ALUMINIUM, EEG AND INTELLECTUAL FUNCTION IN PATIENTS ON REGULAR DIALYSIS TREATMENT (RDT)

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The aim of the present study was to investigate the correlation between serum aluminium level and neurological abnormalities in patients on RDT taking aluminium hydroxide.

Patients and Methods

Aluminium concentrations were determined by flameless atomic absorption [1] in: (a) tap water; (b) deionised water; (c) dialysates prepared by single automatic units; (d) serum of 44 subjects with normal renal function without Al(OH)₃, (e) serum of 69 patients on RDT for 19.43 ± 20.26 months taking Al(OH)₃, 2–5g/daily.

Neurological investigation was carried out 18–30 hours after the last treatment in two groups of patients on RDT taking Al(OH)₃. The first group comprised 11 patients treated for 20.55 ± 19.10 months with normal serum aluminium levels (16.90 ± 5.09μg/L), the second group 11 patients treated for 39.64 ± 27.24 months with increased serum aluminium levels (75.36 ± 31.01μg/L). The programme of investigation was: EEG, detailed neurological examination, Wechsler Adult Intelligence Scale (WAIS), Wechsler Memory Scale (WMS), Corsi’s block-tapping test, verbal fluency, reading fluency, modified Purdue Pegboard Test (PPT).

Results

1. Aluminium content was very low (<3μg/L) in water treated by demineralising and in dialysates prepared by single automatic units.
2. Serum aluminium was higher (Kendall’s S test: P<0.01) in patients on RDT (31.07 ± 24.61μg/L) than in control subjects (15.00 ± 12.60μg/L).
3. Serum aluminium was higher (Kendall's S test: P=0.06) in uraemic patients treated for more than 6 months (38.48 ± 27.37μg/L) than in patients treated for a shorter time (18.04 ± 9.80μg/L), but there was no statistical correlation between serum aluminium levels and time of dialysis and/or administration of Al(OH)₃.
4. EEGs were normal in 7 patients with increased serum aluminium levels and in 4 patients with normal serum aluminium levels, whereas the remaining patients' EEGs showed minor non-specific changes (diffuse slowing and sporadic paroxysmal abnormalities) in the absence of clinical signs.

5. Neurological examination showed clinical features of peripheral neuropathy, which affected predominantly the distal segments of the lower limbs, in 5 patients; of these, 3 had increased serum aluminium levels and 2 had normal levels.

6. Mental status assessment did not differ significantly between the two groups.

Conclusions

1. Our results confirm the intestinal absorption of Al from aluminium-containing antacids, but there is no correlation between serum aluminium and time of Al(OH)₃ ingestion.

2. There are no early neurological abnormalities related to high serum aluminium levels, at least up to levels which we found in our patients (max 150µg/L).

3. The development of neurological abnormalities into dialysis dementia seems closely related to high aluminium content of dialysate.

Reference