11 year-old female with altered mental status in the setting of diabetic ketoacidosis

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Chief Complaint

- 11 yr and 10-mo female with history of Type 1 DM from out of state
- Presented with nausea and elevated blood sugars x 1 day
- Visiting father in Chicago over winter vacation

History of Present Illness

- Day prior to admission:
  - Felt “sick” in the evening, did not sleep well
  - Several episodes non-bloody, non-bilious emesis
  - Generalized abdominal pain
  - Loose stool x3-4
  - Diminished appetite
  - Dehydrated 2º polydipsia -> rehydrated with OJ
  - Polyuria x 1 day, no dysuria
- Blood sugar “high”; + ketonuria → urgent care (no sick day rules attempted)

Diabetes History

- Diagnosis: 9 yo when admitted with DKA
- Hospitalizations for DM: once (at onset)
- Follows Endocrine physician group out of state
  - Last visit: 2 wks PTA
  - Last HgbA1c: 11%
- Insulin pump 2012-13
  - d/c'd 2 wks PTA for non-compliance

Home Diabetes Management

- Home insulin regimen:
  - Lantus 15 units qhs
  - Humalog mealtime 1:15g with BF, L; 1:20g with D
  - Humalog hyperglycemia correction = 1u:75 >100
- Insulin injection sites: abdomen, arms
- BG monitoring: Reported 5x/day

Glucometer Interrogation

	BF	L	D	Bed	4am
12/28 (Admit)					
12/27			357	258	
12/26	62			145	114
12/25	138				
12/24					
12/23	124				
12/22				57	

More History…

- **Past Medical History:**
  - Type 1 DM
- **Surgical History:**
  - None
- **Allergies:**
  - NKDA
- **Medications:**
  - Insulin (as previously described)
- **Social History:**
  - "Good Student"
  - Lives in mother
  - Visiting father in Chicago over holidays
- **Family History:**
  - No diabetes, thyroid disease or other autoimmune disease

Review of Systems (Page 1 of 2)

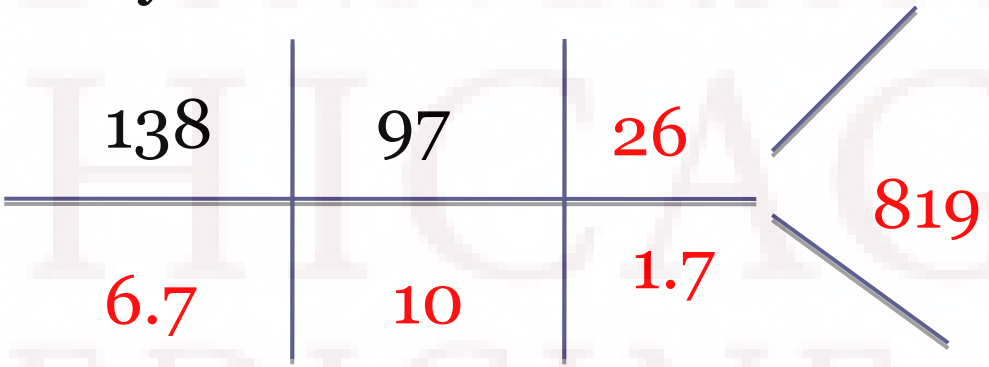
- General:
  - +fatigue, +anorexia, +weight loss (4lb), +polydipsia.
  - No fever or chills.
- HEENT:
  - +dry lips/mouth.
  - Negative for congestion, rhinorrhea, dysphagia, sore throat.
- Cardiac:
  - +chest pain with deep breath and ”heart racing.”
  - No lower extremity edema.
- Pulm:
  - +shortness of breath x 1 day.
  - No cough.

Review of Systems (Page 2 of 2)

- Abdomen:
  - +generalized abdominal pain. +nausea, +vomiting, +loose stool.
- GU:
  - +polyuria
  - No dysuria.
- Skin:
  - +dry skin.
  - No rash.
- MSK:
  - +generalized muscle pain/arthralgias x 1 day.
- Neuro:
  - +confused, +headache 6/10.
  - No seizures.

