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ENDOCRINOLOGY

[SAMSONPLAB ACADEMY](#)

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1. DIABETES MELLITUS – This is high serum blood glucose

DM results from lack or reduced effectiveness of endogenous insulin.

It is imperative that a diabetic person having hypertension should have it well controlled.

TYPE 1 DIABETES

- Usually juvenile onset
- Common in young patients
- It is due to absolute deficiency of insulin
- An autoimmune condition in which there is destruction of the B cells of the pancreas
- **Symptoms:** Polyuria, Polydipsia, Weight loss, Diabetic Ketoacidosis
- First presentation can be Diabetic Ketoacidosis
- There could be history of other autoimmune conditions like Addison's disease, Thyroid disease and Pernicious Anaemia
- **Antibodies** e.g. anti-glutamic acid decarboxylase (GAD) antibodies and islet cell antibodies

TYPE 2 DIABETES

- Usually occurs in the adults mostly in Asian men and above the age of 40 years, most are obese
- Is due to insulin resistance and relative insulin deficiency
- Often it is asymptomatic and may first present with complications like diabetic retinopathy, nephropathy and neuropathy
- **RISK FACTORS:** Pregnancy, obesity, polycystic ovarian syndrome (PCOS), renal failure, lack of exercise

DIAGNOSIS: To make the diagnosis of diabetes, you need to consider the following:

1. If the patient is asymptomatic do the blood tests twice, either:

- Fasting glucose >7.0 OR
- Random blood glucose > 11.0 confirms the diagnosis

Fasting glucose <6.1 is normal

Fasting glucose $6.1-7$ is Impaired fasting glucose

Fasting glucose ≥ 7.1 is Diabetes Mellitus

2. If the patient is symptomatic, perform one of the following tests once.

- Fasting glucose ≥ 7.0 or
- Random blood glucose ≥ 11.1

Do the Oral Glucose Tolerance Test (OGTT) if there is impaired fasting glucose. This is done with 75mg of sugar and blood glucose is measured after 2 hrs.

- Glucose <7.7 is normal
- Glucose $> 7.8-11$ is Impaired oral glucose tolerance
- Glucose ≥ 11.1 confirms Diabetes

NB.

- Only venous blood glucose is used for making the diagnosis.
- Capillary blood glucose is only used for monitoring.
- For monitoring blood glucose control you monitor glycosylated haemoglobin (HbA1c). Normal levels should be less than or equal to 6.1.

Treatment:

Impaired Glucose Tolerance Test

This is treated with diet and exercise.

Type 1 Diabetes Mellitus

In type 1 diabetes Insulin is always used but in different regimes e.g.

1. Twice a day if patient has a regular life style
2. Four times a day plus long acting at bedtime if a patient has variable activity e.g. exercising.
3. Once a day in the morning long-acting when switching from oral hypoglycaemic to Insulin

Type 2 Diabetes Mellitus

Stage 1 (Newly diagnosed):

- Diet and exercise

Stage 2 (Not controlled by diet and exercise alone):

- Diet and exercise +
- Oral hypoglycaemic

Stage 3 (Not controlled by above treatment):

- Diet and exercise +
- Oral hypoglycaemic +

- **Insulin**

Oral hypoglycaemic medication

1. **Biguanides** e.g. Metformin
 - Especially good for patients who are obese. It is always the first choice. Increases insulin sensitivity
 - Weight loss
 - Causes lactic acidosis
 - Does not cause hypoglycaemia
 - If glucose is not controlled add Sulfonylurea.

SE: nausea, vomiting, lactic acidosis.

1. **Sulfonylureas** e.g. Glibenclamide or Gliclazide
 - They increase insulin sensitivity

SE: Hypoglycaemia

1. **Thiazolidines** e.g. Glitazones- Pioglitazone and Rosiglitazone
 - They are used if patient not tolerant to either metformin or Sulfonylurea.
 - They cause weight gain
 - Do not cause hypoglycaemia

COMPLICATIONS OF DIABETES MELLITUS

A. Acute complications

- a. Hypoglycaemia
- b. Diabetic ketoacidosis (DKA)
- c. Hyperglycaemic Hyper-osmolar non ketotic coma (HONK)

A. Chronic complications

- a. Microvascular = diabetic retinopathy, diabetic nephropathy, diabetic neuropathy, autonomic neuropathy and somatic neuropathy.
- b. Macrovascular = Stroke, IHD, Intermittent Claudication, (Peripheral vascular disease) as a result of atherosclerosis

ACUTE COMPLICATIONS

1. **Hypoglycaemia** is glucose less than 3 mmol/l

Symptoms:

Pale, sweating, tremor, jittering, aggressive, tachycardia, seizure, coma.

