

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<sup>5</sup>. Brain activity during sleep contributes to memory consolidation and cerebral maturation.<sup>10</sup> Both REM and NREM sleep and their proper alternation are involved in the consolidation and maintenance of memory;<sup>8</sup> although a correct declarative memory can be maintained in the exclusive presence of NREM, efficiency is less.<sup>8</sup> Patients with Asperger syndrome often present mixed insomnia and decrease of the overall duration, early awakening, daytime drowsiness<sup>11</sup> and, occasionally, parasomnias, PLM and nocturnal breathing difficulties.<sup>12</sup> 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.<sup>13</sup> 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.

## References

1. Khouzam HR, El-Gabalawi F, Pirwani N, Priest F. Asperger's disorder: a review of its diagnosis and treatment. *Compr Psychiatry*. 2004;45:184-91.
2. Gillberg C. Asperger syndrome in 23 Swedish children. *Dev Med Child Neurol*. 1989;31:520-31.
3. Nayate A, Bradshaw JL, Rinehart NJ. Autism and Asperger's disorder: are they movement disorders involving the cerebellum and/or basal ganglia? *Brain Res Bull*. 2005;67:327-34.
4. Tani P, Lindberg N, Appelberg B, Nieminen-von Wendt T, Von Wendt L, Porkka-Heiskanen T. Clinical neurological abnormalities in young adults with Asperger syndrome. *Psychiatry Clin Neurosci*. 2006;60:253-5.
5. Trenkwalder C, Hening WA, Montagna P, Oertel WH, Allen RP, Walters AS, et al. Treatment of restless legs syndrome: an evidence-based review and implications for clinical practice. *Mov Disord*. 2008;23:2267-302.
6. Tsai LY. Asperger syndrome and medication treatment. *Focus Autism Other Dev Disabl*. 2007;22:138-48.
7. Kulisevsky J, Pagonabarraga J. Nuevos tratamientos en los trastornos del movimiento. *Neurología*. 2004;19(Suppl 2):40-63.
8. Shneerson JM. Sleep medicine: A guide to sleep and its disorders. 2nd ed. London: Blackwell Publishing; 2005. p. 194-228.

9. Gabaldón Torres L, Salas Felipe J, Fernández Domínguez J, Vivancos Matellanos F, Izal E, Arpa Gutiérrez F. Restless legs syndrome. Features and impact on sleep. *Neurología*. 2009;24:230-4.
10. Mueller AD, Pollock MS, Lieblisch SE, Epp JR, Galea LA, Mistlberger RE. Sleep deprivation can inhibit adult hippocampal neurogenesis independent of adrenal stress hormones. *Am J Physiol Regul Integr Comp Physiol*. 2008;294:1693-703.
11. Bruni O, Ferri R, Vittori E, Novelli L, Vignati M, Porfirio MC, et al. Sleep architecture and NREM alterations in children and adolescents with Asperger syndrome. *Sleep*. 2007;30:1577-8.
12. Tani P, Tuisku K, Lindberg N, Virkkala J, Nieminen-von Wendt T, Von Wendt L, et al. Is Asperger syndrome associated with abnormal nocturnal motor phenomena? *Psychiatry Clin Neurosci*. 2006;60:527-8.
13. Limoges E, Motttron L, Bolduc C, Berthiaume C, Godbout R. Atypical sleep architecture and the autism phenotype. *Brain*. 2005;128:1049-61.

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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<sup>1</sup> 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.<sup>2-6</sup>

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.<sup>2</sup> In

anterior ischemic optic neuropathy, disc oedema is pale non-hyperaemic and accompanied by a decrease in visual acuity in the form of stroke.

Other entities to be excluded are infiltrative processes (leukaemia, lymphoma) where pupil impairment is predominant. Compressive optic neuropathy, which may be caused by a meningioma of the optic nerve sheath, has the optociliary shunt vessel as pathognomonic sign. Papillitis is usually unilateral, with decreased visual acuity and pupillary alterations and is usually associated with pain upon ocular movements.<sup>2,7,8</sup> Foster Kennedy syndrome, secondary to an olfactory groove meningioma, occurs with papillary oedema in one eye and optic atrophy in the other.<sup>2,9</sup>

In relation to table 1, which sets out the ophthalmoscopic differences, we propose adding that the papilla is not hyperaemic in pseudopapilloedema—unlike in papilloedema. In recent years, we have worked with optical coherence tomography (OCT), and although the authors believe that this study “has not proved effective in differentiating an incipient papilloedema from a pseudopapilloedema, since in both cases there is an increased thickness of the nerve fibre layer of the retina”,<sup>1</sup> we believe along with other researchers<sup>3,10</sup> that, although these measurements in a first consultation have not been useful in establishing differences between both entities, the evolutionary repetitions of the protocol used indeed manage to observe differences.

