Nursing Management of Patient with Disorders of Endocrine System

8
CHAPTER

Learning Objectives

After studying this chapter, the students will be able to:

- Explain anatomy and physiology of endocrine system.
- Describe techniques of assessment of endocrine glands.
- List the various investigations and tests done to evaluate endocrine disorders.
- Discuss disorders of thyroid gland. Hypothyroidism—its clinical manifestations, diagnostic tests and management.
- Explain hyperthyroidism—incidence, etiology, pathophysiology, clinical manifestations, diagnostic tests.
- Write management of hyperthyroidism—medical, surgical and nursing management.
- Describe disorders parathyroid gland—hyperparathyroidism—its clinical manifestation, diagnostic evaluation and nursing care plan.
- Discuss hypoparathyroidism—etiology, pathophysiology, clinical manifestation and medical nursing management.
- Explain disorders of pancreas—diabetes mellitus—etiology, pathophysiology, clinical manifestation, diagnostic evaluation, management and complications.
- Write about disorders of adrenal gland—adrenal tumor—types, management and nursing care plan.
- List pituitary disorders—like gigantism, acromegaly, dwarfism, acromicria, Simmonds' disease, SIADH, dystrophia adiposogenitalis, diabetes insipidus.
- Describe etiology, pathophysiology, clinical manifestations, diagnostic tests and management of pituitary disorders.

Chapter Outline

- Review of Anatomy and Physiology of Endocrine System
- Assessment and Diagnostic Tests

Disorders of Endocrine System

- Pituitary Gland
- Pituitary Disorders

Disorders of Thyroid Gland

- Hypothyroidism
- Hyperthyroidism (Thyrotoxicosis)
- Thyroiditis
- Goiter

Disorders of Parathyroid Gland

- Parathyroid Disorders

Disorders of Pancreas

- Diabetes Mellitus

Disorders of Adrenal Gland

- Addison's Disease
- Adrenal Tumors
- Tumors of Adrenal Glands

Key Terminology

Acromegaly: It is defined as an endocrine disorder that is characterized by progressive enlargement of body parts due to excessive secretion of growth hormone.

Adrenal gland: It is defined as a pair of suprarenal endocrine gland consists of adrenal cortex and medulla to regulate metabolic pathways.

Aldosterone: It is defined as mineral corticoid hormone secreted by adrenal cortex to promote the absorption of sodium and excretion of potassium by kidneys.

Cortisol: Also called stress hormone and defined as steroid hormone which is produced by adrenal cortex and perform functions of inflammation, immune function and metabolism.

Cushing syndrome: It is defined as a group of symptoms that appear due to excessive secretion of adrenocorticotropic hormone and characterized by features of truncal obesity, moon face, buffalo hump, Hypertension, etc.

Diabetes insipidus: An endocrine disorder occurs due to deficiency of vasopressin and characterized by excretion of large volumes of diluted urine.

Diabetes mellitus: A collection of metabolic illnesses known as diabetes mellitus causes elevated blood glucose level as a result of abnormalities in insulin secretion, insulin action, or both.

Ductless glands: The glands present in the human body which secrete their products directly into the bloodstream and are also called as endocrine glands.

Endocrine: It is defined as simply hormone secretion by a ductless gland in the body or something that is internally secreted.

Gigantism: It is an excessively large growth that occurs in childhood before the growth plates close due to an over secretion of growth hormone.

Glucose tolerance test: It is also known as oral glucose tolerance test, the glucose tolerance test finds abnormalities with how your body processes glucose after eating, frequently before your fasting blood glucose level does.

Glycosylated hemoglobin: Glycosylated hemoglobin level is the indicator of how effectively blood sugar has been managed over a longer period of time, every 2–3 months.

Gonads: A gonad is a reproductive gland. Male gonads are the testes, whereas female gonads are the ovaries.

Hashimoto thyroiditis: The autoimmune thyroid illness Hashimoto thyroiditis is characterized by the destruction of thyroid cells by a variety of cell- and antibody-mediated immunological mechanisms.

Hormone: Defined as special type of messengers consists of chemical transmitters that are secreted by one organ of body and carried to target site by bloodstream.

Hypothalamus: It is defined as part of brain which is located inferior to thalamus, serves and a linking structure between nervous and endocrine system and maintains homeostasis of body.

Insulin: Also called investment hormone and is defined as a peptide hormone produced by β cells of pancreas to regulate glucose levels in blood.

Nephropathy: Long-standing history of diabetes and persistent albuminuria lasting 3–6 months in at least 2 out of 3 consecutive urine collections.

Neuropathy: A descriptive word for a demonstrable illness that is either clinically obvious or subclinical and only manifests in peripheral neuropathy when there is diabetes mellitus present.

Pancreas: It is defined as elongated heterocrine gland which consists of digestive exocrine gland and hormone producing endocrine gland located behind the stomach.

Parathyroid gland: An endocrine gland located in neck and embedded posterior to thyroid gland regulates calcium and phosphorus metabolism.

Pineal gland: Also called as epiphysis gland located above hypothalamus behind 3rd ventricle and produces melatonin to regulate circadian rhythm and influences sleep—wakes cycles.

Pituitary gland: Also called hypophysis or master gland of the endocrine system that is round in structure, located in sella turcica and controlled by hypothalamus.

Radiosensitive iodine: Radiosensitive iodine is radioactive form of iodine that is used for imaging test to treat cancer mainly thyroid cancer.

Retinopathy: It is a retinal disease caused by microangiopathy, a long-term impact of diabetes that results in gradual retinal damage and blindness.

Clinical Skills

1. Assessment of endocrine system
2. RBS monitoring
3. Insulin administration

CASE SCENARIOS RELATED TO NURSING MANAGEMENT OF PATIENT WITH ENDOCRINE DISORDER

CASE 1

A 67-year-old postmenopausal woman whose name was Meena was diagnosed to have T2DM 4 years back and is currently on dosage of metformin. Her glycemic levels for the past few weeks have not been controlled well and she visited doctor. She also diagnosed osteoporosis and she had a fragility fracture recently and is taking calcium, Vitamin D, and bisphosphonates for prevention of further fractures. Meena stays alone in a flat and manages her daily activities by herself.

- Discuss the case history and present complaint.
- What precautions or treatment she has to take according to her current situation?
- Prepare the nursing care plan.

CASE 2

Mrs Sushma, 50-year-old housewife came to emergency department with complains of progressive weight gain of 17 kg in 6 months, she also tells that she is feeling dizziness, fatigue and experience loss of memory, slow speech. Sushma was telling to a nurse in ER that she also has dry skin, constipation, and cold intolerance.

Findings of physical examination are:

- Temperature 96.8°F
- Pulse 58/minute and regular
- BP 110/60.

On examination, nurse found that her weight is 92 kg and has a puffy face, with pale, cool, dry, and thick skin.

- Enlist the signs and symptoms.
- What will the diagnosis and what physical findings supported the diagnosis?
- On the basis of initial assessment, prepare the nursing care plan.

CASE 3

Ms Shreya, 23-year-old was observed in April 2022 complaining of polydipsia, polyuria, nocturia and weight loss since November 2021. Doctors suspect Diabetes Insipidus and therefore Diabetes mellitus (DM) was excluded and she was admitted for study of possible diabetes insipidus for various investigations. Water deprivation test was suggestive of Central Diabetes Insipidus. MRI report showed infundibular hypophysitis and no hyperintense signal in the neurohypophysis. She started her therapy with oral desmopressin with clinical improvement.

- Discuss management of diabetes insipidus.
- On the basis of assessment, prepare the nursing care plan.

REVIEW OF ANATOMY AND PHYSIOLOGY OF ENDOCRINE SYSTEM

The endocrine system consists of organ systems that participate in communication in the human body through the secretion of ductless glands, called hormones. Nervous system communicates through its electrical and chemical signaling between neurons and target cells. In this way, endocrine system and nervous system maintain the homeostasis in the human body. There are two types of glands: endocrine glands and exocrine glands. Major differences between endocrine and exocrine glands are described in Table 8.1.

TABLE 8.1: Differences between endocrine and exocrine glands

Endocrine glands	Exocrine glands
• Secrete hormones into the surrounding fluids, which enters into blood and lymph	• Secrete substances directly to target tissues
• Secrete hormones	• Don't secrete hormones
• Ductless glands	• May or may not have ducts
• Example: Pituitary gland, Thyroid gland, ovaries	• Example: Salivary glands, sweat glands, gastric glands

The hormone is secreted by endocrine gland in the extracellular fluid. From there, hormone diffuses into the bloodstream and is transported throughout the body. The hormone, after reaching to the organ, binds to receptor on target cells and induces its specific responses. The endocrine system regulates growth, reproduction, fluid and electrolyte balance, body metabolism and sexual development. In normal healthy individual, the level of a hormone is constant. If hormone concentration in the body increases, its production is inhibited and if its concentration in the body decreases, body produces more hormone for regulation of its concentration in the bloodstream. This mechanism is called negative and positive **feedback control** respectively**.** The hormone levels can be affected by various factors. The nervous system can control the level of hormones by releasing and inhibiting action of hypothalamus. For example,

- Hypothalamus stimulates anterior pituitary to produce TSH
- TSH stimulates thyroid gland to produce T3 and T4
- Nutrition can also affect the level of hormones. For example, iodine is required for production of thyroid hormones T3 and T4.

Classification of Hormones

Hormones are classified into 2 major categories on the basis of their chemical structure or morphology, as Water-soluble hormones and lipid-soluble hormone (Table 8.2).

TABLE 8.2: Differences between water-soluble and lipid-soluble hormones

Water-soluble hormone	Lipid- soluble hormone
• Hormones are soluble in water • Unable to enter into the cell through phospholipid of the plasma membrane • Binds with the receptors tissue on the surface of cell • **Reaction:** Amino-tri-phosphate synthesized into Amino-mono-phosphate inside the cell • **Hormones:** Insulin, epinephrine, HGH and Oxytocin	• Hormones are soluble in lipids • Passes into the plasma membrane • Bind is directly to receptors into the cell nucleus • **Reaction:** cell growth and function through production of RNAs from DNA of the cell. • **Hormones:** Estrogen, Testosterone, Glucocorticoids

Endocrine Glands

The endocrine glands are:

- Pineal gland
- Hypothalamus and Pituitary gland
- Thyroid gland
- Parathyroid glands
- Pancreas
- Adrenal glands
- Gonads (Ovaries and Testes)

The location of the gland is shown in Figure 8.1.

Pineal Gland

The pineal gland, is located in the center of the head also called the pineal body or epiphysis, hormone secreted by the pineal gland is melatonin, a hormone produced at night and related to the regulation of circadian rhythm (or the circadian cycle, the wakefulness-sleep cycle). Melatonin also regulates body's functions related to the night-day cycle (Fig. 8.2).

Hypothalamus and Pituitary Gland

The pituitary gland, also called as hypophysis, is situated in the skull below the hypothalamus. It is often called "Master Gland" because it produces largest number of hormones that control functions of other endocrine glands of the body (Fig. 8.3).

Pituitary Gland has two parts: Anterior Pituitary (or adenohypophysis) and the posterior pituitary (or neurohypophysis) (Fig. 8.4).

