RWANDA
MILITARY HOSPITAL

APPROACH TO DKA

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OUTLINE

Part 1

- Case

Part 2

- introduction to T1 DM,
- Epidemiology
- DKA & Physiopathology

Part 3

- Clinical presentation of DKA
- Management
- Monitoring
- complications of management
- recommendations

Part I: Case

- 15 months old F, presents to a random clinic with oral thrush. As she is unable to feed well, she is admitted for intravenous fluids. Elsewhere NAD
- Hx of polydipsia noted one week ago
- DOA 2, glycemia of 300mg/dl not taken seriously
- DOA 4 found drowsy GCS:13/15, dehydrated with RD plus fruity smell…

Case cont'd

- Glycaemia was in 400mg/dl
- PH:6,89 HCO3: 2,8
- Urine ketone +++

Part II: Introduction to T1DM

- Type 1 DM is due to lack of insulin: autoimmune destruction of Beta cells in the pancreas
- Insulin deficiency causes hyperglycemia hence ketone production from lipid catabolism
- Mgt is insulin
- Genetic susceptibility

EPIDEMIOLOGY

- Worldwide variation, highest in Finland(37-65/100,000), lowest in china, Venezuela (0.1-1,9/100,000)
- 45% present <10 years
- incidence of childhood T1DM is reported to be rising worldwide, increases of 2-5%per yr in Europe, the Middle East, and Australia.
- Incidence studies in lower-income countries in Africa and other regions may underestimate true incidence as new cases are frequently missed and die undiagnosed.
- Rwanda, the number of newly diagnosed children per year(1000s):0.05; number of children with T1DM (1000s): 0.3

- Chris peterson et al. diabetes in the young-a global view and worldwide estimatesof numbers ofchildren with type 1 biabetes. Diabetes research and clinical practice10392014) 161-175

DKA

Definition

- Hyperglycemia: plasma glucose >200 mg/dL
- Metabolic acidosis: PH <7.30 **or** bicarbonate <15 mmol/L
- ketosis (ketonuria or ketonemia)

	DKA severity		
	Mild	Moderate	Severe
pH_{venous}	7.3-7.2	7.1-7.20	<7.10
Serum HCO_3 mmol/L	10-15	5-10	<5

