Review Article

Secondary (Symptomatic) Trigeminal Autonomic Cephalalgia

Debashish Chowdhury
Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, New Delhi, India

Abstract

Primary trigeminal autonomic cephalalgias (TACs) are uncommon group of headache disorders. These are defined and diagnosed by the criteria given by the International Classification of Headache Disorders 3β version. Over the past few decades, a number of secondary (symptomatic) cases have been described in the literature with headache features indistinguishable from primary TACs. Many structural and other pathologies have been found in these patients that can be causally related to the headaches. This review attempts to critically analyze the existing literature including the new cases published during 2015–2017.

Keywords: Cluster-like headache, secondary short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing/short-lasting unilateral headache attacks with autonomic features, secondary cluster headache, secondary hemicrania continua, secondary paroxysmal hemicrania, secondary trigeminal autonomic cephalalgias, symptomatic trigeminal autonomic cephalalgias

Introduction

Trigeminal autonomic cephalalgias (TACs), the term coined by Goadsby and Lipton, is now considered a broad rubric under which four types of primary headache disorders are included (Group 3 in International Classification of Headache Disorders 3β version [ICHD3β]).[1,2] These include cluster headache (CH), paroxysmal hemicrania (PH), short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) and short-lasting unilateral headache attacks with autonomic features (SUNA) (under short-lasting unilateral neuralgiform headache attacks), and hemicrania continua (HC). Although considered uncommon, these headaches are one of the worst pain syndromes known to humankind.

As the nosology and the classification of TACs evolved over the years, many secondary cases were described with an underlying structural pathology. Sometimes, headache features in these secondary TACs were undistinguishable from those with primary TACs, and sometimes, they only apparently mimicked the primary TACs and their secondary nature was belied due to the presence of atypical features. Some excellent reviews are available detailing these cases from 1975 to 2015.[3-8] Some reviews focused only of secondary CH,[4,7] CH being the most common of TACs subtypes. Secondary HC has been included only in one review as it was included in TACs group only in 2013.[8] The purpose of the present article is to critically review the concept of secondary TACs and analyze the new secondary TACs and TACs-like cases reported from February 2015 to July 2017.

Concept of Secondary Headache in the Context of Secondary Trigeminal Autonomic Cephalalgias

For the diagnosis of secondary headache due to a disorder, the causative disorder must be known to cause headache. When new headache occurs in close temporal relationship with such a disorder, it is diagnosed as a secondary headache even though the phenotype is of a primary headache disorder.[2] Although close temporal relationship suggests causation, ICHD3β requires fulfillment of at least one or more of the following conditions, namely worsening and/or improvement of headache with worsening and/or improvement of the presumed causative disorder; headache having characteristics typical of a causative disorder (for example, thunderclap headache for subarachnoid hemorrhage); and existence of other evidence of causation (for example, a biochemical marker-like erythrocyte

Address for correspondence: Dr. Debashish Chowdhury, Room No 504, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, New Delhi - 110 002, India.
E-mail: debashishchowdhury@hotmail.com

Access this article online

Quick Response Code:

Website: www.annalsofian.org

DOI: 10.4103/aian.AIAN_16_18

How to cite this article: Chowdhury D. Secondary (Symptomatic) trigeminal autonomic cephalalgia. Ann Indian Acad Neurol 2018;21:S57-69.

sedimentation rate [ESR] for giant cell arteritis [GCA] or location of headache).[2] Two other situations may also exist. When a preexisting primary headache becomes chronic or becomes significantly worse (more than twofold increase in attack frequency and/or severity) in close temporal relationship with a causative disorder, both the primary and secondary diagnoses are recommended. In the context of TACs, it has been difficult to establish causality with the underlying pathology in many cases. This is because of many reasons. First, because of retrospective nature of most of the case reports, temporal relationship was difficult to establish. Further, imaging was done in a cross-sectional manner. Detection of an abnormality on magnetic resonance imaging (MRI) in a patient at the time of presentation or during one point in patient's subsequent follow-up does not necessarily provide definitive clues about its onset or its progression. A typical example is the presence of pituitary adenoma in many TAC patients. Are these incidental or causal? As pituitary micro-adenomas and macro-adenomas can be found in as high as 1% and 0.2%, respectively, of the population who are asymptomatic,[9] detection of pituitary tumor in a case of TACs does not necessarily point toward causality. Most of the case reports relied on the fact that headache resolved after the treatment of underlying pathology although time of follow-up was not stated in many cases. There may be other reasons for the response such as natural fluctuations or placebo effect. Many cases of secondary CH had responded to the existent treatment (as for the primary CH) as well. Many patients of secondary PH and HC have responded well to indomethacin before their response to the treatment of underlying pathology. Therefore, it is difficult to hold onto this criterion alone for demonstrating causality. To obviate this difficulty, some authors have divided these cases into three groups, namely probable secondary, possible secondary, and unknown.[8]

Overview of the Previously Published Reports

The previous reviews are summarized in Table 1. Although these reviews had varying search modalities and inclusion and exclusion criteria, certain general points which emerged from them are worth noting.

