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Contents

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Contents

World Journal of Clinical Cases

Thrice Monthly Volume 9 Number 14 May 16, 2021

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 9 Number 14 May 16, 2021

Contents

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SYSTEMATIC REVIEWS

Cluster headache due to structural lesions: A systematic review of published cases

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Abstract

BACKGROUND

Cluster headache (CH) is a severe incapacitating headache disorder. By definition, its diagnosis must exclude possible underlying structural conditions.

AIM

To review available information on CLH caused by structural lesions and to provide better guides in the distinguishing process and to ensure that there is not a potentially treatable structural lesion.

METHODS

We conducted a systematic review of 77 published cases of symptomatic CH and cluster-like headache (CLH) in PubMed and Google Scholar databases.

RESULTS

Structural pathologies associated with CH were vascular (37.7%), tumoral (32.5%) and inflammatory (27.2%). Brain mass-like lesions (tumoural and inflammatory) were the most common diseases (28.6%), among which 77.3% lesions were at the suprasellar (pituitary) region. Cases of secondary CH related to sinusitis rose dramatically, occupying 19.5%. The third most common disease was internal carotid artery dissection, accounting for 14.3%. Atypical clinical features raise an early suspicion of a secondary cause: Late age at onset and eye and retroorbital pains were common conditions requiring careful evaluation and were present in at least one-third of cases. Abnormal neurological examination was the most significant red flag for impaired cranial nerves. CLH patients may be responsive to typical CH treatments; therefore, the treatment response is not specific. CLH can be triggered by contralateral structural pathologies. CLH associated with sinusitis and cerebral venous thrombosis required more attention.

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CONCLUSION
Since secondary headache could perfectly mimick primary CH, neuroimaging should be conducted in patients in whom primary and secondary headaches are suspected. Cerebral magnetic resonance imaging scans is the diagnostic management of choice, and further examinations include vessel imaging with contrast agents and dedicated scans focusing on specific cerebral areas (sinuses, ocular and sellar regions). Neuroimaging is as necessary at follow-up visits as at the first observation.

Key Words: Secondary cluster headache; Cluster-like headache; Diagnosis

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Core Tip: Secondary headache could perfectly mimick primary cluster headache, hence neuroimaging should be conducted in patients in whom primary and secondary headaches are suspected. Cerebral magnetic resonance imaging scans are the diagnostic management of choice, and further examinations include vessel imaging with contrast agents and dedicated scans focusing on specific cerebral areas (sinuses, ocular and sellar regions). Neuroimaging is as necessary during follow-up as at the first observation.

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INTRODUCTION

Cluster headache (CH) is classified together with paroxysmal hemicrania, short-lasting neuralgiform headache attacks and hemicrania continua as trigeminal autonomic cephalalgias, which are characterized by unilateral episodes of headache in the trigeminal nerve distribution and ipsilateral cranial sympathetic autonomic features[1].

CH has a period of active cluster bouts when it tends to occur, lasting from 15 to 180 min, with a frequency of one every other day up to eight per day. In episodic CH, the bouts are followed by remission periods; in contrast, there is no remission or a remission of less than 3 mo in chronic CH[1,2]. Other distinguishing features of CH include male prevalence and circadian periodicity[3].

Despite its relatively low incidence in the population, the excruciating headache intensity of CH makes it a severely incapacitating headache disorder that substantially impairs social activities and quality of life[4,5].

However, a structural lesion may be the cause of the headache, and patients with cluster-like headache (CLH) associated with structural lesions have been reported. Accordingly, these findings raise the question of how to search for an underlying lesion. Indeed, how can we obtain the most benefit for patients, *i.e.* achieve high diagnostic standards at comparably low cost?

This systematic review seeks to contribute to the knowledge on CLH caused by structural lesions and to provide better guides in the distinguishing process.

MATERIALS AND METHODS

Study design

A systematic review was conducted to provide an overview of published cases of symptomatic CH and to examine the demographics and clinical characteristics of the patients.

Search strategy

This review was carried out according to the Cochrane Collaboration methodology and is reported according to the PRISMA statement. Relevant articles were identified by searching PubMed and Google Scholar databases using the keywords "secondary cluster headache", "symptomatic cluster headache", "cluster-like headache", "mimicking cluster headache", "causing cluster headache", "presenting cluster headache" and "cluster headache due to". The search of all published English articles of case report was performed in January 2021.

