Symptomatic Trigeminal Autonomic Cephalalgias

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Abstract  Trigeminal autonomic cephalalgias (TACs) are primary headache syndromes that share some clinical features such as a trigeminal distribution of the pain and accompanying ipsilateral autonomic symptoms. By definition, no underlying structural lesion for the phenotype is found. There are, however, many descriptions in the literature of patients with structural lesions causing symptoms that are indistinguishable from those of idiopathic TACs. In this article, we review the recent insights in symptomatic TACs by comparing and categorizing newly published cases. We confirm that symptomatic TACs can have typical phenotypes. It is of crucial importance to identify symptomatic TACs, as the underlying cause will influence treatment and outcome. Our update focuses on when a structural lesion should be sought.

Keywords  Cluster headache · Trigeminal autonomic cephalalgia · Paroxysmal hemicrania · Hemicrania continua · Short-lasting unilateral neuralgiform headache with conjunctival tearing · Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms

Abbreviations  
TACs  Trigeminal autonomic cephalalgias
ICHD  International Classification of Headache Disorders
SUNCT  Short-lasting unilateral neuralgiform headache with conjunctival tearing
SUNA  Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms
MRI  Magnetic resonance imaging

Introduction  
Trigeminal autonomic cephalalgias (TACs) are primary headache syndromes that owe their name to the trigeminal distribution of the pain and the accompanying ipsilateral autonomic symptoms, as defined by the International Classification of Headache Disorders (ICHD)-III beta criteria [1]. The most prevalent TAC is cluster headache, but the category also includes rare diseases such as paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA), and hemicrania continua.

It is well known that an underlying structural lesion can lead to TAC symptoms, which cannot easily be differentiated from those of idiopathic TACs [2, 3•]. Recognizing these underlying pathologies is of crucial importance, as they can influence treatment and outcome. Here, we will give an update of recently published cases with an underlying structural lesion and a TAC phenotype.
| Authors | Publication year | Age (year) | Sex | Headache phenotype | Duration of CH symptoms | Atypical features | Underlying lesion | Treatment | Follow-up | Outcome |
|---------|------------------|------------|-----|-------------------|-------------------------|------------------|-------------------|-----------|-----------|---------|
| Probably symptomatic cluster headache | | | | | | | | | | |
| Edvardsson [13] | 2014 | 49 | M | Cluster headache | 1 month | Nausea, photophobia, and phonophobia | Non-functioning pituitary adenoma (chromophobe adenoma) | Sumatriptan SC, oxygen, verapamil, surgery | 17 months | Pain free |
| Malissart et al. [19] | 2014 | 60 | F | Cluster headache | 3 days | – | Ipsilateral carotid panganglioma | Surgery | Unknown | Pain free |
| Edvardsson et al. [10] | 2013 | 43 | M | Cluster headache | 2 months | Nausea and photophobia/phonophobia | Intrasellar arachnoid cyst | Craniotomy with cyst fenestration | 4 months | Pain free |
| Edvardsson et al. [12] | 2013 | 21 | M | Cluster headache | 3 weeks | – | Maxillary sinusitis | Antibiotics and sinus puncture | 4 years | Pain free |
| Levy et al. [17] | 2012 | 25 | M | Cluster headache | 3 months | – | Ipsilateral pituitary macroadenoma | Cabergoline | Unknown | Pain free |
| Edvardsson et al. [9] | 2012 | 41 | M | Cluster headache | 3 months | Nausea and photophobia/phonophobia | Ipsilateral glioblastoma multiforme | Surgery | 12 months | Pain free |
| Ranieri et al. [22] | 2009 | 39 | M | Cluster headache | 14 years | Maxillary pain next to periorbital pain, continues daily pain during last 7 months, tooth grinding, and frequently waking up at night | Obstructive sleep apnoea diagnosis 14 years after CH diagnosis | Intra-oral device | 12 months | Pain free |
| Sewell et al. [24] | 2009 | 34 | M | Cluster headache | 17 years | At moment of consultation, restless legs syndrome and numbness in fingers | Stroke caused by moyamoya | Two cranial bypasses | 6 years | Pain free |
| Edvardsson [11+] | 2013 | 24 | M | Cluster headache | 4 weeks | – | Acute maxillary sinusitis | Antibiotics and sinus puncture | Several years | Pain free |
| Fontaine et al. [14] | 2013 | 27 | M | Cluster headache | 4 months | – | Ipsilateral hemangiopericytoma | Surgery | 9 months | Pain free |
| Vander Vliet et al. [26] | 2013 | 31 | M | Cluster headache | 2 months | Diffuse headache next to the attacks | Sarcoidosis (also hypothalamic lesion) | Prednisone course | 7 months | Pain free |
| Créac’h et al. [8] | 2010 | 44 | F | Cluster headache | 7 months | Trigger factor: rotation of head to the right | Neurovascular compression caused by fibrosis surrounding both C3 and right vertebral artery | Verapamil for 6 months, microvascular dissection | 2.5 years | Pain free |
| Possibly symptomatic cluster headache | | | | | | | | | | |
| Candeloro et al. [6] | 2013 | 39 | M | Cluster headache | 21 years | Once attack duration of >3 h | Dissection of the right distal internal carotid artery | Heparin | 6 months | Unknown |
| Mijajlović et al. [21] | 2014 | 45 | M | Cluster headache | 7 days | – | Multiple sclerosis | Methylprednisolone course with afterwards verapamil for 1 year | 3 years | Pain free |
| Gil-Gouveia et al. [15] | 2013 | 79 | F | Cluster headache | – | – | 48 h after lens phacoemulsification and intraocular lens implant | Verapamil, sodium valproate, oxygen | 9 months | Decrease in attack frequency |
| Messina et al. [20] | 2013 | 27 | M | Cluster headache | – | – | Angiomyolipoma | Hyperthalamic deep brain stimulation | Unknown | Decrease in attack frequency |
| Donat [2] | 2011 | 33 | M | Cluster headache | – | – | Multiple sclerosis | Verapamil | 10 months | Pain free |
| Choi et al. [7] | 2009 | 52 | F | Cluster headache | 10 years | Attacks sometimes on both sides. This time also blurred vision and central horizontal scotoma | Recurrent posterior scleritis and aseptic meningitis | Prednisone course | 2 months | Unknown |
| Benitez-Rosario et al. [5] | 2009 | 41 | M | Cluster headache | 12 months | Depressive symptoms | Ipsilateral macroadenoma | Cabergoline, hormonal replacement, prednisone course, verapamil | About 1–2 months | Pain free |
Methods

