ENDOCRINE EMERGENCIES

- **OUR TOPICS :**
- DIABETIC KETOACIDOSIS
- HYPERGLYCEMIA HYPEROSMOLAR
- HYPOGLYCEMIA
- ADRENAL CRISIS
- PHEOCHROMOCYTOMA
- THYROID STORM
- MYXEDEMA COMA

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Diabetic ketoacidosis

DONE BY : SHIMA'A RAED

- Diabetic ketoacidosis :It is an acute, Life threatening medical emergency complecation of DM that can occur in both types [1&2] but <u>more common in type 1 DM.</u> **requires very low insulin effects.**

- Biochemical derangement : hyperglycemia , acidosis , ketonemia .
- Precipitating factors :
- - Any type of stress or illness (infection ,truma) [↑ epinephrine]
- - can occur with missed insulin does in type 1 DM [↓ insulin]

Pathogenesis

- When there is insulin deficiency and glucagon or Epi excess , both of which contribute to accelerated sever hyperglycemia and accelerated ketogenesis .
- Sever **hyperglycemia** → osmotic diuresis → **Dehydration and Volume depletion.**
- **High ketones = Anion gap metabolic acidosis**
- **Acidosis** → **Hyperkalemia .**

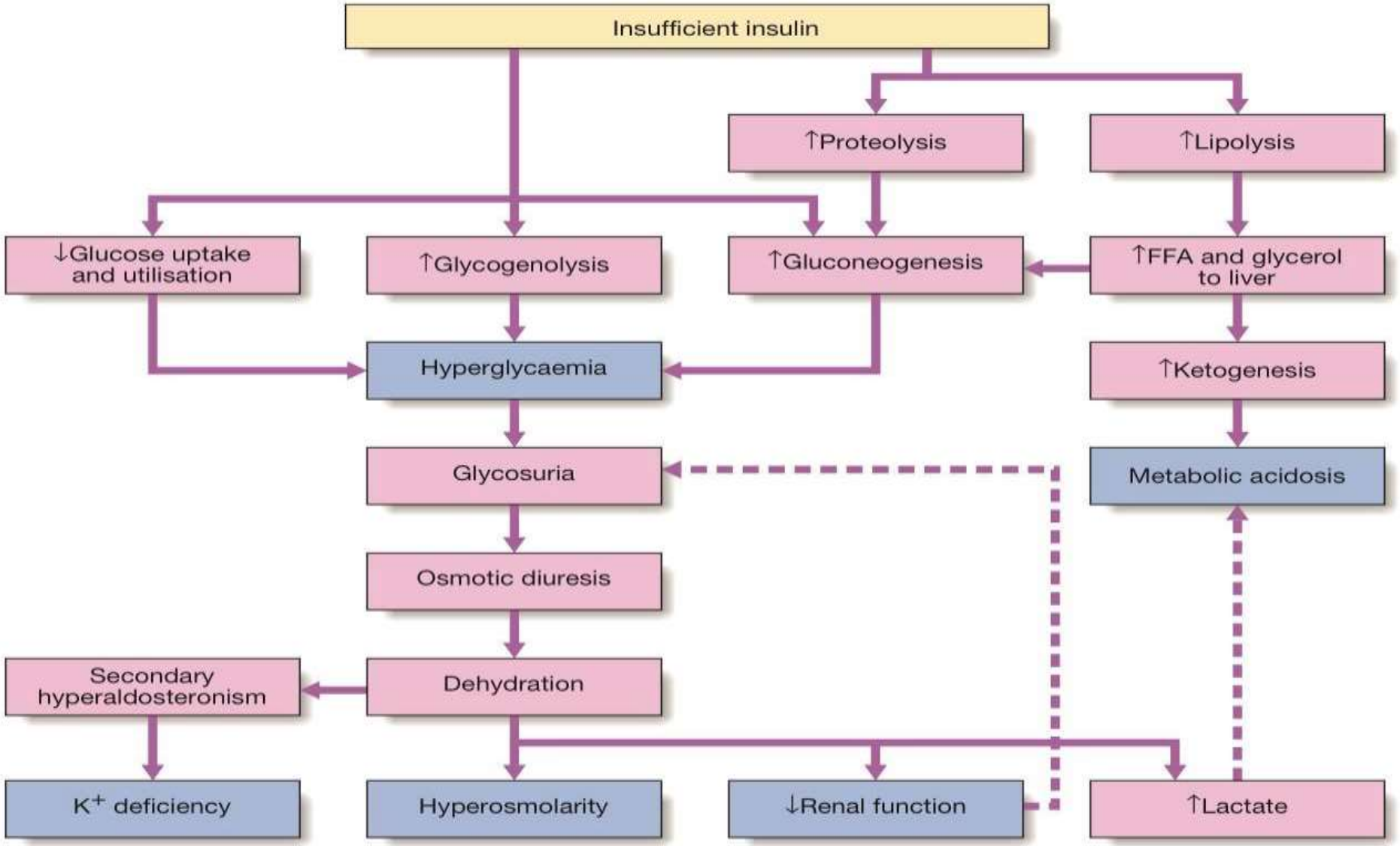


Fig. 21.5 Acute metabolic complications of insulin deficiency. (FFA = free fatty acids.)

Clinical presentation :

- Abdominal pain , nausea and vomiting .
- Volume depletion :
  - dry mucous membranes and
  - low blood pressure.
- Hyperglycemia
- Low bicarbonate.
- Hyperkalemia.
- Elevated plasma and urine ketones
- Glucose in urine

21.16 Clinical features of diabetic ketoacidosis

Symptoms	
• Polyuria, thirst	• Leg cramps
• Weight loss	• Blurred vision
• Weakness	• Abdominal pain
• Nausea, vomiting	
Signs	
• Dehydration	• Air hunger (Kussmaul breathing)
• Hypotension (postural or supine)	• Smell of acetone
• Cold extremities/peripheral cyanosis	• Hypothermia
• Tachycardia	• Confusion, drowsiness, coma (10%)

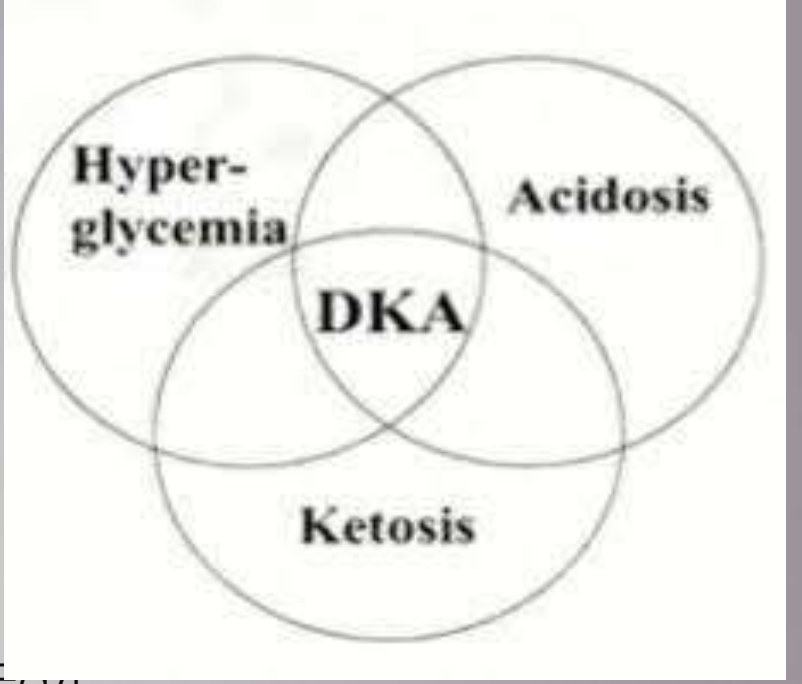
Clinical presentation :

