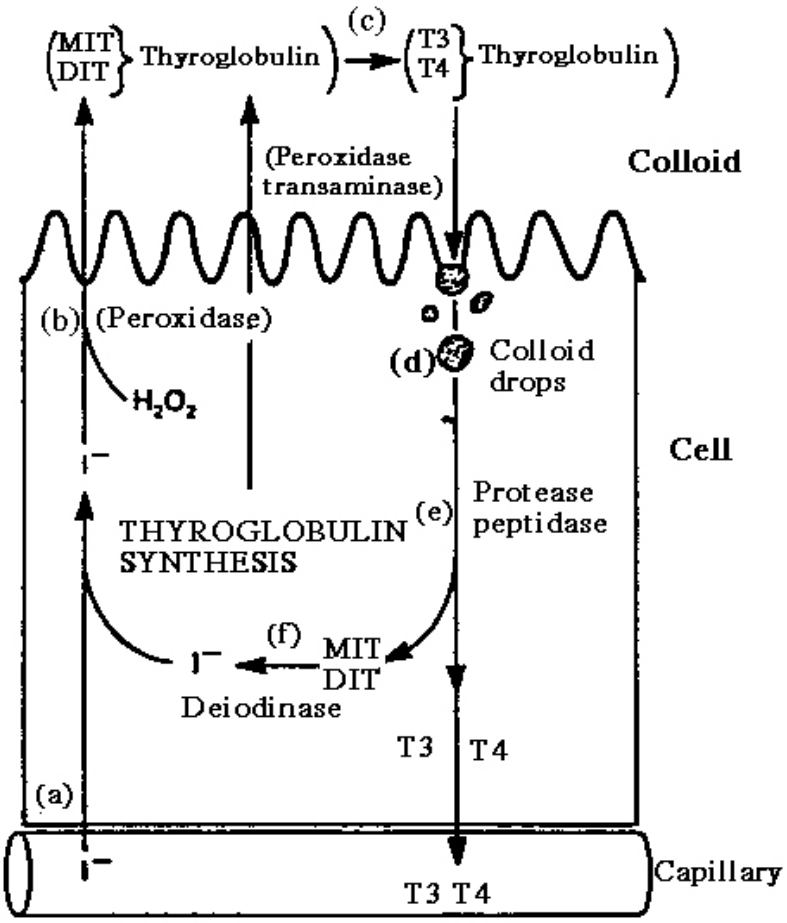


**Thyroxine synthesis**

The production of thyroxine and triiodothyronine are closely related to iodine metabolism as both of these hormones contain high content of iodine. Both of these hormones have similar action but differ in their course and time of action.(12)

The first step in the production of thyroxine is the trapping of iodide by sodium iodide symporter into the lumen of thyroid follicles. Iodide is converted to oxidised iodine with the help of peroxidise and hydrogen peroxide. The oxidised iodine binds with tyrosine aminoacids of thyroglobulin in the presence of iodinase, the process known as organification of thyroglobulin to form mono and diiodotyrosine. They couple to form thyroxine and triiodothyronine which are stored in the thyroglobulin molecules.

The thyroid gland is unique in the fact that it is the only endocrine gland which can store the hormones for months and release it into the circulation slowly when needed. The stores are sufficient for 3 months. Thyroxine when needed, the epithelial cells send pseudopodia into the lumen and they endocytose thyroglobulin. Each thyroglobulin contains 1 to 3 thyroxine molecules and 1 triiodothyronine molecule for each 14 thyroxine. Thyroxine and triiodothyronine are cleaved from the




thyroglobulin molecules with the help of proteases and peptidases present in the lysosomal enzymes and released into the circulation. In the circulation 98% of hormones bind with thyroid binding globulin, thyroid

binding prealbumin and albumin. This is to prevent rapid degradation by liver, kidney and peripheral tissues. In the thyroid gland T4 constitutes 35%, T3 constitutes 5%, and the rest is DIT and MIT.

**T4** is highly protein bound and is stable, it is the extracellular hormone.

**T3** is less protein bound. It is more potent than thyroxine . It enters the cells rapidly and acts. Half life of thyroxine is 6 days while that of triiodothyronine is 1-3 days.

T3 is 3-4 times more potent than thyroxine. Only 20% $T_3$ is produced by thyroid gland while the rest is formed from thyroxine by deiodinase enzyme in the liver, kidney and peripheral tissues.

**Feedback mechanism**

Thyrotropin releasing hormone is produced by hypothalamus.

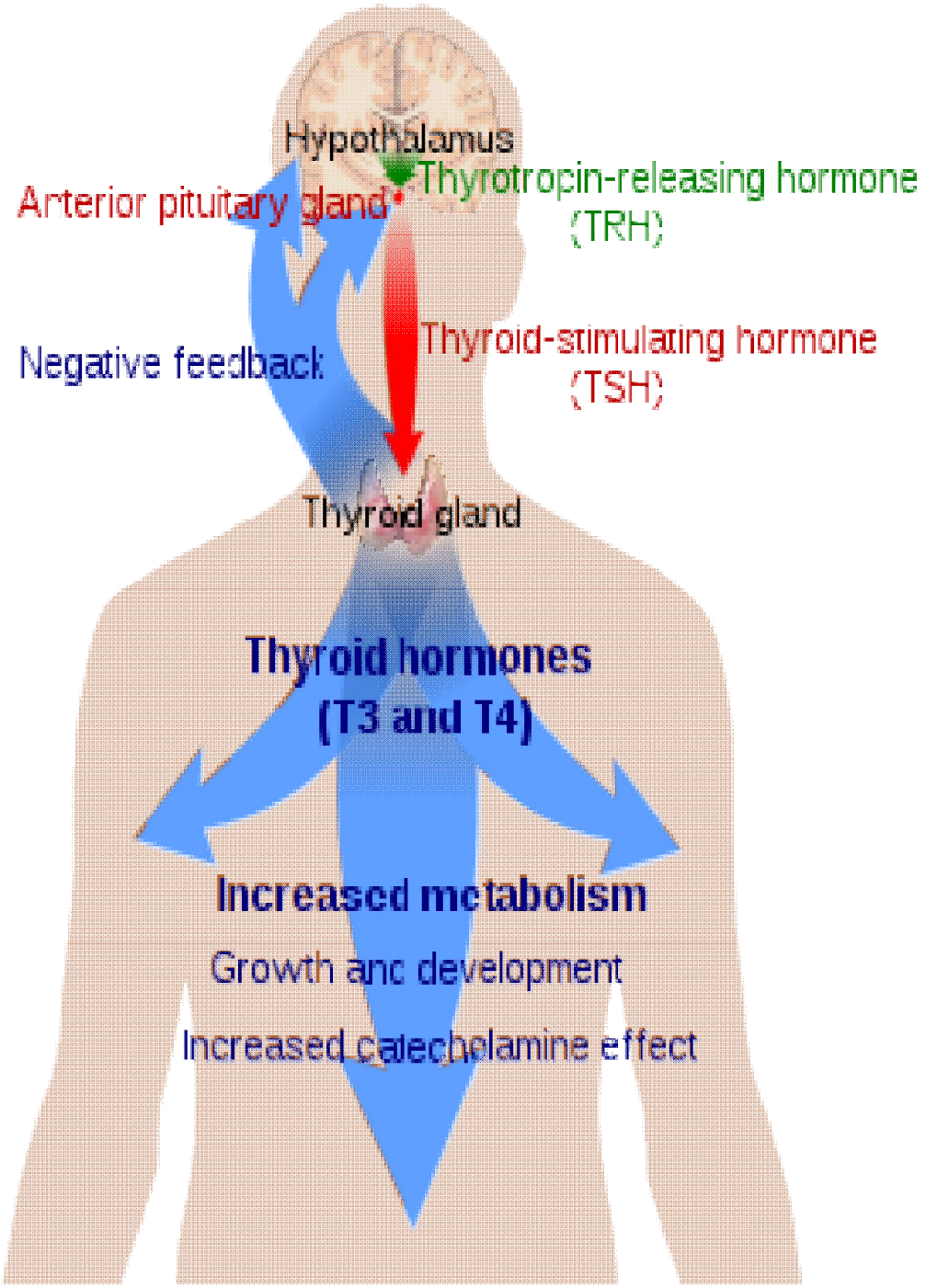
Thyroid stimulating hormone is produced by anterior pituitary.

Thyroxine and $T_3$ are produced by thyroid gland.

All the hormones are highly interlinked. When thyroxine is low, it stimulates the production of Thyrotropin releasing hormone which in turn stimulates the production of TSH which increases the production of thyroxine.

Initially this change occurs only at molecular level. If there is persistent hypothyroidism, excessive TSH will cause hyperplasia of thyroid gland hence goitre occurs. Also in hyperthyroidism there may be enlargement of thyroid gland.

# Thyroid system



**Iodine deficiency disorders:**

Defective iodine intake could lead on to certain bad effects like goiter, mental and growth retardation. This can be prevented by taking adequate iodine in food.. Erosion of soil in riverine area ,excessive tree cutting, overgrazing by livestock have led to loss of iodine from soil. Hence crops grown in that area lack in iodine. Hilly areas are also deficient in iodine. Iodine deficiency can lead to goiter, mental and growth retardation, still birth, defects in speech, hearing and vision. The children living in iodine deficient areas had 13.5 Intelligence quotient points lower than iodine replete areas.

130 countries and two million people were found to be affected with Iodine deficiency disorders, affecting One third of the population. 6.1 crore people in india are suffering from iodine deficiency goiter. And 88 lakh people have growth or mental retardation.

Therefore after a successful trial of iodization of salt in kangara valley, Government of India launched a 100% centrally sponsored programme named National goiter control programme(NGCP) in 1962.

**Objectives of NIDDCP**

1. Magnitude of the problem of IDD in the country must be surveyed;
2. Endemic region must be supplied with iodised salt;
3. Health Education;
4. To monitor the quality of iodized salt by assessing urinary iodine excretion pattern and assessing the magnitude of Iodine Deficiency disorder and
5. Every five years all the areas must be surveyed. The last survey in the endemic areas were not satisfactory.

The National Goiter Control Programme (NGCP) was converted to National Iodine Deficiency Disorder Control Programme (NIDDCP). NIDDCP in 1992, recommended universal salt iodization.

Universal salt iodization is achieved when iodine is present in the food for human and animal consumption.

| Population | Levels recommended |
|---|---|
| Preschool Children | 90µg |
| School Children | 120µg |
| Adults | 150µg |
| Pregnant and breast feeding women | 200µg |

According to WHO declaration, 2001 elimination of iodine deficiency disorders shall be maintained only when

1. The median urinary iodine excretion is 100 µg/l. <20% being below 50 µg/l.
2. > 90% households should be using salt which has iodine content of 15 parts per million or more.

Iodized salt was brought under the revised Prevention of Food Adulteration (PFA) Act of 1988.

WHO recommended that in special circumstances where

1. 20 %loss of iodine from salt from production site to households
2. During cooking loss is 20%
3. Person must take atleast10g per person per day

At the production site concentration of iodine in salt should be 20-40 ppm iodine per kg of salt so that a person avails 150µg of iodine per person per day. Salt must not be transported in gunny bags. Plastic bags must be used.

>90% households are using iodised salt in 30countries. Hence we have achieved the goal of universal salt iodisation. South east asian countries have achieved 70% iodised salt consumption. In India as per the Coverage Evaluation Survey 2009, 91 per cent of households had access to iodized salt, of whom 71 per cent consumed adequately iodized salt. Another 9 per cent consume non-iodised consumed salt.

Iodine is particularly essential for pregnant women as the iodine deficiency can cause abortion, still birth and affect the development of fetus. It is also important for the first two years of life as this is the period where the brain development occurs.

In areas where iodisation is not possible up to the mark, in order to provide essential iodine to children and pregnant women, the following things are done.

1. Administration of iodised oil capsules once in 6-18 months.
2. Lugol's iodine administration once in a month.
3. Water supply must be iodized by adding iodine solution with special instruments.

In 1991,total goiter rate was 13% of the world population affecting 740 million people. Salt coverage had increased to 70% in south east asia.

Between 1994 t0 2006, 94 Countries had conducted urinary iodine survey.

Thus we have data only on 91% of the world's population. Still we are lacking data on 8.9% of the population of the world.

| **Year** | **Number of countries endemic To IDD** |
|---|---|
| 2006 | 47 |
| 2004 | 54 |
| 1993 | 126 |

| Iodine Intake | |
|---|---|
| Adequate | 76% |
| Excessive | 7% |
| Insufficient | 31% |

## SALT IODIZATION PROCESS

Salt is iodized by additional fixed amount Potassium Iodate ($KIO_3$) or Potasium Iodide as an aqueous form or dry powder form, at the production site.

The amount of Iodine supplemented depends on the recommended amount for that country. Iodate is recommended than iodide as it is much more stable. The moisture content of the salt is maintained by improving packaging and storage of the salt. This is required for maintaining the stability of iodine in the salt.

By using small (500 to 1 kg) polyethylene bags loss of iodine was reduced by 30-80%. This is made possible by film insert and polyethylene bags. The loss is 10% over an 18 month period.

This was very much less when iodized salt was stored in large amounts in gunny bags.

## TESTING OF IODINE LEVEL IN SALT

The amount of iodine in salt can be determined in quantity by titration method and by rapid test kits qualitatively.

### Titration method

Titration method involves preparation of 4 solutions and a standard solutions which can be used for a variable time, and then assessing the Iodine content in the salt solution by adding solutions which are already prepared and then titrated.

### Principle

The iodine present in the salt is liberated and in the presence of starch as indicated, iodine is titrated against sodium thiosulphate. This method changes depending on iodate or iodide is used in iodizing the salt.

### Rapid test kits

These kits contain 10 to 15 ml bottle with stabilizing starch containing solution. Then 1 drop of this solution is added to the solution of iodized salt.

Colour change to blue or purple indicates that iodine is present. When the salt is more alkaline, recheck solution is added.

This is only qualitative but not quantitative and must be rechecked by titration method. The advantage of this kit is that they are easy to carry and these tests can be done at the production site.

It can be also be done at the house hold level and we can check the availability of iodine at the household level.

These kits have been provided to environmental health officers, nutrition officer, school teacher, mayors, community midwives and other government workers who are responsible for health. This is helpful in checking the content of iodine at both the production site and house hold level and has helped us in managing the IDD problem.

**INDICATORS OF IMPACT**

1. Urine Iodine
2. Goiter by palpation and by ultra sound
3. Blood levels of TSH and Thyroglobulin

## URINARY IODINE

Only one fifth of the iodine which is ingested by the person is utilized by the thyroid gland. Others will be excreted in urine. Hence the iodine nutritional status of the person can be assessed by measuring the urinary iodine level.

There will be day to day variation in the urinary iodine levels and also there will be variation in the same day in the same individual but this problem can be evened out by measuring the median urinary iodine level of the population.

Urine iodine creatinine ratio was previously used. This is not reliable as the creatinine content of urine of a person varies depending on the protein intake on the previous day. This is also expensive and not needed.

Hence nowadays urinary iodine is calculated in the casual urine samples. These samples doesn’t need refrigeration or addition of any preservatives. These samples can be used even after months by refrigerating the sample. This is done just to prevent the development of bad odour. These samples can be defrozen and refrozen any number of times. But just before taking the aliquots for assessing the urinary iodine level the sample must be completely defrozen.

**PRINCIPLE**

Iodide acts as catalyst where yellow coloured ceric ammonium sulphate is reduced to colourless cerous form in the presence of arsenious acid. This is known as sandell-kolthoff reaction.

Before that the sample is heated using ammonium persulphate or chloric acid to get rid of the interfering substances.

**1. Method A: With ammonium Sulphate**

250 microlitres of urine is digested with ammonium persulphate at $100^0$ C and arsenious acid and ceric ammonium sulphate are added one by one.

After 15 minutes the decrease in yellow colour is measured with the help of spectrophotometer. The values are plotted on a standard curve and the results calculated.

It requires heater and spectrophotometer and basic bio-chemical instruments and is not expensive. It needs expertise and 100 to 150 samples can be run in a single day.

**2. Method B : With Chloric Acid**

This method is the same as method A except that Chloric Acid is used in the place of ammonium persulphate for getting rid of the interfering substances. Chloric Acid is dangerous because the chemical mixture is explosive if it dries in the ventilatory system. Hence they recommend the use of Chloric Acid traps while doing the test.

**3. Modification of Method B**

This method uses ferroin, the redox indicator and stop watch instead of spectro photo meter for measuring the color change. The urine is heated with chloric acid and then the colour changes are read with stop watch in batches.

This method can also be followed with ammonium persulphate digestion method.

**4. Semi Quantitative Method**

Iodide acts as a catalyst in the oxidation of 3,3',5,5'-Tetramethyl benzidine by peracetic acid / hydrogen peroxide to yield yellow coloured products.

Then the results are read comparing with coloured strips and given in 3 ranges, <100 micrograms / litre, 100 to 300 micrograms / litre and >300 micrograms / litre.

This method requires the use of prepacked columns of charcoal which is given by the manufacturer to remove impurities.

**5. Microplate Method**

By this method 400 urine samples can be read in a day. First the samples are heated with ammonium persulphate on microplates kept with in special closed cassettes and boiled to $110^0$C.

Then the contents are changed to another microplate and ceric ammonium sulphate reduction step is done. Manufacturer supply is the crux in conducting these studies.

These test are reliable, fast, inexpensive reproducible and safe.

Method B is hazardous hence method A is used worldwide. The other methods are yet to be checked for their potency.

Most of the methods for assessing iodine in urine are reliable. The range can be extended upward by running the tests with appropriate dilutions. Co efficient of variation is under 10% for all the methods.

30 urine determinants from a group are sufficient for assessing the median urine excretion level in that area.

## INTERPRETATION

For measuring urinary iodine value of a population, median should be used as a measure of central tendency better than the mean.

Percentiles rather than standard deviation must be used as a measure of spread.

Frequency distribution curve will be appropriate if the salt iodine content is also available for the same population.

1. Excessive iodine intake is not a problem. It causes iodine induced hyper thyroidism only in individuals with nodular goiter which could have occurred even if normal iodine was being utilized.
2. When the urine iodine level is more than 300 micrograms per litre in iodine deficient areas, it can cause autoimmune thyroiditis and iodine induced hyperthyroidism in susceptible individuals.

### Thyroid size by clinical method

This is the age old method of surveying iodine deficiency disorders but the increase in thyroid size takes a long time to occur.

The thyroid gland is difficult to palpate in new borns. This method can be used in children of 6 to 12 years of age.

**Technique**

Subject is made to stand in front of the examiner and the examiner looks for any visible thyroid gland enlargement. Then the patient is asked to look up and extend the neck fully. This pushes the thyroid gland forward and it becomes easily palpable. Palpation of the thyroid gland is done by gently sliding the thumb along the tracheal side between the fifth cervical vertebra and first thoracic vertebra.

Another method can be used where the doctor stands behind the person and palpates the gland with the fingers.

**Ultrasound of the thyroid**

It is more reliable, non invasive and safe procedure. The results can be compared with the standard for that age, sex and body surface. It also varies for different population and countries.

In autoimmune thyroiditis, there will be alteration in echogenecity.

## FNAC thyroid

### 1. Obtaining cell samples

To get sufficient amount of samples for studying is not so easy. Studying the cytology is challenging and needs expertise. Dermal anaesthesia may be useful. First the thyroid mass is located with fingers. Then a 22- to 25 gauge needle of size 4.5 cm is poked into three different sites. Syringe may or may not be used. If a cyst is palpated first it must be drained and then FNA of the cyst done. When the nodule is difficult to palpate ultrasound can be done with the help of ultrasound.

The thyroid is a highly vascular organ and hence the aspirate may contain only blood. This can be stopped if the procedure is done within 5 seconds.

Contraindications of Thyroid FNA

Bleeding diathesis is an absolute contraindication to FNAC as an hematoma collection at the aspiration site can cause tracheal compression and cause respiratory distress. Hence bleeding time, PT and APTT is mandatory before FNA thyroid. Tracheal injury and local infection are rare complications. By pressing the puncture site for 5 minutes, subcutaneous hematoma can be avoided. Hemoptysis may occur due to tracheal injury.

**2. Slide preparation:**

The smears must be carefully collected and processed to yield good smears. Different methods are used to prepare smear depending on the nature of the thyroid gland. (a) One drop is kept near one end of slide and smeared by a cover slip. (b) A small drop of thyroid aspirate is put on a glass slide, crushed with a second slide that is then separated vertically from the first one. A thick smear may obscure the details of the cytology of the gland.

