complex auditory hallucinations and CSE, which seems to be an interesting option for further research in the basic field.

References

1. Gasse Ch, Derby LE, Vasilakis C, Hershel J. Incidence of firsttime idiopathic seizures in users of tramadol. Pharmacotherapy. 2000;20:629-34.
2. Gardner JS, Blought D, Drinkard CR, Shatin D, Anderson G, Graham D, et al. Tramadol and seizures: A surveillance study in a managed care population. Pharmacotherapy. 2000;20: 1223-431.
3. Spiller HA, Gorman SE, Villalobos, Benson BE, Ruskosky DR, Stancavage MM, et al. Prospective multicenter evaluation of tramadol exposure. J Toxicol Clin Toxicol. 1997;35:361-4.
4. Tobias JD. Seizure after overdose of tramadol. South Med J. 1997;90:826-7.
5. Potschka H, Friderich E, Loscher W. Anticonvulsant and proconvulsant effects of tramadol, its enantiomers and its M1 metabolite in the rat kindling model of epilepsy. Br J Pharmacol. 2000;131:203-12.
6. Jick H, Derby LE, Vasilakis C, Fife D. The risk of seizures associated with tramadol. Pharmacotherapy. 1998;18: 607-11.
7. Labate A, Newton MR, Vernon GM, Berkovic SF. Tramadol and new-onset seizures. Med J Aust. 2005;182:42.
8. Kahn LH, Alderfer RJ, Graham DJ. Seizures reported with Tramadol. JAMA. 1997;278:1661.
9. Jovanovic V, Martinovic Z, Nesic N. Seizures associated with intoxication and abuse of tramadol. Clin Toxicol. 2006;44: 143-6.
10. Mason BJ, Blackburn KH. Possible serotonin syndrome associated with tramadol and sertraline coadministration. Ann Pharmacother. 1997;31:175-7.
11. Keeley PW, Foster G, Whitelaw L. Hear my song: auditory hallucinations with tramadol hydrochloride. BMJ. 2000;321:1608.
12. Meseguer Ruiz VA, Navarro López V. [Auditive and visual hallucinations secondary to tramadol administration]. An Med Interna. 2003;20:493.
13. Ripple MG, Pestaner JP, Levine BS, Smialek JE. Lethal combination of tramadol and multiple drugs affecting serotonin. Am J Forensic Med Pathol. 2000;21:370-4.
14. Van Puijenbroek EP, Verduijn MM, Koning-Verest IFd. Urineretentie en pseudo-hallucinaties tijdens het gebruik van tramadol. REB-bulletin. 1995;10:6-7.
15. Moreno-Izco F, Ruibal M. Myoclonic seizures induced by tramadol in patients with juvenile myoclonic epilepsy. Rev Neurol. 2006;43:255.
16. Bottaro FJ, Martinez OA, Fernandez-Pardal MM, Bruetman JE, Reisin RC. Nonconvulsive status epilepticus in the elderly: A case-control study. Epilepsia. 2007;48:966-72.

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Autoimmune vestibulopathy associated with autoreactive antibodies and parotid involvement

Vestibulopatía autoinmunitaria asociada con anticuerpos autorreactivos y afección parotídea

Dear Editor:

Autoimmune vestibulopathy is a rare and treatable condition characterised by recurrent episodes of vertigo or progressive clinical signs of instability with clinical data of vestibular hypofunction.[1,2] Its early recognition makes it possible to introduce immunosuppressive treatment to stop its progression.[2,3] We present a patient with autoimmune vestibulopathy and radiological findings of parotid condition.

The patient was a 52-year-old male with no personal or family history of interest, with a progressive condition of tinnitus and hearing loss of 3 years' evolution. For the past year he had suffered unsteady gait sensation, with a tendency to veer to the right. He did not report a sensation of object rotation.

The neurological examination revealed nystagmus after head shakes, corrective saccadic movements with head turns and a decrease in dynamic visual acuity compared to static.

The complementary tests were: normal Ss, normal biochemical results, negative syphilis, normal thyroid hormones, normal vitamin B12, positive ANA, fine structure anti-Golgi, positive Ro/SSA antibodies, positive transglutaminase antibodies; the rest of the results were normal or negative. The coronal sections of cranial magnetic resonance imaging showed enlarged parotid glands with small millimetric hyperintense images that could correspond to small cysts or acini, plus a more prominent one with a lobular aspect in the right parotid gland, with a size of 1.3 cm. This finding could be related to an autoimmune disease (fig. 1).

Prednisone treatment was prescribed, yielding clinical stabilisation and partial improvement of instability and tinnitus. The treatment was suspended after 4 months due to side effects, after which the patient suffered a clinical exacerbation. Immunomodulatory treatment was reintroduced, resulting in clear clinical improvement.

Autoimmune vestibulopathy is a rare entity but it must be recognised, due to the importance of early immunosuppressive treatment.[2,3] It may be the expression of a uniquely vestibular condition or be part of the clinical context of a systemic autoimmune disease (SLE, ulcerative colitis, Cogan's syndrome). Autoantibodies against the inner ear have been described, but it was not possible to determine them in our case.[3,4] In our opinion, clinical suspicion of autoimmune vestibulopathy condition should lead to immunomodulatory treatment, possibly with steroids, to prevent clinical progression and the disability that it generates. The possibility of autoimmune vestibulopathy should be considered in cases of instability and recurrent vertigo. Early treatment is essential to prevent irreversible vestibular hypofunction.