Inclusion criteria

Published articles of case report fulfilling the following criteria were included: (1) CLH presented as the initial main symptom; (2) Headache disappeared instantly after treatment for structural pathologies; and (3) At least a 6-mo follow-up period without attacks. The data obtained included age at onset and diagnosis, sex, headache side and site, attack duration and frequency, accompanying cranial autonomic symptoms, response to typical CH treatment and other associated features.

Exclusion criteria

Exclusion criteria were as follows: (1) CLH did not present as the initial main symptom; (2) Structural pathologies not treated, attacks remained after treating structural pathologies or patient died within 6 mo; (3) Associated with systemic diseases; (4) Associated with head injuries; (5) Associated with surgeries; and (6) Associated with particular triggering factors.

Data abstraction

A data abstraction form was devised based on the Cochrane's recommendations. Abstracts identified from searches were screened by two independent authors. Both authors reviewed full-text versions of the articles and articles were retained if they met inclusion criteria. Data extracted from included articles comprised: Authors and date of studies; demographics of patients (age, sex); clinical characteristics (structural pathologies, ages of headache onset, right or left side, headache regions, positional relation of headache and pathologies, headache attack duration and frequency, accompanying symptoms, conditions in intervals, physical and neurological examinations). All data required to answer the study questions were published within the articles, so no contact with authors was necessary. The study did not involve contact with humans, so the ethical approval was not required.

RESULTS

A total of 124 papers (137 cases) were published between 1988 and January 2021. Seventy-seven cases were in accordance with the inclusion criteria. The screening process and results of selection are summarized in a flow chart (Figure 1).

Anatomical correlates of CLH

Vascular pathologies (37.7%, n = 29) were the first cause of secondary CH: Internal carotid artery dissection (ICAD) (11 cases), intracranial aneurysm (6 cases), cerebral venous thrombosis (CVT) (5 cases), arteriovenous malformation (3 cases), subclavian artery subclavian steal phenomenon (1 case), vertebral artery dissection (1 case), intra-cavernous internal carotid artery thrombosis (1 case) and dural arteriovenous fistulas (1 case) (Table 1).

Secondary CH cases associated with tumoural pathologies were 32.5% (n = 25), including prolactinoma (8 cases), meningioma (4 cases), epidermoid cyst (3 cases), nonfunctioning pituitary adenoma (2 case), Arnold–Chiari type I malformation (2 cases), arachnoid cyst (1 case), glioblastoma multiforme (1 case), pituitary cyst (1 case), pituitary growth hormone-producing adenoma (1 case), inflammatory myofibroblastic tumour (1 case), and paraganglioma around the internal carotid artery in neck (1 case) (Table 2).

Inflammatory pathologies were comparatively less frequent causes, amounting to 27.2% (n = 21): Sinusitis (13 cases), idiopathic orbital myositis (2 cases), pituitary granuloma (2 cases), sinuses mucocele (2 cases), inflammatory orbital pseudotumour (1 case), and posterior scleritis (1 case). In addition, there were 2 cases caused by idiopathic intracranial hypertension but without the discovery of pathological origins (Table 3).

Table 1 Cases of secondary cluster headache associated with vascular pathologies.

