Endocrine Disorders: Common Clinical Scenarios

For Medical Students & House Officers

BY

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&

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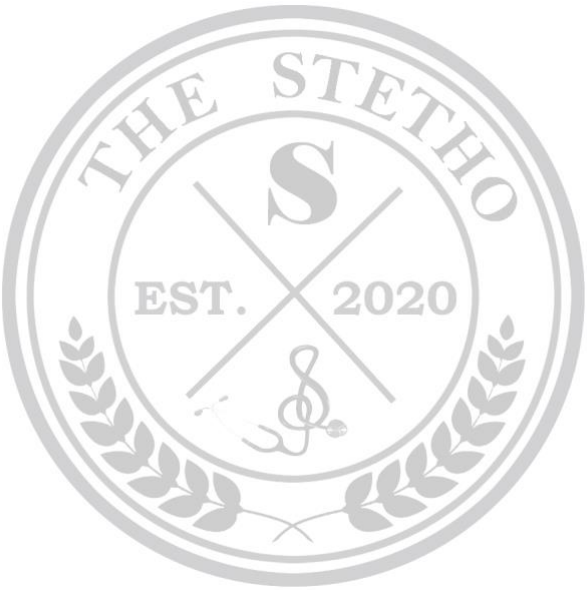
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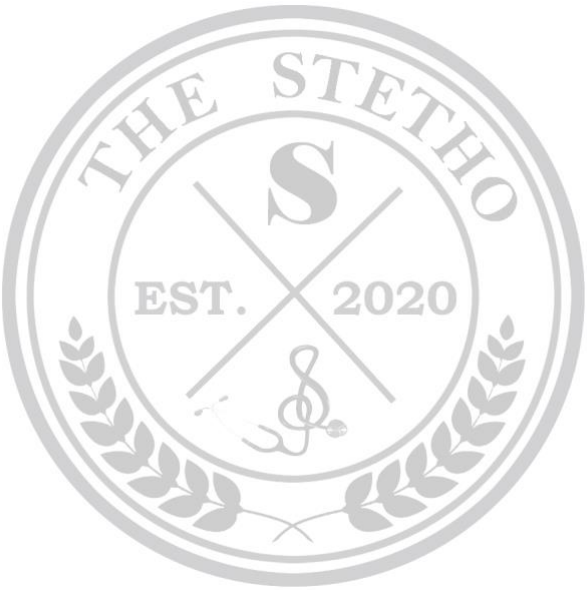
Dr. Asim Khan & Dr. Muhammad Irfan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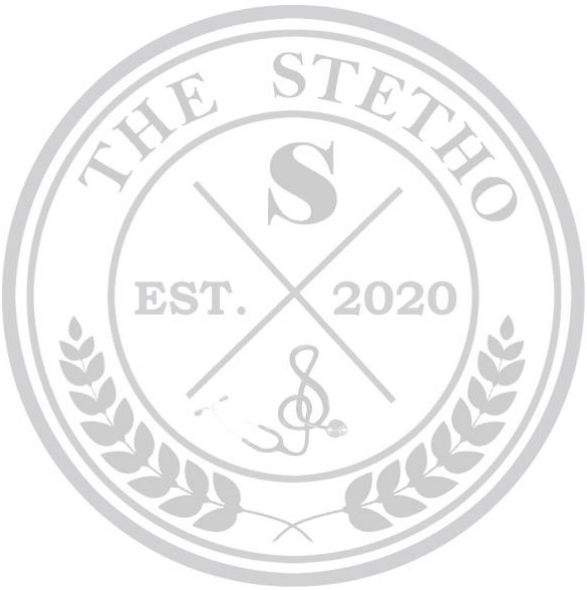
"Hippocrates"

" Whenever a doctor cannot do good he must be kept from doing harm "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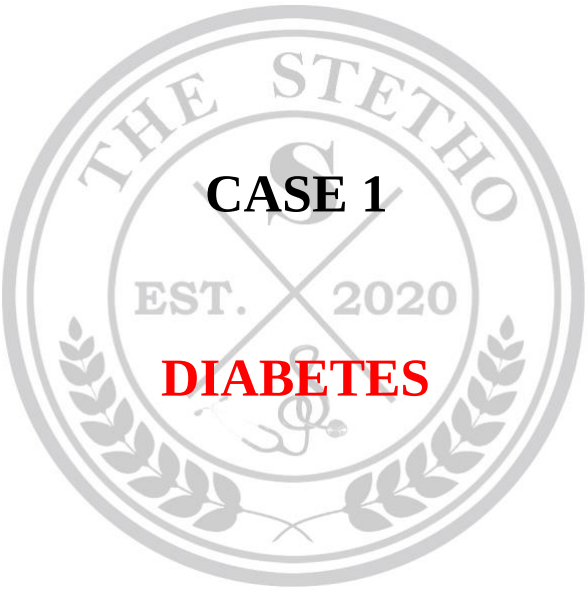


CASE 1

DIABETES

Diabetes mellitus is a syndrome of chronic hyperglycemia due to relative insulin deficiency, resistance or both.

