

Investigation vignette

A 32 Year old Woman Admitted to the Intensive Care Unit with Hypoxia, Generalised Weakness and Agitation

CASE REPORT

A 32 year old woman was admitted to the intensive care unit from the accident and emergency department with progressive generalised weakness, hypoxia and agitation, and a provisional diagnosis of Guillain-Barré syndrome. She had a past history of abdominal trauma due to a motor vehicle accident 12 months ago which necessitated a small bowel resection. Following that admission she had some difficulty in maintaining her 'fitness weight', and had developed a strict diet in an attempt to reduce the number of her fluid bowel motions.

Her vital signs revealed a pulse rate of 88 beats per minute, respiratory rate of 16 per minute, blood pressure 110/45 and temperature of 36.4°C. She was drowsy but eye opening in response to her name and responding purposefully to command. However, she had generalised hypotonia, and pulse oximetry revealed a saturation of 89%. The arterial blood gases revealed a PO₂ of 58 mmHg, PCO₂ of 55 mmHg and a pH of 7.55 while breathing air. A biochemical profile performed on admission (Figure 1) led to the diagnosis.

Name	Age	Sex	Time of Collection Analysis		Date
Ms. J. T.	32	F	1110	1200	21.10.99

Sodium	138	mmol/L	(137 - 145)
Potassium	1.9	mmol/L	(3.1 - 4.2)
Chloride	81	mmol/L	(101 - 109)
Bicarbonate	50	mmol/L	(22 - 32)
Anion Gap	8.9	mEq/L	(8 - 16)
Calc Osmolarity	291	mmol/L	(280 - 300)
Glucose	4.4	mmol/L	(3.0 - 6.0)
Urea	10.7	mmol/L	(3.0 - 8.0)
Creatinine	0.04	mmol/L	(0.05 - 0.12)
Phosphate	0.72	mmol/L	(0.70 - 1.25)
Total Calcium	1.75	mmol/L	(2.10 - 2.55)
Albumin	42	g/L	(39 - 50)
Globulins	31	g/L	(22 - 35)
ALT	25	U/L	(10 - 45)
AST	29	U/L	(10 - 45)
GGT	35	U/L	(0 - 50)
ALP	82	U/L	(30 - 100)
Total bilirubin	28	µmol/L	(4 - 20)

Figure 1. Plasma biochemical profile performed on a venous blood specimen taken from the patient on admission

Diagnosis: Hypokalaemia and hypocalcaemia (caused by hypomagnesaemia?)

This patient had a serum magnesium level of 0.4 mmol/L (normal range 0.7 - 0.9 mmol/L) and her chest X-ray revealed no abnormalities. The hypo-kalaemia in this case caused the profound weakness, metabolic alkalosis and respiratory compensation (with hypercarbia), which led to her hypoventilation and hypoxia.

The hypokalaemia in this patient was probably due initially to her gastrointestinal loss with some renal loss secondary to the severe alkalosis. Usually, excess extrarenal potassium loss will cause an alkalosis in patients who have avid sodium retention from the distal nephron (e.g. in patients with primary or secondary hyperaldosteronism with an adequate distal delivery of sodium). In patients with diarrhoea, an excess gastrointestinal bicarbonate loss tends to occur which causes a characteristic hyperchloraemic acidosis, rather than a hypochloraemic alkalosis.¹ This patient subsequently admitted to diuretic (frusemide) use, which may have also promoted the hypomagnesaemia (and hypocalcaemia) as well as the alkalosis.

Hypokalaemia associated with hypomagnesaemia is usually resistant to potassium therapy if the hypomagnesaemia is not corrected simultaneously,² as a urinary potassium loss occurs with magnesium depletion.^{3,4} Hypocalcaemia caused by hypomagnesaemia is thought to be due to impaired release of PTH and impaired peripheral action of PTH,⁵ which also requires magnesium repletion before a normal calcium balance (and normocalcaemia) returns.⁴ An association has also been described between hypomagnesaemia and hypophosphataemia,⁶ although in this patient the initial phosphate level was within the normal range (0.72 mmol/L).

While hypomagnesaemia and hypocalcaemia may present with tetany, the presence of severe hypo-

kalaemia modifies this clinical feature and in our case was associated with generalised weakness. However, tetany may subsequently develop in patients in whom the extracellular potassium is corrected rapidly.⁷

Our patient improved slowly with both potassium and magnesium supplementation. Her diet was modified to suit her 'short bowel' syndrome and she was discharged from hospital 10 days later.

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