

# Complications of Juvenile Diabetes

Joanna Fernandes

9<sup>th</sup> Semester

### **Acute complications**

- **Diabetic ketoacidosis**
- **Hypoglycaemia**

### **Intermediate complications**

- **Lipoatrophy**
- **Limited joint mobility**
- **Growth failure**
- **Delay in sexual maturity**
- **Hypoglycaemic unawareness**

### **Chronic complications**

- **Retinopathy**
- **Nephropathy**
- **Dyslipidaemias**
- **Coeliac disease**



## Diabetic ketoacidosis

- × Most severe complication of diabetes mellitus
- × State of hyperglycaemic dehydration with ketotic acidaemia
- × Characterised by-
  - Hyperglycaemia - >300mg/dl
  - Ketonaemia – positive at greater than 1:2 dilution
  - Acidosis –  $\text{pH} < 7.3$ ,  $\text{HCO}_3^- < 10\text{mEq/l}$

# Pathophysiology

Insulin deficiency

Hyperglycaemia

Activation of lipolysis

increased catabolism

osmotic diuresis

release of cellular  
 $\text{Na}^+, \text{K}^+, \text{PO}_4^{2-}$

Increased free fatty acids

Increased metabolism of fatty acids

- Dehydration
- Activation of RAAA
- Loss of electrolytes

Increased ketone bodies

Ketonuria

Acidosis

## Clinical features

× Symptoms:

- polydipsia, polyphagia, weakness ; anorexia, nausea, vomiting, abdominal pain. Most patients present with altered sensorium, and around 10% are comatose.

× On examination:

- Fruity or musty odour in breath
- Loss of skin turgor, dry tongue, sunken eyes
- Hypothermia
- Tachycardia
- Hyperpnoea or Kussmaul's breathing
- Hyporeflexia
- Hypotonia,
- Stupor or Coma



## Investigations

- × Serum glucose – >300mg/dl ( N: fasting : 60 -105 mg/dl)
- × Ketones : total concentration from 3mM/lt to 30mM/lt (N: 0.15mM/lt)
- × Acidosis :
  - Serum bicarbonate level <15mEq/lt ( N: 18 – 24mM/L)
  - pH is <7.3 ( N : 7.35 – 7.45)
- × Electrolytes : Serum sodium- hyponatraemia
  - Serum potassium – low, normal or high
  - Serum phosphorus – intracellular hypophosphataemia



## **Management**

- × **General**
  - **Monitor vitals and neurological status**
  - **Monitor serum glucose levels, serum ketones, electrolytes,**
  - **Maintain input output chart**
  - **For a patient in shock, nasogastric tube should be placed and bladder catheterised.**
  - **Continuous ECG monitoring to diagnose potassium fluctuations**



× **Fluids**

**1. Intravascular fluid replacement**

- Mainstay of treatment in DKA
- Isotonic saline or ringer lactate can be used
- Rate of infusion -5-10ml/kg/hour

**2. Insulin therapy**

- Insulin as 0.10U/kg I.V. Infusion
- Insulin should be given until DKA resolves
- Decrease in blood glucose of 50 to 75mg/dl/hr is appropriate.





### **3. Treatment of potassium level derangement**

- Wait for serum K<sup>+</sup> report before adding KCl to the drip
- When hypokalaemia is seen, infuse 20-30mEq/hour with continuous monitoring of potassium levels and ECG

### **4. Bicarbonate**

- Indications are, life threatening hyperkalaemia, lactic acidosis complicating DKA, severe acidosis

**DIABETIC KETOACIDOSIS (DKA) TREATMENT PROTOCOL**

TIME	THERAPY	COMMENTS
1st hr	10-20 mL/kg IV bolus 0.9% NaCl or RL Insulin drip at 0.05 to 0.10 units/kg/hr	Quick volume expansion; may be repeated. NPO. Monitor I/O, neurologic status. Use flow sheet. Have mannitol at bedside; 1g/kg IV push for cerebral edema.
2nd hr until DKA resolution	0.45% NaCl: plus continue insulin drip 20 mEq/L KPhos and 20 mEq/L KAc 5% glucose if blood sugar >250 mg/dL (14 mmol/L)	If K <3 mEq/L, give 0.5 to 1.0 mEq/kg as oral K solution OR increase IV K to 80 mEq/L
Variable	Oral intake with subcutaneous insulin	No emesis; CO <sub>2</sub> ≥16 mEq/L; normal electrolytes
<p>Note that the initial IV bolus is considered part of the total fluid allowed in the 1st 24 hr and is subtracted before calculating the IV rate.</p>		
<p>Maintenance (24 hr) = 100 mL/kg (for the 1st 10 kg) + 50 mL/kg (for the 2nd 10 kg) + 25 mL/kg (for all remaining kg)</p>		
<p>Sample calculation for a 30-kg child:</p>		
<p>1st hr = 300 mL IV bolus 0.9% NaCl or LR</p>		
<p> </p>		