Wolfsdorf J. Diabetes Care 2006; 29:1150

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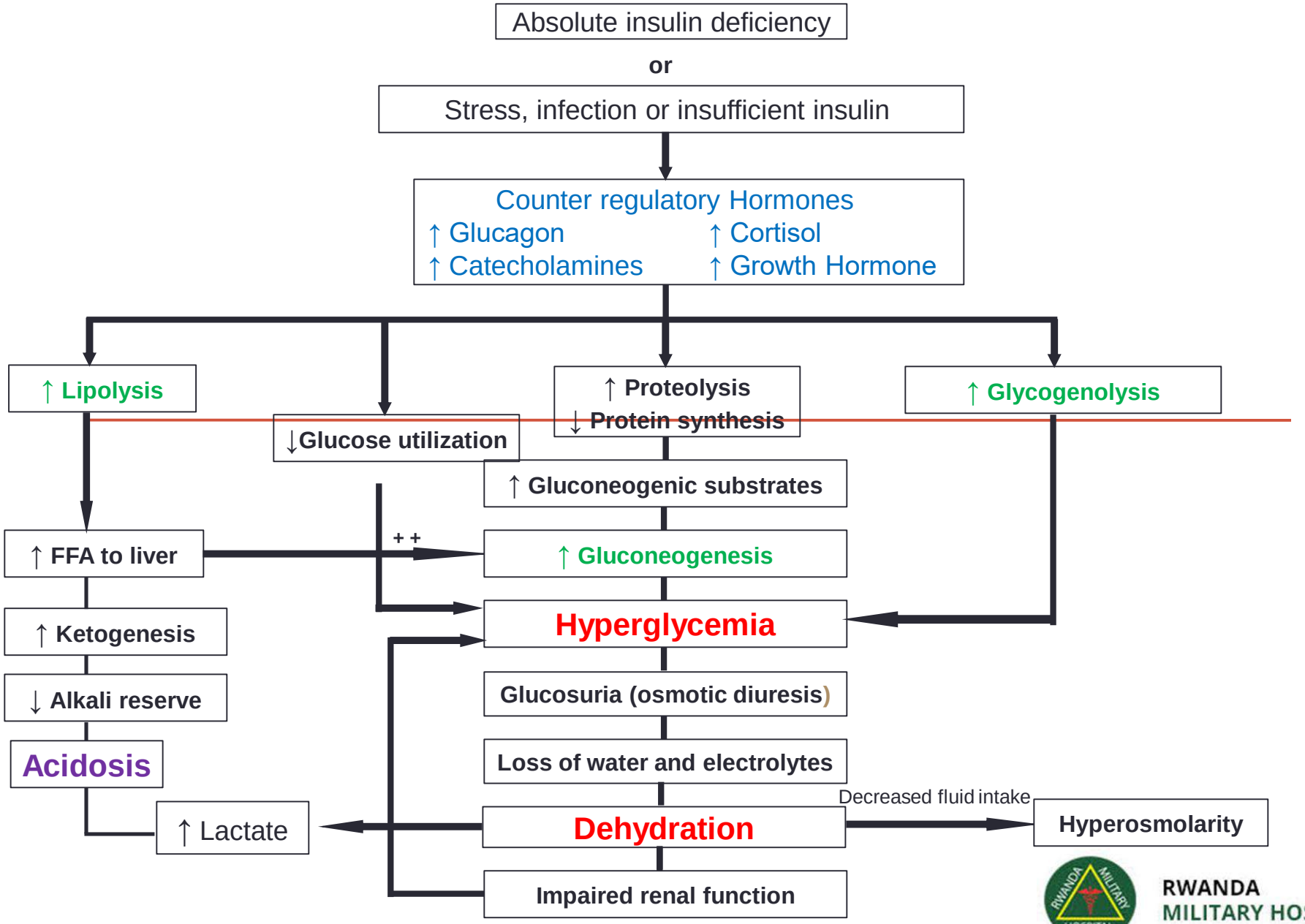
DKA

- Leading cause of mortality and morbidity of T1DM
- Occurs at 1st Dg of T1DM in about 1/3 in the US
- 15-85% newly diagnosed infants and children present with DKA depending on regional incidence
- In established T1DM, rate of DKA is 6-8%/yr

PATHOPHYSIOLOGY OF DIABETIC KETOACIDOSIS



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Part III: Clinical presentation

- Polyuria; polydipsia less obvious in infants
- fatigue;
- signs of dehydration (underestimated due to increased serum osmolality, then fluid shifts from intracellular to extracellular
- Nausea, vomiting, abdominal pain (can mimic acute abd?)
- Rapid, deep (Kussmaul) respiration
- Decreasing LOC related to severity acidosis
- weight loss, nocturia,...
- Occasionally diaper candidiasis

Management

- **Initial evaluation:**
  - Vital signs
  - Signs of shock
  - Level of dehydration, calculate fluid deficit (5-10%)
  - weight
  - Level of consciousness

- **Lab investigations**:
  - blood glucose.
  - Venous pH and serum HCO3
  - Ketosis (ketonemia, ketonuria)
  - Electrolytes (Na, K, Cl)
  - Urea, creatinine.
  - pCO2
  - FBC
  - Ca, phos, Mg
  - Calculate anion gap
  - Severity of DKA

Management goals

- Treatment of dehydration
- Insulin
  - Correct acidosis and reverse ketosis
  - Restore glucose to near normal
- Treatment of precipitating cause
- Close monitoring
- Avoid complications of therapy
  - Cerebral edema
  - Hypokalemia

