over administered his spray in his enthusiasm to cure his enuresis.

The child was admitted to the paediatric ward where management consisted of discontinuing the drug, fluid restriction, and observation. The day after admission his serum sodium was 131 mmol/l. He made a full neurological recovery and was discharged.

Discussion

Treatment with DDAVP nasal spray has improved the quality of life of thousands of patients with nocturnal enuresis since its introduction in 1989.³ It is well tolerated by most patients. Common local side effects include irritation of the nasal mucosa, rhinitis, and epistaxis. Systemic adverse effects include headache, dizziness, flushing, raised blood pressure, and abdominal pains.² Water intoxication and hyponatraemia are life threatening complications which may occur after accidental overdose or excess fluid intake. DDAVP should be given with care to patients with heart failure, or other conditions which might be aggravated by water retention. The recommended fluid intake during treatment is 30 ml per kg body weight during the four hour period before a dose of DDAVP and for 12 hours afterwards.³

The manufacturer recommends monitoring electrolyte concentrations at least once during intranasal DDAVP treatment if its use extends beyond one week.

In the case reported here convulsions developed more than 18 hours after the last dose. This corresponds to the pharmacokinetic properties of DDAVP. The antidiuretic effect of DDAVP becomes effective at 6–26 hours after intranasal application and lasts for approximately 10 hours (plasma half life is 2.2–4.4 hours).⁵

Basic management consists of stopping the drug, fluid restriction, and suppressive treatment for seizures as necessary. Recovery is usually quick, uncomplicated, and complete.

Robson *et al* reported seizures and altered levels of consciousness in patients taking conventional dosages of DDAVP.³ This can occur in patients with cystic fibrosis or nasal polyps who have an abnormal nasal mucosa.⁶

Drug interaction has not been substantiated, but neurologically active drugs such as methylphenidate and imipramine are thought to potentiate the action of DDAVP.⁷

Early symptoms of hyponatraemia are often non-specific and innocuous. They can be easily overlooked or mistaken for a viral illness.² Clear explanation of the hazards of DDAVP treatment to the patient and carers is essential. Children should be supervised when self administering DDAVP nasal spray.

There is not always a clear explanation for the hyponatraemia induced by the use of DDAVP. Further research is required in this area.⁸

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Hyperkalaemic paralysis—a bizarre presentation of renal failure

Gary L A Cumberbatch, Timothy J Hampton

Abstract

Paralysis due to hyperkalaemia is rare and the diagnosis may be overlooked in the first instance. However it is rapidly reversible and so long as electrocardiography and serum potassium measurement are urgently done in all patients presenting with paralysis, it will not be missed. A case of hyperkalaemic paralysis is described and a review of the emergency management discussed.

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Keywords: hyperkalaemia; paralysis; renal failure

Case report

A 53 year old woman presented to the accident and emergency department with a six hour history of dyspnoea and a 24 hour history of ascending weakness of her limbs. She had no past medical history of note and was on no medication. There had been no symptoms of a preceding viral illness.

On examination she was fully conscious, very pale, and unable to speak. Her respiratory rate was 28 breaths/min though shallow, pulse rate 60 beats/min, blood pressure 150/80 mm Hg, and she was apyrexial. She had flaccid paralysis of all limbs and no deep tendon

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Figure 1 Electrocardiogram.

reflexes were elicited. Bedside glucose test was normal and no urine was obtained after catheterisation.

Electrocardiographic (ECG) monitoring revealed very broad QRS complexes, absent P waves and very peaked T waves (see fig 1). The results of arterial blood gas analysis were obtained within 10 minutes: pH 6.99, arterial carbon dioxide pressure 6.0 kPa, arterial oxygen pressure 30 kPa on 60% oxygen, bicarbonate 9 mmol/l, base deficit of 20 mmol/l, and potassium 10.4 mmol/l.

She was given calcium chloride intravenously, followed by infusions of dextrose with soluble insulin and sodium bicarbonate.

Within 15 minutes she was able to talk and explained that she was having difficulty breathing. She was promptly transferred to the intensive care unit. Subsequent blood investigations showed a sodium concentration of 130 mmol/l, potassium 10.5 mmol/l, urea 72 mmol/l, and creatinine 2800 μ mol/l; she had a normochromic, normocytic anaemia with a haemoglobin concentration of 60 g/l, and normal serum calcium, magnesium, and liver function tests.

She was diagnosed as having hyperkalaemia secondary to renal failure and haemofiltration started. Within four hours she had regained almost full power in all her limbs and was able to sit up in bed unsupported.

By the next morning her acid-base balance had corrected, her potassium concentration was 5.9 mmol/l, and she was in sinus rhythm. Further investigation revealed gross bilateral hydronephrosis due to obstruction of both ureters by advanced cervical carcinoma.

She had bilateral nephrostomy drainage and underwent palliative radiotherapy and chemotherapy with full return of power to all limbs and no respiratory difficulties.

Discussion

Paralysis due to hyperkalaemia is rare, with only 10 cases reported in the literature. Return of full power was between five minutes to three hours from the time treatment was initiated in these reported cases.

Most patients with severe hyperkalaemia suffer fatal arrhythmias well before paralysis intervenes as cardiac muscle is much more susceptible to high serum potassium than skeletal muscle. It is not understood why, very occasionally, the converse occurs.

