Aluminum Toxicity from Oral Sucralfate Therapy

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Dear Sir,

Aluminum (Al) toxicity in dialysis patients has been associated with the use of Al-containing antacids and Al-contaminated water used in hemodialysis [1]. With attention to these issues, Al toxicity in this population is less of a problem now than it was a few years ago.

Aluminum is present in other medications however, and the use of these medications may also result in Al absorption [2, 3] and Al intoxication [4, 5]. Sucralfate is the Al salt of sucrose sulfate and a daily therapeutic dose contains 828 mg elemental Al. Herein is a report of a patient who developed clinical and biochemical evidence of Al toxicity while receiving sucralfate therapy. A 67-year-old woman with chronic renal failure secondary to biopsy-proven focal sclerosing glomerulopathy developed renal failure in 1985 and began chronic ambulatory peritoneal dialysis. Due to peritonitis she was converted to hemodialysis later that year. From November 1985 to March 1986 she wintered in the southwestern United States and was given Al hydroxide 15 ml TID as a phosphate binder. This was discontinued on her return home and magaldrate was substituted.

In September 1986, she was investigated for epigastric pain and found to have significant esophagitis and a duodenal ulcer and placed on sucralfate 1 gm QID (by the gastroenterologist) in addition to her ongoing magaldrate. On a return visit to the nephrologist's office in March 1987, the sucralfate was stopped. At that time she complained of myalgias, bone pain in her shoulders and back. She was noted to have postural hypotension (120/70 to 106/50 mm Hg) with no compensatory increase in heart rate. Also, her hemoglobin had been 104 g/l presucralfate therapy and was now 88 g/l despite resolution of the gastrointestinal problems.

Review of serum Al levels (done by nameless atomic absorption technique) revealed that the serum Al level had been 1,556 nmol/l

(September 29) prior to sucralfate therapy, rising to 1,955 nmol/l and 2,919 nmol/l 14 and 24 days postcommencement of therapy, respectively. Serum Al level was 4,434 nmol/l in February 1987, and deferoxamine therapy was given March 1987 to July 1987. This resulted in a lowering of serum Al levels to 3,355 nmol/l in April 1987 and 1,916 nmol/l in July 1987.

She continued to have episodes of confusion and weakness particularly postdialysis, and she became permanently institutionalized.

Aluminum toxicity is a devastating complication in dialysis patients. Although nephrologists tend to avoid or limit the use of Al-containing antacids in these patients, other medications may deliver substantial quantities of absorbable Al [2]. Sucralfate has been recommended for the treatment of acid-peptic disorder, but the significance of the high content of Al despite the low percentage absorption has not been appreciated.

Aluminum is absorbed from medications. Uremia may allow an increase in gastrointestinal absorption of Al [6, 7]. Sucralfate, despite being advertised as 'nonsystemic' does result in Al absorption, with levels that are comparable to those following equivalent doses of Al hydroxide [8]. Prolonged courses should be used with caution or avoided in patients with significant renal impairment or renal failure in order to limit the possibility for Al toxicity.

References

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524

Burgess

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Erratum