Fluid & Electrolyte Therapy

- During **1st HOUR**: Resuscitate in case necessary to restore perfusion if impaired
- 10-20 mL/kg of NaCl 0.9% over 1-2 hours
- Dehydration should be corrected slowly to avoid cerebral edema
- Deficit to be corrected over 48h to be added on daily requirement : usually rate is 1.5-2x daily maintenance
- Provide maintenance fluid and electrolytes

Fluid & Electrolyte Therapy

- For at least 4-6 hours: 0.9% NaCl + K^+
- Then later ½ NaCl+ K^+
- Add 5% dextrose when glycaemia 250 mg/dL
- Adjust dextrose concentration (5%-12.5%) according to glycaemia to avoid hypoglycemia if BG< 150mg/dl before ketoacidosis resolves
- Continue IV fluid therapy until acidosis has been corrected (pH: ≥7.30 or HCO3: ≥ 15; anion gap near-normal) and patient is able to eat and drink

Insulin treatment

- Start **continuous IV insulin infusion**. Dose is 0.05-0.1 unit/kg/hour of Regular insulin.
- Start with the lowest effective insulin dose
- If no syringe pump available, give 0.05-0.1 unit/kg IV every hour or 0.1 unit/kg SC 2 hourly
- Check glycaemia hourly
- Once glucose is below 250 mg/dl (14 mmol/L), change IV fluid to D5%1/2NS with 20meq of KCl/L

Potassium treatment

- if **hyperkalemia** or impaired real function: none until levels fall to normal and adequate kidney function.
- **normokalemic and voiding**, initiate K replacement with the start of insulin therapy at the concentration of 40mEq/L (40 mmol/L).
- If **hypokalemic**, K replacement to be started immediately. Insuline should be delayed until serum K has been restored to a near normal concentration.
- Potassium replacement therapy should continue as long as IV insulin and fluid therapy is still running.

Complete Clinical Evaluation

Evaluate peripheral perfusion

- **Shock** → 0.9% NaCl 20cc/kg Repeat PRN
- **Decreased, no shock** → 0.9% NaCl 10cc/kg over 1-2 hours
- **normal**

Slow rehydration in 48h. Begin with 0.9% NaCl:
4-9 kg: 6 cc/kg/hr
10-19 kg: 5 cc/kg/hr
20-39 kg: 4 cc/kg/hr
40-59 kg: 3.5 cc/kg/hr
60-80 kg: 3 cc/kg/hr

No IV fluids available
- Urgent transfer to other hospital
- Rehydrate PO with ORS 5cc/kg/hr in small sips or via NG tube
- Give ½ of fluids as fruit juice or coconut juice if ORS is not available

Hyperlycemia

Glycosuria
Ketonuria

Start insulin IV or SQ 1-2 hrs AFTER hydration therapy
Child 5y or older → 0.1U/kg/h
Child <5 y → 0.05U/kg/h

Blood glucose Q1H
Goal ↓BG by 100 mg/dl/hr

- BG >100mg/dl → ↓ insulin by 20%
- BG <50mg/dl → ↑insulin by 20%

BG<250 mg/dl

Change IV fluid to D5% (+NaCl)
at same rate (see above)
+
Continue insulin as above
Blood sugar goal: 150-200 mg/dl

Resolution of DKA + NPO
Insulin R 0.1 U/kg Q4H IV for every 50 mg/dl that BG is >150 mg/dl; maximum dose 0.3 units/kg

Resolution of DKA + PO
Insulin SQ: Combination of multi-injection per day
<3y = 0.3U/kg/day
3-12y = 0.5-0.75U/kg/day
12-16y = 0.75-1.0U/kg/day
>16y = 0.75U/kg/day

Potassium

- **K<3.3 mEq/L** → give 40 mEq/L KCl per hr until K >3.3 mEq/L
- **UOP >2cc/kg/hr + 10cc/kg bolus complete** → 20-30 mEq/L until taking PO
- **K >5.5 mEq/L** → no KCl; repeat K in 2 hrs

