Serotonin syndrome

To the editor:

I would like to respond to 2 issues raised in the excellent Toxicology report by Drs. Ip and Renouf regarding serotonin syndrome.[1] The first is the decision to paralyze the patient to stop ongoing seizure-induced rhabdomyolysis. While neuromuscular blockade will certainly limit further myoglobin release, it removes the ability to clinically assess and treat potentially harmful ongoing electrical seizure activity. In the absence of continuous EEG monitoring, more aggressive antiseizure treatment with drugs like barbituates and propofol might be more prudent. Phenytoin should be used cautiously in overdose patients and not at all in those who may have ingested tricyclic antidepressants (TCAs) because phenytoin, like the TCAs, is a sodium channel blocker that may provoke lethal dysrhythmias, including ventricular tachycardia. Hyperthermia can be treated with aggressive external cooling, while the mainstay of therapy for rhabdomyolysis, as the authors indicated, is high volume saline infusion (although urine alkalinization may increase myoglobin solubility and enhance renal clearance).

The second important issue is the irrelevence of urine toxicology screens, which are often performed in overdose patients. While these may provide useful information for a subsequent psychiatric assessment (if it is important to know what drugs the patient is using), "tox screens" have almost no role in the emergency department. In a patient like the one described, with a documented history of benzodiazepine and codeine use, screening for these drugs is not helpful, since they will, of course, be present and clinically insignificant. The presence of cannabis on the screen is similarly irrelevent, as it would never change patient management, and sympathomimetics such as cocaine or amphetamines are diagnosed clinically based on a characteristic toxidrome.

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Reference

1. Ip A, Renouf T. Serotonin syndrome due to an overdose of moclobemide? CJEM 2002;4(2):98-101.

[One of the authors responds:]

Dr. Hadley is correct in saying that paralytic agents interfere with the ability to clinically assess ongoing seizure activity. In this case, however, the patient's persistent muscular rigidity without tonic–clonic movement made it impossible to determine whether she was seizing, even prior to paralysis. Continuous EEG monitoring would have been ideal, but was unavailable. Under the circumstances, we felt it prudent to administer vecuronium to prevent rhabdomyolisis, hyperthermia and metabolic acidosis related to ongoing muscular activity. As Dr. Hadley suggests, thiopental or propofol may have been helpful adjuncts.

We share Dr. Hadley's opinion regarding the limited utility of drug screening, which is well established in emergency practice.[1] Unfortunately, consulting services are fond of ordering a large number of tests, as happened in this case. Our general practice is to base management on clinical toxidromes, to order ASA and acetaminophen levels relatively routinely, and to check tricyclic levels occasionally. Other drug screening seldom influences management.

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Reference

1. Schiller MJ, Shumway M, Batki SL. Utility of routine drug screening in a phychiatric emergency setting. Psychiatr Serv 2000;51(4):474-8.

https://doi.org/10.1017/S1481803500006333 Published online by Cambridge University Press