No specific evidence-based recommendations have been issued on the treatment of spinal cord infarction. The use of fibrinolysis is limited since the exact time of symptom onset is uncertain on many occasions, leading to diagnostic delays that leave patients outside the treatment window. To date, only isolated cases have been reported of successful fibrinolysis.[8]

The prognosis of spinal cord infarction is poor: only 11%-46% of patients are able to walk independently after the event.[9] The main risk factors for poor functional prognosis include initial severity, female sex, old age, and lack of improvement within 24 hours of symptom onset. Although prognosis is usually poor, functional recovery may occur several months or even years after the lesion, which underscores the importance of early, long-term rehabilitation treatment.

In conclusion, while spinal cord infarction is an infrequent complication of bronchial artery embolisation, physicians should be aware of this entity and be familiar with the associated anatomical and technical risk factors; prevention is essential given the poor long-term functional prognosis and associated morbidity. Long-term rehabilitation treatment may achieve substantial functional improvement, even several months or years after the event.

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Opsoclonus-myoclonus syndrome secondary to duloxetine toxicity☆

Síndrome de opsoclono-mioclono secundario a intoxicación por duloxetina

Dear Editor:

Opsoclonus-myoclonus syndrome (OMS) is extremely rare, with incidence estimated at 0.18 cases/1 000 000 person-years.[1] Clinically, it is characterised by the presence of 3 symptoms: opsoclonus, myoclonus, and ataxia. Opsoclonus is defined as involuntary, rapid, multidirectional conjugate saccadic eye movements. Myoclonus most frequently affects the trunk or limbs, and is usually postural or induced by movement. Ataxia may be caused by severe myoclonus or by cerebellar damage. Other associated symptoms include cognitive dysfunction, behavioural alterations, encephalopathy, cranial nerve alterations (cranial nerves IV, V, and VI), and seizures.[2]

OMS in children is well characterised; the most frequent aetiology is paraneoplastic, in the context of neuroblastoma.[3] According to the literature, the most frequent aetiologies in adults are paraneoplastic, infectious, or idiopathic, with metabolic or toxic causes being exceptional.[4]

We present a case of OMS secondary to duloxetine toxicity.

The patient was a 44-year-old woman with history of anxiety and depression, for which she was being treated with lorazepam. She was referred by the emergency department after presenting blurred vision, involuntary movements, and visions of ''hooded people'' in her home, who the patient

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believed had poisoned her; symptoms began upon awakening. Upon arrival, she was haemodynamically stable, but presented tachycardia. In the assessment of higher cognitive functions, she was alert, oriented to time and person but disoriented to place, and inattentive; she spoke little but showed no signs of dysphasia, with preserved comprehension. One reiterative utterance was noted (''I've been poisoned''). In the cranial nerve examination, we observed mydriasis with impaired pupillary light reflex, and rapid, multidirectional, conjugate eye movements compatible with opsoclonus. Finally, she presented action myoclonus in all 4 limbs and the trunk (see Supplementary Material for video). Emergency complementary tests included a complete blood count and biochemistry profile, venous blood gas analysis, urine analysis, urine toxicology, and brain MRI. All results were normal or negative, with the exception of neutrophilic leukocytosis and positive results for benzodiazepines in the urine. We also performed an emergency lumbar puncture; cerebrospinal fluid biochemistry showed no significant alterations, and samples were taken for a microbiology study, oligoclonal banding, an anatomical pathology study, and antineuronal and onconeuronal antibody testing. The patient was admitted with a diagnosis of OMS associated with delusional ideation and visual hallucinations. We started empirical treatment with high-dose corticosteroids (intravenous methylprednisolone dosed at 1 g for 5 days). We also screened for a primary tumour: results for tumour markers were negative, and a mammogram and a chest-abdomen-pelvis CT scan revealed no signs of malignancy. The serology study, anatomical pathology study, and antineuronal and onconeuronal antibody tests of cerebrospinal fluid samples all returned negative results.

Five days after admission, symptoms progressively improved and eventually resolved; the patient acknowledged having consumed 3 packages of duloxetine 30 mg the night prior to symptom onset, with suicidal intent. We tested for duloxetine in the plasma, finding a concentration of 371 ng/mL (therapeutic range, 20-80).

Toxic aetiology of OMS is extremely rare. The literature includes cases of OMS induced by amitriptyline, cocaine, lithium, phenytoin, phenelzine, ciclosporin, ipilimumab/nivolumab, cefepime, and venlafaxine.[5—13] To our knowledge, this is the first case of OMS associated with duloxetine toxicity, and the second case associated with a serotonin-norepinephrine reuptake inhibitor (the first case was reported in a patient receiving venlafaxine). The pathophysiological mechanisms explaining OMS of toxic aetiology are unclear. In general, the syndrome is thought to originate from dysfunction of the omnipause neurons of the nucleus raphe interpositus[2] or disinhibition of the fastigial nucleus in the cerebellum[14]; however, these structures are neither serotonergic nor norepinephrinergic. Necpál and Skorvanek[13] suggest that the case they report of OMS associated with venlafaxine toxicity may have been explained by serotonin syndrome, as the patient presented mental, neuromuscular, and autonomic alterations. Our patient also presented alterations in all 3 of these domains: mental (psychosis), neuromuscular (opsoclonus/myoclonus), and autonomic (tachycardia, mydriasis); this supports the hypothesis proposed by Necpál and Skorvanek.[13] Additionally, while increased production of catecholamines is observed in patients with neuroblastoma,[15] there is very little evidence that a norepinephrinergic effect is involved in the pathogenesis of OMS. Therefore, while we cannot completely rule it out, norepinephrinergic origin seems less likely in these cases.

In conclusion, while it is very infrequent, toxic aetiology is highly relevant in the aetiological diagnosis of OMS. This is the second case of OMS associated with serotonin-norepinephrine reuptake inhibitors; serotonin syndrome may have played a role in pathogenesis.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.nrl.2020.05.009.

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