Trigeminal Autonomic Cephalalgia by Neurogenic Palatine Compression: A Clinical Report and Review

Cefalea Autonómica Trigeminal por Compresión Neurogénica Palatina: Reporte de Caso y Revisión

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SUMMARY: This clinical report presents the evolution of a possible intraoral traumatic trigeminal autonomic cephalalgia simulating a probable cluster headache. A 50-year-old female patient had severe right-hand side pain for 7 years with autonomic signs and symptoms, such as lacrimation, conjunctival injection, rhinorrhoea, nasal congestion, forehead perspiration, myosis and eyelid edema. The episodes of pain lasted 4 or 5 months with 3- or 4-month remission periods between the painful onsets. The headaches presented an episodic pattern (1 to 3 attacks daily) lasting three to six hours. The patient had used five prosthetic sets continuously (24 h) for 20 years and the current prosthesis was 7 years old. Accidentally, after the alleviation of the maxillary denture due to it rested on a marked irritated incisive papilla the symptoms disappear. The patient has been periodically checked over a 5 year period since, without recurrence of her pain and autonomic symptomatology. The possible pathophysiology is discussed.

KEY WORDS: Sphenopalatine Ganglion; Incisive Papilla; Neurovascular Pain; Referred Pain; Temporomandibular Disorders.

INTRODUCTION

Primary and referred pain patterns depend on the intensity, localization and timing of the perceived pain stimulus which can produce neuropathic pain (Sessle *et al.*, 1986). It can also simulate pathologies' apparently unconnected symptomatic behavior. Neuropathic pain pathophysiology and etiology are neither clear nor defined as yet. Peripheral neuropathic pain originates in the pathological change of nociceptive afferent nervous fibers caused by injured tissue being produced during the microtrauma or macrotrauma creating inappropriate peripheral or central pain signals (Moskowitz, 1984). Neuropathic and neurogenic pain can be expressed having vegetative implications, among other neurovascular manifestations (Ertsey *et al.*, 2004).

Autonomic nervous system (ANS) involvement in producing pain is an ongoing debatable matter. The pain influences and is influenced by the ANS. Trigeminal autonomic cephalalgias (TAC) and Complex Regional Pain

Syndrome are presentations of pain having vegetative symptoms expressed as cranial autonomic symptoms which can be associated or disassociated with pain (Bouhassira *et al.*, 1994; Goadsby *et al.*, 2001; Melis *et al.*, 2002; Martins *et al.*, 2004; Putzki *et al.*, 2004). Such headache pain may be accompanied by considerable autonomic reactions in the head depending on parasympathetic and secondary sympathetic dysfunction (Hardebo, 1984; Goadsby & Edvinsson, 1993; Jänig, 2003; Gottselig & Messlinger, 2004).

TAC consists of three separate presentations: cluster headache, paroxysmal hemicrania and SUNCT (short-lasting unilateral neuralgiform headache with conjunctival injection and tearing). Although cluster headache is known to be a primary headache disorder, various clinical reports have stated that it is a secondary presentation from multiple origins (Tfelt-Hansen *et al.*, 1982; Sacquegna *et al.*, 1982; Kudrow *et al.*, 1984; Romoli & Cudia, 1988; Appelbaum & Noronha, 1989; Gawel *et al.*, 1989; Hannerz, 1989; Formisano *et al.*,

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1990; West & Todman, 1991; Koenigsberg *et al.*, 1994; Cremer *et al.*, 1995; Taub *et al.*, 1995; Muñoz *et al.*, 1996; Evers *et al.*, 1997; Heidegger *et al.*, 1997; Rosebraugh *et al.*, 1997; Piovesan *et al.*, 2001; Soros *et al.*, 2001; Albertyn *et al.*, 2004). Cluster headaches mimicking the appearance of these situations' must be kept in mind, such as neurovascular pain disorders or atypical TAC presentations or neurovascular variants (Rozen, 2002).