Urgent Care Course (OSH)

- Serum Chemistry:



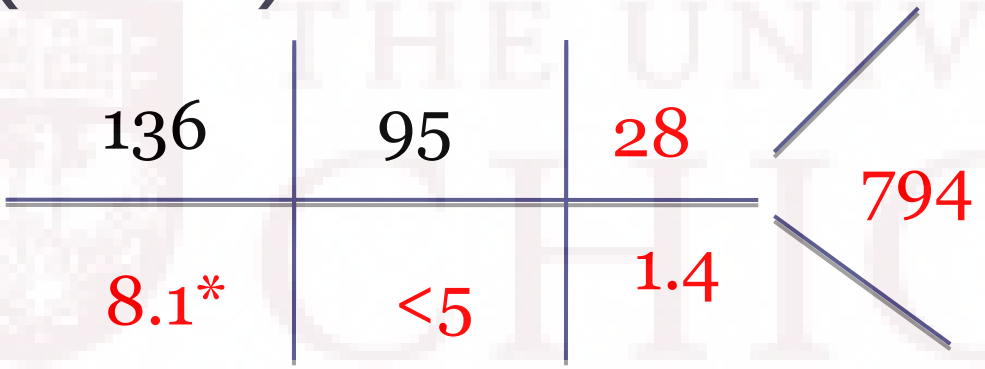
- Anion Gap: 36
- Urine ketones: >160 mg/dL
- Treatment:
  - 20cc/kg bolus NS and transfer to Comer Children's PICU

Physical Exam upon arrival to PICU

- Vitals: T 99.7F, P 147, BP 111/57, R 28, 100% on room air, Wt 35.9kg
- General: appears uncomfortable
- HEET:
  - conjunctiva normal, oropharynx clear.
  - +dry mucous membranes.
- Neck:
  - supple.
  - +thyroid mildly enlarged, symmetric.
- Chest: tanner IV breast.
- CV:
  - +tachycardia
  - no murmur.

- Pulmonary:
  - Deep, labored breathing
  - clear to auscultation
- Abdominal:
  - normal bowel sounds, soft, non-distended
  - +tender diffusely, no guarding or rebound
- Genitourinary: Tanner 4 pubic hair
- MSK:
  - tender diffusely
- Neuro:
  - +decreased muscle tone.
  - waxing and waning mental status
- Skin:
  - warm, cap refill < 3 sec.
  - +diaphoretic. +pallor. +lipohypertrophy on back of the arms.

Laboratory Studies on Admission (22:00)



- Ca: 10.1
- Mag: 2.8
- Phos: 5.7

- VBG: pH 7.061, POC2 15, Base excess -24
- Beta-OHB: 10.14 mmol/L
- Lactate 4.67 mmol/L
- Serum osmolality: 358 (275-295 mOsm/kg)
- Urinalysis: 1.027, 1+ protein, 3+ glu, 3+ketones, (-) LE, (-) WBC
- HgbA1c: 10.6%

