Critically unwell child - Cases

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Airway Case

- 6 week old infant;
- Otherwise fit and well, no feeding issues;
- 3 day history of coryzal symptoms;
- 24 hours of poor feeding and ↑respiratory effort;

O/E:

↑RR, Subcostal recession, Sats 92% in 50% O2;

Capillary gas: pH:7.2, pCO_2: 9 kPa , pO_2: 7 kPa , B.E: -5.0

Airway Case

- Diagnosis?
- Immediate management?
- KIDS team transport arranged…further management for transfer?

Airway Case

- A: Patent or added sounds ?stridor ?wheeze ?dribbling;
- B: Air entry? Added sounds? Accessory muscle use? Tracheal tug? Sternal/intercostal/subcostal recession? Cyanosis? Oxygen saturations?
- C: Cap refil? Heart rate? NIBP?
- D: AVPU, Blood glucose?, Temp?
- E: Urine output: Wet nappies?

Airway Case

Table 2 Signs of increased work of breathing

Ventilatory frequency	
Infant	>50 bpm
Child	>30 bpm
Effort	
Infant	Head bobbing; nasal flaring
Child	'See-saw' chest and abdomen; recession: subcostal, sternal, intercostal, tracheal tug; nasal flaring
Posture	
Infant	Arching backwards
Child	Tripod position
Noise	Infants grunt to generate auto-CPAP; wheezing can occur with an inhaled foreign body; stridor
Ineffective breathing	Hypoxaemia and hypercarbia produce tachycardia, sweating, restlessness and confusion, agitation and anxiety, pallor, or mottling
Impending respiratory arrest	Decreased conscious level, slowing ventilatory frequency, episodes of apnoea, silent chest despite vigorous respiratory effort, bradycardia

Airway Case

- Investigations:
  - Capillary gas;
  - Capillary Blood glucose;
  - FBC, Blood cultures;
  - Throat viral swabs and bacterial cultures;
  - AP and Lateral x-rays;
  - …CT (May require anaesthesia to facilitate this)

Airway Case

Table 1 Causes of respiratory distress

Site	Infective cause	Non-infective cause
Upper airway	Uvulitis	Foreign body
	Epiglottitis	Vocal cord dysfunction
	Croup	Anaphylaxis
	Retropharyngeal/ peritonsillar abscess	Tumours
Lower airway	Tracheitis	Asthma
	Bronchioloitis	Anaphylaxis
		Hilar tumours
		Vascular abnormalities
Pulmonary	Pneumonia	Pulmonary oedema
	Empyema	Pneumothorax
		Pulmonary infiltrations (e.g. fibrosis, oncological, and autoimmune conditions)

Airway Case

Table 1 Correlation between phase of stridor and probable level of airway obstruction

Stridor	Level of obstruction
Inspiratory	Above the cords (extrathoracic); e.g. croup, epiglottitis
Expiratory	Below the cords (intrathoracic); e.g. foreign body
Biphasic	At or below the cords (intra or extrathoracic); e.g. foreign body, bacterial tracheitis

Table 3 Croup score.[4] Mild croup, 0–3; moderate croup, 4–6 (transfer to HDU); severe croup, 7–10 (patient requires tracheal intubation)

Score	0	1	2
Breath sounds	Normal	Harsh, wheeze	Delayed
Stridor	None	Inspiratory	Inspiratory and expiratory
Cough	None	Hoarse cry	Bark
Recession/flaring	None	Flaring, suprasternal recession	Flaring, suprasternal and intercostal recession
Cyanosis	None	In air	In oxygen 40%