## *Hypoglycaemia*

- × Defined as blood sugar less than 60mg/dl
  - × Counter regulatory hormones are secreted- adrenaline, glucagon, and cortisol
  - × Adrenergic symptoms are seen like sweating, palpitations & tremors
  - × If left untreated can cause symptoms like seizures, fainting and coma
  - × Treatment : Rule of 15 - Give 15g of free sugar and check glucose level in 15 minutes
- If the child is unconscious, glucagon is given I.M., 0.3mg in infants, 0.5mg in a child <25kg and 1mg in a child >25kg
- × Prevention : by discussing treatment changes to be followed by the patient in case of increase activity



## Intermediate complications

- × Lipoatrophy- fat atrophy at the injection site. Can be prevented by rotation of injection sites
- × Limited joint mobility- typically in hands, flexor contractures occur causing the 'prayer sign'
- × Growth failure if DM is not well controlled
- × Delay in sexual maturity is associated with inadequate control of diabetes and delayed bone age
- × Hypoglycaemic unawareness: caused by frequent hypoglycaemia along with tight metabolic control. Attacks can be reduced by raising target blood sugar levels and decreasing the possibility(avoidance) of hypoglycaemia.

Lipoatrophy at site of injection of insulin



## PRAYER SIGN

A positive "prayer sign" can be elicited on examination with the patient unable to approximate the palmar surfaces of the phalangeal joints while pressing their hands together.

Seen in diabetics

; This represents:- cervical spine immobility and the potential for a difficult endotracheal intubation.

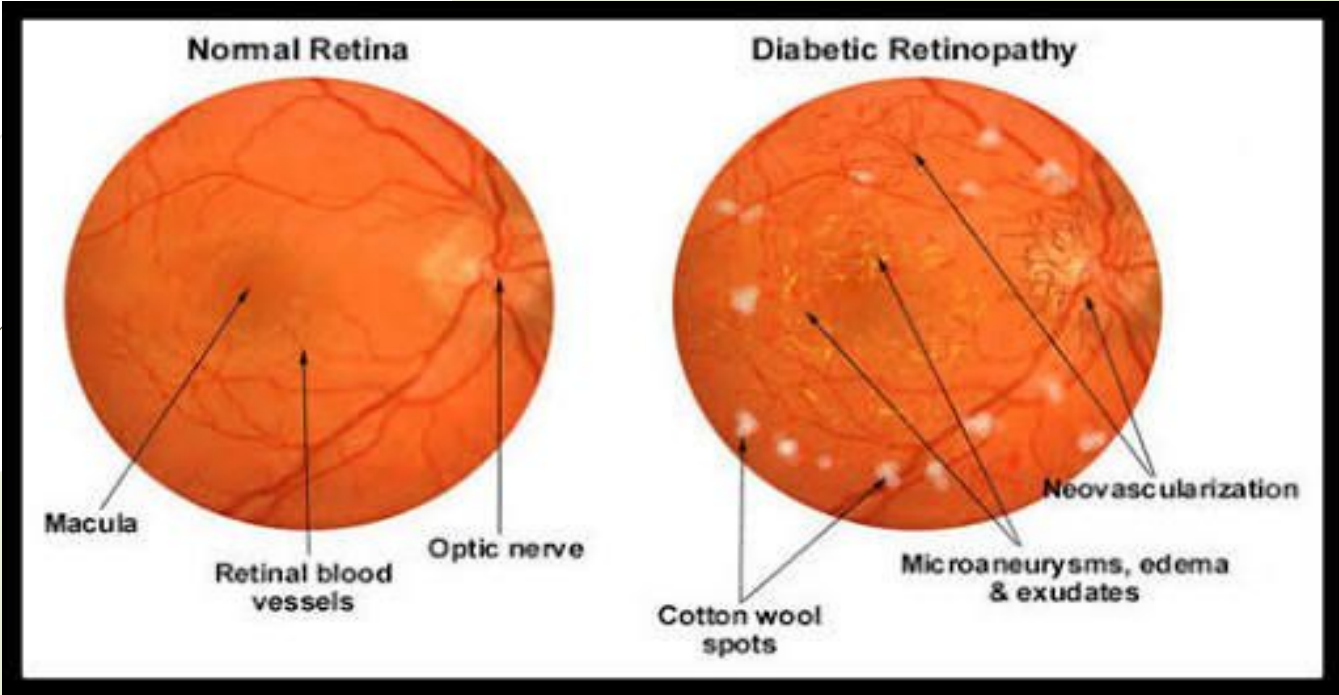




## Chronic complications

× Diabetic retinopathy:

- It can occur within 15 years of diabetes, but it can be postponed to adolescence by intensive management of juvenile diabetes
- It is characterised by micro-aneurysms and proliferative changes in retina
- Ophthalmological examination should be conducted once the child reaches 10yrs of age and has had diabetes for 3-5yrs
- Annual follow-up is suggested





