Painful tic convulsive

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Hemifacial spasm may be associated with trigeminal neuralgia, geniculate neuralgia, or vestibular and/or cochlear nerve dysfunction.

The syndrome of tic convulsif consists of ipsilateral concurrent trigeminal neuralgia and hemifacial spasm. Cushing described in 1920 this syndrome in three patients ¹⁾

Painful tic convulsif is a syndrome restricted to paroxysmal dysfunction of the fifth cranial nerve and seventh cranial nerves causing trigeminal neuralgia and hemifacial spasm together.

It occurs primarily in women over the age of 50 years and is usually associated with vertebrobasilar dolichoectasia and aneurysm²⁾.

Less frequently an arteriovenous malformation or cholesteatoma-which compresses the trigeminal and facial nerve roots in the posterior fossa. In rare instances this syndrome may be caused by disseminated sclerosis $^{3)}$

Diagnosis

Magnetic resonance imaging (MRI), due to its inherent excellent contrast resolution, is an excellent modality for demonstrating the nerve compression by dilated and tortuous vessels seen in this condition. For this purpose, 3D MRI sequences are especially useful like constructive interference in steady state (CISS) and MR angiography. Both of these have been reported to be helpful in the diagnosis of this condition ⁴.

Mittal et al. report a case of PTC in which they were able to document facial and trigeminal nerve compression by VBD on MRI, using CISS and Time of flight magnetic resonance angiography ⁵⁾.

Ten (6.8%) out of 146 patients with trigeminal neuralgia (TN) who underwent SPGR-MRI and 3D-TOF-MRA from August 1993 to October 1996, were found to have vascular compression caused by a tortuous vertebrobasilar system (TVBS). They were mostly males, demonstrated left-sided predominance, and had ipsilateral hemifacial spasm, compared with other 52 patients whose offending arteries were either superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA)or posterior inferior cerebellar artery (PICA). The patients who showed vascular compression by TVBS, presented an artery which compresses and dislocates the rootentry zone (REZ) of the trigeminal nerve, presses the brain stem at REZ and simultaneously compresses the REZ of the facial nerve. In addition, the diameters of the two branches of vertebrobasilar artery were not equal. These features indicate that the atherosclerotic change of the offending artery in TN caused by TVBS is more severe than that caused by SCA, AICA or PICA. This change causes an irregular running of artery which leads a strong compression of the trigeminal nerve REZ and of the brain stem. Consequently, the facial nerve REZ is severely affected leading to the presence of tic convulsif in TN caused by TVBS⁶.

Treatment

The standard modality of treatment is microvascular decompression, which has shown greater effectiveness and control of symptoms in the long-term. However medical treatment, which includes percutaneous infiltration of botulinum toxin, has produced similar results at medium-term in the control of each individual clinical manifestation, but it must be considered as an alternative in the choice of treatment ⁷⁾.

Case series

2011

Nine consecutive cases of coexistent HFS and TN caused by neurovascular confliction in the same side were studied. Except for one, the patients suffered from HFS followed by ipsilateral TN. All patients underwent MVD and were followed up for 3 to 30 months. Each surgery was analyzed retrospectively.

Intraoperatively, a looped vertebral artery (VA) shifted to the suffered side was found in 8 patients. The VA was regarded as the direct or indirect offending artery. After MVDs, the spasm ceased immediately in 6 patients; the other 3 patients had delayed relief within 3 months. The pain disappeared immediately in 7 of 9 patients. One patient felt relief after a week, and 1 had pain but improved slightly. No recurrence or complication was found.

A shifted VA loop may account for this tic convulsif syndrome. MVD is a reasonable and effective therapy with a high cure rate for the disease. The key to the surgery is to move the VA proximally. The dissection should be performed rostrally starting from the caudal cranial nerves⁸.

2009

Bilateral HFS and tic convulsif were encountered in 7 (0.4%) and 6 (0.37%) patients, respectively. Fifty-six (3.4%) patients were younger than 30 years old at the time of microvascular decompression.

HFS can result from tumor, vascular malformation, and dolichoectatic artery. Therefore, appropriate preoperative radiological investigations are crucial to achieve a correct diagnosis. The authors emphasize that distal compression or only venous compression can be responsible for persistent or

recurrent symptoms postoperatively. In cases of bilateral HFS, a definite differential diagnosis is necessary for appropriate therapy. MVD is recommended as the treatment of choice in patients younger than 30 years old or patients with painful tic convulsif⁹.