- Anion gap metabolic acidosis [low bicarbonate] wil cause :
  - Kussmaul breathing : deep labored breathing
  - Hyperventalation to blow off co2 and raise pH
- Fruity smell on breath (acetone)
- Hypophosphatemia .
- Loss of ATP :
  - Muscle weaknees [respiratory failure]
  - Heart failure [contractility]
- Arrhythmias [hyperkalemia]
- Cerebral edema (common cause of death in children)

Diagnosis

- Diagnostic criteria
  - Triad : hyperglycemia , anion gap acidosis and ketones.

- A) hyperglycemia : serum glucose typically > 250mg/dl and < 850mg/dl
- B) metabolic acidosis:
  - Blood Ph < 7.3 and serum HCO3- <18 mEq/l
  - Icreased anion gap due to produc
- C) ketonemia (serum positive for ketones) and ketonuria .



Measurement	criteria
Glucose	>250 mg/dl
Arterial Ph	<7.30
Bicarbonate	< 18 mEq/dl
Urine ketones	positive
Serum ketones	positive

Treatment

- IV fluids : usually normal saline and infused continuously.
- IV insulin : Bolus plus continuous drip
- Add potassium to IV fluids when potassium less than 5.3 mEq/dl
  - Normal potassium 3.6 to 5.2 mEq/dl
- Monitor Mg , Ca and phosphate and replete as needed
- Bicarbonate infusion (usually not necessary)
  - Only indicated with pH <6.9

Monitoring

- Close monitoring of serum glucose and electrolytes
- When glucose approaches 200mg/dl add 5% dextrose to saline infusion
  - Allows continued insulin infusion to suppress ketones .
  - Avoids hypoglycemia
- Diabetic ketoacidosis resolve when:
  - anion gap normalizes (less than 12mEq/l)
  - Beta hydroxybutyrate absent (if available)
  - Patient can eat .

HHS/HHNS and Hypoglycemia

SHATHA SALAMA AL-OMOUSH

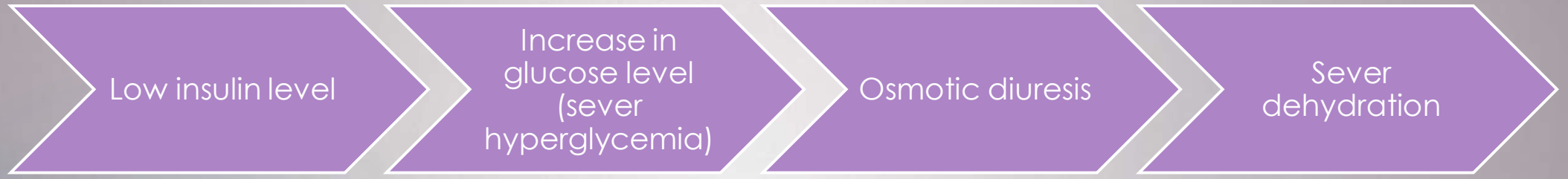
Hyperosmolar hyperglycemic syndrome (HHS)

- This condition, in which severe hyperglycemia develops **without significant ketosis**, is the metabolic emergency characteristic of uncontrolled type 2 diabetes.
- SO it is a state of severe hyperglycemia, hyperosmolarity, and dehydration

Key Features of HHNS : •
Severe hyperosmolarity (>320 mOsm/L)
• Hyperglycemia (>600 mg/dL)
• Dehydration
• Acidosis and ketosis are absent (unlike in DKA)

- ***The classical presentation of HHS :***
- Patients present in middle or later life, often with previously undiagnosed diabetes. Common precipitating factors include consumption of glucose-rich fluids, concurrent medication such as thiazide diuretics or steroids, and intercurrent illness.
- Factors that play a rule in the presentation:
- ***Age.*** The extreme dehydration characteristic of hyperosmolar hyperglycaemic state may be related to age. Old people experience thirst less acutely, and more readily become dehydrated. In addition, the mild renal impairment associated with age results in increased urinary losses of fluid and electrolytes. ***The degree of insulin deficiency:*** This is less severe in the hyperosmolar hyperglycaemic state. Endogenous insulin levels are sufficient to inhibit hepatic ketogenesis, but insufficient to inhibit hepatic glucose production.

Pathophysiology



Ketogenesis is minimal because a small amount of insulin is released to blunt counterregulatory hormone release (glucagon). • Ketosis and acidosis are typically absent or minimal .

Severe dehydration is due to continued hyperglycemic (osmotic) diuresis. The patient's inability to drink enough fluids (either due to lack of access in elderly/bedridden patients or to inadequate thirst drive) to keep up with urinary fluid losses exacerbates the condition

- ***Clinical features:***
- a. Thirst, polyuria
- b. Signs of extreme dehydration and volume depletion (hypotension, tachycardia)
- c. CNS findings and focal neurologic signs are common (e.g., seizures)—secondary to hyperosmolarity.
- d. Lethargy and confusion may develop, leading to convulsions and coma.

- ***Risk factors include:***
- A stressful event such as infection, heart attack, stroke, or recent surgery
- Heart failure
- Impaired thirst
- Limited access to water (especially in people with dementia or who are bedbound)
- Older age
- Poor kidney function
- Poor management of diabetes, not following the treatment plan as directed
- Stopping or running out of insulin or other medicines that lower glucose level

- ***Investigations and diagnosis***
- a. Hyperglycemia: serum glucose typically higher than DKA and frequently >900 mg/dL.
- b. Hyperosmolarity: serum osmolarity >320 mOsm/L.
- c. Serum pH >7.3 (no acidosis); serum $HCO3^-$ > 15.
- d. BUN is usually elevated. Prerenal azotemia is common

Treatment

- a. Fluid replacement is most important (normal saline): 1 L in the first hour, another liter in the next 2 hours. Most patients respond well. Switch to half normal saline once the patient stabilizes. When glucose levels reach 250 mg/dL, add 5% glucose (D51/2NS) as in DKA. • Very rapid lowering of blood glucose can lead to cerebral edema in children (just as in DKA). • In patients with cardiac disease or renal insufficiency, avoid volume overload (can lead to CHF), but generous fluids are still needed. b. Insulin: An initial bolus of 5 to 10 units intravenously, followed by a continuous low-dose infusion (2 to 4 units/hr) is usually appropriate.

