

DIABETES MELLITUS

DEFINITION

- A state of sustained high blood sugars leading to various complications, due to deficiency of insulin or resistance to it.

CLASSIFICATION

- 1. Type 1 DM
- There is severe or absolute lack of insulin, Normally diagnosed at < 30 years.
- 2. Type 2 DM
- Relative deficiency of insulin with resistance to its action. Normally in adults.
- 3. Type 3 DM
- Caused by multiple non-pancreatic diseases

- Especially hormonal like acromegaly and Cushing's disease, administered drugs-steroids. Potent diabetics are those with first degree relatives with diabetes.
- 4. Type 4
- Gestational diabetes. Seen in 4% of all pregnancies. Resolves after delivery.

AETIOLOGY

- 1.Type 1 is autoimmune or idiopathic
- 2.Genetic defects- in beta cell function e.g glucokinase(MODY2), mitochondrial DNA mutations.
- 3. Diseases of pancreas –pancreatitis, pancreatectomy, neoplasm, cystic fibrosis, hemochromatosis,
- 4. Endocrinopathies(hormonal) -

- Acromegaly, Cushings disease, glucagonoma, phaeochromocytoma, hyperthyroidism, somastatinoma, aldosteronoma
- 5. Drugs or chemical induced –glucocorticoids, thyroid hormones, thiazides, diazoxide, phenytoin, beta adrenergic agonists, beta blockers, protease inhibitors
- 6. Infections –congenital rubella, CMV, coxsachie virus

- 7. Other syndromes associated with diabetes
 - Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Friedrick's ataxia, Huntington's chorea, myotonic dystrophy, porphyria
- 8. Gestational diabetes

EPIDEMIOLOGY

- Prevalence has risen dramatically in the past two decades.
- In 2000 prevalence 0.19% in <20 years, 8.6% in >20 years and 20.1% in those > 65 years. Male female ratios basically the same, though slightly higher in men.
- Scandinavia has the highest rates, 35/100,000 per year, in type1 DM, the Asiatics have the lowest, China, Japan, 1-3/100,000 per year and Northern Europe and USA have intermediate rates.

- Type 2 is highest in the Pacific islands, intermediate in India and USA and lowest in Russia and China.
- Incidence variation is due to genetic, environmental and behavioural factors.

RISK FACTORS FOR TYPE 2 DIABETES

- 1. Family history of diabetes in first degree relative
- 2. Obesity, BMI > 25
- 3. Sedentary life
- 4. Race /ethnicity –
- 5. Previously identified IGT (tolerance) OR IFG(fasting)
- 6. History of GDM or baby over 4kg

- 7. Hypertension
- 8. High Triglyceride levels or low HDL cholesterol levels.
- 9. Polycystic ovarian syndrome
- 10. History of vascular disease

CLINICAL PRESENTATION

- Typically Polyuria, polydipsia and polyphagia
- Weight loss
- Repeated infections/wounds
- Dehydration
- Weakness/dizziness/poor vision
- Acidotic breathing and coma

COMPLICATIONS

- 1. Diabetic keto acidosis
- 2. Non-ketotic hyper-osmolar coma
- 3. Hypoglycemic comas
- 4. Repeated infections
- 5. Diabetic foot
- 6. Retinopathy
- 7. Renal disease
- 8. Hypertension

- 9. Strokes – ischemic
- 10. Myocardial infarction(heart disease)
- 11. Diabetic diarrhoea/gastroparesis,
dysmotility
- 12. Neuropathy- peripheral, autonomic
- 13.Cystopathy/erectile dysfunction/female sexual dysfunction/retrograde ejaculation
- 14.Dermatological disorders

TREATMENT

- 1. Diet
- 2. Lifestyle change
- 3. Exercise
- 4. Education on body care/ hygiene/disease
- 5. Insulin/oral drugs
- 6. Follow up for life

PREVENTION

- NEWSTART

DIABETIC COMAS

- CAUSES:
- 1. DKA
- 2. Non-ketotic Hyperosmolar hyperglycemic coma
- 3. Hypoglycemias

DIABETIC KETOACIDOSIS

- Medical emergency
- Sugars – high, $>22.4\text{mmol/l}$
- Polyuria/polydipsia
- Kussmaul's respiration
- Severe dehydration
- Comatose
- Ketones In urine, and ABGS-severe met. Acid.