Pituitary gland is connected by the hypophyseal stalk to the hypothalamus. The hypothalamus is situated on the dorsal side of the pituitary gland. The pituitary gland has two parts. The anterior (or front) pituitary produces hormones that affect the

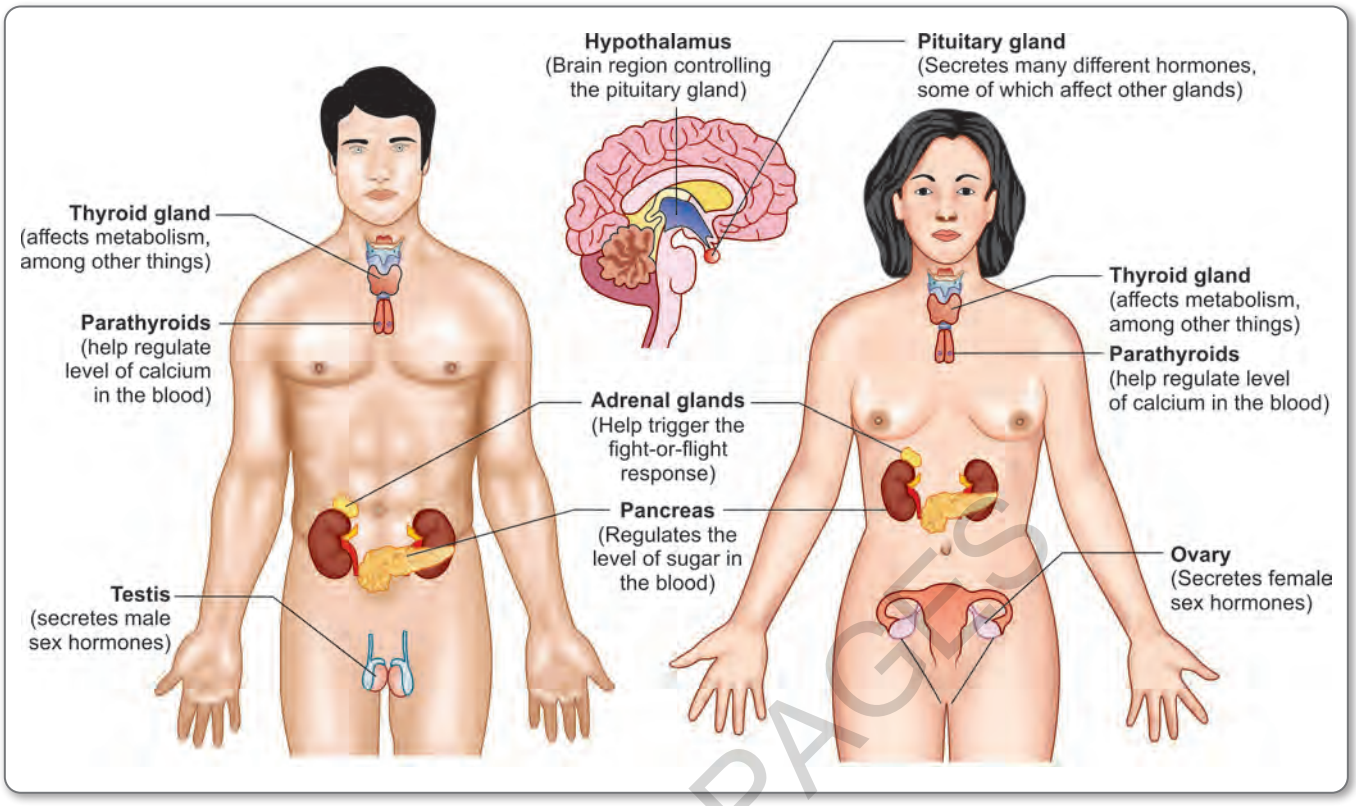

Fig. 8.1: Location of the endocrine glands

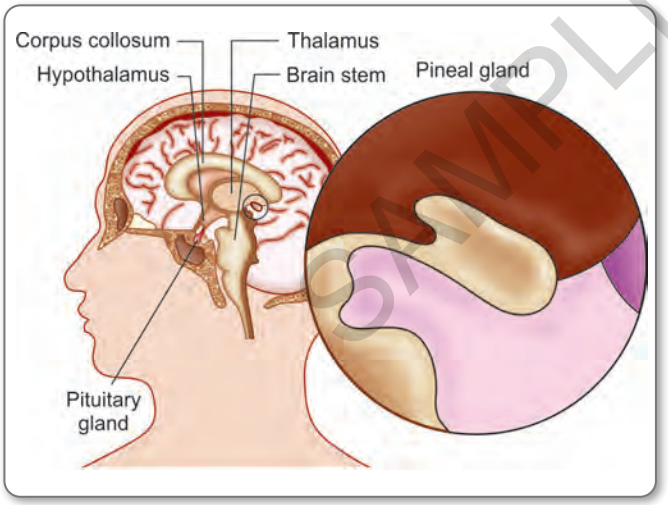

Fig. 8.2: Pineal gland

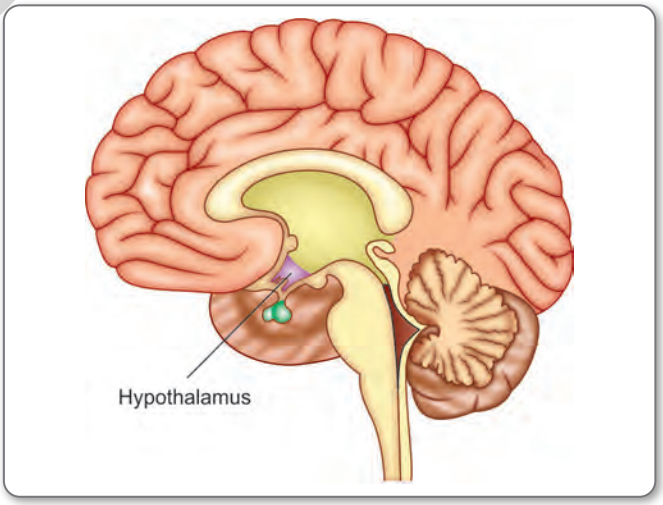

Fig. 8.3: Hypothalamus

breasts, adrenals, thyroid, ovaries and testes, as well as several other hormones. The main glands affected by the posterior (or rear) pituitary are the kidneys. The hormones produced by the anterior pituitary gland are ACTH (adrenocorticotropic hormone), FSH (follicle-stimulating hormone), GH (growth hormone), LH (luteinizing hormone), PRL (prolactin), TSH (thyroid-stimulating hormone. Posterior pituitary gland secretes ADH (antidiuretic hormone, or vasopressin) and Oxytocin. Pituitary hormone secretion (Fig. 8.5) is controlled by hypothalamus by secreting releasing factor.

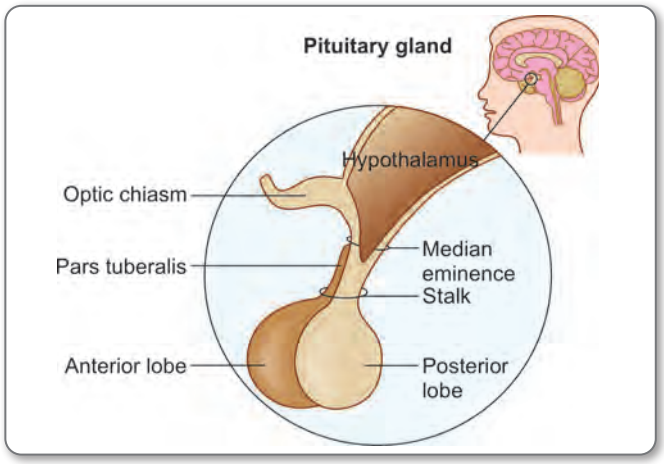

Fig. 8.4: Pituitary gland

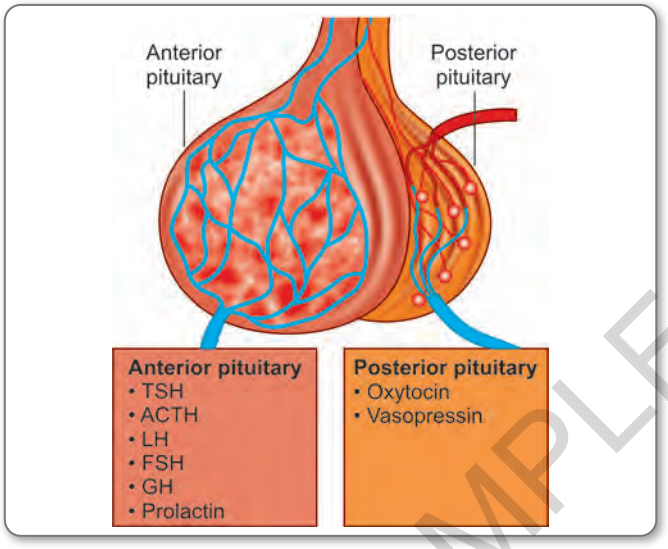

Fig. 8.5: Hormones secreted by pituitary gland

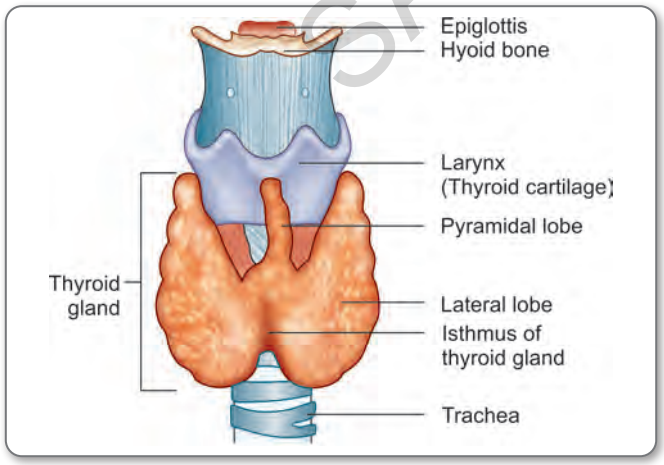

Fig. 8.6: Thyroid gland

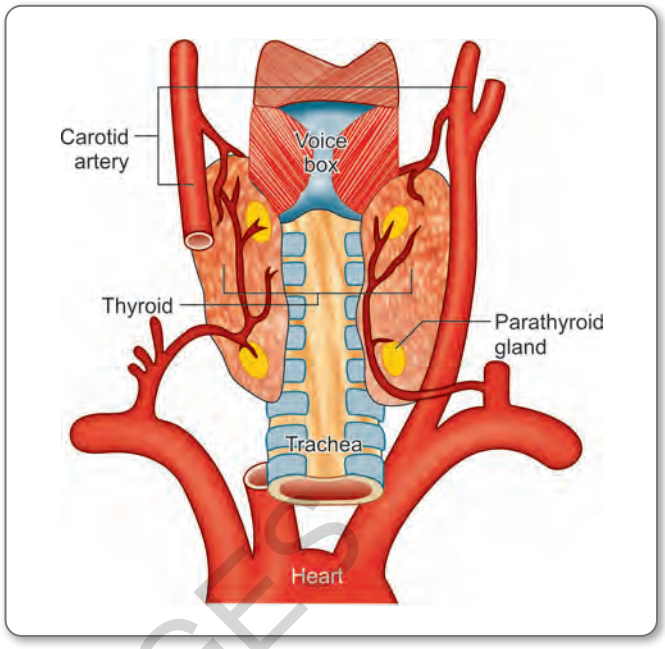

Fig. 8.7: Location of parathyroid gland

Thyroid Gland

The thyroid gland (Fig. 8.6) is located at the anterior and upper part of the trachea. The gland resembles a butterfly in shape. It has two lobes, which are connected by isthmus. Hormones secreted by thyroid gland are called thyroid hormones (TH) and include T3 (triiodothyronine) and T4 (thyroxine). Thyroid hormone increases the metabolism in adults. Calcitonin is also secreted by thyroid gland which decreases excess level of calcium in the body.

Parathyroid Glands

There are four to six parathyroid glands embedded in the posterior lobes of thyroid gland. Hormone secreted by parathyroid gland is called parathyroid hormone (PTH) or parathormone. PTH hormone controls phosphate metabolism. Vitamin D in normal amounts is necessary for PTH effect (Fig. 8.7).

www.parathyroid.com/parathyroid.htm

Pancreas

Pancreas (Fig. 8.8) is situated in the abdomen behind the stomach between spleen and duodenum. It performs both endocrine and exocrine functions. Endocrine cells of pancreas produces hormones (glucagon and insulin) while exocrine secretion includes digestive enzymes (pancreatic polypeptides).

