**Background**

Fanconi syndrome is a generalized transport defect within the proximal renal tubules which leads to inappropriate urinary losses of glucose, amino acids, bicarbonate, uric acid, phosphate, potassium and other organic compounds.

It may be inherited or acquired following exposure to certain xenobiotics.

The medical literature has a few case reports of aspirin (ASA) intoxication leading to its development.

**Research Question**

In cases of ASA toxicity, what proportion of patients develop laboratory findings consistent with Fanconi syndrome?

**Methods**

This is a retrospective review at a tertiary care hospital in an urban setting.

All cases from 2001-2011 with aspirin concentrations >30 mg/dL were reviewed for:

- Proximal renal dysfunctions as evidenced by a change in creatinine of 20% between admission and peak and/or peak and discharge.
- Glucosuria with a serum glucose less than 180 mg/dL.
- Proteinuria

**Results**

One hundred and three patients in 108 independent encounters had ASA levels >30 mg/dL and were analyzed for elevations in creatinine, proteinuria and glucosuria.

Nine cases were identified to meet the study criteria. Complete data was found for 55 cases, thus, 16.4% demonstrated evidence of Fanconi Syndrome.

The average age was 25.8 ± 9.9. Women accounted for 66.7% of all identified cases. Mean ASA concentration was 59.8 ± 20.4 mg/dL. The mean maximum serum glucose was 142.6 ± 30.1 mg/dL, while the the mean maximum urinary glucose was 237.5 ± 174.7 mg/dL. Mean proteinuria was 128.6 ± 75.6 mg/dL, while mean pH was 7.4 ± 1.1.

**Discussion**

A proposed mechanism for aspirin induced Fanconi syndrome involves covalent bonding of salicylate and/or its metabolites to the mitochondria of the proximal tubular cells.

The resultant change to the mitochondria alters its function leading to energy-dependent failure of the active transporters within the tubular cells.

Although, the study was limited by its retrospective design, restriction to a single center, exclusion of cases missing data and a small number of events fitting the definition of Fanconi syndrome, the findings consistent with Fanconi syndrome in patients following ASA overdoses suggests its role in the development of renal tubular dysfunction.

**Conclusion**

Fanconi syndrome was found in 8.7% of all cases, but in 16.4% (9/55) of cases with complete data available.

Further studies in a larger scale may provide better understanding regarding the frequency or risk factors for the development of this syndrome following ASA overdose.

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<th>Sex</th>
<th>Initial Cr</th>
<th>Max Cr</th>
<th>D/C Cr</th>
<th>Max Serum glucose</th>
<th>Max UA glucose</th>
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Aspirin associated Fanconi Syndrome: Is it an occult phenomenon?

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