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CASE REPORTS

MYASTHENIC CRISIS–LIKE SYNDROME DUE TO *CLEISTANTHUS COLLINUS* POISONING

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ABSTRACT

Poisoning with Cleistanthus collinus frequently causes cardiac manifestations such as rhythm disturbances and also results in other manifestations such as metabolic acidosis and hypokalemia. We present the case of a patient who presented with a rare myasthenic crisis–like syndrome requiring assisted ventilation due to Cleistanthus collinus poisoning, which responded to treatment with neostigmine.

Key words: Cleistanthus collinus, neuromuscular blockade, poisoning

INTRODUCTION

Cleistanthus collinus (known as 'Vadisaaku') is commonly consumed for suicidal and homicidal poisoning in the Chittoor district in the state of Andhra Pradesh. Sparse literature is available regarding the uncommon manifestations of poisoning with *Cleistanthus collinus*. Published reports suggest that it causes cardiac rhythm disturbances and also results in other manifestations such as metabolic acidosis, hypokalemia,[1-4] and rarely neuromuscular weakness.[3,5] We present the case of a patient who presented with a rare myasthenic crisis–like syndrome due to *Cleistanthus collinus* poisoning, which responded to treatment with neostigmine.

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CASE HISTORY

A 38-year-old man presented to the emergency room with history of consumption of more than 50 leaves of *Cleistanthus collinus* following a bout of alcohol intake. He received gastric lavage elsewhere and was referred to our tertiary care teaching hospital three days after the consumption as he developed fever, blurred vision, generalized muscular weakness, difficulty in swallowing, and breathlessness. There were no abdominal or gastrointestinal symptoms. On examination he was conscious, coherent; his temperature was 102° F; heart rate, 120/min, regular; and respirations 52/min. He was conscious, and his speech was slurred. He also had bilateral ptosis, restricted ocular movements in all the directions, bilateral facial weakness, and inability to hold the neck up.

Pupils were normal sized; direct and indirect pupillary reflexes were intact. Findings of fundus examination were normal. Fasiculations were absent. He had grade 3/5 power in all four limbs, predominantly proximal muscle weakness, intact deep tendon reflexes, and a normal sensory system. Findings of rest of the physical examination were unremarkable. At admission, hemogram revealed neutrophilic leukocytosis, serum sodium was 132 mEq/L, and serum potassium was 3.6 mEq/L. Otherwise, results of serum biochemistry were normal. Serial electrocardiograms revealed sinus tachycardia. The chest radiograph and echocardiography were within normal limits.

On the second day of hospitalization, the respiratory effort deteriorated arterial blood gas (ABG) analysis while breathing room air at sea level revealed oxygen tension (PaO_2) was 55 mm Hg; carbon dioxide tension ($PaCO_2$), 24 mm Hg; pH, 7.225; bicarbonate, 18 mEq/L; and alveolar-arterial oxygen gradient [A-a (DO_2)], 60.6; and mechanical ventilatory support was instituted. Apart from the supportive care, he received intravenous neostigmine (0.5 mg) and atropine (1 mg) administered once every four hours. Within one hour of administration of neostigmine, ptosis disappeared and the power became normal (grade 5/5) in all the four limbs. A T-piece trial was initiated on the third day of admission, and the patient was extubated by the fourth day. Electroneuromyography (ENMG) and decremental response with repetitive nerve stimulation could only be done while he was receiving neostigmine, and the results of nerve conduction studies at that point in time were normal; no decremental response was noted. Neostigmine and atropine were continued and gradually tapered over a period of 10 days.

Following correction of metabolic acidosis, the ABG normalized by the next 48 h. We could not measure serum chloride levels and urinary potassium levels. From the third day of admission, the patient developed hypokalemia [serum potassium levels, 2.4 mEq/L to 2.8 mEq/L], which was corrected over the subsequent 10 days with potassium chloride, initially intravenously for 72 h, followed by oral potassium chloride supplementation. By the 13th day of admission, serum potassium levels became normal and remained so.

While in hospital, he also developed hospital-acquired pneumonia, which was treated with intravenous antibiotics as per culture and sensitivity report. He was discharged in stable condition on the 24th day of hospitalization.

DISCUSSION

Cleistanthus collinus is an extremely toxic plant-poison. The actual composition of the poison is still to be elucidated. Diphyllin glycosides, cleistanthin A and B — the toxins of *Cleistanthus collinus* — are thought to produce cardiac arrhythmias, urinary potassium wasting, hypoxia, metabolic acidosis, and hypotension.[1]

Neuromuscular manifestations such as those observed in the present patient have occasionally been described earlier,[3,5] but their reversal with neostigmine has never been documented. The experimental evidence in Albino rat model suggests that *Cleistanthus collinus* leaf can result in neuromuscular junctional blockade, particularly at the post-synaptic acetylcholine receptors,[6-8] mimicking myasthenia gravis and the toxic effect of

D-tubocurarine. This *in vivo* experimental neuromuscular blockade in rat model was potentially reversible with the administration of neostigmine.[6-8] These observations prompted the use of neostigmine in this patient.

Hypokalemia was unlikely to be the cause of neuromuscular weakness at the time of admission, as the serum potassium levels were normal. Absence of abdominal symptoms and absence of a characteristic descending weakness, absence of fasiculations, and a dramatic response to neostigmine argue against botulism as a possibility and strongly suggest a defect in the post-synaptic acetylcholine receptor. Oropharyngeal weakness and loss of efferent limb of the cough reflex due to respiratory muscle weakness could have predisposed this patient to develop aspiration pneumonia. Subsequent occurrence of hypokalemia on the third day of admission reiterates the importance of monitoring serum potassium levels and correcting hypokalemia in patients with *Cleistanthus collinus* poisoning.

Awareness regarding myasthenic crisis–like presentation due to *Cleistanthus collinus* poisoning resulting in acute respiratory failure can facilitate early administration of neostigmine, which, along with assisted ventilation, can be potentially life saving.

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