

# Grave`s Disease

## TIG

### **Disorder of Thyroid Hormone:**

*Due to Hypo function of Thyroid Hormone*

### **(A) Cretinism:**

This disease is caused by the hypo secretion of thyroid hormone in childhood. Hypo secretion of pituitary TSH may cause hypo activity of thyroid gland. Congenital lacks of thyroid gland also help in development of cretinism.

### **characteristic features are as follows:**

#### **(i) Morphological features:**

1. Linear growth is severely retarded resulting dwarfism.
2. Shunted skeletal growth.
3. Short club like fingers.
4. Rough, dry, wrinkled and scaly skin.
5. Scanty growth of hairs.
6. Bloated facial appearance.
7. Idiotic look.
8. Thick and enlarged tongue (macroglossia).
9. Excess saliva secretion.
10. Poor muscle growth.
11. Pot-bellied abdomen.
12. Teeth are deformed.

13. Reduced sexual growth.

**(ii) Physiological features:**

1. Decreased heart rate.
2. Low body temperature.
3. Low BMR.
4. Fat is irregularly distributed.
5. Low blood sugar level.
6. Reflexes are sluggish.
7. Immune mechanism is reduced.
8. Low RBC count.

**(iii) Psychological features:**

1. Cretins are frequently deaf and dumb.
2. The child is subnormal in intelligence.
3. Have various degrees of mental retardation and idiocy.

**(iv) Sexological features:**

1. Retarded growth of sex organs and sex glands.
2. Secondary sex characters are underdeveloped.

**(B) Myxoedema (Gull's disease):**

This disease is caused by hypo activity of thyroid gland in adult. It is also caused by the hypo secretion of pituitary TSH.

**It is characterized by following features.**

**(i) Morphological features:**

1. Skin colour becomes yellow due to accumulation of mucinous protein deposit in the cutaneous tissue.
2. Skin shows appearance of puffy swellings.
3. Puffy face.
4. Swelling in neck region.
5. Irregular fat deposition, specially in clavicular, and buttock region.
6. Loss of hair from eyebrows, pubis, axillae.
7. Tongue and larynx are thickened.

**(ii) Physiological features:**

1. Low BMR.
2. Low blood sugar level.
3. Reduced heart rate and cardiac output.
4. Low blood pressure.
5. Nitrogen excretion is reduced.
6. Less appetite.
7. Less peristaltic activity in GI tract.
8. Cholesterol level becomes high.
9. Slow muscle contractibility.

**(iii) Psychological features:**

1. The patient is lethargic and sleepy.
2. Dull in appearance.

3. Suffers from loss of memory.

4. Reflexes are very slow.

**(iv) Sexological features:**

1. Sexual functions are diminished.

2. Primary sex organs and sex glands are degenerated.

3. Loss of libido effect.

4. Failure of fertility power.

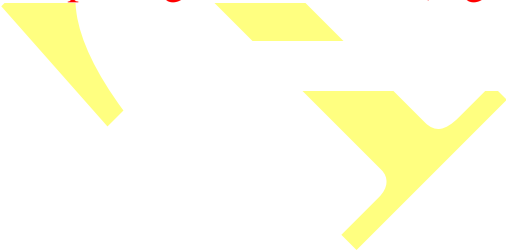
***Due to Hyper Secretion of Thyroid Hormone:***

**Grave's disease (Exophthalmic goiter):**

This disease is caused by hyper secretion of thyroid hormone in both infant and adult life. Hyper secretion of pituitary TSH or thyrotoxicosis may help in development of hyper activity of thyroid gland. This disease may be mediated by an immunoglobulin known as LATS (Long acting thyroid stimulator). LATS binds to TSH receptors and activates thyroid cell functions even in absence of circulating TSH.

**It shows following features:**

**(i) Morphological features: (Fig. 12.9)**





**Fig. 12.9:** Exophthalmic goiter  
(Grave's disease)

1. Enlarged thyroid gland.
2. Decreased body weight.
3. Skin becomes soft and sweaty.
4. Loss of fat depot.
5. Hands are usually warm and moist.
6. Hairs are fine and friable.
7. Muscles become easily fatigued.
8. Large protruded eye ball i.e. exophthalmic eye.
9. Retraction of the upper eye lid.

**(ii) Physiological features:**

1. BMR is greatly increased.
2. Osteoporosis takes place due to loss of calcium and phosphorus.
3. Shows hyperglycemia and glycosauria.