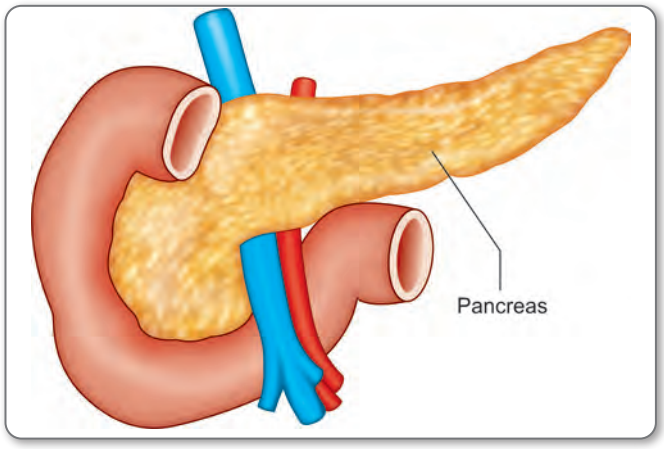

Fig. 8.8: Pancreas

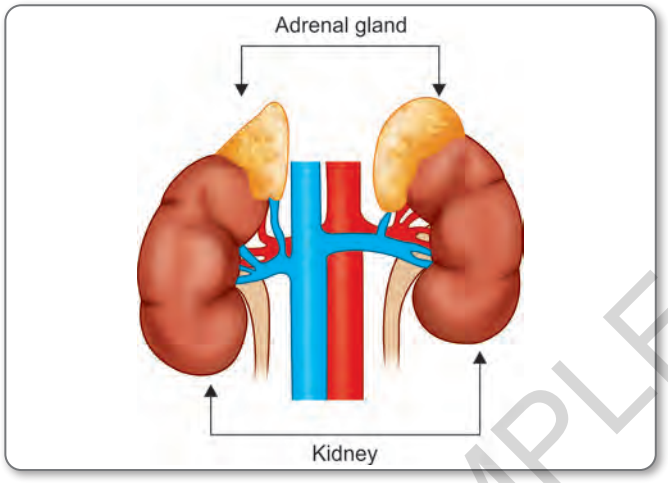

Fig. 8.9: Adrenal gland

Adrenal Glands

Adrenal glands (Fig. 8.9) are located on the upper portion of the kidney. They are two in number. Each gland has two parts; inner part is medulla and outer part is cortex. Adrenal medulla secretes catecholamine and adrenal cortex secretes steroid hormones.

The catecholamine: epinephrine (adrenaline) and nor epinephrine (noradrenaline)

Epinephrine stimulates and pituitary to secrete ACTH. Norepinephrine acts on heart it increases the heart rate and force of cardiac contraction. It also causes vasoconstriction throughout the body.

Gonads

The testes in man and ovaries in female are gonads (Fig. 8.10). Gonads secrete sex hormones, which regulate the growth and promote the onset of puberty. Testis in man produces androgen

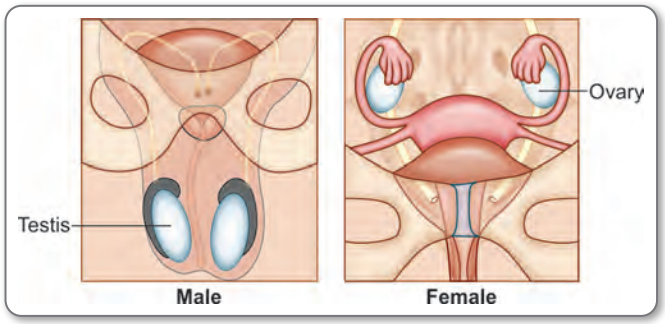

Fig. 8.10: Gonads

(primarily testosterone); in women, ovaries secretes estrogen and progesterone to maintain reproductive functioning.

Evidence Based Practice

A Study conducted by Ed Susman March 07, 2015 revealed— Men with borderline low testosterone levels who are referred to an endocrinologist often show signs of clinical depression or depressive symptoms, researchers reported that out of 200 men in a study, 56% had significant depressive symptoms, had a known diagnosis of depression and/or were using antidepressants when they were seen by Michael Irwig, MD, associate professor of medicine and director of the Center for Andrology at George Washington University, Washington.

ASSESSMENT AND DIAGNOSTIC TESTS

History taking—Nurses must have an adequate knowledge regarding anatomy and physiology of endocrine system, which consists normal function of endocrine glands or their hormones. She will be able to identify the altered functions in the endocrine disorder

Assessment of Endocrine Disorders

History of Past Illness

- Childhood health problems
- Assess an abnormal growth pattern, e.g., head circumference, size of the hand and feet.
- Any history of hair growth on face in women, decreased hair growth in men.
- Take history of immunization.
- History of hospitalization, head trauma (may lead to pituitary dysfunction), past surgical history.

Family History

Family history is important, as many endocrine disorders run in the family. A well collected history will help in diagnosis and management of the disease.

History of exercise, nutrition and habits:

- Assess exercise, food intake, sleep and rest pattern.
- Also explore use of alcohol; alcohol may cause liver and pancreas abnormality.
- Collect demographic data of the client—Age, gender, geographical residence,
- Medical history of some disorders, e.g., Diabetes mellitus, hepatitis, and disorder of gallbladder.

Physical Examination

Nurse will use various methods of physical examination which includes vitals measurement, systematic inspection/head-to-toe inspection, the endocrine glands should be inspected and palpated routinely in all suspected patients. It is important to examine physical, psychological and behavioral changes. Patients suffering with endocrine disorder may exhibit following changes:

Assess clients' chief complaints by system wise assess-ment:

- **Integumentary system**
  - Hyper pigmentation or hypo pigmentation may be due to Addison's disease.

  Changes in hair distribution amount or texture, etc.
- **Cardiovascular system**
  - Circulation (Palpitation, chest pain, dysrhythmias, gallop rhythm, etc.)
  - Kussmaul's respiration (Deep rapid breathing is due to diabetic ketoacidosis.
- **Neurological system**
  - Nervousness, increased irritability, insomnia, fatigue, muscle weakness, etc.
- **Elimination**
  - Polyuria, diarrhea, stool changes anorexia, etc.
  - Food and Fluid (Weight loss/gain, increased thirst, nausea, vomiting, etc.)
- **Musculoskeletal**
  - Generalized weakness
  - Decreased muscle mass
  - Enlargement of bones and cartilage
- **Neurosensory**
  - Speech alteration, mental status changes, etc.
- **Pain**
- **Ego integrity**
  - Anxiety, depression, emotional liabilityetc.
- **Safety**

  Heat/cold intolerance, excessive sweating, exophthalmos, elevated temperature, etc.
- **Reproductive system**
  - Decreased libido, hypomenorrhea, impotence, etc.
  - Fluid deprivation-weight loss/gain
- **Head and Neck**
  - Pituitary gland enlargement or tumor leads to pressure on optic nerve which causes decreased visual acuity.
  - In hyperthyroidism fluid accumulation in eye and retro-orbital tissue can cause eyeball protrusion from orbit.
  - In Cushing Syndrome increased cortical secretion cause moon face and facial fullness
  - In hyperthyroidism patient may have puffiness, periorbital edema,
  - Goiter can be found out due to iodine deficiency

Clinical Manifestation

The clinical manifestation of endocrinal diseases are tabulated as follows (Table 8.3).

TABLE 8.3: Clinical manifestation of Endocrine diseases

- Lethargy
- Muscle hypertonia (Fig. 8.11)

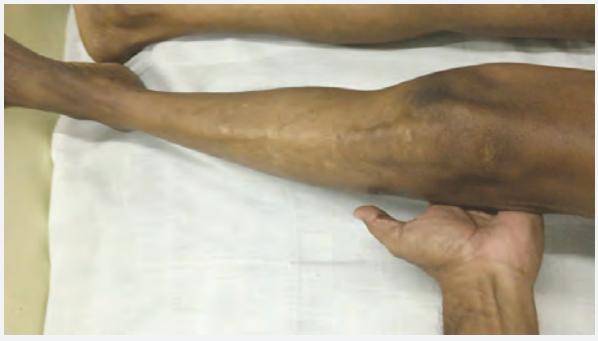

Fig. 8.11: Muscle hypertonia of lower limb

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- Mask-like face (Fig. 8.12)

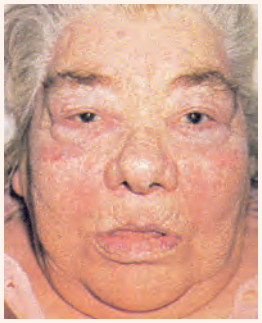

Fig. 8.12: Mask like face as seen in Parkinson's disease

- Vision change (Fig. 8.13)

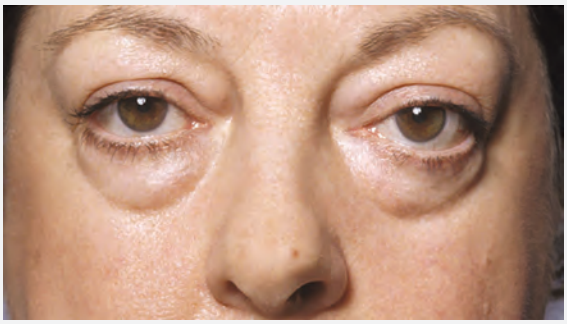

Fig. 8.13: Vision changes in thyroid disease.

- Dull mental process
- Emaciation (Fig. 8.14)

Fig. 8.14: Emaciation

- Exophthalmoses (Fig. 8.15)

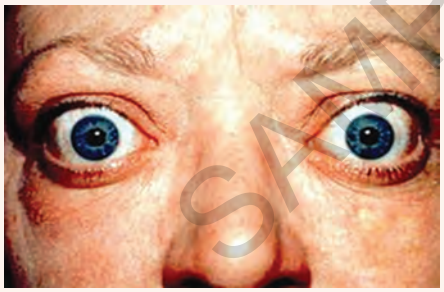

Fig. 8.15: Exophthalmos as seen in Graves disease

- Growth of hair on female face (Fig. 8.16)

Fig. 8.16: Growth of hair on female face

- History of emotional reaction
- Buffalo hump, moon face (Fig. 8.17)

Fig. 8.17: Moon face as evident in Cushing syndrome

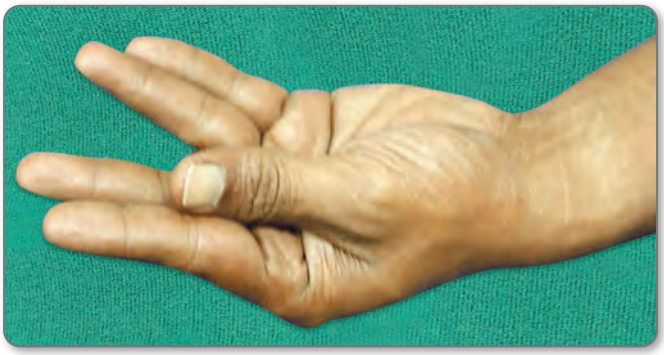

Fig. 8.18: Carpopedal spasm as evident in hypocalcemia

- Measurement of blood calcium, phosphate or serum cortisol level
- Chvostek's sign (the twitching of the facial muscles in response to tapping over the area of the facial nerve)
- Trousseau's sign (carpopedal spasm caused by inflating the blood-pressure cuff to a level above systolic pressure for 3 minutes.) (Fig. 8.18)

Common manifestations during assessment which suggest the endocrine disorders are as heat/cold intolerance, altered energy level, weight gain/loss, growth of hair on female face, alteration in secondary sexual characteristics, vision changes, joint pain, etc. Nurse will document all this collected history in sequence, so that it helps to diagnose the disease (Table 8.4).

Diagnostic Tests

TABLE 8.4: Diagnostic tests to assess endocrine function

Name of the Test	Indication	Normal Value	Patient Preperation
Growth hormone (GH), Human Growth Hormone (hGH)	To assess GH levels to identify GH deficiency (dwarfism) or GH excess (gigantism, acromegaly).	Men: <5 ng/mL Women: <10 ng/mL	Client should not eat or drink anything 8–10 hours prior to having blood drawn. Instruct client to rest for 30 to 60 minutes before blood is drawn
Somatomedin C (Insulin- Like Growth Factor or IGF-1)	This test used to evaluate secretion of growth factor and to identify GH deficiency or excess (as above).	125–250 ng/mL	Fasting previous night of the investigation preferred but not necessary
Water Deprivation Test	This combination of blood and urine test is used to identify causes of polyuria (increased urine output), syndrome of inappropriate antidiuretic hormone, psychogenic polydipsia, ADH or vasopressin is given IM or subcutaneously		Instruct not to smoke, eat or drink after midnight, duration of test will be 8 hours. Every hour assess weight, take postural BP (lying and standing measures separated as ordered) assess urine for volume and specific gravity, and send samples of urine to the lab for osmolality. Blood samples for osmolality are taken when urine samples are collected and when client demonstrates orthostatic hypotension.
Magnetic Resonance Imaging (MRI)	This radiographic study is done to identify tumors of the hypothalamus or pituitary gland.		Explain client that he has to lie still during the examination. Assess for any metallic implants (such as pacemakers, body piercings, shrapnel). If present, test is not performed.
Thyroid–stimulating TSH	In this blood test, TSH and T_4 levels are measured to differentiate pituitary from thyroid causes of hypothyroidism.	<3 ng/mL	Client should not eat shellfish for several days before the test. Evaluate medications: TSH value may be increased by aspirin, steroids, dopamine, and heparin; and decreased by lithium and potassium iodide.
Thyroxine (T_4)	To determine thyroid function and aid in the diagnosis of hyperthyroidism and hypothyroidism.	Free T_4 1.0–2.3 ng/mL	Assess medication: Value of T4 may be altered by chlorpromazine (Thorazine), phenytoin (Dilantin), heparin, lithium, sulfonamides, reserpine (Serpasil), testosterone, propranolol (Inderal), tolbutamide (Orinase), and salicylates in high doses. oral contraceptives, estrogen, clofibrate, and perphenazine (Trilafon).

