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An Unusual Cause of Fulminant Guillain-Barré Syndrome: Angel's Trumpet

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A 5-year-old previously healthy boy presented with typical findings of Guillain-Barré syndrome and unilateral tonic pupil. He was placed on mechanical ventilation for 35 days for respiratory failure. Plasmapheresis and two courses of intravenous immunoglobulin therapy were given to the patient, and he experienced stepwise recovery from his illness. This case of acute motor axonal neuropathy type Guillain-Barré syndrome is novel in that the cause was established as ingestion of a toxic solanaceous plant, angel's trumpet (*Brugmansia suaveolens*; syn. *Datura suaveolens*). Understanding the signs and symptoms of angel's trumpet toxicity can allow for early diagnosis and proper case management.

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Introduction

Guillain-Barré syndrome, an autoimmune-mediated disease, is the most common cause of acute flaccid paralysis in childhood [1]. A variety of infections (e.g., Epstein-Barr virus, cytomegalovirus, hepatitis, varicella, other herpes viruses, *Mycoplasma pneumoniae,* and *Campylobacter jejuni*), as well as immunizations, have been known to precede or to be associated with the illness [2]. Other unusual causes are snake bite, medications (e.g., allopurinol), and Hodgkin disease, as well as events such as surgery and childbirth [3,4]. Guillain-Barré syndrome has been subdivided into acute inflammatory demyelinating polyradiculopathy, acute motor axonal neuropathy, acute motor and sensory axonal neuropathy, and Fisher syndrome [5].

Reported here is a novel case of acute motor axonal neuropathy type Guillain-Barré syndrome caused by angel's trumpet (*Brugmansia suaveolens*; syn. *Datura suaveolens*) plant ingestion. Understanding the signs and symptoms of angel's trumpet toxicity can lead to early diagnosis and proper case management.

Case Report

A 5-year-old previously healthy boy presented at a hospital after developing flaccid paralysis associated with respiratory symptoms. He was provided airway support via face mask and was transferred to the author's pediatric intensive care unit for further management. A day before admission, he fell down while walking and had rapidly progressive weakness. He had no history of febrile illness, vaccination, or any medication during the previous month.

Physical examination at the time of admission revealed difficulty in speaking and swallowing, rapid shallow breathing with a rate of 60/min, pulse of 152/min, blood pressure of 155/55 mmHg, and dry mouth with red, inflamed oral mucosa with coated tongue. Neurologic examinations demonstrated marked hypotonia and diffuse muscle weakness with absent deep tendon reflexes, as well as marked anisocoria, with the left pupil almost totally dilated and minimally reactive to light. Ptosis was not observed, and extraocular movements remained intact. There were no sensory abnormalities. The rest of the examination was unremarkable.

Laboratory studies indicated a white blood cell count of 14,600/mm^3; findings were normal for serum C-reactive protein, electrolyte, and renal and liver function tests. Lumbar puncture revealed white cells (predominantly lymphocytes) at 11/mL, protein at 70 mg/dL, and glucose at 100 mg/dL. Cerebrospinal fluid culture was negative for enteroviruses and bacteria and stool culture was negative for *Campylobacter jejuni* and polioviruses. Serologic findings were negative for *C. jejuni*, *Mycoplasma pneumoniae*, human immunodeficiency virus, Venereal Disease Research Laboratory test, Epstein-Barr virus, and hepatitis B and hepatitis C virus. Findings for cytomegalovirus immunoglobulin M were negative, but positive for immunoglobulin G. Nerve conduction and electromyographic studies were performed on the median, ulnar, tibial sural nerves using conventional procedures. These documented findings compatible with diffuse motor axonal neuropathy (reduced compound muscle action potential with normal conduction and sensory nerve conduction velocities). Postcontrast gadolinium enhanced magnetic resonance imaging revealed marked enhancement of the nerve roots in the conus medullaris and cauda equine (Fig 1). Cranial magnetic resonance imaging did not reveal any enhancement or thickening of the third or sixth cranial nerves.

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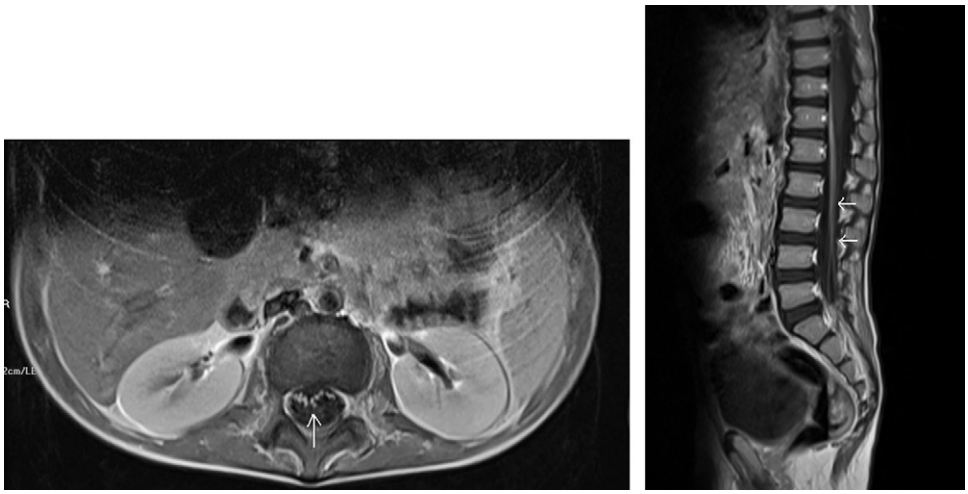

Figure 1. T_1-weighted postcontrast axial (A) and sagittal (B) magnetic resonance image of the cauda equina reveals smooth, diffuse enhancement of the nerve roots.