Year	Ref.	Pathologies	Age at onset, interval between age at onset and diagnosis, yr	Sex
1997	Rosebraugh *et al*[33]	ICAD	34	Male
2003	Frigerio *et al*[34]	ICAD	50	Female
2005	Hannerz *et al*[16]	ICAD	58	Male
2006	Razvi *et al*[35]	ICAD (in the petrous segment of ICA)	44	Male
2007	Hardmeier *et al*[36]	ICAD (petrous segment)	38	Male
2007	Straube *et al*[13] (case 2)	ICAD (cavernous segment)	35	Male
2008	Rigamonti *et al*[37] (cases 1 and 2)	ICAD (case 1, lacerum segment)	50	Male
		ICAD (case 2, lacerum segment)	49	Male
2008	Godeiro-Junior *et al*[38]	ICAD (petrous segment)	53	Male
2008	Tobin *et al*[39]	ICAD (cervical segment)	55	Male
2013	Tsivgoulis *et al*[40]	ICAD (petrous segment)	32	Male
1991	West *et al*[17]	Aneurysm (posterior inferior cerebellar artery, vertebral artery)	39 (12)	Male
2000	McBeath *et al*[31]	Aneurysm (posterior communicating artery)	45 (10)	Male
2006	Gentile *et al*[11]	Aneurysm (multiple cerebral aneurysms)	58	Female
2007	Valença *et al*[41] (cases 1 and 2)	Aneurysm (case 1, posterior communicating artery)	47	Male
		Aneurysm (case 2, intracranial ICA)	57	Male
2009	Sewell *et al*[42]	Aneurysm (moyamoya disease)	18 (16)	Male
2006	Georgiadis *et al*[25]	CVT (superior sagittal sinus)	46	Male
2006	Park *et al*[26]	CVT (right transverse sinus, straight sinus)	20 (6)	Male
2006	Peterlin *et al*[8]	CVT (case 1, left transverse, sigmoid sinus)	38 (9)	Male
		CVT (case 2, left transverse sinus, sigmoid sinus, internal jugular vein)	32 (10)	Male
2008	Rodríguez *et al*[18]	CVT (superior sagittal sinus and bilateral transversal sinuses)	51	Male
1982	Mani *et al*[43]	Arteriovenous malformation (occipital lobe)	22 (14)	Female
1996	Muñoz *et al*[19]	Arteriovenous malformation (case 1, temporal lobe)	52	Male
		Arteriovenous malformation (case 2, frontal lobe)	40 (6)	Male
2001	Piovesan *et al*[44]	Subclavian steal phenomenon	51 (9)	Female
2008	Kim *et al*[45]	Vertebral artery dissection (posterior inferior cerebellar artery)	48	Female
2008	Ashkenazi *et al*[20]	Intra-cavernous carotid artery thrombosis	62	Male
2017	Chang *et al*[46]	Middle meningeal artery dural arteriovenous fistulas	42	Male

CVT: Cerebral venous thrombosis; ICAD: Internal carotid artery dissection.

Headache characteristics

Two cases were ruled out because of an unclear description of age[6,7], and the average age of symptom onset was 38.8 ± 13.0 years. Forty patients (51.9%) were between 20-40-years-old, six (7.8%) were younger than 20-years-old, and 31 (40.3%) were older than 40-years-old. Sixty (77.9%) patients were male and 17 (22.1%) female; the male: Female ratio was 3.53: 1 (60: 17).

CH was strictly unilateral in all patients, with 43 (55.8%) left-sided and 34 (44.2%) right-sided cases. Structural pathologies were ipsilateral to the headache in 61 (79.2%), contralateral in 4 (5.2%) (Case 1)[2,8-10], midline (no visible invasion to the left or right

Table 2 Cases of secondary cluster headache associated with tumoural pathologies (including tumours and cysts)

Year	Ref.	Pathologies	Age at onset (Interval between age at onset and diagnosis, yr)	Sex
1988	Greve *et al*[47] (case 3)	Prolactinoma	57 (1)	Male
2001	Porta-Etessam *et al*[48]	Prolactinoma	28 (8)	Male
2004	Leone *et al*[9]	Prolactinoma	46 (3)	Male
2005	Negoro *et al*[27]	Prolactinoma	14 (3)	Male
2012	Levy *et al*[49]	Prolactinoma	25	Male
2013	Edvardsson *et al*[50]	Prolactinoma	46	Male
2017	Andereggen *et al*[51]	Prolactinoma	46	Male
2017	Pineyro *et al*[52]	Prolactinoma	15 (3)	Female
1991	Levyman *et al*[53]	Epidermoid cyst (posterior fossa)	40 (13)	Female
2006	Massie *et al*[10]	Epidermoid cyst (epidermoid clival lesion)	36 (3)	Male
2007	Eimil-Ortiz *et al*[28]	Epidermoid cyst (pontocerebellar angle)	47	Male
1989	Hannerz *et al*[21]	Parasellar meningioma	28 (18)	Male
1995	Taub *et al*[6]	Meningioma (tentorium cerebelli)	35-40 (20-25)	Male
2003	Bigal *et al*[7]	Inflammatory myofibroblastic tumour (undersurface of the tentorium)	18.5 (9.5)	Male
2008	Alty *et al*[54]	Trigeminal meningioma (cerebellopontine angle)	30	Male
2009	Robbins *et al*[55]	Meningioma (planum sphenoidale)	29 (8)	Male
2012	Edvardsson *et al*[22]	Parietal glioblastoma multiforme	41	Male
2013	Edvardsson *et al*[56]	Supra- and intrasellar arachnoid cyst	43	Male
1982	Tfelt-Hansen *et al*[57]	Pituitary chromophobe adenoma	21 (31)	Male
2014	Edvardsson *et al*[58]	Pituitary chromophobe adenoma	49	Male
1996	Milos *et al*[59]	Pituitary GH-producing adenoma	33 (4)	Male
2016	De Pue *et al*[32]	Pituitary cyst	35 (12)	Male
2004	Seijo-Martinez *et al*[12]	Arnold–Chiari type I malformation, syringomyelia	36	Female
2015	Kao *et al*[60]	Arnold–Chiari type I malformation	26	Male
2014	Malissart *et al*[61]	Carotid paraganglioma	60	Female