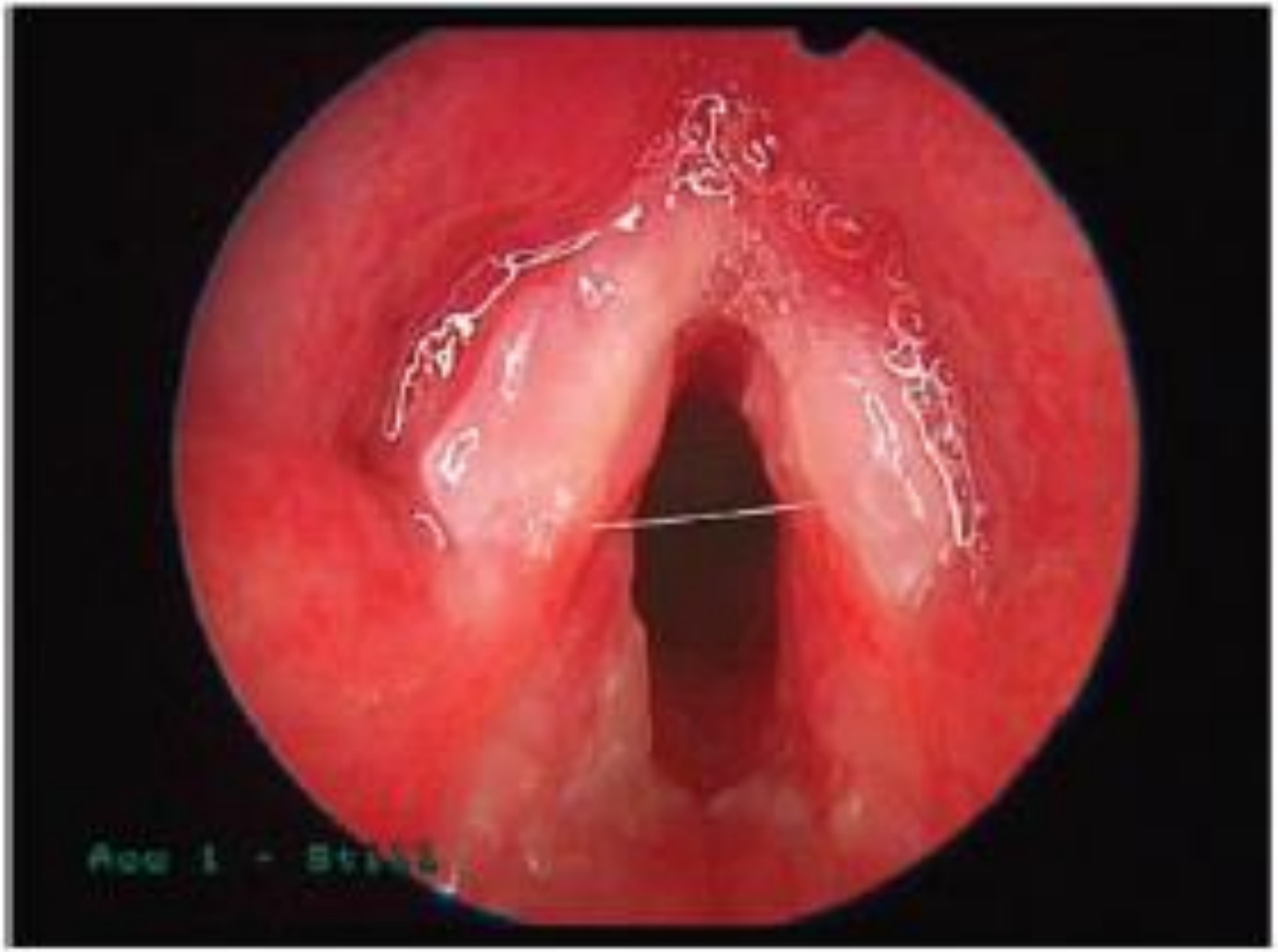


Fig 1 Croup.