Next Step in Management

21:00

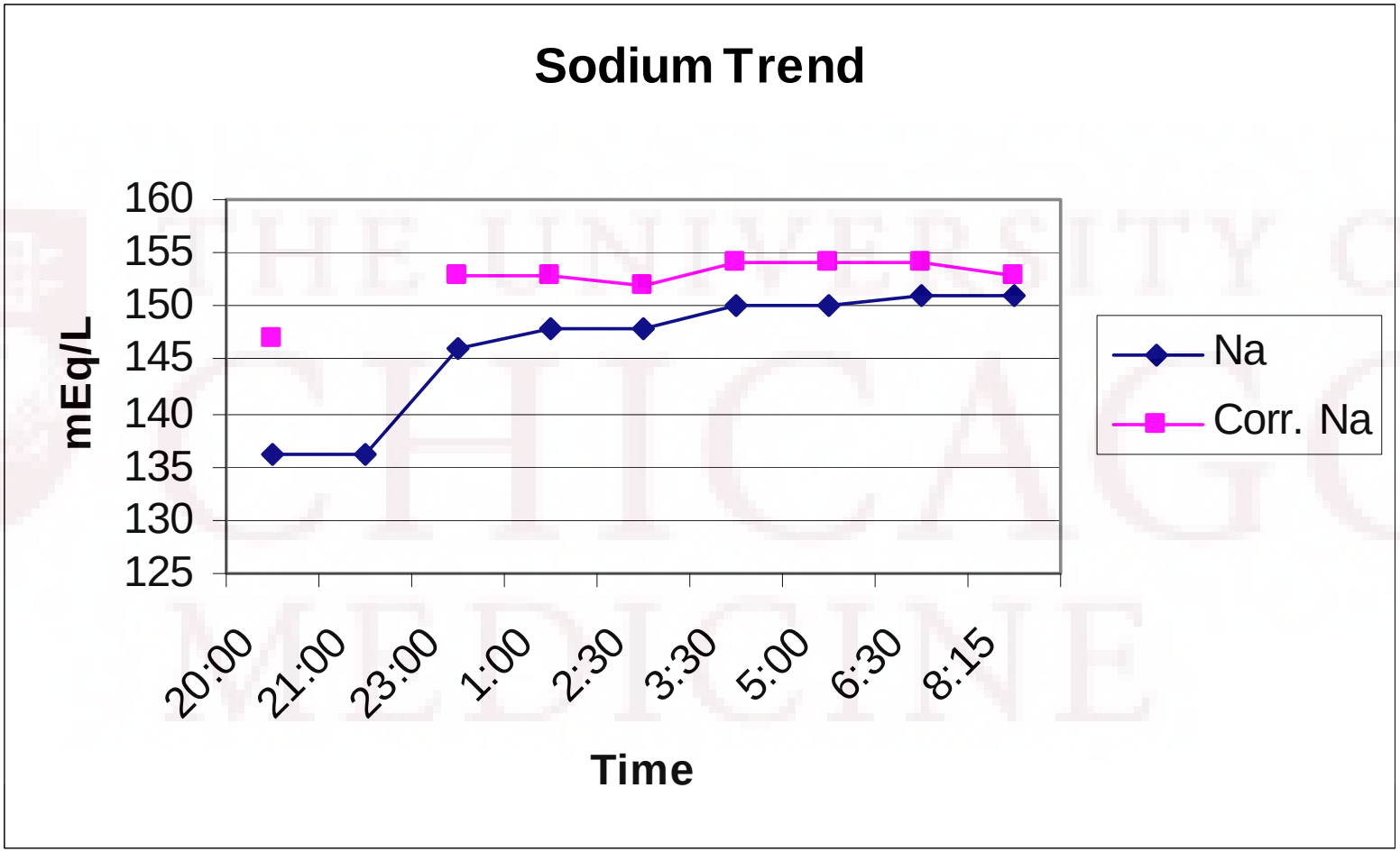
- Started continuous infusion insulin 0.1 units/kg/hr
- Started NS at 110 cc/hr (= MIV rate)
- Attempt A-line for more frequent lab monitoring

Overnight Course

Time	POC Glu	Glu	pH	Na	*Na	HCO3	AG	BHB	Osm	Clinical Course
20:00	> 550	794	7.06	136	147	4.2	48	10.1	358	
21:00		> 700	7.14	136		4.6				Start insulin gtt, NS, attempt A-line
22:30	> 550									Changed fluid to half rate D10NS + half rate NS
23:00		535	7.15	146	153	<5	37	>9		Change in mental status +headache

*Na = corrected Na

Time	Glu	pH	Na	*Na	HCO3	BHB	Osm	Clinical Course
23:00	535	7.15	146	153	<5	>9		Change in mental status +headache
24:00	442	7.18	150	155	7			
01:00	418	7.28	148	153	8			
02:30	342	7.25	148	152	12			Changed to D10NS
03:30	351	7.25	150	154	13	5.05		
05:00	344	7.26	150	154	17			
06:30	284	7.3	151	154	17			
08:15	244	7.33	151	153	21	1.23		