WHO DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

Fasting blood glucose > 7.0 mmol/L (126 mg/dL). Random blood glucose > 11.1 mmol/L (200 mg/dL). One abnormal value is diagnostic in symptomatic individuals; two values are needed in asymptomatic people. The glucose tolerance test is required for borderline cases and for diagnosis of gestational diabetes.

HbA1c>6.5 (48 mmol/L).

Type 1 diabetes

Type 2 diabetes

Polyuria and Polydipsia + Many patients are over 40 years of age and weight loss with age and are obese.

Random plasma glucose of 200 mg/dL + Polyuria and polydipsia.

Ketonuria (11.1 mmol/L) and weight loss are uncommon at time of diagnosis.

Candida vaginitis + Plasma glucose of 126 may be an initial manifestation. 126 mg/dL (7.0 mmol/L) after an overnight fast on more than one occasion.

Two hours after 75g oral glucose; diagnostic values are 200 mg/dL (11.1 mmol) documented on more than one occasion. + Ketonemia, ketonuria, + HbA1c2 6.5%. or both +

Hypertension, dyslipidemia and Islet autoantibodies are often associated.

Glycemic Index: The glycemic Index of a carbohydrate containing food ls determined by comparing the glucose excursions after consuming 50 g of test food with glucose excursions after consuming 50 g of reference food (white bread)

Eating low glycemic Index foods results in lower glucose levels after meals. Low glycemic index foods have values of 55 or less and include many fruits, vegetables, grainy breads, pasta and legumes.

High glycemic index foods have values of 70 or greater and include baked potato, white bread, and white rice.

Glycemic index is lowered by the presence of fats and protein when food is consumed in a mixed meal. Even though it may not be possible to accurately predict the glycemic index of a particular food in the context of a meal, it is reasonable to choose foods with low glycemic index.

STEPS IN THE MANAGEMENT OF THE DIABETIC PATIENT

DIAGNOSTIC EXAMINATION

An attempt should be made to characterize the diabetes as type 1 or type 2 or other specific types such as MODY, based on the clinical features present and on whether or not ketonuria accompanies the glycosuria.

Features that suggest end-organ insulin insensitivity to insulin, such as visceral obesity, acanthosis nigricans, or both, must be identified. The family history should document not only the incidence of diabetes in other members of the family but also the age at onset, association with obesity, the need for insulin, and whether there were complications.

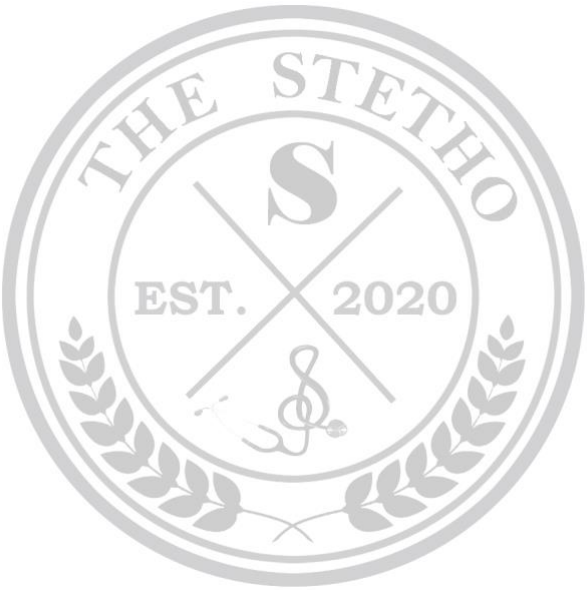
Many patients with newly diagnosed type 1 diabetes still have significant endogenous insulin production, and C peptide levels do not reliably distinguish between type 1 and type 2 diabetes.

Factors that increase cardiac risk, such as smoking history, presence of hypertension or hyperlipidemia, or oral contraceptive pill use, should be recorded.

Laboratory diagnosis of diabetes should document fasting plasma glucose levels above 126 mg/dL (7 mmol/L) or postprandial values consistently above 200 mg/dl (11.1 mmol/L) or HbA1c of at least 6.5% and whether ketonuria accompanies the glycosuria, An HbA1c measurement is also useful for assessing the effectiveness of future therapy.