DKA versus HHNS

TABLE 4-8 Diabetic Ketoacidosis Versus Hyperosmolar Hyperglycemic Nonketotic Syndrome

	DKA	HHNS
Pathophysiology	Insulin deficiency → ketosis, acidosis, dehydration	Insulin deficiency → hyperosmolarity, osmotic diuresis, profound dehydration
Laboratory Findings	• Hyperglycemia (>450) • Metabolic acidosis (anion gap)—serum pH <7.3 • Ketosis	• Hyperglycemia (>900 mg/dL) • Hyperosmolarity (>320 mOsm/L) • Serum pH >7.3 (no acidosis)
Treatment	Insulin, IV fluids, potassium	Aggressive IV fluids, low-dose insulin infusion
Mortality Rate	5%–10%	10%–20%

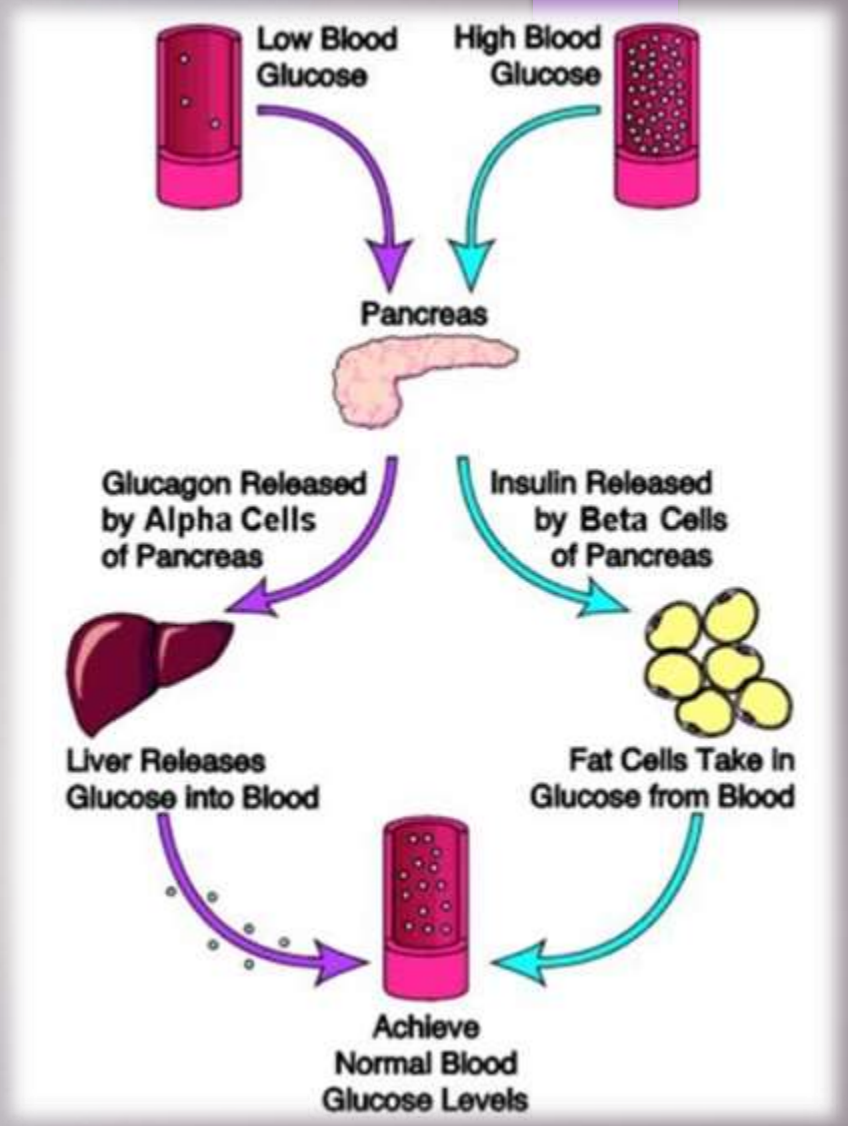
Hypoglycemia :

- is when your blood sugar levels have fallen low enough that you need to take action to bring them back to your target range. This is usually when your blood sugar is less than 70 mg/dL or 3.9 millimoles per liter (mmol/L).
- ** However, could be there a difference in blood sugar targets, and what level is too low for the patient .
- Hypoglycemia happens either in diabetic or non-diabetic patients.

- The glucose level that defines hypoglycemia is variable In people with diabetes, levels below 3.9 mmol/l (70 mg/dl) are diagnostic. In adults without diabetes, symptoms related to low blood sugar, low blood sugar at the time of symptoms, and improvement when blood sugar is restored to normal confirm the diagnosis. Otherwise, a level below 2.8 mmol/l (50 mg/dl) after not eating or following exercise may be used. `In newborns, a level below 2.2 mmol/l (40 mg/dl), or less than 3.3 mmol/l (60 mg/dl) if symptoms are present, indicates hypoglycemia. Other tests that may be useful in determining the cause include insulin and C peptide levels in the blood.

Pathophysiology

- This condition typically arises from abnormalities in the mechanisms involved in glucose homeostasis.
- Normally :the balance of insulin and glucagon to maintain blood glucose
- **Insulin**: secreted by the pancreas in response to elevated blood glucose following a meal.
- **Glucagon**: a fall in blood glucose increases the release of glucagon from the pancreas to promote glucose production



Hypoglycemia classification :

- five distinct categories:
- severe hypoglycemia (an event causing such neurological changes as to require the aid of another person).
- documented symptomatic hypoglycemia (a plasma glucose at or less than 70 mg/dL accompanied by typical symptoms of hypoglycemia).
- asymptomatic hypoglycemia.
- probable symptomatic hypoglycemia.
- relative hypoglycemia (symptoms suggestive of hypoglycemia with a measured plasma glucose concentration greater than 70 mg/dL

In hypoglycemia :

- Physiologic Responses to Hypoglycemia

• When glucose levels approach the low 80s, insulin levels decrease—This decrease is normally enough to prevent hypoglycemia.

• As glucose levels decrease further, glucagon levels increase (glucagon is the first line of defense against more severe hypoglycemia).

• Epinephrine is the next hormone to combat hypoglycemia.

Cortisol and other catecholamines also play a role.

• As glucose levels decrease into the 50s and below, symptoms begin.

