



Life-Threatening Poisoning Associated with Henbane Plant

Hayatı Tehdit Eden Ban Otu Zehirlenmesi

Ökkeş Hakan Miniksar, Ahmet Selim Özkan, Mustafa Said Aydoğan, Aytaç Yücel, Türkan Toğal

Inonu University, Faculty of Medicine, Department of Anesthesiology and Reanimation, Malatya, Turkey

Dear Editor,

Hyoscyamus Niger (HN) is a very common and highly hallucinogenic plant which contains anticholinergic agents. The HN is found in every region in Turkey. It is 25-80 cm tall with yellowish, purple flowers. As an annual herbaceous plant, it opens up with a lid-like cover when it is ripe and contains many seeds in its fruits. HN is commonly known as "stinking nightshade" or "black henbane;" in Turkey, HN is defined as "Ban Plant" (Ban Otu) or "Crazy Bat" (Deli Bat). There are 6 different types of henbane in Turkey. Black henbane and maize henbane are the kinds used in medicine. Maize henbane grows in and around Malatya in Turkey. Its leaves contain hyoscyamine, scopolamine (hyoscine), and several tropane alkaloids. Glycosides is found in the entire body of the plant, though it is dense in the seeds (1). The oral intake can cause central and peripheral anticholinergic effects. It is known that HN poisoning leads to anticholinergic syndromes. In this case report, we are presenting the life-threatening poisoning of a family of four after the accidental oral intake of HN.

10 minutes after cooking and consuming a plant in their garden, which was reported to be HN, all members of the family developed nausea, vomiting, visual and speech disorders, dry mouth, altered consciousness, and palpitations. The investigation about the plant has identified it to be HN. The initial evaluation at the ER revealed that the 77-year-old father of the family had clinical signs like meaningless speech, agitation, visual hallucinations, dilatation in both pupils, difficulty in movement, facial flushing, and respiratory distress. The results of this patient's ER examination was as follows: arterial blood pressure (BP): 90/60mm Hg; heart rate (K): 60/min; filiform pulse; oxygen saturation (SpO₂): 94%. The 67-year-old mother similarly had nausea, vomiting, palpitations, dry mouth, and agitation. This patient had 95/60mm Hg arterial blood pressure, 80/min heart rate, and SpO₂ was 98%. There were two other patients; a male and a female, both aged 37. These patients also had nausea, vomiting, and palpitations. The routine laboratory findings of the patients were normal. Considering this revealing clinical picture, we diagnosed the patients with poisoning related central anticholinergic syndrome (CAS). To monitor the patients closely, we transferred the patients to the intensive care

unit. We performed ECG, BP, and SpO₂ monitoring for each patient. We provided supportive breathing with oxygen masks (5L/min). We applied gastric lavage with nasogastric tube and gave each patient 1mg/kg of activated charcoal. Because the patients did not show any signs of physostigmine, we started a symptomatic treatment. Developing superficial breathing, we administered 3mg of intravenous (iv) midazolam which was followed by an endotracheal intubation. Next we started intermittent positive pressure ventilation (IPPV) with mechanical ventilator support. The arterial blood gas values of the patient was then hemodynamically stable. Achieving strong spontaneous respiration 24 hours after the intubation, the patient was extubated. Other patients received symptomatic treatment. With no further complaints and stable hemodynamics, the patients were discharged after 48 hours.

HN poisoning is a rare kind of poisoning in the literature. HN is reported to have caused central anticholinergic syndrome (SAS) due to the alkaloids it contains. Because of its anticholinergic properties, the intake of HN brings about central and peripheral symptoms (2). Central effects may, in turn, result in confusion, anxiety, delirium, hallucination, myoclonus, dysarthria, choreoathetosis, hyperactive deep tendon reflexes, convulsions, and coma. The peripheral anticholinergic effects that may surface are mydriasis, peripheral vasodilation, hyperpyrexia, tachycardia, urinary retention, decreased gastrointestinal motility, decreased secretions, and respiratory depression. In the treatment of patients with suspicion of intoxication, practitioners should ensure respiratory and circulatory continuity and start antidote treatment in case of gastrointestinal decontamination and other similar conditions that require such treatment (1).

Our patients had both central and peripheral anticholinergic symptoms. Because the signs and symptoms were typical, we were able to initiate the treatment without delay. We needed to provide mechanical ventilation support for the patient who developed respiratory failure and loss of consciousness. In such cases, patients should be monitored closely in terms of tachyarrhythmias and cardiac state, which may develop depending on the degree of vagal impact on the sinoatrial node. Physostigmine, an

acetylcholinesterase inhibitor, may be required in patients with dysrhythmia, significant hypertension, uncontrolled hyperpyrexia, convulsions, and coma (3). If physostigmine could not be found, practitioners should consider symptomatic treatment.

The oral intake of henbane may cause anticholinergic side effects. We believe that close follow-up, respiratory support, and symptomatic treatment may prevent mortality and morbidity resulting from the potentially life-threatening intoxication due to henbane intake.

Kind Regards

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Correspondence/İletişim

Ökkeş Hakan MİNİKSAR
İnönü Üniversitesi Tıp Fakültesi, Anesteziyoloji ve
Reanimasyon Anabilim Dalı, MALATYA, TÜRKİYE
E-mail: hminiksar@yahoo.com



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