GH: Growth hormone.

side on neuroimaging) in 10 (13.0%), and unknown in 2[11,12].

Information about attack duration was missing in 4 cases[7,13-15]. For the remaining 73 cases, 56 patients experienced attacks lasting 15–180 min, and duration was unclear for the other 17 (23.3%) cases. Twelve cases lacked accurate information about attack frequency[7,13-23]; sixty of the remaining sixty-five cases had an attack frequency between one every other day and eight per day, and five (7.7%) did not fulfil the frequency criterion.

The headache predominantly affected orbital and periorbital regions in 47 cases (61.0%), temporal regions in 23 (30.0%), and supraorbital regions in 16 (20.1%). Additionally, 26 patients (33.8%) described pain in the eye and retroorbital regions; 14 (18.2%) described pain in other sites. Regarding accompanying autonomic symptoms, 73 patients (94.5%) reported lacrimation, conjunctival injection, and rhinorrhoea; 40 patients (51.2%) reported miosis and ptosis; and 39 patients (50.6%) reported nasal congestion, eyelid oedema, and forehead sweating. In addition to typical CH autonomic symptoms, 13 patients (16.9%) described migraine-like symptoms of

Table 3 Cases of secondary cluster headache associated with inflammatory pathologies and idiopathic intracranial hypertension

Year	Ref.	Pathologies	Age at onset (Interval between age at onset and diagnosis, yr)	Sex
1988	Takeshima *et al*[62]	Sinusitis (case1, frontal, ethmoidal, maxillary sinusitis)	31	Male
		Sinusitis (case 2, frontal, maxillary sinusitis)	31 (9)	Male
1995	Zanchin *et al*[14]	Sinusitis (sphenoid sinus aspergillus infection)	64	Female
1997	Heidegger *et al*[63]	Sinusitis (sphenoid sinus aspergillus infection)	68	Male
2002	Scorticati *et al*[23]	Sinusitis (foreign body in the maxillary sinus, maxillary sinusitis)	26 (8)	Female
2013	Edvardsson *et al*[64]	Sisitis (maxillary sinusitis)	24	Male
2013	Edvardsson *et al*[65]	Sinusitis (maxillary sinusitis)	21	Male
2018	Balgetir *et al*[66] (cases 1-7)	Sinusitis (frontal, ethmoid, maxillary sinusitis)	23	Male
		Sinusitis (frontal, maxillary sinusitis)	28	Male
		Frontoethmoidal mucocele	33	Male
		Sinusitis (frontal, ethmoid, maxillary sinusitis)	27	Male
		Sinusitis (maxillary, ethmoid sinusitis)	16	Male
		Sinusitis (frontal sinusitis)	31	Male
		Sinusitis (sphenoid and ethmoid sinusitis)	39	Female
2018	Branco *et al*[67]	Sphenoid sinus mucocele	62	Male
2007	Harley *et al*[68]	Inflammatory orbital pseudotumour	33	Female
2009	Choi *et al*[69]	Posterior scleritis	42 (10)	Female
2017	Ersoy *et al*[70]	Idiopathic orbital myositis	34	Female
2019	Douglas *et al*[71]	Idiopathic orbital myositis	19	Female
2007	Favier *et al*[15]	Hypothalamus-pituitary granuloma	26	Female
2013	van der Vlist *et al*[72]	Hypothalamus-pituitary granuloma	31	Male
2006	Volcy *et al*[73]	Idiopathic intracranial hypertension	40	Male
2008	Testa *et al*[74]	Idiopathic intracranial hypertension	28	Female

nausea, phonophobia and vomiting. Persistent miosis and/or ptosis were observed in 11 patients (14.3%). During the intervals between attacks, 18 patients (23.4%) reported additional headaches of different intensities and characteristics. Altered neurological findings were found in 21 (27.2%) patients (Table 4).