In 2009, our group published a comprehensive update of symptomatic TACs, reported until mid-February 2009 [4]. With this report as starting point, we conducted a PubMed search from February 2009 to January 2015 with the following key words: trigeminal autonomic cephalalgia, cluster headache, hemicrania continua, SUNCT, SUNA, paroxysmal hemicrania, secondary, and symptomatic. Only articles written in English were included of which the full text was available. Cases were divided into three categories: probably secondary, possibly secondary, and unknown. Cases were defined as probably secondary when there was a dramatic improvement of the headache after treatment of the underlying lesion. Cases were defined as possibly secondary when the patient was treated but did not become headache free, or was not treated, but where a causal relation was possible based on previous experience with other patients. Efficacy of indomethacin was not considered as treatment response in paroxysmal hemicrania and hemicrania continua, as this is one of the diagnostic criteria and not specifically aiming at an underlying lesion. The category unknown was used for patients in which a causal relation between the phenotype and the lesion was less likely or at least unclear: in most cases, the patient was not treated and a causal relation between the lesion and the TAC was unlikely on anatomical grounds and/or a probable incidental finding.

Results

Cluster Headache

We found 23 cases with a cluster headache-like phenotype in 23 articles [2, 5, 6*, 7–10, 11*, 12–26]. We excluded 3 patients, as they did not fulfill the ICHD-III criteria beta version, all having an attack duration of more than 3 h. We also excluded another patient who did not have a structural lesion [16]. This resulted in 19 patients of whom 12 could be categorized as probably secondary and 7 as possibly secondary (Table 1).

Of the 12 cases in the probably secondary category, 5 had a neoplasm [15, 19, 21, 27, 28]: a non-functioning pituitary adenoma, an ipsilateral carotid paraganglioma, an ipsilateral prolactinoma, an ipsilateral glioblastoma multiforme, and an ipsilateral hemangiopericytoma. A vascular cause, a stroke secondary to moyamoya disease, was found in 1 patient [29]. Other patients had an intrasellar arachnoid cyst, maxillary sinusitis (n=2), compression of the right vertebral artery by fibrosis, sarcoidosis (with a hypothalamic lesion), and obstructive sleep apnoea [8, 10, 11*, 12, 22, 26].

