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Case Report

Airway Management of Hypokalaemic Paralysis with Trismus and Bulbar Palsy Due To Conn's Syndrome

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INTRODUCTION

Hypokalaemic paralysis is an atypical condition of acute muscle weakness caused by a variety of disorders, occurring when plasma potassium ion levels drop below 3.5 mmol/L. This reduction in potassium affects the resting membrane potential of muscle cells, leading to paralysis.[1]

Trismus associated with hypokalaemia is rare, with only one documented case.[2] Trismus is a tonic spasm of the masticatory muscles, particularly the masseter and temporalis, resulting in reduced jaw opening.[1,2] In such cases, respiratory arrest and death due to severe arrhythmia have been reported, primarily due to prolonged hypoxaemia and delays in airway management. Thus, urgent care and emergency physicians must ensure adequate oxygenation, ventilation, and, if necessary, perform rapid sequence intubation (RSI).[2]

This case report examines the clinical evidence on the use of neuromuscular relaxants and RSI in emergency rooms for muscle paralysis due to ionic alterations. The uniqueness of this case lies in the combination of trismus, hypokalaemic paralysis, and Conn's syndrome undergoing surgical intervention.

CASE REPORT

A 45-year-old male gravedigger with a history of high blood pressure treated with enalapril and hydrochlorothiazide presented with 10 days of paraesthesia in his right hand and fingers of his left hand, along with progressive leg weakness. Over the past 3 days, he experienced difficulty swallowing, dysphasia, dysarthria, and cervical muscle weakness. He denied drug, alcohol, or canned food use.

On arrival, the patient was conscious and oriented, with a heart rate of 95 bpm, blood pressure of 170/110 mmHg, and oxygen saturation of 92%. Neurological examination was normal, but he had difficulty opening his mouth and weakness in bulbar and cervical muscles. Oropharyngeal examination showed uvula swelling, likely due to soft palate paralysis.

The patient exhibited motor weakness with upper extremity strength of 2/5 and lower extremity strength of 1/5. Venous blood gas analysis revealed marked alkalosis and severe hypokalaemia (1.3 mmol/L). ECG showed a sinus rhythm with flattened T waves, consistent with hypokalaemia. Treatment included Hydrocortisone 200 mg and intravenous infusion of 40 mEq of potassium chloride in saline. Although corticosteroid treatment can exacerbate hypokalaemia, Hydrocortisone was administered empirically, due to its rapid anti-inflammatory effect, aimed at managing uvular edema and a potential inflammatory process that could compromise the airway. The priority at that point was to ensure patient stabilization and secure adequate airway access.

During observation, the patient developed respiratory failure with cyanosis, requiring unsuccessful face mask ventilation, dropping saturation to 53%. Cisatracurium 20 mg and Midazolam 15 mg were administered for orotracheal intubation, which was difficult due to trismus. ENT specialists performed an emergency tracheostomy. The patient suffered cardiac arrest but was resuscitated after 1 min of chest compressions and adrenaline boluses.

Post-tracheostomy, ventilation improved saturation to 99%, allowing the patient to be moved to surgery for

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tracheotomy completion and mechanical ventilation connection. Serum potassium levels slightly improved to 2.1 mmol/L. Vocal cord integrity was assessed using a flexible fibrobronchoscope, ruling out damage.

The patient was transferred to the ICU, where continuous potassium replacement improved respiratory function and muscle strength. Trismus resolved after 24 h, but hypokalaemia persisted (2.3 mmol/L). Mechanical ventilation was removed 4 days later. The patient remained in the ICU for 20 days before being discharged to internal medicine for further study.

The diagnosis of primary hyperaldosteronism was confirmed by the endocrinology department following an extensive diagnostic workup. Endocrinological tests revealed elevated aldosterone levels with very low plasma renin activity, which were confirmed through the saline infusion test, iodocholesterol scan, adrenal vein sampling, and several other differential diagnostic tests. This process ultimately confirmed aldosterone overproduction by a left adrenal adenoma, leading to the final diagnosis of Conn's syndrome.

