Hypokalaemia, Uraemia, and Tetany

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Tetany is commonly due to hypocalcaemia, but may be associated with other biochemical abnormalities, including hypomagnesaemia and hypokalaemia. We describe a woman with a 10-year history of ulcerative colitis who from 1960 to September 1962 had progressive muscular weakness and stiffness associated with frequent and crippling tetanic spasms. The symptoms were thought to be due to hypokalaemia and uraemia and improved when these biochemical abnormalities were corrected.

CASE HISTORY

A woman aged 43 presented in 1957 with a five-year history of loss of weight and intermittent rectal bleeding with diarrhoea. A diagnosis of ulcerative colitis was made, and this was confirmed by barium enema. Treatment was started with sulphaguanidine. In 1958, because of rapid loss of weight, ileostomy was performed with considerable improvement in her general condition, and in April 1960 resection of the colon and rectum was carried out. Two months after the operation tetanic spasms first developed. They were treated with calcium gluconate up to 10 g. daily, given intramuscularly, though the serum calcium was never below normal on five occasions (range 9.1–10.4). She was also given prednisolone, 5 mg. three times a day. From 1960 to 1962 the tetany persisted, and she developed generalized stiffness of all limbs. Finally she was incapacitated and confined to her bed. She had lost a considerable amount of weight and the ileostomy fluid had increased. The blood urea in April 1960 was 70 mg.% and serum potassium 2.4 mEq. By September 1960 the blood urea had risen to 135 mg.% and persisted at this level (140 mg.% in July 1962). During this time there was clinical evidence of urinary-tract infection; this was treated with antibiotics. In September 1962 she was transferred to Freedom Fields Hospital for further investigation.

On examination she was pale, thin, and ill-looking. She had finger-clubbing. There were frequent muscle twitchings and tetanic spasms of the hands and feet, and generalized hypertonus of the limb muscles. The pulse rate was 80/min., regular. Blood-pressure was 145/75 mm. Hg and heart sounds were normal. In the abdomen was a functioning ileostomy.

Investigations showed the following results: Hb 62%, red cells hypochromic. White blood cells 6,000 with lymphopenia. Serum calcium 10 mg.% (repeat 9.2 mg.%), phosphorus 1.9 mg.%, magnesium 3.5 mg.% (2.9 mEq), sodium 137, potassium 3.2, chloride 100, alkali reserve 26.5 mEq/l., protein 7 g.%, blood urea 52 mg.%. Maximum specific gravity of urine 1008. Urea clearance 80% normal. Tube specimen of urine—heavy growth of *Escherichia coli* on three occasions. Intravenous pyelography was normal. Barium meal revealed no evidence of small-bowel disease. E.C.G. was compatible with hypokalaemia.

Hypocalcaemia and hypomagnesaemia being excluded, hypokalaemia and renal insufficiency remained as possible causes for the tetany. These possibilities were treated consecutively.

In addition to dietary potassium she was given potassium in solution and received 100 mEq/daily for 14 days. The average daily potassium output during this period (measured over three consecutive days) was 37.1 mEq (11.8 in urine); 25.3 in ileostomy fluid. She therefore retained at least 63 mEq/day, a total retention of approximately 880 mEq. The serum potassium rose during this period to 6 mEq and the E.C.G. showed evidence of hyperkalaemia. The potassium was therefore temporarily stopped but later resumed with 50 mEq daily. The output of urinary potassium rose to 37.6 mEq per day as the deficit was corrected.

During this period the tetany improved but did not cease. Renal insufficiency as a cause was therefore investigated. The blood urea on admission had been 52 mg.% and rose again to 135 mg.% during

the next month. Possible causes included active pyelonephritis and potassium nephropathy, but the most important appeared to be the high ileostomy loss and low urinary output. Thus over the 16-day period (2 to 18 October 1962) in which the potassium balance studies had been carried out, the average fluid intake had been 1,585 ml./day, with a daily average output of ileostomy fluid of 587 ml. and a daily urine output of 504 ml. The fluid intake was therefore increased, and between 18 October and 10 November it averaged 2,268 ml./24 hr. The urine output during the same period increased to 908 ml./24 hr. Concurrently urinary-tract infection with *E. coli* was treated with ampicillin and erythromycin. During this period the blood urea fell to 28 mg.%. The tetany became less frequent, the muscles less stiff, and finally tetany stopped altogether.

COMMENT

The association of hypokalaemia and tetany has been previously reported on many occasions. Eliel *et al.* (1950) record tetany associated with potassium deficiency in the post-operative period, while the association of tetany with hypokalaemia in primary aldosteronism is well known. Tetany may also be associated with multiple metabolic disorders, as in the case reported by Staffurth and Allott (1962), when simultaneous hypokalaemia and hypocalcaemia were associated with disturbance of calcium and phosphorus and a moderate degree of renal failure.

Of the total body K⁺ (approximately 4,000 mEq in a man weighing 70 kg.) only 150 mEq is in the extracellular compartment and hence directly measured by the serum K⁺ level. The serum level is not then always a good indication of the total deficit of potassium ions. In primary aldosteronism a positive balance may be achieved only with an intake of 100 mEq/day (Prunty, 1958), and a total of several hundred to a thousand mEq potassium may be retained after removal of the aldosterone-producing tumour. In Staffurth and Allott's case the deficit of 675 mEq potassium was present when the serum level was 2.1 mEq/l. Surawicz *et al.* (1957) stressed the need for large doses of potassium to correct the deficit in their 50 cases of hypokalaemia.

In the present case the tetany was partly attributed to deficiency of potassium ions, and this was supported by the improvement when the deficit was corrected. As in the above studies the deficit (880 mEq) was much greater than was supposed from the serum level (3.2 mEq). Renal failure was also thought to be partly responsible and was considered to be mainly extra-renal in origin. This view was supported by urea clearance of 80% and good dye concentration on excretion urography at three minutes, despite a high level of blood urea. After the fluid intake had been considerably increased the urinary output almost doubled and the blood urea fell to normal. Concurrently all muscle weakness, stiffness, and tetany disappeared.

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