Routine staining methods

Either air-dried and Romanowsky-stained smears or ethanol-fixed and Papanicolaou-stained smears can be prepared as per the need. For Papanicolaou staining, the smears must be fixed quickly before drying with a commercial spray fixative or 95% ethanol. If fixation is delayed, it will result in air-dried artefactual changes and loss of cellular details. After Air-drying smears, one of the Romanowsky modified methods namely May-Grunwald-Giemsa, Wright stain, or Diff-Quik method can be used, as air-drying artifactual changes can be avoided. However, nuclear details will be well-visualized only in wet-fixed and Papanicolaou-stained smears. But the use of air-dried and wet-fixed smears at the same time is usually recommended, as these two staining methods are complementary**.**

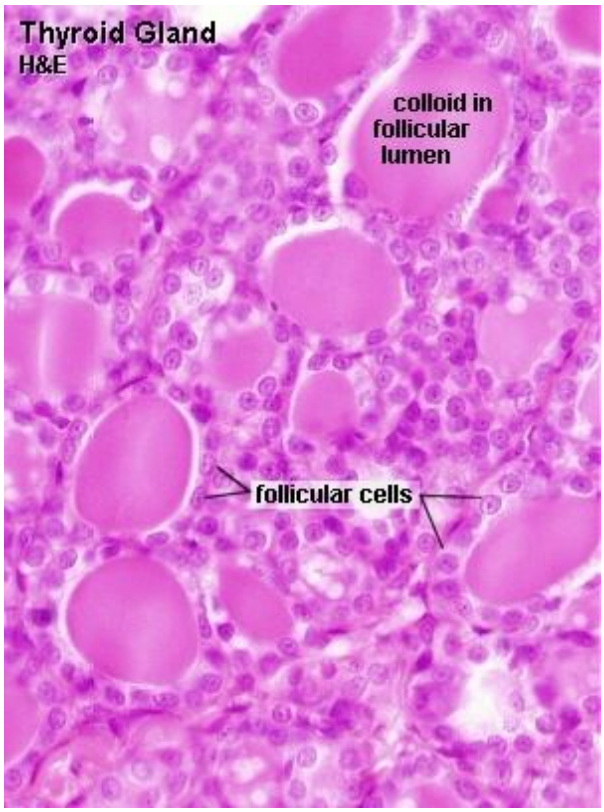
Cytodiagnosis and Its Limitations

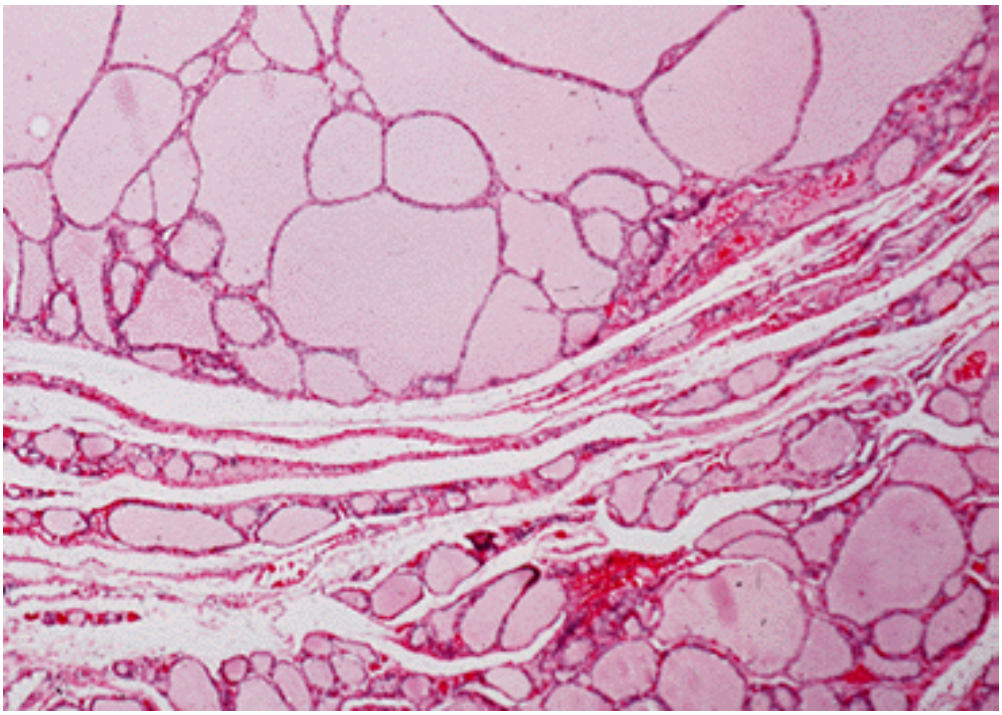
The cytodiagnosis of thyroid nodules by FNA is complex for the following reasons

1. Cytological patterns can overlap between neoplastic and non-neoplastic lesions.
2. The features of different neoplasms may overlap.
3. The same gland may contain different malignancies and also nonneoplatic lesions

.

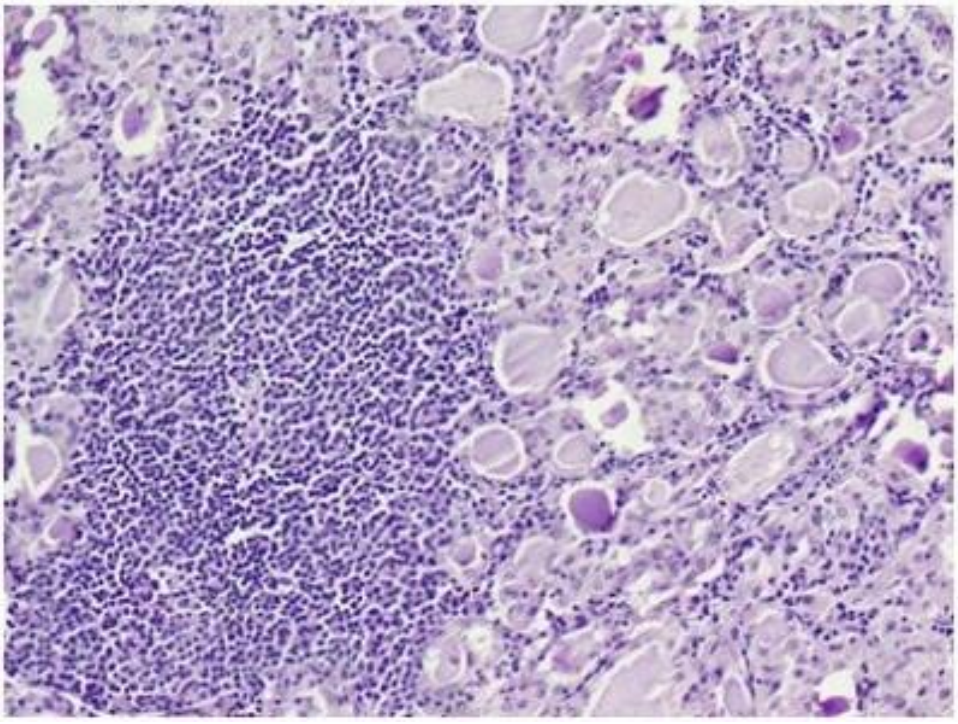
**Normal histology**





In case of colloid goiter, there will be large uniform follicles filled with colloid.

**Autoimmune thyroiditis**



**Grading of thyroid gland**

| | | |
|---|---|---|
| Grade 0 | - | No lymphoid cells |
| Grade 1 (Mild) | - | (Mild) few lymphoid cells infiltrate the follicles |
| Grade 2 (Moderate) | - | Moderate lymphocytic infiltration with hurthle cell change. |
| Grade 3 (Severe) | - | Severe florid lymphocytic infiltration with germinal center formation. |

## Hypothyroidism

May be either congenital or acquired.

## Congenital hypothyroidism

It affects 1 in 3000 infants, worldwide.

Girls are more affected than boys.

This occurs due to the absence of gland or defect in enzymes.

## CAUSES

### 1. Primary hypothyroidism

Defect of fetal thyroid development (dysgenesis)

- Aplasia
- Ectopia
- Hypoplasia

### Defect in thyroid hormone synthesis (dyshormonogenesis)

- Iodide transport defect: there will be mutation in sodium-iodide symporter gene
- Thyroid organification, or coupling defect: presence of mutation in thyroid peroxidase gene
- Defects in $H_2O_2$ generation: mutations present in DUOXA2 maturation factor or *DUOX2* gene
- Thyroglobulin synthesis defect: mutation of thyroglobulin gene
- Deiodination defect: presence of mutation in *DEHAL1* gene

**TSH unresponsiveness**

- $G_s\alpha$ mutation (e.g., type IA pseudohypoparathyroidism)
- Mutation of TSH receptor

Defect in transport of thyroid hormone: mutation present in monocarboxylate transporter 8 *(MCT8)* gene

Iodine deficiency (endemic goiter)

Maternal antibodies: like thyrotropin receptor–blocking antibody (TRBAb, also termed *thyrotropin-binding inhibitor immunoglobulin*)

Maternal Medications

- Iodides, amiodarone
- Propylthiouracil, methimazole
- Radioiodine

**2.Secondary hypothyroidism(pituitary)**

Pit 1 mutations

- TSH deficiency
- Prolactin deficiency
- Growth hormone deficiency

**PROP-1 Mutations**

- TSH deficiency
- Growth hormone deficiency
- Prolactin deficiency
- LH deficiency
- FSH deficiency
- ACTH deficiency

TSH deficiency: mutation present in TSH β subunit gene (Manifests as primary hypothyroidism with elevated TSH level).

Multiple pituitary deficiencies (e.g., craniopharyngioma)

**TRH deficiency**

- Isolated or
- Multiple hypothalamic deficiencies (e.g., septo-optic dysplasia)

TRH unresponsiveness

Mutations present in TRH receptor

**1.** Thyroid dysgenesis - 80-85%

Agenesis-33%

Ectopic gland-66%

**2.** Dyshormonogenesis - 5%

It is an autosomal recessive disorder. Always goiter present.

**3.** Transplacental thyrotropo receptor blocking antibodies-2% TSH cannot act on thyroid gland-hence no hormonal synthesis.

Deiodinase defect-

- Mono and diiodotyrosine cannot be deiodinated.
- Hence there will be loss of iodine in urine leading on to hypothyroidism.

## TRANSIENT HYROTHYROIDISM IN NEWBORN

1. If the mother has autoimmune disorder there will be thyroid receptor blocking antibodies. Ultrasound will show presence of thyroid gland. But is not present in iodide scan.
2. Radioiodine administration in the first year of life.
3. Excessive iodine exposure during delivery (iodine antiseptic, iodine containing weeds).
4. Preterm babies.

## CLINICAL FEATURES

- Increased head size due to myxedema of brain
- Wide open anterior and posterior fontenelle
- Edema of eyelids, hypertelorism, depressed nasal bridge
- Large protruded tongue leading to breathing difficulty
- Delayed dentition
- Brittle hair
- Low hairline
- Protruded abdomen, umbilical hernia
- Delayed bone maturation
- Sluggishness, Sleepiness
- Prolonged cholestasis (decreased glucuronide conjugation)
- Cardiovascular-heart murmurs, pericardial effusion

- General pallor(macrocytic anemia)
- Delayed development
- Hypotonia
- Pseudohypertrophy

**Acquired hypothyroidism**

Incidence of acquired hypothyroidism is 0.3% and subclinical hypothyroidism is 2%. The most important cause is chronic lymphocytic thyroiditis with female preponderance in the ratio of 2:1.

In mild iodine deficiency, the normal thyroid gland tries to adapt and maintains thyroid hormone production within the normal range. However, the prolonged thyroid hyperactivity due to such change leads to thyroid growth, and during proliferation of follicular cell there is a tendency to mutations leading to multifocal autonomous growth and function.

## ETIOLOGIC CLASSIFICATION OF ACQUIRED HYPOTHYROIDISM

Autoimmune (acquired hypothyroidism)

- Hashimoto thyroiditis -
- Polyglandular autoimmune syndrome, types I and II

-down,turner,type 1 DM

Iatrogenic

- Propylthiouracil, methimazole,
  iodides, amiodarone
  lithium,
  thalidomide,valprate,aminogluthemide,stavudine,interferon α

- Irradiation
- Radioiodine
- Thyroidectomy

Systemic disease

- Cystinosis
- Langerhans cell histiocytosis

Hemangiomas (large) of the liver (type 3 iodothyronine deiodinase)

Hepatitis C infection,William syndrome

Hypothalamic-pituitary disease

**Medications**

Amiodarone- inhibits 5'deiodinase enzyme thereby reducing the peripheral conversion of thyroxine to triiodothyronine.

Methimazole and propylthiouracil are antithyroid drugs hence cause hypothyroidism. Interferon α ,stavudine have autoimmunogenic potential-CLL like features. Hence causes thyroxine deficiency.

Expectorants contain iodine hence by feedback mechanism they inhibit thyroxine production.

Thiocyanate causes competitive inhibition of iodide transport into the cell and also prevents thyroglobulin from iodination.

Propylthiouracil-blocks peroxide enzyme and prevents organification of tyrosine to form thyroxine.

Nephropathic cystinosis-impaired thyroid function

Langhan cell histiocytosis-infiltration of the thyroid gland by langhan cells causes thyroid gland destruction and hypothyroidism.

In William syndrome and hepatitis C-there are no antibodies but cellular destruction occurs.

Hemangiomas of liver have increased deiodinase activity and therefore increased thyroxine destruction- and hypothyroidism.

## CLINICAL FEATURES

- Goiter-enlargement of the thyroid gland

  May be diffuse or nodular

  May cause pressure symptoms
- Decrease in thyroxine is compensated by increase in thyroid gland size
- Weight gain, fluid retention and myxedema
- Thin dry skin, puffy face, edematous eyelids
- Cold intolerance, hypothermia
- Bowel hypomotility-constipation, ileus
- Increased sleepiness, decreased energy
- Delirium, dementia, stupor coma
- Muscle cramps, nerve entrapment
- Delayed osseous maturation
- Heart failure, ventilatory failure
- Coagulopathy
- Adolescents-delayed puberty,menomettrorrhagia
- Younger children
- Galactorrhea-increased TRH stimulates prolactin
- Precocious thelarche, macroorchidism -TSH binds to FSH receptor
- Headache, Vision defects
- Hyponatremia-SIADH and impaired free water clearance

- Ventilatory failure-blunted hypercapnia and hypoxia
- Heart failure-impaired ventilatory drive
- Hypothermia, decreased response to sepsis
- Decreased calorigenesis
- Associated adrenal insufficiency

## LABARATORY FINDINGS

| | **TSH levels** | **Thyroxine levels** |
|---|---|---|
| Hyperthyroidism – overt | Decreased | Increased |
| Hyperthyroidism – subclinical | Decreased | Normal |
| Hypothyroidism –overt | Increased | Decreased |
| Hypothyroidism – subclinical | Increased | Normal |

### Lymphocytic Thyroiditis (Hashimoto Thyroiditis)

Hashimoto thyroiditis is the most common cause of thyroid disease in children and adolescents, and accounts for many of the “simple” goiter. It is also the most common cause of acquired hypothyroidism, with or without goiter.

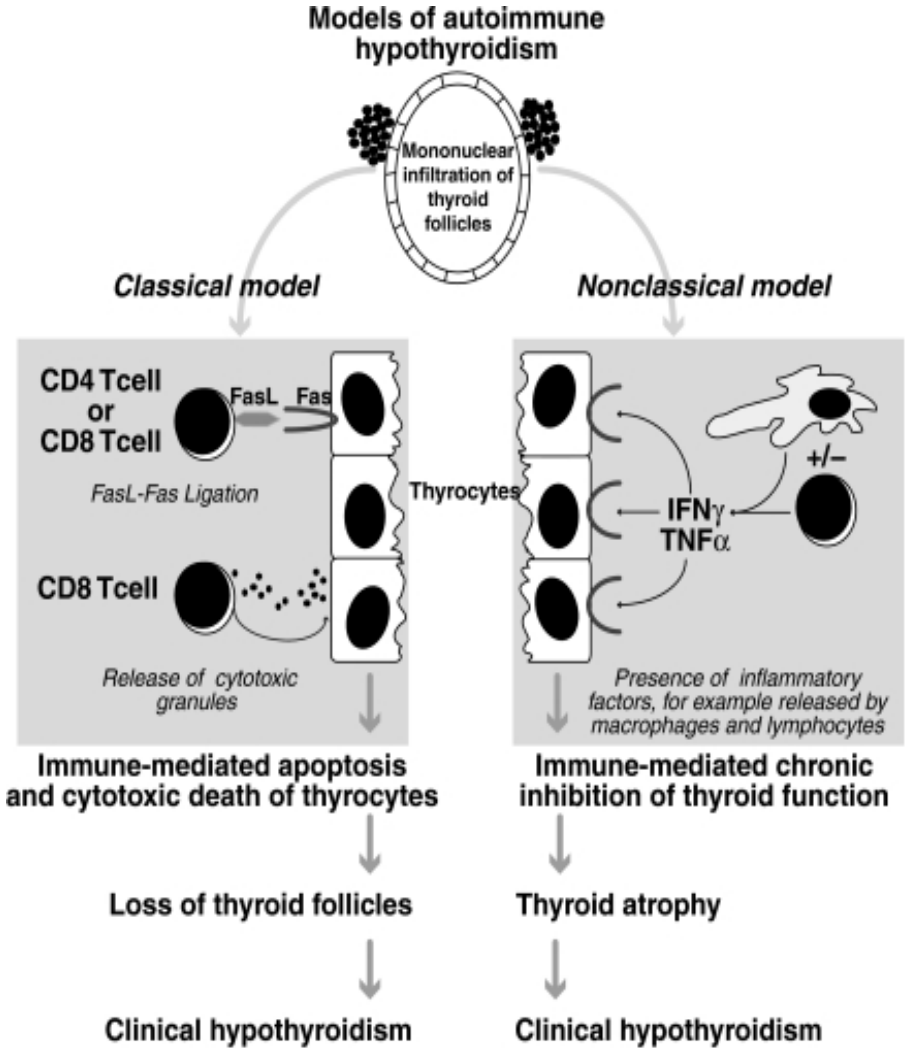
1 to 2% of younger school-going children and 4-6% of adolescents have positive anti thyroid antibodies as evidence of autoimmune thyroid disease.

**Etiology**

This is an typical organ-specific autoimmune disease, characterized histologically by lymphocytic infiltration of the thyroid gland. Earlier, there may be hyperplasia only followed by infiltration of lymphocytes and plasma cells between the follicles, resulting in atrophy of the follicles. Lymphoid follicle formation with germinal centers is the hallmark of autoimmune thyroiditis; the degree of atrophy and fibrosis of the follicles may vary from mild to moderate.

60% of infiltrating lymphoid cells are T cells and 30% are B-cells. The T-cell population is represented by helper ($CD4^+$) and cytotoxic ($CD8^+$) cells.

## Pathogenesis



The CD8+ cytotoxic cells cause thyroid cell destruction either by perforin mediated cell necrosis or by granzyme-B mediated apoptosis. T cell production of cytokines locally, such as tumor necrosis factor (TNF), IL-1, and interferon (IFN-α), may cause thyroid cells to be more susceptible to apoptosis mediated by death receptors, such as Fas, which are activated by their respective ligands on T cells. The cytokines impair thyroid cell function directly, and also induce the expression of many proinflammatory molecules by the thyroid cells themselves, such as cytokines, HLA class I and class II molecules, CD 40,adhesion molecules, and nitric oxide .

With the HLA haplotypes namely HLA-DR4and HLA-DR5 –there is increased risk of goiter and thyroiditis, HLA-DR3 is associated with the atrophic variant of thyroiditis.

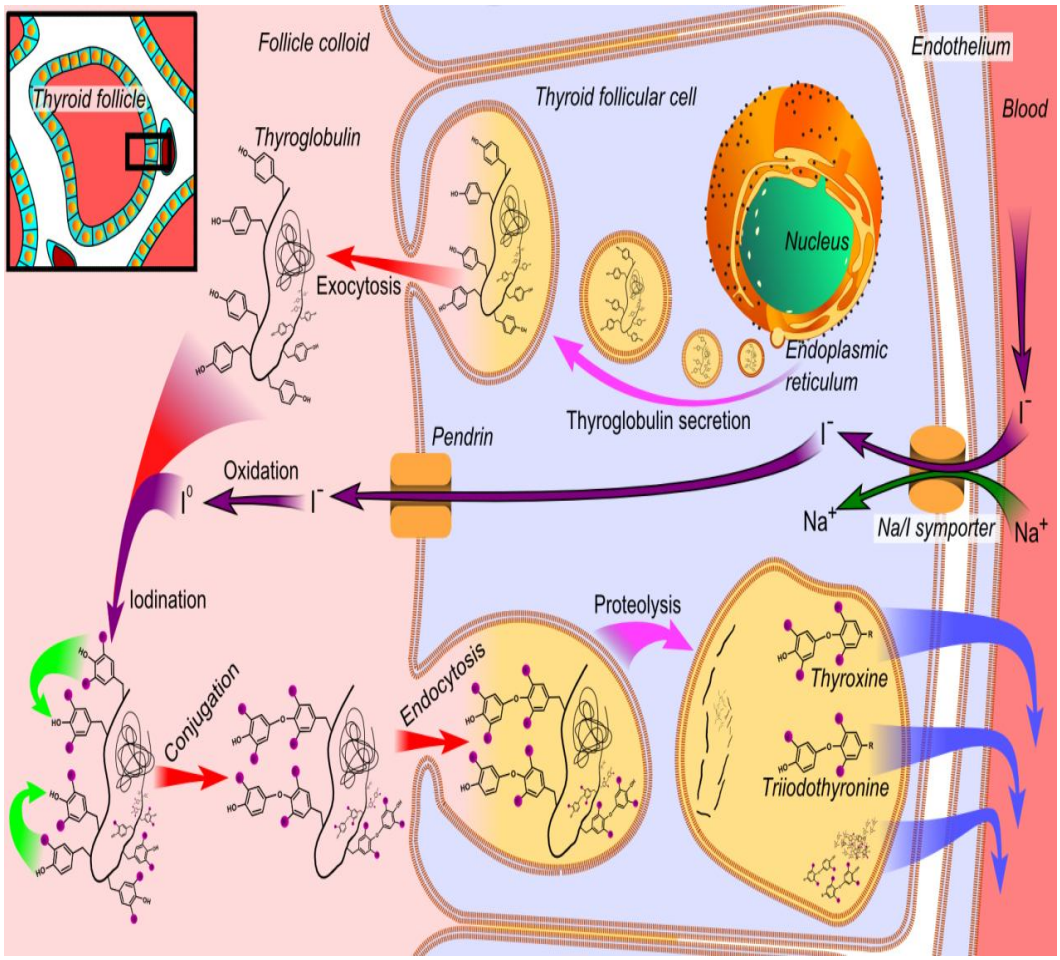
A variety of different thyroid antigen auto antibodies are also involved. Thyroid anti peroxidase antibodies (TPOAbs; also known as antimicrosomal antibodies) and antithyroglobulin antibodies are present in the sera of 90% of children with lymphocytic thyroiditis and also in many patients with Graves disease.

