Haemodialysis for hyperammonaemic encephalopathy

Sir,

We recently reported the usefulness of haemodialysis for treating hyperammonaemic coma complicating urinary diversions [1]. In our previous report, the obtained blood-side ammonia clearances during high-efficiency haemodialysis were similar to those found for urea (with respective values of $261.4 \pm 11.4$ vs $262.6 \pm 47.2$ ml/min) and comparable to those reported in vitro by Cordoba et al. [2]. The purpose of the present communication is to confirm the usefulness of haemodialysis for hyperammonaemic encephalopathy and to verify measured clearances.

Case. A 59-year-old female was brought to the emergency room with decreased alertness over a few few hours. She had known congenital uropathy which had required a right nephrectomy and an ureterosigmoidostomy. History was negative except for a deliberate discontinuation of laxatives the week before. Initial biochemistry serum results were as follows: creatinine 89 $\mu$mol/l, urea 10.3 mmol/l, sodium 147 mmol/l, chloride 119 mmol/l, bicarbonates 14.5 mmol/l. Arterial pH was 7.32 with a pCO$_2$ of 28.5 mmHg. A bicarbonate infusion was initiated and netilmicine was empirically given intravenously. Because of neurological deterioration, she was intubated and transferred to the ICU. Serum ammonia concentration drawn upon admission came back markedly elevated at 228 $\mu$mol/l and increased to 379 $\mu$mol/l 5 h later when the patient deteriorated.

A temporary dialysis catheter was inserted, the bicarbonate infusion was stopped and intermittent haemodialysis was initiated using a CT-190G dialyser ($1.9$ m$^2$ triacetate cel lulosic membrane manufactured by Baxter Healthcare Corporation, McGaw Park, IL, USA) at a blood flow rate of 450 ml/min and a dialysate flow rate of 500 ml/min. Blood was drawn pre- and post-dialyser every hour for ammonia determination during haemodialysis which was maintained over 4 h and 50 min. Mean blood-side ammonia, urea, and creatinine clearances were respectively 289.8 $\pm$ 25.5, 352.2 $\pm$ 16.8, and 180.3 $\pm$ 10.8 ml/min; pre-dialyser blood ammonia was reduced to 4 $\mu$mol/l when dialysis was discontinued; however, a rebound was observed with an increased concentration to 41 $\mu$mol/l 2 h after the end of dialysis.

The patient became progressively more alert over the next 12 hours and an abdominal ultrasound confirmed acute left pyelonephritis. A long-term treatment with lactulose and oral sodium bicarbonate was prescribed and she was discharged from the ICU. Two days later, she was readmitted to the ICU because of decreased alertness. Ammonia level was 72 $\mu$mol/l and the treatment consisted in oral ciprofloxacin, sodium benzoate, and lactulose administered more frequently. Two weeks later, she was finally discharged from the hospital. Urological follow-up has resulted in the modification of the type of urinary diversion with the purpose of reducing associated complications [3].

Comment. The present report emphasizes the importance of recognizing hyperammonaemic encephalopathy complicating urinary diversions and, if possible, preventing its occurrence. In case of a severe encephalopathy, haemodialysis can be a useful adjunct offering rapid lowering of blood ammonia concentrations. The evolution of our patients and the obtained intra-dialytic clearances confirm these findings.

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