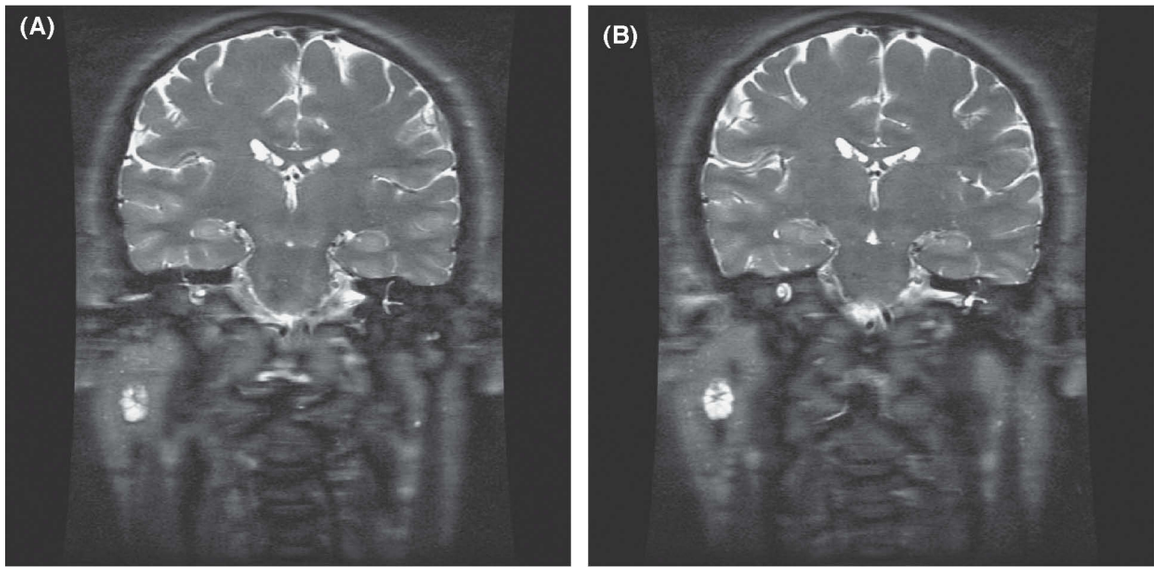

Figure 1 T2-weighted coronal sections of MRI scan showing enlarged parotid glands with small millimetric hyperintense images that may correspond to small cysts or acini, plus a more prominent one (1.3 cm) with a lobular aspect in the right parotid gland.

References

1. Zingler VC, Cnyrim C, Jahn K, Weintz E, Fernbacher J, Frenzel C, et al. Causative factors and epidemiology of bilateral vestibulopathy in 255 patients. Ann Neurol. 2007;61:524-32.
2. Deutschländer A, Glaser M, Strupp M, Dieterich M, Brandt T. Immunosuppressive treatment in bilateral vestibulopathy with inner ear antibodies. Acta Otolaryngol. 2005;125:848-51.
3. Schüler O, Strupp M, Arbusow V, Brandt T. A case of possible autoimmune bilateral vestibulopathy treated with steroids. J Neurol Neurosurg Psychiatry. 2003;74:825.
4. Zingler VC, Cnyrim C, Jahn K, et al. Causative factors and epidemiology of bilateral vestibulopathy in 255 patients. Ann Neurol. 2007;61:524-32.

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Caudal nucleus haemorrhage due to dental anaesthesia

Hemorragia de núcleo caudado por anestesia dental

Dear Editor:

The most common causes of intraparenchymatous cerebral haemorrhage are arterial hypertension (HT), coagulopathy, amyloid angiopathy, cerebral malformations, tumours and strokes with hemorrhagic transformation. However, there are other less common aetiologies in which haemorrhage has a specific trigger, such as substances with sympathomimetic effects on the cardiovascular system; consequently, there have been cases caused by diet pills containing phenylpropanolamine,[1] by amphetamines and methylphenidate[2] for the treatment of attention deficit disorder and by pseudoephedrine[2,3] in nasal decongestants.

The purpose of our letter is to present the case of a patient who suffered a cerebral haemorrhage, probably related to dental anaesthesia.

The patient was a 41-year-old female, with no cardiovascular risk factors. The only relevant personal history was that she was in treatment with paroxetine for depressive syndrome.

Two days before being seen by our department, she underwent a dental intervention for the extraction of several teeth, in which nerve block anaesthesia was applied in both mental foramina using a solution of 90mg lidocaine hydrochloride monohydrate and 18mg noradrenaline tartrate. She immediately suffered sudden headache and vomiting. Her arterial pressure at that time was 140/70mmHg. Due to the persistence of headache, she decided to go to the emergency service. The only finding on the neurological and physical examination was neck rigidity. Neuroimaging studies were performed (computed tomography scan and brain magnetic resonance) (figs. 1 and 2), revealing haemorrhage in the head of the left caudate nucleus. This was open to the ventricles, with minimal dilatation of the temporal horns. Laboratory analyses, electrocardiogram and chest radiograph were normal. Coagulopathy was ruled out by a normal coagulation study, and angiography showed no vascular malformations that would justify a cerebral haemorrhage.