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Name of the Test	Indication	Normal Value	Patient Preperation
Triiodothyronine (T_3)	To diagnose hyperthyroidism and to compare T_3 with T_4 for diagnosis of thyroid disorder	80–200 ng/dL	Evaluate medications: Value can be altered by propylthiouracil, methimazole, lithium, phenytoin (Dilantin), propranenololreserpine, large doses of aspirin, steroids, and sulfonamides. Estrogen, progestins, oral contraceptives, T_3 and methadone.
Triiodothyronine Resin Uptake (T_3RU)	This test is an indirect measure of free thyroxine (T_4). The client's blood is mixed with radioactive T_3 and synthetic resin, and the radioactive T_3 will bind with available thyroid-binding globulin sites. In hyperthyroidism there are few binding sites left, more T_3 is taken up by the resin, and a high T_3 resin uptake results. The opposite occurs in hypothyroidism.	25–35% uptake	No special preparation is needed.
Thyroid Antibodies (TA)	To assess thyroid immune disease (Graves disease, chronic thyroiditis, Hashimoto's thyroiditis).	Antithyroglobulin: negative to titer <1:20 Antimicrosomal: negative to titer <1:100	Assess for recent viral infection (which could trigger autoimmune disease).
Radioactive Iodine Uptake (RIA)	This test provides a direct measure of thyroid activity and is useful in evaluating the activity of solitary thyroid nodules. Based on the rationale that the thyroid gland takes up iodine in any form, radioactive iodine is given orally or intravenously, and the thyroid gland uptake is measured with a scanner at several hourly intervals and at 24 hours.	2–4 hours: 3–19% 24 hours: 11–30%	Nothing orally mouth for 6 to 8 hours before the test. Tell clients not to take supplemental iodine several weeks before the test and thyroid medications should be discontinued.
Thyroid scan	This radiologic study evaluates thyroid nodules. Radioactive isotopes are given orally and a scanner is passed over the thyroid to make a graphic record of the radiation emitted.	A normal thyroid scan has a homogenous pattern of radiation with symmetric lobes.	No special preparation is needed.
Parathyroid hormone (PTH)	A blood test done to identify hypoparathyroidism or hyperparathyroidism; also used to monitor response to PTH therapy.	Intact PTH: 11–54 pg/mL	Do not to eat or drink for 8 hours before the test.
Calcium (Ca)	To check for serum calcium excess or deficit in parathyroid and bone disorders; and to monitor calcium levels.	9.0–11.0 mg/dL, 4.5–5.5 mEq/L, or 2.3-2.8 mmol/L (SI units)	Observe for manifestations of tetany, including positive Chvostek's and Trousseau's signs, if hypocalcemia.
Cortisol	To measure amount of total cortisol in the serum and evaluate adrenal cortex function. It is decreased in Addison's disease and hypothyroidism; increased in Cushing's syndrome and hyperthyroidism.	8 a.m. – 10 a.m.: 138–635 nmol 4 p.m.–6 p.m.: 83–359 nmol <100 mcg/24 hours	Instruct client not to eat or drink and to rest for 2 hours before the test. Evaluate medications: Cortisol is decreased by androgens, phenytoin (Dilantin), and increased by oral contraceptives, estrogen, spironolactone (Aldactone) and Triparanol.

Contd...

Name of the Test	Indication	Normal Value	Patient Preperation
Aldosterone	To assess a deficit or an excess of aldosterone; and to compare blood and urine levels with other lab data to evaluate overhydration with increased sodium and adrenal malfunction. A 24-hour urine test is considered a more reliable measure of aldosterone than a random aldosterone test.	<16 mcg/dL (fasting) 6-25 mcg/24 hours	Assess diet and lab results: Levels are increased by hyponatremia, hyperkalemia, and a low-salt diet. Values are increased by diuretics, hydralazine (Apresoline), diazoxide (Hyperstat), nitroprusside, and oral contraceptives.
Adrenocorticotropic Hormone (ACTH)	To determine if a decreased plasma level of cortisol is due to adrenal cortex hypofunction or pituitary hypofunction.	7 a.m. – 10 a.m.: 8-80 pg/mL 4 p.m.: 5-30 pg/mL 10 p. m. – 12 p. m.: < 10 pg/mL	Instruct the client that food and fluids may be restricted, and to eat a low-carbohydrate diet for 24 hours prior to the test. Assess medications: ACTH values may be increased by metyrapone, vasopressin and insulin; and decreased by steroids, estrogen amphetamines and alcohol.
ACTH Stimulation	Done to check for pituitary hypofunction. The drug metyrapone (Metopirone) is given to block the production of cortisol, thus causing an increased ACTH secretion. If the ACTH level does not increase, the problem is pituitary insufficiency.		Ask medications as for ACTH test.
ACTH Suppression	Done to check for the origin of the condition. The drug dexamethasone (Decadron) is given to suppress ACTH production. If an extremely high dose is needed, the cause is of pituitary origin; if the plasma cortisol continues to be high with ACTH suppression, the cause could be adrenal cortex hyperfunction (Cushing's syndrome). Normally the plasma cortisol level should double in 1 hour.		Tell client to avoid tea, caffeinated coffee, and chocolates; no other food or fluid restriction is needed. Assess medications: False positives may be caused by phenytoin, barbiturates, meprobamate, and carbamazepine. If dexamethasone causes gastric irritation, milk or antacids may be required.
17-Ketpsteroids	This 24-hour urine test is done to measure metabolites in urine and evaluate adrenal cortex function.	Men: 5-25 mg in 24 hours Women: 5-15 mg in 24 hours	Instruct client how to save urine (urine must contain a preservative and be refrigerated). Assess medications and refer to information about the test. Levels are affected by a variety of drugs; if possible, these should be discontinued for 48 hours before the test. Women cannot have the test while menstruating because blood can cause a false-positive finding.
Computerized Tomography (CT) of the Abdomen	This radiologic study is used to assess the adrenal gland for tumors (including size and metastasis).		Determine if contrast medium will be used; if so, assess client for allergy to iodine (shellfish).
Fasting Blood Sugar	This test of serum or plasma is used to identify or confirm a diagnosis of diabetes mellitus. A finding of greater than 125 mg/dL might indicate diabetes.	Serum/plasma: 70-100 mg/dL	Tell the client not to eat or drink for 12 hours before the test. Do not administer insulin until blood specimen is taken. Assess medications: FBS may be increased by cortisone, diuretics, ACTH, levodopa, epinephrine, anesthetics and phenytoin (Dilantin).

Contd...

Name of the Test	Indication	Normal Value	Patient Preperation
Oral Glucose Tolerance Test (OGTT)	If previous fasting blood sugar (FBS) results are elevated or inconsistent, diabetes mellitus should be diagnosed.		Instruct the client to fast for 12 hours prior to the test. Inform the client that they cannot consume any food or liquids during the test, excluding water. Evaluate the medications: Steroids, oral contraceptives, estrogens, thiazide diuretics, and salicylates are medications that may raise OGTT levels. Inform the client that throughout the test, he or she might feel lethargic and perspire; in this case, the nurse should be informed. Although they are typically transient, they could be signs of hyperinsulinism.
Glycosylated Hemoglobin (HbA1c)	The efficacy of treating diabetes mellitus is evaluated using this serum test. The results show the average blood sugar level over a 1 to 4 month period; a high level denotes uncontrolled diabetes mellitus and a higher risk for complications.	5.5–9% of total high	Chronic renal failure, long-term blood loss, and anemias can all lead to decreased levels. Hyperglycemia, alcohol consumption, pregnancy, hemodialysis, and continuous cortisone use can all cause levels to rise.
Computed Tomography (CT) of the abdomen	This radiographic examination is used to find pancreatic cysts or malignancies.		If a contrast medium is given, check for iodine allergies (shellfish).

Desmopressin Response Test

Desmopressin is also known as DDAVP (1-deamino-8-D-arginine Vasopressin). It is a synthetic hormone that temporary boost level of clotting factor in bloodstream. This hormone can be used before any surgery or invasive procedure to stop excessive bleeding.

It also helps to release Von Willebrand factor and factor VIII (clotting factor). Von Willebrand factor is a protein which sticks to the areas of blood vessels that is damaged and helps the bleeding to stop. Less Willebrand factor in the blood cause bleeding for longer period.

Recommendation of Test

The test is carried out for:

- Mild Hemophilia
- VonWillebrand Disease
- Platelet Function Defect

Precautions

It is important to ask the patient before test for:

- Medication for heart problem
- History of high blood pressure
- Pregnancy

Test Procedure

- Before the patient taking DDAVP, nurse must take blood sample to check the factor level and also record blood pressure and pulse
- If readings are normal, give quick injection of DDAVP under skin at upper arm. Patient may feel stinging sensation as the medicine is injected
- After 90 minutes second blood sample should be taken
- After 4 to 6 hours third blood sample should be taken to monitor the increase of factor level in the bloodstream

Side Effects of DDAVP

- Redness on chicks
- Low blood pressure
- Nausea
- Headache
- Allergic reaction at injection site
- Warmth around the face

DAAVP can cause body to retain fluid therefore it is advised to the patient not to drink more than 1 L of fluid during the 24 hours after the injection

DISORDERS OF ENDOCRINE SYSTEM

PITUITARY GLAND

Pituitary gland which is located in the brain is called master gland. It regulates the function of various hormone releasing glands such as thyroid, ovaries, testes and adrenal glands (Fig. 8.19). When the pituitary gland produces an excessive amount or insufficient amount of a certain hormone, pituitary diseases result. A pituitary tumor is the most frequent cause of these conditions.

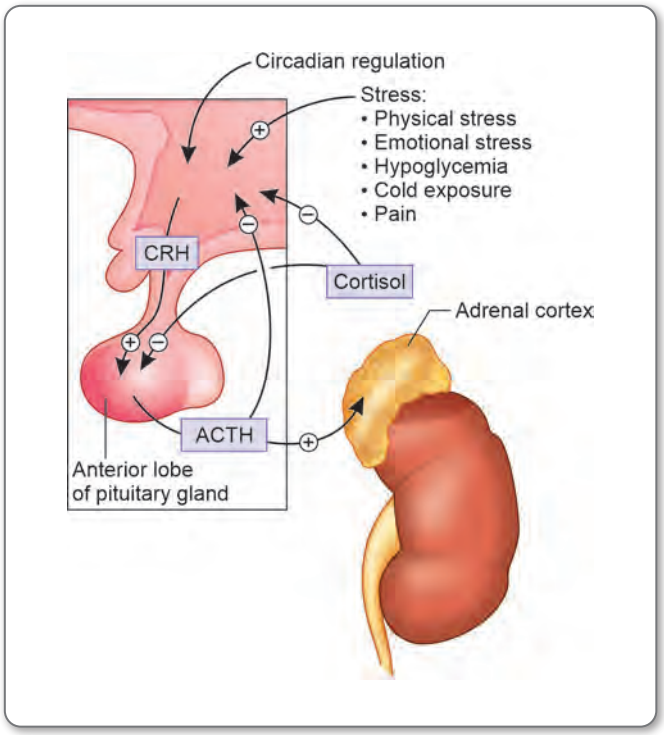

Fig. 8.19: Function of pituitary gland

Causes of Disorder of Pituitary Gland

Pituitary functions may be altered by over secretion or under secretion of the hormones released by the gland. These disorders occur independently in anterior and posterior lobe.