1. Nearly half of the patients had "typical" features of TACs fulfilling ICHD2 and/or ICHD3β criteria despite having a structural intracranial or extracranial pathology to account for their headaches. Hence, it has been suggested that neuroimaging should be done in all patients of TACs.[3,6,8] Although this appears appealing, some authors have argued that definitive recommendations for neuroimaging cannot be generalized to all TAC patients because of retrospective nature of the case reports and small number of cases.[4,5,7] As mentioned previously, even if a structural pathology is found, cause-and-effect relationship is difficult to establish in many cases. There are no population-based studies available to throw light on this issue. On the other hand, since many of the etiologies for secondary TACs are potentially serious, others have argued that neuroimaging should be routinely offered to such patients. Fortunately, TACs are relatively uncommon (as compared to migraine and tension-type headache), and hence, the economic burden for such investigations may not be much of a concern although "incidentalomas" picked up during routine neuroimaging may be potentially confusing
2. Nearly half of the secondary TAC patients also had "atypical features," sometimes very obvious, and sometimes too trivial to attract attention of the clinicians. All TAC patients must therefore be carefully scrutinized for the presence of any "atypical" features and be subjected to investigations if any of these are detected. These atypical features may be present at the time of presentation or may develop subsequently. High index of suspicion must therefore be kept for any atypicality
3. Certain red flags have been identified which must prompt for further investigations. These include older age at onset, abnormal general or neurological examination, attack-related features not fulfilling ICHD criteria, namely duration, frequency, and localization, and unresponsiveness to treatment
4. Many reports also describe patients with TAC-like headaches which only superficially exhibit TAC features. Their onset, duration, frequency, and presence of additional neurological symptoms and signs clearly mark them as mere mimickers rather than true TACs. For example, Mainardi *et al*. identified 156 cluster-like headaches from 1975 to 2008.[10] Eighty were excluded because of inadequate information. Of the remaining 76, only 38 (50%) fulfilled the diagnostic criteria of CH as per the ICHD2
5. About one-third of secondary TAC patients had "episodic pattern" of headaches. Hence, contrary to common belief, even episodic TACs patients can have a secondary cause and should be considered for imaging
6. Overall, the reported occurrence of secondary TACs has been low. However, exact proportion is difficult to ascertain as the reported cases are in the form of case reports and small series, and none of them reported the number of primary TACs cases seen during the same period. Furthermore, lower detection could be due to the fact that previously many of these patients were not imaged. There is a trend toward greater reporting of secondary TACs over the years
7. Intracranial tumors are probably the most common causes for secondary TACs, although in the some recent reviews their proportion has decreased (around 30% as compared to 50% in earlier reviews).[7,8] Among the tumors, the occurrence of pituitary tumors is particularly high. Most of these pituitary tumors are functioning tumors and removal/medical treatment of these has resolved headaches in most cases for at least a certain period in follow-up, thereby lending credence to the supposition of causal relationship
8. Another important cause of secondary TACs is vascular abnormalities mainly in the form of dissections and

Table 1: Summary of the reviews on symptomatic trigeminal autonomic cephalalgias (1975-2015)

Number	Author	Journal/ year	Title	Period of search	Number of cases	Comments	Etiology
1	Favier *et al*.[3]	Arch Neurol., 2007	Trigeminal autonomic cephalgias because of structural lesions: A review of 31 cases	January 2001-December 2005	31	27 previously described cases and 4 cases of their own; 27 cases were excluded. There were 16 cases of CH/PH; 1 cluster tic; 4 SUNCT and rest 10 had atypical TACs	8 had vascular lesions, 16 had cerebral tumors (11 pituitary tumors; 10 functioning) and rest miscellaneous lesions
2	Favier *et al*.[4]	Curr Pain Headache Rep. 2008	Cluster headache: To scan or not to scan	2001-2008	23 (21+2)	2 more CH cases were added	Both had tumors; 1 had pituitary tumor
3	Cittadini and Matharu[5]	The Neurologist 2009	Symptomatic trigeminal autonomic cephalalgias	1975-May 2007	40	24 patients of CH. Of these, 12 had typical and 12 had atypical features; 1 had CH-Tic. 50% responded to abortive treatment; 3 patients with PH; All had atypical features; 10 cases of SUNCT; 5 had atypical features	12 (50%) of CH patients had tumors; 7/12 had pituitary tumors (6 functioning) and 33% vascular lesions. All PH patients had mass lesions; 2 had functioning pituitary adenomas. All PH patients had absolute response to indomethacin. All SUNCT patients had mass lesions; 7 had pituitary adenomas (5 functioning)
4	Wilbrink *et al*.[6]	Curr Opin Neurol. 2009	Neuroimaging in trigeminal autonomic cephalgias: when, how, and of what?	January 2001-2008	56	(33 from reviews of 1 and 2) and 23 additional cases. Described TACs and TACs-like syndromes	27 had tumors (more than 50% were pituitary tumors), 22 had vascular lesions and the rest miscellaneous causes
5	Edvardsson[7]	Springer Plus 2014	Symptomatic cluster headache: A review of 63 cases	From 1993 to May 2013	63	Focused only on symptomatic CH	28 patients had vascular lesions (11 had dissections); 25 had tumors (10 pituitary tumors); 48% had typical presentations while 52% had atypical presentations
6	de Coo *et al*.[8]	Curr Pain Headache Rep 2015	Symptomatic trigeminal autonomic cephalalgias	February 2009-January 2015	53	Updated series from review 4; 53 typical cases were identified; 19 cases with CH, no cases with paroxysmal hemicrania, 6 cases with hemicrania continua, and 28 cases with SUNCT/SUNA; classified as probable, possible and unknown	16 of the 53 cases had tumors (mainly pituitary tumors); more than 40% of patients with SUNCT/SUNA had neurovascular conflict and that most experienced spectacular improvement after surgical decompression

TACs=Trigeminal autonomic cephalalgias, CH=Cluster headache, PH=Paroxysmal hemicranias, SUNCT=Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing, SUNA=Short-lasting unilateral headache attacks with autonomic features

aneurysms, although their reported occurrence had been low. These are potentially sinister etiologies and hence should not be missed. The third important cause is intracranial infections

9. Response to treatment has been a tricky issue in secondary TACs. In many reports, the casual relationship with the detected pathology was demonstrated by showing the resolution of the headache by an intervention aimed at the pathology. However, other possibilities may exist such as spontaneous resolution of an episodic disease (intervention being coincidental), natural fluctuations in disease severity, or placebo effect. Further, a large number of patients of secondary TACs have responded to the standard existent treatment aimed at the primary disorder. Hence, it is difficult to judge whether the effect produced by an intervention was indeed significant. Only long-term follow-up could answer that. Unfortunately, many reports lacked the details of long-term follow-up after intervention
10. There are increasing case reports of the presence of neurovascular conflict in SUNCT/SUNA, and many such patients had dramatic improvement in their headaches following microvascular decompression (MVD). Favoni *et al*.[11] in a review identified 222 cases of SUNCT/SUNA and found neurovascular conflicts in 37 (16.7%). Sixteen patients underwent MVD and 75% reported complete pain relief. While some authors have considered cases of SUNCT/SUNA with vascular loop on the symptomatic

side as secondary, there is no mention in ICHD3β about the nosology of this entity. Interestingly, cases of trigeminal neuralgia (TN) with neurovascular conflict are considered as "classical" in ICHD3β.

New Cases of Secondary Trigeminal Autonomic Cephalalgias (January 2015–July 2017)

Secondary cluster headache

We found 10 new reports for secondary CH and CH-like headaches [Table 2].

Tumors

De Pue *et al*.[12] described a 47-year-old male who suffered from primary right-sided chronic CH for 12 years, never attack-free for more than 1 month. His initial MRI scan was normal. Subsequently, the patient developed continuous interictal pain at the same side, and repeat MRI showed a cystic pituitary lesion consistent with either a Rathke's cleft cyst or cystic adenoma with high prolactin levels. On cabergoline, his headaches were completely resolved.