Insulin

- When glucose levels approach the low 80s, insulin levels decrease—This decrease is normally enough to prevent hypoglycemia

glucagon

- As glucose levels decrease further, glucagon levels increase (glucagon is the first line of defense against more severe hypoglycemia).
- Glucagon is secreted by pancreatic a-cells and acutely raises plasma glucose concentration by stimulating hepatic glucose production via glycogenolysis and gluconeogenesis.

epinephrine

- Epinephrine acts on alpha and beta adrenergic receptors at multiple end organs to effect a more sustained increase in plasma glucose concentration: epinephrine increases glycogenolysis and gluconeogenesis at the liver; reduces insulin secretion while increasing glucagon release from the pancreatic islets; reduces glucose uptake and utilization and increases glycolysis by muscle; and increases lipolysis in adipose tissue

Glucagon and epinephrine are secreted as glucose levels fall slightly below the physiologic range, at approximately 68 mg/dL (3.8 mmol/L)

Activation of autonomic nervous system.

- which increases the amounts of norepinephrine at the nerve terminals and epinephrine in the circulation.

Other hormones

- As glucose levels fall further, other counterregulatory factors are activated. Secretion of growth hormone occurs at a plasma glucose threshold of approximately 66 mg/dL (3.7 mmol/L) and secretion of cortisol at approximately 58 mg/dL (3.2 mmol/L).[11,12] Growth hormone and cortisol induce changes in metabolic processes over longer periods of time (hours) by stimulating lipolysis in adipose tissue and ketogenesis and gluconeogenesis in the liver. These two hormones do not have an immediate role in the recovery from hypoglycemia.

Hypoglycemia causes

- 1. Drug-induced—taking too much insulin is a common problem in diabetic patients attempting tight control of their disease.
- Other drugs :

Sulfonylureas may be used in the treatment of diabetes or may be taken by non-diabetics in suicide attempts.

Quinine may produce severe hypoglycemia in the course of treatment for falciparum malaria

Propranolol can induce hypoglycemia in the presence of strenuous exercise or starvation

Salicylates may cause hypoglycemia; usually accidental ingestion by children

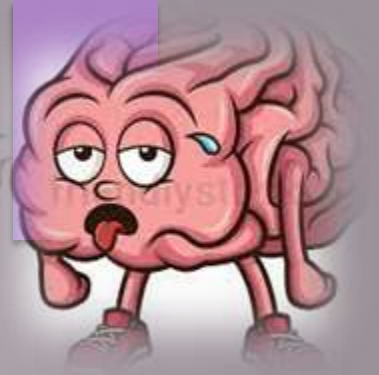
Pentamidine used in the treatment of resistant pneumocystis pneumonia

- 2. Factitious hypoglycemia
- This is a relatively common variant of self-induced disease and is more common than an insulinoma. Hypoglycemia is produced by surreptitious self-administration of insulin or sulfonylureas.
- a. If the patient took insulin surreptitiously, there will be a high blood insulin level and a low blood C-peptide level (because exogenous insulin does not contain C-peptide)
- . b. Patients taking exogenous insulin will also develop anti-insulin antibodies, which can be measured.
- c. If the patient took sulfonylurea, check urine or serum for levels of this drug.

- 3. Insulinoma : Insulinomas are pancreatic islet cell tumours that secrete insulin.
- 4. Ethanol (Alcohol) ingestion—due to:
- a. Poor nutrition that leads to decreased glycogen (and loss of glycogenolysis)
- b. Metabolism of alcohol that lowers nicotinamide adenine dinucleotide levels and decreases gluconeogenesis
- 5. Postoperative complications after gastric surgery (due to rapid gastric emptying)
- 6. Reactive (idiopathic) hypoglycemia—symptoms occur 2 to 4 hours after a meal
- 7. Adrenal insufficiency
- 8. Liver failure
- 9. Critical illness
- 10. Disorders of carbohydrate metabolism (e.g., glycogen storage diseases)—usually diagnosed at a much younger age

Clinical features

- ** Symptoms occur at a blood glucose level of 40 to 50 mg/dL and maybe before
- Shakiness, anxiety, nervousness
- Palpitations, tachycardia
- Sweating, feeling of warmth (sympathetic muscarinic rather than adrenergic)
- Pallor, coldness, clamminess
- Dilated pupils (mydriasis)
- Hunger, borborygmus
- Nausea, vomiting, abdominal discomfo
- Headache



- ***The primary organ at risk in hypoglycemia is the brain***—The brain uses glucose as its main energy source (Unlike other tissues, the brain cannot use free fatty acids as an energy source).
- Therefore, if the amount of glucose supplied by the blood falls, the brain is one of the first organs affected. In most people, subtle reduction of mental efficiency can be observed when the glucose falls below 3.6 mmol/l (65 mg/dl). Impairment of action and judgment usually becomes obvious below 2.2 mmol/l (40 mg/dl). Seizures may occur as the glucose falls further. As blood glucose levels fall below 0.55 mmol/l (10 mg/dl), most neurons become electrically silent and nonfunctional, resulting in coma. These brain effects are collectively referred to ***as neuroglycopenia***.

Diagnosis

- ***Blood glucose level :***Symptoms generally begin when levels drop below 50.

  However, there is no cutoff value to define hypoglycemia
- ***Whipple triad*** (is used to diagnose true hypoglycemia (i.e., hypoglycemia due to underlying disease)
- ***Lab tests :*** for measurement of **serum insulin**, **C-peptide**, and **glucose** when symptoms occur (an overnight fast may be sufficient to produce symptoms).
- ***72-hour fast*** (24 hours is usually sufficient ; used to diagnose insulinoma (if suspected)