4. Protein catabolism is increased.
5. Increased blood pressure.
6. Increased cardiac output.
7. Greater loss of sodium and potassium through urine.
8. Requirement of vit. B complexes increased.
9. Increased appetite.
10. Loud heart sounds.

**(iii) Psychological features:**

1. Patient becomes nervous and emotional.
2. Person becomes restless and sleepless.

**(iv) Sexological features:**

1. Increase in libido effect.
2. Disturbances in menstrual cycle in woman.
3. The fertility power becomes reduced.

**Special note:**

**Goiter:**

It is the enlargement of thyroid gland.

**It can be divided into following types:**

**(A) Simple goiter:**

It is caused by the deficiency of iodine in the body.

**It is again subdivided into three types:**

**(i) Colloid goiter:**

This type is prominent in areas where the drinking water is deficient in iodine. In order to produce normal levels of hormone, the gland becomes

hypertrophied. The follicles are distended with colloid. Intake of iodinated salt is the preventive measure (Fig. 12.10).



**(ii) Diffuse parenchymatous goiter:**

The follicular epithelial cells show hypertrophy and multiply in number due to which lumen of the follicle become obliterated.

**(iii) Nodular goiter:**

This type is characterized by the appearance of nodular swelling in some part of the gland. It is caused by distinct much elevation of  $T_3$  with less elevation of  $T_4$  level.

**(B) Toxic goiter:**

When swelling or enlargement of the thyroid gland results in excess secretion of thyroid hormones, this condition is called toxic goiter (Fig. 12.11).



**Fig. 12.11:** Massive thyroid enlargement due to toxic goiter

The main cause for this goiter is the ingestion of chemicals, like anti-thyroid drugs, like methylthiouracil, propylthiouracil and carbimazole. The presence of goitrogenic substances in cabbage, turnip etc. which contain a chemical substance goitrin. With these effects follicular cells become hypertrophied and hyperplastic. This leads to hyper secretion.

### **(C) Exophthalmic goiter:**

Due to hyper secretion of thyroid hormone, thyroid gland becomes enlarged. As a result there is a protrusion of the eyeball called exophthalmic goiter due to swelling of the extra ocular muscles and connective tissue within the bony wall of the orbit.

### **Table 12.2: Difference between Hypo and Hyperthyroidism**

#### **Hypothyroidism:**

1.  $T_3$  and  $T_4$  levels are decreased
2. BMR becomes low



3. Perspiration becomes decreased
4. Impaired intestinal glucose absorption
5. Low blood cholesterol
6. Decreased protein anabolism
7. Weight gain
8. Decreased appetite
9. Low body temperature
10. Cold intolerance
11. Dry palm
12. Sleepiness, tiredness
13. Goiter may or may not be present
14. Low pulse rate and less cardiac output

**Hyperthyroidism:**

1.  $T_3$  and  $T_4$  levels are increased.
2. BMR becomes high.
3. Perspiration becomes increased.
4. Enhanced intestinal glucose absorption.
5. High blood cholesterol.
6. Increased protein anabolism.
7. Weight loss.
8. Increased appetite.

9. High body temperature.
10. Heat intolerance.
11. Moist palm.
12. Restlessness, insomnia.
13. Goiter present.
14. High pulse rate and increased cardiac output.

**Table 12.2: Difference between Hypo and Hyperthyroidism**

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6. Decreased protein anabolism	6. Increased protein anabolism.
7. Weight gain	7. Weight loss.
8. Decreased appetite	8. Increased appetite.
9. Low body temperature	9. High body temperature.
10. Cold intolerance	10. Heat intolerance.
11. Dry palm	11. Moist palm.
12. Sleepiness, tiredness	12. Restlessness, insomnia.
13. Goiter may or may not be present	13. Goiter present.
14. Low pulse rate and less cardiac output	14. High pulse rate and increased cardiac output.

**(D) Hokkaido goiter:**

During the synthesis of thyroid hormones, inorganic iron is taken up from the blood and converted into organic form by reacting with the amino acid tyrosine. High concentration of iodine inhibits organification of iodine and causes hypothyroidism.

### **Cause of Hypothyroidism:**

Decreased secretion of thyroid hormones may cause hypothyroidism.

### **The causes are given below:**

#### **Nature of Hypothyroidism Causes:**

##### **(a) Primary:**

- i. Iodine deficiency in the food and drinking water.
- ii. Defective synthesis of the thyroglobulin and secretion can lead to hypothyroidism.
- iii. Genetic failure in the development of the thyroid hormone receptors.
- iv. Inborn error in thyroid hormone biosynthesis.
- v. Radioactive iodine therapy may cause hypothyroidism.
- vi. The intake of goitrogenic substances.
- vii. Failure of the conversion of  $T_3$  and  $T_4$  in peripheral tissue.