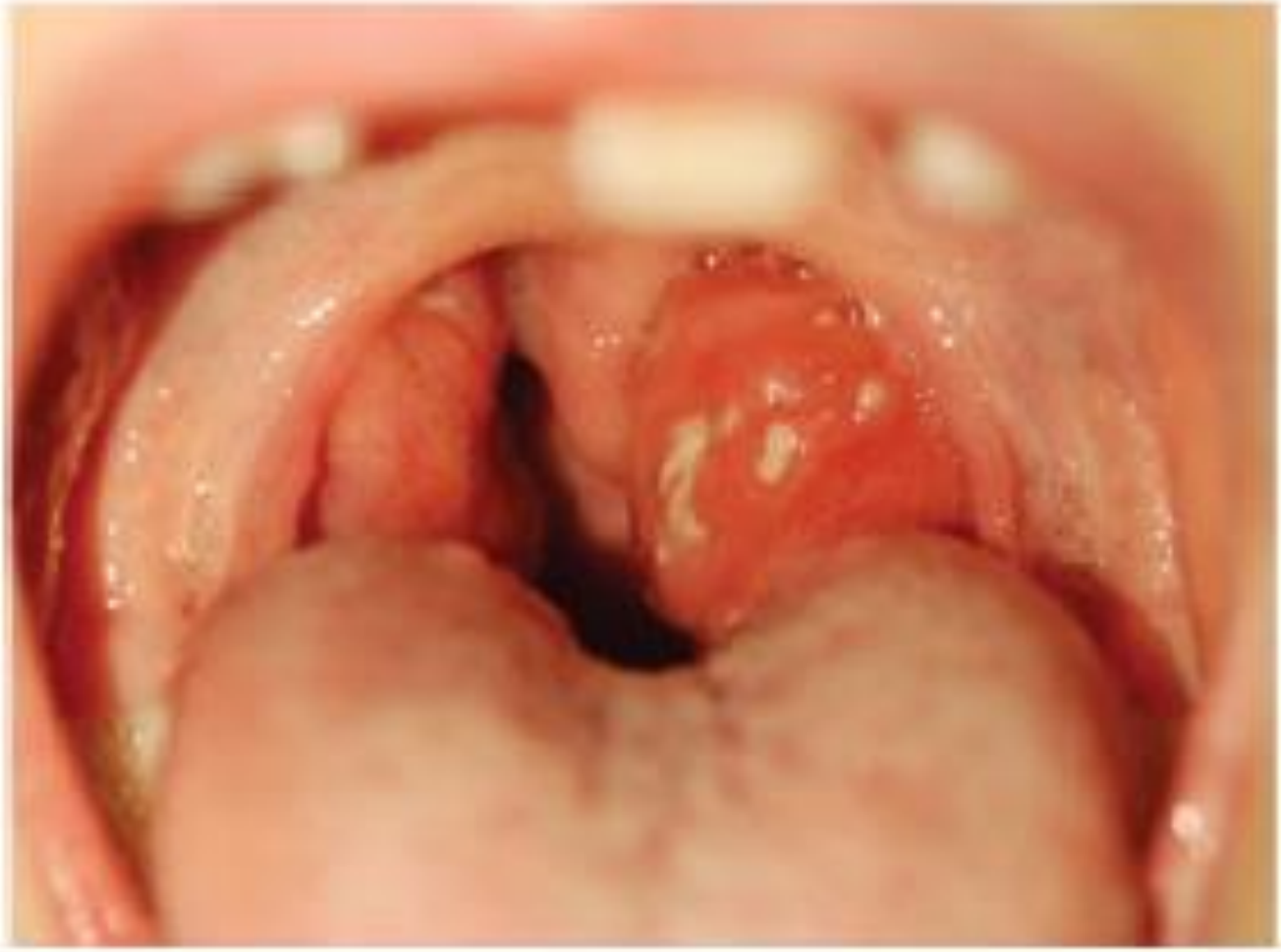


Fig 2 Tonsillitis with airway narrowing.