- May have history of nausea/ vomiting, abdominal pain
- Physical examination shows tachycardia, severe dehydration, hypotension, tachypnea with acidotic breathing, abdominal tenderness, lethargy, obtundation, altered mental state and may be in coma.

- DIAGNOSIS
- History of diabetes usually,
- The typical presentation history
- Presentation may be atypical.
- Physical examination- typical features
- Blood sugar
- Urinalysis
- ABGs

TREATMENT

- 1. Soluble insulin, 10 units IV stat, 10 units IM stat, then 6-8 units per hour infusion in adults. 0.1 units/kg/hr in children. Sugars 1 hourly.
- Sugar should be falling by 5mmol/hr, if not by 2-4 hrs double or triple, max 10 fold increase.
- Once sugar is 10 mmol/l and acidosis resolved then give 0.05-0.1 units/kg/hr till ready to take orally in adults, children 0.02 units/kg/hr.

- When settled, taking orally, calculate total dose used, take $\frac{2}{3}$ of total dose required to control sugars/24hrs, to be the dose of mixtard or lente insulin. Give $\frac{2}{3}$ am, $\frac{1}{3}$ nocte.
- Monitor daily and adjust up or down to control sugars. Goal is Fasting sugar $< 8\text{mmol/l}$.
- At discharge, reduce dose by 10%.

- 2. Rehydration – N/S
- Total fluid lose usually 6-8 litres. Replace $\frac{1}{2}$ in first 4 hours, the rest over 24-36 hours.
- 1 litre in first $\frac{1}{2}$ hour
- 1 litre in next 1 hour
- 1 litre in next 2 hours
- Then 0.45% saline 150-300mls/kg/hr.
- When sugars < 14 mmol/l, alternate 0.45% saline with 5% Dextrose at 100-200mls/hr.

- 3. KCL – always assume a potassium deficit
- Give 10meq/hr if plasma K <5.5meq/l, ECG normal, urine flow and normal creatinine documented. Give 40-80meq/hr if plasma K < 3.5 meq/l or if bicarbonate added.
- If initial serum K is <3.3meq/l, do not give insulin until it is corrected to >3.3meq/l

- 4. PH 7.1 – 7.2, 40Meq HCO/m2
- PH <7.1 , 80Meq/m2
- Infused over 2 hrs
- PH normally corrects itself when there's adequate hydration with saline at > 7.2

- 5. Look for precipitating cause and treat.
- Usually infections, or non-compliance. Others trauma, infarction, drugs-cocaine, alcohol etc
Hemogram, cultures, CXR, ECG
- 6. Measure glucose 1-2 hourly, electrolytes and phosphate, Calcium, magnesium, ABGs and anionic gap 4 hourly for first 24 hrs.
- Monitor BP, pulse, respiration, mental status and fluid input/output every 1-4 hrs

- Phosphate needs replacement only if $<0.32\text{mmol/l}$. Hypomagnesimia may also need replacement

HYPEROSMOLAR NON-KETOTIC HYPERGLYCEMIC COMA

- Usually in elderly living alone, no care, no food, unable to look after themselves. They have mental confusion, lethargy or coma.
- Physical examination reveals profound dehydration, hyperosmolality, hypotension, tachycardia, and altered mental status.
- May be precipitated by AMI or stroke. Others - pneumonia, sepsis, or other serious infection.

- Sugars usually over 55.5 mmol/l, hyperosmolality >350mOsmol/l, prerenal azotemia. Na normal or slightly low. Acidosis or ketonemia is mild or absent.
- Treatment:
- Soluble insulin
- Rehydrate with N/S
- Same treatment regimen. Will require more fluids and longer treatment.

- If serum sodium is $>150\text{mmol/l}$, use 0.45% saline. The water deficit replace over 1-2 days, 200-300mls/hr.
- Initial sol. Insulin same as in DKA, then infusion of 3-7 units per hour. Once sugars $< 14 \text{ mmol/l}$, reduce rate to 1-2 units/hour.
- Graduate as in DKA.

HYPOGLYCEMIC COMAS

- Dramatically recovers on giving sugars, tea, food, IV 50% dextrose.
- Usually due to missing meals, while taking hypoglycemics, overdosing, improvement.
- Education
- Carrying of sugars around.