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LITERATURE REVIEW

Vascular head and face algiae

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ABSTRACT

Vascular head and face algiae represent a physiopathological and therapeutic entity, symptomatically polymorphic, due to the numerous structural and topographic variants where they occur. Pathology of any structure representing the face and the head may begin with or be accompanied by algia at this level. The physiopathological mechanism of vascular face algiae develops in three stages: arterial spasm, vasodilation and edema.

The crises should be distinguished from migraine, although it seems that the physiopathological mechanisms are, at least partially, similar. Also, the differential diagnosis should be made between the classical forms of vascular head and face algiae, the atypical and borderline ones. Management of vascular face algiae needs to consider the crisis, the background therapy, nerve blockages and surgical treatment.

KEYWORDS: algiae, vascular, Sluder syndrome, Charlin syndrome, chronic paroxysmal hemicrania

Vascular head and face algiae are included in the great chapter of headaches. Over 80% of the emergency presentations to the ENT Emergency Room are due to different clinical and topographical forms of headaches.

Pathology of any structure representing the face and the head may begin with or be accompanied by algia at this level.

If pain caused by visceral organs of the various functional anatomic systems in this region (brain, eyes, nose, pharynx, ear etc.) are described in detail in the pathology of the respective organs, herein we will make a strict review of vascular origin head and face algiae.

Vascular head and face algiae represent a physiopathological and therapeutic entity, symptomatically polymorphic, due to the numerous structural and topographic variants where they occur.

Vascular-type algia manifests at the level of the face by:

- pain crisis, usually located at the root of the nose;
- pain is strictly unilateral and very precisely located; it regularly repeats in the form of variable length crises ("cluster headache");
- painful crises are associated with nasal, ocular and vasomotor secretory phenomena.

These crises should be distinguished from migraine, although it seems that the physiopathological mechanisms are, at least partially, similar (there are many similarities). Nevertheless, migraine has clinical manifestations that clearly define it and, in the same time, differentiate it from the other head and face algiae.

Migraine frequently has a prodrome, a strictly fronto-orbital pain topography, the pain being pulsating. The onset of migraine is precocious, in childhood, and especially in adolescence. Pain is accompanied by photophobia and neurovegetative symptoms. They manifest in the ophthalmic, sylvian, basilar areas, in a genetically-prone context.

Vascular head and face algiae are much less common than migraine (0.4% in men and 0.8% in women). They occur most frequently between 20 and 70 years. Their study is necessary in the differential diagnosis of migraine. The common clinical elements of vascular head and face algias are: periodicity, presence of neurovegetative disorders, stress, anxious behaviour.

Periodicity. The painful syndrome occurs most frequently in spring and autumn and it can manifest

between one and three months. The frequency of the crises varies from one per week to more crises per day. Pain has a sudden onset, without being accompanied by a prodrome, and it reaches climax in 15 to 20 seconds. Pain is usually unilateral and topographically distributed on one of the branches of the trigeminal nerve. Remission periods generally exceed two years.

Vasomotor disturbances are characterized by parasympathetic hypertonia, manifested by: lacrimal hy-

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persecretion, conjunctival hyperaemia, unilateral nasal obstruction corresponding to the pain, unilateral rhinorrhea, palpebral miosis and ptosis, bradycardia. Neurovegetative disorders are more frequent and pronounced in shy, emotional individuals.

Role of stress. Intense activity, chronic fatigue, tabacco or alcohol are risk factors. Patients with gastric ulcer or gastrointestinal disorders are most prone to painful face crises of vascular type.

During the crisis, the patient tends to isolate, is afraid of light, has reduced professional performance, anxiety, psychomotor agitation and keeps his head in his hands.

The physiopathological mechanism of vascular face algia develops in three stages: arterial spasm, vasodilation and edema. The three stages occur in the vessels of the territory innervated by the trigeminal nerve and histamine acts as a mediator. Cyclicity, duration and intensity of painful crises depend on the hypothalamus which regulates secretions of melatonin, cortisol, beta-endorphins and prolactine, under the influence of several less-known endogenous factors or exogenous ones (stress, toxic, living conditions, etc.). Hypoxia accentuates pain by influencing chemoreceptors in the carotid corpuscle.