Loss of consciousness and sweating = hypoglycaemia until proven otherwise.

Treatment

1. **If the patient is conscious give an oral sugary drink.**
2. **If the patient is unconscious then treat as follows:**
 - **1st choice is 10% glucose/dextrose**
 - **2nd choice 50% glucose/dextrose**
 - **3rd choice is Glucagon; disadvantage is that it does not work if there is alcohol in the blood or in patients with anorexia nervosa. This is because glucagon acts by converting glycogen into glucose. Glycogen is the storage form of glucose in the liver.**

CAUSES OF HYPOGLYCAEMIA

1. Insulin overdose especially in type 1 Diabetes
2. Glibenclamide and gliclazide in type 2 Diabetes
3. Insulinoma = it is a benign tumour of the pancreas which produces insulin and it causes hypoglycaemia. Usually every time a patient misses meal he loses consciousness or he will have fit. It occurs as part of MEN-1.

Investigation: C-peptides and Insulin level in the blood.

2. Diabetic Ketoacidosis (usually it has gradual onset)

- Occurs only in type 1 Diabetes
- The criteria is pH < 7.3, HCO₃ < 15, ketones in urine or capillary
- ABGs will show metabolic acidosis
- **Precipitating factors:** infection, surgery, MI, Sepsis, UTI, Gastroenteritis and Pneumonia

Symptoms/Clinical features:

Young patient, weight loss, polydipsia, dehydration, lethargy, anorexia, vomiting, abdominal pain, coma, usually there is progressive drowsiness.

On examination patient will have Kussmaul respiration, which is deep and sighing breathing. Fast respiratory rate e.g. 40/min

Treatment:

- **The initial treatment is always IV fluids.**

- Use normal saline until capillary blood glucose is 10 mmol/L then switch to dextrose 5%.
- Give insulin 0.1 units per kg
- Give K⁺ depending on the level. If K⁺ is >5 mmol/L don't give any.

3. Hyperglycaemic Hyper-osmolar Non-Ketotic coma (HONK)

Occurs only in type 2 Diabetes

Usually elderly patient and this could be the first presentation.

Criteria for diagnosis:

1. No acidosis
2. Glucose > 35 mmol/l
3. Plasma osmolarity >350 mosm/kg
4. Ph is normal
5. No ketones in the urine

Symptoms:

1. Dehydration
2. Lethargy, weakness, polyuria, polydipsia, progressive loss of consciousness over days

Treatment: IV fluids (normal saline) and Insulin

CHRONIC COMPLICATIONS OF DIABETES

1. MACROVASCULAR COMPLICATIONS

a. Heart - Myocardial Infarction: Usually patient has silent Myocardial Infarction (MI) or atypical chest pain due to neuropathy.

b. Brain - TIA/Stroke due to atherosclerosis.

c. Peripheral vascular disease - atherosclerosis leads to intermittent claudication. Pain comes on walking and after a short rest it goes away.

2. MICROVASCULAR COMPLICATIONS

Diabetic retinopathy

- A. Background** - Micro-aneurysms, dot and blot haemorrhages and hard exudates
- B. Pre-Proliferative** - Micro-aneurysms, dot and blot haemorrhages, hard exudates and soft exudates (cotton wool spots)
- C. Proliferative** - Micro-aneurysms, dot and blot haemorrhages, soft and hard exudates, new vessel formation (neovascularization)
 - New vessel formation leads to bleeding which may cause retinal detachment which comes as a sudden loss of vision and the patient complains as a curtain coming down. Cataract formation is earlier.

Diabetic Maculopathy - This is when changes develop in the macula

Vitreous haemorrhage - This is when there is bleeding in the vitreous. The patient usually complains of floaters.

