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BRIEF REPORT

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Valsalva-induced cluster headache

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Abstract A case valsalva-induced cluster headache is presented. Cluster attacks induced by valsalva manoeuvres alone were only recently described, and such patients have features of cough headache and cluster headache. Attacks occurred a couple of times a week in the patient presented, solely triggered by valsalva manoeuvres including coughing, sneezing or straining and not by exercising.

Keywords Cluster headache • Valsalva • Melatonin

Cluster attacks of severe unilateral short-lived pain and autonomic features have few identified triggers. Attacks are described as occurring during vulnerable periods of time rather than being induced by triggers, with the exception of alcohol ingestion during vulnerable periods. In addition, cough-induced headache is uncommon but well described, and is not prolonged or associated with autonomic features. The presentation of valsalva-induced cluster headache has characteristics of both cough headache and cluster headache.

Case report

A 59-year-old man was evaluated for a 9-month history of headache attacks precipitated only by coughing, sneezing or, straining (for example while carrying heavy objects), which are all valsalva manoeuvres. Attacks lasted 30–120 min, with a frequency of a couple of times a week and were not triggered by his regular jogging. The side-locked pain was centred in the right periorbital region and between attacks there was a vague sensation in the same area. Triggers such as a sneeze would rapidly bring on severe sharp right periorbital pain and tearing, conjunctival injection with frequent right-sided rhinorrhoea (no congestion). There was no ptosis and at times the patient was agitated, resulting in pacing. More than one attack could be experienced in the same day. Although it was clear that brief increases in intrathoracic pressure could trigger an attack, the mildest forms of valsalva manoeuvre (lifting objects, mild straining at stool, but not sneezing or coughing) were usually free of attacks.

Background medical history was notable for a deep vein thrombosis and a pulmonary embolus, which developed a few months after the start of the headaches. Investigations failed to reveal any explanation for the hypercoagulability and he was maintained on coumadin. The patient did not have the factor V Leiden (R506Q) mutation or the prothrombin G20210A mutation for hypercoagulability. Protein-C and protein-S levels were not obtained, as the patient was already on coumadin and the levels would be expected to be low. There was no history of hypertension. The patient had stopped smoking 20 years previously but had a ten pack-year history of smoking, which ceased 20 years ago. General and neurological

examinations were unremarkable. Specifically there was no allodynia or sensory loss in the face. Brain magnetic resonance imaging (MRI) was normal and there was no Chiari type-1 malformation, posterior fossa crowding or evidence of venous sinus thrombosis. Magnetic resonance angiography of the brain was also normal.

The patient had discontinued a modest dose of verapamil (240 mg daily) due to lack of response prior to initial review. A trial of topiramate prophylaxis was stopped at 75 mg daily due to side effects and lack of response. Sumatriptan 4 mg subcutaneous injection for abortive relief was prescribed and used only once with moderate relief. Prophylaxis then consisted of melatonin 3 mg nightly (increasing to 6 mg) and by two weeks the frequency of attacks reduced. Improvement continued over the following 2 weeks. Over the following 4 weeks the patient became attack-free. After 8 weeks the medication was discontinued and and at the time of writing 12 weeks off medication he has not had a recurrence.

Comments

Valsalva-induced cluster headache was first described by Ko and Rozen [1]. Their patient had attacks typically lasting 4 h, solely triggered by valsalva manoeuvres and responding to daily indomethacin. An earlier paper described 'benign cough cluster headache' in a man with unilateral headache attacks lasting only 1–10 min without autonomic features, characteristics that do not favour a diagnosis of cluster headache [2].

Attacks of cluster headache are not typically associated with an identified trigger, although alcohol is well known to precipitate attacks. Valsalva triggers to headache suggest a diagnosis of cough headache, which warrants scrutiny of the posterior fossa and foramen magnum by MRI for evidence of crowding and Chiari type-1 malformation. Without such abnormalities primary cough headache is ascribed to presentations that fulfil International Headache Society criteria with headache lasting from 1 s to 30 min only and without autonomic features [3]. The patient presented had much longer attacks than primary cough headache and the autonomic features further separate primary cough headache from valsalva-induced cluster headache. The patient's frequent uneventful jogging excludes primary exertional headache.

Secondary cluster-like headache has recently been associated with cerebral vein thrombosis [4]. Magnetic resonance venography was not performed in the case presented. The development of cerebral vein thrombosis causing cluster headache and persisting for many months despite anticoagulation was thought to be remote.

Valsalva manoeuvres reliably increase intracranial pressure by increasing central venous pressure, but rarely is this accompanied by headache. The pathophysiology bridging the rise in central venous pressure to the precipitous onset of cough headache or in this case a cluster attack is unknown. Although exact mechanisms of cough headache are unknown, response to lumbar puncture and acetazolamide have suggested a role for raised intracranial pressure. Cluster headache is easily provoked with nitroglycerin 1 mg sublingually but the delay of 30 to 50 min suggests a different mechanism to cough for instance [5].

Indomethacin, a first line agent for cough headache, was avoided, as the patient was anticoagulated. For the same reason measurement of cerebrospinal fluid pressure (CSF) was avoided. A related compound, melatonin, which also has analgesic and anti-inflammatory effects, among other actions, was started [6]. Melatonin has been shown in a double-blind placebo-controlled study to be of benefit in cluster headache [7]. Indomethacin is known to reduce CSF pressure due to cerebral vasoconstriction and a reduction in cerebral blood flow. It is not known whether melatonin also reduces CSF pressure, but a recent small study suggested that it does not reduce cerebral blood flow [8]. The patient so far has not exhibited a return of events off melatonin, but we cannot assume that he was melatonin responsive, as a natural remission may have occurred. Event-related triggers of cluster are uncommon and valsalva-induced cluster is a distinct subtype of cluster headache.

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