CHOREA, A MANIFESTATION OF HYPONATRAEMIA?

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SYNOPSIS

Choreiform movements have not been recorded as a manifestation of hyponatraemia. We describe a 57 year old lady with hyponatraemia and chorea, and suggest a causative link between the two.

CASE REPORT

A 57 year old Indian lady was admitted to a district hospital in Kluang, Johore on 26/2/80 for exertional dyspnoea for one week and diarrhoea for 2 days. She had a history of a mental illness about 10 years ago but has since been well. She was not on any medication. She was pale and dyspnoeic at rest. There were multiple cervical nodes described as hard and irregular. Blood pressure was 130/80mmHg. There were bilateral crepitations and rhonchi in the lungs. Investigations showed: Blood urea 9mg%, serum sodium 117.5mEq/1, S. K^+ 3.3mEq/1, $C1^-$ 70.9mEq/1. Chest x-ray showed bilateral opacities, sputum smears were repeatedly negative for tubercle bacilli. Barium enema showed a rigid terminal ileum with irregular margins. The patient suffered transient right sided weakness while in hospital. She refused lymph node biopsy and was transferred to Tan Tock Seng Hospital on 25/3/80, still complaining of diarrhoea. On examination, she was afebrile, but thin and lethargic. She was not dehydrated and blood pressure was 120/80mmHg. There were coarse crepitations in both lung bases, and firm enlarged cervical nodes were palpable. No diarrhoea was observed in the ward. Investigations showed: Blood urea 13mg%, serum potassium 3.9mEq/1, Na 128mEq/1, C1 94mEq/1, blood glucose 73mg%. s. calcium 7.7mg%, s. albumin 2.5g%, serum glutamate pyruvate transaminase 13 units, serum alkaline phosphatase 192 units, serum globulin 4.2g%, serum bilirubin 0.6mg%. Abdominal x-ray showed no adrenal calcifications. A right cervical lymph node biopsy under local anaesthesia was done, and this showed tuberculous

lymphadenitis. On 1/4/80, she developed right hemiplegia with right facial palsy. At lumbar puncture cerebrospinal fluid pressure was 15 cm water, and analysis of the fluid revealed no cells, a trace of globulin, 55mg% protein, 52mg% glucose, 649mg% chlorides and no organisms on smear. The cerebro-spinal fluid was V.D.R.L. negative and aerobic culture negative. Her general condition gradually improved, and power in her right limbs recovered a little. She was discharged from hospital on 11/4/80 with anti-tuberculous drugs. The right sided weakness continued to improve at home.

On 22/5/80, she was admitted to Tan Tock Seng Hospital for uncontrollable involuntary movements of all four limbs for 1 day. She was afebrile, confused but fully conscious and not dehydrated. Blood pressure was 120/80mmHg. and there was no oedema. On attempting any voluntary action her limbs would go into attacks of sudden irregular and jerky semipurposive movements associated with facial grimacing. The left side was affected more but the right side was not spared. There was no apparent loss of power even in the previously hemiplegic side, but an accurate assessment of motor power was difficult. There was, however, a residual facial palsy. Muscle tone was normal. She vomited a few times on her first hospital day. Investigations showed: Haemoglobin 11.2g%, blood urea 11mg%, serum potassium 4.7mEq/1, Na 110mEq/1, C1 68mEq/1, blood sugar 130mg%, arterial blood pH 7.36, pCO_2 34.8mmHg., pO_2 115.9mmHg. Plasma osmolality was 229mosm/kg. and urine osmolality 177mosm/kg.

She was given intravenous normal saline 500 mls. six hourly for one day. The next day, 23/5/80, she stopped vomiting, was able to take orally and the choreiform movements improved dramatically and blood urea was 13mg%, serum potassium 3.7mEq/1, Na 121mEq/1 and C1 80mEq/1. The involuntary movement subsided and finally ceased on 27/5/80. Serum electrolytes on 29/5/80 were potassium 4.1mEq/1, sodium 134mEq/1, chloride 91mEq/1. She was discharged on 7/6/80 and has been well since.

DISCUSSION:

Chorea may be senile, posthemiplegic, postneurosurgical, encephalitic, postkernicteric, Sydenham's, Hungtington's, drug-induced or post-traumatic (4). It may also occur in Wilson's disease, and very rarely in hyperthyroidism (1) and polycythaemia rubra vera (3), but has not been described in association with hyponatraemia.

The negative history in this patient excludes postneurosurgical and post-traumatic chorea. Chorea was observed in some patients during the acute phase of encephalitis lethargica during the epidemic but few if any cases of this encephalitis have been seen since 1930. There was no clinical evidence of hyperthyroidism or polycythaemia rubra vera. The age of the patient and the age of onset and the transient nature of the chorea make it unlikely to be postkernicteric, Sydenham's, Hungtington's, senile or due to Wilson's disease. The drugs usually implicated in iatrogenic chorea are the phenothiazines. Rifampicin, ethambutol and isoniazid which the patient was on have not been documented to cause chorea to the best of our knowledge. Moreover, our patient was continued on these drugs without any ill effects subsequenty.

In the postthemiplegic type, the abnormal movements are confined to the side of the weakness and generally spare the face. They are more hemiballismic than choreiform. (4) In our patient, the chorea was bilateral and involved the face.

The major clinical manifestations of hyponatraemia are neurologic in nature and include lethargy, confusion, delirium, coma, muscular weakness and convulsions (2). A classic description of the symptoms of hyponatraemia are recorded by McCance (6) when he and his volunteers induced hyponatraemia in themselves by a saltfree diet and sweating. The severity of the neurologic disturbance is related not only to the level of serum sodium but also to the rate at which it falls. Whether the hyponatraemic encephalopathy results from brain cell swelling or from intracellular potassium depletion is still in dispute (2). The coincidental onset of choreiform movement with the development of hyponatraemia and the rapid cessation of the movements with the correction of the hyponatraemia is compelling evidence that the two are causally related.

The most likely cause of the hyponatraemia was inappropriate anti-diuretic hormone secretion which was probably due either to the cerebrovascular accident or the pulmonary tuberculosis.

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