Baseline values include fasting plasma triglycerides, total cholesterol and HDL cholesterol, electrocardiography, kidney function studies, peripheral pulses, and neurologic,

podiatric. and ophthalmologic examinations to help guide future assessments



PATIENT EDUCATION (Self-Management Training)

Since diabetes Is a lifelong disorder, education of the patient and the family is probably the most important obligation of the clinician who provides care. The best persons to manage a disease that is affected so markedly by daily fluctuations in environmental stress, exercise, diet, and infections are the patients themselves and their families.

The "teaching curriculum" should include explanations by the clinician or nurse of the nature of diabetes and its potential acute and chronic hazards and how they can be recognized early and prevented or treated. Self-monitoring of blood glucose should be emphasized, especially in insulin requiring diabetic patients, and instructions must be given on proper testing and recording of data.

Patients and Signs and symptoms of hypoglycemia: their families Dizziness, unceasing hunger, tremors, and tachycardia, pallor, sweating, lethargy, should be anxiety, blurred vision, headache, difficulty taught to speaking, confusion, irritability, convulsions, recognize signs and coma if not treated on time. and symptoms of hypoglycemia and how to treat low glucose reactions.

Strenuous exercise can precipitate hypoglycemia, and patients must therefore be taught to reduce their insulin dosage in anticipation of strenuous activity or to take supplemental carbohydrate.

Infections can cause insulin resistance, and patients should be instructed on how to manage the hyperglycemia with supplemental rapidly acting Insulin.

Advice on personal hygiene, including detailed Instructions on foot and dental care, should be provided.

THERAPY:

Treatment must be individualized on the basis of the type of diabetes and specific needs of each patient. However, certain general principles of management can be outlined for hyperglycemic states of different types.

CASE 2

TYPE I DIABETES

A combination of rapidly acting insulin analogs and long-acting insulin analogs allows for more physiologic insulin replacement.

Insulin glargine or insulin degludec is usually given once in the evening to provide 24-hour coverage.

There are occasional patients in whom insulin glargine does not last for 24 hours, and in such cases, it needs to be given twice a day.

Insulin detemir usually has to be given twice a day to get adequate 24-hour basal coverage. Alternatively, small doses of NPH (~3-4 units) can be given with each meal to provide daytime basal coverage with a larger dose at night.

The 24-hour basal dosage is usually based on age and body weight. An adolescent might need as much as 0.4 unit/kg/day; young adult (less than 25 years), 0.35 unit per/kg/day; and older adults, 0.25 unit/kg/day.

For example, a 70-kg, 30-year-old person may require a basal rate of 0.7 unit per hour throughout the 24 hours with the exception of 3 am to 8 am, when 0.8 unit per hour might be appropriate (given the "dawn phenomenon" reduced tissue sensitivity to insulin between 5 am and 8 am).

The meal bolus varies based on the time of day and the person age. Adolescents and young adults usually require 1 unit for about 10 g of carbohydrate. Older adults usually require about 1 unit for 15 g of carbohydrate.

The correction factor—how much insulin is needed to lower glucose Levels by 50 mg/dL—can be calculated from the insulin-to-carbohydrate ratios. For example, if 1 unit is required for 15 g of carbohydrate, then 1 unit will lower glucose levels by 50 mg/dL. If 1.5 units of insulin are required for 15 g of carbohydrate (that is, 1 unit for 10 g Carbohydrate), then 1.5 units of insulin will lower glucose levels by 50 mg/dL (that Is, 1 unit will lower glucose level by 33 mg/dL).

For a 70-kg 30-year-old person, bolus ratios of 1 unit for 12-15 g of carbohydrate plus 1 unit for SO mg/ dl of blood glucose over a target value of 120 mg/dL would be reasonable starting point. Further adjustments to basal and bolus dosages would depend on the results of blood glucose monitoring.

One of the more difficult therapeutic problems in managing patients with type 1 diabetes is determining the proper adjustment of insulin dose when the pre-breakfast blood glucose level is high. Occasionally, the pre-breakfast hyperglycemia is due to the Somogyi effect, in which nocturnal hypoglycemia leads to a surge of counter regulatory hormones to produce high blood glucose levels by 7 am. However, a more common cause for pre-breakfast hyperglycemia is the waning of circulating insulin levels by the morning.

The diagnosis of the cause of pre-breakfast hyperglycemia can be facilitated by self-monitoring of blood glucose at 3 a™ in addition to the usual bedtime and 7 am measurements or by analyzing data from the continuous glucose

monitor. This is required for only a few nights, and when a particular pattern emerges from monitoring blood glucose levels overnight, appropriate therapeutic measures can be taken. The Somogyi effect can be treated by lowering the basal insulin dose at bedtime or by eating a snack at bedtime.