It is believed that the high extracellular concentration of potassium prevents repolarisation of the nerve and muscle membrane by reducing the efflux of potassium after depolarisation. The membrane therefore remains depolarised and refractory to any further stimulation. Consequently, skeletal muscle contraction cannot occur so paralysis results.¹

Other contributory factors include metabolic acidosis, hyponatraemia, and hypoxia all of which impair the Na^+/K^+ ATPase membrane pump, thereby interfering with the generation of an action potential.

As this case illustrates, progressive hyperkalaemia from chronic renal failure only occurs when oliguria supervenes because of the ability of the kidney to excrete potassium until renal failure is far advanced. Hyperkalaemic paralysis manifests itself with a sudden attack of flaccid paralysis which progressively ascends from the legs to the trunk and arms. If untreated it progresses to affect speech, swallowing, and finally ventilation.² There is no alteration of conscious level and no sensory loss, though paraesthesiae may occur. It has previously been mistaken for Guillain-Barré syndrome because of the almost identical presentation.³

ECG monitoring invariably reveals the pathognomonic features of hyperkalaemia: peaked T waves, diminution of the P wave, atrioventricular block, widening of the QRS complex, and ventricular arrhythmias. The tracing eventually assumes a sine wave appearance which may degenerate to ventricular fibrillation or asystole.

The emergency management of hyperkalaemic paralysis involves the administration of oxygen to prevent the potentiation of hyperkalaemia by hypoxia and assessing the need to assist ventilation. In only one of the reported cases was intubation necessary and this was

achieved without any muscle relaxants.⁴ If intubation is indicated, suxamethonium is contraindicated as it will exacerbate the existing hyperkalaemia and have no effect on the skeletal muscles as they are already depolarised. An anaesthetic induction agent alone is all that is needed to facilitate intubation and ventilation.

Immediate treatment includes the administration of calcium intravenously to prevent cardiac deterioration by stabilising the myocardial cell membrane and this can be given as either 10% calcium gluconate or 10% calcium chloride. Calcium should be given over five minutes under constant ECG monitoring but its stabilising effect will only be transient.

The hyperkalaemia is treated with a dextrose/insulin infusion which may reduce serum potassium as quickly as 1 mmol/l in 30 minutes because of the resultant intracellular shift of potassium. This occurs because the intracellular formation of phosphorylated glucose intermediates and glycogen bind potassium.

Soluble insulin is used with 50% dextrose in a ratio of one unit per 10 ml (5 g) and 100 ml infused over an hour. Rapid infusion must be avoided as it may cause a transient rise in serum potassium by its osmotic effect on cells.

Sodium bicarbonate should always be considered as it causes a prompt decline in serum potassium for a number of reasons. Firstly, it buffers hydrogen ions in the extracellular space and so promotes the intracellular shift of potassium. Secondly, by reversing a metabolic acidosis, potassium secretion into the distal tubule of the kidney is enhanced. Thirdly, as a hypertonic solution it will have a simple dilutional effect upon potassium and finally, in those patients with coexisting hyponatraemia, the salt load helps correct this and so improves Na^+/K^+ exchange. However, it must be administered cautiously as such a high sodium load can easily precipitate fluid overload especially in patients with renal insufficiency. An 8.4%

solution can be given as 50 ml over an hour and its effect will last two hours. An alternative intravenous route to that used for calcium must be used to avoid the precipitation of calcium bicarbonate.

Recent research now demonstrates that both nebulised and intravenous β agonists such as salbutamol also rapidly reduce serum potassium in patients with chronic renal failure. It is thought that endogenous catecholamines promote the intracellular shift of potassium and by stimulating the release of these catecholamines, β agonists may have a role in the treatment of hyperkalaemia.^{5,6}

Exchange cation resins such as calcium resonium remove potassium from the body and can be given orally or rectally. These resins can rapidly decrease serum potassium within 30 minutes and will continue to act for many hours.

As these pharmacologic agents will only temporarily reduce potassium, starting dialysis or haemofiltration must be the next priority. This will quickly restore normal electrolyte and acid-base balance while the cause for the hyperkalaemia is urgently sought.

We would recommend a 12 lead ECG and urgent serum potassium measurement in any patient presenting with paralysis, as this rare but readily reversible condition will then be quickly recognised.

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An unusual case of urinary retention due to imperforate hymen

David J Hall

Abstract

A 15 year old girl presented to the accident and emergency (A&E) department with a 24 hour history of lower abdominal pain, and was found to have acute urinary retention. She was discovered to have an imperforate hymen with associated haematocolpos and haematometrium. This is rare and is hence a very unusual presentation to the A&E department. Patients presenting with retention of urine should be carefully assessed for the cause.

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Keywords: urinary retention; imperforate hymen

Case report

A 15 year old girl presented to the accident and emergency (A&E) department with a 24 hour history of lower abdominal pain. She reported frequency of micturition and dysuria, and gave a history suggestive of being constipated, although her bowels had been opened the previous day.

Examination revealed her to be in discomfort and to have a fever of 38°C but she was otherwise not systemically unwell. An abdominal mass extending from the pelvis to the umbilicus was noted and a presumptive diagnosis of acute urinary retention secondary to constipation and urinary infection was made.

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