1. Thyroid peroxidise antibodies inhibit enzyme activity and can also stimulate natural killer cell cytotoxicity.

   They also fix complement resulting in destruction of thyroid gland.
2. Anti thyroglobulin antibodies does not have role in the autoimmune destruction of the gland.
3. Thyrotropin receptor–blocking antibodies are also present, mostly in patients with hypothyroidism, and it causes hypothyroidism and thyroid atrophy in patients with autoimmune thyroiditis.
4. Antibodies against pendrin, a protein present in apex on thyroid follicular cells is present in 80% of children with autoimmune thyroiditis.

   The disorder is double the time more common in girls than in boys. It can occur during the first 3 yr of life but becomes more common after 6 year of age it reaches a peak incidence during adolescence. The most common clinical manifestation are goiter and growth retardation. In most of the patients, the thyroid is enlarged diffusely, firm, and not tender. In about 30% of patients, the gland is lobular and nodular. Almost all the affected children are clinically euthyroid and without symptoms; some can also have pressure symptoms in the neck, including difficulty in swallowing and breathing difficulty. Some of the patients also have clinical

signs of hypothyroidism, but clinically euthyroid patients may have subclinical hypothyroidism.




A few children may also have manifestations suggesting hyperthyroidism but there will be evidence in TSH or thyroxine. It may also coexist with graves disease.

The clinical course maybe variable. Some children may persist to have goiter with euthyroid status. Most children who are euthyroid at presentation remain euthyroid, although a percentage of patients acquire hypothyroidism gradually within months or years. In children who

initially have mild or subclinical hypothyroidism i.e.elevated serum TSH, and normal free $T_4$ levels, over several years 50% may revert to euthyroidism, and about 50% continue to have subclinical hypothyroidism, and a few develop overt hypothyroidism. Thyroiditis is the cause of most cases of nongoitrous (atrophic) hypothyroidism.

Familial clustering of lymphocytic thyroiditis are common; the incidence in siblings or parents of affected children is very high as 25%. Autoantibodies to thyroid peroxidase and thyroglobulin in these families appear to be transmitted in an autosomal dominant fashion, with reduced penetrance in males.

Autoimmune thyroiditis can occur in 10% of patients with type I autoimmune polyglandular syndrome, characterized by autoimmune polyendocrinopathy, candidiasis, and ectodermal dysplasia (APCED). APS-1 consists of two of the triad of hypoparathyroidism, Addison disease, and mucocutaneous candidiasis i.eHAM syndrome. This is also inherited as autosomal recessive and is caused by mutations in the autoimmune regulatory *(AIRE)* gene present on chromosome 21q22.3.

Autoimmune thyroiditis can occur in 70% of patients with APS-2 i.e.Schmidt syndrome. APS-2 consists of the association of Addison disease with type 1 diabetes mellitus (T1DM) or autoimmune thyroid disease. The etiology may be unknown. Autoimmune thyroid disease also is associated with vitiligo, pernicious anemia, or alopecia. TPO

Antibodies are found in approximately 20% of white people and 4% of black children with T1DM.

Autoimmune thyroid disease also has an increased incidence in children with congenital rubella.

Lymphocytic thyroiditis is associated with some chromosomal disorders, particularly Turner syndrome and Down syndrome. In Down syndrome children, a study reported that 28% - antithyroid antibodies (mostly anti-TPOs), 7% - subclinical hypothyroidism, 7% - overt hypothyroidism, and 5% - hyperthyroidism. In a study of girls with Turner syndrome, 41% had antithyroid antibodies (again, mostly anti-TPOs), 18%of children had goiter, and 8%of children had subclinical or overt hypothyroidism.

Boys with klinefelter syndrome may also develop autoimmune thyroiditis.

**Hyperthyroidism**

Hyperthyroidism leads to an hypermetabolic state known as thyrotoxicosis characterised by elevated levels of T3 and T4. However this excessive release may be from preformed thyroxine as in thyroiditis or from extrathyroid tissue.

**Symptoms**

**Constitutional symptoms:** The skin is soft, warm, and flushed; heat intolerance and excessive sweating can occur. Increased sympathetic activity and hypermetabolism can result in weight loss despite increased appetite.

**Gastrointestinal:** Stimulation of the gut results in malabsorption, hypermotility, and diarrhea.

**Cardiac:** Palpitations and tachycardia occurs commonly; elderly patients may develop congestive heart failure due to aggravation of preexisting heart disease.

**Neuromuscular:** Patients may frequently experience nervousness, tremor, and irritability. Nearly 50% may be develop proximal muscle weakness known as thyroid myopathy.

**Ocular manifestations:** A wide, staring gaze and lid lag may be present because of the sympathetic overstimulation of the levator palpebrae superioris.

Thyroid storm is used to define the abrupt onset of severe hyperthyroidism. This condition occurs mostly in individuals with underlying Graves disease, probably resulting from an acute rise in catecholamine levels, as might be encountered as during stress.

**Diagnosis**

The diagnosis of hyperthyroidism is based on clinical features and laboratory data.

The measurement of serum TSH concentration using most sensitive assays provides the most useful single screening test for hyperthyroidism, because TSH levels will be decreased even at the earliest stages, when the disease has not manifested.

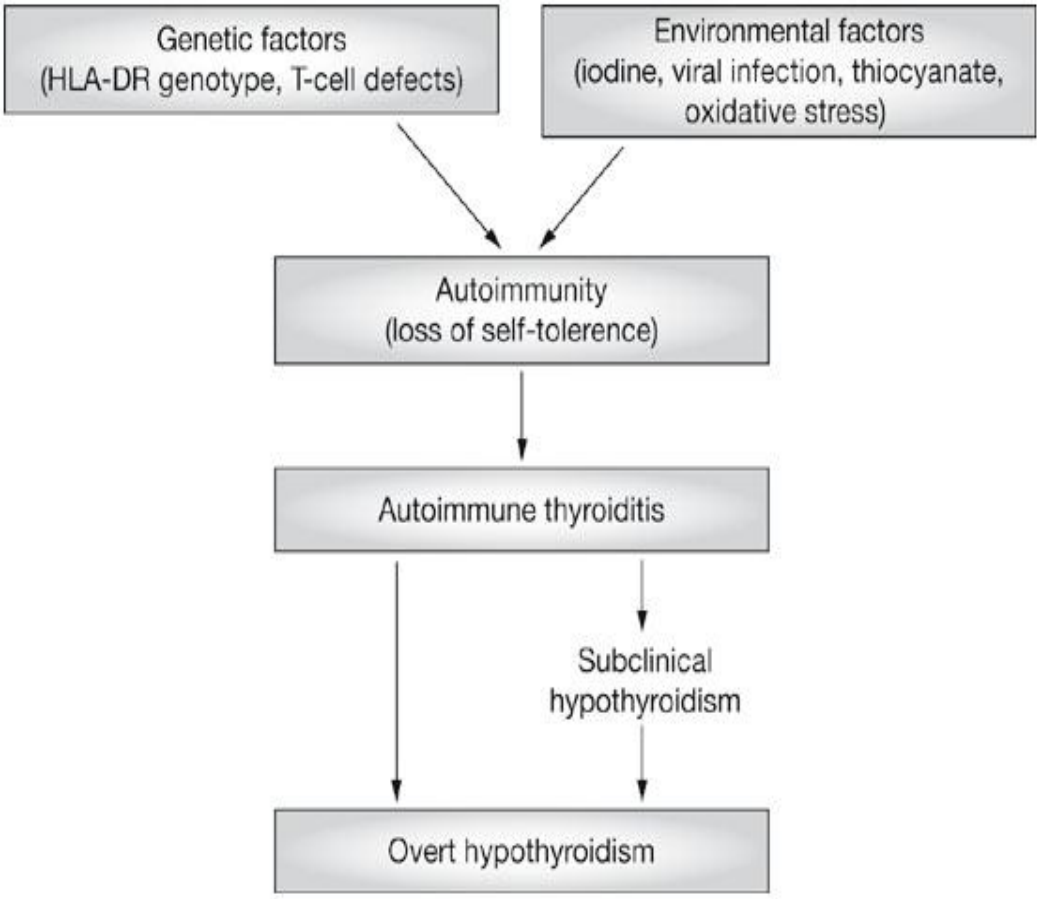
In rare cases of pituitary- or hypothalamus-associated hyperthyroidism, i.e, Secondary, TSH levels may be either normal or raised.

A low TSH value is mostly associated with increased levels of free thyroxine. In some of the persons, hyperthyroidism may result predominantly from increased circulating levels of $T_3$ . In these cases free $T_4$ levels will be decreased, and hence direct measurement of serum $T_3$ must be useful.

Once the diagnosis of thyrotoxicosis is confirmed by a combination of TSH and free thyroid hormone assays, then measurement of radioactive iodine uptake by the thyroid gland can be done to determine the etiology. There may be diffuse increased uptake in the whole gland as in Graves disease, increased uptake in a solitary nodule as in toxic adenoma, or decreased uptake as in thyroiditis.

# REVIEW OF LITERATURE

In a study done by rose et al, in 1999 at Baltimore hospital, Department of microbiology and immunology, Department of Pathology, there was proliferation of T cells from patients of hashimoto's thyroiditis only in the presence of iodinated human thyroglobulin. There was no proliferation in the presence of non iodinated thyroglobulin.



There may be many etiologies for chronic lymphocytic thyroiditis

1. Genetic predisposition
2. Environmental triggers

   Genetic predisposition:

   The occurrence of autoimmune thyroiditis in monozygotic twins varied from 15-50 while in dizygotic twins the occurrence was same as in siblings.

   This proves that susceptibility to autoimmune thyroiditis is due to nonheritable epigenetic factors, that is, it may be due to environmental triggers.

   Environmental triggers:

   Many autoimmune diseases are influenced by infections but there is no such microorganism influencing autoimmune thyroiditis.

   Excessive iodine is the only extensively studied environmental trigger influencing autoimmune thyroiditis.

   Methods by which excessive iodine may trigger autoimmune thyroiditis:

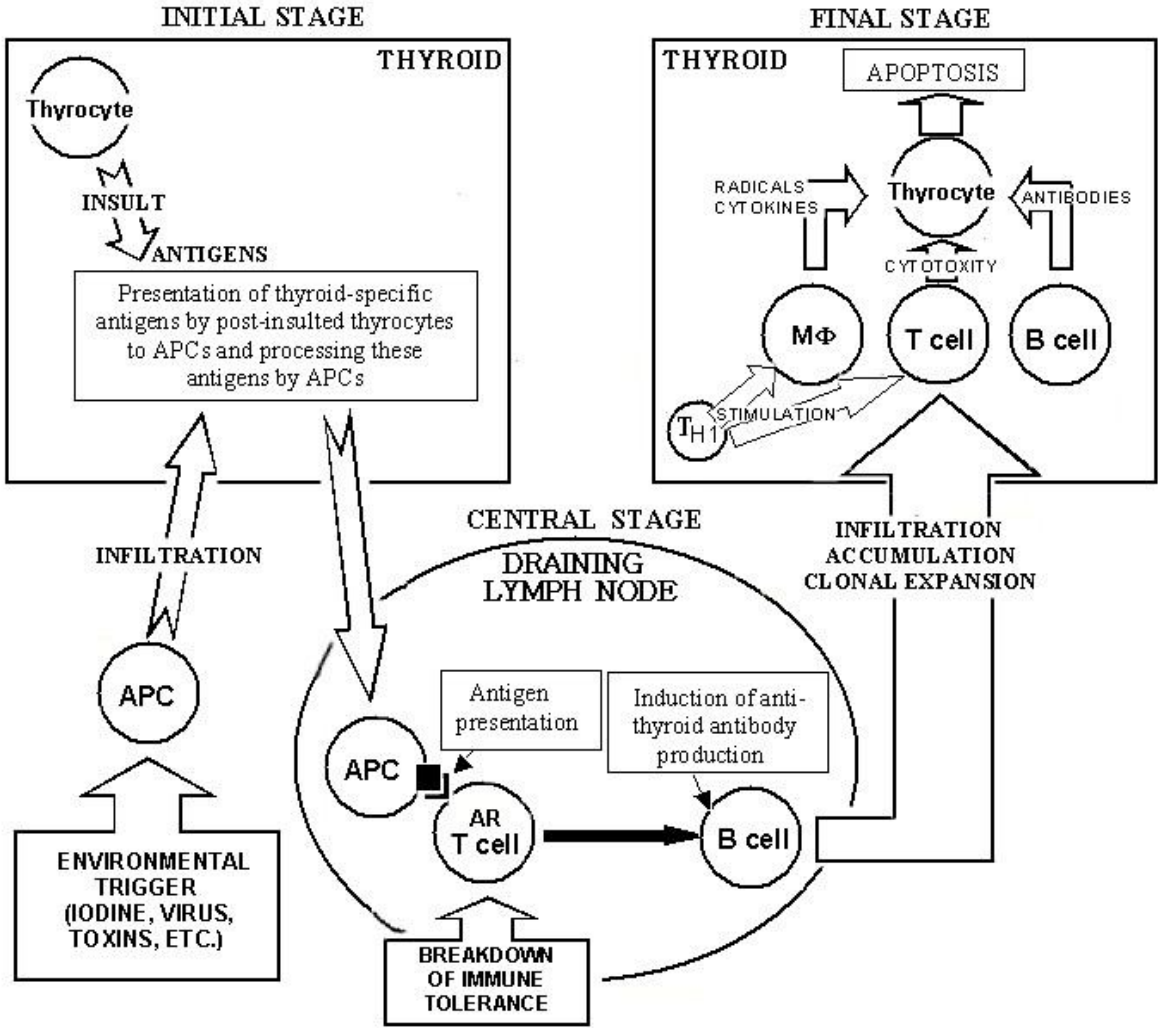
   1. Degeneration of free radicals
   2. Direct injury to thymocytes
   3. Inhibits the sodium iodide pump responsible for trapping iodide.

These tyrosine residues have high affinity for iodine and iodination in early phase occurs only in these 4 tyrosine residues.i.e. they occur only at these specific sites only in particular sequence.

When iodine present in excessive amount, the other tyrosine residues are also iodinated. This produces a conformational change in the thyroglobulin molecule, thereby increasing its stability. The degree of iodization depends on the neighbouring groups and ionization constants and accessibility. When excessively iodinated, the thyroglobulin molecule becomes nondegradable by the cathepsins, which usually degrade normally iodinated thyroglobulin.

Hence the autoimmunogenic potential of iodinated thyroglobulin is increased by the following mechanisms.

1. Changes in processing of antigen
2. Stereochemical shape alteration
3. Production of iodine containing determinants
4. Appearance of cryptic epitopes




Roitt and Coke have demonstrated that murine thyroglobulin reactive T cells can proliferate in the presence of human thyroglobulin only depending on the degree of iodisation of thyroglobulin.

Their study showed that human peripheral T cells from chronic lymphocytic thyroiditis easily proliferated with normal human thyroglobulin while it was not so easy with T cells of

euthyroid individuals. But in the absence of traces of Iodine, none of the T cells were able to proliferate. Thus the degree of iodization of thyroglobulin determines the proliferation of T lymphocytes.

Iodisation effects on immunoreactivity of human thyroglobulin

1. Iodisation alters stereochemical configuration of thyroglobulin making it nondegradable.
2. Creates a novel epitope.

These possibilities were studied by making a panel of murine monoclonal antibodies to human thyroglobulin to proliferate in the presence of both iodinated and noniodinated thyroglobulin. There was significant difference suggesting loss of some epitopes and gain of some epitopes.

One particular monoclonal antibody reacted with only iodinated but not with noniodinated thyroglobulin. The reaction of antibody with noniodinated thyroglobulin was restored once the thyroglobulin was iodinated.

Its affinity was greater for thyroxine but there was less binding for triiodothyronine or reverse triiodothyronine. Hence both the position and number of iodine substitution on thyroxine is important in determination of binding capacity.

Autoimmune thyroiditis was studied in an animal model, NOD $H2^{h4}$ a nonobese diabetic mouse produced by crossing of NOD with B10A(4R) an $H^{2k}$ mouse. NOD $H2^{h4}$ has increased incidence of spontaneous thyroiditis but was absent in either of the parental strains. These mice were given iodine by mixing it in drinking water. When the dose of iodine and duration of feeding was increased, the incidence of thyroiditis also increased in these mice. The severity of the lesion of thyroiditis was also proportional to thyroglobulin specific antibodies.

The disease was transmitted to normal mice when they were given the spleen cells of iodine fed donors.

**Conclusion**

Presence of iodine increases the autoimmunogenic potential of thyroglobulin and the adoptive immunity is also transmissible.

**Chandra AK[(10)]** et al, conducted a cross sectional study in sundarban delta region of West Bengal, Participants were 4656 school children.

**Objective**

Was to study the total goitre rate, thiocyanate excretion pattern of the school going children, urinary iodine and iodine content in edible salt and also in drinking water.

**Methodology**

Children are clinically examined for goiter, urinary iodine and thiocyanate levels are measured, iodine content of drinking water and salt sample are also measured.

**Conclusion**

Goiter prevalence was about 38.2%. Median urinary iodine level was 225 microg/l indicating that there is no biochemical iodine deficiency. They are also consuming dietary goitrogen. Environmental factors other than iodine deficiency may also have role for persistence of endemic goiter.

**Dilip Kumar Das**[9] et al, conducted a Cross-sectional descriptive study in West Bengal, in 2006 which was published in the Journal of the American College of Nutrition, in 2008, Participants were 2400 school children of age 8–10 years.

**Objective**

Was to assess the prevalence of goitre, and to measure urinary iodine excretion levels and to estimate salt iodine content at the household level.

**Methodology**

Goitre was being assessed clinically, urinary iodine level was also analyzed, household salt samples were also tested to measure iodine content .

**Conclusion**

Total goitre rate was found to be 13.7%.Median urinary iodine excretion level was 13g/dL with the normal range being 10–20 g/dL. 80% of salt samples tested was found to have adequate iodine content.

**R.K Marwaha**[(11)], Karak& N.Kochupillai had conducted a Cross sectional study in 6283 healthy school girls of age 10-18years in the year March 1999 at All India Institute of Medical science, Delhi.

**Objective**

Was to evaluate the prevalence of goiter and also thyroid autoimmunity and to assess thyroid functional status in healthy school girls, in the different parts of the country in postiodization phase.

**Methodology**

1810 Goitrous girls 1810 were investigated for T4&TSH, antithyroid antibodies namely TPO and Thyroglobulin, urinary iodine and also cytomorphology by FNAC.

**Results**

The prevalence of goiter was found to be 28%. The prevalence of juvenile autoimmune thyroiditis was found to be 7.5%. Hypothyroidism-6.5%, Subclinical hypothyroidism found to be -15%, Subclinical hyperthyroidism-5.1%.

**Conclusion**

Individuals with Juvenile Autoimmune Thyroiditis were found to have higher prevalence of thyroid dysfunction than without thyroiditis. Their study showed no correlation between urinary iodine and autoimmunity.

**Jinkou Zhao[14] et al,** conducted a cross sectional study in 2371 schoolchildren of age 6 to 15 years, and 607 adults in china and it was published in American Journal of Public Health, October 2000.

**Objective**

To study the relation of iodine content of the household water to the thyroid size and the urinary iodine excretion in the area with high iodine concentration in the water.

**Methodology**

Iodine content of drinking water i.e. well water was measured. Children were assessed for thyroid size, adults were assessed for median urinary iodine excretion.

**Conclusion**

Townships with a higher median level of iodine in well water had a

1. Higher median urinary iodine concentration.
2. Higher prevalence of goiter
3. Higher prevalence of abnormal thyroid volume (P<.001).

This study concluded that excess iodine in water was the cause of endemic goitre.

In the case control study done by **Srinivasan et al**, at Institute of Child Health and Hospital for Children ,Chennai they proved that if UIE is ≥ 300 µg /L then there is 17.94 times more chance of having autoimmune thyroiditis than those who have UIE < 300 µg /L. Cases were autoimmune thyroitis patients, controls were other goiter patients. 90.7 % of cases (n=39) and 37.3% of controls exhibited higher than optimal urinary iodine excretion ( >200µg /L).

Urinary iodine excretion was Significantly higher in children with autoimmune thyroiditis rather children without autoimmune thyroiditis

A descriptive study was done by Duarte et al, in 2009 in sao poaulo cardiology patients.

**Objective**

Complete thyroid workup for patients who are in follow up in cardiology department.

Population-399

Hyperthyroidism i.e overt and subclinical was present in 29 patients (6.5%), whereas hypothyroidism i.e overt and subclinical was found in 32 individuals. Cysts were detected in 2.8% patients. Single nodules were detected in 102 (25.6%), and multinodular goiters were detected in 34 (8.5%). Hashimoto's thyroiditis was present in 16.8% patients, most of whom were women.

Urinary iodine excretion was more in elderly patients than in general population. And also more in men than women. There is higher prevalence of thyroid dysfunction in elderly patients.

In a study conducted by Marwara [(11)]et al,in AIIMS, NEW DELHI goiter patients over a two year period (January 2005-December 2006) were studied.

**Subjects:**

695 school children of age 5-18 years were studied.

**Results:**

Overall, 16% of goitrous children had hypoechogenicity on ultrasound, 15.2% had cytopathological evidence of thyroiditis, 10.6% had positive thyroid peroxidase antibodies and 25.2% had abnormal thyroid function tests. Subjects with hypoechogenicity had higher percentage of thyroiditis on cytopathology, thyroid peroxidase antibody positivity and thyroid dysfunction than those with normal echogenicity.

**Conclusion:**

- Fine Needle Aspiration Cytology is always the gold standard in the diagnosis of autoimmune thyroiditis.
- Thyroid USG is always useful, but has a limited role, in excluding thyroid disease even in children. The sensitivity of thyroid ultrasonography for the diagnosis of autoimmune thyroiditis in children is lesser than that was reported in adults.

A prospective study was done by **Marwara et al,** in Srilanka to study the evolution of autoimmunity during the iodization phase.

**Objective:**

Was to study the evolution of thyroid autoimmunity, in relation to the change in goitre prevalence, during 3 years of iodine prophylaxis in Sri Lanka.