Finally, we celebrate the quality of the photographs that illustrate the text and we reiterate our gratitude to the authors; such reviews, which clarify knowledge about controversial issues, are very necessary for the proper development of neuroophthalmology and neuroscience in general.

## References

- Muñoz S, Martín N. Papiledema: ¿verdadero o falso? *Neurología*. 2009;24:263-8.
- Eguía F, Ríos M, Capote A. Manual de diagnóstico y tratamiento en Oftalmología. Sección 8, Tema 83. Papiledema. La Habana: Ciencias Médicas; 2009. p. 582-8.
- Mendoza Santiesteban C, Mendoza Santiesteban E, Reyes Berazán A, Santiesteban Freixas R. Capítulo 43. Papiledema. Actualización en diagnóstico y tratamiento. In: Ríos Torres M, Capote Cabrera A, Padilla González CM, Eguía Martínez F, Hernández Silva JR, editors. *Oftalmología. Criterios y tendencias actuales*. La Habana: Ciencias Médicas; 2009. p. 537-54.
- Hodelín Tablada R, Fuentes Pelier D, Santiesteban Freixas R, Francisco Plasencia M. Craneosinosis y papiledema. *Rev Neurol*. 1997;25:2051.
- López Valdés E, Bilbao-Calabuig R. Papiledema y otras alteraciones del disco óptico. *Neurología Suplementos*. 2007;3: 1-76.
- Khonsari RH, Wegener M, Leruez S, Cochereau I, Milea D. Optic disc drusen or true papilledema? *Rev Neurol (Paris)*. 2009; 18:234-8.
- Gao X, Zhang R, Mao Y, Wang Y. Childhood and juvenile meningiomas. *Childs Nerv Syst*. 2009;30:345-9.
- Sattar MA, Hoque HW, Amin MR, Faiz MA, Rahman MR. Neurological findings and outcome in adult cerebral malaria. *Bangladesh Med Res Counc Bull*. 2009;35:15-7.
- Acebes X, Arruga J, Acebes JJ, Majos C, Muñoz S, Valero IA. Intracranial meningiomatosis causing Foster Kennedy syndrome

by unilateral optic nerve compression and blockage of the superior sagittal sinus. *J Neuroophthalmol*. 2009;29:140-2.

- Hedges T. Neuro-ophthalmology. In: Schuman JS, Puliafito CA, Fujimoto JG, editors. *Optical coherence tomography of ocular diseases*. 2nd ed. Thorofare, NJ: Slack; 2004. p. 621-30.

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## Reply to: Papillary oedema: True or false?

### Respuesta a: Papiledema: ¿verdadero o falso?

Dear Editor:

We thank Drs Fuentes-Pelier and Hodelín-Tablada for their interest in the review “Papilloedema: true or false?”. We want to clarify that the fundamental purpose of our work<sup>1</sup> is to describe the diagnostic approach for a clinical suspicion of papilloedema and the role of new technologies in this context. That is why we consider it appropriate to present two distinct clinical scenarios like those appearing under the headings “Papilloedema versus pseudopapilloedema” and “Oedema versus papilloedema”.

In the first section, we discuss the changes of the optic disc that may pose reasonable diagnostic doubt, especially at the stage of incipient papilloedema, such as buried drusen, full disc in hyperopia, nasal elevation of the myopic disc and myelin fibre presence. Papillary tumours<sup>2</sup> such as astrocytoma and melanocytoma present differential characteristics (very dark pigmentation in the optic disc that partially or completely obscures the papillary margins, or a round lesion that may indicate a blackberry-like spot superimposed on the disc with intralaminar calcifications, respectively) which, according to our experience, we do not need to include in the differential diagnosis of pseudopapilloedema (figs. 1 and 2). Furthermore, we do not think it appropriate to consider abnormalities in disc development<sup>3</sup> (papillary coloboma, morning glory anomaly and peripapillary staphyloma) in the differential diagnosis for the same reason.

We agree that papillary oedema can be produced by multiple causes and that the differential diagnosis should be carried out with optic neuropathies that eventually appear with papillary oedema at some point in their evolution (ischemic, infectious, infiltrative, tumour or compressive). Therefore, we propose the second clinical scenario oedema versus papilloedema; however, the appearance of papillary oedema in itself may be non-