When a waning insulin level is the cause, then either increasing the evening basal insulin dose or shifting it from dinnertime to bedtime (or both) can be effective. If this fails, insulin pump therapy may be required. The currently available dosed loop systems enable patients to achieve close to normal glucose levels in the morning with a low risk of nocturnal hypoglycemia.

CASE 3

TYPE II DIABETES

The possibility that the individual patient has a specific etiologic cause for their diabetes should always be considered, especially when the patient does not have a family history of type 2 diabetes or does not have any evidence of central obesity or insulin resistance. Such patients should be evaluated for other types of diabetes such as LADA or MODY

Patients with LADA should be prescribed insulin when the disease is diagnosed and treated like patients with type 1 diabetes.

It is also important to note that many patients with type 2 diabetes mellitus have a progressive loss of beta cell function and will require additional therapeutic interventions with time.

WEIGHT REDUCTION:

One of the primary modes of therapy in the obese patient with type 2 diabetes is weight reduction. Normalization of glycemia can be achieved by weight loss and improvement in tissue sensitivity to insulin.

A combination of caloric restriction, increased exercise, and behavior modification is required if a weight reduction program is to be successful, Understanding the risks associated with the diagnosis of diabetes may motivate the patient to lose weight.

For selected patients, medical or surgical options for weight loss should be considered. Orlistat, phentermine/ topiramate, lorcaserin, naltrexone/extended-release bupropion, and high dose liraglutide (3 mg daily) are weight loss

medications approved for use in combination with diet and exercise.

Non-obese patients with type 2 diabetes frequently have increased visceral adiposity—the so-called metabolically obese normal weight patient. There is less emphasis on weight loss, but exercise remains an important aspect of treatment.

GLUCOSE-LOWERING AGENTS:

The current recommendation is to start metformin therapy at diagnosis and not wait to see whether the patient can achieve target glycemic control with weight management and exercise.

When diabetes is not well controlled with Initial therapy (usually metformin), then a second agent should be added. Presence of cardiovascular or kidney disease, or both, will determine the choice of the second agent.

CASE 4

HYPOGLYCEMIA

HISTORY:

This is when your blood sugar levels become too low. The symptoms include shakiness, pale skin, headache, sweating, hunger, fatigue, dizziness and light headedness.

Hypoglycemia is an abnormally low concentration of blood glucose. severe hypoglycemia can be fatal or lead to irreversible neurological damage. Blood glucose levels should be measured whenever possible jn patients presenting symptoms of hypoglycemia. If hypoglycemia is suspected but blood glucose measurement is not available, glucose (or another available sugar) should be given empirically. Always consider hypoglycemia in patients presenting impaired consciousness (lethargy, coma) or seizures.

Clinical features: Rapid onset of nonspecific signs, mild to severe depending on the degree of the hypoglycemia: sensation of hunger and fatigue, tremors, tachycardia, pallor, sweats, anxiety, blurred vision, difficulty speaking, confusion, convulsions, lethargy, coma.

DIAGNOSIS:

Capillary blood glucose concentration (reagent strip test): non-diabetic patients: Hypoglycemia :< 3.3 mmol/liter (< 60 mg/dl) Severe hypoglycemia :< 2.2 mmol/liter (< 40 mg/dl)

Diabetic patients on home treatment: < 3.9 mmol/liter (< 70 mg/dl) tf blood glucose measurement is not available, diagnosis is confirmed when symptoms resolve after the administration of sugar or glucose.

MANAGEMENT OF HYPOGLYCEMIA:

Conscious patients:

Children: a teaspoon of powdered sugar in a few ml of water or 50 mi of fruit juice, maternal or therapeutic milk or 10 mi/kg of 10% glucose by oral route or nasogastric tube.

Adults: 15 to 20 g of sugar (3 or 4 cubes) or sugar water, fruit juice, Soda, etc. Symptoms improve approximately 15 minutes after taking sugar by Oral route.

Patients with impaired consciousness or prolonged convulsions:

Children: 5 ml/kg of 10% glucose by IV route (2 to 3 minutes) or infusion

Adults: 1 ml/kg of 50% glucose by slow IV (3 to 5 minutes). Neurological symptoms improve a few minutes after the injection.

Check blood glucose after 15 minutes. If it is still low, readminister glucose by IV route or sugar by oral route according to the patient's clinical condition.

If there is no clinical Improvement, differential diagnoses should be considered: e.g., serious infection (severe malaria, meningitis, etc.}, epilepsy.