Diabetic Nephropathy

- Micro-albuminuria- loss of >300mg/day of protein.
- It is a big risk factor of IHD and stroke therefore it needs treatment.
- In people who are diabetic, the target BP is ≤ 130/80 and if there is micro-albuminuria then target BP ≤125/75.

Micro-albuminuria leads to diabetic nephropathy and eventually renal failure if not treated. In renal failure insulin sensitivity increases and insulin metabolism decreases therefore insulin needs to be reduced to avoid hypoglycaemic attacks.

Diabetic Neuropathy

1. Peripheral neuropathy (somatic neuropathy) is usually symmetrical in a form of gloves and socks.
2. Mono neuropathy e.g. 3rd, 4th and 6th nerve palsy.
3. Autonomic neuropathy will cause vasovagal syncope, diarrhoea, postural hypotension or urinary retention.
4. Amyotrophy – progressive wasting and weakness of muscles especially the quadriceps muscles.

2. PITUITARY GLAND

Anterior Pituitary produces Growth hormone (GH), Gonadotropins: Follicle stimulation hormone (FSH) & Leutenizing hormone (LH), Prolactin (PRL), Thyroid stimulating hormone (TSH), Adrenocorticotrophic hormone (ACTH)

Posterior Pituitary stores Oxytocin and ADH (Anti-diuretic hormone)

- Oxytocin acts on the uterus and causes contraction.
- ADH acts on the kidneys and cause urine retention.

NB: The ANTERIOR PITUITARY PRODUCES hormones and the POSTERIOR PITUITARY STORES hormones.

Hypopituitarism

Hormones are affected in this order: GH, FSH & LH, PRL, TSH, ACTH

Causes are at 3 levels:

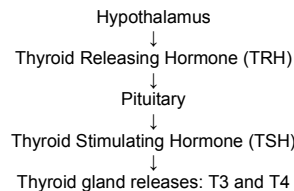
1. **Hypothalamus:** Kallman's syndrome (isolated FSH LH deficiency with anosmia and colour blindness), tumour, inflammation, infection
1. **Pituitary stalk:** Trauma, surgery, compression by a mass lesion (eg. due to a craniopharyngioma), carotid artery aneurysm
1. **Pituitary:** Tumour, irradiation, inflammation, autoimmunity, ischaemia (eg. Sheehan's syndrome due to post partum haemorrhage)

Clinical features: depends on the hormone that is deficient and the underlying cause.

Investigations: Check for the specific hormones and look for the underlying cause eg. MRI for pituitary tumour

Treatment: Hormone replacement and treatment of the underlying cause.

3. HYPERTHYROIDISM



SYMPTOMS OF HYPERTHYROIDISM

1. Weight loss, tachycardia, diarrhoea, oligomenorrhoea, irritability, heat intolerance, tremors, sweating and weight loss despite increased appetite, atrial fibrillation/sinus tachycardia
2. Typical signs of **Graves disease** are exophthalmus, ptosis, diplopia, lid retraction, lid lag

CAUSES:

1. GRAVES DISEASE

It is an autoimmune disease. Antibodies resembling TSH are formed and act on the thyroid and stimulate production of T3 & T4. It is associated with other autoimmune disease like type 1 diabetes, Addison's disease, Vitiligo. There is diffuse enlargement of the thyroid gland. There is bruit and eye signs e.g. diplopia, exophthalmus.

Treatment: Carbimazole. In pregnancy use propylthiouracil. Give beta blockers if no contraindications like asthma.

1. TOXIC ADENOMA

It is a benign tumour of the thyroid gland and it produces thyroxine. It is a solitary adenoma, which means there will be a lump in the thyroid which moves on swallowing.

Treatment is radio-iodine.

3. TOXIC MULTINODULAR GOITER

There are multiple nodules.

Treatment is Carbimazole and radiotherapy

1. SUBACUTE THYROIDITIS - This is also known as De-quervain's thyroiditis

The cause is viral infection ie. Upper Respiratory Tract Infection. The thyroid is usually painful and enlarged.

