Metabolic Acidosis & it’s connection with other diseases

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Case Report

Keywords: ENS: Enteric Nervous System, CNS: Central Nervous System, GERD: Gastroesophageal reflux disease, IBS: Irritable bowel syndrome

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Abstract

After long observation and analysis, a connection between metabolic acidosis and migraine have been found in a female patient. She is suffering from headache from childhood. As, headache is not the primary cause it was difficult to treat. Several medicines worked when patient takes the medicine regularly. Headache is back when the drug is stopped. As the root cause was not found and not treated, there are several other problems started happening like muscle pain, weak muscle, numbness etc. Long-term metabolic acidosis has effect on the Gastroesophageal & Gastrointestinal motility. For the patient, it was observed that “Metabolic Acidosis” is the indicator. Acidosis takes place first.

There can be many reasons for metabolic acidosis, but with consistent disbalance of pH level in body it damages many areas of body. And there can be several diseases added to the list. Most importantly it interrupts the connection between CNS (Central nervous system) and ENS (Enteric nervous system). Due to this disconnection the signals coming from brain cannot reach the GI tract. Very few of the signals from (instruction to contract and relaxation of the GI tract) can reach. The fact is “Less signals, less motility” and the outcome is “lazy gut. There can exist false signals, wrong interpretations of the signals.

Background

For a female migraine patient, it has been observed that, headaches trigger after meals. We tried to maintain a history of food taken and were able to establish the below foods contributing to headache trigger,

List - 1

1. Food that is causing allergic reaction like dairy (e.g., Milk and milk derivatives)
2. Soy & soy products.
3. Coconut, peanut & nut products.
4. Meat.
5. Food that is having flavor – Umami taste (e.g., Asafetida, Garlic, Fenugreek)
6. Caffeine & caffeine derivatives.
7. Seafood.
8. Choco and chocolate product.
9. Fermented food (Alcohol & beverages)
10. Sesame seeds.
11. Wheat.

Case Presentation

When treated with antihistamines, the patient showed improvement and less frequent episodes of headache. H₁ blocker & H₂ blockers are helpful but they cannot stop headaches. The use of calcium channel blockers was helpful. SSRI & SNRI drugs worked partially. But to fix the problem at root, gaining
balance in the body’s pH level is the most important task to do. Proton Pump Inhibitor category drugs were effective. Gaining back body’s bicarbonate level is helpful.

**Conclusion**

Metabolic acidosis is the start and root cause of the several other problems like Migraine, Fibromyalgia, IBS, and muscle problems. Most of the problems are neurological, altered neurotransmitter release, disconnection between CNS & ENS. Body not being able to interpret efferent signals. There can be misinterpretation and false afferent signals.

The problem of “Metabolic Acidosis” can be hereditary.

**Case**

**Beginning of the problem**

The migraine trigger food list (List-1) is long. And if we carefully take a look at the list, we can see very little options are left for eating and fulfilling body’s nutrition requirement. Quality of life becomes worse.

In the list most of the items are in “Food Intolerance” group. Few of them are in the “Food Allergy” group. Food intolerance signifies limited enzyme level. So, GI tract is the place where the problem is starting and then there is alteration of metabolism.

Metabolic acidosis is when the body produces too much of acid and body’s pH level gets disbalanced. After long observation it was found that the patient is showing below symptoms along with the primary complaint of headache,

1. Gas, acid, bloating, nausea.
2. Muscle pain, stiffness in back neck.
3. Weak muscle.
4. Shortness of breath.
5. Fast heart rate.
6. Constipation.
7. Low appetite.
8. Mood swing.
9. Anxiety & Depression.
10. Food craving.
11. Shivering, tremor sensation.

**Discussion**

**Problem explanation & connection with acidosis:**
Shortness of breath or Dyspnea, Fast heart rate

Carbon dioxide (CO$_2$) is a gas that is produced as a normal byproduct of our body’s energy production. As per the normal body’s process, CO$_2$ diffuses into bloodstream so that it can be exhaled from lungs. When CO$_2$ level become elevated, the signal reaches brain. Brain, then sends signal to the respiratory system to control CO$_2$ level, to decrease the level. Lung starts working heavily, it tries to exhale elevated CO$_2$. So, a deep and faster breathing continues until the oxygen & carbon dioxide levels are balanced again. When the signal reaches brain that CO$_2$ level is normal then the respiratory rate becomes normal again. If in the process of metabolism, CO$_2$ production is more due to metabolic disorder then dyspnea is how the body tries to balance it. There is increase in heart rate too.

By the below equation we can see the process of glucose converting into CO$_2$, H$_2$O and energy,

$$C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O + \text{energy}$$

When the CO$_2$ level increases, it gets diffused in red blood cells and the reaction is,

$$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow HCO_3^- + H^+$$

When H$^+$ is increased, blood becomes acidic. The pH level decreases. To make it normal again, H$^+$ level must come down or increased HCO$_3^-$ level is required. When body’s bicarbonate buffer is low, decreasing H$^+$ level is the only solution otherwise we may provide HCO$_3^-$ from external source. Respiratory chemoreceptors identify the CO$_2$ level of blood and create signals (with the help of CNS) to take care of it.

Headache & it’s connection with acidosis: (Migraine?)

It has been observed in the patient, from the pattern of her headache episodes, that the reason of the pain is due to excessive release of neuroexcitatory amino acids.

Histamine induced headache- For the patient, antihistamine drugs showed useful.

Glutamine induced headache-The umami taste is detected in mouth through the umami taste buds and the signal is sent to brain. These receptors are found in GI tract. Glutamate can bind with Umami receptors. This may trigger headache. Calcium channel blockers can prevent this condition.

Blood-Brain-Barrier is what protects the CNS from entering unwanted particles/substances.

From study, it has been established that carbon dioxide and lactic acid are discharged into the circulating cerebrospinal fluid. Thus, affecting the pH level of CNS. This triggers excitatory neurotransmitter release.

Mood, sleep, appetite, IBS & their connection with acidosis: (Lazy gut?)
Another important neurotransmitter to talk about is serotonin. 90% of it is produced in gut. It controls mood, sleep, appetite. It has role in digestion of food and gut motility. Undigested food in gut may create inflammation and prolonged inflammation may cause nerve cell deaths. This can limit the signals to the brain. This affects the bowel movement. Less signals from brain makes the gut lazy. On top of this, for the patient, as calcium channel blockers are taken for headache, that makes the gut lazier.

Central nervous system (CNS) & Enteric nervous system (ENS)

CNS & ENS always work in a synchronized manner. But for any nerve damage in gastrointestinal tract, this connection between CNS and ENS is lost or damaged, resulting in poor GI function including motility of gut.

**Muscle weakness/pain & its connection with acidosis:** (Fibromyalgia?)

Muscles contract when they receive signals from motor neurons. Due to damage in the nerve, there is less signals coming from motor neuron. Increased H⁺ level triggers muscle pain.

**Tremor & it’s connection with acidosis:**

The patient occasionally faces the episodes of tremor or shivering of whole body.

**Papilledema & it’s connection with acidosis:**

Papilledema is also seen in the patient during the episodes of headache.

**Abbreviations**

END
Enteric Nervous System
CNS
Central Nervous System
GERD
Gastroesophageal reflux disease
IBS
Irritable bowel syndrome

**Declarations**

Ethics approval and consent to participate: Not applicable

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal
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DP: Contributed to writing the case and discussion,

SR: Contributed to writing the case and discussion,

JF: Contributed to writing the case and discussion, all authors have read and approved the final manuscript

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Figures
Figure 1

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