MEDICAL VIDEO REVIEW

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CASE

15 year old male w/ T1DM in DKA, coming from outside facility

Questionable compliance with insulin, has a pump

Initial glucose >700, pH 6.95 BD -29 HCO_3 4

20 mL/kg bolus NS and insulin at 0.1 Units/kg/h = 4 Units/h + mIVF

Referring physician wanted to intubate due to tachypnea and give bicarb

CASE

Rapid Cardiopulmonary Assessment

Alert/Drowsy, airway patent & maintained, lungs CTAB, tachypneic, palpable pulses, pale skin, GCS 15

Secondary Survey

PERRL 4→2, Mild abdominal pain

Vitals

T 36.7ºC BP 104/61 (87) HR 144 RR 27 SpO2 100%

CASE

This is a sick diabetic

What are your priorities for care?

What are some of the challenges when accepting care from a patient in DKA transferred from another facility?

How does insulin really work?

How did Gary get so smart?

How should we fluid resuscitate patients in DKA?

What is DKA?

How is DKA different from HHNS?

Do sleepy kids in DKA need a head CT?

Does testing for ketones matter?

Is bicarb a bad idea?

What are the risk factors for cerebral edema?

Does Mary do every Med Video Review?

Fluids first?

Cerebral edema

That’s not our insulin

DKA

2014 International Society for Pediatric and Adolescent Diabetes definition

Hyperglycemia

blood glucose of >200 mg/dL

Metabolic acidosis

venous pH <7.3 or plasma bicarbonate <15 mEq/L

Ketosis

ketones in the blood or urine

DKA

2014 International Society for Pediatric and Adolescent Diabetes definition

Metabolic acidosis severity by pH

Mild 7.2 - 7.3

Moderate 7.1 - 7.2

Severe <7.1

DKA

2014 International Society for Pediatric and Adolescent Diabetes definition

Ketosis

qualitative (Yes/No) measurement of ketones (acetoacetate) underestimates ketosis

serum ß-hydroxybutyrate is more accurate

ß-hydroxybutyrate ≥3 mmol/L (31 mg/dL) is consistent with DKA

DKA

2014 International Society for Pediatric and Adolescent Diabetes definition

PICU criteria at CCHMC

No strict value, but generally <7 = PICU at CCHMC

Kids that are younger and have AMS with pH <7.2

Any kid under 3 years on HD #1 is a good PICU candidate regardless of initial I-STAT pH

DKA is a state of hyperosmolar dehydration

Fluids first?

How do we know whether or not a patient is dehydrated?

Mackenzie et al. Lancet, 1989

In gastroenteritis the 4 best signs of dehydration were:

- capillary refill >2 s
- absent tears
- dry mucous membranes
- overall appearance of illness

Murphy et al. Arch Dis Child, 1998

In gastroenteritis the 4 best signs of dehydration were:

- decreased skin turgor
- dry oral mucosa
- sunken eyes
- altered neurological status

The aforementioned signs and symptoms may not accurately assess hydration status in DKA

Koves et al. Diabetes Care, 2002

Random convenience sample of 37 patients with DKA

Two examiners with satisfactory independent agreement assessed degree of dehydration clinically

Actual dehydration measured by weight changes throughout hospital stay

Results

- 70% had incorrect assessment of hydration
- Overestimated dehydration in patients <6% dehydrated
- Underestimated dehydration in patients >6% dehydrated

Conventional signs assess depletion of extracellular fluid volume

DKA has extra and intravascular dehydration

Metabolic acidosis causes:

- decrease in peripheral vascular resistance
- decrease in cardiac ventricular function
- compensatory hyperventilation

Prolonged insulin deficiency/catabolic state

- lipolysis and proteolysis and subsequent weight loss

In DKA patients lose **both** intracellular and extracellular water - IV fluids mainly effect the latter