Treatment is analgesia or observation

1. MEDICATIONS: Amiodarone, thyroxine & lithium

- For **amiodarone**, the patient will be on treatment for arrhythmia (SVT and VT)
- For **thyroxine**, it is usually patients with hypothyroidism and on replacement therapy with levothyroxine
- For **Lithium** these are usually patients being treated for bipolar mood disorder

1. ECTOPIC TISSUE- this is thyroxine produced by anywhere else other than the thyroid gland.

INVESTIGATIONS:

1. T3, T4, TSH
2. TSH Receptor antibodies

3. If there is a mass in the neck then USS: if the mass is solid then do FNAC and if the mass is cyst then do surgical removal.
4. Isotope scan=to decide if it is a hot nodule or cold nodule.

Hot nodule - usually indicates a benign adenoma. It accumulates iodine as it manufactures thyroxine.

Cold nodule is usually cancer. It does not take up the contrast.

SUBCLINICAL HYPERTHYROIDISM

This hyperparathyroidism with low TSH or symptoms but normal T3 & T4.

Treatment is observation

Medical treatment is needed only if TSH 0.1 or symptoms of AF, weight loss

Treat with carbimazole if treatment required

4. HYPOTHYROIDISM

CAUSES:

1. **Hashimoto's Disease**
(Thyroid is diffusely enlarged)
It's an autoimmune disease and is associated with pernicious anaemia, Diabetes mellitus type 1, Addison disease. Antibodies: anti-peroxidase, Anti-thyroglobulin, anti-microsomal antibodies (thyroid gland is small)
1. **Primary Atrophic Hypothyroidism**
Diffuse infiltrate, which leads to atrophy of the thyroid. It is an autoimmune disease. There is no goitre.
1. **Iodine Deficiency**
Common in Africa where water is not iodized.
1. **Thyroidectomy**
1. **Radio-iodine Therapy**
1. **Medications**
Carbimazole, Lithium (do TFTs and U&E), Amiodarone

SYMPTOMS OF CLINICAL HYPOTHYROIDISM

1. Weight gain, bradycardia, constipation
2. Cold intolerance, Menorrhagia, tiredness, hoarseness, dementia
3. Toad like face, dry skin
4. Goitre
5. Cholesterol raised.

SECONDARY HYPOTHYROIDISM

The cause is low TSH due to problems in the pituitary.

TERTIARY HYPOTHYROIDISM This is due to low TRH in the hypothalamus.

Treatment: Levothyroxine

SUBCLINICAL HYPOTHYROIDISM

High TSH, normal T3 & T4.

Treatment is observation, only treat if TSH > 10 or previous disease or other associations like vitiligo, DM type 1, pernicious anaemia or if there are positive antibodies. Use thyroxine if **treatment** is required.

SUBCLINICAL THYROID DISEASE

TSH High	T3 and T4 Normal	S u b c l i n i c a l Hypothyroidism
TSH Low	T3 and T4 Normal	S u b c l i n i c a l Hyperthyroidism
TSH High	T3 and T4 Low	C l i n i c a l Hypothyroidism
TSH Low	T3 and T4 High	C l i n i c a l Hyperthyroidism

5. PARATHYROID GLAND

1. Produces Parathormone, the main function of which is to increase calcium in the blood.
2. Parathormone increases the production of active vitamin D and in turn vitamin D does the following actions.
 - Increases reabsorption of calcium from the kidney
 - Increases absorption of calcium from the gut
 - Increases release of calcium in from the bones.

THE OVERALL EFFECT OF PARATHORMONE IS TO INCREASE CALCIUM IN THE BLOOD.

HYPERPARATHYROIDISM

1. PRIMARY HYPERPARATHYROIDISM

- The commonest cause of hyperparathyroidism is the adenoma of the parathyroid, usually solitary adenoma.
- The second cause is hyperplasia of the parathyroid.

SYMPTOMS OF HYPERPARATHYROIDISM are mainly due to **hypercalcaemia**. These are weakness, tiredness, depression, polyuria, polydipsia, confusion, thirst and abdominal pain and constipation.

Parathyroid adenoma is usually associated with MEN1 (Multiple Endocrine Neoplasia). MEN syndrome consists of

MEN 1

Pancreas tumour =gastrinoma
Parathyroid adenoma
Pituitary adenoma

MEN 2a

Thyroid tumour
Adrenal adenoma
Parathyroid adenoma

MEN 2b

Thyroid
Adrenal
Parathyroid
Mucosal neuromas

Zollinger-Ellison disease is multiple ulcers in the stomach, duodenum and small intestine, which are poorly responsive to PPI and caused by Gastrinomas occurring as MEN 1.

