Nightmare series Case – The Acidosis Rodeo

38 year-old male patient

Hx: Nausea, vomiting, weakness

Medical Hx: Htn,

Found on Ventilator after intubation: 7.0 ETT, 21cm at teeth

AC-Vol Vt 500, Rate 22, Peep 5

ABG:

pH 6.97
PCO2 - 17
HCO3 - 5
BE -23

ABG dissected

If we use the calculated PCO2 for total or max compensation formula, we come out at a PCO2 of 15.5

How?

(1.5 x HCO3) + 8
(1.5 x 5) + 8 = 15.5

That means that this patient is almost maxed out on respiratory compensation with that low HCO3 and PCO2.

PO2 – 102

NA 126
CL 80

Corrected NA?

Hyperglycemia causes osmotic shifts of water from the intracellular to the extracellular space, causing a relative dilutional hypernatremia.

Na + [(glucose level - 100) x 0.016]
126 +[(1034 – 100) x 0.016]
126 + [(934) x 0.016] = 14.944
126 + 14.944 = 140.9

K+ 8

Does the patient have too much k+ when acidotic?
Does this affect the heart?
DKA patients can be very acidotic. But if we fix this acidosis quickly we drop the K+.. We cannot fix a low K+ fast enough. Never treat high or low K+ in an alkalosis or acidosis without first correcting metabolic problem. It's falsely high or low.

Is it really high…Remember, it's not an excess amount, its that all of the intracellular K+ is now in the wrong spot and in the blood stream. The heart doesn't like this.

Treating increased K+ [A true High]
CaCl (raises the action potential threshold)
NaHCO3 (raises the pH, electrical gradient)
D50 – needs a transport molecule to cross over into the cell.. Cause a K+ shift with that movement (Every 1 amp of D50 = 5-10 units of insulin
Insulin – moves the Dextrose molecule across membrane, thus causing the K+ shift back into the cell
Albuterol – Causes a beta stimulus that has a K+ attached to it and pushes K+ back into the cell.

BUN 39
Cr 2
Acute Renal Failure

WBC 27,000

Lactate – Stress response

Procalitonin - High levels indicate a high probability of sepsis, that is, a higher likelihood of a bacterial cause for the symptoms. They also suggest a higher risk of progression to severe sepsis and then to septic shock.

0.15-2 ng/mL – Mild to moderate bacterial infection, inflammatory response
(>2 ng/mL) – Bacterial Sepsis

Hgb 11
HCT 43

Glucose 1034 mg/dL

Insulin Drip @ 15 units/kg/hr

Anion gap -49

[NA-(CL + HCO3)] + K

[126-(80+3)] + 8

[126- 83] + 8

43 + 8 = 51

How can we recognize Metabolic Acidosis without ABG's??

1. (24) – HCO3 (CO_2) on Chem 9
    a. 24 – 3 = 21
    b. Base Deficit = 21

PCO_2 vs. $EtCO_2$

Carbonic acid buffer system is the primary form of regulation and the fasted to be overwhelmed. –Second to second.
Respiratory system is minute to minute
Kidneys are days. HCO3

PaCO2 Vs. Etco2
Equal or +/- 3-5mmHg in normal healthy person

Four vital components to maintain normal relationship

1. Co2 production must occur the tissue level and diffuse to the plasma
2. Cardiac output must be high enough to carry the blood to the lungs from the tissue
3. Co2 must diffuse from the blood to the alveoli
4. Vt and alveolar MV must move the co2 to the EtCO2 sensor inlet.

3P's of EtCO2

1. Pulse
2. Perfusion
3. pH

How do we manage our patient?

What's the right answer?

Attempt to identify the Acidosis?
Immediate EtCO2 placement
Rule out the 3P's

Attempt to maintain moderate VE.
What was their EtCO2 prior to intubation?
If in doubt, set VE high

Glucose

What limits do you have to identify CBG?
CBG monitors stop reading at 600

Why do you prevent drop of > 100 per hour?
Causes cerebral edema
Recommendation is to turn off insulin drip
ICU can worry about Glucose drop in controlled setting.