ECHO Summary,2/AUG/2024
Session Title: ED Approach to Diabetic Emergencies

Summary Author: Makenna Hittner

Edited by: Jessica Pelletier, DO, MHPE

Disclaimer:
The information presented in this summary is based on the presentation given by the panelists and is intended for general informational purposes only. The authors and collaborating partners do not accept responsibility for any outcomes resulting from the implementation of treatments outlined in this document. It is strongly recommended that individuals verify the information against their national guidelines and seek professional advice before making any decisions related to the content presented herein.

ECHO Session Panelists
Experts:
Dr. Nsubuga Martin
Consultant Physician, Entebbe Regional Referral Hospital

Dr. Kamoga Dickson
EM Resident, MakCHS

Ms. Benda Hannah
In-charge Endocrinology Ward, Kiruddu NRH

Dr. Jjunju Samuel
Physician

Case Presenter:
Dr. Ndegwe Ambrose
MSc Diabetes, Internal Medicine Resident, MAKCHS

Moderator:
Dr. Twinamatsiko Brian

EM Resident, MUST

Definitions

Diabetic Emergencies:

- Diabetic Ketoacidosis (DKA)
    - Blood glucose >11.0 mmol/L or known diabetes mellitus
    - Blood ketone concentration >3.0 mmol/L or 2+ ketonuria
    - Bicarbonate concentration <15.0 mmol/L and/or venous pH <7.3 + anion gap >15
- Hyperosmolar Hyperglycemic State (HHS)
    - Blood glucose usually >50 mmol/L
    - Calculated osmolality (2[Na + K] + glucose) of >290 mOsm/kg
    - No significant ketonuria or acidosis
    - Severe dehydration
- Hypoglycemia
    - Blood glucose <4.0 mmol/L

Epidemiology

- 3.6% of adults in Uganda have diabetes mellitus.[1]
- DKA is more common with type 1 diabetes. HHS is more common with type 2 diabetes.[2]

Risk Factors

- DKA and HHS
    - Newly diagnosed diabetes
    - Poorly controlled diabetes
    - Treatment interruptions
    - Infections
    - Trauma
- Hypoglycemia
    - Overdose of insulin or other anti-diabetic medications
    - Excessive alcohol intake
    - Sepsis and critical illnesses
    - Liver disease
    - Prematurity
    - Starvation
    - Insulinomas
    - Certain drugs (e.g. quinidine)
    - Certain hormone deficiencies (e.g. cortisol, growth hormone)[2]

Clinical features

- **AIRWAY:**
    - Any of these patients may present with a decreased level of consciousness. Secure the airway with intubation if absolutely necessary.
        - Note: If intubating a patient with DKA, hyperventilate (RR 30, flow 60 L/min initially) to help blow off acidosis.

- *Avoid intubation unless absolutely necessary, since these patients are at high risk for decompensation*. The following are tips for safely intubating the DKA patient:
    - Resuscitate the patient to the best of your ability first (if possible).
    - Consider delayed-sequence intubation using ketamine (https://emcrit.org/dsi/) rather than rapid-sequence intubation - this preserves the patient breathing on their own as long as possible, allowing them to blow off carbon dioxide.
    - Avoid succinylcholine if possible, as it may worsen hyperkalemia.
    - Consider administering a bicarbonate bolus, particularly if their bicarbonate level is <10 mmol/L.
    - Along with a high RR, high tidal volume (8 cc/kg) will help with hyperventilation.[3]

