independent partners described the SEMES-SEPAR document as "sound management" (including treatments and measures). Clinical sessions took place in the two groups, while all the HES doctors received help and information regarding the document, requesting its systematic application from 4 October 2008. The incidence rate of CAP was 0.56% in front of 0.59% of patients treated in HES (6-7 cases/1,000 rooms/year in the two groups). The average age was 62±21 years versus 65±19 years. The Charlson comorbidity index (mean±SD) was 1.9±1.6 versus 2.1±1.8, which increased 4.1±2.1 in front of 4.5±2.3 when weighting the age variable. The study and control groups had a distribution of cases that showed no significant differences regarding the risk groups on Fine's score (PSI: Pneumonia Severity Index). We can affirm that the implementation of the guidelines has significantly improved the care process for CAP in our HES. We managed to increase the appropriate empirical treatment and achieve greater aetiological diagnosis, a more appropriate microbiological application for additional tests, a shorter hospital stay, a shorter duration for the antibiotic and a lower rate for re-visits. In addition, and as expected in our study, we achieved a higher rate of appropriate antimicrobial administration within the first 4 hours of the patient's admission to the HES. In terms of absolute percentage mortality during the 30 days following hospital admission (assigned by the study partners to the actual infectious process or derived from complications), this is lower in the study group (8% compared to 11%), although without significant differences, possibly due to the sample size.

Conflict of interest

The authors affirm that they have no conflicts of interest.

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Central Serous Chorioretinopathyas First Sign of Onset of Sleep Apnea-Hypopnea Syndrome

Coriorretinopatía central serosa como forma de debut del síndrome de apneas-hipopneas del sueño

To the Editor:

In recent years, there have been major advances in our understanding of the pathophysiology of the sleep apnoea-hypopnoea syndrome (SAHS). In addition to the traditional relationship with cardiovascular risk or traffic accidents, it has been associated with metabolic disorders (atherogenesis or hydrocarbon metabolism disorders), neurological conditions (cerebrovascular disease or intracranial hypertension) or ocular diseases (drooping eyelid syndrome or glaucoma).[1,2]

We present a case of central serous chorioretinopathy (CSC) in a young patient, which was the first sign of SAHS. As far as we are concerned, this is the first case described in the Spanish literature. The patient was a 37-year-old man, who was an ex-smoker with a history of diverticulitis, which had been treated surgically, and common migraine, also known as migraine without aura. He went to the emergency services due to a 48-hour progressive loss of vision in the right eye, for which he was examined by the ophthalmology department. The examination of the right fundus revealed an accumulation of fluid in the macular area with detachment of the neuroepithelium. Fluorescein angiography was performed, exhibiting a hypofluorescent zone in the area of detachment and a late-stage hyperfluorescent leaking point (fig. 1A). The patient also underwent optical coherence tomography, which showed the detachment of the neuroepithelium with subretinal fluid (fig. 1B). These findings were consistent with the diagnosis of CSC. Given his previous history of snoring, the patient was referred to our sleep unit for evaluation. In the case history, he experienced symptoms of a 3- to 4-month development that was consistent with snoring, apnoeic pauses, watery rhinorrhoea, dry mouth and morning and excessive daytime sleepiness (13 points on the Epworth test). There were no disorders found in the examination of the solid craniofacial or tonsillar hypertrophy. The patient presented with Mallampati II, BMI of 28.5 kg/m^2 and blood pressure of 130/85 mmHg. The radio-allergo-sorbent-assay (RAST) was weakly positive for *D. pteronyssinus* and *D. farinae*. The polysomnography (PSG) revealed an alteration of the sleep architecture, with an increased sleep surface and at the expense of respiratory events (mostly obstructive), with a respiratory disturbance index (RDI) of 64.2 h^{-1}, a 94% average rate of oxygen saturation (SaO_2) and a time below 90% of SaO_2 (TC90) of 5.5%. The patient was treated with continuous positive pressure airway (CPAP) at 7 cmH_2O and antagonists of leukotriene receptors, improving the respiratory symptoms and decreasing daytime sleepiness (6 points on the Epworth test). The eye evolution was favourable, with recovery of the visual acuity in 4 weeks and the disappearance of the fundus alterations.