In all cases, after stabilization, give meal or snack rich in complex carbohydrates and monitor the patients for a few hours.

If patient does not return to full alertness after an episode of severe hypoglycemia, monitor blood glucose levels regularly.

For the treatment of hypoglycemia in a person with impaired consciousness and no established IV access, immediate administration of glucagon is suggested, rather than waiting to establish IV access.

Administration of glucagon (subcutaneous, intramuscular or nasal) will usually lead to recovery of consciousness within 15 minutes, although it may be followed by marked nausea or even vomiting. Dose of glucagon is I mg IM / \V/ SC and 3mg (nasal); May repeat in 15 minutes as needed.

Last but not the least, underlying cause of hypoglycemia should be treated.

Causes other than diabetes: Treat severe malnutrition, neonatal sepsis, severe malaria,

acute alcohol intoxication, etc. End prolonged fast.

Replace drugs inducing hypoglycemia (e.g., quinine IV, pentamidine, ciprofloxacin, enalapril, beta-blockers, high-dose aspirin, tramadol), or anticipate hypoglycemia (e.g., administer quinine IV in a glucose infusion).

In diabetes patients:

Avoid missing meals, Increase intake of carbohydrates, if necessary, Adjust dosage of insulin according to blood glucose levels and physical activity. Adjust dosage of oral anti-diabetics, taking into account possible drug interactions.

CASE 5

DIABETIC KETOACIDOSIS (DKA)

CAUSES: DKA results from insulin insufficiency with a relative or absolute increase in glucagon and may be caused by insufficient or interrupted insulin therapy, infections (pneumonia, urinary tract infection, gastroenteritis, sepsis), infarction (cerebral, coronary, mesenteric, peripheral), emotional stress, excessive alcohol intake, surgery, pregnancy and trauma, and certain drugs such as steroids, cocaine etc.

CLINICAL FEATURES: Polydipsia, polyuria, anorexia, nausea or vomiting, abdominal pain, rapid breathing (kussmaul respiration), fruity breath odor of acetone, fever, tachycardia, hypotension, signs of dehydration (dry skin and mucous membranes and poor skin turgor) and mental status change ranging from altered conscious level to coma.

INVESTIGATIONS: Urgent RBS (RBS > 250 mg/dL), Serum ketones, Urine for Ketones, Serum electrolytes, Serum bicarbonates (>10 mmol/L} and ABGs. Also advise ECG, CXR, urine, sputum and blood cultures.

DIAGNOSIS CONFIRMATION: Elevated blood sugar (RBS > 250), serum/urinary ketones, metabolic acidosis-low serum bicarbonate (< 15) and low blood PH (< 7.3).

MANAGEMENT:

(ADA 2009, AAFP 2013 Guidelines) Admit the patient to ICU for frequent monitoring and pass large bore IV line

1. **Fluid therapy:**

Inf 1 Liter per hour of 0.9% Normal saline over 1-2 hours. After 2 Utre of fluid have been given, the intravenous infusion should be at the rate of 300 400 ml/hr.

Use 0.9% saline the serum sodium is greater than 150 mEq/l, when 045 saine (half normal) saline solution should be used. (CMDT 2022) In our setup for adults usually of L of 0 9% N/S Is given in 1st 30 minutes 4 BP low) 2nd L of 0.9% N/S is given in two-hour and 3rd L of 0.9% N/S is given in four hours. 4th L of 0 9% N/S is given in 8 hours and 5th L of 0 9% N/S is given in 16 hours. Switch to 5% dextrose and 0 45% saline at 150-250 mi/hour when plasma glucose reaches 250 mg/dL.

Monitor labs, urine output, hemodynamics, state of hydration and physical exam to determine adequacy of hydration.

Avoid fluid overload in renal and cardiac patients (Give controlled lV fluids at the rate of 80-100mL/hr).

Excessive fluid replacement (more than 5L in 8 hours) may contribute to acute respiratory distress syndrome or cerebral edema.

2. **Insulin therapy:**

Inj insulin Regular IV (0.1 units/kg) stat, then Inj-insulin Regular IV 0.1 units/kg per hour by continuous IV infusion (a bolus dose is not required if patients are given hourly insulin at 0.14 units/ Kg).

If the plasma glucose level fails to fall at least 10% in the first hour, a repeat loading dose (0.1 or 0.14 unit/Kg) is recommended.

The insulin dosage should be adjusted to lower the glucose concentration by about 5—70mg/dL/hr.

