Obstructive sleep apnoea as a cause of headache presenting to the emergency department

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Case report

A 48-year-old South Asian lady presented to the emergency department complaining of predominantly right-sided temporal headache, associated with lacrimation and swelling around the right eye. She had type 2 diabetes mellitus and chronic kidney disease. She was obese with a body mass index of 58.1 kg/m². Clinical examination revealed no signs of meningism. Computerized tomogram (CT) of her head was normal. Erythrocyte sedimentation rate (ESR) was raised at 97 mm/h. She was started on oral corticosteroids for a presumptive diagnosis of temporal arteritis based on the ESR and discharged.

Three weeks later, while on corticosteroids, she was readmitted with a relapse of the acute headache. Temporal artery biopsy from her previous admission showed no evidence of temporal arteritis. A rheumatology review concluded that her relatively young age, long duration of headaches (6–9 months of headaches prior to presentation) and normal temporal arteries on palpation, made temporal arteritis unlikely. She was discharged with analgesics.

A few weeks later, she was reviewed in outpatient clinic and her history of headaches revisited. She suffered with chronic daily headaches and despite corticosteroid therapy, she had had further acute episodes, in addition to her daily headaches. She was gaining weight; her glycaemic control deteriorated. Other investigations to determine the cause of raised ESR excluded autoimmune disorders, myeloma, infection (including occult tuberculosis) and Cushing’s syndrome. Corticosteroids were discontinued slowly over the ensuing weeks.

Meanwhile, following an unrelated referral, she underwent an overnight pulse oximetry which showed 4% oxygen saturation dip-rate of 17.7/h, diagnostic of moderate-severe obstructive sleep apnoea (OSA). The patient was commenced on Continuous Positive Airway Pressure (CPAP) ventilation with full-face mask, pressure at 11.8 cm H₂O. Within days, her headache started to settle down. She continued to use the CPAP successfully, for an average of 5.35 h/night.

In the following months, her body weight issues were addressed and diabetes treatment optimized; HbA1C improved from 9.0% to 6.8%. She lost nearly three stones in weight since stopping corticosteroids. In January 2010, it was felt that her sleep apnoea status needed reviewing, in view of the significant weight loss. An overnight multi-channel portable sleep study showed that apnoea–hypopnoea Index (AHI) was 2.9/h (OSA = AHI ≥ 5/h). She has since come off CPAP therapy and remains headache-free. Figure 1 shows the patient’s clinical progress.
Discussion

OSA is associated with cluster headache, migraine and tension-type headache as well as patterns like chronic daily headache, morning headache and awakening headache. Cluster headache is a severe unilateral orbital, supraorbital, and/or temporal pain lasting 15–180 min untreated, usually associated with one or more of the following autonomic symptoms on the affected side—conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, facial sweating, miosis, ptosis or eyelid oedema. When association between cluster headache and OSA have been reported, showing an 8.4-fold increase in incidence of OSA in those with cluster headache. Conversely, studies on patients with a diagnosis of OSA show that they have a high incidence of headaches of various patterns.

This patient’s acute presentation to emergency department can be attributed to cluster headache in association with OSA. The nature and location of the headache, ipsilateral eyelid oedema and lacrimation fulfil the criteria for diagnosis of cluster headache. The lack of improvement with corticosteroids and remission with OSA treatment supports this conclusion. The possible contributors to elevated ESR included chronic renal failure with an estimated glomerular filtration rate of 35–45 ml/h, obesity,4 metabolic syndrome5 and potentially obstructive sleep apnoea.6

Headaches, both acute and chronic types, are common presentations to hospital-based and community-based clinical practice. Sleep disorder-related headaches are well-recognized. Clinicians should be aware of this association to avoid subjecting patients to unnecessary investigations looking at the cause of headache. Patients, particularly men, with obesity, increased neck circumference, hypertension, age >65 years, family history of OSA and those who use alcohol or sedatives are at increased risk of developing OSA.7 Such patients presenting with headaches should have sleep apnoea formally investigated; sleep-questionnaires are often insufficient for diagnosis. This will benefit their headache and sleep-related disorders, as well as potentially reducing their cardiovascular risk.8

References


