

DISEASES OF THE THYROID GLAND

HYPERTHYROIDISM

- Over activity of the thyroid gland.
- Women : men ratio (8:1).
- activity of gland :
 - a)- 5- 10 times increase in secretion.
 - b)- 2-3 times increase in size.

CAUSES

1- Graves' disease:

- an autoimmune disorder.
- increased circulating level of thyroid- stimulating immunoglobulins (TSI).
- 95%.
- 4 – 8 times more common in women than men.

2- Thyroid gland tumor:

- 95% is benign.
- 5% is malignant.
- history of head and neck irradiation and family history.

3- Exogenous T3 and T4:

(rarely cause)

4- Excess TSH secretion:

- diseases of the hypothalamus (TRH).
- diseases of the pituitary (TSH).

DIAGNOSIS

■ Symptoms:

1- Goiter in 95%.

2- skin:

- smooth, warm and moist.
- heat intolerance, night sweating.
- patchy pigmentation.

3- musculo skeletal:

- Muscle atrophy.

4- Neurological:

- tremor.
- enhanced reflexes.
- irritability.

5- Cardiovascular:

- increase heart rate.
- increase stroke volume.
- arrhythmias.
- hypertension.

6- G.I tract:

- weight loss.
- diarrhea.

7- Renal function:

- ↑ glomerular filtration rate.

8- Exophthalmos:

- anxious staring expression.
- protrusion of eye balls.

9- Others:

- menstrual cycle disturbance.



INVESTIGATIONS

1- Serum T3, T4 measurement.

In primary hyperthyroidism:

high T3, T4 and low TSH .

In secondary hyperthyroidism:

high T3, T4 and high TSH.

TREATMENT

1- Medical therapy:

e.g. propylthiourcal.

- usually for 12-18 months course.

- with 3-4 monthly monitoring.

2- Surgery:

- Subtotal thyroidectomy.

- Indication for surgery:

a)- Relapse after medical treatment.

b)- Drug intolerance.

c)- Cosmetic.

d)- Suspected malignancy.

HYPOTHYROIDISM

Under activity of the thyroid gland more in woman (30- 60 years).

CAUSES

1- inherited abnormalities of thyroid hormone synthesis :

- peroxidase defect.
- Iodide trapping defect.
- thyroglobulin defect.

2- Endemic Colloid Goiter:

- before table salt.
↓ iodide → ↓ hormone formation → ↑ TSH → ↑ Thyroglobulin
→ ↑size (>10 times)



3- Idiopathic Nontoxic Colloid Goiter:

- I⁻ intake is normal.
- thyroiditis?

inflammation → ↑ cell damage → ↓ hormone secretion
→ ↑ TSH → ↑ of activity of normal cells → ↑ size

4- Gland destruction (surgery).

5- Pituitary diseases or tumor.

6- Hypothalamus diseases or tumor.

DIAGNOSIS

1- skin and hair:

- dry skin.
- cold intolerance.

2- Musculo skeletal:

- muscle bulk.
- in skeletal growth.
- muscle sluggishness
- slow relaxation after contraction.

3- Neurological:

- slow movement.
- impaired memory.

4- Cardiovascular:

- blood volume.
- ↓ heart rate
- ↓ stroke volume.
- Arteriosclerosis.

5- G.I tract:

- constipation
- increase weight.

6- Renal function:

- decrease glomerular filtration rate.

7- Myxoedema:

An edematous appearance through out body.

8- others:

- loss of libido.
- menstrual cycle disturbance.



INVESTIGATIONS

1- Serum T3, T4 are low.

- TSH is elevated in primary.
- TSH is low in secondary hypothyroidism.

TREATMENT

- L- thyroxine
- Starting dose is 25-50 μg .
- Increase to 200 μg .
- At 2-4 weeks period.

The first response seen is the weight loss.