2 cohorts of girls were studied. One in 1998 and the other one in 2001. Prevalence of thyroid Antibody in 2001 was also lower among those with the Antibodies, 34.8% had Thyroglobulin Antibody alone and 46.9% had a combination of Thyroglobulin Antibody and TPO Ab, compared with 82.0% Thyroglobulin Antibody alone in 1998. In 2001, subclinical hypothyroidism was more frequent in Ab+ (6.3%) than Ab-negative girls (1.0%). Only 10 of them (23.8%) remained Antibody positive (mostly TPO Ab and Thyroglobulin Ab) in 2001. Goiter and autoimmunity prevalence was significantly lower than in 1998.

According to an article published by **Lauberg** et al in the Best Res Endocrine Journal in 2000, even small changes in urinary iodine deleteriously affects the thyroid gland.

Both low and high urinary iodine levels cause thyroid dysfunction. Hence they suggested that the iodisation must be curtailed according to the needs of the population after proper monitoring.

In a study done by **weiping** tang in the department of endocrine metabolism in China

| | **Mild iodine deficiency** | **More than adequate** | **Excessive intake** |
|---|---|---|---|
| Overt hypothyroidism | 0.2% | 0.5% | 0.3% |
| Subclinical hypothyroidism | 0.2% | 2.6% | 2.9% |
| Autoimmune thyroiditis | 0.2% | 1% | 1.3% |

Maintenance of TSH levels between 1 to 1.9 reduces the incidence of thyroid dysfunction. Taking Iodine more than needed causes hypothyroidism.

# STUDY JUSTIFICATION

The existing Universal Salt Iodization program has led to the reduction in IDD in endemic population groups. (1)

- Inspite of this prevalence of goiter-13% (2)
- Childhood hypothyroidism is 3%
- Subclinical hypothyroidism of 10% are still seen in our country.

Experience from southeast Asian countries also shows increase in detection of Juvenile autoimmune thyroiditis in post –iodization assessment.

Urinary iodine-prime indicator of a person's iodine nutritional status (3). Hence this study is done to assess the iodine nutritional status in children with acquired hypothyroidism and healthy school children.

# AIM & OBJECTIVE OF THE STUDY

## Primary Objective

To find out the iodine nutritional status in children with acquired hypothyroidism of age 2-12 years attending tertiary care centre.

## Secondary objective

To assess changes in iodine nutritional status in autoimmune thyroiditis.

# MATERIALS & METHODS

**Study Design :** Descriptive study

**Study Place :** 1. Department of Pediatric Endocrinology
Institute of Child Health and Hospital for Children, Egmore.
2. Institute of Biochemistry
Madras Medical College.

**Study Period :** August 2014-September 2014

**Study Population :** 61 cases and 102 healthy children

Two cohorts were selected.

One cohort consisted of cases of acquired hypothyroidism attending endocrinology OPD, ICH and HC during the study period.

**Inclusion Criteria :**

All children with acquired hypothyroidism of age 2-12 years attending Endocrinology OPD during the study period i.e with TSH levels>5μIU/ml

**Exclusion Criteria :**

1. Children with proven congenital hypothyroidism.
2. Children on thyroxine and Iodine containing drugs like cough suppressants.

Other cohort consisted of healthy school children of age 6-12 years from two different schools.

**Ethics involved**

Ethical committee clearance was obtained from the institutional review board.

Informed consent was obtained from the parents/guardian of all the children involved in the study

**Manoeuvre**

Cases were recruited based on the inclusion and exclusion criteria and informed consent was obtained. History taking and clinical examination was done and entered in the data form.

Goiter if present was graded according to WHO criteria

Grade 0 - Thyroid gland invisible and not palpable

Grade 1 - Neck thickening not visible in normal neck position but palpable, thickened mass moves with deglutition

Grade 2 - Neck thickening visible during normal neck position corresponding to the enlarged thyroid gland found on palpation

**Thyroid function tests**

Then free thyroxine and thyroid stimulating hormone levels were measured using enzyme immunoassay method

Free T4 (47-128 ng/ml) and TSH (0.5 – 5µÌU/ml)

**Autoimmune antibody levels**

Autoimmune antibody levels namely, anti thyroid peroxidise antibodies otherwise known as anti microsomal antibodies and thyroglobulin antibodies were measured using electrochemiluminescence assay.

anti TPO > 34 IU/ml

anti TG > 115 IU/ml were considered significant

Ultrasound of thyroid gland was done in all children

**FNAC**

Fine needle aspiration cytology of the thyroid gland was done in all children with goiter.

In case of autoimmune thyroiditis, there will be

1. Marked lymphocytic infiltration of the thyroid with formation of germinal center.
2. Thyroid follicular atrophy is accompanied by oxyphil metaplasia.
3. Absence of colloid.
4. There may be mild to moderate fibrosis.

In case of colloid goiter, there will be large uniform follicles filled with colloid.

Children with FNAC and or autoimmune antibodies positive were diagnosed to have autoimmune thyroiditis.

**Measurement of urinary iodine**

5 ml of urine was collected in plastic screw topped containers from all cases and were stored in - 20 degree centigrade until it reached the labarotory. Then urinary iodine level was measured using Sandell-Kolthoff reaction.

**Principle :**

Urine is digested using ammonium persulphate. Iodine acts as a catalyst in the reduction of yellow coloured ceric ammonium sulphate to colourless cerous form and rate of colour disappearance is noted.

**Procedure :**

First, Urine is mixed to suspend all sediments. Then 250µl of urine is pipetted into 13*100 mm size test tubes. Iodine standards are also prepared. Then all the test tubes are heated to 100°c for 60 minutes and then cooled to room temperature. 2.5 ml of arsenious acid solution is added to all test tubes and made to stand for 15 minutes.300 µl of ceric ammonium sulphate solution is added to each test tube with quick mixing. Exactly after 30 minutes of adding ceric ammonium sulphate ,its absorbance is read at 420 mm using calorimeter.

**Inference :**

A calibration curve is constructed on a graph paper by plotting iodide concentrations on the x axis and its optical density at 405µg/l($OD_{405}$).

**Epidemiological criteria to assess iodine nutrition based on median urine iodine concentration in school children**

| Sl.No. | Median urinary iodine excretion in µg/L | Iodine nutritional status |
|---|---|---|
| 1 | <20 | Severe Iodine deficiency |
| 2 | 20-49 | Moderate Iodine deficiency |
| 3 | 50-99 | Mild Iodine deficiency |
| 4 | 100-199 | Adequate iodine nutrition |
| 5 | 200-299 | Above requirements |
| 6 | >300 | Excessive causing adverse side effects |

**Second cohort:**

A cohort of 102 healthy children from two different school in Chennai were selected. 5 ml of urine was collected from the children and urinary iodine levels were calculated.

Children with high urinary iodine levels (>200 µg/L) were taken as cases. In those children, whose parents gave consent for invasive investigations, thyroid function tests and autoimmune antibody levels were done.

In Children with visible goiter, ultrasound and FNAC thyroid was done. As all the children had high urinary iodine levels, the same test was also done in a group of 50 children in another school.

# RESULTS

## Age Distribution

Our study population consisted of children with acquired hypoyhyroidism of children between the age group of 2-12 years.

Mean age of presentation of acquired hypothyroidism was 9.45 years. The standard deviation was 2.54. Our lowest age of presentation was 3 years which was a case of autoiimune thyroiditis.

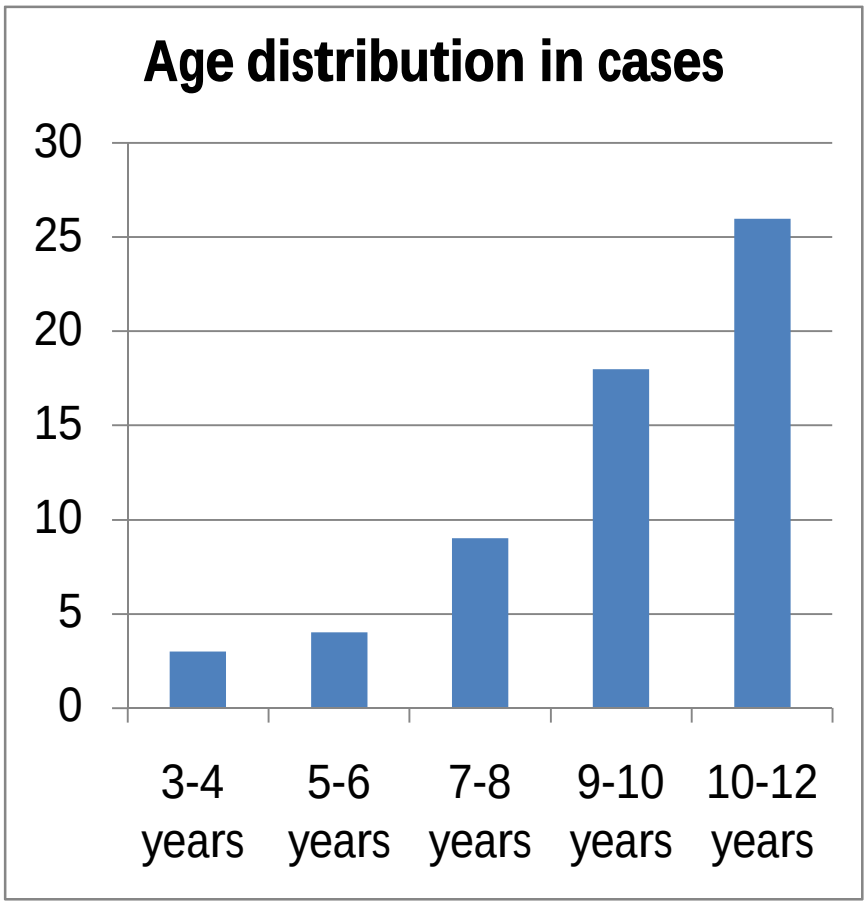



**Chart - 1**

**Sex distribution among the cases**

The Female : Male ratio in our study was 2.7:1.

There was a definitive female preponderance as in all other studies involving autoimmune thyroiditis.

| Gender | Number | Percentage (%) |
|---|---|---|
| Girls | 48 | 78 |
| Boys | 13 | 22 |

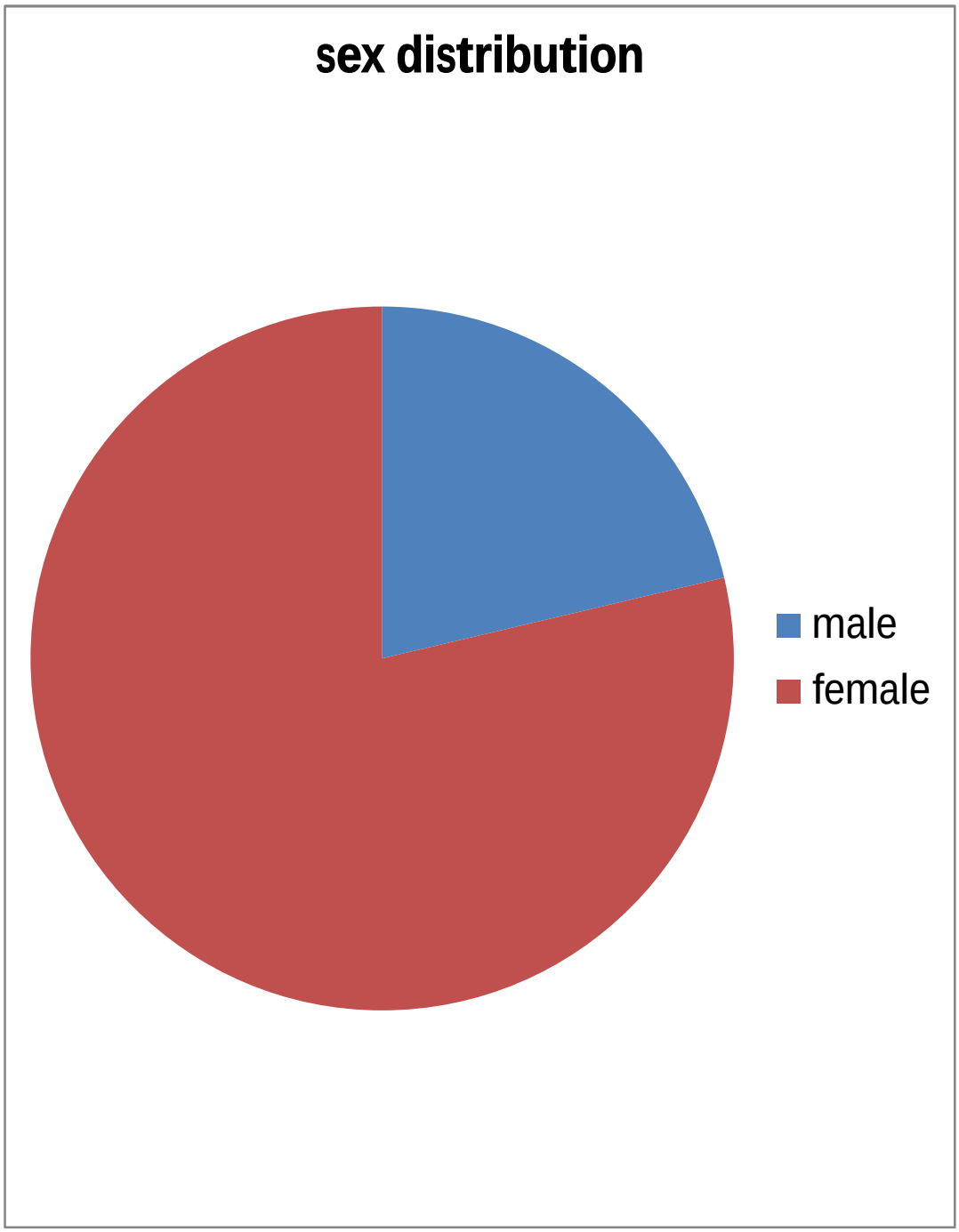



**Chart - 2**

**Autoimmune Thyroiditis**

A diagnosis of autoimmune thyroiditis was done when FNAC and/or antithyroid antibodies were positive.

There were 43 cases of autoimmune thyroiditis others had some other cause.

43 out of 61 had autoimmune thyroiditis with the percentage of 70.

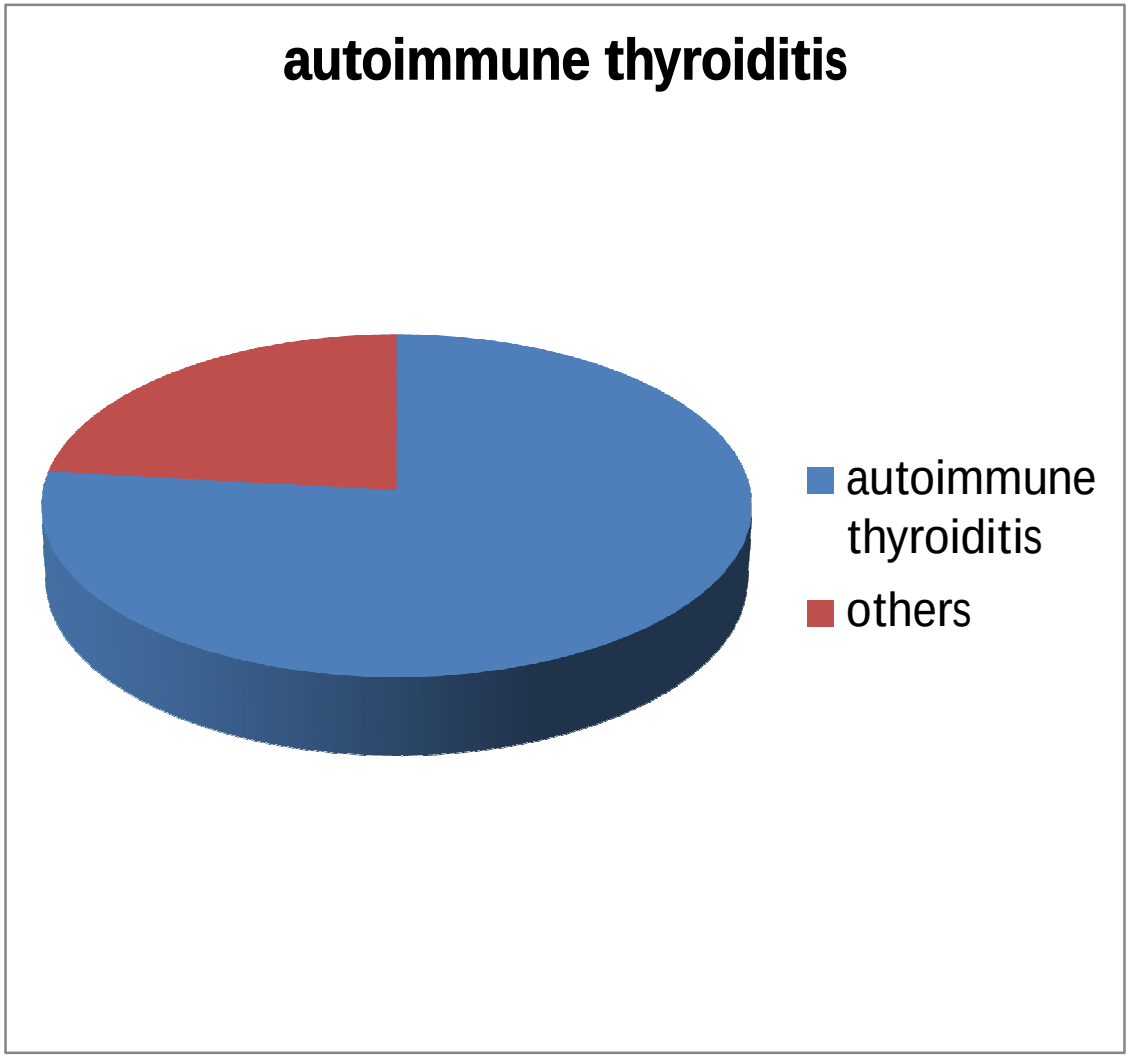



**Chart - 3**

**Ultrasound Thyroid**

Ultrasound of the thyroid gland was done in all the cases of acquired hypothyroidism as it is the sensitive test to pick up thyroid volume as there will be inter-observer error in assessing grade 0 and grade 1.

Ultrasound had reported thyromegaly in 34 cases and Thyroiditis in 25 cases. But only 19 out of 43 cases of autoimmune thyroiditis i.e 44 % had the correct USG diagnosis of thyroiditis.

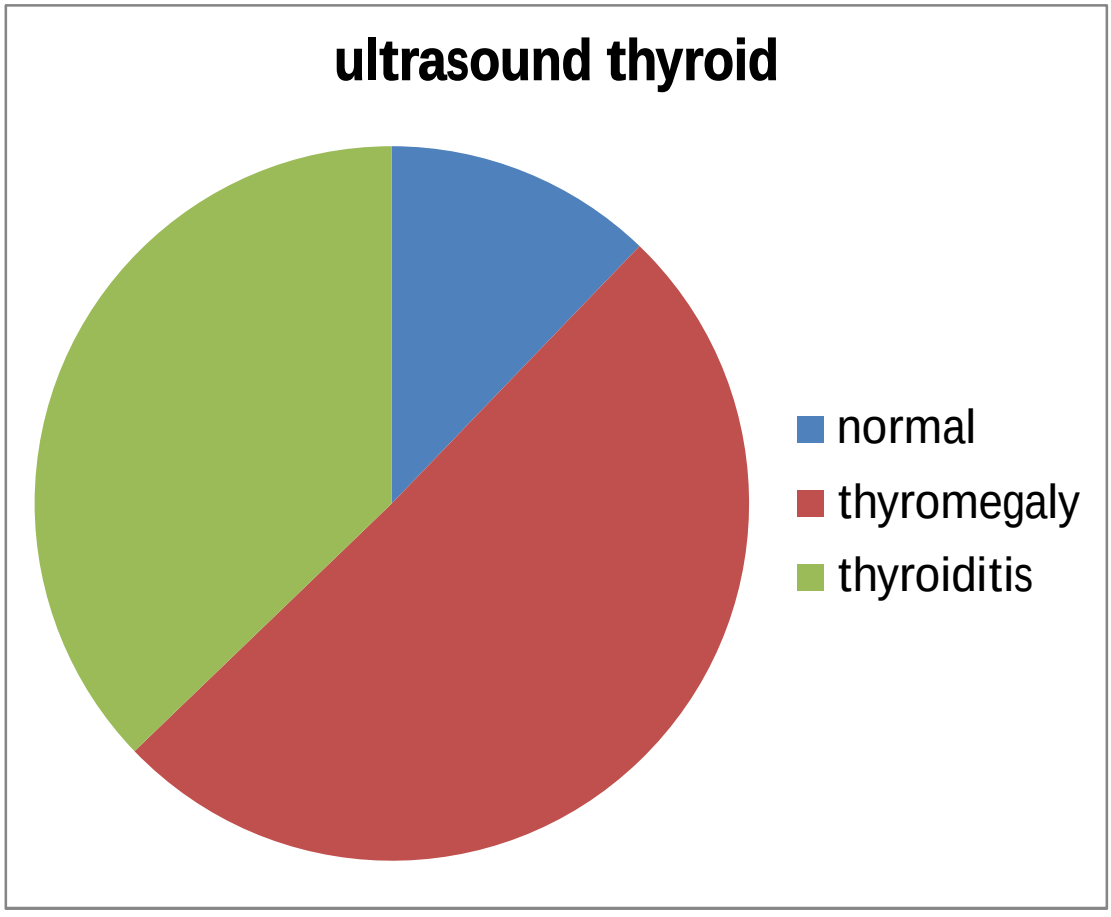



**Chart - 4**

**Results of FNAC**

FNAC is the gold standard for diagnosing autoimmune thyroiditis. In one case FNAC was not done as it was not a case of goiter and the child had came with the complaints of excessive weight gain.

22 out of 43 cases of autoimmune thyroiditis had lymphocytic infiltration in FNAC.38 cases showed excessive colloid accumulation in the thyroid gland.