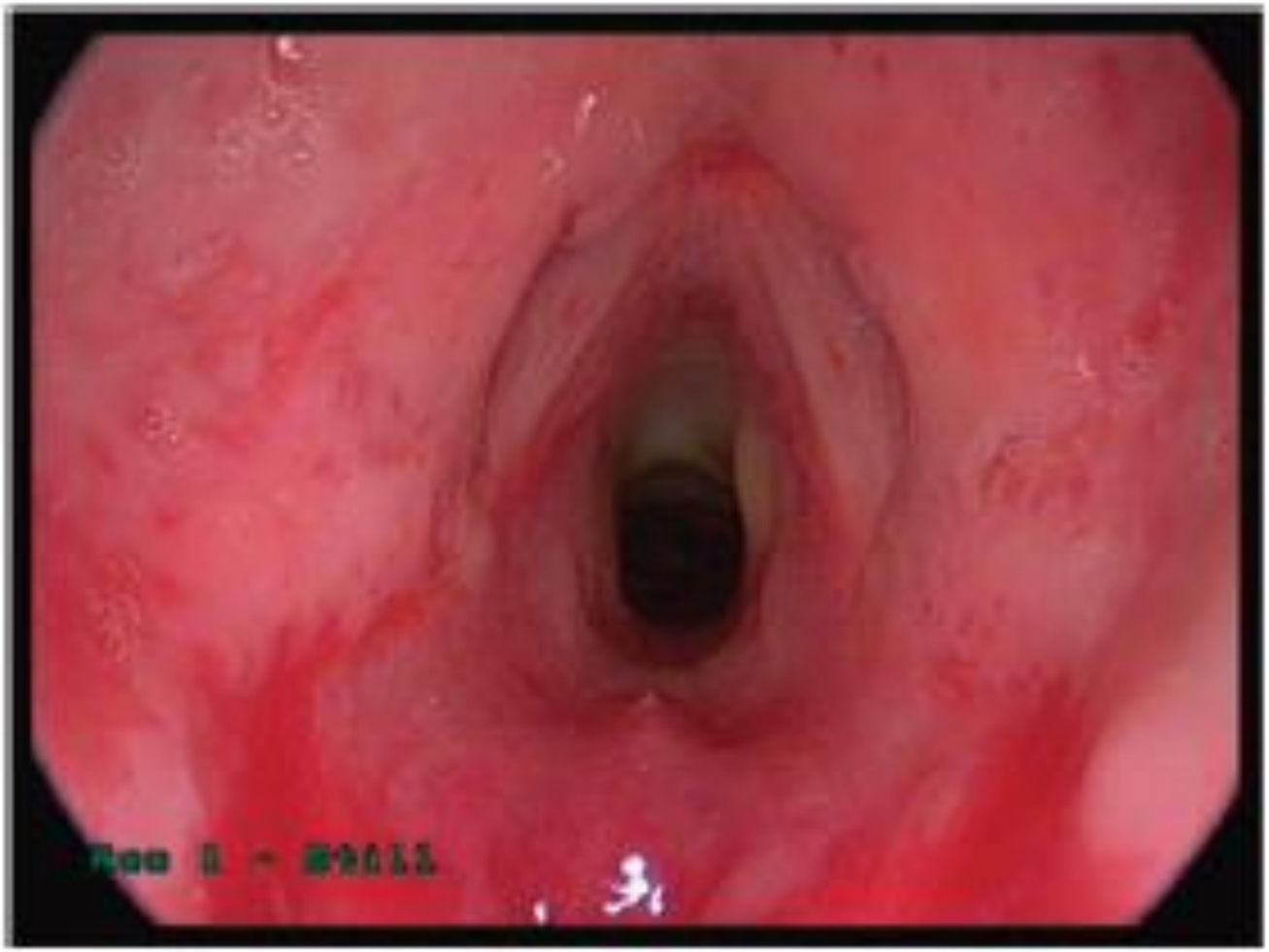


Fig 4 Bacterial tracheitis significant with erythema and oedema.

Sepsis Case

- 5 day old;
- 24 hour history of poor feeding and unresponsive;

In A&E:

A: Patent, B: ↑RR, C: ↓cap refil, weak pulses + unrecordable BP, D: ↓Blood glucose (1.2 mmols), E: AVP**U**

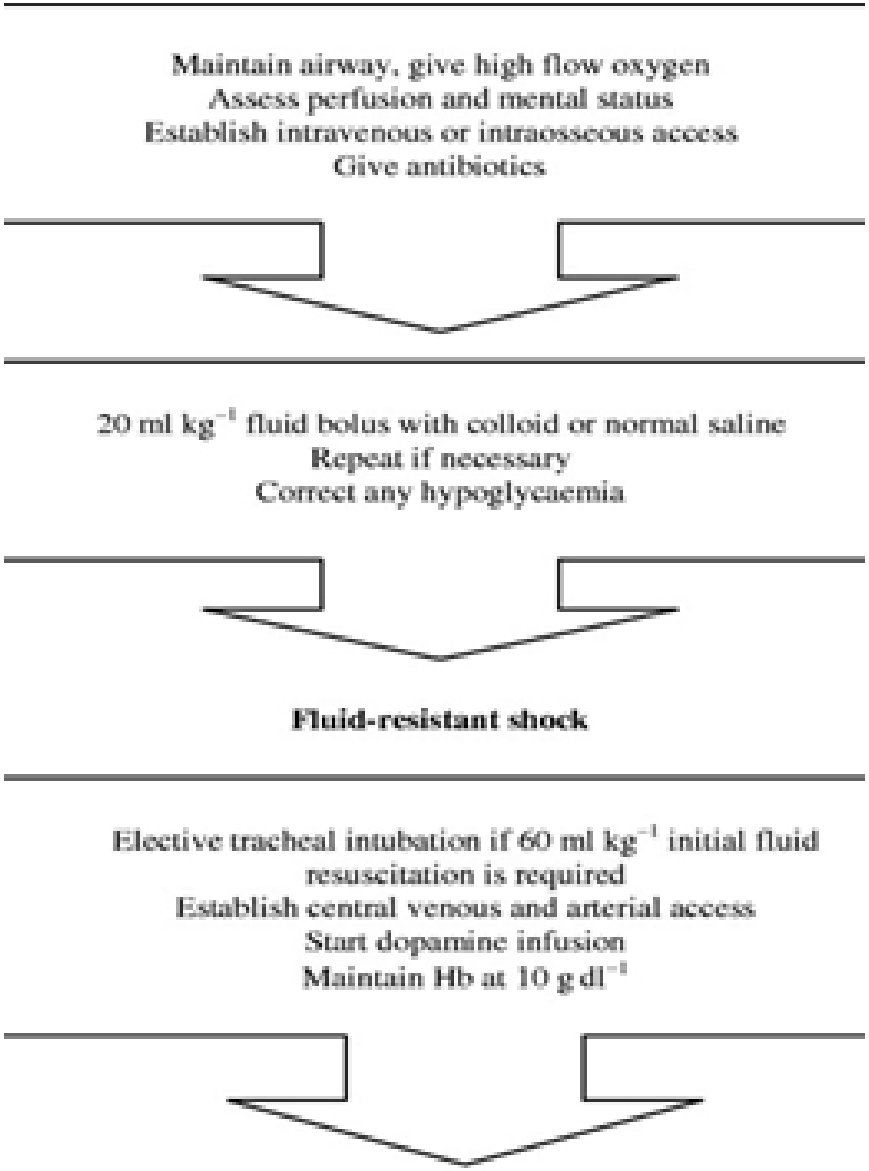
Fluid bolus: 60 mls/kg of 10% Dextrose + 0.9% Saline