2006

Boscá-Blasco et al. report the cases of four patients with combined TN and HFS out of a total of 247 patients with HFS who were treated with botulinum toxin. One patient had TN that was contralateral to the HFS, while the other three were ipsilateral, and one of these had bilateral HFS. In all four cases both the HFS and the TN improved with botulinum toxin treatment.

These four patients with TN and HFS suggest a common aetiology for the two disorders, due either to central neuronal hyperactivity or to vascular compression of several cranial nerves. The beneficial effect of botulinum toxin in both disorders supports the idea of this toxin having a central mechanism of action that acts by controlling neuronal hyperactivity in the brain stem, as well as its peripheral action ¹⁰.

1984

Since Cushing's 1920 description of this syndrome in three patients, 37 additional cases have been reported in the world literature. Of the 15 with adequate operative descriptions, 10 had vascular abnormalities and five had tumors. The authors report 11 cases of tic convulsif treated by microvascular decompression of both the fifth and seventh cranial nerves. At operation, 21 of 22 nerves were found to have root entry zone vascular compression. One trigeminal nerve was considered normal. One seventh nerve had a tumor displacing the anterior inferior cerebellar artery into its root entry zone. The average follow-up period in this series was 6 years 2 months (range 1 to 8 1/2 years). Eight patients (73%) were pain-free, two (18%) had frank recurrences, and one (9%) had mild discomfort. Eight patients (73%) were totally free of facial spasm, and two others (18%) had only a trace of residual spasm. These results are comparable to those achieved by treating the individual syndromes with microvascular decompression. Therefore, microvascular decompression of both the fifth and seventh cranial nerves is recommended as the treatment of choice in tic convulsif ¹¹.

Case reports

2017

Fenech et al. describe a unique presentation of bilateral PTC in a man with bilateral hemifacial spasm and trigeminal neuralgia secondary to neurovascular conflict of all four cranial nerves. Following failed medical and radiofrequency therapy, microvascular decompression of three of the four involved nerves was performed, where the offending vessels were mobilised and Teflon used to prevent conflict recurrence. He continues to respond to Botox for right hemifacial spasm. Since surgery, he remains pain free bilaterally and spasm free on the left¹².

2014

Rare case of cerebello-pontine angle meningioma causing painful tic convulsif¹³.

2013

Jiao et al. report a case of a 77-year-old woman with coexistent trigeminal neuralgia and hemifacial spasm who had experienced Bell palsy half a year ago. The patient underwent microvascular decompression. Intraoperatively, the vertebrobasilar artery was found to deviate to the symptomatic side and a severe adhesion was observed in the cerebellopontine angle. Meanwhile, an ectatic anterior inferior cerebellar artery and 2 branches of the superior cerebellar artery were identified to compress the caudal root entry zone (REZ) of the VII nerve and the rostroventral cisternal portion of the V nerve, respectively. Postoperatively, the symptoms of spasm ceased immediately and the pain disappeared within 3 months. In this article, the pathogenesis of the patient's illness was discussed and it was assumed that the adhesions developed from inflammatory reactions after Bell palsy and the anatomic features of the patient were the factors that generated the disorder. Microvascular decompression surgery is the suggested treatment of the disease, and the dissection should be started from the caudal cranial nerves while performing the operation ¹⁴.

2012

Verghese et al. report an Posterior fossa arachnoid cyst that caused PTC in a 50-year-old woman. Her radiological evaluation revealed a median, well-circumscribed, cystic lesion of the posterior fossa suggestive of arachnoid cyst, pushing the cerebellum and brainstem anteriorly. Midline suboccipital craniotomy and marsupialization of cyst was performed with complete recovery of symptoms. This is the first report of a retrocerebellar arachnoid cyst causing PTC¹⁵.

Painful tic convulsif caused by an arteriovenous malformation ¹⁶.