No serum K available
- No UOP → do NOT give K
- UOP present: give 20mEq/L x4h, then increase to 40 mEq/L/hr

Monitoring

Parameter	Trequency
Vital signs	Hourly
Fluid balance (In/Out)	Hourly
Level of consciousness (neuro status)	Hourly
Blood glucose	Hourly
U&Es, venous blood gaz	2-4h
ketosis	2-4h

Discontinuing the insulin infusion

The insulin infusion should continue until all of the following conditions are met:

- Venous pH >7.30, **or** serum bicarbonate (HCO_3) >15 mEq/L
- Blood glucose <200 mg/dL (11.1 mmol/L)
- Serum anion gap reduced to normal (12 ± 2 mEq/L) **or** serum BOHB ≤1 mmol/L (10.4 mg/dL)
- Patient is tolerating oral intake

Discontinuing the insulin infusion

- There might be mild hyperchloremic acidosis and/or ketonuria for some time after the above conditions are met.
- So Hyperchloremic acidosis with a normal anion gap is not a contraindication for switching the patient to subcutaneous insulin.
- SC insulin to be initiated at least 30 min before stopping continuous insulin

Complication of mgt

- **Cerebral edema**
- insufficient rehydration
- Hypoglycemia
- Hypokalemia
- Hyperchloremic acidosis

Cerebral edema

- Risk factors:

Severe acidosis at presentation.

Substantially elevated BUN at presentation.

Severe hypocapnia.

Young child (<5 years) and/or new onset of diabetes

Muir AB. Cerebral edema in childhood diabetic ketoacidosis. Diabetes Care 2004; 27:1541.

Cerebral edema

- Diagnosis

Minor criteria (moderately suspicious findings)

Headache – Although headache is frequently present at diagnosis, worsening or recurrence of headache during treatment is suspicious for cerebral injury.

Vomiting – Vomiting is suspicious if it develops or recurs during treatment.

Irritability, lethargy, or not easily aroused from sleep – These features are suspicious particularly if they occur or worsen after initiation of therapy.

Elevated blood pressure (eg, diastolic BP >90 mmHg).

Major criteria (very suspicious findings)

Abnormal or deteriorating mental status after initiation of therapy, agitated behavior, or fluctuating level of consciousness.

Incontinence inappropriate for age.

Inappropriate slowing of heart rate – eg, decline more than 20 beats per minute that is not attributable to improved intravascular volume or sleep state.

Diagnostic criteria (signs of significant brain injury, increased intracranial pressure, or brain herniation)

Muir AB. Cerebral edema in childhood diabetic ketoacidosis. Diabetes Care 2004; 27:1541.

Cerebral edema: mgt

Indications:

•Child with DKA and One diagnostic criterion, or
•Two major criteria, or
•One major and two minor criteria, or
•One major and one minor criterion (if child under 5 years of age)

The decision to treat should be based on signs and symptoms. **Do not rely on neuroimaging to make or exclude the diagnosis.**

Interventions:

- Give mannitol, 0.5 to 1 g/kg intravenously over 15 minutes. The mannitol dose may be repeated in 30 minutes, if there is no initial response.[Δ]
- Adjust fluid administration as indicated to maintain normal blood pressure and optimize cerebral perfusion.
- Avoid hypotension that might compromise cerebral perfusion pressure.
- Neurosurgery consultation regarding further management, including possible invasive monitoring of intracranial pressure in selected cases.

Muir AB. Cerebral edema in childhood diabetic ketoacidosis. Diabetes Care 2004; 27:1541.

Recommendations

- Full assessment of patient (Hx, P/E..) and… to repeat if something is not correlating with current working Dg
- Remember to take glycaemia and interpret patient investigation results !!!
- Use of protocol in mgt of DKA

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- Thank you
- Comments???