TABLE 9-9. Pathophysiology of Thyroid Hormones

	Hyperthyroidism	Hypothyroidism
Symptoms	Increased basal metabolic rate (BMR) Weight loss Negative nitrogen balance Increased heat production Sweating Increased cardiac output Dyspnea (shortness of breath) Tremor, muscle weakness Exophthalmos Goiter	Decreased basal metabolic rate Weight gain Positive nitrogen balance Decreased heat production Cold sensitivity Decreased cardiac output Hypoventilation Lethargy, mental slowness Drooping eyelids Myxedema Growth retardation Mental retardation (perinatal) Goiter
Causes	Graves' disease (increased thyroid-stimulating immunoglobulins) Thyroid neoplasm Excess TSH secretion Exogenous T ₃ or T ₄	Thyroiditis (autoimmune or Hashimoto's thyroiditis) Surgery for hyperthyroidism I ⁻ deficiency Congenital (cretinism) Decreased TRH or TSH
TSH levels	Decreased (feedback inhibition of T ₃ on the anterior lobe)	Increased (by negative feedback if primary defect is in thyroid gland) Decreased (if defect is in hypothalamus or anterior pituitary)
Treatment	Propylthiouracil (inhibits peroxidase enzyme and thyroid hormone synthesis) Thyroidectomy ¹³¹ I (destroys thyroid) β-Adrenergic blocking agents (adjunct therapy)	Thyroid hormone replacement therapy



CRETINISM

Extreme hypothyroidism during infancy and childhood (failure of growth).

CAUSES

- 1- Congenital lack of thyroid gland (congenital cretinism).
- 2- Genetic deficiency leading to failure to produce hormone.
- 3- Iodine lack in the diet (endemic cretinism).

SYMPTOMS

- 1- Infant is normal at birth but abnormality appears within weeks.
- 2- Protruding tongue.
- 3- Dwarf with short limbs.
- 4- Mental retardation.
- 5- Often umbilical hernia.
- 6- teeth.



TREATMENT

Changes are irreversible unless treatment is given early.