- **BREATHING**:
    - Look for Kussmaul's respirations (deep, labored breaths) which are a sign of DKA.
    - Breath with a fruity odor is another sign of DKA.
    - DKA is often triggered by infection. Assess for signs of respiratory infection and monitor O2 saturation; give supplemental oxygen if needed.
- **CIRCULATION**:
    - DKA and HHS are associated with severe dehydration. Check blood pressure and capillary refill to assess the patient's volume status. Give fluids according to protocol below.
    - Get an EKG to monitor for arrhythmias secondary to electrolyte abnormalities caused by DKA and HHS.
- **DISABILITY:**
    - Use the Glasgow Coma Scale to assess level of consciousness.
    - Always check a fingerstick blood glucose in a patient with a decreased level of consciousness.
        - If blood glucose <4.0 mmol/L, treat hypoglycemia immediately (see below).
        - If blood glucose >11.0 mmol/L, consider DKA.
        - If blood glucose > 50 mmol/L, consider HHS.
        - *There can be overlap between DKA/HHS, as noted in the case presentation in the ECHO session.* Patients with DKA/HHS overlap syndrome will have both acidosis AND hyperosmolality.
    - HHS usually presents with stupor/altered level of consciousness but MAY present with focal neurologic deficits or seizures.
        - Treat seizures as you normally would. You can check out a quick summary from the Continuing Medical Education on Stick (CMES) Journal Club, July 2024:

https://docs.google.com/document/d/1Z8_eGq0LqLuq9Q8gfz0EpHLLVa5bLMm-W-oC6iGUWsM/edit?usp=sharing

- Ensure that you exclude alternate causes of seizures such as eclampsia or head trauma.

- Obtain brain imaging with noncontrast CT for patients with new-onset seizures or focal neurologic deficits to exclude alternate causes (such as intracranial hemorrhage or mass lesions).
  - *Imaging findings may be NORMAL in patients with cerebral edema, as changes on brain imaging may lag behind the clinical presentation.*[4]
- Assume the patient has **cerebral edema** if they have one diagnostic criterion, two major criteria, or one major and two minor criteria:
  - "Diagnostic criteria
    - Posturing
    - Abnormal response to pain
    - Cranial nerve palsy
    - Neurologic respiratory pattern
  - Major criteria
    - Altered or fluctuating mental status
    - Sustained decrease in heart rate of 20 beats per minute without an obvious cause such as fluid resuscitation
    - Incontinence in a normally continent patient
  - Minor criteria
    - Vomiting (after treatment, if the patient initially presented with vomiting)
    - Headache
    - Lethargy
    - Diastolic blood pressure >90 mm Hg
    - Age <5 y"[4]

- **EXPOSURE**:
  - Check temperature to assess for fever, as infection is a common trigger for DKA.
  - If the patient seized prior to arrival, check for associated trauma such as tongue lacerations and injuries from falls

- Presentation of DKA:
  - Dehydration, tachycardia, tachypnea, hypotension, decreased level of consciousness, the smell of ketones on breath, abdominal pain, and/or vomiting.
- Presentation of HHS:
  - Severe dehydration, tachycardia, tachypnea, hypotension, altered mental status, and/or stroke-like symptoms (i.e. hemiplegia, visual blurring, hemianopsia), seizures
    - *Remember that it is difficult to differentiate seizures from HHS vs. seizures from cerebral edema* - see diagnostic criteria above
- Presentation of hypoglycemia:

- Sweating, palpitations, shaking, hunger, confusion, drowsiness, speech difficulty, incoordination, headache, nausea

Diagnostics

- Random blood sugar
  - If <4.0 mmol/L = hypoglycemia
  - If >11.0 mmol/L = DKA vs HHS vs severe hyperglycemia
- Urinalysis - look for ketonuria, glucosuria, and evidence of UTI
  - DKA: glucosuria and 2+ ketonuria
  - HHS: glucosuria but no significant ketonuria
- VBG or ABG - check pH for acidosis
  - DKA: high anion gap metabolic acidosis
  - HHS: no significant acidosis
- Serum electrolytes - bicarbonate, potassium (K+), sodium, chloride, anion gap
  - DKA: bicarb low, anion gap high, K+ falsely normal/elevated
  - HHS: osmolality typically >290 mOsm/kg; calculate osmolality with the following equation: (2[Na + K] + glucose)
    - Use *uncorrected* sodium to calculate the osmolality!
  - Remember: markedly elevated glucose affects sodium!
    - Use *uncorrected* sodium to calculate anion gap: (Na - [Cl + HCO3]) = anion gap
    - Use *corrected* sodium to evaluate fluid status: (Na + [Glucose/4]) = corrected Na+
- EKG - look for arrhythmias caused by electrolyte abnormalities as well as ischemia as possible trigger for DKA
- Complete blood count (CBC) - look for signs of infection
- CXR, urine/blood cultures - look for signs of infection
- Renal and liver function tests - check for signs of end organ damage due to potential hypovolemic shock secondary to severe dehydration
- HbA1C - assess for long-term blood glucose control
- Test for TB, HIV, and malaria - rule out as causes of decreased level of consciousness AND check for potential triggers for DKA