Sodium Trend
mEq/L
160
155
150
145
140
135
130
125
20:00
21:00
23:00
1:00
2:30
3:30
5:00
6:30
8:15
Time
Na
Corr. Na

Time	Glu	pH	Na	Corr Na	HCO 3	AG	BHB	Osm	Clinical Course
08:15	244	7.33	151	153	21		1.23		
09:00	190								
10:00	132								Headache continues
11:00	167								
12:00	189	7.31	152		20		0.25	319	Endo examined -> confusion
20:30	177	7.4	145	146	20	10			

Case Summary

- Pubertal female w/ T1DM under the care of a parent unfamiliar with DM sick-day management
- Presented with severe dehydration, DKA and hyperosmolarity in setting of insulin resistance +/- insulin omission
- Had steep drop in BG soon after initiation of treatment for DKA
- Developed mental status changes and treated with 3% HS with significant improvement

Cerebral Edema in DKA

- Life-threatening consequence of DKA
- Occurs in 0.5-1% of children with DKA
- Mortality is 21-24%
- Young children > adolescents > young adults
- Pathophysiology not well-understood
  - 1) Cytotoxic edema
  - 2) Vasogenic edema

Bohn, Desmond. Pediatric Critical Care Medicine 2010.
Shore, Paul. Pediatric Critical Care Medicine 2011.
Wolfsdorf et al. Pediatric Diabetes 2009.

Clinical Questions

1. What are the most sensitive and specific signs/symptoms of cerebral edema in children?
2. Which osmotic agent is more effective in treating cerebral edema: hypertonic saline or mannitol?

Cerebral Edema in Childhood Diabetic Ketoacidosis

Natural history, radiographic findings, and early identification

Table 1—*Bedside evaluation of neurological state of children with DKA*

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 (e.g., grunting, tachypnea, Cheyne-Stokes respiration, apneusis)
Major criteria
Altered mentation/fluctuating level of consciousness
Sustained heart rate deceleration (decline more than 20 bpm) 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

Signs that occur before treatment should not be considered in the diagnosis of cerebral edema.

Muir et al. Diabetes Care 2004.