2011

Giglia et al. present the case of a 50-year-old man suffering from "painful tic convulsif", on the left side of the face, i.e., left trigeminal neuralgia associated with ipsilateral hemifacial spasm. An angio-MRI scan showed a neurovascular confliction of left superior cerebellar artery with the ipsilateral V cranial nerve and of the left inferior cerebellar artery with the ipsilateral VII cranial nerve. Neurophysiological evaluation through esteroceptive blink reflex showed the involvement of left facial nerve. An initial carbamazepine treatment (800 mg/daily) was completely ineffective, so the patient was shifted to lamotrigine 50 b.i.d. that was able to reduce attacks from 4 to 6 times per day to 1 to 2 per week. Considering the good response to the drug, the neurosurgeon decided to delay surgical treatment ¹⁷.

2009

A 67-year-old woman who presented with a typical left hemifacial spasm of 8-month duration. After 2 months, she experienced lacinating and sharp shock-like pain in the left side of her face affecting the

V1 and V2 territories and a discrete attenuation of nauseous reflex on the left side. CT angiography and MRI revealed significant compression of left cranial nerves V, VII, VIII, IX and X by a giant and tortuous vertebro-basilar arterial complex. This case illustrates the nonlinearity of the relationship between the presence of the stressor factor and the actual manifestation of the disease ¹⁸.

2007

A case of right-sided HFS after which left TN developed, which is an unusual form of PTC. Both disorders were caused by bilateral vascular compression of the cranial nerves and successfully treated with botulinum toxin and carbamazepine. As PTC is benign in nature and can be treated with botulinum toxin, neuroradiological investigations should be performed for an accurate aetiological diagnosis, particularly in young patients with atypical disease manifestations ¹⁹.

Bilateral hemifacial spasm and trigeminal neuralgia: a unique form of painful tic convulsif²⁰.

2004

A 80-year-old woman had a 10-year history of left trigeminal neuralgia and ipsilateral hemifacial spasm. She presented with intermittent left facial twitching and pain, especially upon swallowing. MRI revealed compression of the left trigeminal nerve by the left anterior inferior cerebellar artery and of the ipsilateral facial nerve by the posterior inferior cerebellar artery. Microvascular decompression of the lesions via left lateral suboccipital craniotomy resulted in immediate and complete symptom improvement. The case demonstrates that different arteries can affect the trigeminal and facial nerve at a stage that precedes compression by a tortuous vertebrobasilar artery. They suggest that the presence of PTC should be considered in patients with a tortuous vertebrobasilar artery, irrespective of the offending arteries ²¹⁾.

2002

A 70-year-old man with hemifacial spasm associated to trigeminal neuralgia secondary to an ectatic basilar artery. He was treated with botulinum toxin type A, 2.5 mouse units over five sites at the orbicularis oculi and one over the buccinator muscle. After botulinum toxin injections, relief was gained not only from twitching but also from pain. When the effects of the toxin vanished, spasms and pain recurred. Further infiltrations were given every 12 weeks following the same response pattern. This observation further validates the increasing role of botulinum toxin in pain management ²².

2001

A case is presented of painful tic convulsif caused by schwannoma in the cerebellopontine angle (CPA), with right trigeminal neuralgia and ipsilateral hemifacial spasm. Magnetic resonance images showed a 4 cm round mass displacing the 4th ventricle and distorting the brain stem in the right CPA. The schwannoma, which compressed the fifth and seventh cranial nerves directly, was subtotally removed by a suboccipital craniectomy. Postoperatively, the patient had a complete relief from the

hemifacial spasm and marked improvement from trigeminal neuralgia. The painful tic convulsif in this case was probably produced by the tumor compressing and displacing the anterior cerebellar artery directly ²³⁾.

1995

A case of painful tic convulsif (trigeminal neuralgia and ipsilateral hemifacial spasm) caused by cerebellopontine angle epidermoid tumor is presented. This tumor was compressed to the trigeminal nerve, and became attached to the facial and auditory nerves. The facial nerve exit-zone of brain stem was also compressed by the tumor along with a branch of the posterior inferior cerebellar artery. Total removal of the tumor was carried out and neuralgia and facial spasm disappeared. Painful tic convulsif caused by brain tumor is rare (eight cases in the literature plus our case), but epidermoid tumor is not rare as a cause of this complaint (seven in eight cases). In preoperative examination of this case, we could not detect this epidermoid in the cerebellopontine angle, because this tumor was the same intensity as CSF liquid on magnetic resonance imaging (T1 and T2 weighted image) and exerting hardly any mass effect on the brainstem. On encountering a case of painful tic convulsif of unknown origin despite the usual preoperative examinations, it may be useful that same kind of brain tumor, especially, epidermoid might be concealed in the cerebellopontine angle lesion ²⁴.