If clinical circumstance prevents the use of an insulin infusion, then the insulin can be given intramuscularly. An initial dose 0.15 unit/Kg of regular insulin is given intravenously and at the same time, the same dose is given intramuscularly. Subsequently regular insulin is given IM hourly at a dose of 0.1 unit/Kg until the blood glucose falls to around 250mg/dL, when the insulin can be given subcutaneously.

If initial serum potassium is < 3.5 mEq/L, do not administer insulin until the potassium is corrected to > 3.5 mEq/L.

3. **Assess the patient:**

What precipitated the episode (noncompliance, infection, trauma, Infarction, cocaine)? Initiate appropriate workup for precipitating event (cultures, chest x-ray, ECG). The agent or event that precipitated DKA should be aggressively treated. Give IV antibiotics in case of infection. If the patient is vomiting or has altered mental status, a nasogastric tube should be inserted to prevent aspiration of the gastric contents.

4. **Measure Electrolytes:**

Random blood glucose 1 hourly, measure electrolytes (especially K+, bicarbonate,

phosphate and magnesium) and anion gap 2 hourly for first 24 hrs.

5. **Monitor:**

blood pressure, pulse, respiration, mental status, fluid intake and output every 1-4 hours.

6. **Electrolytes repletion:**

If the potassium > 6mEq/L, don't give potassium. If the Potassium level is 4.5-6 mEq/L, give 10mEq/hr of KCI. If the potassium level is 3-4.5, give 20 mEq/hr of KCI. Goal is to keep potassium levels at 45 mEq/L. Potassium can be given as follow: two thirds as KCI and one third as KPO4. If pH<6.9, give 100ml of 1.4% sodium bicarbonate (2 amp) in 400 mL sterile water (isotonic solution) with 20 mEq KCI at a rate of 200 mL/h for 2 hours until venous pH is >7.0; if

necessary, repeat every 2 hours until pH >7.0 (AAFP 2013recommends > 6.9)

In DKA, replace phosphate if any of the following: Cardiac dysfunction, anemia, respiratory depression, or phosphate levels <1.0 mEq/dL, or symptoms of hypophosphatemia (there are no Studies on phosphate repletion in HHS). The average deficit of 4050 mmol of phosphate should be replaced intravenously at a rate No greater than 3-4 mmol/hr in a 60 to 70-Kg person.

7. Continue above until patient is stable:

once glucose goal level is achieved (150-250 mg/dL) and acidosis is resolved. Insulin infusion may be decreased to 0.05-0.1 units/kg per hour. When the patient become stable, calculate the

dosage of insulin according to the units of short acting insulin given in the last hours.

8. Administer intermediate or long-acting Insulin:

Insulin as soon as patient is eating. Allow for overlap in insulin Infusion and SC insulin injection.

9. **Transition to Subcutaneous Insulin Regimen:**

Once the DKA is controlled and the patient is awake and able to eat, subcutaneous Insulin therapy can be initiated.

CASE 6

HYPERGLYCEMIC HYPEROSMOLAR STATE (HHS)

It is also known as hyperosmolar non-ketotic state (HONK). It is characterized by severe hyperglycemia (> 600mg/ dL) Serum osmolality greater than 310 m Osm/kg

No acidosis; blood PH > 7.3
Serum bicarbonate greater than 15 mEq/L. Normal anion gap (less than 14 mEq/L).
Serum Osmolality = 2 x Na + BUN / 2.8 + glucose /18 (BUN = 2.14 x Blood urea)

Treatment for HHS remain the same as for DKA except

1. Fluid therapy:

If hypovolemia is present as evidenced by hypotension and oliguria, fluid therapy should be initiated with 0.9% saline. In all other cases, 0.45% saline appears to be preferable as the initial

replacement solution because the body fluids of these patients are markedly hyperosmolar.

As much as 4-6 L of fluid may be required in the first 8-10 hours.

Careful monitoring of the patient is required for proper sodium and water replacement. An important end point of fluid therapy is to restore urinary output to 50 mL/h or more. Once blood glucose reaches 250 mg/dl (13.9 mmol/L), fluid replacement should include 5% dextrose in either water, 0.45% saline solution, or 0.9% saline solution, The rate of dextrose infusion should be adjusted to maintain glycemic levels of 250 300 mg/dL (13.9-16.7mmol/L) in order to reduce the risk of cerebral edema.

2. Insulin therapy: Less insulin may be required to reduce the hyperglycemia in nonketotic patients as compared to those with diabetic ketoacidotic coma. In fact, fluid replacement alone can reduce hyperglycemia considerably by correcting the hypovolemia, which then increases both glomerular filtration and renal excretion of glucose. Insulin treatment should therefore be delayed unless the patient has significant Ketonemia (beta-hydroxybutyrate more than 1 mmol/L).