There were seven cases defined as possibly secondary. Multiple sclerosis was found in two, of whom both became pain free under verapamil or prednisone, which are used as prophylactic cluster headache medication and therefore are not strictly aiming at the underlying lesion [2, 21]. Another
| Authors                      | Year | Age  | Sex | Headache phenotype | Duration symptoms | Atypical features                                                                 | Underlying lesion                                      | Treatment                                                                 | Follow-up | Outcome                       |
|------------------------------|------|------|-----|--------------------|------------------|-----------------------------------------------------------------------------------|---------------------------------------------------------|----------------------------------------------------------------------------|-----------|-------------------------------|
| Probably symptomatic SUNCT/SUNA |      |      |     |                    |                  |                                                                                   |                                                         |                                                                            |           |                               |
| Favoni et al.[49]            | 2013 | 53   | F   | SUNCT              | 3 years          | –                                                                                 | Compression of trigeminal nerve by right superior cerebellar artery       | Microvascular decompression trigeminal nerve                | 11 months | Pain free                     |
| Chitsanikul et al.[37]       | 2013 | 45   | M   | SUNCT              | 3 years          | Improvement by vigorous activity                                                   | Ipsilateral mixed gangliocytoma and pituitary adenoma                   | Surgery                                  | 4 years   | Pain free                     |
| Chitsanikul et al.[37]       | 2013 | 51   | F   | SUNCT              | 4 years          | Right arm and facial numbness during attacks, irregular menstruation, decrease in libido, galactorrhoea | Ipsilateral pituitary adenoma                                             | Surgery                                  | 18 months | Improvement in frequency and intensity |
| Civen et al.[38]             | 2013 | 57   | F   | SUNCT              | 3 years          | –                                                                                 | Aneurysm                                                               | Surgery                                   | Unknown   | Pain free                     |
| Domingos et al.[40]          | 2012 | 46   | M   | SUNCT              | 3 months         | Blurred vision outside attack                                                       | Compression trigeminal nerve by superior cerebellar artery               | Microvascular decompression                      | 1 year    | Pain free                     |
| Guerriero et al.[42]         | 2009 | 57   | M   | SUNCT/SUNDAT        | 3 months         |                                                                                   | Ipsilateral macroadenoma                                                | Surgery                                  | Unknown   | Pain free                     |
| De Louvres et al.[39]        | 2009 | 50   | M   | SUNCT              | 4 years          | –                                                                                 | Ipsilateral epidermoid tumour in cerebellopontine angle                  | Cabergoline                               | 7 months   | Pain free                     |
| Rodgers et al.[46]           | 2013 | 33   | M   | SUNCT              | 6–8 months       | Triggered by head movements, chewing, jaw opening                                  | Compression trigeminal nerve by superior cerebellar artery               | Gabapentin, duloxetine, pregabalin, phenoxybarbital, morphine, steroids, carbamazepine all ineffective, afterwards surgery | 6 months   | Pain free                     |
| Williams et al.[3•]          | 2010 | 71   | M   | SUNCT              | 6 years          |                                                                                   | Compression trigeminal nerve by superior cerebellar artery               | Lamotrigine, carbamazepine, gabapentin, baclofen, and prednisolone without benefit, surgery | 32 months   | Pain free                     |
| Williams et al.[3•]          | 2010 | 54   | M   | SUNCT/SUNA         | 1–2 months       | Compression trigeminal nerve by superior cerebellar artery | Lamotrigine, valproic acid, and topiramate without benefit, surgery | Surgery                                  | 32 months   | Pain free                     |
| Williams et al.[3•]          | 2010 | 46   | M   | SUNCT              | 3 years          | Compression trigeminal nerve by superior cerebellar artery | Lamotrigine and carbamazepine without benefit, surgery | Lamotrigine, prednisolone, and morphine without benefit, surgery | 30 months   | Pain free                     |
| Williams et al.[3•]          | 2010 | 56   | M   | SUNA                | 1 year          | Compression trigeminal nerve by superior cerebellar artery | Lamotrigine and carbamazepine without benefit, surgery | Lamotrigine and topiramate without benefit, surgery | 9 months   | Pain free                     |
| Williams et al.[3•]          | 2010 | 49   | M   | SUNCT              | 5 years          | Compression trigeminal nerve by superior cerebellar artery | Lamotrigine and carbamazepine without benefit, surgery | Lamotrigine and topiramate without benefit, surgery | 20 months   | Pain free                     |
| Possibly symptomatic SUNCT/SUNA |      |      |     |                    |                  |                                                                                   |                                                         |                                                                            |           |                               |
| Favoni et al.[49]            | 2013 | 55   | M   | SUNCT              | 9 years          | –                                                                                 | Compression trigeminal nerve by superior cerebellar artery               | Gabapentin, verapamil, pregabapentin, and iv corticosteroids course, indomethacin for 1 month without effect, response on carbamazepine | Unknown   | Pain free                     |
| Chitsanikul et al.[37]       | 2013 | 25   | F   | SUNCT              | 6 years          | –                                                                                 | Ipsilateral pituitary adenoma                                           | Indomethacin, lamotrigine, topiramate, carbamazepine, gabapentin, oxycodone, and greater occipital nerve block all without effect, surgery | 1 year    | No improvement                |
| Chitsanikul et al.[37]       | 2013 | 56   | F   | SUNCT              | –                | –                                                                                 | Ipsilateral pituitary tumour                                           | Surgery                                  | 6 months   | No improvement                |
| Chitsanikul et al.[37]       | 2013 | 30   | F   | SUNCT              | 12 years         | –                                                                                 | Ipsilateral pituitary adenoma                                           | Surgery                                  | 20 years   | No improvement                |
| Cascella et al.[29]          | 2011 | 57   | F   | SUNCT              | 1 month          | –                                                                                 | Lung adenocarcinoma                                                     | Greater occipital nerve block and indomethacin without effect, valacyclovir, and prednisone course, chemotherapy, gabapentin | 5 months   | Pain free                     |
| Kutchenko et al.[44]         | 2010 | 81   | F   | SUNCT              | 5 months         | –                                                                                 | Ipsilateral meningioma                                                  | Gabapentin                               | Unknown   | Pain free                     |
| Bogorad et al.[36]           | 2010 | 61   | F   | SUNCT              | 2 years          | –                                                                                 | Multiple sclerosis                                                      | Carbamazepine, steroids, and indomethacin                            | 1 day     | Pain free                     |
the outcome after treatment remained unclear [6]. Other diagnoses in this category are as follows: recurrent posterior scleritis and a specific meningitis (treated with prednisone), post-operative cluster headache (lens phacoemulsification and intraocular lens implant), an angiomyolipoma, and an ipsilateral macroprolactinoma [5, 7, 15, 20]. The latter two patients responded completely or partly to treatment of the underlying lesion, but only in combination with preventive cluster headache treatment.