DISCUSSION

When establishing a diagnosis of secondary headache, an important causal relationship criterion is to confirm the disappearance or clear relief of headache after the associated condition is removed. In our study, we excluded associated conditions like systemic diseases, injuries and surgeries because the pathologies cannot be removed, in general terms. Associated conditions of specific triggering factors (drug, emotion, movement) were also excluded, for the same reason. Cases of CLH presenting as an accompanying symptom, which means other clear defect symptoms of nervous system lesions occur in the meantime, were also ruled out of the analysis because they were less likely to be confused with primary CH. Therefore, we selected the patients that were both strong "confusing" and in a strong causal link with structural lesions and focused the evaluation on these patients.

Clinical implications

CLH-associated conditions: Vascular pathologies (37.7%, n = 29) and tumoural pathologies (32.5%, n = 25) were the most common in CLH patients, followed by inflammatory pathologies (27.2%, n = 21). A previous comprehensive reappraisal of

Table 4 Demographic profile and characters of the headache

	Number (%)/mean ± range
Age at onset (n = 66)	
Average age	38.3 ± 13.0
20-40 yr	40 (51.9%)
< 20 yr	6 (7.8%)
> 40 yr	31 (40.3%)
Sex	
Male	60 (77.9%)
Female	17 (22.1%)
Side	
Left	43 (55.8%)
Right	34 (44.2%)
Sides of headache and structural pathologies (n = 66)	
Ipsilateral	61 (79.5%)
Midline	10 (13.0%)
Contralateral	4 (5.2%)
Unknown	2 (2.6%)
Attacks not fulfilling ICHD-3	
Duration (n = 65)	17 (23.3%)
Frequency (n = 57)	5 (7.7%)
Headache sites	
Orbital/periorbital	47 (61.0%)
Temporal	23 (30.0%)
Supraorbital	16 (20.1%)
Eye/retroorbital	26 (33.8%)
Other	14 (18.2%)
Associated autonomic symptoms	
Lacrimation/conjunctival injection/rhinorrhoea	73 (94.5%)
Miosis/ptosis	40 (51.2%)
Nasal congestion/eyelid oedema/forehead sweating	39 (50.6%)
Nausea/phonophobia/vomiting	13 (16.9%)
Persistent miosis/ptosis	11 (14.3%)
Additional headaches between attacks	18 (23.4%)
Altered neurological examination	21 (27.2%)

ICDH: International Classification of Headache Disorders.

CLH in 2010[24] reported that pathologies associated with CLH were the vascular ones (38.5%), followed by tumours (25.7%) and inflammatory infectious diseases (13.5%). The review also included post-traumatic and other neurological, iatrogenic and dental pathologies in the analysis, which may explain the different figures comparing to our study.

Brain mass-like lesions (tumoural and inflammatory) were the most common diseases (28.6%, n = 22), among which 77.3% (17 in 22) of lesions were at the suprasellar (pituitary) region. The second most common diseases was ICAD, accounting for 14.3% (n = 11) of CLH patients; all the dissections were situated at the

