METABOLIC ACIDOSIS

Sushma Bhusal

1.6.2015
CASE PRESENTATION

• CC: 50 F Caucasian F transferred to BHC on 11/15/14

• Presented to OSH with 4 days of
  • Diffuse abdominal pain
  • Watery diarrhea, several episodes of vomiting
  • Fevers to 102

HPI

• Several Hospital admissions
CASE PRESENTATION

PMH: DM2, HPL, Recurrent UTIs, PVD with SMA thrombosis

PSH:
  • 1998 Cholecystectomy
  • 6/2013: Recanalization of SFA and Popliteal Atherectomy and Balloon Angioplasty, R iliac - femoral bypass
  • 9/2013: SMA thrombosis s/p ex lap with small bowel ascending/transverse colon resection at OSH (patient left with 19 cm of jejunum distal to ligament of Treitz and L colon)
  • 10/2013 Ex-lap, wash out and primary anastomosis: c/b C diff, prolonged intubation, started on TPN
CASE PRESENTATION

- Was on home TPN for about a year, tolerated well

- **10/16/14**: Admitted to OSH for sepsis from UTI, treated with antibiotics, developed severe abdominal pain and hematemesis, EGD showed multiple gastric ulcers with nodular mucosa and ischemia

- Transferred to Bellevue on 10/22/14. CTA of abdominal aorta showed new celiac artery stenosis in addition to chronic SMA thrombosis

- Celiac angiogram and angioplasty with stent. Discharged on 10/24/14

- Readmitted on 11/15/14
CASE PRESENTATION

➢ Social: Smoker 30 PPD, no alcohol or illicit drug use

➢ FH: Non contributory

➢ Allergies: NKDA

➢ Medications:
  • ASA 81 mg PO daily
  • Simvastatin 40 mg PO daily
  • Plavix 75 mg daily
  • Cefuroxime 500 mg bid
  • Methadone 5 mg PO q8hr
  • Oxycodone 10 mg PO q8hr prn
  • Cyclobenzaprine 10 mg PO tid
  • Mirtazapine 5 mg PO tid
  • Meclizine 12.5 mg tid
  • Nexium 40 mg PO daily
PHYSICAL EXAM

- Vitals: T 98.9, HR 95, RR 19, BP 116/57, 99% RA
- Gen: Emaciated, Alert, NAD
- HEENT: dry mucous membranes
- Resp: CTABL
- CV: S1S2+, regular, no m/r/g
- Abdomen: Diffuse tenderness, BS+, well healed surgical scars
- Ext: Thin, no edema
**LABS**

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UA: Large blood
Protein Mod
RBC – 15 – 30
Many bacteria
Mod yeast
LE / Nit - Neg

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Blood cultures drawn
IMAGING

- CXR: Plate-like atelectasis

- CE CT A/P: Patent Celiac artery stent, distal anastomosis site mild hyper-enhancement with mesenteric fluid s/o ischemia, heterogeneous liver s/o congestive hepatopathy, Patent R iliac femoral bypass
HOSPITAL COURSE

• Blood cultures: Candida albicans

• Treated for Fungemia with Voriconazole

• TPN stopped, Tunneled Cath removed on 11/19/14

• On PPN 11/25/14 - 12/1/14 when TPN restarted after PICC placement

• Meanwhile developed tachycardia and pleuritic chest pain, CT PE on 12/1/14 revealed segmental PE, started on heparin drip, extensive hypercoagulability work up revealed anti-thrombin III deficiency (58%)
HOSPITAL COURSE

• Nephrology consulted on 11/28/14 for hyperkalemia with EKG changes

• Deemed to be from excessive K replacement when GFR had halved (Cr 0.4-0.8) (160mEq)

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HOSPITAL COURSE

- Treated with lasix, insulin, dextrose, Ca gluconate, kayexelate

- EKG changes and hyperK resolved

- Developed fluctuating metabolic acidosis (AG + Normal AG)
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**VBG 12/3/14**: pH 7.30 / PCO2 34 / HCO3 16

