

# INTERNAL CAROTID ARTERY DISSECTION PRESENTING AS CLUSTER HEADACHE

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Cluster headache (CH) is a clinical entity characterized by strictly unilateral head pain attacks accompanied by ipsilateral autonomic phenomena. The attacks are severe, short-lasting (15–180 minutes), and may occur several times a day<sup>1</sup>. Symptomatic CH cases have been described in association with different kind of lesions located in the middle fossa, near the sellar or parasellar structures although lesions of the internal carotid artery (ICA) were also described: aneurysma of ICA and internal carotid artery dissection (ICAD)<sup>2</sup>.

We report a patient with symptomatic CH secondary to ICAD, and complete remission of his symptoms with usual treatment for CH. We obtained informed consent from the patient for publication.

## CASE

A 53-year-old white man presented to the emergency room of Hospital Israelita Albert Einstein with 10-day history of recurrent right peri-orbital headache. Headaches were sudden, severe, pulsating, of thirty minutes duration and occurred once or twice a day. Headache was accompanied by ipsilateral ptosis, conjunctival injection, lacrimation and nasal stuffiness. He was on treatment for mild hypertension with hidroclorotiazide 25 mg once a day. Examination during headache revealed right Horner's syndrome with partial ptosis and miosis, accompanied by ipsilateral lacrimation, eyelid edema and conjunctival injection. After this episode, he remained with mild right ptosis. The remaining cranial nerves and neurological examination were normal.

The above features satisfied criteria "A to D" for CH of the International Classification of Headache Disorders (number, site and duration, accompanying symptoms, frequency of headaches)<sup>1</sup>. Criterion E stipulates that other causes for the headache must be ruled out. We therefore performed further investigations. Computed tomographic imaging of the brain was normal. Fat suppression T1-weighted magnetic resonance imaging of the carotid canal level displayed high signal compatible with blood products within the wall of the right internal carotid what was suggestive of arterial dissection (Figure).



Figure. T1-weighted axial section, at the carotid canal level. Fat suppression technique reduces the adipose tissue signal, which becomes slightly hypointense while the methaemoglobin subintimal blood maintains its hyperlucency signal. A mural haematoma is evident with consequent narrowing of the vessel lumen.

He was admitted in our critical care unit and complementary investigation was performed. Blood cell count, erythrocyte sedimentation rate, coagulogram, protein C and protein S, lipido-gram were normal. Results from a heart survey, including Holt-er's monitor and echocardiography, were normal.

The patient was anti-coagulated. Verapamil and prednisone were initiated as prophylactic therapy for cluster headache in our patient. There was complete resolution of headaches in one week and the patient was entirely asymptomatic at follow-up six months later.

## DISSECÇÃO DA ARTÉRIA CARÓTIDA INTERNA APRESENTANDO-SE COMO CEFALÉIA EM SALVAS

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## DISCUSSION

Clinical signs of ICAD are often dissociated with initial pain and local signs followed by retinal or cerebral ischaemic events<sup>3</sup>. Headache is the most common symptom preceding stroke in patients with ICAD<sup>4</sup>. Frontal and parietal localizations were significantly associated with internal carotid artery dissection<sup>5</sup>. Pain in ICAD is postulated to be caused by stimulation of trigemino vascular system and it can mimic different primary headaches, including CH<sup>6</sup>. In a series of 65 ICAD patients, pain was the presenting symptom in 58.5%, typically unilateral and localized, a painful Horner's syndrome was the initial sign in 29%, and in two cases, pain mimicked CH<sup>6</sup>. Similar cases have been reported: in all Horner's syndrome persisted in pain free intervals, autonomic parasympathetic signs variably included lacrimation, conjunctival injection and nasal congestion, duration and character of pain was less stereotyped than in CH<sup>2</sup>.

Examination during both primary cluster headache and ICAD may demonstrate a Horner's syndrome. Persistent ptosis and miosis between headaches are widely accepted as features of primary cluster headache but data regarding carotid vasculature in such circumstances is lacking<sup>7</sup>. In routine practice, individuals with cluster headache have diagnosis made upon clinical grounds with structural neuroimaging requested in a substantial proportion. Few patients have carotid imaging. Identification of ICAD is important as it has substantial risk of stroke (55%) with alternate management and prognosis<sup>3</sup>. It is postulated that certain features may help to distinguish ICAD from cluster headache. Headache duration more than 3 hours, lack of diurnal periodicity and absence of precipitation by alco-

hol intake should raise suspicion of ICAD. Atypical pain, localisation of pain over face or eye, and accompanying neck pain suggest ICAD rather than cluster headache. Lack of anhidrosis accompanying Horner's syndrome during a headache has been postulated to suggest ICAD (as fibres pass along the external carotid plexus)<sup>8</sup>.

In conclusion, individuals presenting with new onset of a cluster headache disorder may have an underlying internal carotid artery dissection. Once clinical features may not be helpful in distinguishing ICAD from primary cluster headache, and further investigation must be performed, ideally an carotid angiogram. We also believe that in patients with a previous history of CH, but with atypical symptoms in the presenting attack, complementary investigation must also be performed.

## REFERENCES

1. The International Classification of Headache Disorders, 2<sup>nd</sup> Ed. Cephalalgia 2004;24(Suppl 1):S9-S160.
2. Rigamonti A, Iurlaro S, Reganati P, Zilioli A, Agostoni E. Cluster headache and internal carotid artery dissection: two cases and review of the literature. Headache 2008;48:467-470.
3. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Eng J Med 2001;344:898-906.
4. Pieri A, Spitz M, Valiente RA, Avelar WM, Silva GS, Massaro AR. Spontaneous carotid and vertebral arteries dissection in a multiethnic population. Arq Neuropsiquiatr 2007;65:1050-1055.
5. Campos CR, Calderaro M, Scaff M, Conforto AB. Primary headaches and painful spontaneous cervical artery dissection. J Headache Pain 2007;8:180-184.
6. Biousse V, D'Anglejan-Chatillon J, Massiou H, Bousser MG. Head pain in non-traumatic carotid artery dissection: a series of 65 patients. Cephalalgia 1994;14:33-36.
7. Razvi SS, Walker L, Teasdale E, Tyagi A, Muir KW. Cluster headache due to internal carotid artery dissection. J Neurol 2006;253:661-663.
8. Frigerio S, Buhler R, Hess CW, Sturzenegger M. Symptomatic cluster headache in internal carotid artery dissection - consider anhidrosis. Headache 2003;43:896-900.