Patient complaints almost fulfilled the criteria for a cluster-type headache in this clinical report. The present symptomatology closely resembled a probable cluster headache (IHS ICHD-II, 2004). Cluster headache is an important, but uncommon, type of headache, having an estimated 10-70 per 1,000 people prevalence, whereas migraine is in the 100-150 range. This neurovascular disorder has a rare family history. This pathology commonly begins to presents itself in people aged 30 to 40 and is more frequently present in men than women (86% male preponderance)(Sjaastad & Bakkeiteig, 2003). Ekbohm *et al.* (2002) found significant variance in the male to female ratio regarding age of onset (predominance of males aged 30-49 but more common in females after age 50). The pain is intense and excruciating, having a strictly unilateral location that is commonly manifested in the trigeminal maxillary and ophthalmic areas (oculo-fronto-temporal areas), but can involve other regions (van Vliet *et al.*, 2003). Episodes of pain are abruptly manifested with visual aura reaching maximum level in approximately 15 minutes and lasting up to 3 hours. Ipsilateral signs of autonomic response may usually be present, such as lacrimation, rhinorrhoea, nasal congestion, forehead and facial perspiration, myosis, ptosis, eyelid edema, conjunctival injection and temporal arterial inflammation. The pain is normally periodic and present for 8 weeks, 1 to 8 attacks occurring daily, accompanied by 6-month to 2-year remission periods (Dodick *et al.*, 2000).

CLINICAL REPORT

A 50-year-old woman was sent from an otolaryngology clinic to an orofacial disorders' center as she was suffering from headaches and craniofacial pain. The patient had had severe pain for 7 years with unilateral presentation (right-hand side), which did not subside with pharmacologic (AINES, triptans and indometacine) and oxygen management. The episodes of pain lasted 4 or 5 months with 3- or 4-month remission periods between the painful onsets. The headaches presented an episodic pattern (1 to 3 attacks daily) lasting three to six hours. Present pain intensity was measured on a 1-to-10 Visual Analogue Scale (VAS), 10 being the worst possible pain experienced by a

patient; the patient in question was rated 8 on the VAS. She described the headache as starting during the night while she was asleep and waking her up. There was no history of snoring or episodes of apnea. Intraorbital pain was associated with complete presentation of autonomic signs and symptoms: lacrimation, conjunctival injection, rhinorrhoea, nasal congestion, forehead perspiration, myosis and edema of the eyelid. Additionally, the patient also reported xerostomy, intraoral metallic taste, visual hypostesia and secondary scalp hyperalgesia during the episodes of pain.

Craniofacial pain was exhibited having an anterior-posterior itinerary from the right eye to the ipsilateral suboccipital zone involving the ear and superior maxilla. No cigarette or alcohol consumption was reported. Each episode lasted too long (6 hours) to be a conventional cluster headache (according to diagnostic criteria); a probable cluster headache was therefore diagnosed.

A temporomandibular disorder (TMD) was also found consisting of left-hand side TMJ capsulitis and local muscle soreness in chewing muscles with bilateral temporal tendonitis. Dental evaluation revealed an unstable maxillary denture occluding natural mandible teeth. Prosthesis retention and stability was compromised with compression on the palatine incisive papilla. Moreover, an over-closed over-bite with altered vertical dimension was observed. The incisive papilla was intraorally positioned in the anterior residual ridge crest towards the vestibule area due to severe reabsorbed maxillar alveolar crest (Fig 1). The patient had used five prosthetic sets continuously (24 h) for 20 years and the current prosthesis was 7 years old (when these headaches started).

The maxillary denture rested on reabsorbed maxillary bone and hyper-movable anterior crest with marked incisive papilla. The prosthesis bordered and pressured this papilla irritating this area (Fig. 2). The prosthesis was alleviated in this area during the first appointment to eliminate erythema and inflammation, although the patient did not express pain. This procedure was made because a replacement manufacture of the old prosthesis was accorded with the patient

An appointment was scheduled at the otolaryngology clinic for a sphenopalatine ganglion (SG) block following the above evaluation. The patient had been checked during a previous appointment at the orofacial disorders' center before the SG block procedure was begun; she reported spontaneous healing of her symptomatology. The patient was instructed to estimate her degree of relief on a 1-to-10 VAS scale (10 being the initial pain experienced by the patient). It was established that the pain had diminished from 10 to 1 on the VAS. When asked about referred and autonomic

symptomatology, she stated that total resolution of these symptoms had occurred. The SG block was then stopped due to accidentally the pain finished. The relief of pain was measured during the second week, using VAS again; it was 0 over 10. The patient has been periodically checked over a 5 years and 6 months period since, without recurrence of her pain and autonomic symptomatology.

