Commentary

The opsoclonus–myoclonus syndrome (OMS) is a pluriethiological neurological disorder, seldom encountered in the medical practice, affecting 1 in 10 000 000 people annually, producing about 60 infections worldwide every year. In children, the most frequent cause of OMS is the neuroblastoma, in more than 50%

of the cases, appearing precociously between 1.5 and 2 years. In adults, it is associated as a paraneoplastic manifestation during the evolution of the breast neoplasm, small-cell lung cancer, kidney or pancreas carcinoma or gall bladder, but also with the autoimmune diseases, the Hashimoto disease, in the presence of autoantibodies oriented against neurons—anti-neuronal antibody type 2—or the cerebellar Purkinje cells.[1] Other causes of the OMS may be streptococcal infections,[2] the celiac disease, hydroelectrolyte imbalance, cerebral anoxia or cerebral hemorrhage. The infectious causes involved in the appearance of the OMS are frequently viral: Coxsackie B2, B3, Epstein Barr, the Saint Louis encephalitis virus, hepatitis C virus, rubella, mumps, cytomegalovirus, the Whipple disease, the herpes simplex virus,[3] HIV,[3] the West Nile virus, but there are also descriptions of infections with *Mycoplasma pneumonia*,[4] Rickettsia or Borrelia burgdorferi as triggering factors of the OMS. Among the medications associated with OMS, amitriptyline, haloperidol and diazepam have been quoted. In the article published by Verma R, Kumar S, Biyani S, Singh A "Opsoclonus Myoclonus Syndrome induced by phenytoin intoxication" in Journal of Neurosciences in Rural Practice the association with phenytoin is described for the first time.[5]

The OMS is to be translated by opsoclonus-involuntary, rapid, multivectorial and conjugated movements of the eyes, even during the sleep, without concomitant modifications of the visual field; myoclonus – short, involuntary movements of the torso, with or without ataxia or other cerebellar signs, sleepiness or speech disorders – dysphasia, strabismus or vomiting. The traditional treatment of OMS is with ACTH (adrenocorticotropic hormone), corticotherapy – prednisone or methylprednisolone – in high doses of 500 mg – 2 g/day for 3-5 days, intravenous immunoglobulin for 3-5 days and weekly plasmapheresis for 6 weeks. In the cases associated with the administrations of anti-epileptic drugs, it is necessary to change the therapy in order to solve the neurological episode. The cases which are refractory to the treatment are treated with chemotherapy – cyclophosphamide (Cytoxan), the association of cyclophosphamide and dexamethasone, azathioprine or cyclosporine. The bio-therapy with Rituximab[6] which ties the antigen CD20 of the mature B cells by reducing the level of B cells within the cerebrospinal fluid proved to be beneficial in the cases of OMS appeared as paraneoplastic manifestations. The cases associated with tumoral affections require surgical interventions—the extirpation of the tumor while the OMS appeared in the evolution of viral infections is generally in remission once the infectious trigger regresses or concomitant to the administration of antibiotics in the case of the Lyme disease, for example, ceftriaxone sodium.

The symptomatic treatment with clonazepam or trazodone is efficient in ameliorating the irritability and sleeping disorders.

According to the etiology, the prognostic of OMS is variable but it is admitted that the long-term evolution is associated with neurological sequels in 80% of cases—frequently manifested by speech and behavior disorders, which can be controlled by a immunomodulatory therapy.[1]

We consider that every case of OMS published contributes to the deepening of the etiology of this syndrome, the precocious recognition of the diagnosis and the prompt initiation of the treatment.

This case expands the spectrum of OMS etiology and has an important impact in the long-term management of patients with phenytoin treatment.

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References

1. Brunklaus A, Pohl K, Zuberi SM, de Sousa C. Outcome and prognostic features in opsoclonus-myoclonus syndrome from infancy to adult life. Pediatrics 2011;128:e388-94.
2. Candler PM, Dale RC, Griffin S, Church AJ, Wait R, Chapman MD, *et al*. Post-streptococcal opsoclonus-myoclonus syndrome associated with anti-neuroleukin antibodies. J Neurol Neurosurg Psychiatry 2006;77:507-12.
3. Klaas JP, Ahlskog JE, Pittock SJ, Matsumoto JY, Aksamit AJ, Bartleson JD, *et al*. Adult-onset opsoclonus-myoclonus syndrome. Arch Neurol 2012;69:1598-607.
4. Huber BM, Strozzi S, Steinlin M, Aebi C, Fluri S. Mycoplasma pneumoniae associated opsoclonus-myoclonus syndrome in three cases. Eur J Pediatr 2010;169:441-5.
5. Verma R, Kumar S, Biyani S, Singh A. Opsoclonus myoclonus syndrome induced by phenytoin intoxication. J Neurosci Rural Pract 2014;5:S109-10.
6. Pranzatelli MR, Tate ED, Verhulst SJ, Bertolone SJ, Bhatla D, Granger M, *et al*. Pediatric dosing of rituximab revisited: Serum concentrations in opsoclonus-myoclonus syndrome. J Pediatr Hematol Oncol 2010;32:e167-72.

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