Robin *et al*.[13] described a very interesting case of CH being "cured" by a glioblastoma. A 49-year-old male had refractory right chronic CH (CCH) for 4 years who failed various preventive treatments, blocks, and occipital nerve stimulation. He refused deep brain stimulation. Subsequently, he developed fresh neurological symptoms in the form of dizziness, ataxia, and hemiparesis. However, interestingly, it was found that 2–3 weeks before the onset of these neurological symptoms, his chronic headaches had stopped. Glioblastoma multiforme was detected involving right cingulate gyrus. Following operation, he only had few mild attacks for 1½ months which completely resolved thereafter till he succumbed to his illness 1 year later.

Escuti[14] described a case of 50-year-old man who 9 years back had generalized headaches to an occipital lipoma, resection of which provided him with considerable albeit not full relief. Following this, for the past 2 years, the patient started having CH-like attacks. Repeat imaging showed regrowth of the lipoma at C1–C2 level. Headaches were relieved by sumatriptan injections. The lipoma was not operated and the patient reported decrease in attacks following significant weight loss.

Vascular

Eswaradass *et al*.[15] reported a case of 44-year-old male patient who presented with episodic right-sided headaches for 1 month, 1–3 times a day with narrowing of palpebral fissure and mild lacrimation. The patient had restlessness and agitation during the episodes. There was no significant response to analgesics and 100% oxygen. MRI brain was normal. Digital subtraction angiography revealed right indirect carotid cavernous fistula. Following endovascular embolization, the patient had complete relief in headache.

Bellamio *et al*.[16] reported two cases of secondary CH due to vascular etiology. First was a 71-year-old male who had features of left-sided episodic cluster for the past 20 years. He had been taking various preventives with moderate relief. An MRI scan at some point in his illness showed a left pontine cavernous angioma, which was considered an incidental finding. Then, few years later, his headache type changed suddenly from episodic CH (ECH) to CCH without any response to preventive medications. Repeat MRI showed increase in size of the cavernous angioma. Surgery resolved his headaches completely, and he remained headache-free till 6 years of his last follow-up. The second patient was a 29-year-old male with right-sided CH. His first cluster period lasted for 2 weeks with attacks occurring at fixed times of the day with accompanied by autonomic symptoms. Three months later, he presented with the next cluster period of 1 month with similar features. After 1 month, he developed fever, meningeal irritation, and features of intracranial hypertension. His MRI showed left external jugular vein thrombosis climbing up to sigmoid sinus. Treatment with warfarin and acetazolamide successfully resolved his headache. He was pain-free at 1 year.

de Coo *et al*.[17] reported a patient with cluster-Tic syndrome. Initially, the patient had only TN. After 6 months, she started developing redness and tearing. She did not respond to indomethacin. One year later, she developed CH attacks occurring up to four per day, each lasting 30–90 m. Previous two MRIs were reported as normal. However, review of the last MRI showed compression of the left trigeminal nerve by the petrosal vein. Vascular decompression abolished the TN and decreased CH attacks although they did not disappear completely.

Semnic *et al*.[18] reported a case of 49-year-old male with features superficially resembling left-sided ECH. However, there were no autonomic symptoms associated with headache attacks and restlessness was not mentioned. Furthermore, mild numbness and hyperalgesia of the affected area were present. Similar headache occurred 3 years back for 45 days with complete resolution of symptoms. MRI showed segmental cavernous carotid ectasia. Treatment details were not mentioned.

Miscellaneous

Kao and Hsu[19] reported a case of 26-year-old male who presented with a thunderclap-like headache involving right forehead precipitated by cough. Subsequently, the headache persisted for 2 weeks in episodic form lasting 2–3 h, 3–4 times/day and got precipitated by cough and exertion. It was associated with rhinorrhea and tearing from right eye. Subsequently, he developed persistent anhidrosis of his right face and trunk. In addition, he developed persistent numbness of right half of the body. MRI showed caudal descent of cerebellar tonsils compression of posterolateral aspect of the right cervical spinal cord and syringomyelia. His headaches resolved completely after decompression surgery.

Pelikan *et al*.[20] reported a case of 42-year-old female with sudden-onset left-sided headache with ipsilateral autonomic symptoms. She visited the emergency department (ED)

Table 2: Secondary cluster headache and cluster headache-like headaches (February 2015 to June 2017)

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment before the diagnosis of secondary pathology	Treatment after the diagnosis of secondary pathology	Outcome/ follow-up duration after the treatment of the pathology
1	Bellamio *et al.* (2017)[16]	71/ male	ECH-CCH	Left	20 years	Sudden change of ECH to CCH	Pontine cavernous angioma	Left	ECH responded to conventional treatment; CCH did not respond	Surgery	Headache resolved completely/6 years
2	Bellamio *et al.* (2017) [16]	29/ male	ECH	Right	4 months	Sudden change in headache character; fluctuating vision loss; meningeal irritation, fever, raised ICP	External jugular vein thrombosis	Left	Not mentioned	Acetazolamide and warfarin	Headache resolved completely/1 year
3	de Coo *et al.* (2017)[17]	41/ female	Cluster-Tic	Left	6 years	Initially, attacks of TN occurred only upon standing from sitting	Petrosal venous compression of trigeminal nerve	Left	CH attacks responded to oxygen; verapamil had not effect	Surgery	TN resolved completely. CH frequency and intensity decreased/ follow-up period not mentioned
4	De Pue *et al.* (2016)[12]	47/ male	CCH	Right	12 years	Change in headache characteristic; continuous interictal headache	Rathke's cleft cyst/ cystic adenoma (prolactinoma)	Right	Relief with sumatriptan injections; modest response with verapamil	Cabergoline	CCH stopped; 6 months later probable SUNA evolved/1.5 years
5	Pelikan *et al.* (2016)[20]	42/ female	ECH	Left	4 months	Numbness in lip, face, and tongue; ataxia and clumsiness of left hand	MS; Demyelinating plaques; one large plaque in right centrum semiovale	Right	Responded to dihydroergotamine	Dimethyl fumarate	Headache resolved completely/1 year
6	Escuti (2015)[14]	50/ male	ECH	Right	2 years	Previously operated for occipital lipoma for generalize headaches	Regrowth of lipoma at C1-C2 level	Left	Relief with sumatriptan injections	No further surgery offered	Headache decreased in severity on weight reduction/ spontaneously
7	Kao and Hsu (2015)[19]	26/ male	ECH	Right	2 weeks	Thunderclap type presentation followed by episodic headaches; precipitated by cough, exertion; sensory loss; anhidrosis	Type 1 Chiari malformation	Right cord compression	Not mentioned	Decompression	Complete relief in headache/few days
8	Robin *et al.* (2015)[13]	49/ male	ECH-CCH	Right	4 years	Change of headache character to CDH; 2-3 weeks prior had dizziness, ataxia and hemiparesis	GBM involving cingulated gyrus	Right	Failed standard treatment, blocks and ONS; refused DBS	Headache subsided 2-3 weeks before development of fresh neurological symptoms. Resective surgery performed	Headache disappeared completely following 6 months of surgery

Contd...