Mainly of two reasons are there:

1. Hyperactivity
2. Hypoactivity

Types of Pituitary Disorders (Table 8.5)

TABLE 8.5: Types of pituitary disorders

Part Involved	Hyperactivity	Hypoactivity
Anterior pituitary	1. Gigantism 2. Acromegaly 3. Acromegalic gigantism 4. Cushing's Syndrome	1. Dwarfism 2. Acromicria 3. Simmonds' disease
Posterior pituitary	Syndrome of Inappropriate ADH (SIADH)	Diabetes insipidus
Anterior and posterior pituitary	—	Dystrophia adiposogenital

PITUITARY DISORDERS

Simmonds' Disease

In 1939, the German Medical Society proposed renaming anterior pituitary deficiency Simmonds Disease after Maurice Simmonds. Pituitary cachexia is another name for Simmonds' disease. It is attributed to hypophysis (anterior portion of pituitary gland) destruction or physiology exhaustion. Tumor, embolic infarction, tuberculosis, and syphilis cause destruction. Simmonds' disease symptoms include premature senility, gonad and genital atrophy, amenorrhea, breast atrophy, loss of pubic hair, loss of libido, constipation, hypotension, and muscular weakness.

SIADH

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) occurs when the body produces excessive levels of antidiuretic hormones, causing the body to retain water and sodium in the blood.

SIADH is most commonly caused by hypothalamic disease such as cancer, meningitis (inflammation of the meninges, the membranes that cover the brain and spinal cord), encephalitis (brain inflammation), brain tumors, psychosis, lung diseases, head trauma, Guillain-Barré syndrome, and certain medications, as well as damage to the hypothalamus or pituitary gland during surgery (Fig. 8.20). In more severe cases of SIADH, the following symptoms may occur: nausea or vomiting, cramps or tremors, depression, memory impairment, irritability, personality changes such as combativeness, confusion, and hallucinations, seizures, and stupor or coma. A complete medical history and physical examination, as well as blood and urine tests to measure sodium, potassium, and osmolality (concentration of solution in the blood and urine), will be required to confirm SIADH. Specific treatments for SIADH include medications that inhibit the action of ADH (also known as vasopressin), surgical removal of an ADH-producing tumor, and other medications to help regulate body fluid volume.

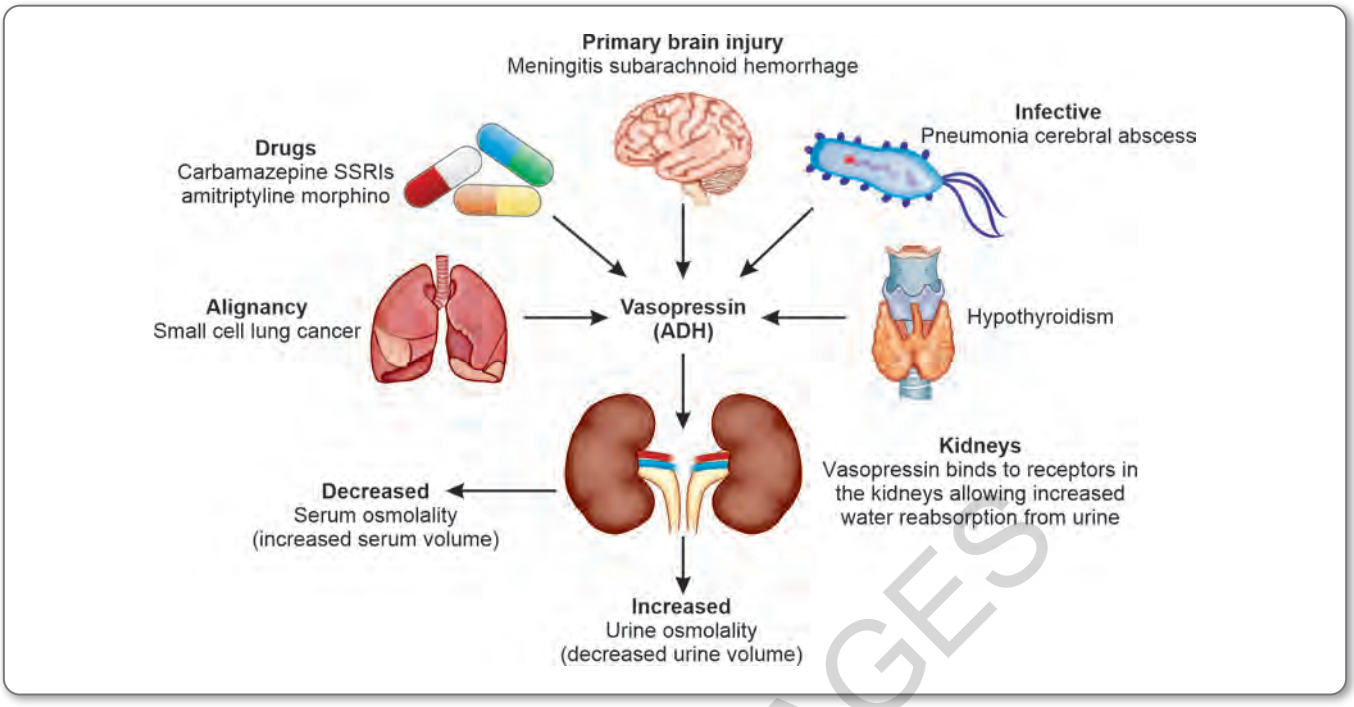

Fig. 8.20: Causative factors associated with SIADH

Pathophysiology

Pathophysiology of SIADH has been depicted in Figure 8.21.

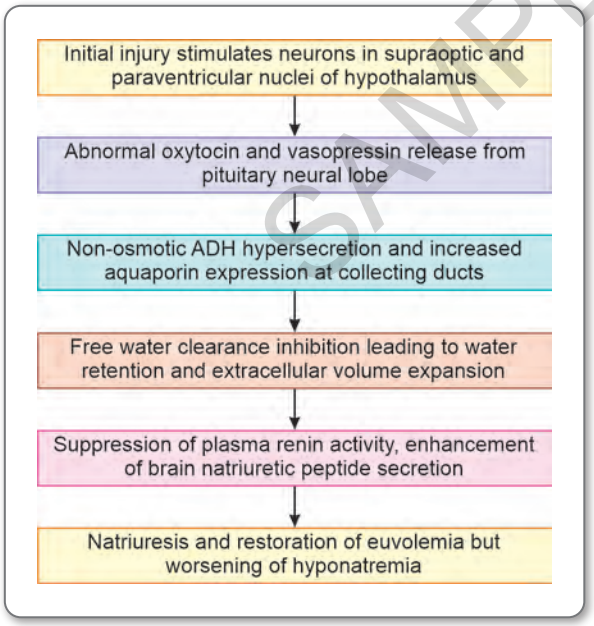

Fig. 8.21: Schematic diagram explaining pathophysiology of SIADH

Management of SIADH (Figs 8.22 and 8.23)

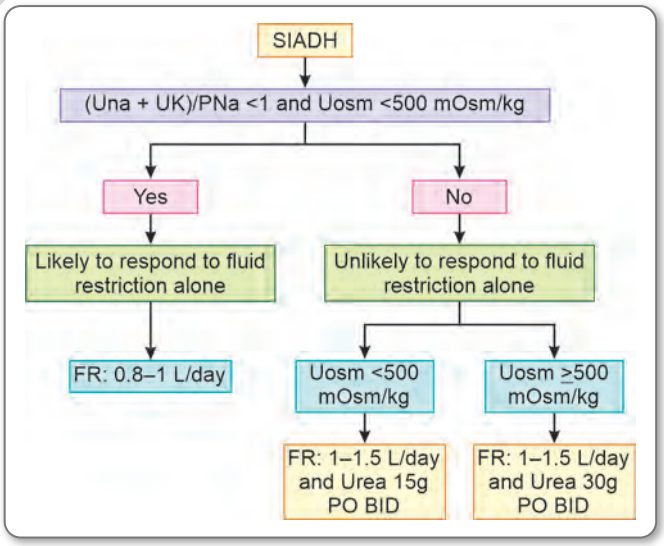

Fig. 8.22: Flowchart explaining management of SIADH

Indirect Modalities

- Fluid restriction
- Treatment of underlying pathology

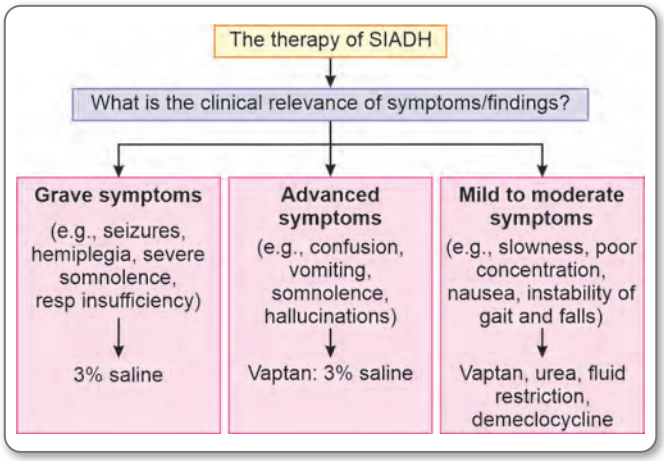

Fig. 8.23: Recommended fluid therapy for SIADH

- Hypertonic saline
- Loop Diuretics
- Urea
- Demeclocycline, lithium
- Hemodialysis, CVVH, SLEDD

Direct Modalities

- **Vaptan treatment:** Vaptans are a new class of pharmaceuticals that have been developed to treat hypervolemic and euvolemic hyponatremia. These medications are nonpeptide vasopressin antagonists that compete with V2 receptors in the kidney to inhibit the hormone's antidiuretic effect. This blockage causes water diuresis (aquaresis), which reduces body water content and raises plasma sodium levels if not offset by increased fluid intake. As a result of this increase in plasma sodium, thirst and plasma vasopressin concentrations rise, ostensibly limiting the effects of vasopressin antagonists. Nonetheless, vaptans are especially useful in treating hypervolemic hyponatremia caused by severe congestive heart failure or chronic liver failure, because the only other treatments currently available, such as fluid restriction and diuretics, are slow-acting and ineffective. Vaptans can also be used to treat euvolemic hyponatremia caused by the syndrome of inappropriate antidiuretic hormone (SIADH), especially when it is chronic and/or symptomatic. Vaptans, on the other hand, are less useful than hypertonic saline infusion in cases of acute, severe, and symptomatic hyponatremia because their effects vary unpredictably from patient to patient. Vaptan therapy is completely contraindicated in hypovolemic hyponatremia (low total body water) and in the vasopressin-independent form of inappropriate antidiuresis caused by constitutive activating V2 receptor mutations.

Anterior Pituitary Disorders

Gigantism and Acromegaly

Gigantism and acromegaly are the disorders of abnormally hypersecretion of growth hormone. Tall stature is a hallmark of gigantism, which develops when growth hormone hypersecretion takes place prior to the union of the long bone epiphysis. Acromegaly develops when the epiphysis fuses and GH hypersecretion continues, resulting in large extremities and a distinctive facial appearance (Fig. 8.24).

Etiology and Risk Factors

- Pituitary adenomas that secrete growth hormone are the primary cause of acromegaly and gigantism in around 95% of cases.
- Acromegaly can be due to an ectopic secretion of growth hormone-releasing hormone (GHRH) release from neuroendocrine tumors in the pancreas or lungs.
- Acromegaly can occasionally be caused due to an ectopic growth hormone release by abdominal and hemopoietic cancers.
- The genetic syndromes such as multiple endocrine neoplasia-1 (MEN-1), neurofibromatosis are linked to gigantism and acromegaly.

Clinical Manifestations

- Enlargement of the hands and feet is seen due to the bony expansion and soft tissue swelling.
- Hyperhidrosis and skin tags are present in about 98% of cases of acromegaly. Skin tags are due to the epithelial cell hyperproliferation induced by GH.
- Acromegalic facies: Prominent supraorbital ridge, broad nose, acne, large lips, overbite, prognathism, tongue enlargement, and coarsening of facial features form the characteristic acromegalic facies.

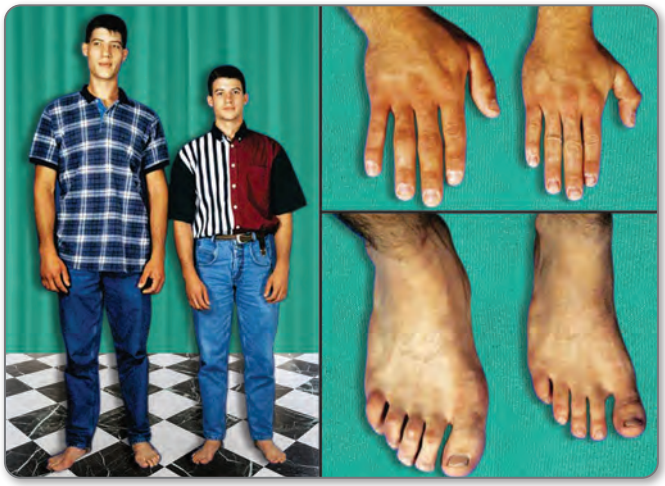

Fig. 8.24: Pituitary gigantism

- Generalized weakness and lethargy are common symptoms.
- Elongation of the jaw can lead to teeth malocclusion and temporomandibular joint pain.
- Abnormal curvature of spine from side to side and from front to back also known as kyphoscoliosis.
- Carpal tunnel syndrome due to the compression of median nerve by overgrown tissues.
- Delayed puberty or hypogonadotropic hypogonadism is also evident.
- Also, the heart, liver, kidneys, spleen, thyroid gland, parathyroid glands, colon, and pancreas are larger than normal.