Capillary blood gas: pH: 6.9, pCO_2:3.5 kPa, pO_2: 4.0 kPa, B.E: -20, HCO_3^-: 7.0, Lactate: 18, BS: 4.6

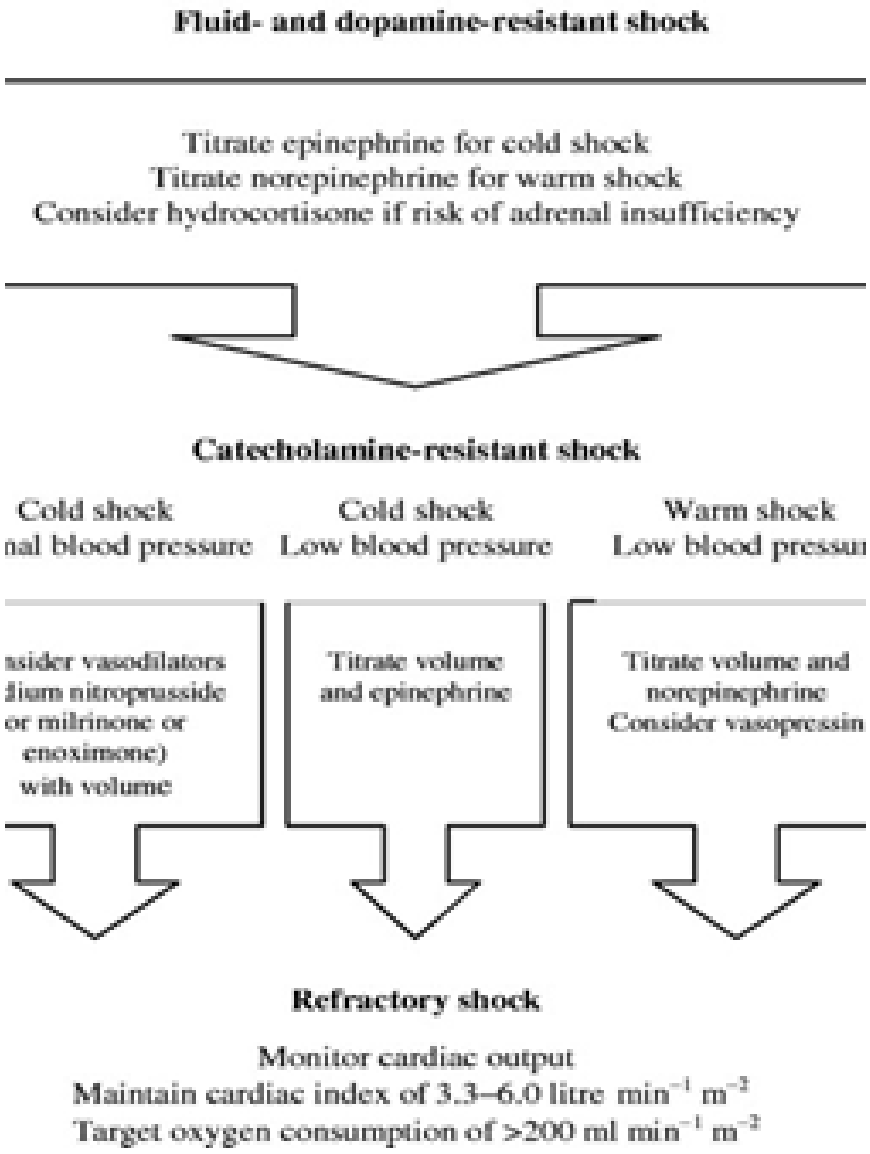
Sepsis Case

- Diagnosis?
- Immediate management?
- KIDS team transport arranged…further management for transfer?

Sepsis Case


Maintain airway, give high flow oxygen
Assess perfusion and mental status
Establish intravenous or intraosseous access
Give antibiotics
20 ml kg^{-1} fluid bolus with colloid or normal saline
Repeat if necessary
Correct any hypoglycaemia
Fluid-resistant shock
Elective tracheal intubation if 60 ml kg^{-1} initial fluid resuscitation is required
Establish central venous and arterial access
Start dopamine infusion
Maintain Hb at 10 g dl^{-1}

Sepsis Case


Fluid- and dopamine-resistant shock
Titrate epinephrine for cold shock
Titrate norepinephrine for warm shock
Consider hydrocortisone if risk of adrenal insufficiency
Catecholamine-resistant shock
Cold shock
nal blood pressure
Cold shock
Low blood pressure
Warm shock
Low blood pressu
nsider vasodilators
lium nitroprusside
or milrinone or
enoximone)
with volume
Titrate volume
and epinephrine
Titrate volume and
norepinephrine
Consider vasopressin
Refractory shock
Monitor cardiac output
Maintain cardiac index of 3.3–6.0 litre min^{-1} m^{-2}
Target oxygen consumption of >200 ml min^{-1} m^{-2}

