Pontine Myelinolysis Following Correction of Hyponatraemia

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Summary

A patient with severe hyponatreamia secondary to chronic renal failure was treated with peritoneal dialysis (PD). On the third day of admission, she developed progressive obtundation. Neurological examination showed bilateral brisk reflexes with intact brain stem reflexes. Magnetic resonance imaging demonstrated patchy demyelination of the pontine area indicating central pontine myelinolysis (CPM). Despite supportive measures, the patient died on the fifteenth day of admission. The rate of correction of hyponatraemia with peritoneal dialysis can be rapid and detrimental to hyponatraemic chronic renal failure patients and careful monitoring of serum sodium level is advocated.

Key Words: Hyponatraemia, Chronic renal failure, Peritoneal dialysis, Central pontine myelinolysis

Introduction

Hyponatraemia is the commonest electrolyte abnormality in hospitalized patients. Chronic renal failure as a cause of hyponatraemia in hospital patients accounts for 2% - 12% of all causes[1,2]. There is definite risk of CPM following rapid correction of chronically hyponatraemic patients[1,2]. Correction of hyponatraemia using PD has the added advantage of treatment of uraemia and is considered to be a slow gentle form of correction. This case report however illustrated that treatment of hyponatraemia with PD can lead to rapid correction of hyponatraemia and CPM.

Case Report

A 35-year-old Chinese housewife presented to the nephrology clinic, Kuala Lumpur General Hospital in late January, 1994 with a history of ankle oedema, nausea and vomiting for the last 3 months. She had a history of hypertension for a year and in early January was detected to have proteinuria at the outpatient clinic. An ultrasound in October, 1993 showed both normal sized kidneys of 12 cm but there was increased echogenicity of the renal parenchyma indicating renal parenchymal disease. Renal profile in early January, 1994 showed urea of 17.3 mmol/l, sodium of 133 mmol/l, potassium of 3.7 mmol/l and creatinine of 630 umol/l. Physical examination was unremarkable except for mild ankle oedema for which oral frusemide at doses of 80 mg prn was prescribed.

Five days later, she was admitted to the nephrology ward for abnormal behaviour. She was noted to be quiet, speaking irrelevently and neglecting her children. The renal profile done at the nephrology clinic showed urea of 21.5 mmol/l, sodium of 101 mmol/l, potassium of 3.0 mmol/l and creatinine of 556 umol/l. In view of the severe hyponatraemia occurring in the setting of

chronic renal failure, PD was chosen as a form of gentle correction of the hyponatraemia. Serial monitoring of the serum sodium and creatinine showed rapid correction of sodium level to 126 mmol/l at day 2 and 138 at day 3. Figure 1 shows the serial readings of serum sodium and creatinine.

On day 4, patient developed progressive obtundation and was not responsive to call. Neurological examination showed bilateral brisk reflexes but brain stem responses were intact. Computerized tomographic scan and lumbar puncture were normal. She did not recover despite supportive PD. Magnetic resonance imaging as shown in Figure 2 at day 11 of admission demonstrated 2 foci of hyperdensity at the pontine area at T2 weighted images consistent with a diagnosis of CPM. The patient died at day 15 of admission.

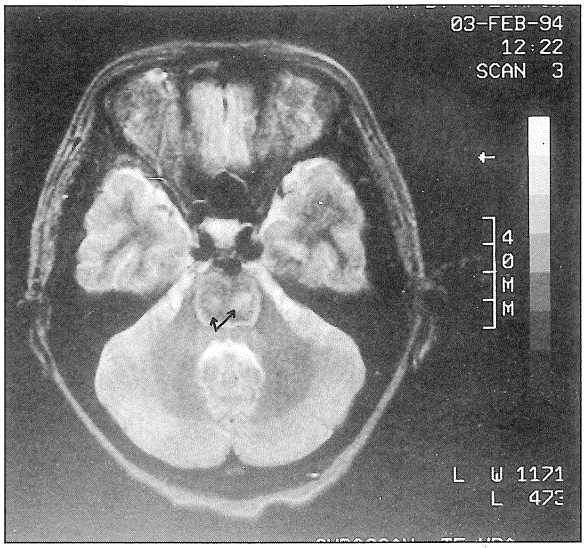

Fig. 2: MRI at T2 weighted density at the level of the brain stem. Arrow indicates the 2 demyelinated opacities characteristic of central pontine myelinolysis