CSC has been described, in particular, among the ophthalmologic alterations of SAHS.[3-5] This condition produces a chorioretinal alteration that is consistent with the serous detachment of the neurosensory retina in the macular area, allowing the passage of fluid from the choroid to its final destination in the subretinal space. The condition usually occurs in young and middle-aged men, who

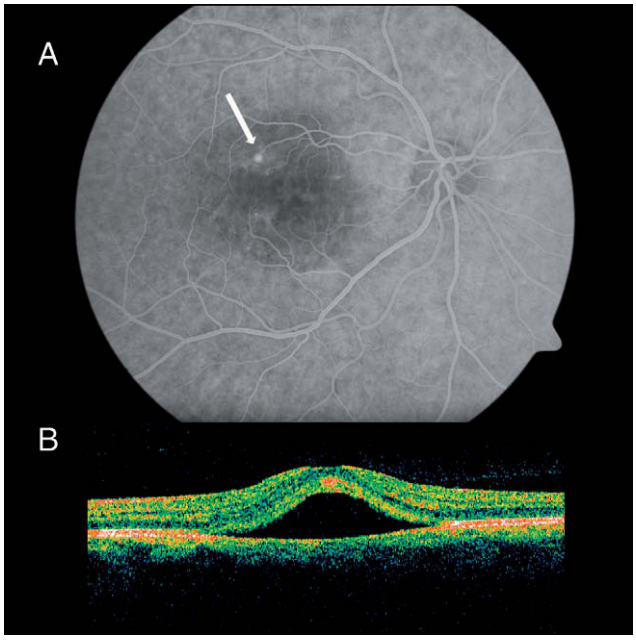

Figure 1. A. The fluorescein angiography shows a hypofluorescent area corresponding to the zone of detachment and to a hyperfluorescent leaking point (white arrow) in late-stage arteriovenous filling. B. The optical coherence tomography shows the detachment of the neurosensory retina with subretinal fluid.

refer impaired visual acuity and metamorphopsias.[3-5] The aetiopathogenesis of the condition consists of two theories: the reversal of the polarity of the retinal pigment epithelium and the increase in vascular permeability of the choroidal circulation.[5] The risk factors for its occurrence include: type A personality, pregnancy, Cushing's syndrome and treatment with corticosteroids or sympathomimetic medicines.[4] This condition is associated with increased levels of catecholamines,[3] having noted this finding as a possible link with SAHS.

In patients with SAHS, blood pressure at night usually follows a *Non-Dipper* pattern, because the repeated obstruction of the upper airway causes a release of catecholamines, the levels of which are detectable in serum and urine, thus increasing the sympathetic activity, with a clear association between dose and response.[1,6]

In this context, it seems logical that an increase in the catecholamine levels, which occurs in patients with SAHS, would favour the development of CSC in predisposed individuals. This was the case with our patient, where no risk factors were present, nor was he taking medication that could have been responsible for the development of CSC. Furthermore, the patient's blood pressure limit served as a marker of sympathetic activation, which effect was implicated in the development of CSC.

In previous studies, Leveque et al.[3] found a higher risk of SAHS among patients with CSC (OR 3.67). Kloos et al.[4] found a 22% prevalence of SAHS among 36 patients with CSC. Furthermore, a case was recently described in which treatment with CPAP, not eye therapy, cured the CSC, thus supporting the theory that associates both conditions.[5]

The ocular alterations in SAHS were recently reviewed,[2] although it is difficult to separate the findings associated with conditions such as diabetes or hypertension from the specific contribution of SAHS; thus, research in this field is still ongoing.

Given these findings, we may conclude that the sympathetic activation that occurs in patients with SAHS may act as a trigger for CSC in predisposed individuals. For this reason, patients with CSC would have a higher risk of SAHS than the general population. Therefore, its remission should be assessed from ophthalmology specialists to sleep units screening SAHS.

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