Aluminium in Plasma

Introduction

Because of the ubiquitous distribution of aluminium compounds, natural human exposure is unavoidable, and moderate amounts of the element enter the body constantly through inhalation of atmospheric dusts and ingestion of food and drink. Despite an oral intake ranging from 5 to 10 mg daily little Aluminium is absorbed and serum levels of 0.07–0.30 µmol/L are usually found. Tissue Aluminium levels are very low. No biological function for the metal has been found. With normal renal function aluminium is readily excreted in the urine.

Moreover, life has evolved in an environment so rich in aluminium that it would be surprising if human beings could not tolerate substantial variations in exposure without ill effects. Under most circumstances this tolerance appears to hold. Industrial aluminium toxicity is rare and tissue concentrations of the metal have apparently been little affected by extensive use of aluminium products and cooking ware. The application of thousands of kilograms of aluminium products as antiperspirants has not caused toxicity except for occasional local irritation. Indeed, a considerable body of experimental data gathered over many years suggests the presence of formidable epithelial barriers to aluminium absorption in the lung, the gastrointestinal tract and the skin.

However, high levels of aluminium can accumulate in the tissues of patients with chronic renal failure on longer term haemodialysis treatment. This increased tissue load of aluminium may be derived from the following sources:

- Intestinal absorption may follow the administration of aluminium hydroxide gels used to control the high plasma phosphate levels found in chronic renal failure (may also be used as an antacid). In the past patients have ingested up to several kilograms of elemental aluminium over their dialysis ‘career’
- Water used for haemodialysis may contain aluminium which will dialyse across the dialysis membrane and lead to raised serum aluminium and tissue aluminium levels. A single dialysis may expose the patient’s blood to as much as 250 L of water (39 000 L per year)
- The dialysis concentrate used to prepare the dialysate may contain high levels of aluminium and lead to substantial contamination of the dialysate fluid

Increased tissue content of aluminium appears to be the major factor in the aetiology of dialysis dementia and dialysis osteodystrophy. The prevention of iatrogenic aluminium poisoning involves caution in the use of aluminium containing oral phosphate binders, together with regular monitoring of (i) the aluminium content of the dialysate, (ii) the domestic tap water used to prepare the dialysate, and (iii) serum aluminium levels in patients on long term haemodialysis treatment.

Sample requirements

7 mL of blood is collected into 7 mL Navy K2 EDTA Plus BD Trace Metal tube. After blood collection, the plasma must be separated from the red cells within 4 hours of collection using ‘aluminium free’ transfer pipettes and serology tubes. Specimens are stored at 4°C and analysed weekly.

Interpretation

Normal reference range 0.07 – 0.30 µmol/L

Raised levels are found in renal failure, particularly in patients on haemodialysis where aluminium hydroxide is administered as a phosphate binder to control plasma phosphate levels. In these patients serum aluminium greater than 7.4 µmol/L generally leads to clinical symptoms of aluminium toxicity. Levels greater than 3.7 µmol/L are of clinical concern and close surveillance is required, while levels greater than 2.2 µmol/L need attention.

Keywords: Trace Element:

Information last updated on 17 March 2017