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Hypokalaemic paralysis secondary to thiazide diuretic abuse: an unexpected outcome for cauda equina syndrome

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ABSTRACT

Study design: Case report.

We present the case of a 55-year-old woman with cauda equina syndrome, and hypokalaemic paralysis secondary to thiazide diuretic abuse.

The authors have no conflict of interest.

Institutional Review Board was sought for this research in association with informed patient consent. The article in no way refers to the identity of the individual involved.

CASE REPORT

We present the case of a 55-year-old woman, previously healthy, who presented to the emergency department with an 18-hour history of progressive leg weakness, inability to pass urine, a 2-week history of intermittent diarrhea, 3 weeks of bilateral leg pain, and fatigue during the previous 3 months. During the preceding 6 months she lost 25 kg, dropping from 95 kg to 70 kg. The woman has a smoking history of 30-pack/year. She had previous mild episodic lumbar back pain but none on this occasion. She reported mild depression.

On catheterization, she had a urine output of 1150 mL clear urine. Neurological examination of her lower limbs revealed only lower motor neuron signs. Power was 3/5 at L2 and L3, 4/5 at L4 and L5, and normal in the S1 myotome bilaterally, with no sensory disturbance. Her tone and coordination was normal but her reflexes were diminished symmetrically. Neurological examination of the upper limb was entirely normal. A rectal examination showed a decreased anal tone.

Laboratory results are shown in **Table 1**. Result of an electrocardiogram was normal. A medical review was sought. Initial medical management included potassium replacement of 40 mmol/h in a normal saline solution. The pa-

tient was scheduled the following morning for a magnetic resonance imaging scan of the complete spine to rule out a compressive lesion or occult malignancy.

She was examined in the orthopaedic trauma ward 3 hours later because she had developed a cough, was dyspnoeic, and had occipital pain. The tone of her voice had lowered; she was slurring her words and was plethoric. She was now quadraparetic. Her lower limb power had deteriorated. The weakness in her upper limbs was again mainly proximal: 3/5 in C4 and C5, 4/5 in C6 and C7, and 5/5 in C8 and T1 myotomes. Oxygen saturation was falling continuously. The patient was pre-arrest. She was intubated and placed on ventilation, and transferred to the intensive care unit. She had a supraventricular tachycardia following intubation which was reversed medically, and she became hypotensive and required inotrope support.

The hypokalemia was corrected slowly during 48 hours to the normal range. The patient made a rapid and complete neurological recovery. She subsequently admitted to self-administering double doses of bendroflumethiazide, a thiazide diuretic, for months in an attempt to lose weight. A serological test result was positive for avian influenza, H5N1 subtype, and deemed the probable cause of her diarrheal illness.

Table 1 Initial venous and arterial blood tests.

Component	Result	(Reference range)
HB	11.4	(12–15 g/dL)
WCC	29	(3.5–10 x10 ⁹ /L)
Plts	643	(140–400 x10 ⁹ /L)
CRP	17.8	(0–5 mg/L)
U	25	(7–21 mg/dL)
Cr	233	(50–90 mg/dL)
K	1.8 (Haem)	(3.5–5.0 mmol/L)
Na	138	(135–145 mmol/L)
Cl	99	(98–106 mmol/L)
Glucose (fast)	6.8	(4–6 mmol/L)
eGFR	19	(> 90 L/min/1.73 m ²)
Ca	2.16	(2.1–2.5 mmol/L)
Phos	0.64	(0.8–1.5 mmol/L)
pH	7.42	(7.34–7.44)
pCO ₂	4.3	(4.4–6.0 kPa)
pO ₂	9.4	(10–13 kPa)
chCO ₃	20.6	(18–23 mmol/L)
Base excess	-2.8	(-3–+3 mEq/L)

DISCUSSION

Our patient's initial presentation was not typical of cauda equina syndrome and a review by the on-call medical team was obtained in the emergency department. The overriding priority was to admit the patient for a magnetic resonance imaging scan of her spine to rule out a causative lesion at the spinal cord.

