Case Report

Meperidine-induced serotonin syndrome in a susceptible patient

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We present a patient with a history of clomipramine-induced serotonin syndrome 5 yr prior who developed serotonin syndrome after a single dose of meperidine. This report heightens appreciation of population at risk and also recognition of potential toxicity in meperidine.

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Meperidine is one of the most commonly used opioids to relieve acute pain.

Through blocking presynaptic serotonin reuptake, meperidine may induce or interact with other serotonergic agents and result in serotonin hyperstimulation. We present a patient with a history of clomipramine-induced serotonin syndrome 5 yr prior who developed serotonin syndrome after a single dose of meperidine.

Case report

This 41-yr-old male presented for surgical management of his right clavicle fracture. Five years ago, he developed a life-threatening episode which included palpitations, chest tightness, chills, limb numbness, hypertension (BP: 181/116 mm Hg), tachycardia (HR: 151 beats min⁻¹), dilated pupil size (5.5 mm), and hyperthermia (37.9°C), after taking clomipramine for premature ejaculation. He was treated with supportive care and recovered without any sequelae.

On admission, he had been taking bisoprolol and amlodipine for hypertension. The laboratory tests were unremarkable except hyperlipidaemia. Anaesthesia was induced with i.v. fentanyl 150 μg, propofol 150 mg, glycopyrrolate 0.2 mg, and rocuronium 50 mg. After tracheal intubation, anaesthesia was maintained with pure oxygen and desflurane as required. At the end of procedure, neuromuscular block was reversed and the tracheal tube was removed uneventfully. He received i.v. meperidine 30 mg for pain relief in the post-anaesthesia care unit. One minute after the injection, he developed dizziness, nausea, numbness of his limbs, and palpitations. Subsequently, he became nervous, sensed chest pressure, and difficult breathing. Mild tremors along with a few clonic jerks were also noted in both feet. Hypertension (BP: 150/90 mm Hg), tachycardia (HR: 151 beats min⁻¹), tachypnoea (23 bpm), and elevated body temperature (37.2°C) were also found. He was treated with i.v. labetalol 12.5 mg to attenuate the haemodynamic changes. After 30 min of close observation, he reported relief of all the symptoms.

Discussion

Serotonin syndrome is a toxic state resulting from serotonin hyperstimulation at postsynaptic neurone. This adverse drug reaction occurs most commonly in patients who are using single or multiple medications that increase postsynaptic serotonin levels. Furthermore, inherited and acquired deficits in peripheral serotonin metabolism may contribute to the development of serotonin syndrome. Hypertension, atherosclerosis, and hyperlipidaemia are all associated with a reduction in endothelial MAO activity and thus a reduced capacity to metabolize serotonin. The presence of predisposing factors in peripheral serotonin metabolism deficit coupled with the use of serotonergic drugs may increase the likelihood of clinical presentation.

For diagnosis, the Hunter serotonin toxicity criteria is now a preferred proposal. These rules demonstrate that, in the presence of a serotonergic agent, if spontaneous clonus

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is present, then serotonin toxicity may be reliably diagnosed; else if inducible clonus is present with agitation or diaphoresis serotonin toxicity may be reliably diagnosed. In this case, the diagnosis of serotonin syndrome was suggested by his previous episode, and symptoms and signs with the abrupt onset after meperidine administration.

Given the large number of available serotonergic agents for the treatment of psychiatric disorders and the possible interaction with meperidine, this report heightens appreciation of population at risk and also recognition of potential toxicity in meperidine.

References

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