The treatment consisted of laparoscopic surgery to remove the adrenal adenoma, which was successfully performed. A long-term endocrine follow-up was conducted to monitor for possible recurrences and ensure the complete resolution of hypokalaemia and hyperaldosteronism.

DISCUSSION

Trismus and Hypokalaemic Paralysis

Trismus and bulbar palsy are extremely rare in severe hypokalaemia, with only a few reported cases.[1,2] This case is the first linking hypokalaemic paralysis to Conn's syndrome from an aldosterone-producing adenoma.

Gupta[2] described a 28-year-old man with barium carbonate poisoning, hypokalaemia, flaccid quadriparesis, and trismus. Trismus as a manifestation of localized myotonia due to hypokalaemic paralysis is unusual but linked to ion-sensitive channels and possibly genetic predisposition.[2,4] Other causes of trismus include tetanus, rabies, poisoning, drug reactions, multiple sclerosis, and brainstem disease.[1,2]

Hypokalaemic periodic paralysis is a common cause of acute weakness with metabolic and electrolyte abnormalities (Table I).[3,4] Common causes include familial periodic paralysis, renal tubular acidosis, severe diarrhea, and excessive caffeine intake (Table I).[3,4]

Conn's syndrome accounts for 33% of primary hyperaldosteronism cases, leading to excess aldosterone, increased sodium reabsorption, and renal potassium excretion. While symptoms can be managed with spironolactone, surgical removal of the adenoma is preferred.[3,4]

TABLE I.
Causes of Hypokalaemia.

Potassium depletion – renal
Increased aldosterone
Diuretics
Hypomagnesemia
Renal tubular acidosis (Type I and II)
Metabolic alkalosis
Liddle's syndrome
Conn's syndrome
Potassium depletion – extrarenal
Decreased intake
Vomiting/Diarrhea
Zollinger–Ellison syndrome
Fistulas
Potassium shift into cells
Increased insulin
Alkalosis
Thyrotoxic periodic paralysis
Familial Hypokalaemic paralysis

Management of Hypokalaemia and Rapid Sequence Intubation

Emergency physicians must rapidly recognize and treat hypokalaemia to prevent respiratory failure. The recommended treatment is an intravenous dose of 20–60 mEq of potassium chloride in 1 h, preferably through a central line, with continuous ECG and serum potassium monitoring.[3]

Airway control in hypokalaemic paralysis poses challenges due to limited mouth opening and neck movement. Delays in securing a definitive airway increase morbidity and mortality. RSI aims to minimize time between loss of airway reflexes and endotracheal intubation to prevent regurgitation of gastric contents.[5]

Ideal neuromuscular relaxants for RSI should allow rapid intubation and quick recovery of spontaneous breathing with minimal hemodynamic changes. Succinylcholine is preferred for RSI due to rapid onset and short duration but is unsuitable for patients with renal failure, brain injury, burns, neuromuscular diseases, or potassium imbalances, as in this case.[3,5]

Rocuronium, a non-depolarizing muscle relaxant, provides rapid action and is an excellent alternative to succinylcholine, especially with sugammadex, its specific antagonist, allowing rapid reversal of deep blockade. Studies show rocuronium with sugammadex allows faster recovery of spontaneous ventilation compared to succinylcholine.[5] However, cricothyrotomy or tracheostomy remains the gold standard for airway management in "cannot intubate, cannot ventilate" scenarios.

In this case, cisatracurium was used initially, followed by rocuronium to improve trismus. Combining two non-depolarizing muscle relaxants is uncommon but reported to have a synergistic effect.[5]

CONCLUSION

This case emphasizes the need for rapid recognition of severe hypokalaemia to prevent cardiorespiratory failure and highlights a rare instance of quadriparesis with bulbar palsy and trismus due to an aldosterone-producing adenoma. The combination of trismus with hypokalaemia is extremely rare, and emergency physicians must be proficient in airway management and RSI, including the use of rocuronium and sugammadex.

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