Table 2: Contd...

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment before the diagnosis of secondary pathology	Treatment after the diagnosis of secondary pathology	Outcome/ follow-up duration after the treatment of the pathology
9	Semnic *et al.* (2015)[18]	49/ male	ECH	Left	1 month; similar headache 3 years back for 45 days	Mild numbness and hyperalgesia of the affected area	Segmental cavernous carotid ectasia	Left	Not mentioned	Not mentioned	Not mentioned
10	Eswaradass *et al.* (2015)[15]	44/ male	ECH	Right	1 month	No significant response to 100% oxygen	Indirect carotid-cavernous fistula	Right	No relief with analgesics and 100% oxygen	Endovascular embolization	Headache resolved completely/not mentioned

*At the time of diagnosis of secondary pathology. ECH=Episodic cluster headache, CCH=Chronic cluster headache, ICP=Intracranial pressure, CDH=Chronic daily headache, GBM=Glioblastoma multiforme, SUNA=Short-lasting unilateral headache attacks with autonomic features, TN=Trigeminal neuralgia, ONS=Occipital Nerve Stimulation, MS=Multiple sclerosis, DBS=Deep brain stimulation

where she was given dihydroergotamine and her headaches resolved. However, she developed numbness on the left side of lips and facial numbness in the mandibular and maxillary areas. Two months later, other neurological symptoms such as hand clumsiness and gait ataxia developed. She continued to have similar headache attacks. Her MRI showed multiple demyelinating plaques including a large plaque in the right centrum semiovale. She was started on dimethyl fumarate and her headaches completely resolved.

Secondary paroxysmal hemicrania

We found three new reports for secondary PH and PH-like headaches [Table 3].

Ljubisavljevic *et al*.[21] reported a 40-year-old female patient who presented with two types of right facial pain for 2 years. The first type was TN consisting of paroxysm of attacks in V2/V3 distribution and was triggered by various stimuli; the second one was throbbing orbital and frontal pain with ipsilateral autonomic symptoms such as conjunctival injection, lacrimation, and aural fullness. This pain lasted most often between 15 and 20 m. Thus, a diagnosis of CPH-Tic syndrome was considered. On MRI, there were multiple hyperintense paraventricular lesion and hyperintense lesion in the right trigeminal main sensory nucleus and root inlet. All were hypointense on T1 and did not show any enhancement on contrast. Thus, a diagnosis of clinically isolated syndrome presenting as CPH-Tic syndrome was made. Headaches were completely relieved by indomethacin and lamotrigine at 6 months.

Choi *et al*.[22] described a 43-year-old male presenting with severe paroxysmal left periocular and frontal headaches 10–12 times/day lasting 10–15 m with ipsilateral conjunctival congestion and lacrimation for 1 year. Initial MRI was normal. The patient did not get relief with indomethacin, various other drugs, and occipital and sphenopalatine ganglion blocks. A repeat MRI 1 year later showed left superior oblique mass. On further evaluation, it was found to be metastatic leiomyosarcoma arising from a primary focus in the right thigh. The patient had complete relief of his headache following gamma knife surgery of his orbital leiomyosarcoma.

Taga *et al*.[23] reported isolated attacks of headache resembling PH after tadalafil and sildenafil (phosphodiesterase-5 inhibitors) administration in a 35-year-old man. The patient was a case of Alcock syndrome (pudendal nerve entrapment) and had erectile dysfunction. Indomethacin completely relived these headaches triggered by tadalafil and sildenafil.

Secondary short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing/short-lasting unilateral headache attacks with autonomic features

We found 10 new reports for secondary SUNCT/SUNA and SUNCT/SUNA-like headaches [Table 4]. We excluded the case reports with neurovascular conflict.

Vascular

Lambru *et al*.[24] described a case of 58-year-old male with right-sided chronic SUNCT and TN for 16 years which

Table 3: Secondary paroxysmal hemicranias and paroxysmal hemicranias-like headaches (February 2015 to June 2017)

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment before the diagnosis of secondary pathology	Treatment after the diagnosis of secondary pathology	Outcome and follow-up duration after the treatment of the pathology
1	Ljubisavljevic *et al.* (2017)[21]	40/ female	CPH-Tic	Right	2 years	Hypoesthesia in the region of right maxillary and mandibular nerve	Multiple demyelinating lesions especially at right trigeminal main sensory nucleus and root inlet and right corticospinal tract at the medulla oblongata presenting as CIS	Right	Partial response to carbamazepine and amitriptyline	Indomethacin and lamotrigine	Complete pain relief at 6 months
2	Choi *et al.* (2017)[22]	43/ male	CPH	Left	1 year	None	Orbital (left superior oblique) metastatic leiomyosarcoma arising from the thigh	Left	No response to indomethacin, other drugs and SPG and occipital blocks	Gamma knife	Complete pain relief at 3 months
3	Taga *et al.* (2017)[23]	35/ male	EPH	Right	Not mentioned	Always provoked by tadalafil, sildenafil intake	Phosphodiesterase inhibitors administration	Not applicable	No response to analgesics	Indomethacin/ not mentioned	Complete relief

CIS=Clinically isolated syndrome, CPH=Chronic paroxysmal hemicranias, SPG=Sphenopalatine ganglion, EPH=Episodic primary headache, *At the time of diagnosis of secondary pathology

started after 3 weeks of right dorsolateral medullary stroke due to vertebral artery (VA) dissection. Recent MRI showed bilateral neurovascular conflict without any compression, indentation, or distortion. The patient could not tolerate lamotrigine and had only 40% response to carbamazepine and gabapentin.

Jin *et al*.[25] reported a case of 64-year-old male presented with left SUNCT attacks 13 days, following a stroke involving left dorsolateral medulla (VA occluded). His symptoms resolved spontaneously after 14 days.