Treatment

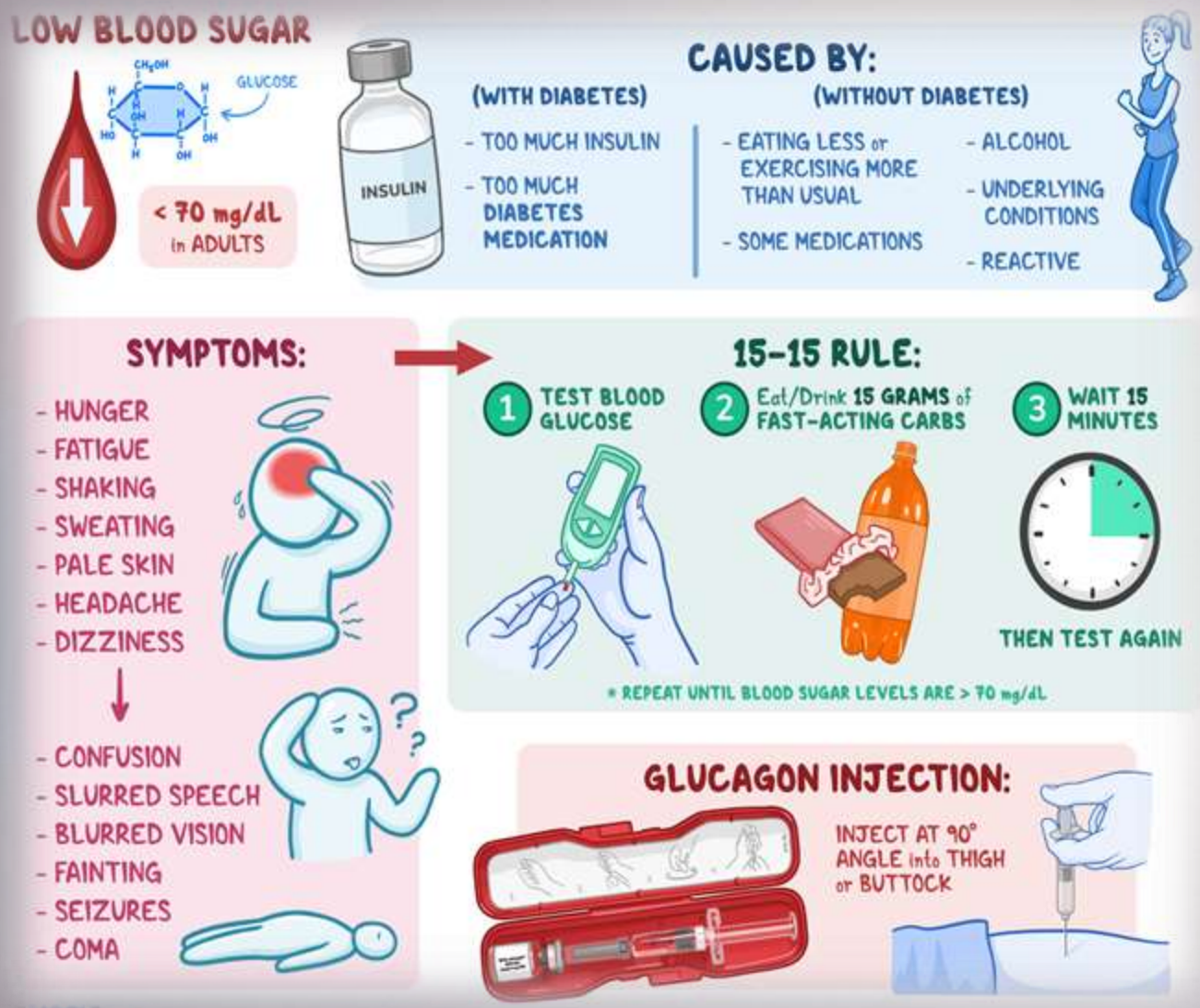
Acute treatment of hypoglycemia.

- a. If the patient can eat, give sugar-containing foods; Blood glucose can be raised to normal within minutes by taking (or receiving) 10–20 g of carbohydrate. It can be taken as food or drink if the person is conscious and able to swallow.
- If a person has such severe effects of hypoglycemia that they cannot (due to combativeness) or should not (due to seizures or unconsciousness) be given anything by mouth, medical personnel such as paramedics, or in-hospital personnel can give intravenous dextrose, concentrations varying depending on age (infants are given 2 ml/kg dextrose 10%, children are given dextrose 25%, and adults are given dextrose 50%).
- Repeat administration of D50W as necessary, but switch to D10W as clinical condition improves and glucose level is approximately >100 mg/dL.

Dasiglucagon was approved for medical use in the United States in March 2021, to treat severe hypoglycemia in diabetic patients .

- ***After treating hypoglycemia acutely , then :***
- Appropriate management of underlying cause (e.g., diabetes, insulinoma) .
- If reactive hypoglycemia is suspected, dietary interventions are appropriate.
- If the patient is an alcoholic (or suspected alcoholic), give thiamine before administering glucose to avoid Wernicke encephalopathy.

Summary



21.21 Emergency treatment of hypoglycaemia

Mild (self-treated)

- Oral fast-acting carbohydrate (10–15 g) is taken as glucose drink or tablets or confectionery
- This should be followed with a snack containing complex carbohydrate

Severe (external help required)

- If patient is semiconscious or unconscious, parenteral treatment is required:
  IV 75 mL 20% dextrose (= 15 g; give 0.2 g/kg in children)*
  Or
  IM glucagon (1 mg; 0.5 mg in children)
- If patient is conscious and able to swallow:
  Give oral refined glucose as drink or sweets (= 25 g)
  Or
  Apply glucose gel or jam or honey to buccal mucosa

*Use of 50% dextrose is no longer recommended.

Adrenal crisis

Farah itkedek

Adrenal crisis

- An acute and severely symptomatic stage of adrenal insufficiency that is caused by an abrupt or sudden failure on the part of adrenal cortex to produce cortisol.
- **Primary**: due to destruction or defect of the adrenal cortex.
- **Secondary**: due to a hypothalamopituitary disease or suppression of hypothalamopituitary-adrenal (HPA) axis.

Clinical features

- **Clinical features**:
- Loss of cortisol:
- Hypotension, shock ,Cardiovascular collapse ,abdominal pain, lethargy, tiredness, weakness,, hypoglycemia, nausea, and vomiting.
- Loss of aldosterone:
- Potassiun retention, hyperkalemia,acidosis,sodium loss in urine and hypovolemia.

Causes

- Withdrawal of steroid therapy in a patient on long term steroid therapy.
- Adrenal gland is the most commonly involved endocrine organ in patients infected with HIV, most of them being critically ill.
- Medications that interfere with cortisone synthesis include: ketoconazole, rifampin, phenytoin and megesterol acetate.
- The most dramatic adrenal crisis is due to adrenal hemorrhage or infarction.,
- due to meningococcal septicemia:
- --Any stress (trauma, surgery, infection ,dehydration) can precipitate the crisis