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**ABG 12/11/14**: pH 7.01 / PCO2 21 / HCO3 5
**ADDITONAL LABS**

**UA:**
- Blood: 3+
- pH: 6
- Protein: 2+
- WBC: 2-5
- RBC: 5-10

**VBG**
- pH: 7.30
- PCO₂: 34
- PO₂: 35
- HCO₃⁻: 17

**BMP**
- 135
- 110
- 10
- 292
- Ca⁺: 8.6

**Urine AG:**
- (Na⁺ 24 + K⁺ 21) - Cl⁻ 71 = -26

**Urine Osm:**
- 270
DIFFERENTIALS: NORMAL ANION GAP ACIDOSIS

- Diarrhea, loss of bicarbonate
- Hyper alimentation from TPN
- Distal RTA (Urine AG negative)
- Use of PPI (no other culprit medications)
DIFFERENTIALS: ELEVATED ANION GAP ACIDOSIS

- Lactate negative
- D lactic acidosis from short gut
- Diabetic ketoacidosis, no ketones
- TPN related
TREATMENT

• Patient treated with bicarbonate drip, PO K citrate, K bicab, TPN adjustments for electrolytes, acetate

• Still with intermittent severe acidosis

• Mainly contributed by bicarbonate losses in the GI tract
SHORT BOWEL SYNDROME AND METABOLIC ACIDOSIS

• Gut electrolyte processing
• Renal bicarbonate handling
  • Reabsorption
  • NAE
• Small bowel syndrome
• D Lactic acidosis
• Gut processes about 8-9 L fluids per day

• Derived from oral intake and endogenous secretions

• Absorption process functions with 98% efficiency, only 100-200 ml excreted per day

Sleisenger and Fordtran. Chapter 99. 9th Ed
CJ ASN 2008
GUT AND ACID BASE HOMEOSTASIS

• Large amounts of H⁺ and HCO₃⁻ traverse specialized epithelia of various segments of gut

• Under normal circumstances only 30-40 mmol of bicarbonate lost in stool

• As opposed to Kidneys (acid base balance), intestines designed for absorptive function

• Fluid and electrolyte transport primarily driven by Na/K-ATPase in basolateral membrane and various apical transporters
GI ELECTROLYTE TRANSPORTERS

Stomach

Gastric parietal cell

Lumen

Pancreas

Pancreatic duct cell

Lumen

Small Intestine

Absorptive cell

Secretory cell

Lumen

Colon

Absorptive cell

Secretory cell

Lumen

*Cystic fibrosis transmembrane conductance regulator
**Downregulated in adenoma gene product
***Short chain fatty acids

CJ ASN 2008
OVERVIEW OF GUT SECRETION

Understanding Acid Base: Abelow
• A 70-kg human contains a free [H+] of 40 nM in about 42 L of water

• Consumption of a high-protein Western diet results in a net production of 50–70 mEq of H+ per day

• Lack of appropriate buffer, the daily production of H+ will decrease pH < 3 within an hour

• The kidney is the primary organ that controls plasma [HCO3 2- ]

• Kidneys have to excrete acid equivalent: Daily Net H+ plus filtered HCO3 2 –

**CJASN 9: 1627–1638, 2014**
BICARBONATE REABSORPTION

Secretion of H⁺ in the α-Intercalated Cell of the Cortical Collecting Duct

Lumen

Interstitium

Voltage +10 mV

-30 mV

O mV

Active transport ➞ Channel ➞ Passive transport
Kidneys excrete acid (reclaim bicarbonate) in the form of titratable acidity and ammonia excretion

Net Acid Excretion: \((U\ Am\ V + U\ TA\ V) - U\ HCO3\ V\)

Under normal conditions: 40% NAE (TA), 60% Ammonia and bicarbonate 0
AMMONIUM EXCRETION