DKA patients are **usually** 5-10% dry

Goal of rehydration in DKA

Restore effective circulating volume and restore GFR to aid in glucose and ketoacid clearance while minimizing the risk of cerebral edema

In moderate to severe DKA start with 10 mL/kg NS or LR over 30-60 minutes

If ECV is still abnormal repeat with additional 10 mL/kg

After ECV is replenished start maintenance fluids at 3000 mL/m^2 divided over 24h

By the book initial goal is 50% of the total fluid deficit in the first 12 hours

Unless severe hypernatremia (calculated Na >155) or HHNS 3000 mL/m^2 will work just fine

Be more careful with young children and new onset DKA

Poorly controlled older children are anecdotally more fluid resilient per some Endocrine attendings

Common pitfalls for fluid administration in DKA

Just giving 1L NS to everyone

Not calculating a BSA (or calculating it accurately)

Not calculating the true fluid deficit in a kid (weight)

Not going slower if extreme hypernatremia (>155)

If a patient in DKA is hypotensive resuscitate with isotonic fluids until they are not

Is it possible to be too aggressive with fluids in DKA?

Cerebral edema

Clinically significant cerebral edema occurs in 0.5 - 1% of patients with DKA

Mortality rate is at least 20%

50 - 80% of DKA deaths are from cerebral edema

Pathophysiology

Insulin and ECV volume depletion lead to osmotic shift into brain cells

Inflammation from cerebral hypoxia/ischemia

Vasogenic edema

Defective vasoregulation despite hypocapnia - CBF actually increases

increased cerebral blood flow

disruption of cell membrane ion transport

Clinical presentation

Usually occurs 4-12 hours after treatment initiation

80% develop during treatment

Headache is usually first symptom

Early signs often preclude changes on CT

- Altered level of consciousness
- Sustained heart rate deceleration
- Age-inappropriate incontinence

Risk factors associated in the literature

Younger children

New onset DKA

Failure of serum sodium to rise enough during therapy

Higher BUN at presentation

More severe acidosis

Use of bicarbonate to correct acidosis

Lower initial CO_2

Risk factors associated in the literature

Rate of fluid administration

Rapid correction of dehydration with hypotonic fluids

Rate of insulin administration

Edge et al. Diabetologia, 2006

Prospective case control study of cerebral edema in DKA

- Acidosis
  pH <7.05 OR=0.22
- Baseline electrolyte abnormalities
- Insulin in the first hour
  OR=4.7 (95% CI 1.5–13.9, p<0.007)
- High volumes of fluid (>4L in first 3-4h)

Caveat emptor Most studies on cerebral edema risk factors are limited by lack of sample size and no matched controls

Especially true for fluid and insulin administration rates

Glaser et al. NEJM, 2001

Risk factors for cerebral edema in a retrospective case control study

Compared with matched and random controls

Multivariate analysis

Glaser et al. NEJM, 2001

Cerebral edema more likely in matched control multivariate analsysis if:

- Higher BUN
  Every incr of 9 mg/dL RR=1.8 (95% CI 1.2-2.7), p=0.008
- More severe hypocapnia
  Every decr 7.8 m Hg RR=2.7 (95% CI 1.4-5.1) p=0.002
- Smaller increases of Na during therapy
  Incr of 5.8 mmol/L/hr RR=0.6 (95% CI 0.4-0.9) p=0.01
- Treatment with bicarb
  RR=4.2 (95% CI 1.5-12.1) p=0.008

Glaser et al. NEJM, 2001

Factors not associated with cerebral edema

- Initial serum glucose concentration
- Rate of change in the serum glucose concentration during therapy
- Rates of fluid, sodium, and insulin administration

Cerebral edema develops less frequently prior to therapy

Rate and volume of fluid administration may be less associated with cerebral edema than initially hypothesized

Ischemia may be the more important causative factor

Hypocapnia causes cerebral vasoconstriction

Extreme dehydration decreases perfusion

Hyperglycemia superimposed on an ischemic insult increases:

- Extent of neurologic damage
- Blood–brain barrier dysfunction
- Edema

Pediatric brains need more oxygen and thus are more sensitive to ischemia

While monitoring a patient in the ED how can we assess for the development of cerebral edema?