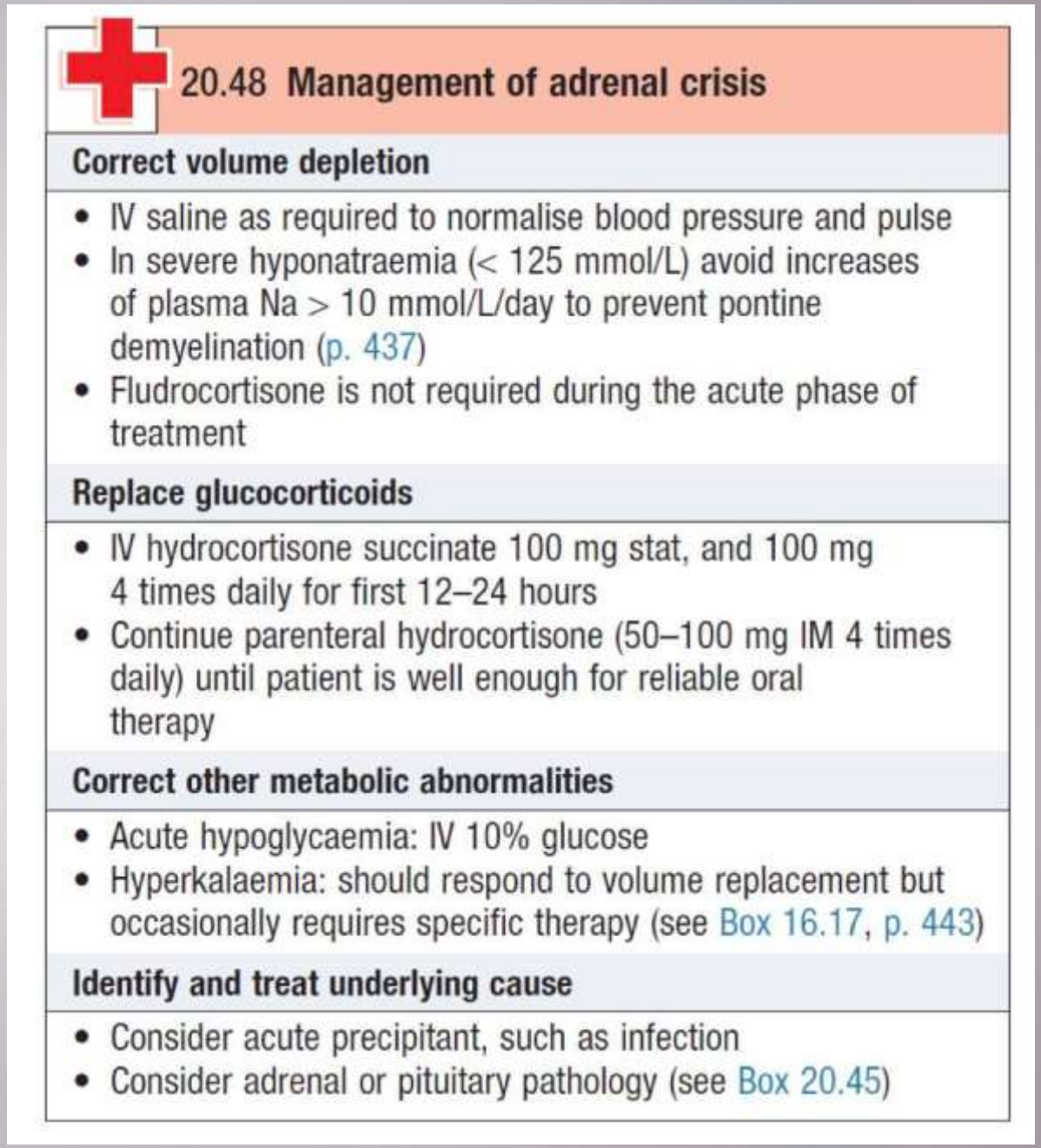
Diagnosis

- Cortisol level (to assess the level of glucocorticoids).
- ACTH (cosyntropin) stimulation test : Failure of cortisol to rise above 552 nmol/L 30 min after administration of 0.25 mg of synthetic ACTH IV.
- Imaging : Abdominal CT reveal enlargement of adrenals.
- Hyponatremia and hyperkalemia (Hyponatremia might be obscured by dehydration)+unresponsive hypotension, THINK ABOUTADDISON

Management

- The patient will necessarily be in a ICU setting (ABC).
- A rapid infusion of dextrose-saline is setup. u 100 mg of hydrocortisone is injected IV and vasopressors are started if hypotension persists.
- Hydrocortisone is repeated in the same dose every 6 hours till the condition stabilizes in 24-48 hours and then the dosage is gradually reduced to 100 mg every 8 hours for the next 48 hours and then changed to oral replacement.
- Treat precipitating diseases

Management of adrenal crisis



20.48 Management of adrenal crisis

Correct volume depletion

- IV saline as required to normalise blood pressure and pulse
- In severe hyponatraemia (< 125 mmol/L) avoid increases of plasma Na > 10 mmol/L/day to prevent pontine demyelination (p. 437)
- Fludrocortisone is not required during the acute phase of treatment

Replace glucocorticoids

- IV hydrocortisone succinate 100 mg stat, and 100 mg 4 times daily for first 12–24 hours
- Continue parenteral hydrocortisone (50–100 mg IM 4 times daily) until patient is well enough for reliable oral therapy

Correct other metabolic abnormalities

- Acute hypoglycaemia: IV 10% glucose
- Hyperkalaemia: should respond to volume replacement but occasionally requires specific therapy (see Box 16.17, p. 443)

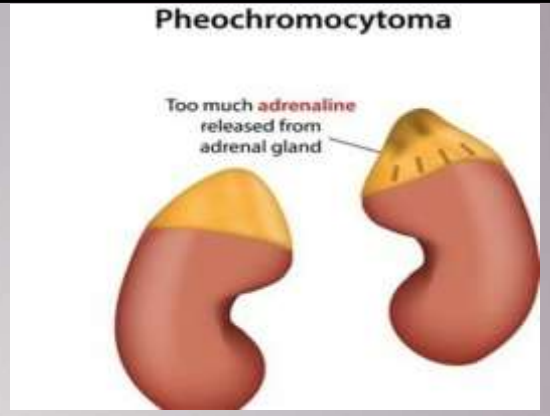
Identify and treat underlying cause

- Consider acute precipitant, such as infection
- Consider adrenal or pituitary pathology (see Box 20.45)

Pheochromocytoma

Done by :Balqees Faisal

Pheochromocytoma



- Phaeochromocytomas are catecholamine-secreting tumors arising from chromaffin cells in the adrenal medulla.
- Curable if diagnosed and treated, **but may be fatal if undiagnosed**.

- any patient presenting with acute hypertension and tachycardia should be considered at risk of phaeochromocytoma, especially if **young or in an at risk group.**

- **Rule of 10%:**

  Malignant, Bilateral, Extra-Adrenal (found within nervous tissue outside of the adrenal glands),**In Children**,**Familial**,**Multiple.**

Clinical feature

- Patients will often present with a history of poorly controlled or accelerated hypertension.
- Presenting features include:

  Episodic hypertension, tachycardia, palpitations, sweating, sever pounding headache, anxiety, feeling of impending death,, nausea and vomiting, abdominal pain (tumor hemorrhage).

  Laboratory findings: hyperglycemia, hyperlipidemia, hypokalemia.

Diagnosis

- 1. Urine screen—test for the presence of the following breakdown products of catecholamines: Metanephrine, Vanillylmandelic acid, homovanillic acid, normetanephrine

- 2. Plasma metanephrines have been proposed by some investigators as a superior test to urine metanephrines, especially when clinical suspicion is high.