Sepsis Case

- Preparation for transfer:
  - Intubate + ventilate any unstable child;
  - Induction agents;
  - Cuffed ETT;
  - Lines: IV access;
    - (KIDS: Arterial + CVC)
  - Inotropes: Dopamine, Noradrenaline, Adrenaline, Milrinone, Vasopressin;
  - Steroids;
  - Investigations: Radiology images, lab results, microbiology results;
  - Communication/Handover, Photocopy notes, drug Kardex/Discussions

DKA

- 4 yo – unwell, vomiting, abdominal pain, lethargic
- History of polyuria, weight loss
- In A&E – sats 99%, tachypnoeic, sighing, tachycardia, hyperglycaemia (18), afebrile
- VBG – acidaemia pH7.2, bicarb 16, urine ketones ++, K+ 3

- Differential diagnosis
- Immediate management

Differential diagnosis

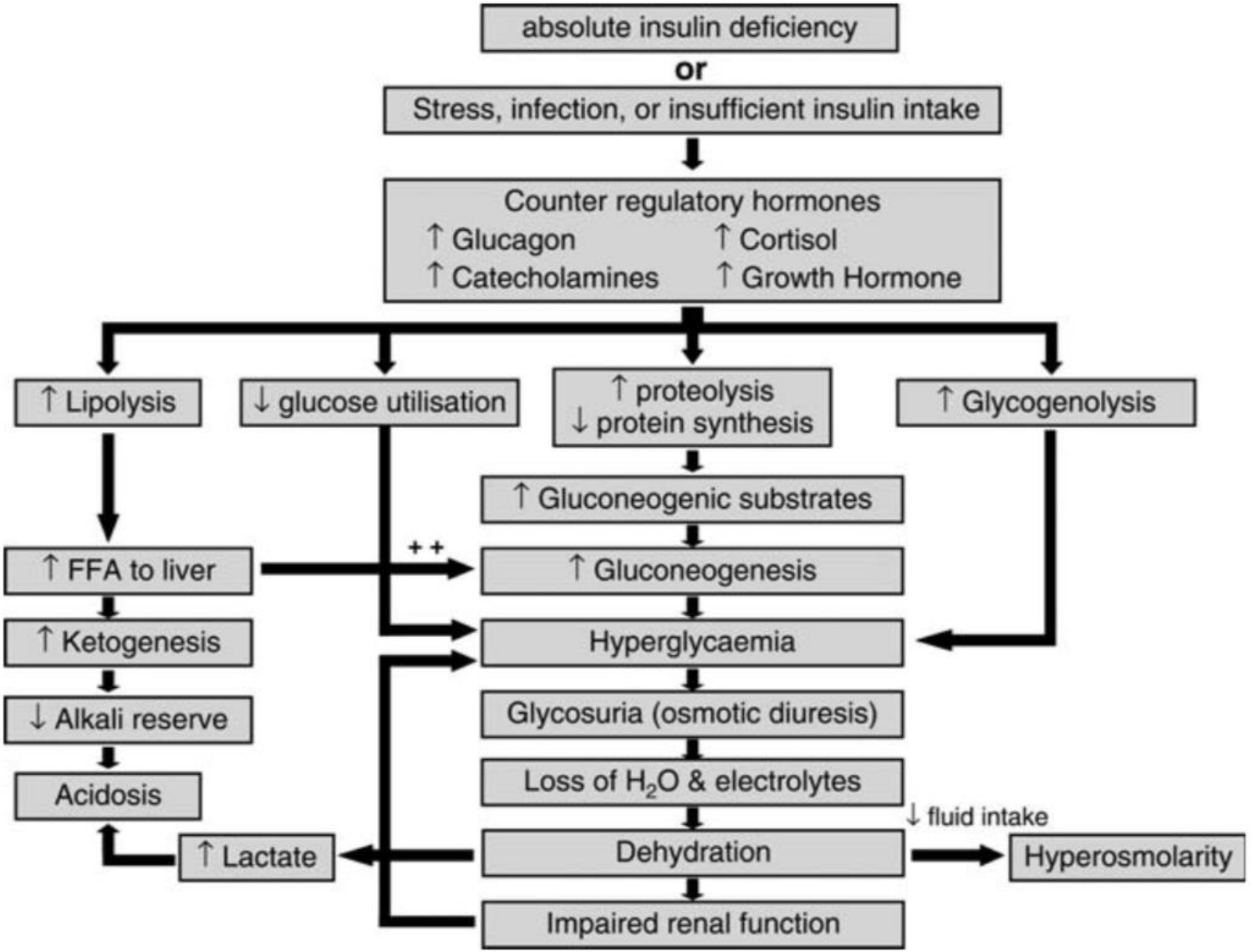
- Acute/surgical abdomen
- Sepsis – meningitis, urinary, chest, other…
- Toxins – salicylate or ethanol poisoning, other?
- Metabolic disorder

