Diabetic Emergencies...

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Alabama EMS Challenge
Goals and No Disclosures

- Diabetes
  - Definitions
  - Some Pathophysiology and input on current treatments
- Hyperglycemic complications and treatment
  - DKA
  - HHS (HONK)
  - Hyperglycemia
- Hypoglycemic complications, risk factors, and treatment
- Key points in EMS care of the diabetic patient
May not be this...

But, hopefully not this...
Diabetes - Definitions

- A group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.
- In the 1950s, 1/5 people died within 20 years after a diagnosis of type 1 diabetes. 1/3 died within 25 years of diagnosis.
- Now—can live relatively normal life span (before insulin, they all died)
Type I Diabetes:

- 10% of all Diabetic cases
- Auto immune destruction of *Islet of Langerhans* in Pancreas
  *(Isle of Dread)*
- So....no or little insulin
  - Usually diagnosed in children and young adults.....*but*...
Fig. 2: The actions of insulin.
Treatment

On January 11, 1922 a 14-year-old diabetic who lay dying in Toronto, was given the first injection of insulin.

Now we have:
Long acting
Intermediate acting
Short and ultra fast acting
Sliding scales/Infusion pumps
Hyperglycemia – Acute or chronic: either way its bad...

- Present in all types of DM and is a main factor leading to long term complications now.

- End organ damage
  - Neuropathy (osmol)
  - Renal disease
  - Retinal disease
  - Chronic “stressor”
    - (platelet activation)
    - Catecholamines
Type II Diabetes:

- ~80-90% of all Diabetic cases
- failure of circulating insulin to act on various tissues – INSULIN RESISTANCE
- a “relative” insulin deficiency
  - Insulin may or may not part of treatment at first
- Usually diagnosed after 40 years old (Ha!)
Oral Treatment’s

- Sulfonylurea
- Metformin
- Invokana

Drug Sites of Action

- Glucose absorption:
  - GLP-1, DPP-IV inhibitors delay gastric emptying
  - Alpha glucosidase inhibitors block breakdown of complex carbs into glucose

- Muscle:
  - Improved insulin sensitivity: thiazolidinediones (TZDs), metformin (lesser effect)

- Liver:
  - Glucose production:
    - Metformin (biguanide)

- Pancreas:
  - Increased insulin secretion:
    - Sulfonylureas, non-sulf. insulin secretagogues, GLP-1, and DPP-IV inhibitors

- Kidney:
  - Increased glucose and sodium excretion:
    - Sodium-glucose co-transporter 2 (SGLT2) inhibitors
Treatment

- Sulfonylurea
  Wear it out...
- Hypoglycemia
Metformin

- Lactic Acid
**Transport Maximum**

- There is a limit to the amount of solute that the renal tubules can reabsorb.
- Limited by the number of transport proteins in the plasma membrane.
- If all transporters are occupied as solute molecules pass, excess solutes appear in urine.
- **Transport maximum** is reached when transporters are saturated.

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- **Invokana**
  - Sweet pee...
- Sulfonylurea
  - Hypoglycemia
  - Wear it out

- Metformin
  - Lactic

- Invokana
  - Sweet pee...
  - Hypoglycemia
  - Dehydration with good glucose control
• 50 y/o male  call for AMS
• Vital Signs
  • Pulse 124/ resp 34/Bp 95/60, Spo2 96% , end tidal 9,  D-stick “Hi”
• Exam
  • Alert, “Ethyl” , dry,
- Altered....
- ? PMH... “sugars”
- Vital Signs
  - HR 124
  - Bp 95/60
  - RR 30 +
  - Spo2 96
  - D-stick “Hi”
  - EtCo2- 9
Diabetic Ketoacidosis

- Metabolic derangements in DKA are indirect or direct consequences of the lack of insulin! (initial DM diagnosis or med non compliance)

- Insulin insufficiency = inability of glucose to enter cells = hyperglycemia and cellular starvation

- Cellular starvation = release of stress hormones (glucagon) = and increased free fatty acids

- Free fatty acids = ketone bodies = acidosis

- Dehydration
- Acidotic—Aerobic for several reasons
- Hyperkalemia
- Lack of insulin –Infection
- Acute MI/ CVA—Stress response
- Trauma/Surgery—Stress response/dehydration
Treatment

D stick  Usually > 250 (higher)

- IVF
- Calcium
- Insulin !!!!
- Sodium Bicarb
- Did I say fluids...
- Respiratory control ?
  - These patient’s usually have large volume deficits

- What can you do in the field?
- Can u diagnosis this ? Yep...
HHS/ HONK

- Hyperosmolar Hyperglycemic State
- Characterized by progressive hyperglycemia and hyperosmolality typically found in a debilitated patient with poorly controlled or undiagnosed type II diabetes
- Development of HHS related to:
  - Insulin resistance or deficiency or BOTH
  - Increased hepatic gluconeogenesis and glycogenolysis
  - Osmotic diuresis and dehydration
HHS Pathogenesis

- Mild hyperglycemia and stress -> increased insulin resistance -> increased insulin levels -> increased gluconeogenesis and glycogenolysis -> increased glucose without ketones -> increased osmolality. Increased dehydration and diuresis with sodium depletion.

