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ANTICHOLINERGIC TOXIDROME DUE TO THORN APPLE SEED INGESTION IN AN ELDERLY COUPLE

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ABSTRACT

Introduction: Sudden onset of reduced consciousness, psychomotor agitation and mydriasis are all indicative of an anticholinergic toxidrome. It is important to note that numerous drugs, as well as certain herbs and plants, possess anticholinergic properties.

Case description: An 84-year-old female patient had sudden nocturnal onset of uncoordinated hand movements and altered mental status. Shortly after, the patient's 83-year-old husband developed symptoms of dysarthria, gait ataxia, vertigo, and delirium.

Conclusion: Anticholinergic syndrome consists of a combination of central and peripheral anticholinergic symptoms. Physostigmine given intravenously resulted in rapid reversal of symptoms. Thorn apple seeds had been accidentally ingested and were identified as the cause.

KEYWORDS

Anticholinergic toxidrome, physostigmine

LEARNING POINTS

- The clinical presentation of an anticholinergic toxidrome includes both central and peripheral symptoms such as altered consciousness, mydriasis, dry mucous membranes and skin, and tachycardia.
- Prompt recognition of anticholinergic toxidrome and the administration of physostigmine can lead to rapid reversal of symptoms and improved patient outcomes.
- Treatment with physostigmine is indicated when a patient with an agitated delirium does not respond adequately to titrated benzodiazepines.

INTRODUCTION

Sudden onset of reduced consciousness, psychomotor agitation and mydriasis are all indicative of an anticholinergic toxidrome. Anticholinergic syndrome consists of a combination of central and peripheral anticholinergic symptoms. The neurotransmitter acetylcholine acts as an

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agonist of muscarinic receptors present both centrally and peripherally[1]. Inhibition of cholinergic transmission can lead to the clinical signs.

Numerous drugs, as well as certain herbs and plants, possess anticholinergic properties and these substances can often lead to toxicity. A comprehensive medical history and medication are mandatory to identify any potential causative agents. Drugs known to induce anticholinergic symptoms include antiparkinsonians, antihistamines, antitussives, antidepressants, and antipsychotics. Many anticholinergic drugs and plants also possess other toxic effects that may obscure a true anticholinergic toxidrome.

CASE DESCRIPTION

An 84-year-old female patient, known to have a medical history of lumbar spinal stenosis and spinocerebellar ataxia type 3 (SCA3), had a sudden nocturnal onset of neurological symptoms, characterised by uncoordinated hand movements, and altered mental status. Her husband promptly sought medical assistance. At the emergency department (ED), neurological examination revealed psychomotor agitation, a decreased level of consciousness with a Glasgow coma scale of E4M5V1, unresponsive dilated pupils and stereotypical grasping movements of both arms that were not rhythmic or jerking. Her symptoms fluctuated over time. Computed tomography and angiography of the brain showed no abnormalities. Point-of-care ultrasound showed urinary retention, which was managed with the placement a Foley catheter.

The patient's 83-year-old husband developed progressively worsening symptoms of dysarthria, gait ataxia, vertigo, and delirium during his stay at the ED. Examination revealed reactive dilated and isocoric pupils, dry flushed skin and urinary retention.

After administering 2 mg of physostigmine intravenously, a significant recovery was observed, and all symptoms resolved within a few minutes. In response to the escalating delirium symptoms observed in the spouse, physostigmine was subsequently administered, resulting in a similarly rapid recovery.

We identified the black seeds as thorn apple seeds. Afterwards, the male patient remembered to have put some mixed nuts in a bowl the previous evening, which they both ate. The black seeds were also kept in this bowl, with the intention of growing the distinctive apple seed flowers the following spring.

DISCUSSION

Sudden onset of reduced consciousness, psychomotor agitation and mydriasis are all indicative of an anticholinergic toxidrome. The differential diagnosis for the observed symptoms is broad and may include drug overdose, thyrotoxicosis, accidental hyperpyrexia, encephalitis, brainstem lesions, epilepsy, and neuroleptic malignant syndrome, particularly in elderly patients.

Central nervous system anticholinergic activity is manifested by altered (mostly decreased) consciousness, convulsions, hyperpyrexia, and mydriasis that does not respond to light. Peripheral symptoms mostly result from autonomic nervous dysregulation and include dry mucous membranes and skin, tachycardia, urinary retention, and decreased bowel movements. It is important to note that focal neurologic signs do not occur as part of an anticholinergic toxidrome and should alarm the treating physician to consider alternative causes.

In case of delirium, an anamnesis with the next of kin can have additional value. In our patients, the children confirmed that the couple lived independently and exhibited no signs of depression or intentions of self-harm. At their parents' home they looked for the possible causative agent. They found small black seeds on two chairs in the living room and in a small bowl mixed with edible nuts standing on a table in between.

Physostigmine can serve as a diagnostic and therapeutic agent, particularly in a patient with agitated delirium not adequately managed with titrated benzodiazepines[2,3].

Physostigmine, a cholinesterase inhibitor used therapeutically in patients with anticholinergic toxicity, produces reversible inhibition of acetylcholinesterase and accumulation of acetylcholine. The increased concentration of acetylcholine overcomes the postsynaptic muscarinic receptor blockade induced by anticholinergic agents[2,4]. Unlike quaternary amines such as neostigmine, physostigmine is a tertiary amine that can cross the blood–brain barrier, enabling its effects within the central nervous system[3].

The recommended dosage of physostigmine is 0.04 mg/kg body weight[3]. Administration of the drug should be performed slowly (evenly over 3 minutes) while monitoring heart rhythm and blood pressure. Treatment should only be done in a monitored environment capable of providing full resuscitative care. Furthermore, it is essential to rule out any cardiac conduction abnormalities prior to administration[3]. The effects typically become evident within 30 seconds to 5 minutes following intravenous administration, and its action persists for approximately 90 to 120 minutes.

As in our patients, prompt recognition of anticholinergic toxidrome and the administration of physostigmine can lead to rapid reversal of symptoms and improved patient outcomes.

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