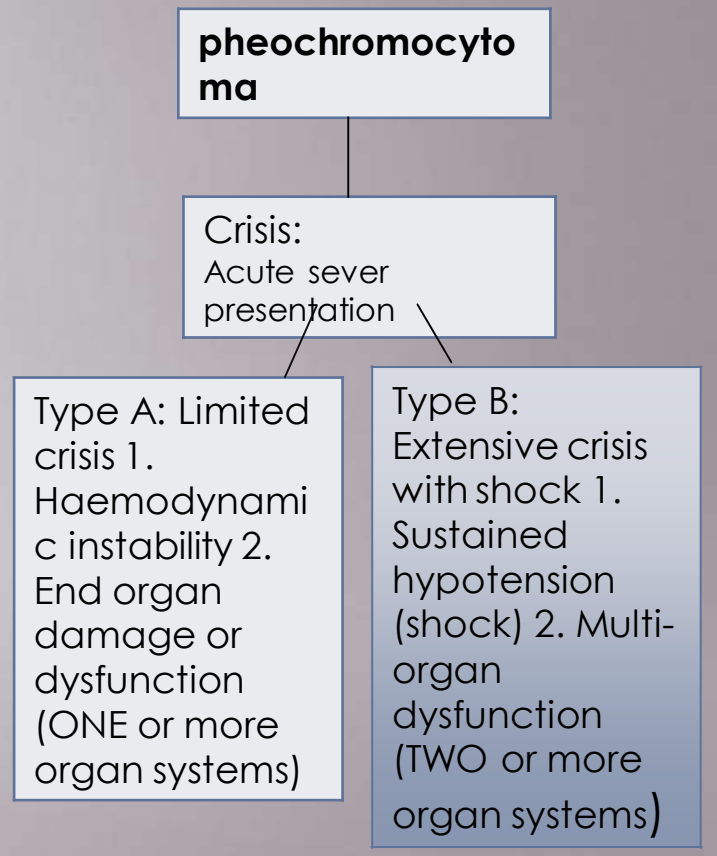
- 3. Urine/serum epinephrine and norepinephrine levels—if the epinephrine level is elevated, the tumor must be adrenal or near the adrenal gland (organ of Zuckerkandl) because nonadrenal tumors cannot methylate norepinephrine to epinephrine

- 4. Tumor localization tests—CT, MRI.

Pheochromocytoma crisis
Hypertensive crisis

- Phaeochromocytoma crisis (PCC) has been defined as the acute severe presentation of catecholamine-induced haemodynamic instability causing end-organ damage or dysfunction is a fatal condition characterized by multi-organ system failure, severe hypertension and/or hypotension, high fever and encephalopathy
- Catecholamines, acting primarily on a-adrenergic receptors, cause profound arterial vasoconstriction leading to hypertension and relatively reduced intravascular volume. This leads to reduced end-organ perfusion and tissue ischaemia and is the primary mechanism for organ failure in PCC.
- A patient could potentially progress from type A to type B in the course of a crisis,

Type A crisis is four times more common than type B crisis

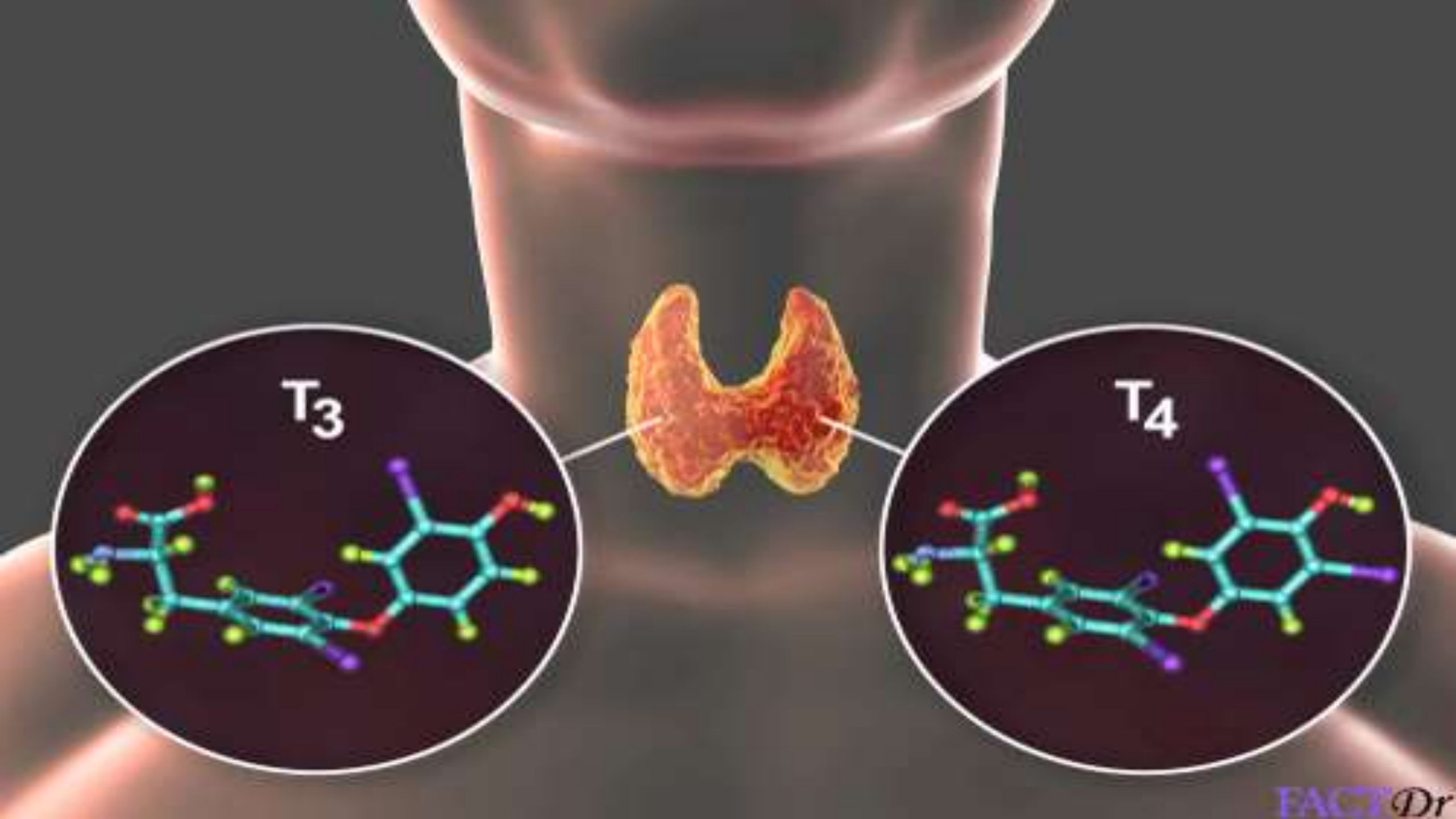


Managem ent

- Surgical tumor resection with early ligation of venous drainage is treatment of choice.
- patients should be treated with α-blockade (typically phenoxybenzamine) for 10 to 14 days prior to surgery as well as β-blockade (i.e., propranolol) for 2 to 3 days prior to surgery.
- The α-blockade is used to control BP, and the β-blockade is used to decrease tachycardia.

THYROID STORM

Done by: Doa Khalid


T3
T4

Thyroid Storm

THYROID STORM IS A RARE, LIFE-THREATENING COMPLICATION OF THYROTOXICOSIS. IT IS THE 2ND THYROID EMERGENCY THAT CARRIES A HIGH MORTALITY RATE, UPTO 20% OF PATIENTS ENTER A COMA OR DIE. THUS, IT IS VERY IMPORTANT TO RECOGNIZE IT EARLY AND START AGGRESSIVE TREATMENT TO REDUCE MORTALITY. THYROID STORM IS OFTEN PRECIPITATED IN PATIENTS WITH UNDIAGNOSED OR INADEQUATELY TREATED HYPERTHROIDISM.

