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Ask the expert*

What treatment would you advise for a 1-year-old child with chronic renal failure who has been on aluminum hydroxide as a phosphate binder for 6 months and then develops epilepsy and is found to have a grossly raised plasma aluminum concentration?

Key word: Aluminium encephalopathy

The immediate approach advises discontinuation of aluminum hydroxide as a phosphate-binding agent and other pharmacological agents containing aluminum (Al), such as Sicralfate or Kaopectate. On a statistical basis, this young child is likely to have obstructive uropathy with renal dysplasia and renal bicarbonate wasting, and probably should be treated with Shol's solution. The most likely diagnosis in this child is that gastrointestinal absorption of Al has resulted in encephalopathy and probably, as well in microcytic anemia and vitamin D-resistant osteomalacia [1]. Severe Al intoxication may develop when Al-containing binders and buffered citrate solutions are ingested together, since citrate markedly enhances the intestinal absorption [2].

In some children who have sufficient residual renal function that permits Al excretion, intoxication can resolve after administration of Al-containing binders is stopped. As described, this child already has convulsions, which presumably are not due to uremia or another metabolic disorder, but rather to Al-induced encephalopathy. Thus, therapy with deferoxamine (DFO) is indicated.

DFO is effective in treating Al intoxication in non-dialyzed patients with chronic renal failure (CRF) and in patients undergoing all forms of dialysis. Because Al is tightly bound to proteins, essentially none is removed by dialysis in the absence of a chelating agent such as DFO. DFO forms a freely diffusable chelate which can be given by the intravenous, intramuscular, or intraperitoneal route. Long-term therapy with DFO should be reserved for those patients with well-documented evidence of Al toxicity, including bone disease and neurological involvement [3, 4]. DFO can cause lens abnormalities, leukopenia, cardiac arrhythmias and allergic reactions. It recently has been associated with fatal infections involving *Yersinia* and *Rhizopus* (a fungus, [5]). Thus, DFO should be administered cautiously at a dosage of 10–20 mg/kg once a week; plasma Al levels should be monitored after DFO therapy is begun. Plasma Al levels should not exceed 800–1000 µg/l, because greater values may exacerbate the encephalopathy. The duration of DFO therapy is difficult to estimate. However, because of its toxicity, DFO should be discontinued as soon as maximal clinical improvement is noted.

Al-induced encephalopathy, osteomalacia and microcytic anemia are iatrogenic diseases; hence, they are preventable. Because the main cause of Al intoxication in a child with CRF is the gastrointestinal absorption of Al, Al-containing antacids should not be given. Infant diet can be regulated adequately by using a low phosphate formula with the addition of calcium carbonate as needed [6]. If small amounts of Al are needed to control serum phosphate concentrations, calcium citrate should be avoided as this enhances the intestinal absorption of Al.

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* The editors invite questions for this section