Diabetic ketoacidosis

- Absolute or relative insulin deficiency

- Absolute = type 1 DM or failure of therapy
- Relative = endogenous insulin < catabolic hormones (glucagon, catecholamines, growth hormone, cortisol)
  - = gluconeogenesis, glycolysis, lipolysis
  - = hyperglycaemia, hyperketonaemia, hyperosmolality
  - = polydipsia, polyuria, loss of electrolytes
- Metabolic picture: ketoacidosis
- DKA at presentation of T1DM more common in children <5yo
- DKA risk in T1DM is 1-10% per year
- UK mortality rate 0.31% - mostly due to cerebral oedema

Pathophysiology



Presentation

- More common in younger than older children
- 30-40% paediatric cases present with DKA
- Symptoms & signs
  - Weight loss
  - Polydipsia/polyuria
  - Dehydration
  - Lethargy/confusion
  - Abdominal pain +- vomiting
  - Kussmaul's respiration
  - Ketotic (fruity, pear drops) smelling breath
  - Fever (precipitant for DKA)
  - Cerebral odema
  - Shock
  - Coma

Diagnostic criteria DKA

- Hyperglycaemia: BSL >11mmol/L
- Acidaemia: Venous pH <7.3 or bicarbonate <15mmol/L
- Ketonaemia: urine ketones ++ or plasma

- Severity
  - Mild: pH 7.2-7.3, bicarb 10-15
  - Moderate: pH 7.1-7.2, bicarb 5-10
  - Severe: pH,7.1, bicarb <5

Complications

- DKA can be fatal due to:
  - Cerebral oedema
    - More common in younger children
    - Mortality 25%
  - Hypokalaemia
    - H+/K+ exchange, diuresis, loss of K+
  - Aspiration pneumonia
    - Intubate drowsy or unconscious and site NG tube
- Other complications
  - Hypoglycaemia, hypokalaemia, venous thromboembolism, missed intercurrent illness

Management

- Immediate capillary glucose, ketones, pH, bicarb
- 2 IV access
- Weight, ECG, FBC, U&E, cultures, conscious level
- ABCDE
- Replace fluid*, insulin, electrolytes
- Hypotension: 10ml/kg 0.9% saline bolus

Replace fluids

- Risk of cerebral oedema with liberal fluids
- Degree of dehydration difficult to assess
- Only give bolus (10ml/kg 0.9% saline) if hypotensive
- Assume *defecits*:
  - Mild-moderate DKA (pH>7.1): 5% deficit
  - Severe DKA (pH<7.1): 10% deficit
  - Replace the deficit volume over 48 hours
- *Maintenance*:
  - <10kg: 2ml/kg/hr
  - 10-40kg: 1ml/kg/hr
  - >40kg: fixed volume of 40ml/hr

Which fluid?

- 0.9% saline with 20mmol K+ in 500ml until BSL <14mmol/L
- Neonates will need larger volumes (ml/kg) – involve PICU early
- If child's condition improves and they commence oral intake, ensure IV rate adjusted down
- BSL <14, add glucose to the fluid

Replace K+

- In IV fluid
- Hypokalaemia may be masked
  - H+/K+ shift intracellular K+ to extracellular
  - Total body hypokalaemia masked until insulin treatment commenced (driving K+ back into cells)
  - Assume hypokalaemia and replace K+ in IVF unless renal failure

Replace insulin

- Essential – to cease lipolysis and ketogenesis and reduce glucose
- But: early insulin replacement increases risk of cerebral oedema
  - Commence (cautious) fluid and K+ replacement first
- 0.05-0.1 units/kg/hr infusion – no boluses
- When pH>7.3, ketones <3, BSL <14, reduce insulin infusion rate to 0.05units/kg/hr
- When ketones <1, child alert and tolerating oral intake, recommence SC insulin and cease insulin infusion 30 mins later

Monitoring child with DKA

- Hourly obs: RR, HR, BP
- Hourly capillary blood glucose
- Hourly fluid balance – input and output
- ECG until electrolytes normal
- 2 hourly blood or urine ketones
- 2-4 hourly VBG, osmolality, FBC, U&E
- Neurological obs – hourly, or in severe DKA and children under 2 years, half-hourly
- Twice daily weight
- Paeds HDU or PICU

Cerebral oedema

- More common in younger and new onset DM
- Mortality rate 25%
- Symptoms
  - Headache
  - Confusion
  - Vomiting
  - Hypertension and bradycardia
  - Hypoxia
  - Late: papilloedema, unequal pupils, cranial nerve palsies