Liapounova *et al*.[26] described a case of 15-year-old male who presented to the ED with right SUNCT-like attacks. There was a history of self-limiting similar attacks 2 years back. His MRI showed right pontine capillary telangiectasia and developmental venous anomaly abutting right pons. Surprisingly, initially, there was partial response to oxygen inhalation; later, he responded to carbamazepine.

Gocmen *et al*.[27] reported a case of 43-year-old male with left-sided headaches with autonomic features for 1 month. Headaches started following lifting of heavyweight. He had these attacks 20–30 times a day, lasting 5–10 min. The headache was accompanied by ipsilateral tearing and conjunctival hyperaemia but no phonophobia, photophobia, nausea, or vomiting. His MRI showed left pontine capillary telangiectasia. He had only partial response to indomethacin but responded to bilateral greater occipital and left supraorbital nerve block with methylprednisolone and lidocaine.

Miscellaneous

Berk *et al*.[28] described a 33-year-old female who had an episode of pituitary apoplexy in 2010 from hemorrhage in a pituitary mass. She recovered and remained asymptomatic with hormonal therapy. In 2012, she started having dull holocephalic headaches and a surveillance MRI showed the presence of the pituitary mass abutting right cavernous sinus which was partially excised. The histological diagnosis was pituitary adenoma. She subsequently received radiation therapy. Within 1 month of the radiotherapy, she developed up to 40 attacks of sharp right-sided headaches lasting 60–120 s, with autonomic features. These attacks were provoked by alcohol intake, physical or emotional stress, insomnia, and orgasm. She partially responded to lamotrigine.

Mathew *et al*.[29] reported two cases of SUNCT secondary to postherpetic infection of V1 distribution of trigeminal nerve. The first patient a 58-year-old man developed SUNCT attacks 1 year following the infection, while the second patient, a 60-year-old man developed it after 1 month. They responded well to pregabalin and lamotrigine.

Nagel *et al*.[30] reported a 47-year-old man who developed left ophthalmic distribution zoster, treated with oral valacyclovir for 7 days. Two days after discontinuing valacyclovir, he experienced SUNCT headaches which responded to a repeat course of valacyclovir. He remained asymptomatic for 2 years. Thus, this case describes an association of SUNCT with overt zoster.

Table 4: Secondary short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing/short-lasting unilateral headache attacks with autonomic features and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing/short-lasting unilateral headache attacks with autonomic features-like headaches (February 2015 to June 2017)

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment of headache before the diagnosis of secondary pathology	Treatment after the diagnosis of secondary pathology	Outcome and follow-up duration after the treatment of the pathology
1	Lambru *et al*. (2017)[24]	58/ male	Chronic SUNCT and TN	Right	16 years (started 3 weeks after stroke)	None	Infarct in right dorsolateral medulla due to VA dissection; bilateral neurovascular conflict without any compression, indentation or distortion	Right	Multiple drugs	Carbamazepine and gabapentin; could not tolerate lamotrigine	40% relief
2	Liapounova *et al*. (2017)[26]	15/ male	Episodic SUNCT	Right	Diagnosed after admission in ED; history of self-limiting previous episode 2 years back	Mild hyperesthesia right side of the face	Right pontine capillary telangiectasia and developmental venous anomaly	Right	Not applicable	Initially, partial response to oxygen; later carbamazepine	Complete response; 1 year
3	Mangaraj *et al*. (2017)[32]	22/ female	Episodic SUNCT	Left	10 years	Secondary amenorrhea, galactorrhea	Macroprolactinoma	Whole of pituitary with suprasellar extension	Not mentioned	Cabergoline; pituitary mass also decreased on drug	Complete response; 1 year
4	Mathew *et al*. (2016)	58/ male	Episodic SUNCT	Right	1 year	None	Herpes zoster in V1 (previous)	Right	Not applicable	Pregabalin and lamotrigine	Complete relief; 5 years
5	Mathew *et al*. (2016)[29]	60/ male	Episodic SUNCT	Left	1 month	None	Herpes zoster in V1 (previous)	Left	Not applicable	Pregabalin	Complete relief; 3 months
6	Nagel *et al*. (2016)[30]	47/ male	Episodic SUNCT	Left	1 month	None	Herpes zoster in V1 (acute infection)	Left	Valacyclovir	Repeat course of valacyclovir	Complete relief; 2 years
7	Berk and Silberstein (2016)[28]	33/ female	Episodic SUNCT	Right	1 month	None	Postradiation to a pituitary adenoma invading cavernous carotid artery	Right	Not applicable	Lamotrigine	Partial response
8	Jin *et al*. (2016)[25]	64/ male	Episodic SUNCT	Left	13 days	None	Infarct in left dorsolateral medulla; left VA occluded	Left	Not applicable	No drug given	Spontaneous resolution of headache within 14 days; 5 months

Contd...

Table 4: Contd...

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment of headache before the diagnosis of secondary pathology	Treatment after the diagnosis of secondary pathology	Outcome and follow-up duration after the treatment of the pathology
9	Rojas-Ramirez (2016)[31]	57/ female	SUNCT	Left	2 years	Palpation of left trapezius resulted in attacks; no other triggers	Head and neck trauma	None	None	Lamotrigine (could not tolerate); gabapentin and melatonin	Significant response; 2 years
10	Gocmen *et al.* (2015)[27]	43/ male	SUNCT	Left	1 month	History of lifting heavy objects just before the start of the headache attacks; transient left-sided sixth nerve palsy	Pontine capillary telangiectasia	Left	Not mentioned	Partial response to indomethacin; followed by bilateral greater occipital and left supraorbital nerve block with methylprednisolone and lidocaine	Complete response; 8 months

SUNCT=Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing, TN=Trigeminal neuralgia, VA=Vertebral artery, ED=Emergency department, *At the time of diagnosis of secondary pathology

Rojas-Ramirez *et al.*[31] reported a 57-year-old female who following a violent head-and-neck trauma sustained 2 years back developed (time of onset from the trauma not mentioned) recurrent attacks (4–30/day) of shooting sharp pain on left temporal and supraorbital areas lasting 10–60 s with autonomic features. MRI brain was normal. She had significant response to gabapentin and melatonin.

Tumor

Mangaraj *et al.*[32] reported a case of 22-year-old female with secondary amenorrhea who had SUNCT-like headaches occurring 15 days to 1 month for 10 years. She had macroprolactinoma which was treated with cabergoline and her headaches completely resolved in 3 months.

Secondary hemicrania continua

We found 11 new reports for secondary HC and HC-like headaches [Table 5].

