Case Presentation

A 15-year-old previously healthy female reportedly ingested 65 g of aspirin in a self-harm attempt. Within two hours, she experienced nausea, vomiting, abdominal pain and decreased hearing.

Physical Exam:
- Vitals: 36.1 118 20 137/93 98%
- Head: Normocephalic, atraumatic
- Eyes: PERRL, EOMI
- Nose/Mouth: MMM, clear oropharynx
- Neck: Supple
- Heart: Tachycardia
- lungs: Tachypnea, CTABL
- Abdomen: S/NT/ND, actively vomiting
- Extremities: Warm, well-perfused
- Back: No CVA tenderness

Initial Laboratory Studies:
- CMP: Glucose = trace
- UA: Protein = 2+
- VBG: 
  - pH = 7.5
- Acetaminophen: <5
- Salicylate: 72
- Urine pH: 5
- Potassium Chloride: 40 mEq in Bicarb gtt
- Sodium Bicarbonate: 143
- Potassium Chloride: 40 mEq PO

Management:
- Based upon history and presentation, patient was started on a bicarbonate infusion.
- Once elevated salicylate level returned, bicarbonate therapy continued.
- Patient was transferred to PICU.
- Over next 3 days developed acute renal injury.

Results
- Creatinine peaked at a level of 1.21 mg/dL.
- Her urinalysis was notable for elevated protein at 100 mg/dL, glucose greater than 500 mg/dL despite normal serum glucose concentrations, rising urinary pH and the presence of red blood cells.

Discussion

There is limited data available on the role of salicylate intoxication as a cause of proximal tubular dysfunction in humans and it is not previously described in the toxicology literature.

Proposed mechanism involves the covalent bonding of salicylate or its metabolites to the mitochondria of the proximal tubular cells, altering mitochondrial function and causing energy-dependent dysfunction of the active transporters.

In this case, there was no alternative explanation for the etiology of this transient Fanconi syndrome other than the salicylate exposure.

Conclusion

We present a case of transient Fanconi syndrome following a significant aspirin overdose. Further studies may provide better understanding regarding the frequency or risk factors for its development following salicylate overdose.

Background

Fanconi syndrome is a generalized transport defect within the proximal renal tubules leading to inappropriate urinary losses of glucose, amino acids, bicarbonates, uric acid, phosphorus, potassium, magnesium, calcium and other organic compounds which may lead to excessive wasting. This syndrome may be inherited and be due to inborn errors of metabolism. It may be acquired following the development of a hematologic malignancy or it may occur following an exposure to certain xenobiotics.

Hypothesis

We hypothesized that salicylates may lead to Fanconi syndrome.

Methods

This is a single patient chart review.