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Case Report

Paroxysmal hemicrania associated to carotid artery dissection: a case report

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Abstract

There are numerous case reports relating trigeminal autonomic cephalalgias to structural injuries. However there is no description of the association between paroxysmal hemicrania and carotid artery dissection. We describe a previously healthy 63-year-old male presented with the onset of severe, throbbing pain in the right frontal region, lasting between 10 and 30 minutes, with a frequency of approximately two to three attacks per day, which began two days before seeking medical care. Pain was associated with ipsilateral tearing, semiptosis and nasal congestion. A cervical arterial magnetic resonance angiography demonstrated left carotid artery dissection in the C1/C2 segment of the left internal carotid artery. The patient became asymptomatic after indomethacin use. We conclude that The possibility of investigating carotid dissection should be considered in patients with paroxysmal hemicrania.

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Introduction

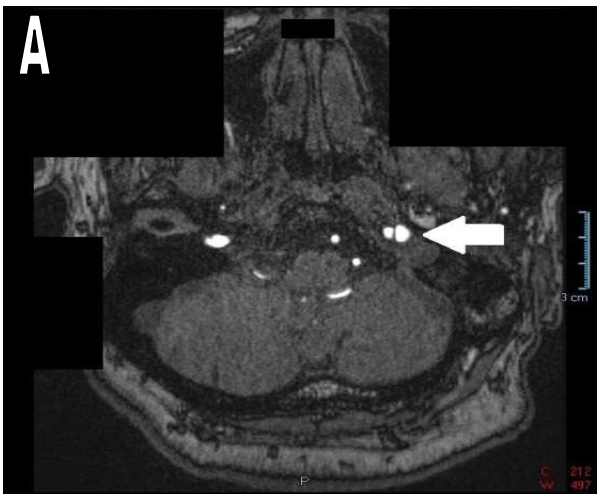
Paroxysmal hemicrania is a disorder characterized by severe, unilateral headache, lasting between two to thirty minutes, with an orbital, supraorbital and/or temporal localization, associated with ipsilateral autonomic symptoms. A marked response to indomethacin is essential for diagnosis.[1]

Although trigeminal autonomic cephalalgias are classified as primary headaches[1], there are numerous case reports relating such headaches to structural injuries.[2-4] To the best of our knowledge, there is no description of the association between paroxysmal hemicrania and carotid artery dissection. Herein, we describe one case that may demonstrate this association.

Case report

A previously healthy 63-year-old white man presented with the onset of severe, throbbing pain in the right frontal region, lasting between 10 and 30 minutes, with a frequency of approximately two to three attacks per day, which began two days before seeking medical care. Pain was associated with ipsilateral tearing, semiptosis and nasal congestion. There was no photophobia, phonophobia, nausea or vomiting associated with pain. There was not any history of head or neck trauma. Physical and neurological examination was normal. The physical examination was not suggestive of other disorders such as Marfan or Ehlers-Danlos syndrome.

Magnetic resonance imaging and intracranial magnetic resonance angiography were normal. A cervical arterial magnetic resonance angiography demonstrated left carotid artery dissection in the C1/C2 segment (Figure 1 A and B). This exam did not find any specific cause for the carotid dissection.



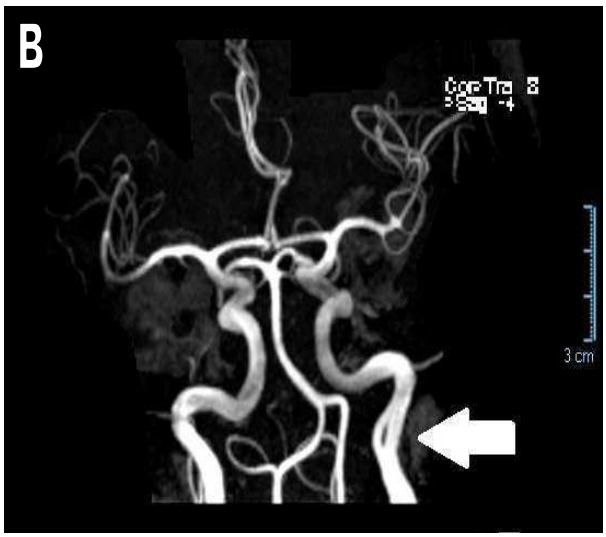


Figure 1. A and B) Cervical arterial resonance angiography demonstrates dissection in the left carotid artery in the C1/C2 segment.

For the treatment of arterial dissection, acetylsalicylic acid and atorvastatin were used. For the treatment of headache, indomethacin (50 mg every 8 hours) was initiated orally, with a significant pain response in only 3 days. The patient became asymptomatic. He took indomethacin for 15 days.

He has had no headaches for three years and has not had any stroke. He still uses acetylsalicylic acid and atorvastatin. He made follows-up magnetic resonance angiography every six months. The last magnetic resonance angiography was performed three years after the headache and the dissection is stable.

The patient had given his written consent for the case report.

Comments

Our patient had no signs or symptoms on the same side as the dissection. Between 26 to 36% of patients who have carotid dissection have no head, face or neck pain[5] and 25.5% of these patients have no local signs or symptoms.[6]

There are descriptions of headache compatible with paroxysmal hemicrania associated with intracranial secondary lesions such as expansive lesions in the sella turcica, pituitary apoplexy, intraparenchymal pontomesencephalic hemorrhage, type I Chiari malformation and giant cell arteritis.[2, 7-11]

Our patient fulfilled the criteria for paroxysmal hemicranias, except for the frequency of pain, which was less than five attacks per day, on most days. We cannot completely rule out the possibility that the association between headache and dissection was a coincidence. However, there was a temporal relationship between the dissection and headache and, the patient had no previous headache. This reinforces a cause and effect relationship between dissection and headache. There was also a temporal relationship between the use of indomethacin and the patient's improvement. We cannot be certain whether this response was due to the use of the medication or to the natural history of the disease.

We did not find other reports about the association between paroxysmal hemicrania and carotid dissection. However, there were an association between carotid dissection and cluster headache[2] and between carotid dissection and hemicrania continua.[12] In all these reported cases of cluster headache and hemicranina continua, the dissection was ipsilateral to pain.[2, 12]

Our patient presented with contralateral headache regarding the side of the arterial dissection. The pathophysiology of autonomic trigeminal headaches involves abnormalities of the hypothalamic function, trigeminal-autonomic reflex disinhibition, cranial and trigeminovascular autonomic activation.[13, 14] In cluster headaches, studies with positron emission tomography and functional magnetic resonance demonstrate activation of the ipsilateral posterior hypothalamus during the pain attack.[14] In the paroxysmal hemicrania, activation occurs of the contralateral posterior hypothalamus and the contralateral ventral midbrain to the pain during the attacks.[14] This may justify the fact that the dissection was contralateral to our patient's headache since it would be ipsilateral to the activated hypothalamus. Another case of paroxysmal hemicrania associated with intraparenchymal hemorrhage also presented contralateral headache to the lesion, corroborating this explanation.[7]

Conclusion

In conclusion, in patients with paroxysmal hemicrania, the possibility of investigating carotid dissection should be considered.

Key points

- Carotid dissection may present as paroxysmal hemicrania.
- Paroxysmal hemicrania can also be a secondary headache.
- The headache presentation can be contralateral to the lesion.

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Author's contributions:
Felipe Araújo Andrade de Oliveira: Conceptualization, Data curation, Writing original draft
Pedro Augusto Sampaio Rocha-Filho: Supervision, Writing review and editing

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