Vascular

Brilla *et al.*[33] described five cases of HC-like headaches following cervical artery dissection, in three cases with specific indomethacin response. In two cases, comorbidity of fibromuscular dysplasia was noted. However, all cases had atypical features for HC. The first case was a 50-year-old Caucasian female with subacute onset of headaches over the span of a day, initially in the left ear and occiput, then behind her left eye. Her magnetic resonance angiography (MRA) showed a left internal carotid artery (ICA) dissection from the bifurcation to the petrous segment. She returned 7 weeks after symptoms onset because her constant left-sided headaches had worsened, and she complained of hypersensitivity of the scalp in the temporoparietal area. There was mild swelling of the inferior periorbital area and mild ptosis. She had complete relief with indomethacin. The second case was a 38-year-old man with a 4-day history of right-sided temporo-orbital headache of moderate-to-severe intensity accompanied by eyelid and temporal edema, ptosis, and miosis. The headache responded completely to indomethacin. Ultrasound and later MRI and MRA confirmed the diagnosis of right ICA dissection. The third case was a 44-year-old physician who one morning noted Horner's syndrome in the mirror. MRI and MRA of the head and neck confirmed the dissection. The patient was treated with warfarin. Two days later, the patient returned complaining a continuous right-sided periorbital headache with episodic worsening accompanied by lacrimation and more pronounced signs of Horner's syndrome and a feeling of sweating in the right face. There was complete relief with indomethacin 200 mg/day. The fourth case was a 47-year-old right-handed female who noted to have a small pupil on the right side; she later also noted intermittent ipsilateral ptosis, especially when tired. The next day, she developed a sudden-onset frontotemporal headache. She was found to have an extensive dissection of the right ICA extending from the C2/3 level into the skull base. The fifth case was a 42-year-old male patient who presented with left retro-orbital headaches occurring for 3 weeks since he lifted a heavyweight. There was background

Table 5: Secondary hemicrania continua and hemicrania continua-like headaches (February 2015 to June 2017)

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment before the diagnosis of secondary pathology	Treatment	Outcome and Follow-up duration after the treatment of the pathology
1	Brilla *et al.* (2017)[33]	50/ female	HC	Left	Few days	Pain in left ear and occiput to begin with	Carotid dissection with co-morbid FMD	Left	Gabapentin/ NSAIDs	Indomethacin; later withdrawn due to FMD-related renal involvement	Complete relief while on indomethacin/1 year
2	Brilla *et al.* (2017)[33]	44/ male	Probable HC	Right	Headache started later	Presented initially with only Horner's; headache started 2 days later	Carotid dissection	Right	Warfarin	Indomethacin after 2 days	Complete relief; indomethacin stopped after 3 months
3	Brilla *et al.* (2017)[33]	47/ female	Probable HC	Right	Headache started later	Presented initially with only miosis and intermittent ptosis; headache started 1 day later	FMD, carotid dissection, ACOM aneurysm	Right	NA	Stenting and clipping	Headache resolved in 6 months with analgesics
4	Brilla *et al.* (2017)[33]	42/ male	Probable HC	Left	3 weeks	Headache started after lifting heavyweight	Occlusion of the ICA, with intramural hematoma indicating dissection	Left	NA	Methylprednisolone and acetaminophen	Headache relief in 4 weeks
5	Russo *et al.* (2017)[35]	62/ male	HC	Left	1 year	Initially raised ESR; after 1 year, left 6th nerve palsy	Widespread but asymmetrical pachymeningeal thickening (more prominent in the left side) and left transverse and sigmoid cerebral sinus thrombosis	Left	Steroids, indotest followed by oral indomethacin	Methylprednisolone and enoxaparin	Headache resolved/2 weeks
6	Mainardi *et al.* (2017)[36]	66/ male	Probable HC	Right	4 months	None	TNP	NA	None	Withdrawal of TNP	Relief in headache within 24 h
7	Zhang *et al.* (2017)[34]	31/ male	HC	Left	4 months	Left 6th nerve palsy on follow up after 1 month	Nasopharyngeal carcinoma	Left	Complete response to indomethacin	Chemoradiotherapy	Headache gradually ceased
8	Alim-Marvasti *et al.* (2016)[38]	40/ male	Side-switching HC	Left to right to left	3 months	Abnormal optic disc appearances and lack of absolute response to indomethacin	Recurrent alternating scleritis	Left > right	NSAIDs, triptans, partial response to indomethacin	Prednisolone	Complete relief; follow-up not mentioned
9	Gantenbein *et al.* (2015)[37]	44/ male	Probable HC	Left	3 months	Started 3 months following cranial surgery	Operated for vestibular schwannoma	Left	NSAIDs	Indomethacin	Complete relief in headache; 4 months

Contd...

Table 5: Contd...

Number	Author (year)	Age/ sex	Headache phenotype	Laterality of headache	Duration of headache diagnosis*	Atypical features	Underlying pathology	Laterality of the pathology	Treatment before the diagnosis of secondary pathology	Treatment	Outcome and Follow-up duration after the treatment of the pathology
10	Gantenbein *et al.* (2015)[37]	26/ male	HC	Left	Few days	Started few days after cranial surgery	Sided selective amygdalohippocampectomy for refractory temporal lobe epilepsy with secondary osteomyelitis with 2 revisions of skull bone	Left	NSAIDs	Indomethacin	Complete relief in headache; not mentioned
11	Gantenbein *et al.* (2015)[37]	51/ female	HC	Right	4 months	Started 4 months following cranial surgery	Operated for vestibular schwannoma	Right	NSAIDs, triptans	Indomethacin, gabapentin	Complete relief in headache with indomethacin; could not tolerate; partial response to gabapentin

Case 2 of the series published by Brilla *et al.* was originally published in 2005. Details are included in the text but not in this table as the period under consideration are from 2015-2017. HC=Hemicrania continua, FMD=Fibromuscular dysplasia, NSAIDs=Nonsteroidal anti-inflammatory drugs, ACOM=Anterior communicating artery, NA=Not available, ICA=Internal carotid artery, ESR=Erythrocyte sedimentation rate, TNP=Transdermal nitroglycerine patch, *At the time of diagnosis of secondary pathology

continuous headache radiating to the left temporal and parietal area, with superimposed stabbing attacks, with a frequency of 5–10 times/day, with duration of 30 s, and they could be triggered by forced looking to the left and could be stopped by intense squinting of the left eye. There was increased lacrimation during the attacks. Computed tomography angiography and MRI/MRA of the head revealed occlusion of the left ICA, with intramural hematoma indicating dissection. The headache was relieved by intravenous methylprednisolone, with the occasional addition of acetaminophen.