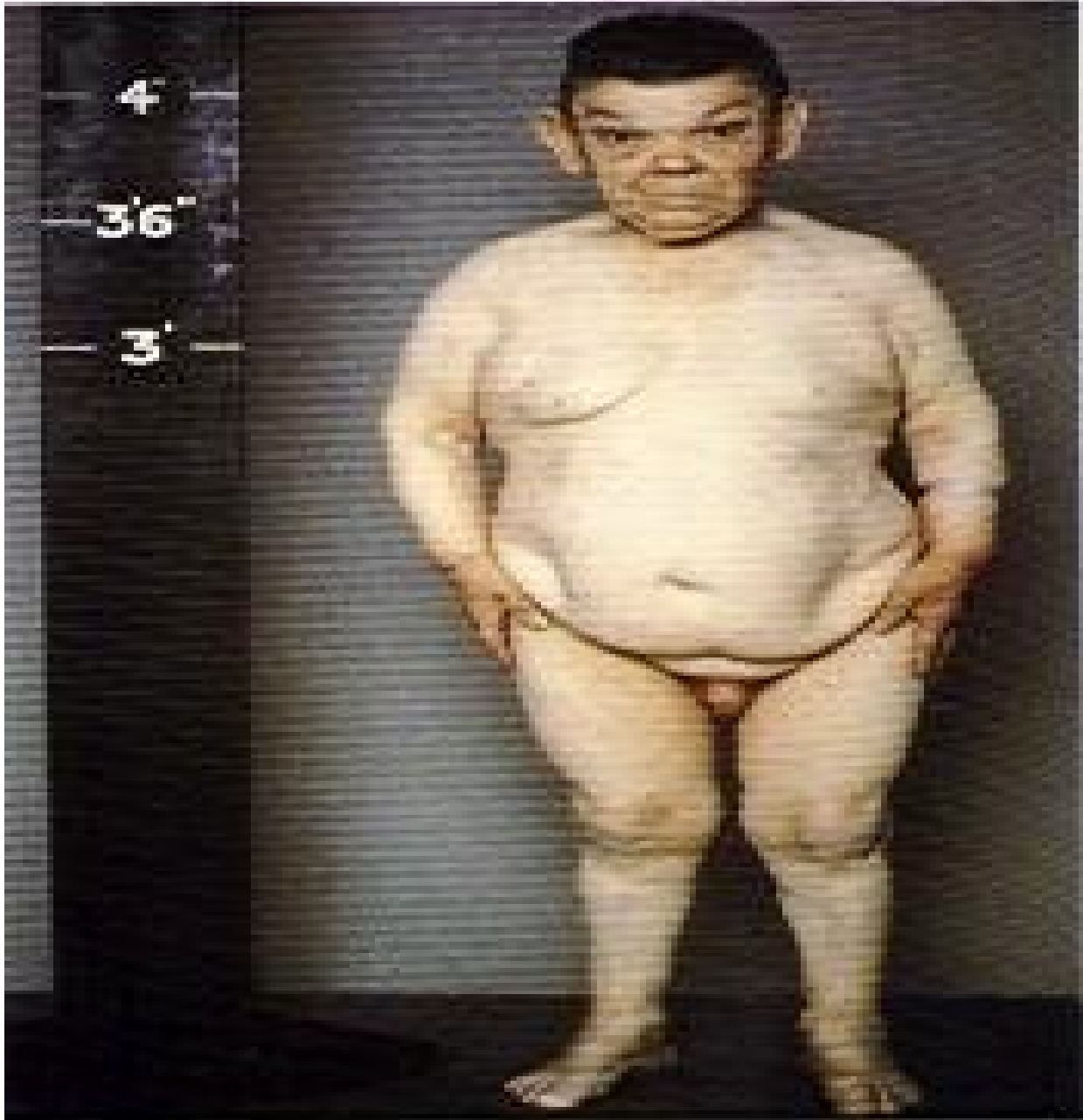


Table 20.19 Hormones in serum		
Hormone	Reference range	Units
Adrenocorticotrophic hormone (ACTH) (plasma)	7–51 (07:00–10:00 h)	ng/l
Cortisol	150–550 (at 08:00 h) < 200 (at 22:00 h)	nmol/l
Follicle-stimulating hormone (FSH)	1.5–9.0	U/l
Male	3.0–15 (early follicular)	U/l
Female*	Up to 20 (mid-cycle) > 30 (post-menopausal)	
Gastrin (plasma)	Up to 120	ng/l
Growth hormone (GH)	Very variable, usually less than 2, but may be up to 50 with stress	mU/l
Insulin	Highly variable and interpretable only in relation to plasma glucose and body habitus	mU/l
Luteinising hormone (LH)	2.5–9.0 (early follicular)	U/l
Female*	Up to 90 (mid-cycle) > 20 (post-menopausal)	
Male	1.5–9.0	U/l
Oestradiol-17β	110–180 (early follicular)	pmol/l
Female	550–1650 (mid-cycle) 370–770 (luteal)	
Male	< 150 (post-menopausal) < 200	pmol/l
Parathyroid hormone (PTH)	10–65	ng/l
Progesterone	< 2.0	nmol/l
Male	< 2.0 (follicular)	nmol/l
Female	> 15 (mid-luteal) < 2.0 (post-menopausal)	
Prolactin (PRL)	60–390	mU/l
Testosterone	10–30	nmol/l
Male	0.4–2.8	nmol/l
Female		
Thyroid-stimulating hormone (TSH)	0.15–3.5	mU/l
Thyroxine (free) (free T₄)	10–27	pmol/l
Tri-iodothyronine (T₃)	1.0–2.6	nmol/l
TSH receptor antibodies (TRAb)	< 7	U/l
* Luteal phase values similar to follicular phase.		
<i>Notes</i>		
1. A number of hormones are unstable, and collection details are critical to obtaining a meaningful result. Refer to local hospital handbook.		
2. Values in the table are only a guideline; hormone levels can often only be meaningfully understood in relation to factors such as sex (e.g. testosterone), age (e.g. FSH in women), time of day (e.g. cortisol) or regulatory factors (e.g. insulin and glucose, PTH and [Ca ²⁺]). Also, reference ranges may be critically method-dependent.		