DISCUSSION

Differential diagnosis must be made with pathologies concerning autonomic system involvement in symptomatic presentation. Trigeminal branch participation accompanied by vegetative symptoms is known as trigeminal autonomic cephalalgias (TAC). Among the closest pathologies,

paroxysmal hemicrania, SUNCT syndrome and neurovascular variants must be discarded because of the similar vegetative symptoms so presented; however, the sudden appearance, localization, intensity, and paroxysmic presentation of these pathologies make the difference. The anatomical connections from the incisive papilla, the vascular patterns and the somatosensorial distribution may clarify the referred symptoms and the pathophysiological justification based on these associations.

Moskowitz affirms that the trigeminal nerve provides algescic afference routes in the pathophysiology and the transmission of headache in humans. Stimulating the trigeminal nerve results in dural vessel dilatation. The ophthalmic and maxillary branches of the trigeminal nerve innervate the cerebral, cerebellar posterior and basilar arteries, as well as the dural and pial arteries and the medial and anterior fossa. Signs of peripheral chronic pain, as in this patient, can arise from adverse conditions affecting the trigemino-vascular neurons thereby producing alteration in the vascular brain flow, no single central origin initiating these headaches' vascular events. Massive vascular effects on efferent nerves can be expected when it is taken into account that the abnormal sensorial and vegetative input coming from the incisive papilla trauma can excite potent vasodilator-mediated afferent nerves (Ertsey *et al.*).

Fig 1. Incisive papilla zone over severe reabsorbed anterior crest.

Fig 2. Prosthesis pressuring incisive papilla zone.

The trauma in the incisive papilla area can involve nociceptive, sympathetic and parasympathetic sensorial impulses. Stimulating the neurovascular network which emerges from the incisive foramen in the incisive papilla area can bring relevant somatosensorial and autonomic consequences. The incisive papilla receive innervation from the trigeminal maxillary branch (V2) taking its autonomic component from the SG. This ganglion receives its autonomic component from the pterygoid channel nerve (vidian nerve). This nerve's sympathetic component (deep petrosal nerve) comes from the common carotid plexus and the superior cervical sympathetic ganglion. The parasympathetic or secretomotor component (greater petrosal nerve from SG) comes from the upper salivatory nucleus having fibers which are shared with the facial nucleus (intermedius nerve). The SG provides vegetative and somatosensorial terminals for the incisive papilla, through the nasopalatine nerve, and a wide neighboring area including the nasal septum, hard palate, soft palate, pharynx, nasal mucous and lachrymal gland areas (Goadsby & Edvinsson).

Sphenopalatine artery (greater incisive artery) septal branch irrigation emerges in the incisive papilla area providing irrigation for the nasal septum; this is mixed with the superior lip and ophthalmic artery and also vasomotor-innervated by the SG. The ganglion is the lachrymal, minor salivary, nasal and pharynx glands' secretomotor component. Activating vegetative neurons in the pterygopalatine ganglion produces cranial blood vessel vasodilatation, secretomotor activation and sensitization of the trigeminovascular afferents (neurogenic inflammation) due to the presence of an inflammatory mediator (Jänig).

The above explains why sympathetic and parasympathic complex neurovascular anatomy stimulation in the palatine area produces autonomic changes resembling a probable cluster headache. Parasympathic stimulation in the area can dilate the intraocular blood vessels and raise blood-pressure. Such vegetative activation also generates ocular conjunctive and nasal vessel expansion. Hardebo has explained how neuron stimulation of the trigeminal nerve in the cornea, iris and around blood vessels (derived from ciliary and conjunctival arteries) causes vasomotor responses in the choroidal artery thereby increasing intraocular pressure and explaining the pain, hypoesthesia and autonomic changes to the patient's right eye. This kind of vegetative activation can also explain changes in perspiration and higher frontal temperature (Gottselig & Messlinger).