CLINICAL FORMS OF VASCULAR HEAD AND FACE ALGIAE

1) Sluder Syndrome is an unilateral vascular cephalalgia, pain being located in the root of the nose. Pain occurs in crises and radiates to the mastoid and neck, being accompanied by unilateral nasal congestion, rhinorrhea and tearing on the same side as the pain. Otalgia and clogged ear sensation may also occur, as well as taste changes. In many cases, the painful crisis may be preceded by hypersialorrhea. The clinical phenomena immediately cease as soon as the sphenopalatine ganglion is anesthetized.

2) Charlin Syndrome is characterized by a pain localized to the internal angle of the eye, and it is associated with tearing, photophobia, blepharospasm, conjunctival hyperaemia, palpebral edema, rhinorrhea with unilateral hyperaemia of the nasal turbinates.

3) Other pain syndromes in vascular head and face algiae

a. Harris's ciliary neuralgia is manifested by temporal pain and tearing on the same side with the pain. The other clinical signs described in previous syndromes are absent.

b. Vail's Vidian neuralgia is characterized by pain at the root of the nose, which radiates to the ear, neck and shoulder.

c. Monbrun - Benistry Syndrome is manifested by a unilateral retro-ocular pain that radiates to the occiput and is accompanied by hemifacial cutaneous vasodilation with profuse sweating and cutaneous hyperesthesia.

ATYPICAL OR BORDERLINE VASCULAR HEAD AND FACE ALGIAE

Their clinical manifestation is similar to classical forms of vascular face algia. However, the difference consists in location, periodicity, evolution and therapeutic response. Five clinical entities are more frequent in practice:

Cluster tic is a face neuralgia accompanied by vasomotor changes on the painful territory. It quickly recedes after administration of Carbamazepine.

Chronic Paroxysmal Hemicrania is characterized by short, but very frequent, painful crises (they can reach up to 40 in 24 hours) and it is found most often in women. Crises recede after administration of Indomethacin.

SUNCT = short lasting unilateral headache with conjunctival injection and tearing is characterized by short and very frequent painful crises, accompanied by conjunctival injection and tearing. Crises are sometimes subintrant. Their frequency may vary for one per day to 30 crises per hour. It receded with Indomethacin.

Tolosa - Hunt syndrome is clinically characterized by stabbing pain, ophthalmoplegia, moderate nasal inflammatory syndrome. Symptoms remit after corticosteroid therapy.

Raeder's paratrigeminal syndrome, also known as *"orbital apex" syndrome*, is characterized by throbbing pain, deeply located at the back of the orbit, hypoesthesia in the ophthalmic territory. Sympathetic participation highlighted by miosis - Claude Bernard Horner syndrome. In the presence of a painful orbital apex syndrome we should also suspect the possibility of a meningioma in the petrous region.

Management of vascular face algie needs to consider the crisis, the background therapy, nerve blockages and surgical treatment.

Treatment of the crisis consists of the following remedies, in sequence or in combination: oxygen therapy, DHE (ergotamine) - per os or by injection, Sumatriptan (Imiject) injection, moderate, pain-relieving doses of cortisone, including morphine, sometimes associated with anxiolytics.

Background therapy. Intermittent administration of antiserotoninics (Metysergide) in progressive doses from 60 mg to 140 mg per day, moderate corticotherapy (40 mg per day), lithium, calcium blockers (Verapamil - Isoptin), anti H1 agents (Sibelium), GABA - mimetics at the level of the hypothalamus (Sodium Valproate).

Nerve blockages have the role of reducing the parasympathetic hyperactivity in the sphenopalatine ganglion. There are four ways of making the anaesthesia:

- contact - with an anaesthetic at the bottom of the middle inferior turbinate;
- infiltration - percutaneous infiltration in the orbito-zygomatic angle;
- through the naso-palatine duct - 1 cm inside the penultimate upper molar;
- anaesthetic infiltration of the Arnold nerve at the C2 level.

Surgical treatment is a last resort after failure of all previous ones. Surgical procedures consist of: thermocoagulation of the Gasser ganglion or simple infiltrations with glycerol, sectioning of the great superficial petrous nerve by intracranial neurosurgical intervention.

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