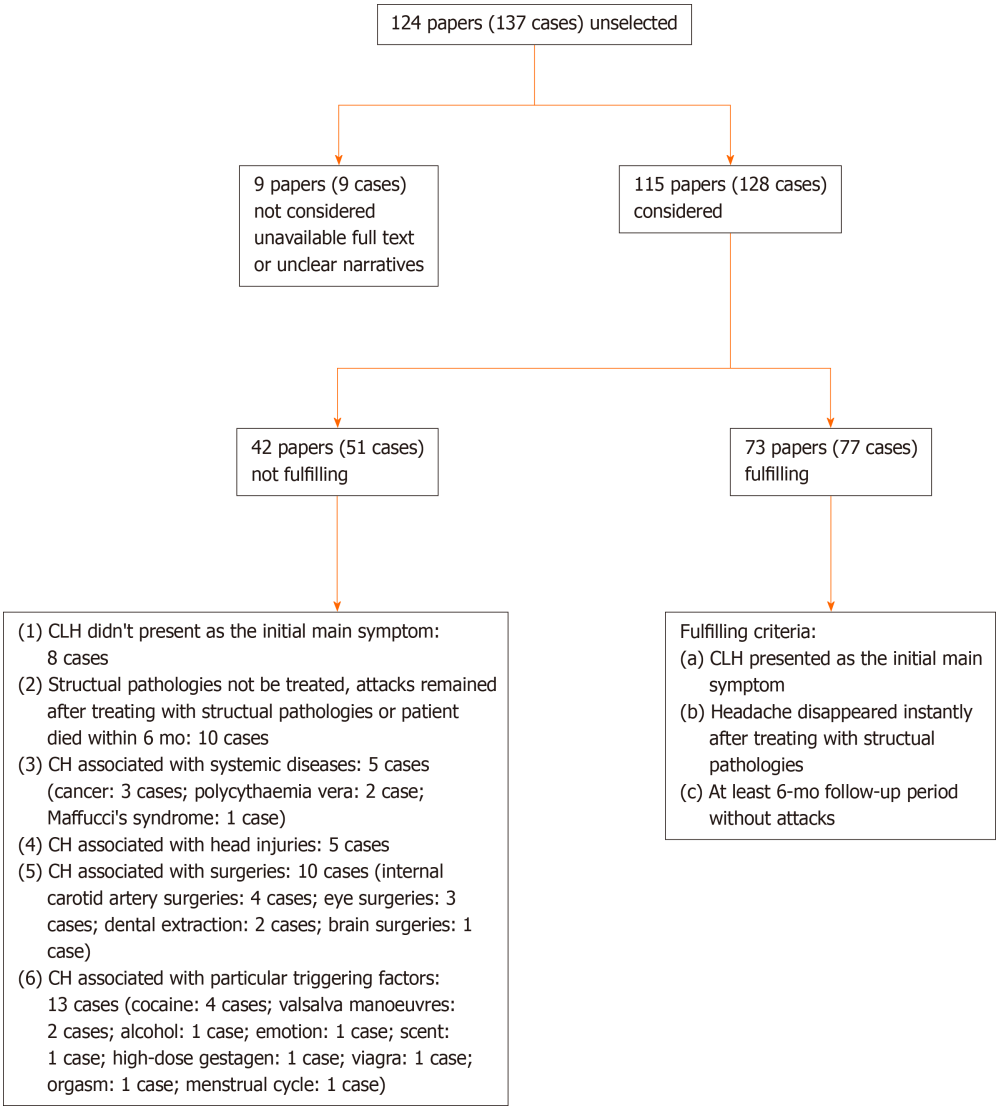

Figure 1 Flow chart of selection of 124 papers found in the literature. CH: Cluster headache; CLH: Cluster-like headache.

extracranial segments of internal carotid artery. An interesting finding is that the figures of CLH related to sinusitis were dramatically higher than a previous report; 15 cases (19.5%) in our study compared to only six cases prior[24], which appears to indicate increased attention related to sinusitis causing CLH. It must be noted that a lack of contributing factors of coagulopathies cannot exclude CVT. In the 3 cases of CVT patients for whom coagulopathies were examined, evidence of coagulopathies was found in none[18,25,26], which may indicate a potential higher incidence of CVT in CLH-associated conditions.

Structural pathologies in CLH were mostly located in the ipsilateral hemisphere; however, under certain circumstances, CLH may be triggered by contralateral structural pathologies (case 1)[2,8,27,28]. Explanations might be as follows: (1) Fos-producing neurons in the dorsal horns of C1 and C2 produce Aδ and C fibres (nociceptive input tracts) that innervate the majority of ipsilateral pain-producing structures and a few contralateral structures[29]. Due to anatomical variation of input fibres or specific anatomical sites, the trigeminocervical complex is sensitive to contralateral nociceptive inputs *via* the few contralateral Aδ and C fibres; (2) The contralateral mass effect might occur through traction of meningeal structures, producing trigeminal nerve enlargement and trigeminovascular system activation; and (3) Coexistence of contralateral structural pathologies and headache, which is possible but unlikely; the CLH disappears after treatment for structural pathologies is started, strongly indicating causality. CLH with midline structural pathologies may be explained by mild ipsilateral invasions that are not well distinguishable on neuroimaging.