A case is presented of painful tic convulsif caused by a posterior fossa meningioma, with right trigeminal neuralgia and ipsilateral hemifacial spasm. Magnetic resonance images showed an ectatic right vertebral artery as a signal-void area in the right cerebellopontine angle. At operation the tentorial meningioma, which did not compress either the fifth or the seventh cranial nerves directly, was totally removed via a suboccipital craniectomy. The patient had complete postoperative relief from the trigeminal neuralgia and her hemifacial spasm improved markedly with decreased frequency. From a pathophysiological standpoint, the painful tic convulsif in this case was probably produced by the tumor compressing and displacing the brainstem directly, with secondary neurovascular compression of the fifth and seventh nerves (the so-called "remote effect") ²⁵⁾.

Painful tic convulsif caused by a brain tumor undiagnosed preoperatively ²⁶.

1992

Patient with painful tic convulsif caused by a brain tumor. The patient was admitted with right trigeminal neuralgia and ipsilateral facial spasm, i.e., painful tic convulsif. Preoperative computed tomography scans showed no apparent abnormalities; however, surgery revealed that these symptoms were associated with a pearly tumor located in the cerebellopontine angle. Subtotal resection for the decompression of the right trigeminal and facial nerves was performed and resulted in complete relief of the symptoms. Histological examination demonstrated the tumor to be an epidermoid cyst ²⁷⁾.

1991

A 77-year-old woman had developed trigeminal neuralgia 12 years before admission and ipsilateral facial spasm 2 years before admission. Upon operation, the superior cerebellar artery was found to impinge upon the entry zone of the fifth nerve. In addition, the anterior inferior and posterior inferior cerebellar arteries were found to bend along the seventh nerve. Teflon sheets were placed between the nerves and offending arteries. She has been pain-free and spasm-free for the past 18 months. Pathomechanism of the association of the multiple compression syndromes and the treatment are discussed ²⁸⁾.

The case of trigeminal neuralgia and ipsilateral hemifacial spasm-painful tic convulsif-is presented. Microsurgical exploration revealed compression of the fifth and seventh cranial nerves by a tortuous contralateral vertebral artery. Neurovascular decompression of the roots entry/exit zone completely relieved preoperative facial pain and spasm²⁹.

1989

A case of epidermoid tumor presenting with a painful tic convulsif was reported. A 35-year old male with trigeminal neuralgia and ipsilateral hemifacial spasm was diagnosed as having an epidermoid by CT and metrizamide CT cisternography and the symptoms were completely eliminated after the operation. In this case, metrizamide CT cisternography was very useful for preoperative diagnosis by demonstrating the characteristic findings of the epidermoid. It should be taken into consideration that there are some cases with trigeminal neuralgia and/or hemifacial spasm whose symptoms are due to brain tumors ³⁰.

1984

A patient had combined otalgia and intractable unilateral facial spasm, relieved by microsurgical vascular decompression of the seventh and eighth cranial nerve complex in the cerebellopontine angle without section of the intermediate nerve. A dolicho-ectatic anterior inferior cerebellar artery compressed the seventh and eighth cranial nerves complex, suggesting that vascular compression of the intermediate nerve or of the sensory portion of the facial nerve may cause geniculate neuralgia. "Tic convulsif" seems to be a combination of geniculate neuralgia and hemifacial spasm. This combination could be due to vascular compression of the sensory and motor components of the facial nerve at their junction with the brainstem³¹.

1983

A 49-year-old man with an epidermoid tumor had a hemifacial spasm on the left and ipsilateral trigeminal neuralgia-i.e., painful tic convulsif. Computed tomography scanning after metrizamide enhancement clearly demonstrated a cerebellopontine angle tumor. In the year since complete removal of the epidermoid tumor, the patient has been relieved of the facial pain and the hemifacial spasm is improved with decreased frequency of the spasm ³².

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