FNAC has diagnosed 51% of autoimmune thyroiditis cases.

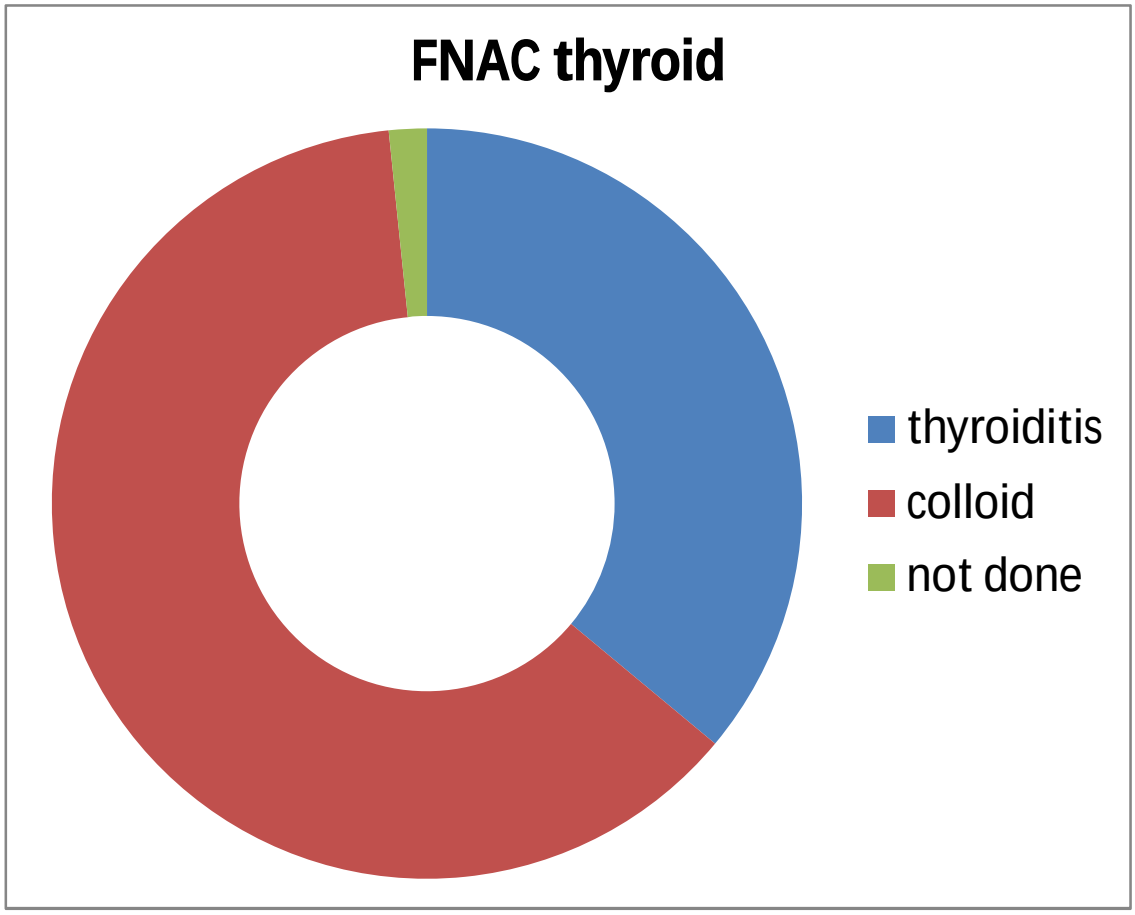



**Chart - 5**

**Comparison of FNAC and ultrasound in diagnosing autoimmune thyroiditis**

FNAC had diagnosed 51% of autoimmune thyroiditis and USG thyroid had diagnosed 44%.

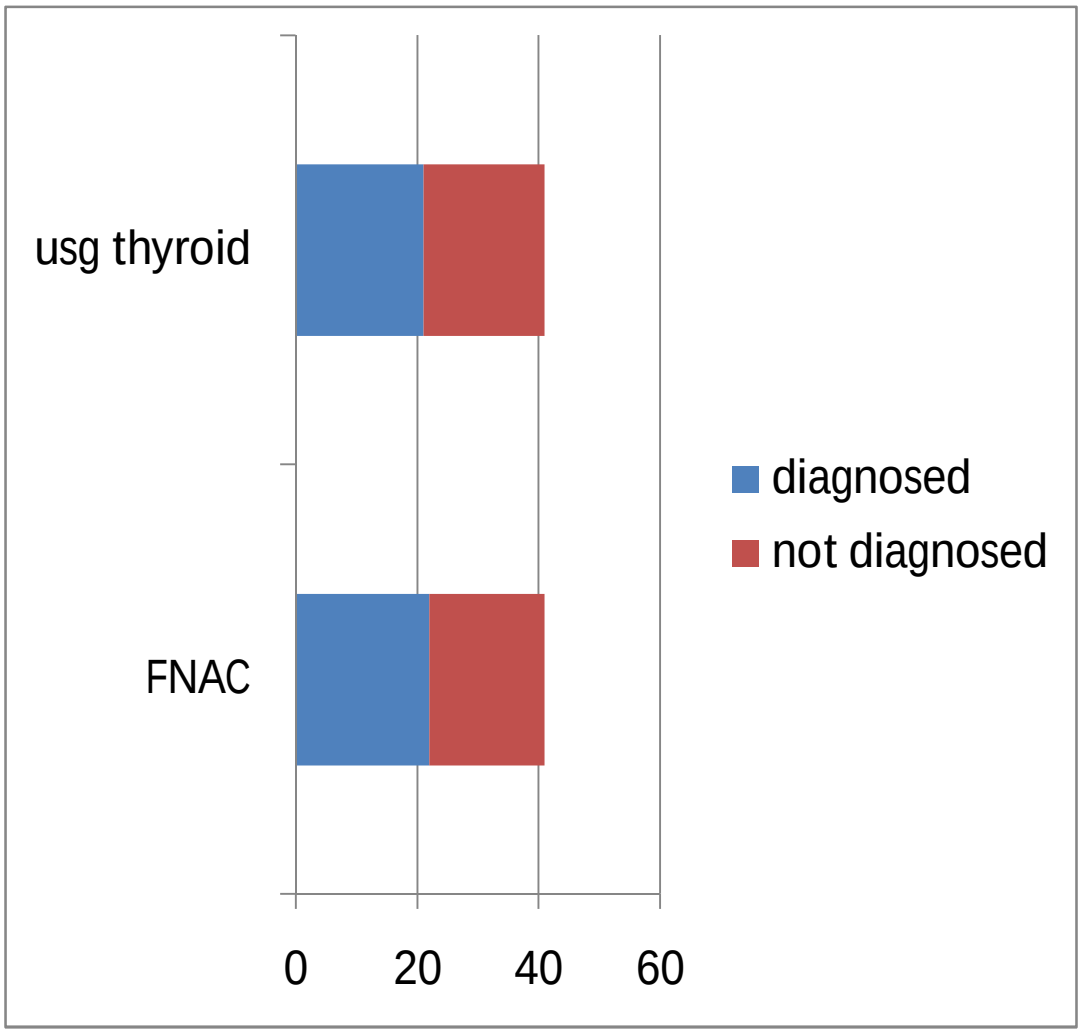



**Chart - 6**

| | **USG** | **FNAC** |
|---|---|---|
| Cases | 25 | 21 |
| Proportions | 0.555556 | 0.489 |

Two sample test for proportions is the test done here.

FNAC is the gold standard test. Hence here the accuracy of Ultrasound in diagnosing autoimmune thyroiditis is tested.

Test statistics is 0.1781

P value is 0.673

That is the hypotheses that ultrasound thyroid diagnosed thyroiditis by chance is ruled out. USG thyroid is as efficient as FNAC in diagnosing thyroiditis.

**Distribution of Anti TPO Antibodies**

The measurement of anti-TPO antibodies had diagnosed 39 out of 43 cases which showed a percentage of 90%.

The lowest value was 0.18 and the highest value was 2700.

The mean value was 373.the standard deviation was 484.

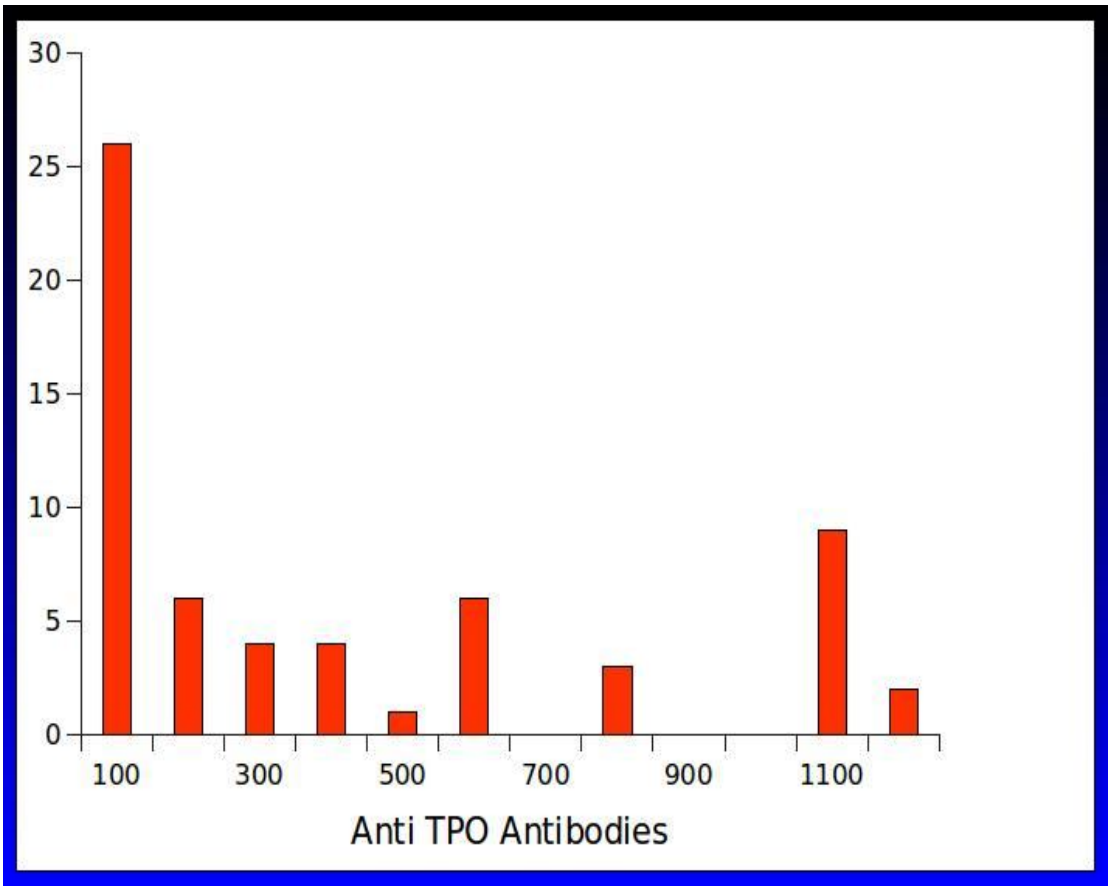



**Chart - 7**

25 cases had values below 100.34 IU/L is considered as positive for diagnosing autoimmune thyroiditis. 37 cases out of 61 had positive anti TPO antibodies.39 out of 43 autoimmune thyroiditis cases had positive TPO antibodies.

**Thyroglobulin Antibodies**

The mean value was 276, the standard deviation was 555.this test was positive in 29 out 0f 43 autoimmune thyroiditis cases. It detected 67% cases of autoimmune thyroiditis.

The smallest value was 0.46.it also had an eccentric value of > 4000.

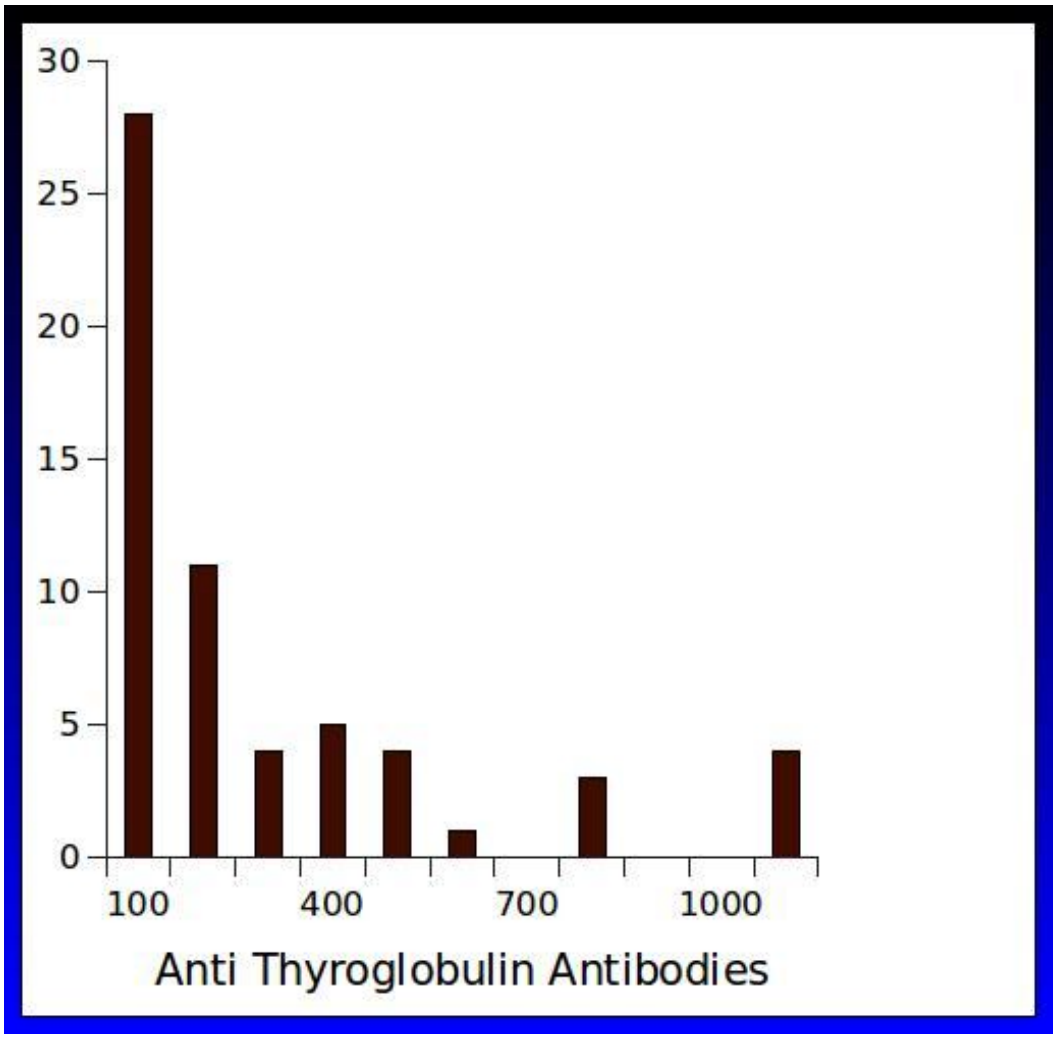



**Chart - 8**

**TSH Levels**

The mean was 33 IU/ml. The standard deviation was 108. 17 cases had TSH value more than 25. There was an eccentric value of 843.

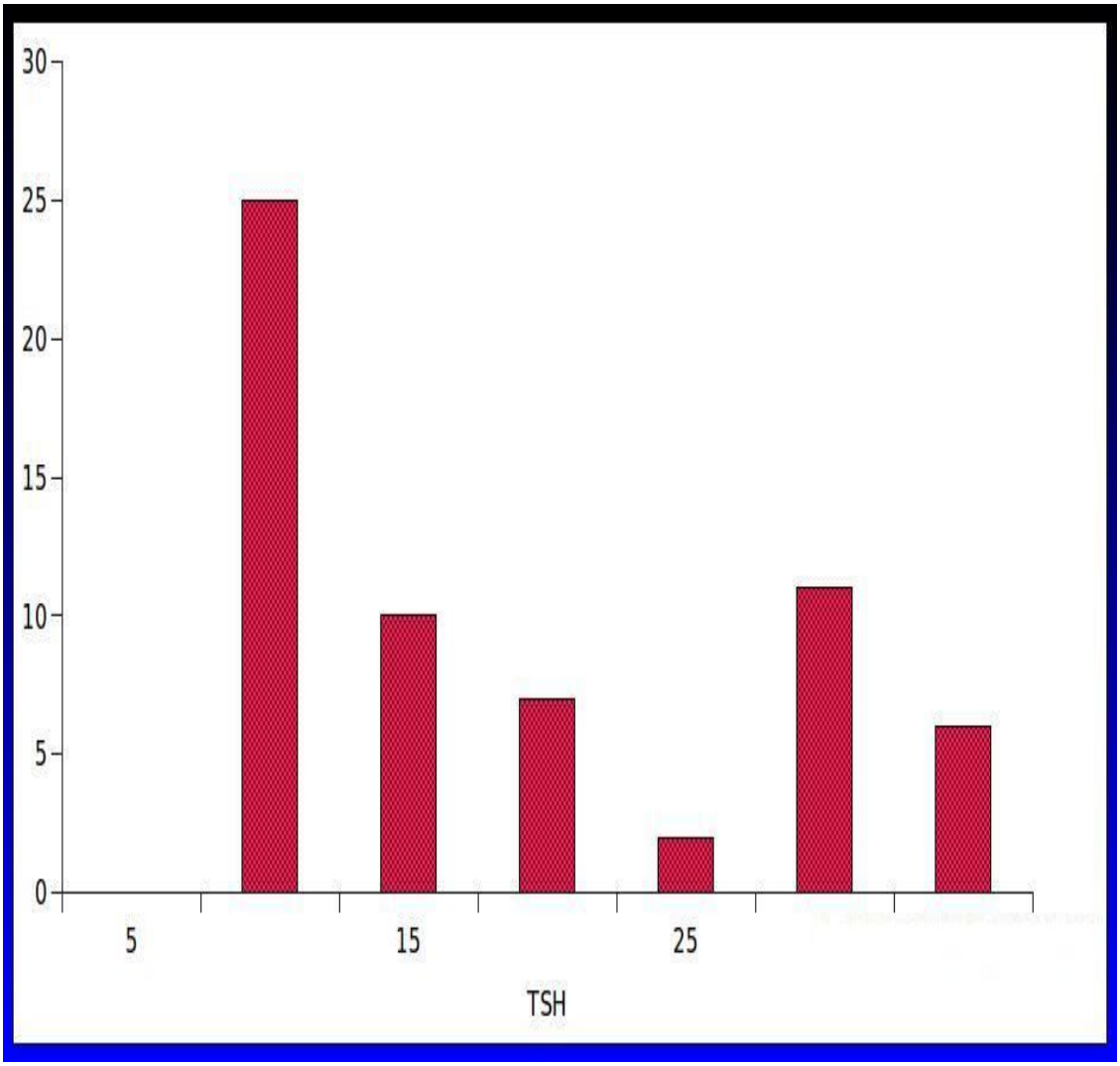



**Chart - 9**

**Goiter Grading by Palpation**

2 cases had no Goiter.  While 52 cases had grade 2 goiter.

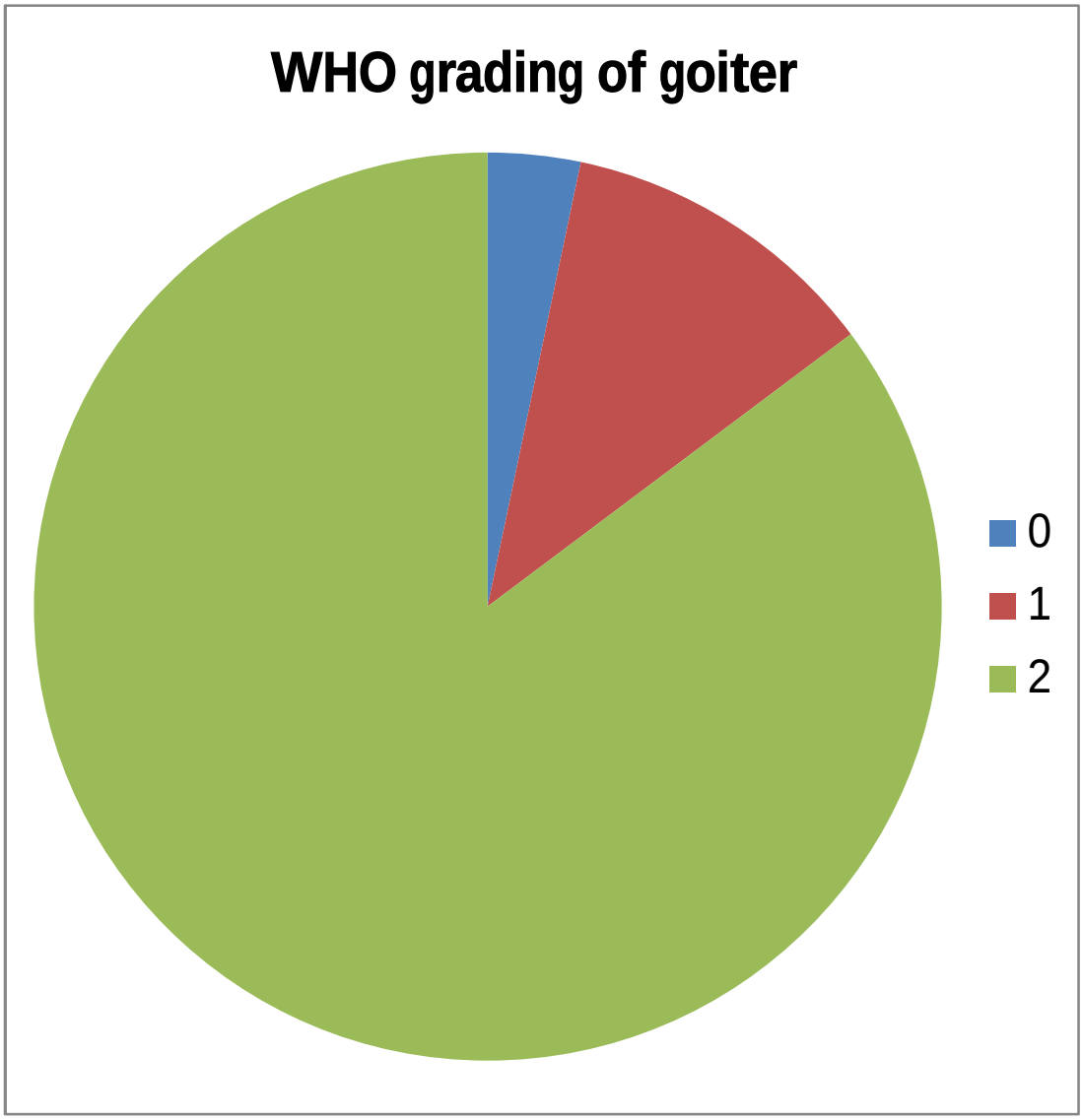



**Chart - 10**

**Urinary Iodine level Distribution**

| Sl.No. | Median Urinary Iodine excretion in µg/L | Iodine Nutritional Status | Cases |
|---|---|---|---|
| 1 | <20 | Severe Iodine deficiency | 0 |
| 2 | 20-49 | Moderate Iodine deficiency | 0 |
| 3 | 50-99 | Mild Iodine deficiency | 1 |
| 4 | 100-199 | Adequate iodine nutrition | 6 |
| 5 | 200-299 | Above requirements | 13 |
| 6 | >300 | Excessive causing adverse side effects | 41 |

Thus only one case had iodine deficiency. While 6 cases had normal iodine excretion,41 cases were having excessive urinary iodine levels.