Cerebral Edema Diagnostic Criteria

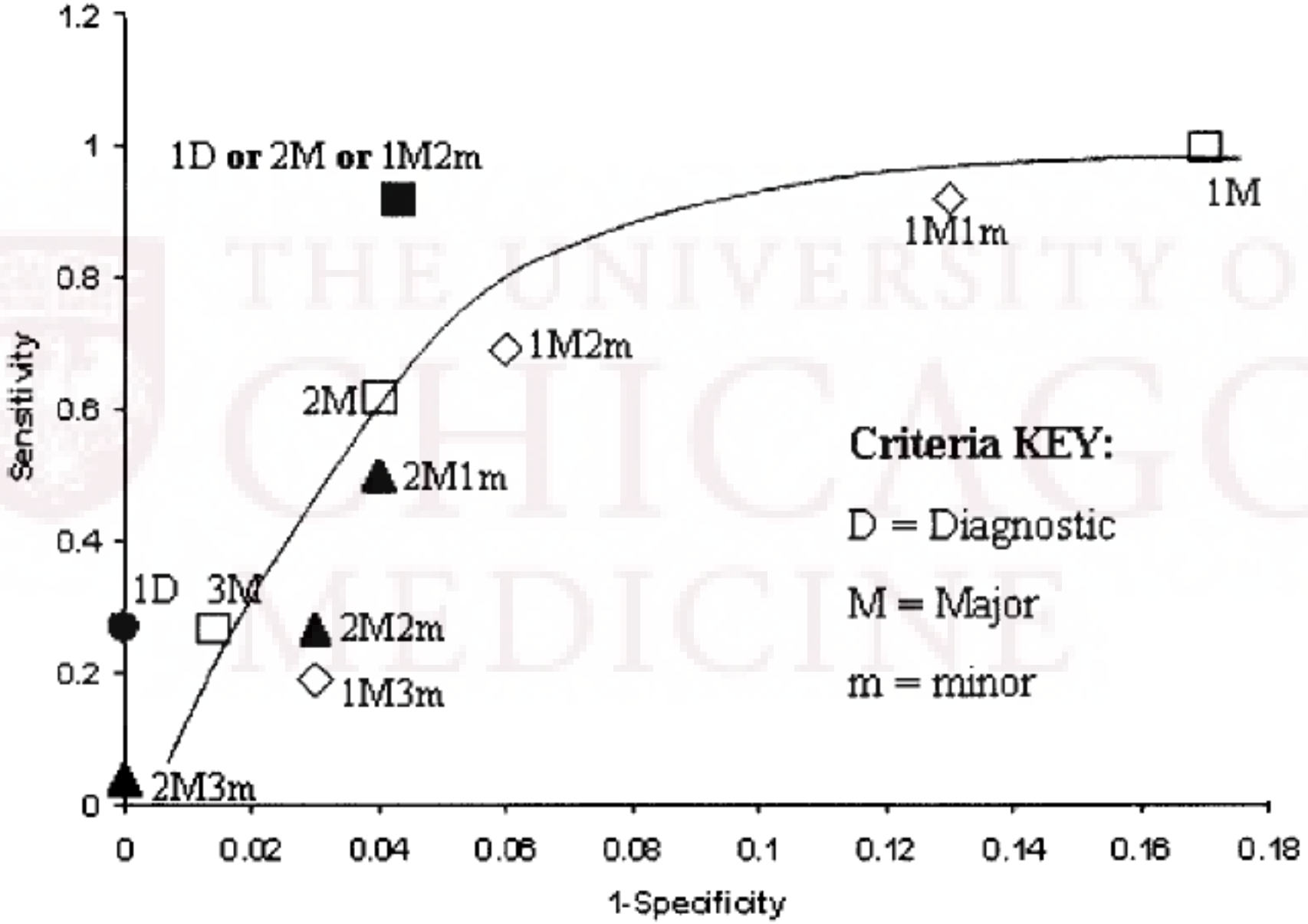
- Abnormal motor or verbal response to pain
- Decorticate or decerebrate posture
- Cranial nerve palsy (esp III, IV, VI)
- Abnormal neurogenic respiratory pattern

Muir et al. Diabetes Care 2004.

Early Indicators of Cerebral Edema

- Major Criteria
  - AMS
  - Sustained HR deceleration not attributable to improved intravascular volume
  - Age-inappropriate incontinence
- Minor Criteria
  - Vomiting
  - Headache
  - Lethargy/Not easily aroused
  - Diastolic BP >90mmHg
  - Age <5

Muir et al. Diabetes Care 2004.