The patient told of a diffuse craniofacial pain accompanied by a sensation of disesthesia caused by constant painful stimulus over a 7-year period. The pattern of pain was explained as being a heterotopic presentation, including

projected pain from V2 and referred pain from central excitatory effect coming from the incisive papilla's neurovascular area (Sessle *et al.*). The trigeminal nerve's (subnucleus caudalis) spinal nucleus neurons receive these craniofacial nociceptive signals which can suffer nerve plasticity and mistake the cortical location of the source of pain caused by the sensitization of non-related afferent interneurons.

Muscular involvement further complicated the pain experienced by this particular patient, leading to a more multifaceted symptomatic presentation, making its diagnosis difficult. Such symptomatology can create a state of protective muscular adaptation to prevent the pain, producing a painful cycle of muscle-skeletal dysfunction which can also produce secondary TMD (Lund *et al.*, 1991).

Periodic presentation of cluster and probable cluster headaches stresses the difficulty of emphasizing that remission of pain and its associated autonomic symptomatology in this patient was achieved by the accidental alleviation of the incisal papilla pressure. The above pathophysiological scenario, involving evoked peripheral pain signals integrating ANS, cannot exclude the fact that a change in vegetative headache may be CNS expression reflected in sensory somatomotor, autonomic and neuroendocrine changes due to central somatosensory cortex-mediated abnormalities and hypothalamus-modulating nociceptive input (van Vliet *et al.*). However, the findings from this clinical report provide a valid new perspective in orofacial differential diagnosis pain origin in spite of the probable intraoral cause and prosthesis alleviation procedure seeming so coincidental with the end of the cluster headache that had lasted by 5 years. It is difficult to determine whether or not the pain relief was a coincidence or not but periods of intense headache, followed by liberation from such pain, has not happened again during the last almost five years. Also, it should be stressed that the last prosthesis was worn for 7 years (when these headaches started) and that painful episodes lasted four or five months, with three or four months' remission between painful onsets. Integrating different pathophysiological models (peripheral and central) can be especially remarkable when no unifying explanation of cluster headache is yet available and cavernous sinus hypothesis, periodicity (circadian rhythm), mitochondrial dysfunction and trigeminovascular-parasympathetic pathways have failed as individual justification (Dodick *et al.*).

Diagnosing a cluster headache can be a complicated task due to variable pain localization and its episodic presentation added to possible intraoral traumatic origin. Interdisciplinary management, including a dental specialist in craniofacial pain, offers a key tool to medical staff during these symptoms' conservative phase. Clinical success

depends on each specialist's ability to study the different aspects of the same problem. Doctors specializing in just one discipline cannot always solve a patient's symptomatology by themselves unless aided by the invaluable support of a multidisciplinary management team.

Every specialist contributes his/her specific knowledge towards differential diagnosis addressing a correct treatment plan. This clinical study represents a useful tool in the differential diagnosis origin of painful orofacial neurovascular disorders.

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RESUMEN: Se presenta un caso de cefalea autonómica trigeminal que simula una cefalea en cluster probable con potencial origen traumático intraoral. Mujer de 50 años que reportaba dolor derecho de intensidad severa con 7 años de evolución. El dolor se presenta con síntomas vegetativos como epifora, irritación de la conjuntiva ocular, rinorea, congestión nasal, sudoración frontal, miosis y edema palpebral. Los episodios de dolor se presentaban intermitentemente en el año, con duración de 4 a 5 meses y periodos de remisión de 3 a 4 meses. Las cefaleas se presentaban de manera episódica en el día (1 a 3 episodios) que duraban de 3 a 6 horas. Se observa prótesis intraoral en mal estado, con 7 años de realizada. La paciente reporta la elaboración de 5 juegos protésicos hasta la fecha y que usa constantemente en el día y la noche. Accidentalmente, y antes de la remisión a otro especialista, los síntomas desaparecen después del alivio mecánico en la zona anterior de la prótesis, que comprimía e irritaba la papilla incisiva. La paciente ha estado bajo controles periódicos durante 5 años y medio, sin recurrencia de los síntomas. La posible fisiopatología es discutida.

PALABRAS CLAVE: Ganglio esfenopalatino; Papila incisiva; Dolor neurovascular; Dolor referido; Desórdenes témporomandibulares.

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