Diagnostic Investigations

- A thorough clinical examination, a detailed patient history and specialized procedures like blood tests, a glucose tolerance test, magnetic resonance imaging (MRI), or computed tomography are used to confirm the diagnosis.
- Cortical thickening, enlargement of the frontal sinuses, and enlargement and erosion of the sella turcica are evident on CT, MRI, or skull x-rays.
- X-rays hands shows tufting, and the soft tissue thickening.
- Patients with suspected acromegaly should undergo monitoring for serum Insulin growth factor-1 levels, because they are often markedly high (3-fold to 10-fold).
- Normal plasma GH levels are high. Blood should be drawn before the patient consumes breakfast (basal state).
- Additional testing can be done, such as echocardiography for determining the involvement of heart.
- Prolactin levels should be checked, especially if the patient presents with galactorrhea or hypogonadism.

Management

Surgery, pharmacological management, and radiation therapy are the three main therapeutic modalities for acromegaly.

Surgical Management

Transsphenoidal surgery is typically used to treat acromegaly, which involves removing all or part of a pituitary tumor. Surgery frequently produces a quick therapeutic response, alleviating pressure on nearby brain structures immediately and reducing growth hormone levels. Surgery is more successful for people with microadenomas than it is for people with larger tumors (macroadenomas).

As long as the tumor is still accessible, surgery is also the treatment of choice in the event of a recurrence.

Diabetes insipidus and deficits in the anterior pituitary are examples of post-operative consequences.

Pharmacological Therapy

Acromegaly patients are frequently treated with three major kinds of drugs. These include dopamine antagonists, growth hormone receptor antagonists, such as pegvisomant, and analogs of the somatostatin hormone, such as octreotide, lanreotide, and pasireotide.

Radiation Therapy

Radiation therapy is used as a treatment for the patients in whom the surgery was ineffective to lower the hormone levels or tumor size. It is administered over the course of four to six weeks and daily treatments are often required for conventional fractionated radiation therapy.

Complications

The following complications arise due to disease conditions or treatment modalities:

- Cardiomyopathy
- Hypertension
- Obstructive sleep apnea
- Hypopituitarism
- Arthropathy
- Colon polyps

Cushing Syndrome

Cushing syndrome is a hormonal disorder results from excessive or high level of adrenocorticol activity. Cushing syndrome is also known as "Hypercortisolism" commonly caused by over use or continuous use of corticosteroid medications which lead to overproduction of corticosteroids from adrenal gland. Functions of cortisol

- Maintain Blood Pressure
- Maintain blood sugar level by balancing the effect of insulin
- Lower inflammation
- Convert food into energy
- Responding to stress

Cushing syndrome is more common in women than men. Sometimes malignancies such as bronchogenic carcinoma can cause Cushing Syndrome through ectopic production of ACTH.

Causes and Risk Factors

Over production of cortisol in the body causes Cushing Syndrome.

- Long-term use for corticosteroids medications such as prednisone

- Malnutrition
- Alcoholism
- Depression
- Panic disorders
- Athletic training
- High stress level
- Tumors produces too much ACTH such as – Pituitary gland tumors, ectopic tumors and adrenal gland tumors

Pathophysiology of Cushing Syndrome

When activated by ACTH, the adrenal gland releases cortisol and other steroid hormones as shown in Figure 8.25. In response to stimulation by corticotropin-releasing hormone (CRH) from the hypothalamus, the pituitary gland produces and releases ACTH into the petrosal venous sinuses. Just before waking up, ACTH is released at its peak, and throughout the day, levels decrease. Cortisol's negative feedback mechanism keeps CRH and ACTH secretion under control.

Cushing's disease adenomas secrete excessive amounts of ACTH and retain some negative feedback responsiveness to high glucocorticoid doses.

Symptoms

- Weight gain, especially in upper body parts
- Mood shaped face
- Rounded, puffy face
- Thinning of skin
- Tiredness
- Fatigue
- Weakness

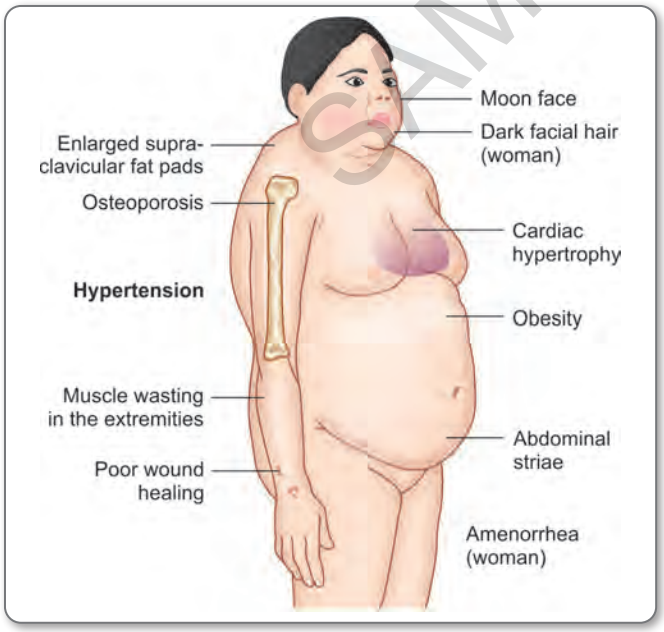

Fig. 8.25: Cushing's syndrome

- Acne
- Buffalo Hump (fatty deposits between the shoulder and upper back)
- High Blood sugar level
- High Blood pressure level
- Osteoporosis
- Depression
- Sleep Problem

Additional symptoms are

In children

- Obesity
- Slow Growth
- High Blood Pressure

In women

- Develop extra facial and body hair
- Irregular menstruation
- Decreased fertility

In men

- Erectile dysfunction
- Low sex drive or interest
- Decreased fertility

Diagnostic Investigations

- **Physical examination and history:** Sometimes Cushing Syndrome can be difficult to diagnose by physical examination itself, therefore history collection is must. Nurse should ask the following questions
  - What symptoms have you experienced or notice?
  - When did you first notice them?
  - Does anything cause symptoms worst or better?
  - What medications are you taking?
- **Laboratory test**
  - **24-hours urinary free cortisol test:** In this test patient will be asked to collect urine for 24 hours to measure the level of cortisol.
  - **Salivary cortisol level:** In normal person physiologically cortisol level drops in the evening. This test measures the level of cortisol in saliva. The saliva sample must be collected in the night to see if the levels are high or low.
  - **Dexamethasone suppression test:** In this test a low dose steroid (Dexamethsone) pill will be given to the patient at late night. The blood will be tested in the next morning to check the level of cortisol. Dexamethasone drops the cortisol level. In Cushing Syndrome patient, it won't occur.
  - **Blood ACTH (Adrenocorticotropin Hormone) Test**: To confirm the tumor is the cause for Cushing Syndrome a blood test is recommended to check the level of ACTH. Low level of ACTH indicates an

adrenal tumor is likely cause and high level of ACTH indicates pituitary or ectopic tumor

- **Petrosal sinus sampling:** In this test blood samples takes from a vein near the pituitary and also from a vein away from pituitary. Patient will get a shot of corticotrophin-releasing Hormone (CRH). High level of ACTH in blood sample taken from near the pituitary vein indicates a pituitary tumor and similar level of ACTH from both the veins sample indicates an ectopic tumor.

- **Imaging studies:**
  - To visualize the tumors in the adrenal and pituitary glands imaging test like CT scan and MRI can be done.

Treatment

The treatment of Cushing Syndrome depends upon the cause of excessive or high level of cortisol.

Elaborate Treatment of Cushing syndrome

The goal of Cushing syndrome treatment is to reduce the body's high cortisol levels. The treatment options are determined by the causative factors.

Reducing Corticosteroid Use

The dosage of the medication will be gradually decreased over time if prolonged usage of corticosteroids is the cause of Cushing syndrome. Patients are not allowed to stop or reduce the dosage of corticosteroid medications by themselves. It is always advisable to gradually taper off the corticosteroid medications, as it enables the body to produce cortisol normally.

Surgery

If a tumor is the cause of Cushing syndrome, surgical removal of the tumor or removal of the adrenal gland, known as "adrenalectomy," is recommended.

Radiation Therapy

Radiation therapy is recommended for a patient whose tumor cannot be completely removed, Patient who is not surgically suitable.

Radiation can be administered in small doses using a technique known as stereotactic radiosurgery. The latter procedure involves delivering a large, one-time dose of radiation to the tumor.

Medications

Cortisol production is controlled with medications.

In people who have become very sick with Cushing syndrome, medications are sometimes used before surgery to improve signs and symptoms and reduce surgical risk. There are medications available to control excessive cortisol production by the adrenal gland. Some of which are included below:

- Ketoconazole
- Mitotane (Lysodren)
- Metyrapone (Metopirone)
- Pasireotide (Signifor)
- Osilodrostat (Isturisa)
- Mifepristone (Korlym, Mifeprex) for patient who have type 2 diabetes or glucose intolerance.

These medications can cause fatigue, nausea, vomiting, headaches, muscle aches, high blood pressure, low potassium, and swelling as side effects.

Lifestyle Modification

Dietary component is an important component of keeping cortisol levels at normal and to prevent the complications of Cushing Syndrome

- **Restrict calorie intake:** Weight gain is the main symptom of Cushing Syndrome, Have food which does not increase weight
  - *Avoid drinking alcohol:* Alcohol consumption cause increased level of cortisol
  - *Blood sugar monitoring:* Cushing Syndrome condition may lead to high blood sugar level
  - *Low sodium intake*: Hypertension or High Blood pressure is a symptom of Cushing Syndrome
- **Take enough calcium and vitamin D:** Cushing Syndrome can weaken the bones which can lead to have patient more prone to fractures

Complications

- Frequent Infections
- Heart Attack
- Stroke
- Hypertension
- Depression
- Enlargement of Tumor
- Type 2 Diabetes
- Cognitive Difficulties
- Bone Loss or Fracture

Dwarfism

The term "dwarfism" in medicine refers to small stature. It is defined as height-vertex below two standard deviations (–2 SD) or in the third percentile for a given age and sex.

As the term suggests, proportional short stature (PSS) refers to a person's proportionately short limbs and trunk. Disproportionately short stature (DSS), on the other hand,

denotes that the person's trunk or extremities are small and there is a big disparity between their sitting and standing heights.

Etiology

Either an underlying medical condition or factors leading to abnormal growth can account for the short stature. Dwarfism has several pathological causes, including:

- Familial Short Stature (FSS): Familial short stature can be identified by a favorable family history and the absence of underlying pathological causes of dwarfism.
- This pattern of small stature depends on heredity, malnutrition during pregnancy or childhood, or both.
- Deficiency of growth hormone: Lack of growth hormone is a common contributor to dwarfism. GH stimulates bone elongation and the growth of soft tissue and cartilage, its deficiency causes dwarfism.
- Achondroplasia is an autosomal dominant genetic disorder, which occurs due to the mutation in the Fibroblast growth factor receptor-3 (FGFR-3) gene. It limits the conversion of cartilage to bone.
- Genetic diseases such as Down's, Turner's, Prader-Willi syndrome can also lead to short stature
- Systemic diseases: Undernutrition, juvenile idiopathic arthritis, inflammatory bowel disease (IBD), celiac disease, chronic kidney disease (CKD) are some systemic conditions that have a secondary impact on growth.

Clinical Manifestations

- Stunted skeletal growth, marked by maximum height approximately 3 feet
- Head becomes slightly larger in relation to the body, and flat nasal bridge is evident.
- Short and wide hands and feet
- Mental activity is normal without any abnormality
- Bowing of legs that progressively worsen over time.