× Diabetic nephropathy :

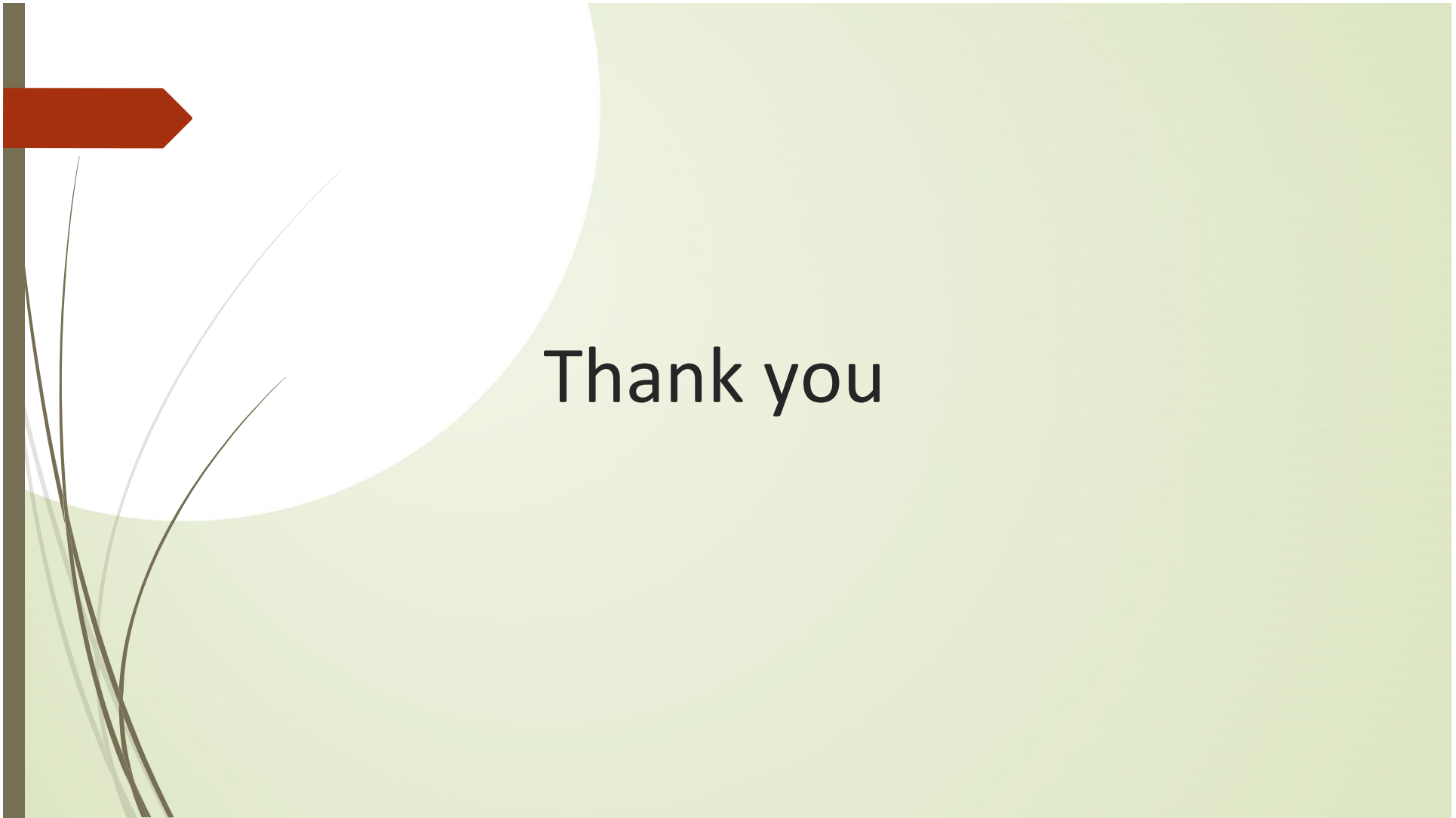
- Defined as albuminuria, which may be preceded by micro-albuminuria
- It causes significant morbidity and mortality in adulthood
- Annual screening needs to be done, once the child reaches 10 years of age or has had diabetes for 5 years
- Patients with raised micro-albumin to creatinine ratio (N:<30mg/g) should receive ACE inhibitors to delay progression to nephropathy





× **Dyslipidaemias**

- **Fasting lipid profile is performed on all pre-pubertal children, if there is a family history of elevated cholesterol or cardiovascular event**
- **If there is no relevant family history of dyslipidaemias, screening is started after the onset of puberty**
- **If profile is normal, test is repeated every 5 years. If lipid profile are abnormal, annual monitoring is recommended**
- **Intervention is required if fasting LDL is greater than 100mg/dl after glucose control has been established**
- **Initial therapy is diet based**
- **If it fails, pharmacological therapy is added**



Thank you