Tumor

Zhang *et al*.[34] described a 31-year-old man presented with a 4-month history of continuous left temporal pain with frequent exacerbations. The exacerbations were associated with ipsilateral conjunctival injection and a sense of restlessness. The pain entirely resolved with indomethacin. His initial MRI had very subtle changes and nasopharyngoscopy was noncontributory. However, a repeat contrast MRI a month later showed a tumor in the nasopharynx which on biopsy turned out to be nasopharyngeal carcinoma. After chemotherapy, his headaches ceased.

Miscellaneous

Russo *et al*.[35] described a case of a 62-year-old man with 12 months of constant moderate pain, strictly localized in the left orbital and temporal regions with throbbing exacerbations and with ipsilateral conjunctival injection, tearing, and mild ptosis. His examination and plain MRI brain were normal. ESR was raised. He was treated as a case of GCA, but he did not improve with steroids. His subsequent contrast MRI revealed hypertrophic pachymeningitis. All secondary causes of hypertrophic pachymeningitis were ruled out. He responded to oral methylprednisolone and azathioprine.

Mainardi *et al*.[36] described a case of 66-year-old man, with a history of migraine who developed a new headache for 4 months. It was dull, mild frontoparietal headache with several severe exacerbations daily, lasting 45–60 min, accompanied by ipsilateral lacrimation, ptosis, and nose stuffiness; onset of headaches had temporal relationship with transdermal nitroglycerine patch (TNP), recommended for coronary heart disease (unstable angina). With temporary withdrawal of TNP, the headache disappeared within a day. The reintroduction of TNP after 10 days brought the reappearance of the headache within hours; replacement of TNP by ranolazine resulted in permanent resolution of headaches.

Gantenbein *et al*.[37] reported a series of three patients who developed a continuous hemicranial headache after cranial surgery. The first was a 44-year-old male who 3 months after excision of a left-sided vestibular schwannoma started had continuous pain without any cranial autonomic symptoms. He responded completely to indomethacin. The second patient was a 26-year-old man was treated for refractory temporal lobe epilepsy with a left-sided selective amygdalohippocampectomy with extirpation of temporomesial dysplasia. He had a skull bone infection with osteomyelitis,

which had to be revised with two more operations. A few days after last intervention, he developed an ongoing left-sided headache with moderate-to-severe pain with ptosis and lacrimation of the left eye during pain exacerbations. He responded completely to indomethacin. The third patient was a 51-year-old female who 4 months after resection of right-sided vestibular schwannoma developed a continuous right-sided headache of moderate intensity. With exacerbations of the pain, she reported ipsilateral lacrimation and facial hot flushes. She was pain-free on indomethacin.

Alim-Marvasti *et al*.[38] described a 40-year-old right-handed woman who woke up with a severe left-sided persistent sharp headache associated with unilateral lacrimation. The pain was mainly orbitofrontal. A few days later, her left eye appeared red. The unilateral left-sided headache was unremitting for 2 weeks with superimposed exacerbations. Then the pain, lacrimation and red-eye switched to the right side for a further 2 weeks before reverting to the left eye. Her optic disc showed drusens. All her investigations were normal, except the presence of oligoclonal bands in the cerebrospinal fluid. B-scan ultrasound scan confirmed a diagnosis of scleritis (left > right). Although not mentioned in the treatment part, authors mention in the discussion that the patient had some response to indomethacin (not absolute). She responded to corticosteroids.

A Word About Nomenclature

There is confusion regarding nomenclature for TACs which are due to some underlying cause. Some authors have used the term "secondary," whereas others have used the term "symptomatic." The word symptomatic is probably undesirable because it connotes a definitive relationship with a structural pathology which may not be the case and the symptoms of TACs are not specific to any etiology. Further, ICHD only recognizes the term "secondary headache." However, the word secondary is also not without confusion as some primary TACs may evolve from episodic to chronic forms and some authors have used "secondary" (versus *de novo*) to underline such transitions. For example, CH may evolve from ECH to CCH and some authors have used the term secondary CCH to distinguish this group from primary CCH which were chronic from the beginning.[39] Similarly, the words such as TACs-like (or cluster-like) have been used with varied interpretations and definitions. It is hoped that ICHD Committee will sort this out in the future and advocate a standardized nomenclature.

Conclusion

Nearly half of secondary TAC patients fulfill ICHD criteria for primary TAC headaches. Hence, many experts advocate routine neuroimaging in all TAC patients. Contrarily, patients of TACs with atypical features and red flags are more likely to have a secondary pathology and hence must be investigated. While reporting a case of secondary TACs, the authors should clearly mention whether the ICHD criteria are met or not, whether there are any atypical features, and whether the case is just TACs-like (mimicker). Categorization into probable, possible, and unknown based on the type of secondary cause and its treatment response along with duration of follow-up will add further clarity. Population-based studies on secondary headaches are very difficult if not impossible to conduct. Hence, real magnitude of the secondary TACs will be difficult to assess.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Goadsby PJ, Lipton RB. A review of paroxysmal hemicranias, SUNCT syndrome and other short-lasting headaches with autonomic feature, including new cases. Brain 1997;120 (Pt 1):193-209.
2. Headache Classification Committee of the International Headache Society (IHS).The International Classification of Headache Disorders, 3rd ed. (beta version). Cephalalgia 2013;33:629-808.
3. Favier I, van Vliet JA, Roon KI, Witteveen RJW, Verschuuren JJGM, Ferrari MD, *et al*. Trigeminal autonomic cephalgias because of structural lesions: A review of 31 cases. Arch Neurol 2007; 64:25-31.
4. Favier I, Haan J, Ferrari MD. Cluster headache: To scan or not to scan. Curr Pain Headache Rep 2008;12:128-31.
5. Cittadini E, Matharu MS. Symptomatic trigeminal autonomic cephalalgias. Neurologist 2009;15:305-12.
6. Wilbrink LA, Ferrari MD, Kruit MC, Haan J. Neuroimaging in trigeminal autonomic cephalgias: When, how, and of what? Curr Opin Neurol 2009;22:247-53.
7. Edvardsson B. Symptomatic cluster headache: A review of 63 cases. Springerplus 2014;3:64.
8. de Coo IF, Wilbrink LA, Haan J. Symptomatic trigeminal autonomic cephalalgias. Curr Pain Headache Rep 2015;19:39.
9. Ezzat S, Asa SL, Couldwell WT, Barr CE, Dodge WE, Vance ML, *et al.* The prevalence of pituitary adenomas: A systematic review. Cancer 2004;101:613-9.
10. Mainardi F, Trucco M, Maggioni F, Palestini C, Dainese F, Zanchin G, *et al.* Cluster-like headache. A comprehensive reappraisal. Cephalalgia 2010;30:399-412.
11. Favoni V, Grimaldi D, Pierangeli G, Cortelli P, Cevoli S. SUNCT/SUNA and neurovascular compression: New cases and critical literature review. Cephalalgia 2013;33:1337-48.
12. De Pue A, Lutin B, Paemeleire K. Chronic cluster headache and the pituitary gland. J Headache Pain 2016;17:23.
13. Robin AM, Pabaney AH, Mitsias PD, Schwalb JM. Further evidence for a pain pathway involving the cingulate gyrus: A case of chronic cluster headache cured by glioblastoma. Stereotact Funct Neurosurg 2015;93:194-8.
14. Escuti N. Headache in a patient with an extracranial lipoma: Report of a new case. J Neurol Disord 2015;3:4.
15. Eswaradass VP, Gnanashanmugham G, Pranesh MB, Parimalam N. A rare cause of symptomatic cluster headache. Med J DY Patil Univ 2015;8:810-2.
16. Bellamio M, Anglani M, Mainardi F, Zanchin G, Maggioni F. Cluster headache: When to worry? Two case reports. Cephalalgia 2017;37:491-5.
17. de Coo I, van Dijk JM, Metzemaekers JD, Haan J. A case report about cluster-tic syndrome due to venous compression of the trigeminal nerve. Headache 2017;57:654-7.
18. Semnic R, Kozić D, Semnic M, Trifunović J, Simić S, Radojičić A, *et al.* Segmental cavernous carotid ectasia in a patient with cluster-like headache. Neurol Neurochir Pol 2015;49:70-3.
19. Kao YH, Hsu YC. Chiari malformation type I presenting as cluster-like headache. Acta Neurol Taiwan 2015;24:122-4.