Major criteria

- Altered mentation/fluctuating level of consciousness
- Sustained heart rate deceleration (≥ 20 beats per minute) not attributable to improved intravascular volume or sleep state
- Age-inappropriate incontinence

Minor criteria

- Vomiting
- Headache
- Lethargy or being not easily aroused from sleep
- Diastolic blood pressure >90 mmHg
- Age <5 years

Diagnostic criteria

- Abnormal motor or verbal response to pain
- Decorticate or decerebrate posture
- Cranial nerve palsy (especially III, IV, and VI)
- Abnormal neurogenic respiratory pattern (eg, grunting, tachypnea, Cheyne-Stokes, apneusis)

Muir et al, Diabetes Care, 2004

After therapy initiated diagnose cerebral edema if:

Any diagnostic criteria

OR

2 major criteria

OR

1 major and **2 minor**

Role of imaging

In small studies (CT and MRI) cerebral ventricle narrowing is seen in DKA

Children with GCS <15 appear to have a slightly higher rate of abnormalities on imaging

Obtain prior to sending patient to PICU if:

- Any concern for cerebral edema clinically
- GCS <15
- Cannot rule out trauma

Treatment

Reduce rate of fluid administration

3% Saline or mannitol

Avoid hyperventilation after intubation

Driving CO_2 too low (especially ≤22) leads to deleterious cerebral vasoconstriction

Outcome

Risk factors for death or persistent vegetative state

Marcin, J Pediatr, 2002

Retrospective review of <18 years with DKA and cerebral edema

Poor outcomes associated with GCS <7, high BUN and intubation with hyperventilation to CO_2 <22 mmHg

Let’s talk about patients being transferred on an insulin drip

That’s not our insulin

An insulin drip is generally started if ketone + and serum HCO_3 <15

Regular insulin drip 0.1 units/kg/hr

No bolus dose required

If hyperglycemic or and HCO_3 >15 may forgo insulin drip and start rapid-acting insulin SQ

When receiving a patient from another facility

Pertinent history

Initial glucose and glucose prior to transfer

Last dose of insulin or concentration/rate of insulin drip (when was it started)

Is the patient hemodynamically stable?

Total and current IVF with composition and rate

Mental status?

When receiving a patient from another facility

Who is transporting the patient?

PICU is medical control for all CCHMC Transport Team patients

MD taking the referral should place orders for insulin drip prior to arrival

Discuss between MD, Charge RN, Triage nurses for placement upon arrival (Triage, STS, PICU)

Make sure Endocrinology is aware of imminent arrival

When receiving a patient from an outside hospital on an insulin drip

STS if:

- Hemodynamically unstable
- AMS
- Insulin needs to be disconnected prior to a new bag being started

ED room if:

- The MD that took the referral has already placed the order for the insulin and it is in the ED
- The transporting unit can stay with the patient until the insulin arrives

Make sure the pump is removed!

There is no current CCHMC guideline or policy regarding how to manage patients arriving to the ED with an insulin drip infusing

Getting the patient ready for admission

Remove the pump!

- The patient or family may need to disconnect since most staff are unfamiliar

Make sure the pump and sensor gets sent to the PICU/floor

- They are expensive
- Data can be downloaded from it

Tell families to expect 3-4 days of extensive education during hospitalization for new onset diabetes

Take home points

Assessing hydration status in DKA is hard - so make sure you get weight and height ASAP

In the young and very acidotic pH <7 as well as new onset DKA be careful with the volume of fluids

Start with 10 mL/kg over 30 minutes

Game-plan before the patient from an outside hospital on insulin arrives

Cerebral edema may be more related to ischemia than volume/composition of fluids

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