**Paroxysmal Hemicrania**

We identified three cases of paroxysmal hemicrania, of whom all were excluded as they did not fulfil the ICHD-III criteria beta version [27, 30, 31]. The missing criterion in two patients was an unknown response to indomethacin [30, 31], and the third reported bilateral instead of unilateral facial pain [27].

**Hemicrania Continua**

We identified seven cases [28, 32–35] of symptomatic hemicrania continua of whom one was excluded as the patient did not receive indomethacin [28]. We categorized two cases as *probably symptomatic*, three as *possibly symptomatic*, and one as *unknown* (Table 2).

The underlying lesions in the cases defined as *probably symptomatic* were a cerebral venous thrombosis and brain metastases of a primary lung adenocarcinoma [33, 35]. Both patients responded to treatment of the underlying cause, and indomethacin could be withdrawn.

In patients defined as *possibly symptomatic*, the possible causes were post-traumatic and twice post-operative [34]. All received indomethacin as treatment for their hemicrania continua.

One case was classified as *unknown*. This patient was diagnosed with an orbital pseudotumour, treated with prednisone and indomethacin [32].

**SUNCT and SUNA**

We found 29 cases of SUNCT and SUNA [3•, 29, 36–48] of whom 1 was excluded because of bilateral pain during the attacks [48]. There were 14 cases defined as *probably symptomatic*, 12 as *possibly symptomatic*, and 2 as *unknown* (Table 3).