Understanding Acid Base: Abelow
SMALL BOWEL SYNDROME: ACID BASE

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<td>Salivary</td>
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<td></td>
</tr>
<tr>
<td>Gastric</td>
<td>2L</td>
<td>-</td>
</tr>
<tr>
<td>Pancreas</td>
<td>2L</td>
<td>70-120 mmol/L</td>
</tr>
<tr>
<td>Small bowel</td>
<td>1L</td>
<td>30 mmol/L</td>
</tr>
<tr>
<td>Bile</td>
<td>1L</td>
<td>40-60 mmol/L</td>
</tr>
<tr>
<td>Colon</td>
<td>600 ml</td>
<td>&gt;200/d</td>
</tr>
</tbody>
</table>

- Colon reabsorbs 100 ml of fluid (10-25% of capacity)
- Short gut:
  - 200 cm of JI segment
  - Without functional colon remnant SB < 100 cm
  - With functional colon, remnant SB < 60 cm
- Dependent on TPN, severe diarrhea and bicarbonate losses
D LACTIC ACIDOSIS

• Rare disorder first described in short gut syndrome by Oh et al in 1979

• Maybe more common than believed

• Defined as metabolic acidosis with D-lactate ≥ 3 mmol/L

Electrolyte & Blood Pressure 4:53-56, 2006
PATHOGENESIS

Glucose

Pyruvate dehydrogenase → Acetyl-CoA → CO₂ + H₂O

D-2-hydroxy acid dehydrogenase

D-Lactate

Overgrowth of acid resistant Gram-positive anerobes (such as Lactobacillus, the major D-lactate producer)

High carbohydrate loading

Glucose fermentation in the colon

Organic acids production

Short or bypass small intestine

Kidney International (2010) 77, 261–262
METABOLISM

• Metabolized to Pyruvate by d-alpha-hydroxy acid dehydrogenase

• Mitochondrial transporters: D lactate/H symporter, D lactate/oxocacid antiporter, D lactate/malate antiporter

• Renal tubular absorption decreases > 30% when levels > 3mmol/L

• Transported to tissues via proton dependent MCT

Electrolyte & Blood Pressure 4:53-56, 2006
CLINICAL PRESENTATION

- Recurrent episodes of encephalopathy and metabolic acidosis in short gut syndrome
- Episodes last from few hours to several days
- Always accompanied by various neurological manifestations

Electrolyte & Blood Pressure 4:53-56, 2006
DIAGNOSIS AND MANAGEMENT

• Increased AG, normal L lactate

• Clinical setting

• Measured enzymatically using D lactate DH specific assay

• Treatment: Na HCO₃, low CHO diet, antibiotics

Electrolyte & Blood Pressure 4:53-56, 2006
CONCLUSION

• Our patient’s metabolic acidosis mainly normal anion gap acidosis from gut losses

• Adequate replacement needed mainly in TPN, as GI absorption poor

• D lactic acidosis should be considered in patient’s with short gut syndrome
HAPPY 2015
TPN AND METABOLIC ACIDOSIS

• Causes:
  • Hyperchloremic metabolic acidosis from synthetic L amino acids
  • Increased titrable acidity from addition of HCl to decrease pH

• Study by Sugiura et al
  • Done on rabbits, 3 groups TPN –HCl (75 Cl/54 acetate ions)/TPN-AA (35/94) /TPN C (35/54)
  • TPN given for 7 days
  • Serial studies of blood acid-base status, pH, serum electrolyte conc and urinary acid-base status were performed
<table>
<thead>
<tr>
<th></th>
<th>TPN -CI</th>
<th>TPN- AA</th>
<th>TPN-C</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>4.30 +/- 0.01</td>
<td>4.71 +/- 0.01</td>
<td>5.49 +/- 0.02</td>
<td></td>
</tr>
<tr>
<td>TA</td>
<td>69.0 ± 0.5 mmol/L</td>
<td>67.1 ± 0.4 mmol/L</td>
<td>25.6 ± 0.2 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>
RESULTS
RESULTS