

## A Case of Acute Iatrogenic Atropine Poisoning

### *Intoxicación aguda por atropina de causa iatrogénica*

#### To the Editor,

A 75-year-old man came to our emergency room for syncope. He reported no adverse reactions to medicines but in previous weeks had presented presyncope attributed to a vasovagal reaction, rather than transitory ischemic accidents, and which had been scheduled for study. In emergency room, an electrocardiogram (ECG) recorded complete atrioventricular block, 24 bpm escape rhythm and 120 ms QRS. Intravenous perfusion of isoprenaline was initiated and the patient presented rhythmic narrow QRS tachycardia at 150 bpm, which was poorly tolerated. We then implanted a temporary pacemaker. During the procedure, the patient developed extreme bradycardia with 80/35 mmHg blood pressure and Stokes-Adams syndrome, which was treated with 1 mg IV atropine, producing a partial response. This was repeated twice with 1 mg IV each time, at 3 min intervals. The patient remained asymptomatic after adequate pacemaker rhythm had been achieved, with normal cardiorespiratory status. At 30 min post-procedure, the patient reported an extremely dry mouth and at 1 h presented dyspnea and psychomotor agitation that intensified and required 1.5 mg IV midazolam. He responded within a few minutes, remaining with his eyes closed and being unable to make any verbal response, despite painful stimuli. He presented a posture resembling decerebration, cutaneous reflex responses of the bilateral plantar extensor, and mydriatic, non-reactive pupils with conserved corneal and oculocephalic reflexes. The patient had extreme reddening and congestion of the upper thorax, neck and face, with generally dry skin. Axillary temperature was 38 °C and groin fold temperature 36 °C. He was hemodynamically stable, with ECG monitoring in pacemaker rhythm and 98% oxygen saturation (FiO<sub>2</sub> 0.4 in Ventimask®). Urgent analysis revealed nothing significant (hemogram with three normal series and normal coagulation; glucose level, renal function, ions and venous gasometry all within normal ranges).

His clinical condition led us to consider various diagnoses: a) iatrogenic pneumothorax following puncture of the right subclavian vein used to establish central vascular access; b) cardiac taponade, discounted following echocardiography; c) pulmonary thromboembolism; or d) thrombosis in vena cava superior, both justifiable given recent central vein manipulation, and e) stroke, most probably embolic-ischemic, related to energetic maneuvers close to the neck. Urgent cranial and chest computerized tomography found nothing to justify the patient's condition. Finally, we considered diagnosing a state of coma secondary to atropine poisoning. As physostigmine was unavailable, we administered 0.5 mg IV neostigmine.<sup>1,2</sup> All signs and symptoms caused by alteration of the peripheral nervous system reversed in a few minutes and, moreover, his Glasgow coma scale score improved; in 48 h the patient had fully recovered.

In the emergency room, coma is a common syndrome. Not infrequently, following initial evaluation of signs and specific

complementary tests, we conclude it may be caused by drug poisoning.<sup>3</sup> The most commonly found drug groups are: anticholinergics, sympathomimetics, tricyclic antidepressants, antihistaminics, antipsychotics and antiparkinson agents. All block the muscarinic receptors (cholinergic block).<sup>3,4</sup> Our patient presented data indicating intense cholinergic block with danger of imminent death, secondary to acute atropine poisoning, usually described in an accidental context: drinking leaf tea, using anticholinergic eye drops or intravenous use in addicts.<sup>3</sup> Treatment of severe atropine poisoning includes measures of general support plus the administration of anticholinesterase agents as an antidote. The preferred drug is physostigmine, with a 0.5-2 mg IV dose administered slowly,<sup>1,2,4</sup> followed at 15 min by an IV infusion at 4 mg/h<sup>1,2</sup> or by repeating the initial dose every 10 min as required,<sup>4</sup> and monitoring respiratory function, level of consciousness and ECG.<sup>1,2,4</sup> Physostigmine is a tertiary amine that acts on the central and peripheral nervous system. Neostigmine or pyridostigmine, quaternary amines, do not cross the hematoencephalic barrier and only act in the peripheral nervous system.<sup>4,5</sup> All of this explains our patient's slow detoxification process, which ended when the atropine was completely metabolized. In the literature, we find reports of patients whose condition has reversed without the use of antidotes, but with respiratory and hemodynamic support, after 48 h.<sup>3</sup> The interest in our case is justified by the habitual use of atropine in cardiology, both in diagnosis and therapy. We have described a severe anticholinergic syndrome, which is highly infrequent but occurred in a common clinical context, that we should be prepared for and know how to treat adequately.

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