INVESTIGATIONS FOR HYPERPARATHYROIDISM

1. Serum Calcium
2. Parathyroid level
3. Bone scan for osteoporosis
4. USS of the parathyroid and thyroid

Treatment: SURGERY

2. SECONDARY HYPERPARATHYROIDISM

Causes:

1. Deficiency of vitamin D
2. Chronic renal failure→ Active vitamin D is formed in the kidney.
3. Malabsorption

Treatment: Active vitamin D and calcium.

HYPOPARATHYROIDISM

CAUSES:

1. Thyroidectomy: Usually during thyroidectomy the parathyroid glands are removed as well.
2. Symptoms are those of hypocalcaemia ie. tetany and peri-oral parasthesia

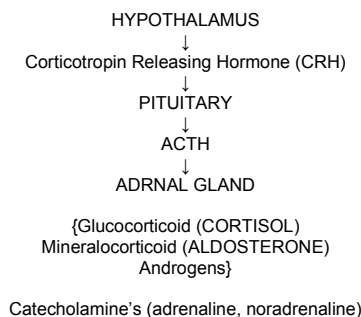
Chovestek sign- when tapping on the angle of the jaw there is twitching of the muscles of the face.

Trousseau's sign- when you tie the BP cuff on the arm there is flexion of the forearm and fingers. This sign is also called carpal pedal sign.

Treatment:

1. **Calcium Gluconate intravenously if severe**
2. **Calcium supplements if mild (Oral Ca tablets)**

1. ADRENAL GLANDS



DYSFUNCTION OF ADRENAL GLANDS

A. HYPO FUNCTION OF THE ADRENAL GLANDS

Addison's Disease - low production of cortisol mainly due to autoimmune disease and infection e.g. tuberculosis

A. HYPER FUNCTION OF THE ADRENAL GLANDS

1. **Pheochromocytoma**- tumour of the adrenal glands from the medulla.
2. **Conn's disease**- adenoma of the adrenal cortex producing aldosterone
3. **Cushing's syndrome**- see below
4. **Virilization**- increased production of the androgens

CUSHING'S SYNDROME

This is excess of cortisol from any cause

CAUSES:

1. **Cushing disease**
This is high cortisol due to pituitary adenoma
This is the commonest cause
Cushing disease is when the tumor is located in the pituitary and produces high ACTH which stimulates the adrenal gland to produce high cortisol
1. **Adenoma of the hypothalamus**
This leads to high production of corticotrophin releasing hormone- high production of ACTH leads to high production of cortisol in the adrenal glands.
1. ACTH produced by lung cancer usually caused by **small cell lung cancer**
1. **Iatrogenic** i.e. patient on treatment for Addison's disease or asthma or COPD.
1. **Adrenal adenoma** - this tumour of the adrenal glands.

Symptoms of Cushing Syndrome:

1. Weight gain
2. Mood changes
3. Central obesity
4. Acne
5. Amenorrhea or irregular menstrual
6. Hirsutism
7. Moon face
8. Buffalo hump
9. Impaired glucose tolerance test
10. Hypertension
11. Abdominal striae
12. Acanthosis Nigricans

DIAGNOSIS

- 1st LINE INVESTIGATIONS (SCREENING TEST)
Overnight Dexamethasone suppression test or 24 hour urinary free cortisol.
- 2nd LINE INVESTIGATION (CONFIRMATION TEST)
If any of 1st line test is positive go for 2nd line.
Which are 48hr Dexamethasone suppression test OR Midnight cortisol/diurnal cortisol
- 3rd LINE INVESTIGATION (LOCALIZATION TEST): To find where is the lesion.

Plasma ACTH: It is usually not detectable in blood.

- A. **If it is increased or detectable**- (May be Ectopic or may be Pituitary cause). Perform high dose Dexamethasone suppression test.
 1. If cortisol is suppressed, the diagnosis is Cushing disease. Do MRI of the pituitary because the most likely locations the pituitary gland.
 2. If cortisol is not suppressed, the diagnosis is likely to be due to an ectopic tumour. Do CT scan to locate the carcinoid tumour.