20. Pelikan JB, McCombe JA, Kotylak T, Becker WJ. Cluster headache as the index event in MS: A Case report. Headache 2016;56:392-6.
21. Ljubisavljevic S, Prazic A, Lazarevic M, Stojanov D, Savic D, Vojinovic S, *et al.* The rare painful phenomena-chronic paroxysmal hemicrania-tic syndrome as a clinically isolated syndrome of the central nervous system. Pain Physician 2017;20:E315-E322.
22. Choi HA, Lee MJ, Chung CS. Chronic paroxysmal headache secondary to an orbital metastatic leiomyosarcoma: A case report. Cephalalgia. 2018;38:389-392. Doi: 10.1177/0333102416687538. [Epub 2017 Jan 6].
23. Taga A, Russo M, Genovese A, Manzoni GC, Torelli P. Paroxysmal hemicrania-like headache secondary to phosphodiesterase inhibitors administration: A Case report. Headache 2017;57:663-4.
24. Lambru G, Trimboli M, Tan SV, Al-Kaisy A. Medullary infarction causing coexistent SUNCT and trigeminal neuralgia. Cephalalgia 2017;37:486-90.
25. Jin D, Lian YJ, Zhang HF. Secondary SUNCT syndrome caused by dorsolateral medullary infarction. J Headache Pain 2016;17:12.
26. Liapounova NA, VanderPluym JH, Bhargava R, Kolski HH. Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing-like attacks in a pediatric patient found to have a pontine capillary telangiectasia and developmental venous anomaly: A case report exploring the root of the problem. Cephalalgia 2017;37:1093-7.
27. Gocmen R, Kurt E, Arslan S, Unal-Cevik I, Karli Oguz K, Tezer FI, *et al.* A rare association of trigeminal autonomic cephalgia: Pontine capillary telangiectasia. Neuroradiol J 2015;28:145-7.
28. Berk T, Silberstein S. Case report: Secondary SUNCT after radiation therapy—A novel presentation. Headache 2016;56:397-401.
29. Mathew T, Srinivas M, Badachi S, Nadig R. Post herpes zoster SUNCT like syndrome: Insights from two case reports. Cephalalgia. 2018;38:399-401. Doi: 10.1177/0333102416677052. [Epub 2016 Oct 31].
30. Nagel MA, Burns TM, Gilden D. SUNCT headaches after ipsilateral ophthalmic-distribution zoster. J Neurol Sci 2016;366:207-8.
31. Rojas-Ramirez MV, Bertoli E, Smith JH. Short-lasting unilateral neuralgiform headache with conjunctival injection and tearing secondary to head and neck trauma: Literature review and case report. J Oral Facial Pain Headache 2016;30:68-72.
32. Mangaraj S, Mishra PK, Choudhury AK, Mohanty BK, Baliarsinha AK. Prolactinoma presenting as short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing syndrome. J Neurosci Rural Pract 2017;8:S158-S161.
33. Brilla R, Pawlowski M, Evers S. Hemicrania continua in carotid artery dissection-Symptomatic cases or linked pathophysiology? Cephalalgia. 2018;38:402-5. Doi: 10.1177/0333102416686346. [Epub 2017 Jan 5].
34. Zhang Y, Wang D, He Z, Wu Q, Zhou J. Hemicrania continua-like headache secondary to nasopharyngeal carcinoma: A case report. Cephalalgia. 2017;37:1005-7. Doi: 10.1177/0333102416654884. [Epub 2016 Jun 10].
35. Russo A, Silvestro M, Cirillo M, Tessitore A, Tedeschi G. Idiopathic hypertrophic pachymeningitis mimicking hemicrania continua: An unusual clinical case. Cephalalgia. 2017:333102417708773. Doi: 10.1177/0333102417708773. [Epub ahead of print]
36. Mainardi F, Zanchin G, Maggioni F. Hemicrania continua-like headache related to transdermal nitroglycerine therapy. Headache 2017;57:494-6.
37. Gantenbein AR, Sarikaya H, Riederer F, Goadsby PJ. Postoperative hemicrania continua-like headache-a case series. J Headache Pain 2015;16:526.
38. Alim-Marvasti A, Ho J, Weatherall M, Patel M, George S, Viegas S, *et al.* Trigeminal autonomic cephalgia caused by recurrent posterior scleritis. Pract Neurol 2016;16:455-7.
39. Torelli P, Cologno D, Cademartiri C, Manzoni GC. Primary and secondary chronic cluster headache: Two separate entities? Cephalalgia 2000;20:826-9.