Treatment of cerebral oedema

- Exclude hypoglycaemia
- Hypertonic (3%) saline 3-5ml/kg over 10-15mins
- Reduce IVF replacement (1/2 rate)
- IPPV when indicated and PICU
- CT brain to exclude cerebral thrombosis, heaemorrhage, infarction

New onset seizures

- 2 year old, previously well, URTI last 2 days, seen by GP – viral
- Seizure at home – PR diazepam by paramedic
- In A&E – fitting, normal glucose
- IV lorazepam, paraldehyde, phenytoin

- Differential diagnosis
- Immediate management

Differential diagnosis

- Febrile convulsion
- Epilepsy
- Traumatic brain injury or non-accidental injury
- Poisoning
- Pseudo-epilepsy

Status epilepticus

- Convulsive or non convulsive
- Convulsive
  - Seizure lasting >30mins or (“continuous”)
  - Recurrent seizures over 30mins without recovery to normal neurological status (“intermittent”)
- Non-convulsive
  - Diagnosed by EEG
  - Altered mental state or conscious level >30mins

SE

- Aetiology varies with age
  - Highest incidence <1yo
    - Immature brain
    - Low seizure threshold
    - Triggers include acute febrile illness
  - <15% patients with CSE have history of epilepsy
- Common triggers
  - Fever
  - Cerebral hypoxia
  - CNS infections
  - Metabolic: hypo-glycaemia, natraemia, calcaemia
  - Hepatic encephalopathy
  - Withdrawal of anticonvulsant in known epileptic

Outcomes

- Dependent on aetiology, age and duration of SE
- Mortality ~<5% - higher when due to CNS causes (encephalitis, TBI etc) than febrile SE
- Febrile SE usually isolated event, recurrence rate 3-4%
- Long term complications
  - secondary epilepsy
  - behavioural problems
  - neurological deficits

Management

- Terminate seizure
  - 1st line drug treatment
  - Longer acting anticonvulsant therapy
- Prevent injury
- Avoid secondary complications
  - Cerebral hypoxia
  - Hypercarbia
  - Hypoglycaemia
  - Progressive lactic and respiratory acidosis
  - Neuronal damage
  - Sympathetic discharge – CNS, CVS and metabolic complications

Complications

Cerebral

Hypoxic/metabolic cerebral damage
Excitotoxic cerebral damage (seizure related)
Cerebral oedema and raised intracranial pressure
Cerebral venous thrombosis
Cerebral haemorrhage and infarction

Cardiorespiratory and autonomic

Hypo- or hypertension
Cardiac failure
Tachy or bradyarrhythmias
Respiratory failure
Pulmonary oedema, hypertension, aspiration, pneumonia
Hyperpyrexia
Hypersecretion, tracheobronchial obstruction

Metabolic and systemic

Dehydration
Electrolyte disturbances (especially hypoglycaemia, hyponatraemia and hypokalaemia)
Acute renal failure (acute tubular necrosis)
Acute hepatic failure
Acute pancreatitis
Disseminated intravascular coagulation
Multiorgan failure
Rhabdomyolysis
Fractures or joint dislocations

Investigations

- Blood
  - Glucose
  - ABG
  - U&E, FBC, LFT, Ca, Mg, PO4, coags, anticonvulsant drug levels
  - Cultures and toxicology as indicated
- Radiology
  - CXR
  - CT/MRI brain
- Other
  - EEG
  - LP – if indicated and when raised intracranial pressure excluded

Drug treatment

Emergency drug treatment

Hypoglycaemia is treated with 2ml/kg 10% dextrose

1st stage (0-10 minutes)

Lorazepam 0.1mg/kg IV given over 30-60 seconds
Diazepam 0.5mg/kg PR or midazolam 0.5mg/kg buccal if no IV access

2nd stage (10-20 minutes)

Lorazepam 0.1mg/kg IV repeated
Paraldehyde 0.4mg/kg PR if still no IV access

3rd stage (>15 minutes)

Call for senior help
Phenytoin 18mg/kg IV or IO over 20 minutes or
Fosphenytoin 15-20mg/kg IV or IO over 20 minutes or
Phenobarbitone 20mg/kg IV over 10 minutes if already on phenytoin
Give paraldehyde 0.4mg/kg PR if not already given

Ongoing management

- Ongoing SE: RSI with thiopentone or propofol
  - Thiopentone – 4-8mg/kg bolus then infusion up to 6mg/kg/min or midazolam 30mcg/kg/min
- Sepsis, encephalitis or uncertain: broad spectrum antibiotics
  - Cefotaxime, aciclovir, erythromcyin
- Monitor in PICU with EEG and aim for burst suppression