Up to 20% of hospitalized patients are hypokalemic, with clinical significance in 4%–5% of them. Potassium levels have been shown to be normal in 95% of patients with eating disorders, so it is difficult to only make the association with low potassium levels [1]. Severe hypokalemia is relatively uncommon. Loss of potassium typically occurs in renal or gastrointestinal disorders or poor dietary intake but can also occur from medication, such as diuretics. Symptoms are often due to the underlying cause of the hypokalemia rather than hypokalemia itself and such was the focus of investigation in this case. The 2 weeks of diarrhea were a likely source of acute potassium loss. The absence of electrocardiographic abnormalities may reflect a background of chronic hypokalemia secondary to the patient's abuse of thiazide diuretic.

Hypokalemic acute flaccid quadriplegia has been described, particularly in association with renal tubular acidosis [2, 3]. All these patients had a positive urinary anion gap, as had our patient—20 mmol/L. In a similar case, a patient's neuromuscular status deteriorated on initiation of the recommended potassium infusion and required endotracheal intubation and positive pressure ventilation. Similarly, there was no evidence of electrocardiographic abnormalities [4]. Thyrotoxic hypokalemic periodic paralysis has been described in two patients 2–3 weeks after consuming thyroxine containing weight-loss supplements [5].

A rapidly evolving neurological deterioration with this presentation could have had a neuropathic, myelopathic, neuromuscular junction or myopathic origin; and differential diagnoses include Guillain-Barre syndrome, uremic polyneuropathy, transverse myelitis, poliomyelitis, or botulism.

Hypokalemic paralysis is rare. With atypical presentations, potential causes of neurological disorders should be considered and medical input should be obtained in the emergency department, particularly when such patients can deteriorate fast, as in our case. It is possible that the rapid potassium replacement may have triggered the development of cardiac and respiratory conditions. One must be aware of the dangers of diuretic abuse, particularly when intentional weight loss and depression are concerned. Hypokalemia can be the sole cause of flaccid paralysis especially when renal or its associated disorders are present.

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COMMENTARY

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Cawley et al describe an interesting case of progressive leg weakness and urinary retention caused by severe hypokalemia. The neurological presentation somewhat mimicked cauda equina syndrome, although there was no back or leg pain and no saddle anesthesia.

On a cursory review of the literature, I could find no other cases of cauda equina syndrome caused by thiazide-induced hypokalemia. However, there is a recent report [1] of a patient with lower extremity weakness, sensory disturbance, and intermittent urinary incontinence from Gitelman syndrome, which is a rare inherited defect in the distal convoluted tubule of the kidneys that manifests as hypochloremic metabolic alkalosis, hypokalemia, and hypocalciuria. People with Gitelman syndrome present with a metabolic profile almost identical to those treated with thiazide diuretics [2].

It is important to remember that cauda equina syndrome has a wide differential diagnosis including compressive, ischemic, and/or inflammatory neuropathy of multiple lumbar and sacral nerve roots [3]. The literature is rife with examples of unusual organic explanations for this clinical presentation, including transverse myelitis, vasculitis, spinal dural arteriovenous fistula, spinal ischemic stroke, inflammatory polyradiculopathy (autoimmune or infectious), and meningeal carcinomatosis (lymphomatous or metastatic) [4].

Although a structural cause, such as a large lumbar disc herniation, is important to rule out, Rooney et al [5] reported that 48% of patients seen in the emergency department for cauda equina syndrome turned out to have no obvious structural abnormality on magnetic resonance imaging. While some patients had an alternate organic cause, most cases were “non-organic” (ie, functional) in nature.

The authors have provided a valuable contribution to the literature, not only because they made an astute diagnosis, saving a patient who was in a “pre-arrest” state to a full recovery but also by adding to the broader differential diagnosis of cauda equina syndrome.

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EDITORIAL PERSPECTIVE

EBSJ thanks the authors for providing this fascinating case and reminding us of the bewildering variety of conditions that may present as spinal disorder. The commentary provided by Dr. Fourney underscores the rarity of this disorder and adds another variant to electrophysiological imbalance mimicking neurologic dysfunction with the description of Gitelman syndrome. While there remains strong hope for increasing cost efficiency in healthcare by more widespread use of care pathways in common conditions such as low back pain, cases like these remind us that there is no substitute for practicing good medicine when encountering each individual patient.