Start the insulin infusion rate at 0.05 unit/kg/h (bolus is not needed) and titrate to lower blood glucose levels by 50-70 mg/dl per hour (2.8-3.9 mmol/L/h). Once the patient has stabilized and the blood glucose fails to around 250 mg/dL (13.9 mmol/L), insulin can be given subcutaneously.

3. Electrolytes replacement:

With the absence of acidosis, there may be no initial hyperkalemia unless associated end-stage chronic kidney disease is present. This results in less severe total potassium depletion than in DKA, and less potassium replacement is therefore needed.

Potassium chloride (10 mEq/L) can be added to the initial infusion of fluids administered if the patient's serum potassium is not elevated.

If severe hypophosphatemia (serum phosphate less than 1 mg/dL [0.32 mmol/L]) develops during insulin therapy, phosphate replacement can be given as described for ketoacidotic patients (at 3 mmol/h).

CASE 7

HYPOTHYROIDISM AND MYXOEDEMA

Autoimmune (Hashimoto) thyroiditis is the most common cause of hypothyroidism.

Fatigue, cold intolerance, constipation, weight change, depression, menorrhagia, hoarseness.

Dry skin, bradycardia, and delayed return of deep tendon reflexes.

FT4 level is usually low

TSH elevated in primary hypothyroidism.

Investigations: All BLIS, ECG, TFTS, Serum calcium, CXR, and anti-thyroid peroxidase antibody.

MANAGEMENT

Before beginning therapy with thyroid hormone, hypothyroid patient requires clinical assessment for adrenal insufficiency and angina.The presence of either condition requires further evaluation and management.

TREATMENT OF HYPOTHYROIDISM:

Otherwise, healthy young and middle-aged adults with hypothyroidism may be treated initially with levothyroxine in average doses of about 1.6 mcg/kg/day.

Tab Levothyroxine 50-200 ig (1.6mcg/kg) 1 tab PO x OD.

CASE 8

HYPOTHYROIDISM AND PREGNANCY

Pregnant women with overt hypothyroidism or myxedema should be treated immediately with levothyroxine at full replacement doses of 1.6 mcg/kg/day

Tab Levothyroxine 100-150 mcg 1 tab PO x OD.
Patients with stable coronary artery disease or those who are over age 60 years are treated with smaller initial doses of levothyroxine, 25-50 mcg orally daily.
Tab Levothyroxine 25-50 mcg 1 tab PO x OD.
The dose can be increased by 25 mcg every 1-3 weeks until the patient is euthyroid.

Dose after delivery.
Measure TSH after 2 months of Levothyroxine or if dose change symptoms relieve at 3 to 6 months after normal TSH.

CASE 9

MYXEDEMA CRISIS

TREATMENT OF MYXEDEMA CRISIS:

Myxedema crisis is a medical emergency and need ICU admission for close monitoring.

Airway management and mechanical ventilation should be instituted for respiratory failure (Patients with hypercapnia).

The hypothermic patient is warmed only with blankets, since faster warming can precipitate cardiovascular collapse. Hypoglycemic patients are given 5% dextrose intravenously.

Hyponatremic patients with a serum sodium 120-130 mEq/mL are administered 0.9% NaCl intravenously, while patients with a serum sodium below 120 mEq/mL are treated with boluses of 100 mL of 3% NaCl intravenously

with Inj-Lasix (furosemide) 20-40 mg IV x BD (to promote water diuresis).

Patients in whom concomitant adrenal insufficiency is suspected are treated with hydrocortisone, Inj Solucortef (hydrocortisone) 100 mg IV x stat, then 25-SO mg Vx QID.

Myxedema crisis requires larger initial doses of levothyroxine intravenously, since myxedema itself can interfere with intestinal absorption of oral levothyroxine.

IV T4: initial dose of 300-S00mcg, followed by daily |V doses of 50-100mcg until the patient can take oral T4. If there is no improvement within 24~48h, add IV T3 (Tropstat 10mcg) IV x TDS for the first 48 hours, Infections must be detected

and treated aggressively with broad spectrum antibiotics accordingly.

NOTE:

Always start with low dose and adjust according to TSH levels: Dose may be doubled during pregnancy and returned to normal.

Follow up TSH at 2nd and 3rd year once TSH levels are normal.

CASE 10

HYPERTHYROIDISM (THYROTOXICOSIS)

Characteristic Features: Sweating, weight loss or gain, anxiety, palpitations, loose stools, heat intolerance, menstrual irregularity.

Tachycardia; warm, moist skin; stare; tremor.