B. **Decreased or undetectable** - do CT Scan of adrenal glands. If no mass is visible on the CT scan then perform Adrenal Vein Sampling.

TREATMENT

- Surgery
- If iatrogenic - Remove the cause.

ADDISON'S DISEASE- Low Cortisol

Causes

1. TB (Most common in developing world)
2. Autoimmune (Commonest Cause)
3. Metastasis
4. Steroid
5. HIV
6. Waterhouse Friderichsen Syndrome (Haemorrhage in the adrenal gland if patient has meningococemia)

Symptoms

1. Fatigue
2. Abdominal Pain
3. Nausea
4. Vomiting
5. Hypotension (hyperkalemic hypotension)
6. Weight loss
7. Anorexia
8. Diarrhoea
9. Constipation
10. Hyperpigmentation
11. Vitiligo

INVESTIGATIONS

Short ACTH Stimulation test (Synacthen test) - Definitive Investigation.

Other investigations: U&E = K+ increased, Na+ decreased, Glucose decreased.

RISK FACTORS

1. Surgery
2. Infection
3. Sepsis
4. Trauma

TREATMENT

- **Replace steroids**
- **Hydrocortisone**
- **If Postural Hypotension- Fludrocortisone**

CONGENITAL ADRENAL HYPERPLASIA

Congenital autosomal recessive disease characterised by cortisol deficiency, with or without aldosterone deficiency and androgen excess.

It has 2 types: **classic and non-classic**.

CLASSIC: severe form. It's either salt losing or non-salt losing

Symptoms:

FEMALES: Classically presents with ambiguous genitalia with enlarged clitoris and one combined sinus instead of a separate urethra and vagina. May experience salt-losing adrenal crisis.

MALES: classically present with no signs at birth.

- Those with salt-losing form typically present at 7-14 days with vomiting, weight loss, lethargy, dehydration, hyponatraemia and hyperkalaemia.
- Those with non-salt-losing form present with virilisation at age 2-3

NON-CLASSIC: mild or late-onset form, they present with hyperandrogenism in later childhood and early pubarche, infertility, hirsutism, amenorrhoea, polycystic ovaries.

Investigations:

Renal function, electrolytes, blood glucose, serum 17-hydroxyprogesterone, corticotropin stimulation test, pelvic ultrasound, bone age

Treatment:

Classic: Standard hormone replacement, these include glucocorticoids, mineralocorticoids

Non-classic: Treatment only symptomatic.

CONN'S DISEASE

This is excess aldosterone, which causes Na+ retention

CAUSES:

1. Adrenal adenoma
2. Bilateral hyperplasia

SYMPTOMS:

1. K+ decreased

2. Na+ Increased or normal
3. Weakness
4. Cramps
5. Polyuria
6. Polydipsia
7. Parasthesias
8. HTN

INVESTIGATIONS

1. Aldosterone/ Renin ratio altered or altered Serum Aldosterone
2. CT adrenal

TREATMENT

1. Hyperplasia- Medicine (Spironolactone/Amiloride)
2. Adenoma- Surgery (Spironolactone given 4wks pre-op)

PHEOCHROMOCYTOMA

This is due to increased effect of catecholamines, usually due to adrenal tumour.

Rule of 10

10% Malignant
 10% Bilateral
 10% Extra-adrenal
 10% Children
 10% Familial
 Increased Catecholamines, associated with MEN-2.

SYMPTOMS:

1. Episodic Hypertension and headaches
2. Anxiety
3. Sweating
4. Palpitation
5. Flushing
6. Nausea
7. Vomiting
8. Abdominal pain

Episodes or intermittent symptoms

INVESTIGATIONS:

- 24 hours urinary collection for Catecholamines/Metanephrines

TREATMENT:

A. CRISIS:

- i. Phentolamine
- ii. Labetalol

A. STABLE PATIENT:

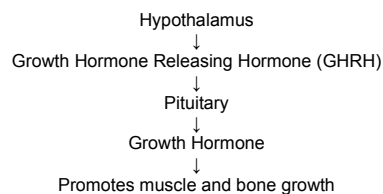
- i. Alpha- blocker (Phenoxybenzamine) followed by
- ii. Beta- blocker (Propranolol)

Surgery is done after 2 weeks of BP control.