Precipitated Factors Include:

- Surgery
- Infections
- Stress
- In rare cases, thyroid storm can be caused by treatment of hyperthyroidism with radioactive iodine therapy for Graves disease.
- Withdrawal of anti-thyroid drugs
- Diabetic keto acidosis

Clinical manifestations

- Similar to symptoms of hyperthyroidism but more severe:

1. High fever
2. Tachycardia +/- AF
3. Agitation or Psychosis
4. Delirium
5. Some patients have GI symptoms of nausea, vomiting, and diarrhea.

Point Scale for the Diagnosis of Thyroid Storm

Thermoregulatory dysfunction

Temperature (8F)	
99.0–99.9 5	0
100.0–100.9	10
101.0–101.9	15
102.0–102.9	20
103.0–103.9	25
>104.0	30

Cardiovascular

Tachycardia (beats per minute)	
100–109	5
110–119	10
120–129	15
130–139	20
>140	25

Atrial fibrillation	
Absent	0
Present	10

Congestive heart failure	
Absent	0
Mild	5
Moderate	10
Severe	20

Gastrointestinal-hepatic dysfunction

Manifestation	
Absent	0
Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
Severe (jaundice)	20

Central nervous system disturbance

Manifestation	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizure, coma)	30

Precipitant history

Status	
Positive	0
Negative	10

Scores totaled

>45 Thyroid storm

25–44 Impending storm

<25 Storm unlikely

Diagnosis:

- Diagnose the condition with measurement of TSH and T3,T4. In virtually all cases, Low TSH and high T3 and T4
- Findings are not different than that of hyperthyroidism, but the difference is in the setting and severity.
- Severe metabolic stress that the patient can no longer tolerate. This severe stress results in a relative adrenal insufficiency, even though the adrenal glands may be functioning, Patients in thyroid storm die from cardiovascular collapse. A very important aspect of treatment is supraphysiologic doses of glucocorticoids.

Management aims to:

- Decrease sympathetic outflow
- Decrease peripheral conversion of T4 toT3
- Decrease production and release of thyroid hormone.

Management:

- First of all we should monitor there Heart Rate, Blood Pressure, Respiration, Temperature.
- IV fluids (With glucose and thiamine)
- Keep the environment cool, quiet (cooling blankets + sedatives)
- No food with iodine
- Avoid Aspirin (can displace thyroid hormones from binding proteins)

Treatmen of Thyroid Storm includes the following:

- Glucocorticoids
- Interrupt the physiologic response to excess thyroid hormone: IV propranolol or esmolol.
- Block new hormone synthesis: high-dose thionamide (PTU or MMI).
- Block release of preformed hormone from the gland: stable iodide.
- Block peripheral conversion of T4 to T3: iodinated contrast agent, propranolol, and corticosteroids. PTU also does this (but not MMI).
- Give empiric broad-spectrum antimicrobial coverage until infection is excluded.
- Provide supportive care in the ICU with diligent attention to volume status, temperature, and heart rate.

Drug	Typical adult dose	Action
Antithyroid agents		
propylthiouracil (also called PTU)	1,200 – 1,500 mg/day, given in 200 – 250 mg increments PO or via gastric tube	Prevents production of more T_4 and T_3 in the thyroid, and blocks the conversion of T_4 to T_3 outside the thyroid.
methimazole (Tapazole)	120 mg given in 20 mg increments PO or via gastric tube	Prevents production of more thyroid hormone.
Iodides		
Lugol's solution	10 drops twice a day PO or via gastric tube	Blocks release of stored thyroid hormone from thyroid gland.
saturated solution of potassium iodide (Pima, SSKI)	8 drops every 6 hours PO or via gastric tube	
Glucocorticoids		
dexamethasone (Decadron)	2 mg every 6 hours, PO or IV	Blocks conversion of T_4 to T_3.
hydrocortisone	100 mg IV every 8 hours	
Beta-blockers		
propranolol (Inderal)	1 mg/min IV as required, then 60 – 80 mg every 4 hours PO or via gastric tube	Reduces symptoms (tachycardia, tremor, restlessness) caused by a heightened response to catecholamines; blocks conversion of T_4 to T_3.
esmolol (Brevibloc Injection)	500 mcg/kg/min for 1 minute, then 50 – 100 mcg/kg/min for 4 minutes	

Myxedema Coma

SARAH FRAIJ

Myxoedema coma

- End stage of untreated or insufficiently treated hypothyroidism
- Rare .
- High mortality rate 50%-75% even with treatment.
- Myxoedema coma occurs almost exclusively in persons aged 60 years and older with female preponderance.

Clinical manifistations

- Hypothermia (24-32.2 C)
- Respiratory depression with CO2 retention.
- Depressed state of conciousness (Confusion/Coma)
- Signs and symptoms of hypothyroidism.
- Hyponatremia
- Hypoglycemia

- **Myxedema coma often occurs after a long history of hypothyroidism. It’s more common during cold winter months.**

- **Any of the following can trigger it:**

- **stopping hypothyroid treatment medication**
- **sudden illness, like heart attack or stroke**
- **Infection(UTI, pneumonia)**
- **trauma**
- **certain drugs that suppress the central nervous system**
- **exposure to cold**
- **stress**

Diagnosis

- High TSH and low T4
- if the T4 is low and TSH low normal consider pituitary hypothyroidism
- Blood gasses
- Electrolytes and creatinine

- The diagnosis of myxedema coma is initially based upon the history, physical examination, and exclusion of other causes of coma. If the diagnosis of myxedema coma is suspected, a blood sample should be drawn

Management

- **1- Supportive care**:
- Gradual warming
- respiratory support (Ventilation)
- Cardiovascular support (Monitor cardiac output and pressure).
- Avoid sedation and fluid overload.

- **2- Parenteral thyroxine**
- (Levothyroxine) Loading dose of 300 – 400 µg
- Then 50-100 µg daily

- **3- Hydrocortisone** 40 – 100 mg every 8 hr for 1 week IV, then taper
- - Controversial but necessary in hypopituitarism or multiple endocrine failure .

Monitoring

- Serum T4 and T3 should be measured every one to two days to confirm that the therapy is working and that very high levels of T3 are avoided. Once there is improvement (regained consciousness, improved mental status, improved pulmonary and cardiac function), the patient can be treated with oral T4 alone. The initial oral T4 dose should be determined based on body weight, age, coexistent cardiovascular disease, and the recent intravenous dose.

Resources

- Step Up Medicine 5th Edition
- Medstudy 19th Edition
- Pathoma
- Davidson's Principles and Practice of Medicine
- Oxford Handbook of Clinical Medicine
- Kumar and clark

Thank you