Graves' disease: most common cause of hyperthyroidism; palpable goiter (sometimes with bruit) in most patients; ophthalmopathy also common.

Suppressed TSH in primary hyperthyroidism; usually increased T4, FT4, T3, and FT3.

MANAGEMENT

Tab Inderal (Propranolol) 40mg, 1-3 tablets PO x QID "OR" 0.5-2 mg IV every four hours. Use cautiously in the presence of heart failure. weeks then 1 tab BD or TDS for 6-18 months "OR"

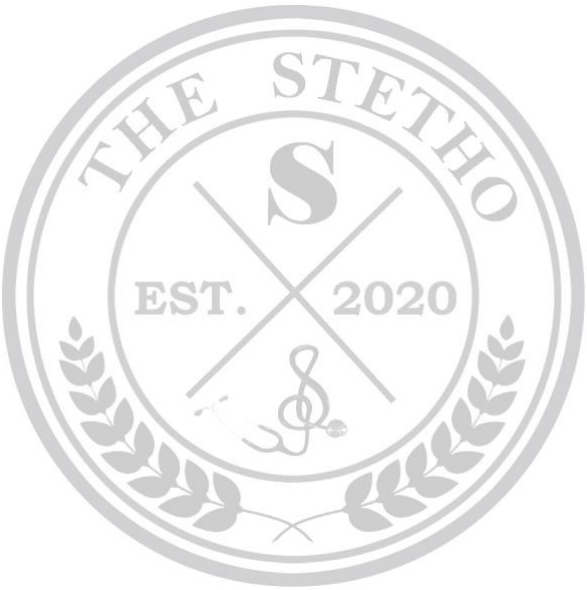
Tab Procarbizole (prophyithiouracil) SOmg, 2-3 tablets PO x QID (300-600mg/day in 4 divided

doses). During pregnancy the dose of prophylthiouracil is kept below 200 mg/day to avoid goitrous hypothyroidism in the infant.

In-case.of Thyrotoxicosis, also: Use iodized salt and Schiller's iodine 2 drops (460 micrograms) once daily for one year. Response may be obtained within 6 months "OR" Lugol's solution 3 drops (21mg) once each month for up to one year until the patient become euthyroid.

CAUTION: Carbimazole may induce bone marrow suppression, Patients should be told to report any type of infection. The drug should be stop ed immediatel if there is neutropenia. Tab Neo-mercazole (Carbimazole) 5mg, 2-4 tablets PO x TDS

Symptomatic therapy for other complaints and treatment of the Definitive surgery with radioactive iodine or surgery is delayed



CASE 11

IMPOTENCE / ERECTILE DYSFUNCTION

Erectile dysfunction can have organic and psychogenic etiologies, and the two frequently overlap.

Organic erectile dysfunction may be an early sign of cardiovascular disease and requires evaluation.

Peyronie disease is a common, benign fibrotic disorder of the penis that causes pain, penile deformity, and sexual dysfunction.

MANAGEMENT

Life style modification and reduction of cardiovascular risk factors are important components of any treatment plan. This should potentially include Smoking cessation, reduction of alcohol intake, diet, exercise, and treatment of diabetes, dyslipidemia, and hypertension.

Hormone replacement therapy.

Counselling and anti-depressants i.e Tab Everlong (Dapoxetine) 60mg, 1 tab PO 3 hours before activity) 50-100mg, 1 tab PO x OD for 3 months.

Tab Freedep (Sertraline)

Tab Viagra (sildenafil) 25-100mg, 1 tab PO 30min before activity

Vacuum erection devices and penile prosthetic surgery.

CASE 12

COMMONLY USED MEDICATIONS IN ENDOCRINOLOGY UNIT

- Drug Brands Gliclazide Tab. Diamicron MR 30, 60mg

- Carbimazole Tab. Neomercazole 5 mg Maint. Dose: 5-15 mg daily

- Tab. Daonil 5 mg Glibenclamide

- Tab. Euglucon 5 mg

- Tab. Getryl 1,2,3,4 mg

- Tab. Evopride 1,2,3,4 mg

- Tab. Evopride Plus Glimepiride + 1/500, 2/500 mg Metformin

- Tab. Getformin 1/500, 2/500 mg

- Hydrocortisone Inj. Solu-Cortef 100,250,500 mg

- Tab. Glucophage Metformin 250,500,850 mg,1g

- Tab. Piozer 15,30,45 mg Pioglitazone

- Tab. Glitos 15,30,45 mg Pioglitazone

- Tab. Piozer Plus Pioglitazone + Metformin 15/500 or 15/850 mg

- Prednisolone Tab. Deltacortil 5 mg

- Tab. Prednisolone 5mg