- Key points: Hyperglycemic, Hyperosmolar, NO ACIDOSIS. (But...)

- HHS usually develops more slowly – sometimes over weeks.
Etiology of HHS

- Infection
- MI
- CVA
- GI Bleed
- Uremia
- Medications
- Trauma
- Pulmonary Embolism
- Heat related illness
- Rhabdomyolysis
- Non compliance with insulin therapy
- New onset diabetes
HHS: Clinical Presentation

- Most pronounced and consistent findings in these patients are Neurologic
  - 10% actually present in coma
  - Most have some degree of AMS
  - Focal Neuro deficits are NOT uncommon
- Typical presentation is a myriad of vague complaints
  - Weakness, fatigue, dehydration, anorexia, blurry vision, exacerbation of co morbid disease
- Sings of dehydration are usually clinically apparent
  - Tachycardia, postural hypotension, dry membranes,
HHS Initial Evaluation and Management

- D stick ...diagnose it... HR/RR/Capnography
- As with DKA, let your history guide you as to potential cause....
- ABC’s!
  - These patients most often have some degree of neurological manifestation – even coma!
- Fluid bolus
  - The average fluid deficit in these patients is 8-12 L
Hyperglycemia

- Acute hyperglycemia defined as BG greater than 300
- Absence of ketosis
- Without labs this can be difficult to identify but let your clinical picture guide you
  - Sick/ Not sick, end tidal
- Can present in several ways!
- Lab abnormality in an otherwise asymptomatic patient
- Can be secondary to poor DM management or initial presentation of DM or ATP (acute Twinkie poisoning)
Hyperglycemic Spectrum: Take home points

- A good history can go a long way
- Prompt diagnosis of some eliciting causes is vital to save lives – EKG!
- IV fluids/ IV Fluids / IV Fluids
- In the intubated DKA patient – hyperventilate
  
  (That’s the only patient population to hyperventilate)

\[ D\text{-stick} \ < \text{“high” and not sick-hyperglycemia} \]
\[ D\text{-stick} \ > \text{“high” and not sick- HONK} \]
\[ D\text{-stick} \ > \text{250 ish and sick, Increased HR/RR -> DKA} \]
Hypoglycemia

- Clinical Definition:
  - Symptoms consistent with diagnosis
  - Low glucose level
  - Symptoms resolve with glucose administration
- Some estimate that over 99% of hypoglycemic episodes occur in diabetic patients on insulin therapy / Sulfonylureas
- Although there is individual variation, signs and symptoms of severe hypoglycemia do not usually occur until the BG is less than 50: (ha)
  - Diaphoresis
  - Tremulousness
  - Tachycardia
  - Altered sensorium
  - Focal neurological deficits
  - Seizures
Case

34 y/o female, HX DM, has Insulin pump, 55 Kg
HR 94, BP 112/68, spo2 96%, glucose “low”
diaphoretic, non verbal, can’t get IV access

What you do?
Hypoglycemia: Evaluation and management

- D stick
- Obtain a history if able
- If patient is awake and alert – they can eat
- IV glucose is the most effective treatment (is there too much?)
- IM glucagon can be used when IV access is not readily available and/or if the patient is not elderly or alcoholic (decreased glycogen stores)
- rectal meds?
Hypoglycemia: Some take home points

- Obtain a history!
- Hypoglycemia is usually a complication of treatment!
  - Most frequently insulin and sulfonylurea's
- **Patients who are hypoglycemic due to sulfonylurea medications (glipizide, glyburide) they may rebound after treatment! (Like Narcan)**
  - Push these patients for transfer
  - They can PRT but document your discussion
- Remember there is both long and short acting insulin
  - Ask the patient what they took! **Look it up....**
- If the patient is awake, oral glucose is fine
- Remember – **hypoglycemia is one of the H’s and T’s**
Bonus points....

- 84 y/o abd pain, hx of HTN, but not DM : )
- Glucose 400, Resp Rate 32, spo2 98, end tidal 9
- BP 90/60 and HR 116
- EKG with prolonged PR and peaked t waves, wide QRS...
Case: Confusion

What’s going on?

What you do?

- 84 y/o abd pain, hx of HTN, but not DM : )
- Glucose 400, Resp Rate 32, spo2 98, end tidal 9
- BP 90/60 and HR 116
- EKG with prolonged PR and peaked t waves, wide QRS...
Diabetic Mimics

- Sick people get naked...and a complete exam....more history
  - Abd exam tight, tender...recent “colon infection“ now with bowel perforation -->
    - Stress response
    - Septic
    - Needs the OR...
  - But our initial treatment is still OK !!!!
    - Unless we miss the belly exam...
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