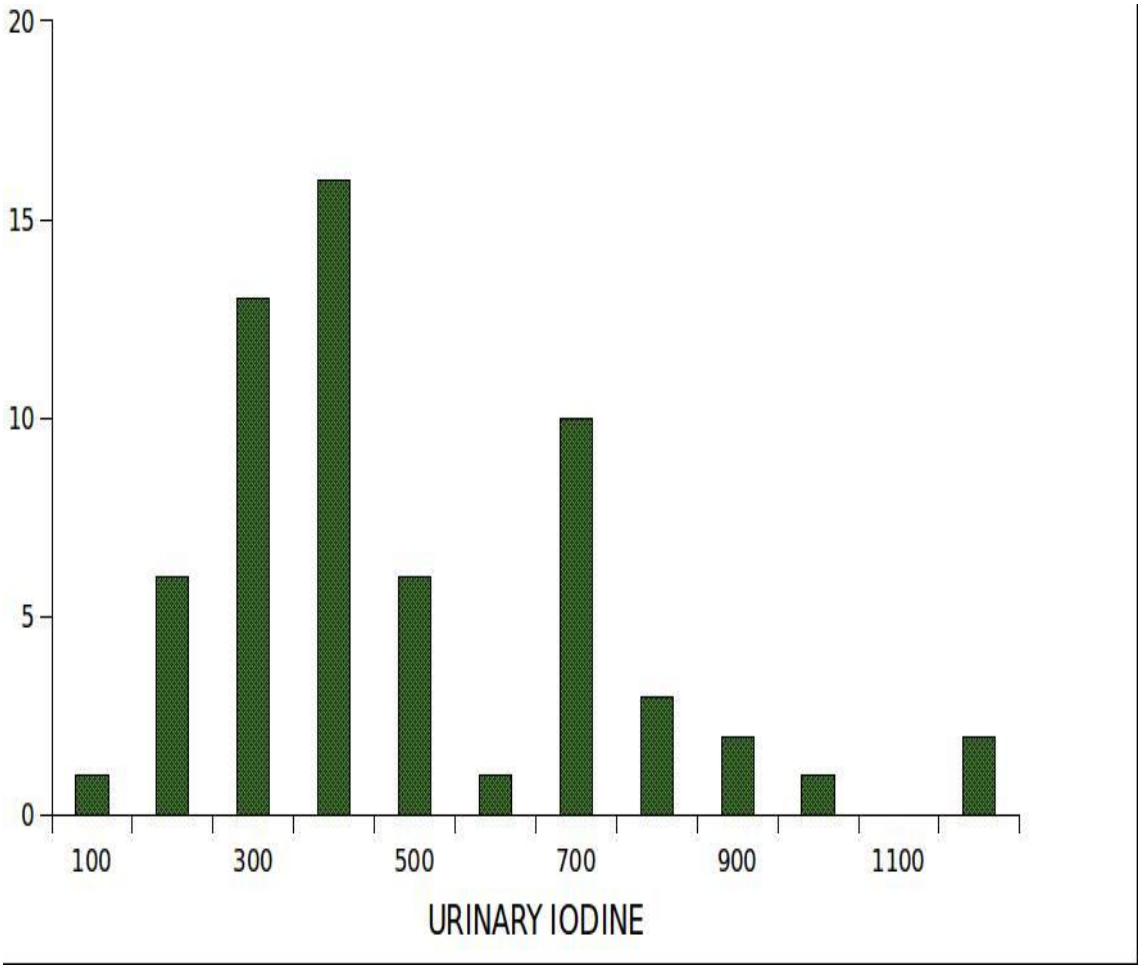



**Chart – 11**

There was an Eccentric value of 1208

**UIE Comparison within the cases**

| Group Statistics | | | | | | |
|---|---|---|---|---|---|---|
| | **AIT** | **N** | **Mean** | **Std. Deviation** | **Std. Error Mean** | **P-value** |
| UIE | 1 | 43 | 495.65 E2 | 288.38140 | 43.97775 | 0.009 |
| | 0 | 18 | 307.11 E2 | 224.13225 | 52.82848 | |

Within the cases group, mean UIE was 495 for autoimmune thyroiditis group which was significantly higher than the other group of hypothyroidism.

*** UIE Group Cross Tabulation**

| | | | UIE Group | | | | Total |
|---|---|---|---|---|---|---|---|
| | | | **<100** | **100-199** | **200-299** | **>300** | |
| VAR00011 | Control | Count | 0 | 0 | 1 | 49 | 50 |
| | | % within control | .0% | .0% | 2.0% | 98.0% | 100.0% |
| | | % within UIE group | .0% | .0% | 7.1% | 54.4% | 45.0% |
| | Cases | Count | 1 | 6 | 13 | 41 | 61 |
| | | % within cases | 1.6% | 9.8% | 21.3% | 67.2% | 100.0% |
| | | % within UIE group | 100.0% | 100.0% | 92.9% | 45.6% | 55.0% |
| Total | | Count | 1 | 6 | 14 | 90 | 111 |
| | | % within VAR00011 | .9% | 5.4% | 12.6% | 81.1% | 100.0% |
| | | % within UIE group | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% |

Fisher exact test-17.701, P-value -0.000

Among the cases 1.6% had iodine deficiency.67% were consuming excessive iodine in salt.

Among the healthy children group, 2 had iodine deficiency with the lowest value of 2.5.

**Comparing Urinary Iodine with Anti TPO antibodies in cases**

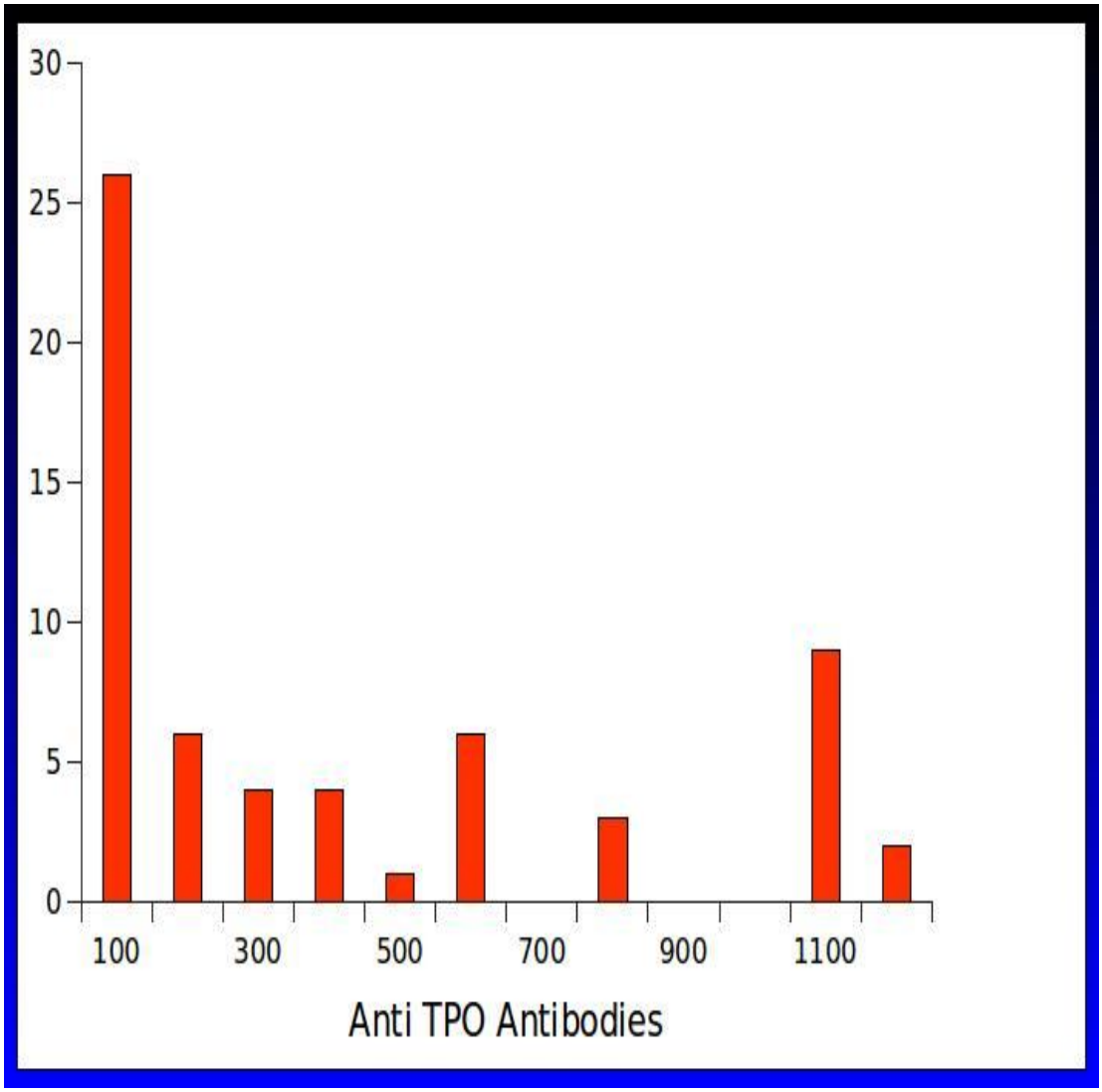



**Chart – 12**

R value is 0.35

$R^2$ value is 0.127. The correlation between the change in antibody level and urinary iodine is very weak.

t – 2.933

Degree of freedom = 59

P value = 0.04724

**Comparing urinary iodine with thyroglobulin antibodies in autoimmune thyroiditis**

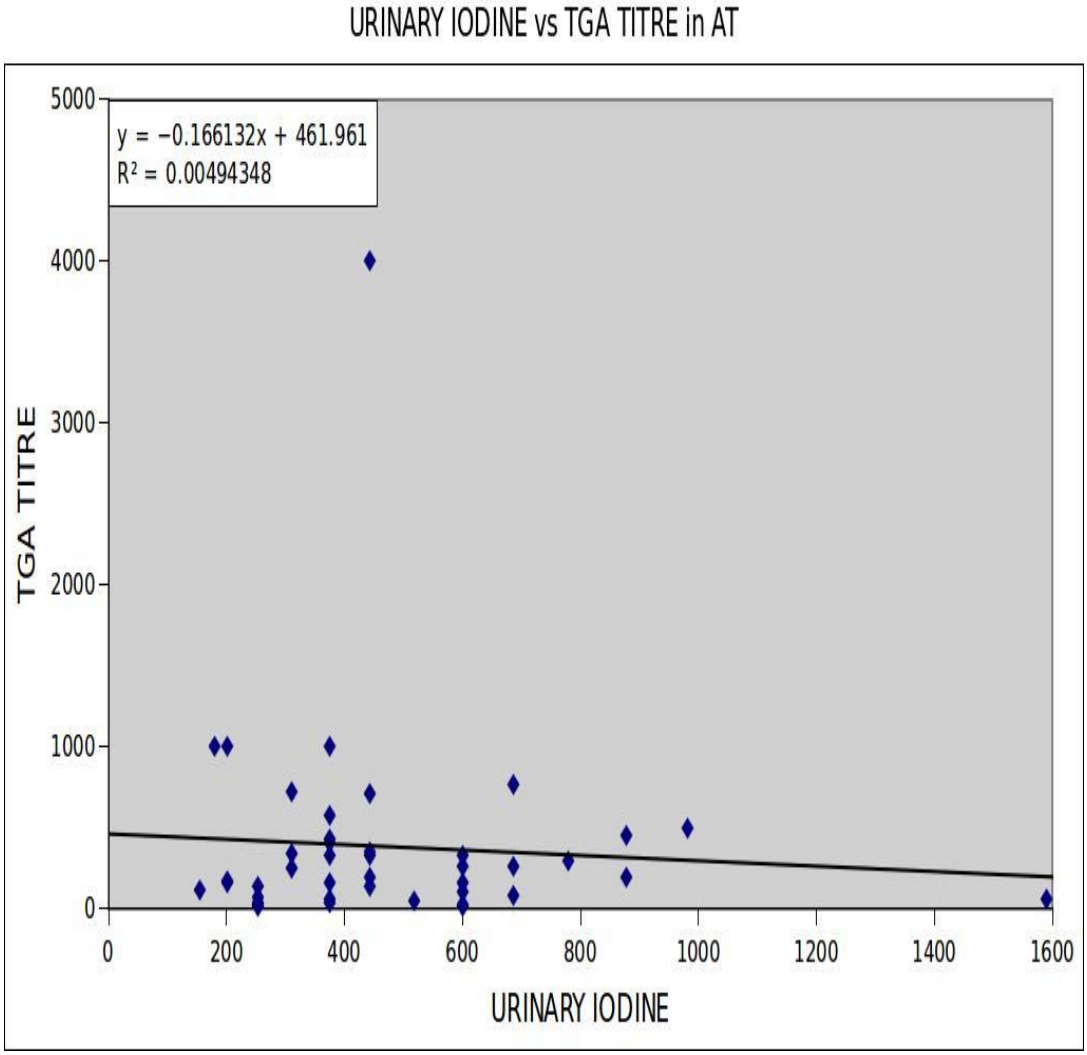



**Chart - 13**

R value is 0.35

$R^2$ value is 0.049. The correlation between the change in antibody level and urinary iodine is very weak.

t- 0.4458, degree of freedom=40, p value=0.65

Correlation by pearson's test is very weak. There is no correlation between TGA and urinary iodine in autoimmune thyroiditis patients.

**Comparing urinary iodine values of autoimmune thyroiditis patients and healthy school children**

Wilcxon-Mann whitney test was used to test the median between urinary iodine values of autoimmune thyroiditis and healthy school children.

P value is 0.0000

It is highly significant.

There is no correlation between the urinary iodine values of autoimmune thyroiditis and healthy school children.

**Comparing urinary iodine with anti TPO antibodies in patients with autoimmune thyroiditis**

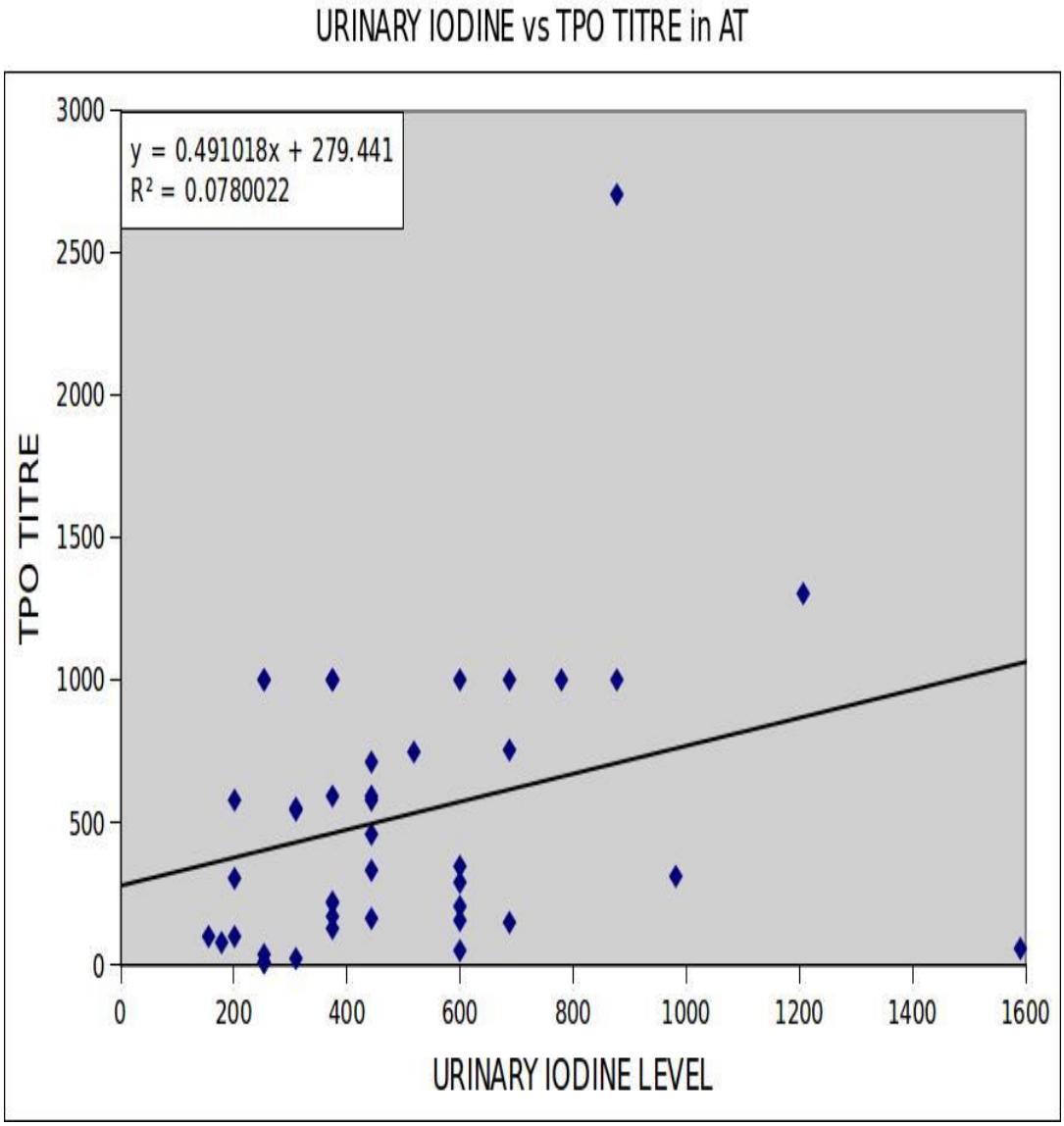



**Chart - 14**

$R^2$ value is 0.078. The correlation between the change in antibody level and urinary iodine is very weak.

t- 1.8624

Degree of freedom=41

P value= -0.06972

95% confidence interval (-0.02- -0.534)

Correlation by pearson's test is very weak. There is no correlation between anti TPO antibodies and urinary iodine in autoimmune thyroiditis patients.

**Comparing anti TPO antibodies and urinary iodine in healthy school Children**

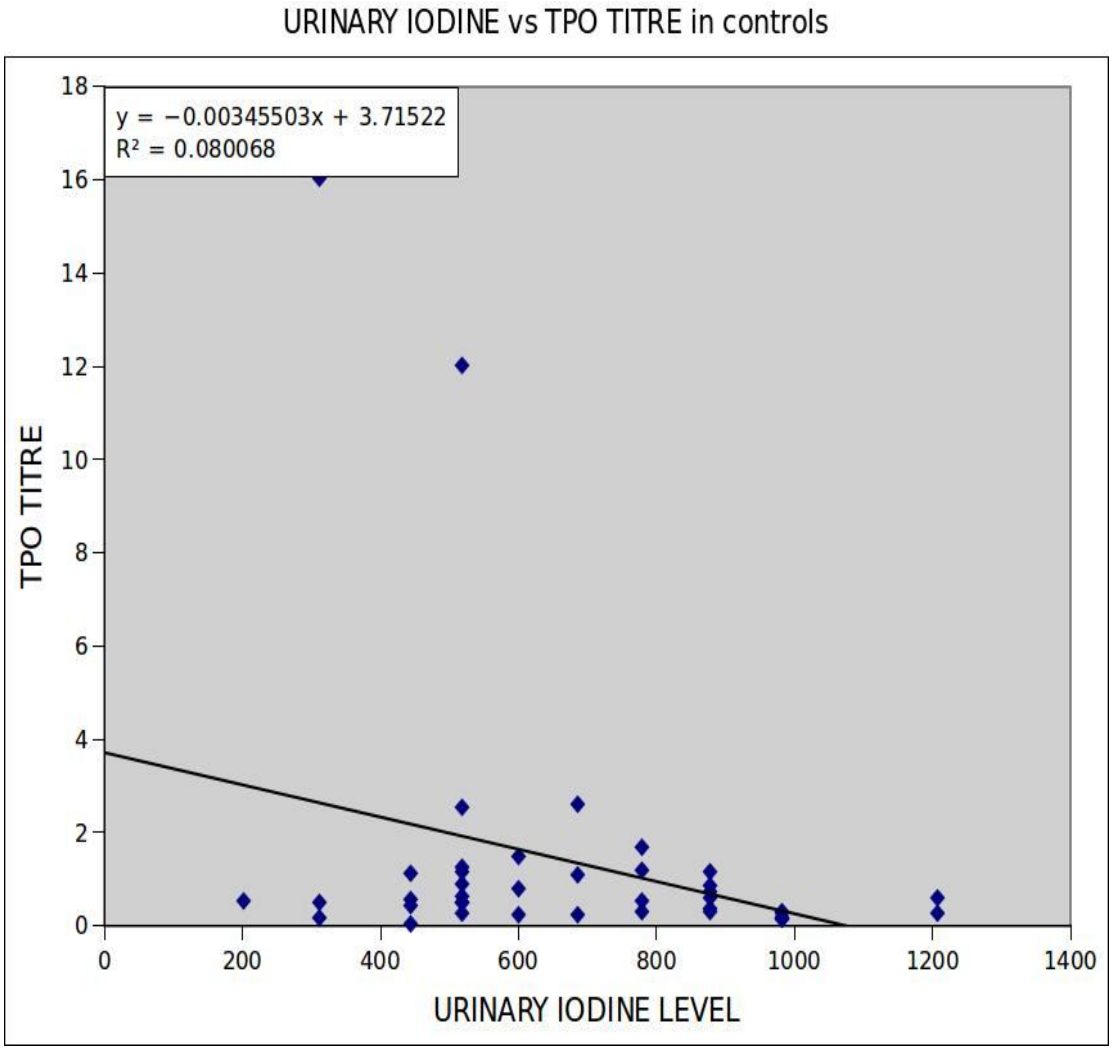



**Chart - 15**

t -1.8186

df 38

p -0.076

correlation –0.2829

There is no correlation between TPO and urinary iodine in healthy children.