Extra Edge

Simmonds' Disease

Simmonds' disease is a term used to describe panhypopituitarism that results from the destruction of pituitary gland by tumors, infiltrative processes (such as lymphocytic), or trauma.

Simmonds' disease has a subclassification called Sheehan's syndrome that only occurs during the peripartum stage.

Disorders of Posterior Pituitary Gland

Posterior pituitary gland secretes the hormone like antidiuretic hormone (ADH), disorder may be there if ADH secretion is increased or decreased. Destruction of posterior pituitary gland by any disease is rare, if the gland is surgically removed, then also there may not be deficiency of hormone because hypothalamus continues to synthesize oxytocin and ADH.

Disorder of posterior lobe is known as syndrome of inappropriate ADH secretion and diabetes insipidus.

Syndrome of Inappropriate Hypersecretion of Antidiuretic Hormone

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is characterized by persistent production or activity of arginine vasopressin (AVP) in the presence of normal or elevated plasma volume.

In hospitalized patients, SIADH is the most frequent cause of euvolemic hyponatremia.

Etiology and Risk Factors

- Ectopic production of ADH as seen in brain related diseases such as stroke, head injury and meningitis
- **Drug-induced:** Some drugs of class antidepressants, anticonvulsants, antipsychotic have propensity to cause SIADH.
- **Neoplastic disorders:** Tumors of lungs (meosthelioma, small cell lung cancer), Gastrointestinal (pancreatic cancer, duodenal cancer), and genitourinary system (cancer cervix) can cause SIADH.
- **Pulmonary disease:** pneumonia, TB and cancer also increases the risk of SIADH.
- **Surgery:** ADH hypersecretion is frequently linked to surgical procedures.

Note: Mnemonic for remembering causes of SIADH

Mnemonic

MAD CHOP
M – Major Surgery
A – ADH production by tumors (Ectopic)
D – Drugs (antidepressants, psychotropics)
C – CNS disorders
H – Hormone deficiency
O – Others (Guillain-Barre Syndrome, HIV)
P – Pulmonary disorders (TB, Pneumonia)

Pathophysiology

ADH maintains plasma tonicity by altering the water balance. The Osmoreceptors in the hypothalamus detects change in the plasma osmolality. A decrease in tonicity inhibits ADH release and subsequent water retention. And, ADH release is stimulated when tonicity rises,

In SIADH, levels of ADH are high even in the presence of decreased plasma osmolality or hyponatremia, which causes excessive water absorption leading to increased blood volume.

Clinical Manifestations

Hyponatremia and decreased ECF osmolality can induce water to move into the cells, leading to cerebral edema.

- Nausea and malaise are some of the first clinical signs of acute hyponatremia, which occurs when the serum sodium concentration falls below 125 to 130 mEq/L,
- Headache, drowsiness, obtundation, and eventually seizures can develop with a more severe and sudden reduction in sodium concentration.
- If the serum sodium level drops below 115 to 120 mEq/L, coma and respiratory arrest may occur.
- Chronic hyponatremia causes the brain to adapt, and the individuals remain asymptomatic even when their serum salt level falls below 120 mmol/L. Chronic hyponatremia can lead to various non-specific symptoms such as: cramping, nausea, vomiting, abnormal gait, memory loss, and cognitive difficulties.

Diagnostic Investigations

- **History taking:** Patient should be enquired about past head injuries, smoking, weight gain, pulmonary symptoms, drug use, or substance abuse (particularly heroin).
- **Physical examination:** Physical examination should include checking intake-output, blood pressure and skin turgor. Euvolemia is often indicated by moist mucous membranes without jugular venous pulsation or edema.

Investigations for SIADH

- **Serum osmolality and serum sodium:** Hyponatremia (i.e., serum Na^+ <135 mmol/L) is a defining feature of SIADH.
- **Urinary osmolality:** The urine osmolality is typically submaximally diluted (i.e., >100 mOsm/kg) in SIADH patients.
- **Imaging studies:** Chest radiography can reveal an underlying pulmonary etiology of SIADH. In some cases, A head MRI or CT scan may reveal the signs of cerebral edema or other cerebral conditions such as brain tumor.

Complications

- Brain Herniation
- Decreased Consciousness

Management

Treatment for SIADH includes correcting underlying problems such hypothyroidism, lung infection, or CNS infection as well as maintaining appropriate salt levels. More than 130 mEq/L is the desired sodium correction level.

Emergent Care

- For people with moderate-to-severe hyponatremia that has a recorded duration of less than 48 hours, urgent therapy should be carefully considered.
- Serum Na^+ levels should be maintained at a maximum level of 125–130 mEq/L, by raising serum sodium by 0.5–1 mEq/h.
- Along with treating hyponatremia, caution should be taken to avoid developing severe hypokalemia.

Acute Setting

Patients presenting with moderate symptoms such as confusion, delirium, disorientation, nausea, and vomiting, should be treated with 3% hypertonic saline, loop diuretics with saline, vasopressin-2 receptor antagonists, and water restriction.

Chronic Setting

Fluid restriction and Vasopressin receptor antagonists are the main treatment choices for asymptomatic patients with persistent SIADH. Other therapy approaches include chronic loop diuretics with increased salt intake, urea, mannitol, and demeclocycline.

Diabetes Insipidus

Diabetes insipidus (DI) is characterized by the passing of significant amounts of diluted urine (>3 L/24 h). It is primarily caused due to the deficiency in arginine vasopressin.

There are two types of diabetes insipidus, i.e., central and nephrogenic diabetes insipidus.

Nephrogenic DI is caused by kidney resistance to the hormone arginine vasopressin (AVP), whereas central DI is caused by a lack of AVP hormone in the pituitary gland or the hypothalamus.

Etiology and Risk Factors

- Malignant and benign tumor of brain such as craniopharyngiomas
- Head injury or cranial surgery
- Subarachnoid hemorrhage
- Hypoxic brain injury
- Multiple drug treatments, including lithium, antibiotics, antifungals, and antineoplastic agents.
- Familial and hereditary inheritance as evident in case of nephrogenic DI.

Pathophysiology

Central DI

Traumatic or pathological injuries to the posterior pituitary gland or hypothalamus results in damage to hormone-

secreting cells in those regions, which interferes with the normal secretion and release of ADH. A diuretic phenomenon results from the renal collecting ducts' loss of capacity to accomplish enough water reabsorption, which is necessary for maintaining the body's volume.

Nephrogenic DI

The malfunction of the kidneys' ADH receptors is the cause of nephrogenic DI. AVPR1 and AVPR2 are two receptors that often react to rising ADH levels in the bloodstream. Vasoconstriction and prostaglandin release are mediated by AVPR1, whereas antidiuretic response and some coagulation factors (factor VIII and von Willebrand's factor) are mediated by AVPR2 receptors.

Clinical Manifestations (Fig. 8.26)

- The three main symptoms of diabetes insipidus are polyuria, polydipsia, and nocturia. The daily urine volume varies greatly from patient to patient, ranging from 3 to 20 L, but it is generally consistent for each patient.
- Children may have severe dehydration, constipation, vomiting, fever, irritability, failure to thrive, and growth retardation as nonspecific symptoms.
- Patients with diabetes insipidus may also experience weakness, lethargy, fatigue and myalgias.
- The findings of the exam may be completely normal.

Note: Mnemonic for manifestations of DI

Mnemonic

DDD
D – Diabetes insipidus
D – Deficient ADH (Central DI) or ADH Doesn't work (Nephrogenic DI)
D – Dilute urine (<300 mOsm/L)

Polyuria

- Excretion of large quantity of dilute urine with increased frequency of voiding is called polyuria. Daily output is 4 to 12 liters.
- Due to absence of ADH, the epithelial cells of distal convoluted tubule in the nephron and the collecting duct of the kidney becomes impermeable to water

Polydipsia

Intake of excess water. Because of polyuria, thirst center in hypothalamus stimulate which results in intake of large quantity of water.

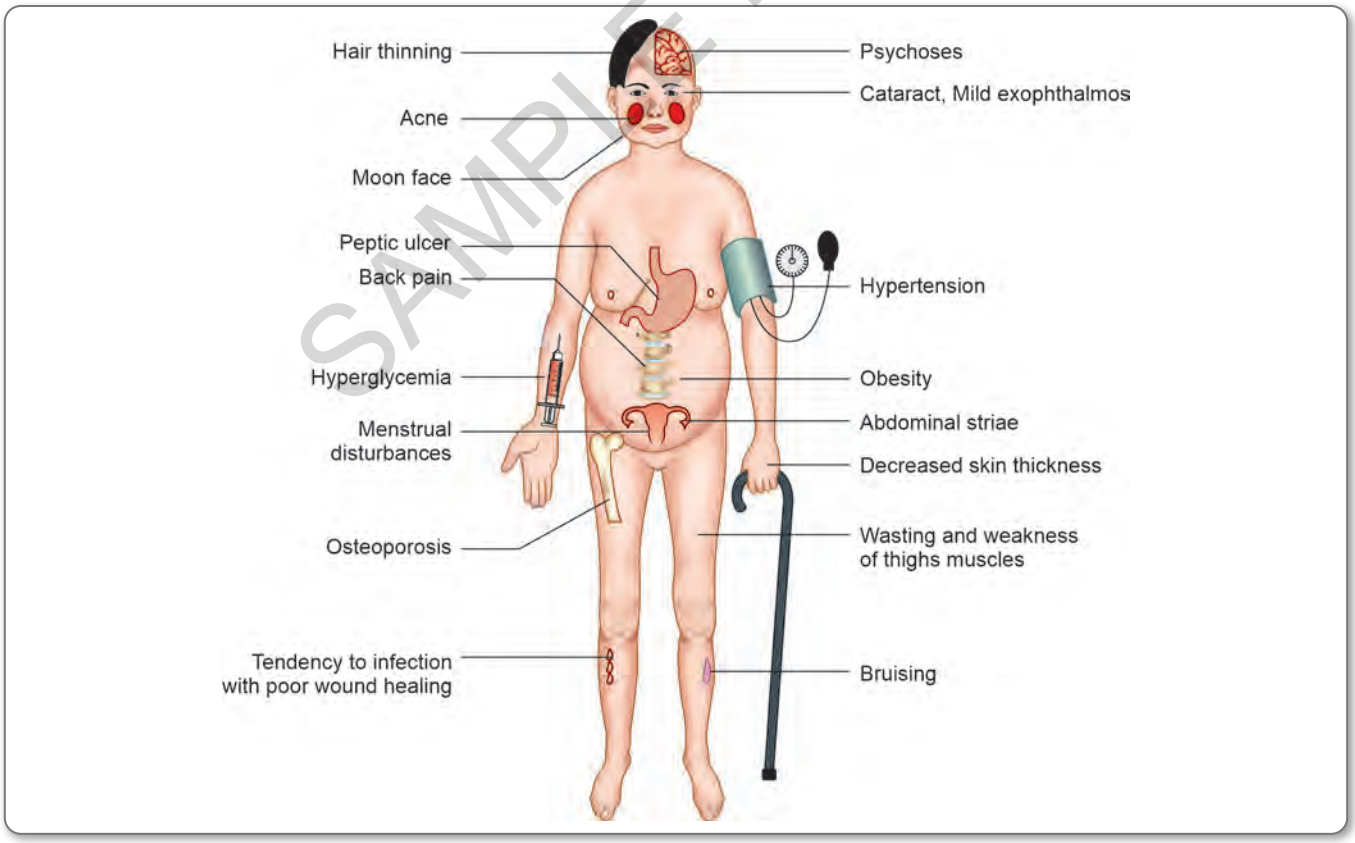

Fig. 8.26: Signs and symptoms of diabetes insipidus

Dehydration

In some cases, the thirst center in the hypothalamus is also affected by the lesion. Therefore, water intake decreases in these patients and, the loss of water through urine is not compensated.

Diagnostic Investigations

- 24-hour urine collection (volume)—typically 3–20 L of urine per day
- Urine osmolality—low results <300 mmol/kg
- Serum osmolality—normal or elevated
- Urinalysis—to rule out other causes of manifestations such as Diabetes mellitus (DM)
- Serum glucose—for determining coexisting

Water Deprivation Test/Dehydration Test

This is done to differentiate between central and nephrogenic DI. If the response occurs to synthetic form of AVP (i.e., desmopressin), the type is central DI (marked by reduction in urine output and increase urine osmolality of >50%.

