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Sleep disorders and restless legs syndrome in an adult with Asperger's disorder, a case report

Alteraciones del sueño y síndrome de piernas inquietas en un adulto con síndrome de Asperger, a propósito de un caso

Dear Editor:

Asperger syndrome[1] is a pervasive developmental disorder (PDD), whose prevalence ranges from 0.1 to 3.6 cases / 1,000 children, with predominance in males. It is characterised by restrictive and repetitive interests and altered social relations.[2] Common symptoms are motor clumsiness, difficulty in planning, sequencing of motor tasks and coordination alterations.[3] It is often associated with other neuropsychiatric syndromes: anxiety disorder, obsessive-compulsive disorder, bipolarity, depression, attention deficit and hyperactivity disorder, Gilles de la Tourette syndrome, epilepsy, movement and sleep disorders.[4] Disturbances of sleep architecture are usually functional, in sleep stability and efficiency; structural alterations are not frequent.[5] Melatonin has been used to treat such disturbances with moderate effectiveness.[6]

Restless legs syndrome (RLS) is characterised by a restlessness that requires moving the legs to relieve the symptoms, which worsen in the evening. Its incidence in the general population is 5-10%.[7] Some 80% of patients with RLS have periodic leg movements (PLM) that hinder sleep.[8] The delay time from inception to diagnosis may be elevated.[9] The first-line drugs are dopamine agonists and L-Dopa, although benzodiazepines and anticonvulsants have also been used; the most effective include gabapentin, clonidine, opioid agonists and beta blockers.[8,9] The typical symptoms of Asperger syndrome, RLS and sleep disorders overlap, thus complicating diagnosis and treatment.

We present the case of a 25-year-old male who attended consultation due to insomnia and hypothymia. At age 16 he was diagnosed with Asperger syndrome and depression, after his academic performance dropped and he complained of fatigue, sadness, hyphedonia, conciliation insomnia and mnemonic and concentration difficulty. Serotonergic antidepressant treatment was initiated, with partial improvement. From age 16 to age 20 he consulted various specialists for apathy, nervousness and conciliation insomnia, receiving antidepressant treatments, benzodiazepines and melatonin. At age 21 he attended consultation because the depressive manifestations remained, as did the difficulty in sleeping from discomfort and an internal feeling of tension in the lower extremities, which appeared at bedtime and which forced him to wander about, preventing relaxation. No temporal relationship was found between symptoms (suggestive of RLS) and the establishment of drug treatment. Physical and analytical examinations were normal. Magnetic resonance imaging, electroencephalogram, magnetoencephalography and cranial spectroscopy did not detect any significant abnormalities. Polysomnography detected, repeatedly, in two tests carried out at 1-year intervals, unstable sleep, very low percentage of deep sleep and a small proportion of REM sleep (table 1). PLMs were also observed. Treatment with escitalopram, 20 mg / day, and alprazolam, 1 mg / day, were started, with partial remission of RLS and depressive symptoms. Subsequently, gabapentin was added up to 600 mg / day, with only slight

Table 1 Polysomnographic recordings from 2004 and 2005

Polysomnography	2004	2005
Total sleep (%)*		
Phase 1	15.6	80
Phase 2	48.5	
Phase 3	18.7	11
Phase 4	17.2	3
REM	0	6
Sleeping time (min)	421	430
Sleep latency (min)	11	56
Sleep efficiency (%)	84	23.46
Awakenings (n)	7	Numerous
Intra-sleep awake time (min)	39	66

*Normal conditions of total sleep: NREM 1 (4%), 2 (25%), 3 and 4 (45%) and REM (25%).

improvement in the night symptoms and the insomnia. Insomnia in this patient was multifactorial (Asperger syndrome, depressive disorder, RLS and PLM) and was causally related to daytime symptoms of fatigue, sleepiness, decreased initiative and secondary abandonment of daily living activities. There are few studies investigating the motor symptoms in patients with Asperger syndrome and its relationship with RLS has not been specifically described. However, both diseases present dysfunctions in the serotonin and dopamine systems, which could point to a common pathophysiology. In patients with pervasive developmental disorders, sleep disorders are common from infancy[5]. Brain activity during sleep contributes to memory consolidation and cerebral maturation.[10] Both REM and NREM sleep and their proper alternation are involved in the consolidation and maintenance of memory;[8] although a correct declarative memory can be maintained in the exclusive presence of NREM, efficiency is less.[8] Patients with Asperger syndrome often present mixed insomnia and decrease of the overall duration, early awakening, daytime drowsiness[11] and, occasionally, parasomnias, PLM and nocturnal breathing difficulties.[12] There are few studies of sleep structure in Asperger syndrome in adults and their results are not consistent. A decrease of the duration of phase III and IV NREM sleep has been observed, as has an increase of K complexes in stage II, a decrease of REM latency and signs of altered oculomotor activity in this phase.[13] In this case, the most significant polysomnographic finding was the absence of REM sleep, which had not been described previously in patients with Asperger syndrome. It points to the primary nature of polysomnographic abnormalities in this patient, thus reinforcing the hypothesis that depression could be, in part, secondary to sleep disturbance.

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Papillary oedema: True or false?

Papiledema: ¿verdadero o falso?

Dear Editor:

We read the work "Papilloedema: true or false?" by Muñoz et al[1] with interest and, as a neuroophthalmologist and neurosurgeon, we would like to express some considerations about it. Firstly, we commend the authors for their clarity in the exposition of their ideas on this highly controversial and necessary issue. It is very important to establish, with scientific truth, when a case is really a papilloedema, which confronts the patient with the possibility that an intracranial tumour may be present; the precocity of this diagnosis is essential in disease evolution.

Pseudopapilloedema is a non-pathological elevation of the papilla that may occur in some disorders, especially congenital. Other causes of pseudopapilloedema which we must keep in mind are: tilted disc syndrome and oblique implantation of the papilla, disc hypoplasia, double optic disc, disc staphyloma, melanocytoma, disc coloboma, morning glory anomaly and astrocytic hamartoma.[2-6]

Papilloedema can also be mistaken for malignant hypertensive retinopathy when there is a history of arterial hypertension and haemorrhage and the white, cotton-like foci extend to the peripheral retina. In the occlusion of the central retinal vein, the condition is usually unilateral and associated with sudden and painless loss of visual acuity.[2] In