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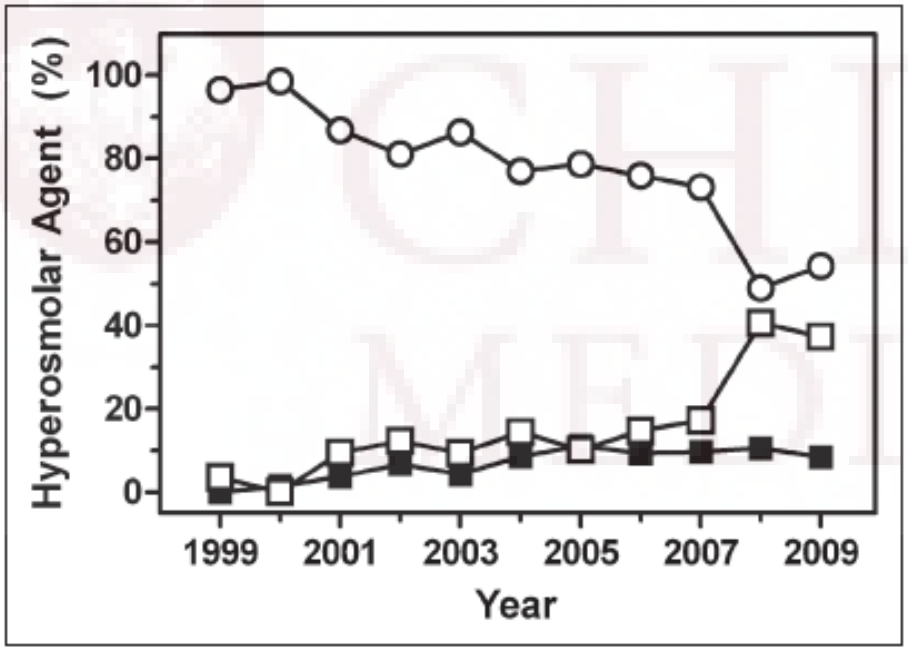
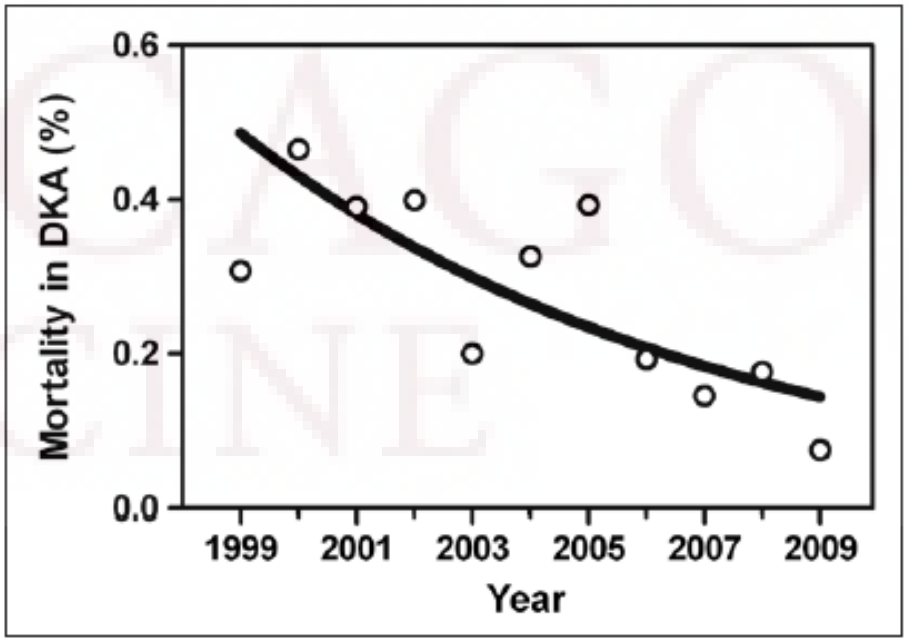
Clinical Question

- Which osmotic agent is more effective in treating cerebral edema: hypertonic saline or mannitol?

Mannitol vs. Hypertonic Saline for Treatment of Cerebral Edema





Decourcey et al. Pediatric Critical Care Medicine 2013.

TABLE 3. Adjusted Odds Ratio of Mortality in Patients Treated for Cerebral Edema in Diabetic Ketoacidosis

Characteristics	OR (95% CI)	Adjusted OR (95% CI)[a]
Treatment with hypertonic saline alone	2.03 (0.94–4.39)	2.71 (1.01–7.26)
Male gender		3.45 (1.79–6.65)
Mechanical ventilation		22.8 (10.7–48.9)
Brain imaging with CT		2.14 (1.00–4.57)
International Classification of Diseases, 9th Revision code		
250.2		3.84 (1.29–11.4)
250.3		3.31 (1.46–7.47)

OR = odds ratio.

Decourcey et al. Pediatric Critical Care Medicine 2013.

Learning Points

- Cerebral edema is a devastating complication of DKA.
- Mechanism of cerebral edema in DKA is still unknown, but is likely multi-factorial.
- Cerebral edema is a clinical diagnosis and should be identified early with the bedside examination.
- Hypertonic saline may be associated with higher mortality than mannitol for the treatment of DKA but there are no definitive prospective trials comparing the two treatments.

Works Cited

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