Treatment

- <u>DKA</u>
  - Goals of treatment: correct life-threatening electrolyte abnormalities, restore intravascular volume, correct acidosis, identify and treat causes and complications.
  - Use intensive- or high-dependency units if the patient has altered mental status, decreased level of consciousness, systolic BP <90 mmHg, bicarb <10 mmol/L, or pH <7.0. If ITU/HDU unavailable, monitor *closely* on the wards.
  - IV fluids - these patients have an average fluid deficit as high as 6 L.[5]
    - Give 1L normal saline over the first hour.
    - Give 1L normal saline in the next hour.

        - Give 2L normal saline over the next 2-4 hours.
        - Give 1L normal saline 4-6 hourly.
        - Reduce the rate in the elderly, those with cardiac disease, or those with only mild DKA.
        - Once blood glucose <14 mmol/L, switch to 5% glucose 1L 8 hourly and continue normal saline concomitantly if the patient is still volume depleted.
    - IV insulin
        - Fixed rate infusion at 0.1 units/kg
        - The insulin infusion is made up of 50 units of soluble human insulin in 49.5 mL normal saline (i.e. 1 unit/mL).
        - Once blood glucose <14 mmol/L, reduce the dose to 0.05 units/kg and add a dextrose infusion (5-10%).
        - Switch to subcutaneous insulin when anion gap becomes normal, bicarb >18, and pH >7.3. If these labs are unavailable, use clinical judgment to decide.
    - Potassium
        - If serum K+ >5.5, do not give potassium. Recheck in 2 hours.
        - If serum K+ 4-5.5, add 20 mmol potassium chloride to each liter of fluid.
        - If serum K+ <4, add 40 mmol potassium chloride to each liter of fluid.
    - Potential complications of treatment for which to watch out: hypoglycemia, hypokalemia, aspiration, cerebral edema.
    - The *target of insulin therapy in DKA is resolution of acidosis* (NOT resolution of hyperglycemia). Continue insulin until two of the following are found on repeat lab draws:
        - “Serum bicarbonate ≥15 mmol/L
        - pH >7.3
        - Anion gap ≤12 mmol/L”[5]
- <u>HHS</u>
    - Goals of treatment: correct life-threatening electrolyte abnormalities, restore intravascular volume, identify and treat causes and complications
    - Management is the same as for DKA except for the following:
        - Insulin fusion rate should be halved (0.05 units/kg and then 0.0025 units/kg).
        - If serum Na+ >155 mmol/L, consider giving 0.45% sodium chloride initially, although normal saline can still be used.
        - These patients have an increased risk of thromboembolic disease, so anticoagulate fully if there are no contraindications.
        - *These patients may require more crystalloid than patients with DKA, as their total fluid deficit may be as high as 9 L.*[4] Reassess fluid status regularly (you can learn more here: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9994306/).
- <u>Cerebral edema</u> (not covered in the ECHO Session)
    - This is a CLINICAL diagnosis - see diagnostic criteria above.

        - A noncontrast brain CT is wise to exclude alternate causes, but is not necessary to make this diagnosis.
    - If you suspect cerebral edema:
        - "Stop IV fluids.
        - Elevate the head of the bed.
        - Perform osmotic diuresis:
            - Mannitol, 1 g/kg IV over 20-30 min or
            - Hypertonic sodium chloride 3%2.5-5 mL/kg IV over 15 min"[4]
- <u>Hypoglycemia</u>
    - Goals of treatment: increase blood glucose
    - In patients who are conscious, oriented, and able to swallow:
        - Start with ABCDE.
        - If the patient is on insulin infusion, stop the infusion.
        - Give 150-200 mL pure fruit juice or 3-4 heaped teaspoons of sugar dissolved in water.
        - Recheck blood glucose 10-15 minutes later. If still <4 mmol/L, repeat above.
        - Monitor in 30-45 minutes. Patient should have a meal.
    - In patients who are unconscious and/or seizing and/or aggressive:
        - Start with ABCDE.
        - If the patient is on insulin infusion, stop the infusion.
        - If IV access is available, give 100 mL of 20% glucose at 400 mL/hr or 200 mL of 10% glucose at 800 mL/hr over 15 minutes.
        - Recheck blood glucose 10 minutes later. If still <4 mmol/L, repeat above.
        - Once the patient recovers, they should have a normal meal.