Management

Hypernatremia management

- IV hypotonic fluids (5% dextrose and 0.45% sodium chloride)
- Frequent monitoring of electrolytes

Central diabetes insipidus

- Desmopressin
- Oral or IV fluid replacement (only in acute settings)

Nephrogenic diabetes insipidus

- Maintenance of adequate per oral fluid
- High dose desmopressin
- Sodium restriction
- Hydrochlorothiazide
- Treat underlying cause: Such as uncontrolled diabetes, chronic hypercalcemia or hypokalemia

Nursing Management

Monitor intake and output, assess for thirst, urination, assess urine specific gravity, teach the patient and family.

Disorders of Both Anterior and Posterior Pituitary

Froehlich Syndrome

Adiposogenital dystrophy, another name for the constellation of endocrine disorders known as Froehlich syndrome, is thought to be the result of injury to the hypothalamus, a region of the brain that connects the neurological system to the endocrine system through the pituitary gland.

Symptoms of the Froelich syndrome include increased or excessive eating that results in obesity, undersized testes, and a delayed onset of puberty. Delays in physical development and the emergence of secondary sexual traits are also frequent in children with Froehlich syndrome.

Pituitary Tumor

The majority of pituitary tumors are benign. However, a tumor that develops on or close to the pituitary gland may:

- Cause changes in hormone production leading to weight gain, excessive or stunted development, high blood pressure, decreased sex drive, or mood swings.
- Tumor mass can also compress optic nerve leading to vision loss and frequent headaches.
- Releasing excess of one or more hormone.
- Not releasing any hormones
- Causing pressure on nearby structures (Example- blurred vision due to the pressing on the nerve of the eye

Pituitary gland diseases are diagnosed using brain imaging (CT and MRI scan) and blood studies. Treatment for pituitary abnormalities depends upon the underlying cause. Hormone insufficiency may need to be corrected with lifelong hormonal supplements, while abnormal cancerous growth requires resection.

Nelson Syndrome

Dr Don J Nelson first reported Nelson syndrome (NS), also known as post adrenalectomy, in 1958. This is a potentially fatal condition that develops following a therapeutic bilateral adrenalectomy. The time between bilateral adrenalectomy and NS diagnosis ranges from 0.5 to 24 years. The loss of feedback inhibition of the hypothalamic-pituitary-adrenal axis is thought to lead to the development of an adrenocorticotropic hormone (ACTH) secreting pituitary tumor. The surgical removal of the tumor is the recommended treatment for Nelson syndrome.

DISORDERS OF THYROID GLAND

HYPOTHYROIDISM

Introduction

Hypothyroidism is a deficiency of thyroid hormone resulting in slowed body metabolism, decreased heat production and decreased oxygen consumption by the tissues.

Incidence and prevalence: Prevalence rates are greater in elderly women (10%) than in elderly men (2.3%). Even though highest incidence is in adults between 30–65 years, it is seen in newborn also.

Definition: Hypothyroidism is a condition in which the thyroid gland does not make enough thyroid hormone. The most common cause of hypothyroidism is inflammation of the thyroid gland, which damages the gland's cells.

Types of Hypothyroidism

- **Primary hypothyroidism:** TH levels are low and TSH levels are elevated indicating that pituitary is attempting to stimulate the secretion of thyroid hormones but the thyroid is not responding. It is most frequently autoimmune in origin but can also be related to iodine deficiency.
- **Secondary hypothyroidism**: Secondary hypo-thyroidism, which occurs in only 5% of cases, is caused by a failure of the pituitary gland to stimulate the thyroid gland or a failure of the target tissues to respond to the thyroid hormones resulting in decreased TSH levels. For example, the pituitary gland and hypothalamus produce hormones that trigger the release of thyroid hormone. A problem with one of these glands can make your thyroid underactive.
- **Tertiary or central hypothyroidism**: It develops if the hypothalamus cannot produce TRH and subsequently does not stimulate pituitary to secrete TSH. It may be due to a tumor, other destructive lesion in hypothalamic region. When this occurs, both TSH and TH levels are again low in serum.
- **Hashimoto's disease**: comes under primary hypothyroidism classified as an autoimmune disorder in which antibodies develop that destroys thyroid tissue. Functional thyroid tissue is replaced with fibrous tissue and TH level decreases. This decreasing level of TH prompts gland to enlarge to compensate, causing a goiter.
- **Cretinism:** It is caused by thyroid hormone deficiencies during fetal or early neonatal life. It can be caused by maternal iodine deprivation or congenital thyroid abnormalities.
- **Iatrogenic hypothyroidism:** It is caused in process of treating hyperthyroidism with radioactive iodine treatment and radiation therapy results in over destruction of thyroid tissue.

Causes of Hypothyroidism (Table 8.6)

TABLE 8.6: Causes of hypothyroidism

- **Autoimmune or Hashimoto's thyroiditis:** In which the immune system attacks the thyroid gland, is the most common example. With Hashimoto's, your body produces antibodies that attack and destroy the thyroid gland. Thyroiditis may also be caused by a viral infection.
- **Postpartum thyroiditis:** Some women develop hypothyroidism after pregnancy

Contd...

- **Radiation therapy:** Radiation to the neck as in case of lymphoma damages cells of the thyroid gland and predisposes a patient to develop hypothyroidism.
- **Radioactive iodine treatment:** Radioactive iodine therapy is prescribed to the patients with overactive thyroid gland. It can destroy the cells of thyroid glands and can predispose a person for hypothyroidism.
- **Medications:** Certain drugs used to treat cancer, psychological disorders, and heart diseases can have an impact on thyroid hormone production. These include lithium, interferon alpha-amiodarone (Cordarone), and interleukin-2.
- **Thyroid surgery:** Following a complete thyroidectomy, patients develop hypothyroidism and need lifelong thyroid hormone medication.
- **Iodine deficiency:** Iodine deficiency impairs thyroid hormone production, which in turn has an impact on the developing brain, liver, kidney, heart, and muscle. Iodine deficiency disorders arise when iodine levels in the soil are low, food products have low iodine concentrations and intake of iodine is poor. The thyroid gland may no longer be able to produce enough thyroid hormone if iodine requirements are not met.
- **Problems with the thyroid at birth:** Some infants could be born with a thyroid gland that did not function properly or developed inadequately. The condition is known as congenital hypothyroidism.
- **Pituitary gland damage or disorder:** A pituitary gland disease can prevent the thyroid hormone from being produced. Thyroid-stimulating hormone (TSH), which is produced by the pituitary gland, has a stimulating effect on the thyroid gland.
- **Disorder of the hypothalamus:** If the hypothalamus does not produce enough thyroid-releasing hormone (TRH). TRH influences thyroid-stimulating hormone (TSH) secretion, a hormone produced by the pituitary gland.

Clinical Manifestations (Fig. 8.27)

Fatigue/Feeling Tired

Fatigue is one of the most common symptoms of hypothyroidism. Thyroid hormone controls energy balance and

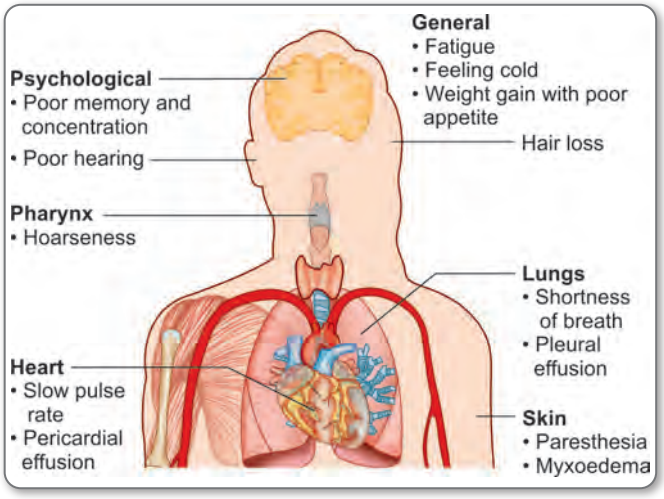

Fig. 8.27: Signs and symptoms of hypothyroidism

metabolism and low thyroid hormone level cause exhausted and sluggish. Patient with hypothyroidism feel un-rested, even though they may be sleeping more.

Weight Gain

Unexpected weight gain is also a symptom of hypothyroidism. When thyroid levels are low, metabolic changes occurs and instead of burning calories for growth and activity, the amount of energy use at rest decreases and as result it tends to store more calories from the diet as fat. The low thyroid hormone levels cause weight gain. Hypothyroidism signals to eat more and burn few calories which lead to weight gain.

Feeling Cold

Heat is a byproduct of burning calories. In cases of hypothyroidism, basal metabolic rate decreases, reducing the amount of heat generate.

That's why low levels of thyroid hormone cause to feel colder. Low thyroid hormone slows body's heat production and cause feeling cold.

Weakness and Aches in Muscles and Joints

Low thyroid hormone flips the metabolic switch toward catabolism and during catabolism, muscle strength decreases, potentially leading to feelings of weakness. Low levels of thyroid hormone slow down metabolism and cause painful muscle breakdown.

Hair Loss

The hair follicles are regulated by thyroid hormone. Low thyroid hormone causes hair follicles to stop regenerating and affects rapidly growing cells (hair follicles), resulting in hair loss.

Itchy and Dry Skin

Like hair follicles, skin cells are characterized by rapid turnover. In Hypothyroidism the dead skin may take longer to shed, leading to flaky, dry skin. Myxedema is specific to thyroid problems, which shows dry skin and red swollen rash on skin.

Feeling Depressed

Hypothyroidism cause depression and anxiety, the cause is still unknown but it is recommended to consult to a physician or therapist.

Trouble Concentrating or Remembering

Many patients with hypothyroidism complain of mental concentrating. Sudden or severe episodes of difficulties in memory or concentration could be a signal of hypothyroidism.

Constipation

Low thyroid levels put the brakes on colon and cause constipation.

Heavy or Irregular Periods

Thyroid hormone interacts with the hormones that control the menstrual cycle, and abnormal levels of it can disrupt their signals.

Risk Factor

- Gender- Women are more likely to develop hypothyroidism than men.
- Type I Diabetes, Multiple Sclerosis, Rheumatoid arthritis, Celiac Disease, Pernicious Anemia or Vitiligo.
- Genetic defects resulting in faulty metabolism of iodine and ingestion of large amount of medical goitrogens (Adrenergic antagonists, glucocorticoids, dopamine, lithium, rifampin, carbamazepine, propylthiouracil and thiocarbamides, aminothiazonate) or nutritional goitrogens (cabbage, soybean, peanuts, peaches, peas, strawberries, spinach and radishes).
- Family history of thyroid disease.
- Iodine deficient area (endemic goiter area).
- Pregnant and lactating women.
- Elderly men receiving aminoglutethimide (1,000 mg day or greater) for prostate cancer.

Pathophysiology

Iodine is required for the synthesis and secretion of thyroid hormone. The hypothalamus regulates the pituitary secretion of TSH by negative feedback system. The hypothalamic–pituitary–thyroid axis plays a key role in maintaining thyroid hormone levels within normal limits. Production of TSH by the anterior pituitary gland is stimulated in turn by thyrotropin-releasing hormone (TRH), released from the hypothalamus (Fig. 8.28). Production of TSH and TRH is decreased by thyroxine by a negative feedback process.

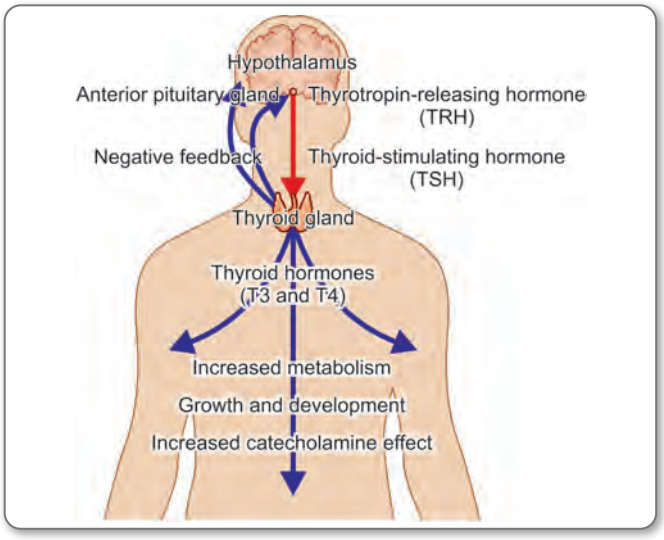

Fig. 8.28: Normal physiology of thyroid hormone production