Treatment. Surgical removal of adenoma

7. ACROMEGALY

1. Increased growth hormone (GH)
2. Pituitary tumour (tumour compressing on the optic chiasma)



SYMPTOMS

1. Increase in ring & shoes size
2. Spade like hands
3. Widespread teeth
4. Hoarse voice
5. Carpel tunnel syndrome
6. Excessive sweating
7. Visual field defect→Bi-temporal Hemianopia

8. Coarsening of facies
9. Prognathism
10. Macroglossia

COMPLICATIONS

1. Impaired Glucose Tolerance Test
2. Increase BP
3. Cardiomegaly, Hypertrophy
4. Increase IHD

INVESTIGATIONS:

1. Definitive- OGTT (Oral glucose tolerance test)
2. MRI of the pituitary gland
3. Serum insulin like growth hormone

TREATMENT: Surgery.

8. SIADH -Syndrome of Inappropriate Anti Diuretic Hormone

This is due to overproduction of ADH which leads to reduced production of urine.

Symptoms:

- Water Retention leading to hyponatraemia and hypertension
- Confusion, nausea, and seizure.

CAUSES:

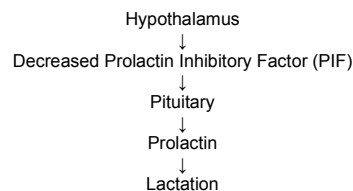
1. Lung cancer- Small cell lung cancer
2. Pancreas Cancer
3. Prostate Cancer
4. As a complication of Meningitis and Head Injury

DIAGNOSIS:

Urine Osmolarity over than 500 mosmol/kg

Plasma Na+ < 125 mmol/kg, plasma osmolality < 260 mosmol/kg

9. HYPERPROLACTINAEMIA



This is the commonest hormonal disturbance of the pituitary gland. Raised level of Prolactin (PRL) leads to hypogonadism, infertility, and osteoporosis.

- Normal PRL level is <400 mU/L
- If the PRL is mildly elevated (400-1000 mU/L) then repeat before referral consider look for causes other than a prolactinoma.
- Very high PRL >5000 mU/L usually means that a prolactinoma is present.

CAUSES OF RAISED PLASMA PROLACTIN:

1. Excess production from the pituitary gland by a Prolactinoma
2. Disinhibition, by compression of the pituitary stalk, reducing local dopamine levels.
3. Use of dopamine antagonist

Physiological: Pregnancy, breastfeeding

Drugs: Metoclopramide, haloperidol, antipsychotics

SYMPTOMS:

1. Amenorrhea
2. Infertility
3. Galactorrhea (milk discharge from the nipples)
4. Reduced libido
5. Weight gain
6. Dry vagina
7. Erectile dysfunction in men

INVESTIGATION

- Serum prolactin levels

TREATMENT

- Dopamine agonist e.g.; Bromocriptine.
- Surgery for adenoma.

10. DIABETES INSIPIDUS

This is passage of large volume, greater than 3 litres per day of dilute urine, due to impaired water reabsorption by the kidney

There are two types

1. **Neurogenic/Cranial Diabetes Insipidus:** Reduced ADH secretion from the Posterior pituitary
2. **Nephrogenic Diabetes Insipidus:** Impaired response of the kidney to ADH

SYMPTOMS:

1. Polyuria
2. Polydipsia
3. Dehydration (In dehydration Na⁺ is high)
4. Hypernatraemia

CAUSES OF NEUROGENIC DI:

1. Idiopathic
2. Congenital
3. Tumor
4. Trauma
5. Hypophysectomy
6. Autoimmune hypophysitis
7. Infiltration → Sarcoidosis
8. Vascular
9. Infection

CAUSES OF NEPHROGENIC DI:

1. Inherited
2. Metabolic
3. Drugs e.g. lithium
4. Chronic renal disease
5. Post-obstructive Uropathy

DIAGNOSIS:- The water deprivation test.

TREATMENT:

-For Cranial DI- Find the cause (MRI- head)
 -For Nephrogenic- Treat the cause
 -Desmopressin for therapeutic trial

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