**Iodine Nutritional Status in healthy children**

| Sl.No. | Median urinary iodine excretion in µg/L | Iodine Nutritional Status | Healthy School Children |
|---|---|---|---|
| 1 | <20 | Severe Iodine deficiency | 1 |
| 2 | 20-49 | Moderate Iodine deficiency | 0 |
| 3 | 50-99 | Mild Iodine deficiency | 1 |
| 4 | 100-199 | Adequate iodine nutrition | 1 |
| 5 | 200-299 | Above requirements | 1 |
| 6 | >300 | Excessive causing adverse side effects | 98 |

**Iodine Nutritional Status in healthy children**

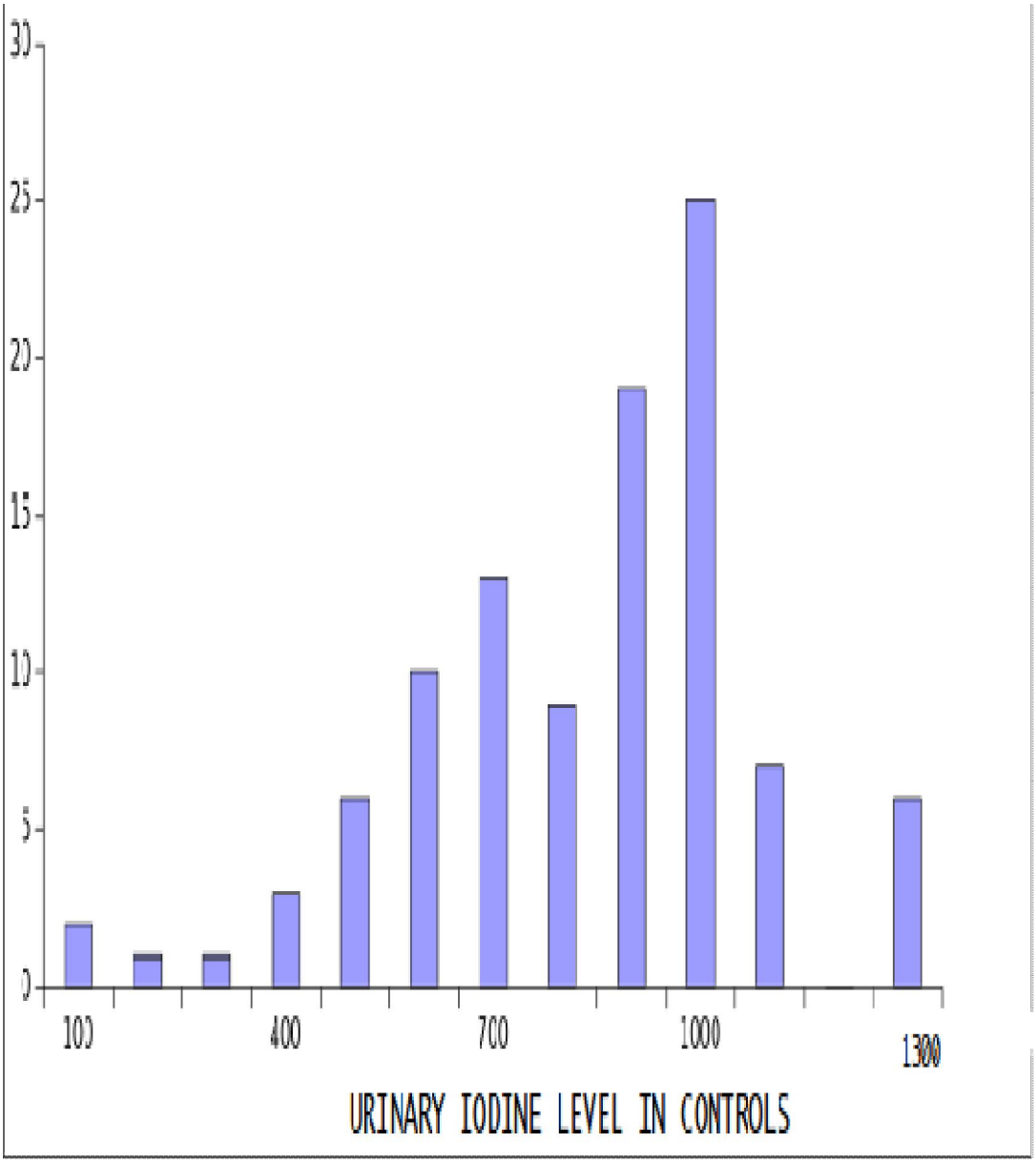



**Chart - 16**

The mean for urine iodine excretion is 789.9. The standard deviation is 269.2.

Among the schoolchildren 1 had mild iodine deficiency and 1 had severe iodine deficiency.

The urinary iodine excretion was higher than the cases. There was an eccentric value of 1590.9.

**Comparing urinary iodine excretion and TGantibodies in healthy children**

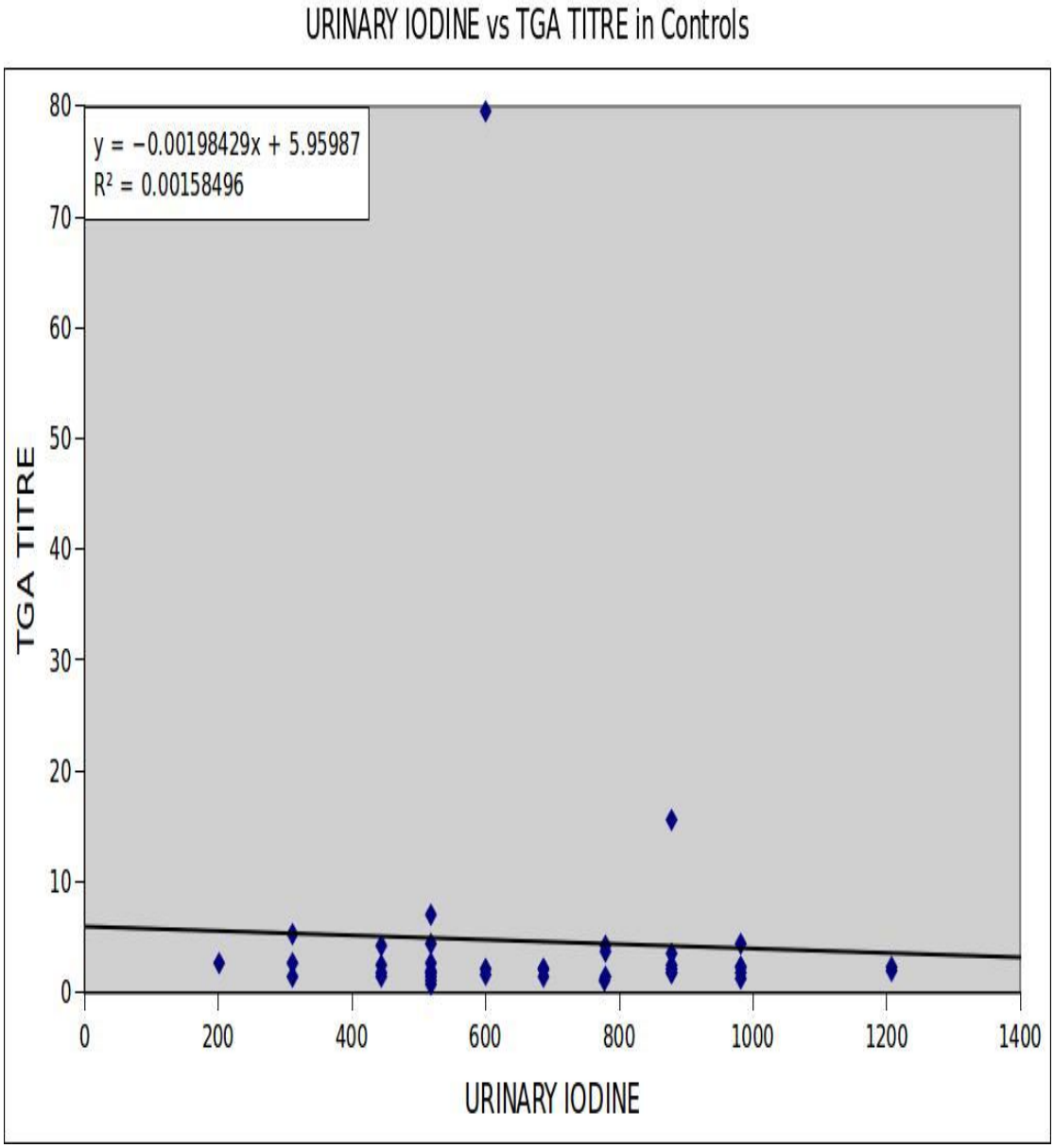



**Chart – 17**

t=-0.2456

Degree of freedom-38

P value -0.8073

Correlation –0.0398

There is no correlation between urinary iodine and TG antibodies in healthy children.

# DISCUSSION

Autoimmune thyroiditis is the most common cause of acquired hypothyroidism, which has genetic predisposition and is also influenced by environmental triggers. This has a slight female preponderance.

Our study also showed that 70% of the acquired hypothyroidism cases were due to autoimmunity. The female : male ratio was 2.7:1.mean age of presentation was 9.45 years.

Chandra K et al showed the mean urine iodine value to be 225 µg/L. Our study showed the mean urine iodine value to be 449 in cases and 789.9 in healthy children.

Jinkuo Zhao et al, and Weiping et al in their studies showed that there is no correlation between urinary iodine and hypothyroidism.

Srinivasan et al showed that excessive urinary iodine may trigger autoimmune thyroiditis.

Chandra et al showed that environmental trigger may play a role in triggering autoimmunogenic potential.

But our study showed that there is no correlation between urinary iodine and antibody levels both in cases as well as healthy school children.

Dilip kumar et al showed that no longer iodine deficiency is present. And 80% of the household were using salt which were adequately iodized. Our study did not measure the level of salt iodisation.

Marwara et al showed that Ultrasound of the thyroid gland though useful, is not sensitive in diagnosing thyroiditis. But our study showed that ultrasound is as good as FNAC in diagnosing thyroiditis.

# CONCLUSION

The cause for acquired hypothyroidism and goiter is no longer iodine deficiency.

The median urinary iodine excretion in the healthy school children was higher than that of the autoimmune thryoditis group in our study.

In our study there was no correlation between the urinary iodine excretion and antithyroid antibodies.

Thus our study contradicts the results of previous studies.

**Postulated hypothesis for excess urinary iodine excretion**:

Universal salt iodisation is successful in our state. The excess urinary iodine excretion is attributed to intake of some other goitrogens or iodine containing substances.

# LIMITATIONS

a. Auto immune antibody levels for all the school children could not be done as the parents did not give consent for invasive blood testing.
b. Assessment of iodine content at the house hold level and production level was not done.
c. Excessive urinary iodine in autoimmune thryoditis could not be attributed to excessive salt iodisation as preiodisation data is lacking.

# RECOMMENDATIONS

Iodine supplementation programme must be tailored only to the iodine deficient areas as in pre1992 era with proper monitoring of the iodine content at the household level and also the median urinary excretion level.

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    Iodine intake as a determinant of thyroid disorders in populations.

    Laurberg P[1], Cerqueira C, Ovesen L, Rasmussen LB, Perrild H, Andersen S, Pedersen IB, Carlé A

# PROFORMA

1. Name:
2. Age:
3. Gender:
4. Endocrine department No:
5. Parent’s name:
6. Address:
7. Phone number:
8. History :

- History suggestive of hypothyroidism-
- H/o intake of goitrogens
- H/o drug intake
- H/o salt intake, whether iodised?
- History suggestive of hyperthyroidism
- H/o irradiation
- Family history:

9. Anthropometry:
   -height
   -weight
   -BMI percentile
10. WHO grading of goiter:

11.Thyroid function tests:

T3:

T4:

TSH:

12.FNAC thyroid:

13.Thyroid auto antibodies levels:

Anti-TPO:

Anti-thyroglobulin:

14.Urinary Iodine levels:

# INFORMATION SHEET

## IODINE NUTRITIONAL STATUS IN CHILDREN WITH ACQUIRED HYPOTHYROIDISM

Investigator name : Dr. Divya.M

Guide : Dr. S.Sundari MD ,Dch.

(To be read to caretakers in the presence of witness)

Hypothyroidism can cause significant growth and mentalretardation. Till the last decade iodine deficiency has been implicated as the prime cause of hypothyroidism.. Recent studies have suggested that increased iodine levels may be associated with autoimmune thyroiditis. Hence this study is done to find out the iodine nutritional status in children with acquired hypothyroidism.

How is the study being done?

Children with acquired hypothyroidism will be thoroughly evaluated by clinical history taking, clinical examination and investigations like thyroid function tests , FNAC, autoantibodies levels and urinary iodine levels .Healthy schoolchildren will be evaluated by history, clinical examination and urinary iodine levels. If urinary iodine levels are high, the children will be evaluated as cases. 2 ml of blood will be taken for doing thyroid function tests and autoimmune antibody levels.

Can I refuse to join the study?

You may refuse to participate or withdraw from the study at any time. In both cases your child will be treated in the usual manner in the hospital.

Is there any benefit or harm from this study?

Your child's health status is known & also the data obtained can be used for community benefit & for the advancement of medical research.

There is no harm to your child from this study.

**Confidentiality:**

The data collected from the study will be used for the purpose of the study only. If the results of the study are to be published personal information of the children participating in the study will be kept confidential. There will not be any disclosure about your child's information without your permission.

How will your decision to not participate in the study affect your child?

Your decisions to not to participate in this research study will not affect your child's medical care or your relationship with the investigator or the institution. Your doctor will still take care of your child and the child will not lose any benefits to which he/she is entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during course of the study without giving any reasons.

I have been fully informed about the study and the benefits to my child and the possible harm that can happen.

This authorisation is valid only for this study. “I have understood and receive a copy of the consent form”. I agree for my child’s participation in this study.

Signature/Thumb print of the parent/guardian:

Signature of the investigator:

Witness signature:

Date:

Principal investigator:

Address:

Phone:

# CONSENT FORM

**STUDY PLACE : INSTITUTE OF CHILD HEALTH AND HOSPITAL FOR CHILDREN, ENDOCRINOLOGY OPD**

**TITLE OF THE STUDY: IODINE NUTRITIONAL STATUS IN CHILDREN WITH ACQUIRED HYPOTHYROIDISM**

Name of the Investigator : Dr.M.DIVYA

Name of the Participant : Age: Sex:

Hospital number : Endocrine no:

1. I have read and understood this consent form and the information provided to me regarding the participation in the study.

2. I have had the consent document explained to me.

3. I have been explained about the nature of the study.

4. I have been explained about my rights and responsibilities by the investigator.

5. I have informed the investigator of all the treatments I am taking or have taken in the past including any native (alternative) treatment.

6. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms. *

7. I have not participated in any research study in the past.

8. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital. *

9. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent. *

10. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.

11. I have understood that my identity will be kept confidential if my data are publicly presented

12. I have had my questions answered to my satisfaction.

13. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document. For adult participants:

Name and signature /parents/guardian

Name_____ Signature_____ Date_____

Name and Signature of impartial witness:

Name_____ Signature_____ Date_____

Name and Signature of the investigator or his representative obtaining consent:

Name_____ Signature_____ Date_____

# MASTER CHART

## School Children

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | V.MANIKANDAN | 3.10.06 | 7 | M | 26 | 1.24 | 16.9 | 878.1 | | | | |
| 2 | J.PRABHAKARAN | 31.12.06 | 7 | M | 19 | 1.14 | 14.6 | 779.1 | 1.29 | 3.15 | 1.08 | 0.53 |
| 3 | S.AKASH | 23.10.06 | 7 | M | 18 | 1.17 | 13.2 | 600.4 | 1.27 | 3.97 | 1.98 | 0.22 |
| 4 | T.MATHEW JOHN | 7.01.07 | 7 | M | 18 | 1.18 | 12.9 | 311.6 | 1.42 | 3.18 | 1.42 | 0.16 |
| 5 | D.YUGESH | 7.04.07 | 7 | M | 20 | 1.21 | 13.7 | 202.1 | 1.78 | 1.61 | 2.53 | 0.52 |
| 6 | A.ALVINRAJ | 23.04.06 | 8 | M | 21 | 1.21 | 14.3 | 878.1 | | | | |
| 7 | D.DEVAKUMAR | 21.11.06 | 7 | M | 21 | 1.26 | 13.2 | 444.3 | | | | |
| 8 | J.GNANASEKAR | 19.06.06 | 8 | M | 20 | 1.23 | | 519.4 | 1.47 | 1.88 | 0.94 | 0.49 |
| 9 | A.HARIDOSS | 1.06.06 | 8 | M | 16 | 1.15 | 13.2 | 1208 | 1.46 | 1.89 | 2.17 | 0.57 |
| 10 | V.SANJAY | 2.10.06 | 7 | M | 22 | 1.2 | 15.3 | 519.4 | 1.42 | 1.88 | 1.77 | 1.15 |
| 11 | NAINAR MOHAMMED | 27.04.07 | 7 | M | 20 | 1.18 | 14.4 | 600.4 | 1.18 | 7.87 | 79.35 | 0.78 |
| 12 | AJITH KUMAR | 26.10.06 | 7 | M | 25 | 1.24 | 16.3 | 444.3 | 1.62 | 2.91 | 4.17 | 0.41 |
| 13 | N.ANGEL JAYANTH | 10.09.06 | 8 | F | 26 | 1.26 | 16.4 | 444.3 | 1.36 | 2.5 | 2.43 | 0.56 |
| 14 | EMIMA | 26.12.06 | 7 | F | 20 | 1.23 | 13.2 | 444.3 | 1.18 | 2.63 | 1.68 | 1.12 |
| 15 | A.JAYAPRAKASH | 14.03.07 | 7 | M | 25 | 1.33 | 14.1 | 600.4 | 1.33 | 2.25 | 1.49 | 1.46 |
| 16 | C.VIJAY | 31.05.07 | 7 | M | 18 | 1.16 | 13.4 | 444.3 | 1.28 | 1.43 | 1.41 | 0.04 |
| 17 | B.KARTHIKEYAN | 18.05.07 | 7 | M | 22 | 1.24 | 14.3 | 519.4 | | | | |
| 18 | V.RADHAKRISHNAN | 27.08.07 | 7 | M | 23 | 1.22 | 15.5 | 519.4 | 1.29 | 2.66 | 0.71 | 1.24 |
| 19 | M.MUTHU | 6.09.06 | 8 | M | 20 | 1.23 | 13.2 | 519.4 | 1.39 | 2.86 | 1.71 | 2.54 |
| 20 | D.SAMUEL | 10.03.07 | 7 | M | 25 | 1.33 | 14.1 | 311.6 | 1.64 | 1.34 | 2.51 | 0.47 |
| 21 | D.IMMANUEL | 04.06.07 | 7 | M | 22 | 1.2 | 15.3 | 779.7 | 1.24 | 2.47 | 1.35 | 1.17 |
| 22 | S.KISHORE KUMAR | 6.12.06 | 7 | M | 23 | 1.19 | 16.2 | 687.1 | 1.29 | 1.69 | 1.99 | 1.08 |
| 23 | D.SARATHY | 24.08.06 | 8 | M | 18 | 1.2 | 12.5 | 600.4 | | | | |
| 24 | harish | 17.02.07 | 7 | M | 23 | 1.32 | 13.2 | 1208.3 | 1.47 | 1.99 | 1.89 | 0.26 |
| 25 | S.SANJAY | 20.02.07 | 7 | M | 21 | 1.17 | 15.3 | 982.3 | 1.33 | 4.12 | 1.72 | 0.13 |
| 26 | P.SANTHOSH | 08.03.07 | 7 | M | 21 | 1.22 | 14.1 | 444.3 | | | | |
| 27 | M.ABIRAMI | 24.02.07 | 7 | F | 20 | 1.14 | 15.4 | 878.1 | 1.29 | 2.3 | 2.02 | 0.85 |
| 28 | P.RUBINI | 06.06.07 | 7 | F | 20 | 1.14 | 15.4 | 600.4 | | | | |
| 29 | S.SWAPNA | 06.07.05 | 9 | F | 26 | 1.28 | 15.8 | 982.3 | 1.21 | 1.76 | 4.38 | 0.25 |
| 30 | R.MALINI | 24.08.06 | 8 | F | 22 | 1.27 | 13.6 | 878.1 | 1.55 | 1.87 | 1.75 | 1.13 |
| 31 | C.S.ASHMITHA | 06.04.07 | 7 | F | 24 | 1.21 | 16.4 | 779.7 | 1.54 | 3.05 | 4.11 | 0.3 |
| 32 | S.SUBATHRA | 13.02.07 | 7 | F | 20 | 1.23 | 13.2 | 519.4 | 1.19 | 1.47 | 6.87 | 12 |
| 33 | S.KEERTHANA | 16.06.07 | 7 | F | 20 | 1.16 | 14.9 | 687.1 | 1.74 | 1.79 | 1.37 | 0.21 |
| 34 | S.SUJI | 10.04.07 | 7 | F | 20 | 1.24 | 13 | 687.1 | | | | |
| 35 | A.MONISHA | 20.06.07 | 7 | F | 18 | 1.21 | 12.3 | 878.1 | 1.3 | 2.06 | 3.44 | 0.3 |
| 36 | P.NALINI | 20.06.05 | 9 | F | 20 | 1.14 | 15.4 | 982.3 | 1.4 | 4.99 | 2.19 | 0.29 |
| 37 | A.PRAVEENA | 20.06.06 | 8 | F | 25 | 1.24 | 16.3 | 600.4 | | | | |
| 38 | L.SAMUEL | 11.07.06 | 8 | M | 15 | 1.19 | 10.6 | 982.3 | 1.44 | 3.92 | 1.21 | 0.15 |
| 39 | T.ABISHEK | 29.03.06 | 8 | M | 18 | 1.2 | 12.5 | 600.4 | | | | |
| 40 | R.VIGNESH | 31.03.06 | 8 | M | 13 | 1.29 | 7.8 | 519.4 | 1.14 | 2.06 | 2.55 | 0.89 |
| 41 | Y.VASANTH | 7.02.06 | 8 | M | 25 | 1.39 | 12.9 | 519.4 | 1.41 | 3.07 | 1.41 | 0.49 |
| 42 | A.YASWANTH | 14.10.05 | 8 | M | 22 | 1.29 | 13.2 | 519.4 | 1.4 | 3.6 | 4.24 | 0.63 |
| 43 | M.MANO | 13.04.06 | 8 | M | 17 | 1.27 | 10.5 | 779.7 | 1.56 | 3.03 | 3.61 | 1.68 |
| 44 | S.BARATH | 19.06.06 | 8 | M | 27 | 1.33 | 15.3 | 687.1 | 1.5 | 0.973 | 2.12 | 2.59 |
| 45 | A.DHANALAKSHMI | 16.11.04 | 7 | F | 23 | 1.28 | 14 | 878.1 | 1.36 | 1.55 | 1.63 | 0.34 |
| 46 | S.DARSINI | 17.03.05 | 9 | F | 25 | 1.32 | 14.3 | 878.1 | 1.24 | 0.914 | 15.51 | 0.73 |
| 47 | P.SUSEELA | 1.10.05 | 8 | F | 16 | 1.23 | 10.6 | 311.6 | 1.38 | 2.36 | 5.22 | 16.01 |
| 48 | D.VIJAYALAKSHMI | 13.04.05 | 9 | F | 27 | 1.36 | 14.6 | 519.4 | 1.48 | 1.62 | 1.8 | 0.27 |
| 49 | CHRISTOPHER | 22.09.06 | 8 | M | 48 | 1.4 | 24.5 | 878.1 | 1.22 | 3.59 | 2.47 | 0.6 |
| 50 | K.SUGUNA | 11.11.05 | 8 | F | 19 | 1.23 | 12.6 | 982.3 | 1.31 | 2.1 | 2.29 | 0.14 |
| 51 | K.UDHAYAKUMAR | | 8 | M | | | | 156.1 | | | | |
| 52 | S.SAKTHIVEL | | 8 | M | | | | 982.3 | | | | |
| 53 | K.SANJAYRAMAN | | 8 | M | | | | 989 | | | | |
| 54 | M.SARAVANAN | | 8 | M | | | | 878.1 | | | | |
| 55 | M.DHANASEKAR | | 8 | M | | | | 1208 | | | | |
| 56 | M.DINESHKUMAR | | 8 | M | | | | 989 | | | | |
| 57 | M.LOKESH | | 8 | M | | | | 982.3 | | | | |
| 58 | R.VIMALADHITHAN | | 8 | M | | | | 989 | | | | |
| 59 | J.K.VENKATESH | | 8 | M | | | | 878.1 | | | | |
| 60 | AFRA THASLIM | | 9 | F | | | | 878.1 | | | | |
| 61 | P.BHARATHY | | 8 | F | | | | 878.1 | | | | |
| 62 | A.LOSHINI | | 8 | F | | | | 878.1 | | | | |
| 63 | N.BARATHRAJ | | 8 | M | | | | 982.3 | | | | |
| 64 | M.ASHOK | | 10 | M | | | | 982.3 | | | | |
| 65 | R.UDHAYAKUMAR | | 9 | M | | | | 989 | | | | |
| 66 | M.MANIKANDAN | | 10 | M | | | | 878.1 | | | | |
| 67 | D.PRAVEEN FRANCIS | | 9 | M | | | | 878.1 | | | | |
| 68 | J.SHETRAJ | | 8 | M | | | | 1208 | | | | |
| 69 | S.YUVARAJ | | 9 | M | | | | 53.1 | | | | |
| 70 | ASARUDEEN | | 8 | M | | | | 1012 | | | | |
| 71 | A.GAYATHRI | | 9 | F | | | | 1012 | | | | |
| 72 | V.SHANTHALAKSHMI | | 8 | F | | | | 2.5 | | | | |
| 73 | D.DIVYA | | 10 | F | | | | 989 | | | | |
| 74 | M.DEEPASHREE | | 9 | F | | | | 878.1 | | | | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 75 | K.BANUPRIYA | | 8 | F | | | | 779.7 | | | | |
| 76 | B.MALINI | | 9 | F | | | | 989 | | | | |
| 77 | A.VELANKANNI | | 9 | F | | | | 878.1 | | | | |
| 78 | P.RAKSHANA | | 9 | F | | | | 982.3 | | | | |
| 79 | S.FATHIMA | | 9 | F | | | | 989 | | | | |
| 80 | A.ANBUSELVI | | 7 | F | | | | 779.7 | | | | |
| 81 | B.SWETHA | | 7 | F | | | | 878.1 | | | | |
| 82 | N.RAJALAKSHMI | | 9 | F | | | | 1208 | | | | |
| 83 | M.MUTHARASU | | 8 | F | | | | 982.3 | | | | |
| 84 | A.IMANRAJ | | 7 | M | | | | 982.3 | | | | |
| 85 | M.KEERTHANA | | 10 | F | | | | 687.1 | | | | |
| 86 | S.DIVYA | | 10 | F | | | | 779.7 | | | | |
| 87 | T.MONISHA | | 10 | F | | | | 982.3 | | | | |
| 88 | K.LAVANYA | | 11 | F | | | | 982.3 | | | | |
| 89 | S.MEENATCHI | | 11 | F | | | | 779.7 | | | | |
| 90 | B.SWATHI | | 11 | F | | | | 687.1 | | | | |
| 91 | R.NAVEENKUMAR | | 11 | M | | | | 779.7 | | | | |
| 92 | S.GOPINATH | | 11 | M | | | | 982.3 | | | | |
| 93 | R.SURYAPRAKASH | | 10 | M | | | | 878.1 | | | | |
| 95 | D.DHAYABABU | | 11 | M | | | | 1012 | | | | |
| 96 | S.HARISH | | 10 | M | | | | 1012 | | | | |
| 97 | S.HARISHKUMAR | | 10 | M | | | | 982.3 | | | | |
| 98 | M.JEEVA | | 10 | M | | | | 998 | | | | |
| 99 | A.MANIKANDAN | | 10 | M | | | | 1012 | | | | |
| 100 | R.DINESH | | 10 | M | | | | 1012 | | | | |
| 101 | A.RAKESH | | 11 | M | | | | 1012 | | | | |
| 102 | K.LOKESH | | 10 | M | | | | 982.3 | | | | |
| 94 | M.SANJAY | | 11 | M | | | | 1590.9 | | | | |

# MASTER CHART
## Patients

| sl.no. | name | age | sex | endo.no | weight | height | BMI | goitre | TSH | T4 | FNAC | USG | TPO ab | TGA ab | U.iodine | AIT or not | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Madani | 6 | F | 88/12 | 22 | 1.12 | 17.54 | 0 | >25 | 24 | nd | N | 74 | >1000 | 180 | 1 | OVERT |
| 2 | aswini | 11 | F | 61/13 | 24 | 1.25 | 15.40 | 1 | 18.05 | 11.1 | colloid | thyromegaly | 579 | >4000 | 444.3 | 1 | OVERT |
| 3 | revathy | 11 | F | 63/13 | 27 | 1.26 | 17.00 | 1 | 11.9 | 150 | colloid | thyromegaly | 25 | 104 | 253.9 | 0 | SUBCLINICAL |
| 4 | divya | 10 | F | 37/13 | 25 | 1.22 | 16.80 | 2 | 7.2 | 84 | colloid | thyroiditis | 22 | 34 | 202.1 | 0 | SUBCLINICAL |
| 5 | shalini | 12 | F | 1172/13 | 16.8 | 1.16 | 15.90 | 2 | >25 | 68 | colloid | thyromegaly | 215 | 567 | 375 | 1 | SUBCLINICAL |
| 6 | dinesh | 3 | M | 597/13 | 10 | 0.91 | 12.10 | 2 | 6.27 | 11 | colloid | thyromegaly | 28 | 45 | 311.6 | 0 | OVERT |
| 7 | sameena | 9 | F | 612/13 | 26 | 1.27 | 16.00 | 2 | 19.94 | 5.26 | thyroiditis | thyroiditis | >100 | >1000 | 202.1 | 1 | SUBCLINICAL |
| 8 | udayakumar | 3 | M | 125/12 | 11 | 0.9 | 13.60 | 2 | 12.4 | 67 | colloid | thyromegaly | 153.6 | 16.8 | 600.4 | 1 | SUBCLINICAL |
| 9 | mohana | 8 | F | 439/13 | 24 | 1.16 | 17.8 | 1 | >25 | 58 | thyroiditis | thyromegaly | 1000 | 185.73 | 878.1 | 1 | SUBCLINICAL |
| 10 | Gowri | 10 | F | 547/13 | 24 | 1.25 | 15.40 | 2 | 28.1 | 5.03 | thyroiditis | thyromegaly | 4.98 | 64 | 253.9 | 1 | SUBCLINICAL |
| 11 | preetha | 11 | F | 557/13 | 26 | 1.28 | 15.80 | 1 | 12 | 6.2 | colloid | thyromegaly | 23.6 | 22.26 | 375 | 0 | SUBCLINICAL |
| 12 | lekhasree | 11 | F | 232/13 | 25 | 1.27 | 15.50 | 2 | 16 | 67 | colloid | thyroiditis | 0.72 | 1.21 | 375 | 0 | SUBCLINICAL |
| 13 | divya | 11 | F | 628/13 | 27 | 1.26 | 17.00 | 2 | 149 | 0.51 | colloid | thyroiditis | 124 | 325 | 375 | 1 | SUBCLINICAL |
| 14 | lokesh | 9 | M | 636/13 | 27 | 1.26 | 17.00 | 2 | 15.33 | 34 | colloid | thyromegaly | 456 | 700 | 444.3 | 1 | OVERT |
| 15 | jenifer | 10 | F | 648/13 | 27 | 1.26 | 17.00 | 2 | 7.2 | 56 | colloid | thyromegaly | 16 | 58 | 311.6 | 0 | SUBCLINICAL |
| 16 | anbarassi | 5 | F | 650/13 | 18 | 1.08 | 15.40 | 2 | >25 | 46 | thyroiditis | thyroiditis | >1000 | 34.19 | 375 | 1 | OVERT |
| 17 | rekha | 8 | F | 652/13 | 22 | 1.24 | 14.3 | 2 | 8.3 | 86 | thyroiditis | thyroiditis | 548.27 | 711.92 | 311.6 | 1 | SUBCLINICAL |
| 18 | abirami | 10 | F | 123/13 | 26 | 1.27 | 16.00 | 2 | 6.2 | 76 | colloid | thyomegaly | 163 | 320 | 444.3 | 1 | SUBCLINICAL |
| 19 | harini | 10 | F | 726/13 | 27 | 1.26 | 17.00 | 2 | 9.4 | 10.3 | colloid | thyromegaly | 28 | 15 | 116 | 0 | OVERT |
| 20 | ajay | 11 | M | 423/13 | 21 | 1.32 | 12.42 | 2 | 6.8 | 65 | thyroiditis | thyomegaly | 23 | 329 | 311.6 | 1 | SUBCLINICAL |
| 21 | sabana | 10 | F | 759/13 | 24 | 1.25 | 15.40 | 2 | 8.1 | 7.6 | thyroiditis | thyroiditis | >1000 | 52.34 | 375 | 1 | SUBCLINICAL |
| 22 | hariharan | 10 | M | 241/13 | 27 | 1.26 | 17.00 | 2 | 7 | 35 | colloid | N | 31.5 | <15 | 253.9 | 1 | OVERT |
| 23 | naveen kumar | 8 | M | 2451/13 | 23 | 1.22 | 15.5 | 1 | 8.9 | 92 | colloid | thyromegaly | 303.2 | 162 | 202.1 | 1 | SUBCLINICAL |
| 24 | gajendran | 7 | M | 696/12 | 24 | 1.18 | 17.23 | 2 | 7.17 | 0.7 | colloid | thyroiditis | 1.02 | 0.66 | 53 | 0 | SUBCLINICAL |
| 25 | ammu | 11 | F | 797/13 | 24 | 1.16 | 17.80 | 2 | 12.3 | 7.78 | thyroiditis | thyroiditis | 98 | 105 | 156.1 | 1 | OVERT |
| 26 | harini | 9 | F | 829/13 | 24 | 1.25 | 15.40 | 2 | 12.6 | 58 | thyroiditis | thyromegaly | 52 | 48 | 1590.9 | 1 | SUBCLINICAL |
| 27 | abirami | 11 | F | 13/14 | 27 | 1.25 | 17.30 | 2 | 6.8 | 94 | colloid | thyroiditis | <3 | 1.53 | 116 | 0 | SUBCLINICAL |
| 28 | aswini | 11 | F | 579/13 | 32 | 1.28 | 19.50 | 2 | >25 | 84 | thyroiditis | thyroiditis | 170 | 402 | 375.4 | 1 | SUBCLINICAL |
| 29 | nishanthi | 12 | F | 56/14 | 24 | 1.16 | 17.80 | 2 | 6.4 | 102 | colloid | thyroiditis | 0.24 | 1.58 | 311.6 | 0 | SUBCLINICAL |
| 30 | jeyamani | 11 | F | 821/13 | 23 | 1.21 | 15.70 | 2 | 8 | 5.33 | colloid | thyromegaly | 329.64 | 192.3 | 444.3 | 1 | OVERT |
| 31 | yamini | 10 | F | 42/14 | 24 | 1.25 | 15.40 | 2 | 7 | 42 | colloid | thyomegaly | 24 | 42 | 116 | 0 | OVERT |
| 32 | sneha | 12 | F | 117/14 | 23 | 1.21 | 15.70 | 2 | 23 | 1.62 | colloid | thyromegaly | 21 | 55 | 779.3 | 0 | SUBCLINICAL |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 33 | riswana | 10 | F | 148/14 | 25 | 1.27 | 15.50 | 2 | 6.5 | 11.5 | colloid | thyromegaly | 24 | 46 | 687.1 | 0 | OVERT |
| 34 | indra | 11 | F | 154/14 | 28 | 1.31 | 16.30 | 2 | 27.87 | 5.5 | thyroiditis | thyroiditis | 540 | 242 | 311.6 | 1 | OVERT |
| 35 | nivedha | 9 | F | 155/14 | 25 | 1.27 | 15.50 | 2 | 33.7 | 44.4 | thyroiditis | thyroiditis | 343 | 156 | 600.4 | 1 | OVERT |
| 36 | lokeshwaran | 7 | M | 186/14 | 20 | 1.23 | 13.2 | 2 | 10.2 | 54 | colloid | thyroiditis | >1000 | 322 | 600.4 | 1 | SUBCLINICAL |
| 37 | karima | 12 | F | 487/14 | 28 | 1.31 | 16.30 | 2 | 84.64 | 17.7 | colloid | thyroiditis | 4.55 | 136.58 | 253.9 | 1 | OVERT |
| 38 | thulasi | 11 | F | 206/14 | 16.8 | 1.16 | 15.90 | 2 | 42 | 22 | colloid | thyromegaly | 592 | 423 | 375 | 1 | OVERT |
| 39 | gayathri | 11 | F | 215/14 | 24 | 1.16 | 17.80 | 2 | 23.23 | 0.53 | thyroiditis | thyroiditis | 577.97 | 156.58 | 202.1 | 1 | SUBCLINICAL |
| 40 | vinodhini | 8 | F | 213/14 | 20 | 1.3 | 11.80 | 1 | 7.3 | 6.95 | colloid | thyroiditis | >1000 | 8.24 | 253.9 | 1 | OVERT |
| 41 | mohanasri | 5 | F | 259/14 | 20 | 1.09 | 16.80 | 2 | 6.5 | 64 | colloid | thyromegaly | 750.45 | 72.2 | 687.1 | 1 | SUBCLINICAL |
| 42 | monisha | 11 | F | 277/13 | 43 | 1.55 | 17.90 | 2 | 8.3 | 56 | thyroiditis | thyroiditis | 289 | 4.15 | 600.4 | 1 | SUBCLINICAL |
| 43 | uthra | 10 | F | 288/13 | 29 | 1.42 | 14.40 | 2 | 15 | 3 | thyroiditis | thyroiditis | 1300 | | 1208 | 1 | OVERT |
| 44 | akila | 11 | F | 305/14 | 24 | 1.22 | 16.12 | 2 | 14 | 96.5 | colloid | thyroiditis | >1000 | >1000 | 375 | 1 | SUBCLINICAL |
| 45 | riya | 9 | F | 389/14 | 30 | 1.32 | 17.20 | 2 | 7 | 53 | colloid | thyroiditis | 32 | 39 | 116 | 0 | SUBCLINICAL |
| 46 | nagamani | 11 | F | 401/14 | 36 | 1.45 | 17.20 | 2 | 7.26 | 65 | colloid | thyromegaly | 6.6 | 4.75 | 779.7 | 0 | SUBCLINICAL |
| 47 | gokulraj | 11 | M | 498/14 | 21 | 1.32 | 12.05 | 2 | 9.7 | 45 | colloid | thyromegaly | 592 | 340 | 444.3 | 1 | SUBCLINICAL |
| 48 | ajithkumar | 11 | M | 504/13 | 23 | 1.21 | 15.70 | 2 | 843 | 0.65 | thyroiditis | thyroiditis | 747.16 | 42.87 | 519.4 | 1 | SUBCLINICAL |
| 49 | vani | 11 | F | 530/14 | 28 | 1.31 | 16.30 | 2 | 7.73 | 0.84 | colloid | thyroiditis | >1000 | 27.8 | 253.9 | 1 | SUBCLINICAL |
| 50 | bavani | 11 | F | 547/14 | 35 | 1.31 | 20.40 | 0 | 13.71 | 5 | colloid | thyromegaly | 18 | 42 | 202.1 | 0 | OVERT |
| 51 | yuvasree | 11 | F | 113/13 | 28 | 1.31 | 16.30 | 2 | 6.5 | 96 | colloid | thyromegaly | 709.85 | 133.41 | 444.3 | 1 | SUBCLINICAL |
| 52 | jeyasakthi | 12 | F | 25/13 | 45 | 1.5 | 20.00 | 2 | >25 | 1 | colloid | thyromegaly | >1000 | 762 | 687.1 | 1 | SUBCLINICAL |
| 53 | mithra | 8 | F | 334/13 | 23 | 1.28 | 14 | 2 | >25 | 49 | colloid | thyomegaly | 215 | 158 | 375 | 1 | SUBCLINICAL |
| 54 | chandra | 9 | F | 806/13 | 25 | 1.27 | 15.50 | 2 | 12 | 56 | thyroiditis | thyromegaly | 143 | 260 | 687.1 | 1 | SUBCLINICAL |
| 55 | nisha | 4 | F | 429/13 | 13 | 0.96 | 14.1 | 2 | 19 | 5.4 | thyroiditis | thyromegaly | 201 | 253 | 600.4 | 1 | OVERT |
| 56 | kaviyarasu | 8 | M | 412/13 | 26 | 1.22 | 17.4 | 2 | 13.17 | 0.91 | colloid | thyromegaly | 0.18 | 0.93 | 211 | 0 | SUBCLINICAL |
| 57 | issac | 10 | M | 704/13 | 25 | 1.28 | 15.26 | 2 | 116.5 | 4.3 | thyroiditis | thyroiditis | 2700 | 450 | 878.1 | 1 | OVERT |
| 58 | priyadarsini | 7 | F | 286/11 | 25 | 1.33 | 14.1 | 2 | >25 | 67 | thyroiditis | thyromegaly | 305 | 495 | 982.3 | 1 | SUBCLINICAL |
| 59 | lokesh | 6 | M | 73/12 | 21 | 1.11 | 17.00 | 1 | 7.3 | 49 | thyroiditis | thyomegaly | 45.89 | 102.15 | 600.4 | 1 | SUBCLINICAL |
| 60 | amul | 12 | F | 504/12 | 25 | 1.24 | 16.26 | 2 | >25 | 94 | colloid | thyromegaly | 2.1 | 4.3 | 211 | 0 | SUBCLINICAL |
| 61 | kavya | 9 | F | 677/12 | 24 | 1.28 | 14.60 | 2 | 18 | 16 | thyroiditis | thyromegaly | >1000 | 287 | 779.7 | 1 | OVERT |