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Approach to Hyperglycemia and Its Crises

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Introduction

Hyperglycemia may be simple due to uncontrolled or newly discovered DM, but may also be due an underlying serious condition like infection, stroke or even MI. Also hyperglycemia may be complemented with hyperglycemic crises, namely Diabetic Keto-Acidosis (DKA) and Hyperglycemic Hyperosmolar State (HHS).

Finger prick random blood sugar should be part of any initial vitals chart in order to avoid hyperglycemia from being easily missed. Hyperglycemia is defined as RBS more than 200mg/dl Management of hyperglycemia should focus on identifying any underlying life-threatening causes such as infections, Stroke, MI, acute abdomen, etc. And then properly treating these causes. Simple hyperglycemia is a diagnosis of exclusion which is regarded once more serious causes have been considered and excluded. Our initial approach as always is ABCDE, through which we will be able to identify and treat any underlying cause of hyperglycemia.

For example; examining the chest for signs of infection or distress during the B phase, noticing any signs of lateralization during our D phase or detecting fever or an abscess during the E phase of our ABCDE approach.

Hyperglycemia without any life-threatening causes can be controlled with adequate hydration. Patients with established DM can get 0.1units/kg of regular insulin SC.

Admit the patients according to their underlying illnesses.

Discharge patients with uncomplicated hyperglycemia with instructions for a healthy life-style including adequate water intake, exercise and omission of sugar or replacing it with diet sugar or stevia along with follow-up at the internal-medicine/ endocrinology clinic.

Diabetic Keto-Acidosis

DKA is caused largely by a relative or absolute lack of insulin. It is characterized by hyperglycaemia, volume depletion, ketonemia and metabolic acidosis. DKA is usually precipitated by uncontrolled DM, stress, infection, trauma, stroke, MI and also pregnancy. Basically, any stress factors can lead to anti-insulin hormonal (catecholamines, steroids and Growth hormone) gushes with subsequent hyperglycemia, relative insulin deficiency, ketosis and dehydration due to osmotic diuresis from glycosuria and ketonuria in a patient with DM.

DKA is common in but not exclusive to the paediatric and adolescent population and can be the first presentation of type 1 DM.

DKA is a serious illness and accounts for 16% of DM related fatalities in the developed world.

Consider DKA and order a VBG in patients with a RBS of 250mg/dl or higher. These patients may present with non-specific symptoms like malaise, headache, dizziness, blurred-vision, **vomiting, abdominal pain**, polydipsia, polyuria, fatigue, weakness, **breathlessness**, etc. They may also show symptoms of precipitating factors as shock, fever, sepsis, MI, trauma, stroke, etc.

Management

- Start with your **ABCDE** survey.
- Start a bolus of **2 liters of crystalloids**, better be Ringer's solution to avoid the risk of hyperchloremic acidosis, which may accompany high volumes of NS infusion. If head injury is suspected, please use NS, as Ringer's is mildly hypotonic and may cause brain enema. The fluid deficit is usually 100ml/ kg of total body weight, thus deficits may be as much as 8 liters. Response to therapy and acidosis should guide fluid therapy.
- Start IV insulin and do not give IV boluses, **only infuse at a rate of 0.1 units/kg/hour or give 0.1 units/ kg subcutaneously every 2 hours**. Insulin infusion is continued until the anion gap is closed completely.
- **Monitor K** levels as they may drop rapidly after initiation of fluid therapy. During acidosis, K shifts extracellularly, and then gets lost with osmotic diuresis. Thus, the patient may appear eukalemic despite actually being K-depleted. Add K to IV fluids once serum K drops below 5.5 and infuse at a rate of 20 mmol/hour. If K drops below 3-stop insulin infusion until K is replete.
- Withdraw blood and send lab requests for at least VBG, CBC, Na, K, Mg, PO4 (phosphate), serum glucose, Troponin, urea, creatinine, CRP and urine analysis.
- **Avoid correction of acidosis with Bicarb** unless fear of decompensated metabolic acidosis and respiratory failure is present, which usually takes place at PH less than 6.9. Boluses of 100-150 ml of 1.4% concentration of sodium bicarb can