Discussion

CPM is a rare but serious disorder associated with severe hyponatraemia. There is good evidence clinically and experimentally that CPM occurred after rapid correction of hyponatraemia[3]. In a series of severe hyponatraemic patients (serum sodium < 116 mmol/l), CPM occurred in 8% of patients[3]. Animal experiments have shown a considerable risk of neurological injury with a rise in serum sodium of 15 mmol/l in 24 hours or 24 mmol/l in 48 hours. The rate of correction of hyponatraemia has been investigated by Stern et al and found that a rise of 12 mmol/l or more per day carries a danger of CPM[3]. The duration of hyponatraemia is also important in the development of CPM. Acute hyponatraemia of less than 48 hours is less susceptible to the development of CPM. In our patient, the duration of hyponatraemia was at least 5 days and the rate of rise was 25 mmol/l in the first day of treatment and 12 mmol/l in the second day. This rate of correction is definitely a risk factor in the development of CPM.

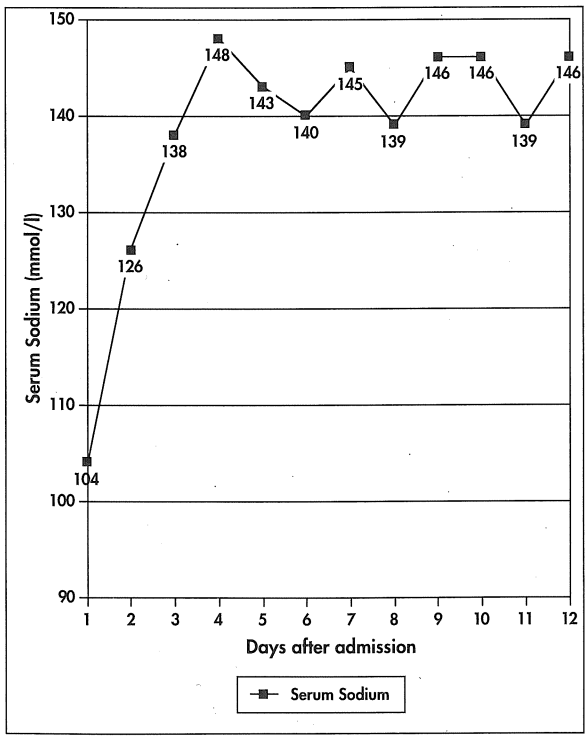

Fig. 1: Serial serum sodium levels of patient

The clinical manifestation of CPM can vary from asymptomatic to characteristic pontine lesion findings of quadriparesis, pseudobulbar palsy and lock-in syndrome. The presence of brain stem reflexes in this patient does not rule out CPM since atypical features may be seen.

The clinical course of CPM is not invariably fatal. Asymptomatic cases and cases with clinical and radiological resolution have been documented. Magnetic resonance imaging is superior to computerized tomography in detecting CPM.

The rate of rise in the correction of hyponatraemia can theoretically be measured by calculating the deficit and correcting it over time by hypertonic saline. However, in practice the corrected sodium level may be more difficult to predict. Hence, there is no substitute for more frequent estimation of serum sodium level in the management of hyponatraemic patients.

Acknowlegdement

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Clonorchiasis/Opisthorchiasis in Malaysians – Case Reports and Review

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Summary

Clonorchiasis and opisthorchiasis are snail-transmitted trematode infections. The disease is endemic in many parts of Asia. Local case reports have been predominantly in Chinese with a history of travel to endemic countries. Thus far, 20 cases of liver fluke infestation have been reported in this country. This report presents another two cases of clonorchiasis and a case of opisthochiasis. We also briefly review pertinent aspects of the disease.

Key Words: Liver fluke infestation, Clonorchiasis, Opisthorchiasis