



5. Magnesium

Draft CARI Guidelines

- a. In dialysis patients, serum magnesium should be kept within the physiological range of 0.70-1.05 mmol/L at all times. (Level B evidence)
- b. Dialysate magnesium concentrations between 0.25 mmol/L and 0.50 mmol/L will maintain normomagnesaemia in the majority of both haemodialysis and CAPD patients, but levels in some case may have to be individualised. (Level B evidence)
- c. Serum magnesium should be monitored regularly, particularly if magnesium oral phosphate binders are used. With haemodialysis this needs to be both pre-and post-dialysis. (Level B evidence)

Practice tips

- Measure serum magnesium monthly in patients who are taking magnesium salts or using dialysate with a magnesium concentration out of the range 0.25-0.50 mmol/L.
- Measure magnesium in other dialysis patients 3-6-monthly.

What is the evidence?

- Free ionised magnesium comprises 55% of total serum magnesium. Serum levels are remarkably constant, unaffected by age, gender or race. Physiological range for total serum magnesium is 0.70-1.05 mmol/L with a mean of 0.85 mmol/L (Johansson 1979, Stendig-Lindberg et al 1985). The kidney is the major regulator of plasma magnesium (Dirks 1983) and accumulation may occur with progressive renal failure leading to hypermagnesaemia (Contiguglia et al 1972, Mordes and Wacker 1977). Once dialysis is begun, the major determinant of magnesium balance is the dialysate magnesium concentration (Mountokalakis 1990).
- Hypermagnesaemia may cause vomiting, lethargy, muscle weakness and skin burning (Freeman et al 1967, Mordes and Wacker 1977), hypotension and cardiac arrhythmias (Dycknrer and Wester 1982). Hypomagnesaemia is associated with a variety of neuromuscular disturbances including cramps, myoclonic jerks, paraesthesia, dyskinesia and even tetany (Dycknrer and Wester 1982, Kingston et al 1986). Serum magnesium influences PTH secretion. Low levels of magnesium in dialysis solutions decrease PTH (Pletka et al 1974) but may adversely affect crystal maturation in bone (Alfrey and Ibel 1973).



- Gonella et al (1988) evaluated bone histology over 1 year in a group of hypermagnesaemic haemodialysis patients before and after reducing dialysate magnesium from 0.5 to 0.25 mmol/L. Serum magnesium levels fell into the normal range with the lower concentration. A significant reduction in bone osteomalacic pattern with no change in resorption pattern was seen.
- Chronic magnesium depletion occurs with very low magnesium dialysate levels resulting in increased serum levels of PTH but impaired sensitivity of bone to PTH (Parsons et al 1980). In a longitudinal study of CAPD patients, lower serum magnesium levels have been associated with increased vascular calcification (Meema et al 1987).
- Serum ionised magnesium levels (the biological active fraction) were studied in 26 haemodialysis (dialysate magnesium 0.375 mmol/L) and 10 CAPD (dialysate magnesium 0.25mmol/L) patients and compared to 66 matched control subjects by Markell et al (1993). Both haemodialysis (0.55 ± 0.2 mmol/L) and CAPD (0.50 ± 0.02 mmol/L) had significantly lower ionised magnesium levels than the controls (0.60 ± 0.004 mmol/L). Ionised magnesium levels showed a strong correlation with total serum magnesium in both haemodialysis ($r = 0.93$) and peritoneal dialysis ($r = 0.92$)
- The use of magnesium-containing oral phosphate binding agents may result in hypermagnesaemia. However, Delmez et al (1982) in a randomised cross-over study of haemodialysis patients showed that magnesium carbonate ($MgCO_3$) together with calcium carbonate ($CaCO_3$) used in conjunction with an 0.3 mmol/L magnesium dialysate compared to $CaCO_3$ alone (double dose) was equally effective in restricting serum phosphate and permitted higher doses of pulse IV calcitriol for PTH suppression without causing hypercalcaemia. Serum magnesium was similar in both phases.
- The use of magnesium-free dialysate together with oral magnesium phosphate binders has in the short term also been shown to maintain normomagnesaemia in both haemodialysis (Breuer et al 1987) and CAPD (Shah et al 1987). However, in the more long term, hypomagnesaemia will develop in the majority of haemodialysis patients and Kenny et al (1987) have shown that normomagnesaemia can be restored with the use of an 0.25 mmol/L dialysate.
- In CAPD, a dialysate magnesium of 0.75mmol/L causes hypermagnesaemia (Kohaut et al 1983, Rahman et al 1987). However, reduction to 0.25 mmol/L maintains normomagnesaemia in most patients (Nolph et al 1983, Hutchison et al 1992).
- There are no published randomised or morbidity/mortality end-point trials comparing the effects of differing dialysate magnesium concentrations in either haemodialysis or CAPD

What do the other guidelines say?

DOQI: No recommendation.

BRA: No recommendation.

CSN: No recommendation.



Implementation and audit

Data on pre- and post-haemodialysis plus CAPD patient serum magnesium levels should be collected by ANZDATA now and repeated 1 year after promulgation of the above guidelines – together with a survey of dialysate magnesium concentrations. Further cross-sectional data could be obtained if questions on PTH levels or symptoms such as cramps were included.