##### **(b) Secondary:**

- i. Due to pituitary adenoma, pituitary ablation or pituitary destruction.
- ii. Pituitary TSH deficiency.
- iii. Defect in TSH receptor of thyroid gland.
- iv. Defect in secretion of TSH-RH of hypothalamus.
- v. Damage of Hypothalamohypophysial system.

Nature of Hypothyroidism	Causes
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(b) Secondary	<ul style="list-style-type: none"> <li>(i) Due to pituitary adenoma, pituitary ablation or pituitary destruction.</li> <li>(ii) Pituitary TSH deficiency.</li> <li>(iii) Defect in TSH receptor of thyroid gland.</li> <li>(iv) Defect in secretion of TSH-RH of hypothalamus.</li> <li>(v) Damage of Hypothalamo-hypophysial system.</li> </ul>

### Causes of hyperthyroidism:

Excess secretion of thyroid hormones may cause hyperthyroidism.

### It is caused by following ways:

- (i) Graves' disease is an autoimmune disease in which circulating antibodies formed against TSH receptor. The antibodies are TSH-R-Ab. It may bind to TSH receptors on thyroid follicle membrane and thus stimulating the secretion of  $T_3$  and  $T_4$ .
- (ii) Lesions on thyroid gland may cause of hyper secretion of  $T_3$  and  $T_4$  even under low TSH.

### Thyrotoxicosis:

It results when tissues are exposed to over-secretion of thyroid hormones. Graves' disease and Plummer's disease are good examples.

### Thyroiditis:

It is the inflammatory disorder of thyroid gland.

### Hashimoto's disease:

Hashimoto (1912) observed this pathophysiology of thyroid gland. It is caused due to damage of thyroid gland tissue and hypo function. The clinical features are—exaggeration of normal lobular pattern, destruction of epithelial cells, degeneration of follicular basement membrane, infiltration of lymphocytes etc. The diagnosis of this disease is

confirmed by the finding of high titers of thyroid autoantibodies in the serum (Fig. 12.12).

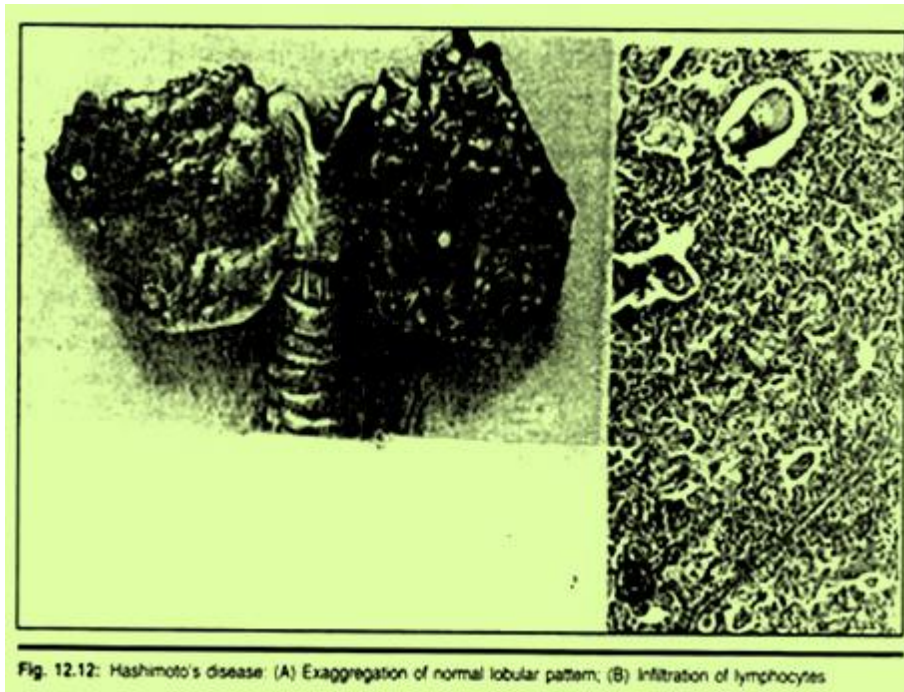


Fig. 12.12: Hashimoto's disease: (A) Exaggeration of normal lobular pattern; (B) Infiltration of lymphocytes

