CLINICAL VIGNETTE

Tree Tobacco Poisoning

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A 68-year-old female with a past medical history significant for hypertension and obstructive sleep apnea developed nausea and diarrhea 30 minutes after eating dinner. The patient reported that dinner included eating a plant growing in her backyard that she had not eaten before. She developed nausea with multiple episodes of non-bloody diarrhea. She denied any chest pain or shortness of breath and presented to the emergency room for further evaluation. Her husband, who had eaten the same food, also developed diarrhea and nausea with vomiting. The initial vital signs were Temp 98.1, BP 159/108, HR 86, RR18, and O2 sat 99% on room air. The diarrhea resolved in the emergency room, but she developed dyspnea approximately 2 hours later. A chest x-ray and EKG were both normal, but she subsequently developed tachycardia with a pulse of 120 and hypoxemia, requiring supplemental oxygen. She initially improved with 2 liters/minute, via nasal canula, but subsequently required 4 liters/minute of oxygen, and continued to desaturate. Bipap was started and her oxygen saturation improved to 100%; however, her mental status began to decline. A new rash was noted on the patient's rash and abdomen, and she received IV solumedrol. However, the patient became minimally responsive and was intubated to protect her airway. The patient's condition stabilized after intubation and was eventually extubated after 24 hours. The plant that the patient and her husband ingested was subsequently identified as nicotiana glauca.

Nicotiana glauca, also known as wild tobacco, or tree tobacco, is a multi-branched perennial shrub up to 5 meters in height. It is characterized by smooth green hairless branches; tubular yellow flowers; and large, alternate, ovate, and glaucous leaves.[1] The word, *Glauca,* was derived from Greek meaning "bluish-gray," which refers to the blue-green powdery coating on the plant.[1] Indigenous to South America, it has become a weed plant in Arizona, California, Texas, Mexico, Hawaiian Island, the Mediterranean region, and Australia.[1]

Unlike other species in *Nicotiana* genus, the predominant nicotinic alkaloid in Nicotina glauca is anabasine rather than nicotine. Anabasine, also known as neonictoine, is a highly toxic basic compound, which exists as enantiomers in equal amounts.[2] The substance has been smoked in rituals, used as medicine, as well as a botanical insecticide in the past.[2,3] Like nicotine, it acts on the nicotinic-type of acetylcholine receptors (nAChRs). Varying in structure, these receptors are all essentially sodium-gated receptors and are widely distributed in the central and autonomic nervous systems and at neuromuscular junctions.[4] When activated, these receptors act as a channel for positively charged ions, mainly sodium. The increase in sodium ion influx through the channel leads to prolongation of membrane depolarization and subsequent enhancement of action potential propagation, which in turn, produces various physiologic effects.

The clinical presentation of nicotinic intoxication displays a biphasic pattern. Initially, the patients may experience stimulatory adrenergic effects of high blood pressure and tachycardia accompanied by parasympathetic signs of miosis, salivation, lacrimation, nausea, vomiting, and diarrhea.[4] Neurological effects of ataxia, tremor, restlessness, headache, visual and hearing disturbances, confusion, dizziness, muscle fasciculations, tremor, miosis, and seizures may also be observed.[4] After the initial stimulatory phase, a period of direct depressive effects ensues due to the paradoxical inhibition of the nicotinic cholinergic receptors following a sustained agonistic action to the point where receptors enter a refractory state.[2] This paradoxical phenomenon of autonomic ganglionic and neuromuscular blockade leads the major toxic complications of hypotension, sinus bradycardia, cardiac conduction abnormalities, arrhythmias, and respiratory depression.[4]

Onset of symptoms usually occurs within 60-90 minutes of ingestion of the plant but may be delayed up to 4 hours. Symptoms can last from 1-2 hours in mild exposure but may last up to 1-3 days in severe intoxications.[4] Diagnosis is largely clinical and is made based on patient's history. Diagnostic urine and serum tests are not readily available in most hospital laboratories. Samples of the suspected plant can be sent to a botanist for formal identification via gas chromatography and mass spectrometry analysis.

Given the rapid absorption of Nicotinic alkaloids and the frequent presence of vomiting as a symptom, treatment with activated charcoal has a very limited time window but can be considered in patients who have ingested a potentially toxic amount of the plant within an hour. Other elimination techniques such as acidification of urine or hemodialysis have not been recommended due to lack of proven efficacy.[4] There is no known antidote for nicotinic poisoning and treatment is largely supportive and symptomatic.[1] Adrenergic stimulation is typically brief and does not require any specific treatment. For patients with more serious intoxications, supportive measures including securing airway, assisted ventilation, seizure control, and hemodynamic stabilization may be necessary. Our patient displayed the typical biphasic pattern

with nicotinic intoxication, an adrenergic response with nausea and vomiting followed by a refractory state due to prolonged stimulation that resulted in respiratory depression requiring intubation in this patient. She has no long-term symptoms related to her acute illness and the remaining plants growing in her garden have been removed.

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