be given when indicated and repeated every 30 minutes as necessary. Avoid correction of acidosis with Bicarb as this **may worsen hypokalaemia** and cause paradoxical intracellular acidosis.
- Order an **ECG** and a CXR/CT chest.
- Check for neurological deficits and order a CT brain if you detect any.
- Order a urine dipstick for glucose and ketones to establish a baseline. Dipsticks detect one type of ketone body (acetone) while our bodies produce three types. Hence, an initial ketone negative urine test should not rule out DKA. Hydroxyl-butyrate; a ketone that is usually produced in severe DKA is **undetectable** with dipstick. With hydration and improved tissue perfusion Hydroxyl-butyrate is converted into acetone in extra-hepatic tissues causing the so-called **paradoxical worsening**.
- Provide IM or slow IV Mg replacement.
- Monitor for and beware of **hypophosphatemia** which may ensue with insulin treatment and lead to neurological deficits and cardio toxic effects in the form of disturbed consciousness, decreased cardiac contractility, cardiac ischaemic changes in ECG and even positive troponins (all amenable to correction with minimal doses of IV phosphate as low as 10-50 mmol). Insulin causes an intracellular shift of phosphate, which is at times already depleted, with the osmo-duretic effect of glycosuria caused by hyperglycemia and DKA. Phosphate is important for the production of ATP (adenosine triphosphate) and the reduction of ATP production causes an ischaemia-like state. **Do not empirically give phosphate**, provide only whenever phosphate levels are below 2.2 mmol. Hypophosphatemia is rare but is never considered as a cause of persistent DCL and cardiotoxicity with treatment unless the clinician is **aware** of it in the first place.
- **Always look for and treat the underlying cause of DKA**.
- **Admit** all patients with DKA to the ICU.

Hyperglycemic Hyperosmolar State
Is characterized by altered mental status, severe hyperglycemia (above 900mg/dl), hyperosmolarity, a PH above 7.3(unless coupled with lactic acidosis in late cases) and no ketoacidosis.

Symptoms can be non-specific such as weakness, vomiting, thirst, polyuria, dehydration, reduced skin turgor, dry mucous membranes and symptoms of associated underlying causes. However, **altered mental status** in the form of coma or lethargy is characteristic of HHS.

Diagnosis of HHS is suspected once a patient is hyperglycemic, mentally altered, non-academic and shows signs of dehydration.

Management of HHS focuses on correction of **electrolyte disturbances** and **volume** depletion.
- **ABCDE** approach is how we approach all our patients in the ED.
- Hyperglycemia is usually corrected by **IV fluids** alone, but if it persists after euvolemia, you can add 0.1unit/kg bolus given IV (maximum 10 units).
- Boluses of 250 ml of crystalloids are given until euvolemia is achieved, then further resuscitation is guided by the patient's cardiogenic and osmolar status.
- If the patient's corrected Sodium is elevated, resuscitate with 0.45% Saline (half water-half normal saline).
- Monitor serum **Potassium** and give 20mmol/hr. once serum K drops below 4.5.
- Avoid bicarb unless PH is below 6.9.
- Routine labs include CBC, serum glucose, urea, creatinine, electrolytes, VBG, CRP, Troponin, urine analysis.
- Obtain an ECG, CXR/CT-chest and a CT-brain.
- Direct your investigations towards **finding the precipitating factor** e.g. stroke, MI, infection, etc.
- **Treat the cause** of this crisis.
- **Admit** all patients to the ICU.

Glossary:
ED Emergency department
ABCDE universal approach to patient Airway, breathing, circulation, disability then exposure
DKA Diabetic keto-acidosis
DCL Disturbed conscious level
HHS Hyperglycemic hyperosmolar state
Na sodium
K potassium
Mg magnesium
Mg milligrams
L liter
D L deciliter
ICU intensive care unit
Mmol millimole
HR hour
PH Potenz hydrogen ion (a scale for acidity from 1 to 14)
IM intramuscular
IV intravascular
CBC complete blood count
VBG venous blood gases
CRP C-reactive protein

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