Complications

- DKA and HHS
    - Life-threatening arrhythmias secondary to electrolyte abnormalities
    - Hypovolemic shock
    - Cerebral edema - more common in kids (rare but serious)
    - Hypoglycemia secondary to over-treatment
- Hypoglycemia
    - Altered mental status, decreased level of consciousness, coma, and death

Disposition

- Ideally, patients in DKA or HHS should be admitted to an intensive care unit for careful monitoring.
- Once the patient's pH, electrolytes, and fluid status have been stabilized, they can be switched from IV to subcutaneous insulin.
- *Educate the patient* on diabetes and insulin use before sending them home!

Special Notes

- A normal blood glucose does not necessarily rule out DKA. Euglycemic DKA is possible in the following situations: pregnancy, use of insulin before ED, starvation, and use of SGLT-2 inhibitors.
    - These patients should be treated the same as those with usual DKA. They will need initiation of dextrose-containing fluids at the same time as their insulin infusion.
    - The end goal of insulin therapy is the same - resolution of acidosis.
- When giving IV fluids to pediatric patients with DKA, do not give a fluid bolus unless they are in shock, as there is a risk of cerebral edema. Instead, give 10 mL/kg IV over 1 hour.
- The goal of treatment of diabetic emergencies is NOT treating hyperglycemia. The goal is to manage the associated complications (e.g. electrolyte disturbances, dehydration, acidosis).
- Normal saline in large volumes can cause hyperchloremic metabolic acidosis. This makes it challenging to know whether DKA has been fully treated, since the patient may still be acidotic but have closure of their anion gap.
    - If you have access to balanced crystalloids (i.e. lactated ringers), these may actually be preferable for treating DKA. However, you should always follow your institutional protocols.[5]

Collaborating Partners

1. Ministry of Health of the Republic of Uganda
2. Seed Global Health
3. Techies Without Borders

References

1. “Uganda.” 2023. International Diabetes Federation. April 25, 2023. https://idf.org/our-network/regions-and-members/africa/members/uganda/. Accessed August 14, 2024.
2. The Republic of Uganda Ministry of Health. *Uganda Clinical Guidelines 2023: National Guidelines for Management of Common Health Conditions.;* 2023. Accessed August 15, 2024. The Republic of Uganda Ministry of Health - 2023 - Uganda Clinical Guidelines 2023 National Guidelin.pdf
3. Helman, A. Baimel, M. Sommer, L. Tillmann, B. Episode 146 – DKA Recognition and ED Management. Emergency Medicine Cases. September, 2020. https://emergencymedicinecases.com/dka-recognition-ed-management. Accessed August 20, 2024.
4. Ravert D. Hyperosmolar Hyperglycemic State. In: Johnson W, Nordt S, Mattu A and Swadron S, eds. CorePendium. Burbank, CA: CorePendium, LLC. https://www.emrap.org/corependium/chapter/recuOCVHwMPsX4XWz/Hyperosmolar-Hyperglycemic-State#h.4jglllhinfto. Updated July 8, 2020. Accessed August 20, 2024.
5. Ravert D. Diabetic Ketoacidosis. In: Johnson W, Nordt S, Mattu A and Swadron S, eds. CorePendium. Burbank, CA: CorePendium, LLC. https://www.emrap.org/corependium/chapter/recvMoKn79II3JOa9/Diabetic-Ketoacidosis#h.9zdb6o6nz21i. Updated December 8, 2020. Accessed August 20, 2024.