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Case Report Open Access

Severe *Nicotiana glauca* Poisoning: A Case Report

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Abstract

Nicotiana glauca is an herbaceous plant with high toxicity due to its primary alkaloid anabasine content which acts as an agonist at peripheral nicotinic receptors.

Case report: We describe an uncommon and severe case of Nicotiana glauca poisoning in a seventy-five-year old man. He developed gastrointestinal and muscular signs two hours after ingestion of green shrubs with glaucous leaves that he mistook for spinach. Soon, in the emergency room he developed coma without localizing signs and acute respiratory failure without abnormalities in the chest X–ray that required intubation and mechanical ventilation for one day. The patient fully recovered and was discharged at home without any residual sequelae.

Conclusion: The ingestion of anabasine, the principal alkaloid in the Nicotiana glauca may result in severe poisoning manifested by acute respiratory failure. Supportive care should make a full recovery.

Keywords: *Nicotiana glauca*; Anabasine; Poisoning; Acute respiratory failure

Introduction

Nicotiana glauca is a species of wild tobacco known by the common name tree tobacco. It is a common roadside weed with much branched shrub that normally grows to a height of more than 2 m. It can reach 7 m in height. Its leaves are thick and rubbery up to 20 cm long. It has yellow cannular flowers about 5 cm long and 1 cm wide (Figure 1). It is a plant native of tropical America, Mexico and the West Indies. It was introduced in Tunisia in 1830 from Europe. *Nicotiana glauca* is an uncommon cause of poisoning in human [1-8]. Anabasine a nicotinic receptor agonist with an additional weak acetylcholinesterase inhibitor activity is the predominant alkaloid (98%) in *Nicotiana glauca* [9-12]. It produces a nicotinic-cholinergic syndrome with muscle weakness and autonomic instability as the main manifestation [1]. We describe a rare case of human poisoning by ingestion of boiled leaves of *Nicotiana glauca* complicated by abrupt acute respiratory failure and coma.

Case Report

A 75 year old man with medical history of diabetes and hypertension was admitted to intensive care unit for acute respiratory failure and coma, shortly after ingestion of green shrubs with glaucous leaves that he mistook for spinach. Two hours after ingestion of *Nicotiana glauca* he vomited several times, followed by the onset of muscle weakness of lower limbs. He was transferred by his family to the hospital. At the Emergency Department the patient was afebrile and awake and his neurological exam didn't find localizing signs and his pupils were in intermediate positions and reflective. One hour later, he developed acute respiratory failure with an oxygen saturation of 60% (PaO_2: 35 mmHg, $PaCO_2$: 59 mmHg) and became unconscious with Glasgow scale at 3, his systolic blood pressure was 180 mm H_2O and heart rate was 72 beats/min.

Figure 1: *Nicotiana Glauca.*

The rest of the physical exam was normal. Initial laboratory blood tests work up at the emergency department showed no abnormalities. The patient was intubated and mechanically ventilated. No other confounds were present in our patient. Indeed, the head computed tomography did not reveal any focal finding and twenty hours later the patient was extubated after the recovery of a normal neurologic status, his blood pressure decreased to 135/65 mmHg, and heart rate to 70 beats/min. Thereafter, the patient reported tiredness and headache which resolved spontaneously without any residual sequelae.

Diagnostic assays for urine and serum levels of anabasine weren't done because they were not available in our laboratory.

Discussion

There are very few cases of human *Nicotiana glauca* poisonig in the literature [1-8]. Often victims confused the plant with another one such as spinach [1]. The onset of symptoms in acute Nicotiana glauca poisoning ranges between 5 min and 3 hours after ingestion [1-8]. Our patient has developed symptoms two hours after ingestion.

Toxicity of this nicotine alkaloid is a result of additional actions at ganglionic sites, motor end plates and smooth muscles. Nicotine alkaloids initially stimulated the ganglia of the sympathetic and parasympathetic nervous systems by a direct acetylcholine like action on the ganglion. This is quickly followed by prolonged gonglionic blockage due to persistent depolarisation [5].

After ingestion, vomiting is a result of stimulation of the emetic chemoreceptor trigger zone [5]. The central nervous system (CNS) signs depend upon the dose and vary from mild stimulatory effects such as tremors, hyper excitability and auditory and visceral disturbances, to severe excitatory signs such as marked incoordination and convulsions. Anabasine transiently stimulates and then severely depresses the CNS [13]. Stimulation of sympathetic ganglia and the adrenal medulla combined with discharge of catecholamines from sympathetic nerve endings and chromaffin tissues of different organs are assumed to be responsible for the cardiovascular signs.

In skeletal muscles, anabasine initially stimulates nicotinic receptors of the motor end plates and in large doses it blocks the receptors causing total paralysis. Ultimately, sudden death is from respiratory paralysis of the diaphragm and chest muscles, resulting from descending paralysis and depolarization block of neuromuscular junction [14]. Our patient had most of the signs described above.

The diagnosis of *Nicotiana glauca* poisoning is typically made on the basis of the patients' history and a presentation with features of anabasine poisoning (like our case) or by high–performance liquid chromatography/photodiode array/mass spectrometry used to detect and quantify anabasine [14].

There is no specific antidote for *Nicotiana glauca* poisoning, and the treatment is largely supportive and symptomatic. Out of 17 reported cases in the literature, 9 recovered without sequellae and 8 resulted in death which occurred as a result of sudden respiratory failure [1].

Conclusion

Ingestion of *Nicotiana glauca* can cause severe systemic intoxication due to its nicotinic receptor agonist action with respiratory muscles paralysis.

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