On the basis of the clinical and laboratory findings, the child was diagnosed as having acute motor axonal neuropathy type Guillain-Barré syndrome. The eye drop test with 0.125% pilocarpine induced a marked miosis in left pupil, which became smaller than the right pupil, and there was accommodation paresis with no pupillary near response. These findings confirmed the diagnosis of tonic pupil. In the absence of patient cooperation, segmental palsy of the sphincter was not assessed biomicroscopically.

Because of increasing respiratory failure, the patient was intubated and placed on mechanical ventilation on the day of admission. Four sessions of therapeutic plasmapheresis were performed every other day. Despite the plasma exchange treatment, the patient lost the gag reflex, and spontaneous breathing stopped. Subsequently, he was given intravenous immunoglobulin at the dose of 0.4 g/kg per day for 5 days with no improvement. On the 25th day of admission, the patient was given a repeat course of intravenous immunoglobulin therapy. After 35 days of mechanical ventilation, he was extubated and ventilation via face mask was started for respiratory support. He was able to sit on 45th day of admission. On the 60th day he could walk with assistance.

A few days after withdrawal of mechanical ventilation, the child was fully capable of speaking and was asked if he had possibly consumed any toxic substances before admission. He reported having eaten ''too many'' white flowers 1 day before and also almost 2 weeks before admission, when he had been playing in the backyard of the house. The parents made the plant identification by comparing the plants in their backyard with available pictures (obtained from the Internet), narrowing it down to angel's trumpet.

Discussion

Angel's trumpet (*Brugmansia suaveolens*; syn. *Datura suaveolens*) (Fig 2) is classified as a member of the nightshade family (Solanaceae). It has a high potential for abuse, in that it contains tropane alkaloids such as scopolamine, hyoscyamine, and atropine [6]. It is highly toxic, especially if ingested, and can cause life-threatening illness.

There have been numerous reports of angel's trumpet plant poisoning over the last 40 years, but the majority have been case reports or small case series. Hall et al. [7] described the cases of 10 adolescents with angel's trumpet intoxication within 3 month period. Delirium, dilated pupils, systolic blood pressure elevation, disorientation, dryness of skin and mucous membranes, and deep tendon reflex hyperactivity were the most common features in these patients. Three of the 10 had flaccid paralysis, and one of them died. Convulsions and flaccid paralysis were observed only in those subjects reporting the ingestion of more than six flowers. In the present case, the patient presented with Guillain-Barré syndrome with similar findings to those reported by Hall et al. [7], except for absent deep tendon reflexes; he reported having eaten ''too many'' flowers.

The unusual aspects of this case were that patient had unremarkable antecedent history for Guillain-Barré syndrome and also had mydriasis without ophthalmoplegia. The differential diagnoses of mydriasis include third nerve palsy, tonic pupil, traumatic mydriasis, and pharmacologic mydriasis [8]. In this case, third nerve palsy was excluded because there was no ptosis or motility deficit and also because magnetic resonance imaging findings revealed no cranial nerve pathology. The patient had no history of any medication and trauma. Tonic pupil is diagnosed by the topical intraocular treatment with 0.125% diluted pilocarpine, which induces a stronger miotic response in the affected eye, indicating denervation hypersensitivity of the pupil, and a defect of the postganglionic parasympathetic pathways.

Guillain-Barré syndrome accompanied by tonic pupil is very rare, and only two cases have been reported in the literature, both adults. One was a 55-year-old woman with Guillain-Barré syndrome associated with bilateral tonic pupil preceded by herpes simplex virus infection [9]. The other was a 66-year-old woman with acute polyneuropathy involving the motor, sensory, and autonomic nervous system [10]. Either topical exposure or ingestion of the flowers of angel's trumpet can cause unilateral or bilateral tonic mydriatic pupil [11]. In the present case, the patient had ingested angel's trumpet more than once within a month. The conclusion was, therefore, that angel's trumpet could have triggered an immune reaction against peripheral nerve myelin, leading to Guillain-Barré syndrome.

Figure 2. Leaves and flowers of angel's trumpet (Brugmansia suaveolens; syn. Datura suaveolens). A poisonous member of the nightshade family (Solanaceae), angel's trumpet is cultivated as an ornamental garden plant. Flower color varies from white to pale yellow or pink, and sometimes orange or red.

To our knowledge, this is the first reported case of Guillain-Barré syndrome caused by angel trumpet plant poisoning. This case report adds to the differential diagnosis of Guillain-Barré syndrome in otherwise healthy children. It is important for parents and garden suppliers to be aware of the potential adverse effects of angel's trumpet plants.

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