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Correspondence

CKJ

Encephalopathy in a dialysis patient treated with lanthanum carbonate (LC)

Sir,

Dr Fraile *et al.* [1] report the interesting case of a dialysis patient who was treated with lanthanum carbonate (LC; 2000 mg daily for 8 months increased to 3500 mg daily for the subsequent 10 months) and developed acute confusional syndrome. Conduct disorders and delirious ideas were also observed. The neurological examination was normal and the electroencephalogram tracing, as well as brain Computed Tomography scan, was also referred as normal. After removing LC from therapy, the patient improved in 72 h.

Interestingly, Dr Fraile assessed LC concentration in cerebrospinal fluid (CSF) and in serum and the levels were very high (33.85 and 49 μg/L, respectively). The LC serum and CSF levels were measured also 10 weeks after the admittance and were found surprisingly undetectable. Of interest, the CSF protein levels that was >100 mg/dL in two occasions.

The authors speculate on the fact that high-serum levels of free LC could be a consequence of reduced serum protein (serum albumin was 2.3 g/dL at admittance) and that blood–brain barrier (BBB) could be also altered by LC itself or by pre-existing pathological conditions.

In their final conclusions, the AA are rising the hypothesis that LC might potentially damage and cross and the BBB thus increasing the risk of neurological side effects in dialysis patients.

The authors also claim that no brain deposit of LC or cognitive disorders have been described so far in patients treated with LC, forgetting, however, the important contribution by Altmann *et al.* [2] who showed—in a prospective randomized multicenter study—that cognitive function deteriorates in haemodialysis patients over a 2-year time period and that LC, as a phosphate binder, does not adversely affect cognitive function compared with standard therapy. This study confirms previous studies performed in animals, exposed to large doses of LC, in which it has been shown that Lanthanum ions cannot pass between the tight junctions of the BBB [3]. Moreover, additional contributions confirmed the safety of LC therapy in dialysed patients followed for up to 6 years [4].

In sum, it is not surprising that a deranged BBB could allow a high LC concentration in CSF in this patient. The real surprise is that this patient also had likely a concomitant increased intestinal permeability, combined with a severe liver cirrhosis, a typical condition in which LC should never be administered.

Conflict of interest statement. None declared.

Editorial Note: Dr Fraile *et al*. had been invited to reply to this letter but we did not receive a response.

[1]Renal and Dialysis Unit, Simone Martini, Milano, Italy
[2]Renal Division, Department of Medicine, Surgery and Dentistry, San Paolo Hospital, University of Milan, Milan, Italy

Diego Brancaccio[1]
Emilio Rivolta[1]
Mario Cozzolino[2]

Correspondence and offprint requests to: Diego Brancaccio; E-mail: diego.brancaccio@tiscalinet.it

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