Most cases were defined as *probably symptomatic*. SUNCT was most often compression of the trigeminal nerve by an artery (8 out of 14), followed by malignancies as a mixed gangliocytoma, an epidermoid tumour, and prolactinomas [3•, 37, 39, 42, 46, 49]. Furthermore, an aneurysm and cavernous sinus dural fistula were found [38, 40]. All patients responded completely to treatment of the underlying cause, which was most often surgery.
Tumours were most often the underlying cause in the category possibly symptomatic SUNCT/SUNA: an ipsilateral prolactinoma (n=2), an ipsilateral pituitary tumour, a lung adenocarcinoma, and an ipsilateral meningioma [29, 37, 44]. Furthermore, trigeminal nerve compression (n=4), multiple sclerosis, a mild hypothalamic-pituitary dysfunction by optical nerve hypoplasia, and a viral meningitis were reported. Five patients became pain free under preventive SUNCT treatment [3*, 36, 43, 47, 49].

There were two cases categorized as unknown. One patient developed a varicella zoster virus meningoencephalitis 1 week after the SUNCT attacks and died within several weeks from arrhythmia secondary to myocarditis, likely as consequence of the viremia [41]. In the other patient, a small posterior skull and a cerebellar hypoplasia, without dysplasia, were found. A causal relation between the development of SUNCT and this anomaly is uncertain [45].

Conclusion

The goal of this review was to give an update on underlying structural lesions associated with TACs, published between February 2009 (since the last review) and January 2015. We identified 53 typical cases: 19 cases with cluster headache, no cases with paroxysmal hemicrania, 6 cases with hemicrania continua, and 28 cases with SUNCT/SUNA.

Tumours were reported in 16 of the 53 cases diagnosed with a TAC, mainly pituitary tumours. Prolactinomas were found in 2 cluster headache and 4 SUNCT patients, followed by pituitary adenomas (n=2). It has indeed been reported that pituitary tumours account for a large portion of the secondary causes of SUNCT [50]. The other way around, various types of headache including TACs have been reported as a frequent symptom of pituitary tumours [51]. An association between the side of the tumour and side of the headache has been suggested [52••]. In most of the reported cases of secondary SUNCT and secondary cluster headache, surgery or medical treatment of the pituitary tumour resulted in improvement.

A vascular lesion as an underlying cause was less often found. An intracranial or extracranial dissection was reported in only 1 of the 19 cluster headache patients. This patient was diagnosed with cluster headache several years before he experienced a cluster headache attack with prolonged duration, which was probably caused by a carotid dissection [6*]. Dissection as a cause for cluster headache is rare but has been reported in earlier reviews [4, 50]. Recognition is of crucial importance as it can have serious consequences for patients. Cases with carotid dissection have shown improvement of the headache after antiaggregant or anticoagulant therapy. Most patients did not even need preventive cluster headache treatment. Repeated contrast-enhanced magnetic resonance imaging (MRI) should be considered if the characteristics of the headache attacks change over time.

In 12 SUNCT patients, a trigeminal nerve compression by vascular structures as possible cause of SUNCT was found. Eight of 11 surgically treated patients became headache free, whereas only 3 patients had no benefit of the procedure. This is an important finding as SUNCT is often considered medically intractable. Trigeminal nerve compression was found in 42.8 % of this series.

A sinusitis was considered probably causal in 2 cluster headache patients. Sinusitis is a common misdiagnosis in cluster headache. Lainez et al. showed that 14 of 75 cluster headache patients (18.7 %) were initially misdiagnosed as having a sinusitis [53••]. It is sometimes very difficult to make a clear distinction between sinusitis and a TAC [54].

In summary, we found 53 typical cases of secondary TACs in our literature study covering the period from February 2009 to January 2015. Secondary underlying lesions seem to be rare in TACs. However, physicians should be aware of possible underlying pathology, as, for example, prolactinomas or glioblastomas, arteriovenous malformations, dissections, and various inflammations can cause a TAC-like phenotype. In our opinion, not only a contrast-enhanced cerebral MRI should be considered once in every patient to exclude a causal underlying pathology but also imaging of cervical vascular structures.

Most of our findings are in accordance with those of Wilbrink et al. [4]. Of additional importance is the more recent observation that in more than 40 % of patients with SUNCT/SUNA, a trigeminal nerve compression by the superior or inferior cerebellar artery was present and that most of these patients experienced spectacular improvement of their headache after surgical decompression. In contrast to other reviews, we found less frequently an intracranial or extracranial dissection causing cluster headache [4, 50]. This could be explained by the fact that there are already various case reports about intracranial and extracranial dissections causing cluster headache [55–57]. The importance of a cerebral MRI to exclude underlying lesions is shown in the current review, as cerebral lesions (e.g. pituitary tumours) were associated with TACs.

Compliance with Ethics Guidelines

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