Age at onset and gender: Primary CH usually occurs between the second and fourth decades[1]. The average age at onset of CLH in our study was 38.8 ± 13.0 years; however, 40.3% of the patients were older than 40 years. It has been suggested that the late age at onset requires attention for structural pathological causes in the previous review[24], which is in accordance with our study that older age at onset is the most frequent "red flag".

The sex ratio of secondary CH was calculated to be 3.53:1, which is close to the ratio of primary CH; males experience CH three times more often than females[1]. The prevalence of male sex in secondary CH is unexpected because the structural causes as a whole are not sex-related. Mainardi *et al*[24] explained the male prevalence by possible anatomical and physiological differences of the hypothalamus in males and females, whereby males possess a greater volume of suprachiasmatic nucleus than do females, which makes males more susceptible[30].

Suspect elements: Atypical headache features can help raise an early suspicion of a secondary cause. The red flags are, in descending order of occurrence, attacks with eye and retroorbital pain (33.8%), altered neurological examinations (27.2%), additional headache between attacks (23.4%), atypical attack duration (23.3%), migraine-like automatic symptoms (16.9%), persistent partial Horner's syndrome (14.3%) and atypical attack frequency (7.7%). An altered neurological examination is the most significant red flag, which highlights impaired cranial nerves and nuclei, among which ophthalmic symptoms and signs are the most frequent, including impaired vision and visual field, diplopia, ocular motility disorders and dysfunctional pupil reflexes.

Attacks with eye and retroorbital pain are described in 33.8% of CLH cases, and they are particularly common in pituitary pathologies (64.3%, 9 in 14), ICAD (63.6%, 7 in 11) and eye diseases (75%, 3 in 4). Another particular "red flag" for ICAD is persistent miosis or ptosis, which is present in 72.7% of cases: 8 in 11. Haematoma in the vessel wall may damage the structure of the sympathetic fibres that travel along the carotid artery, resulting in unresolved sympathetic deficit symptoms between attacks. CH is a strictly paroxysmal cephalalgia in which patients are normal between attacks. Additional headaches during the intervals may suggest a nociceptive input of a different type, representing an element that requires attention. Additional headaches of different characteristics were observed in 23.4% of patients.

Responses to typical CH treatments: Resistance to typical CH treatment should raise suspicion of a secondary origin; however, CLH may be responsive (Table 5), particularly to acute treatment medications, with 46.9% effective responses for acute treatments. In 35.8% of responses, preventive treatments were reported to be effective. Nonetheless, the response to one drug can be completely opposite, even in two cases of the same category of structural origin. This may be due to different formulations, dosages, doses or delivery approaches. There is evidence that a CLH patient with pituitary prolactinoma did not respond to verapamil 80 mg but did respond to verapamil 120 mg three times a day[9]. Therefore, it must be stressed that response to CH treatment is not a sufficiently reliable criterion to discriminate between CH and CLH.

Research implications: Neuroimaging can confirm the diagnosis of underlying structural lesions, which should be performed in patients in whom both primary and secondary headaches are suspected. Brain magnetic resonance imaging (MRI) scans should be the method choice because they can detect most pathologies.

Vascular pathologies normally cannot be revealed by MRI interpretations, or they might present slight and unspecific signs on MRI scans. Computed tomography angiography and magnetic resonance venography are precise diagnostic tools for vascular pathologies. Other investigations include dedicated MRI scans focusing on particular areas, especially the sinuses and the ocular and sellar regions.

There is a remarkable finding that neuroimaging results may be normal in the headache trajectory before diagnosis[9,17,27,31,32]. Explanations could be minor structural lesions at that time that were not visible or because of low resolutions of neuroimaging. As a result, neuroimaging should be considered during the follow-up period in primary CH patients. Changes in headache characteristics or resistance to treatment may indicate progression of structural lesions and especially indicate repeated neuroimaging.

Limitations

Our review has limitations. Reporting bias and publication bias cannot be overlooked in our review because we selected the published cases of CLH as samples. Rare causes

Table 5 Cluster-like headache patients' responses to typical cluster headache treatments

	Effectiveness[a] (*n*, %)
Acute treatment	
Triptans	19/28 (67.9%)
Oxygen inhalation	13/24 (54.2%)
Ergotamine/ergometrine	6/20 (30%)
NSAIDs	7/31 (22.6%)
Analgesics	6/8 (75%)
Caffeine	2/2 (100%)
Total	53/113 (46.9%)
Preventive treatment	
Verapamil	9/17 (52.9%)
Glucocorticoid drugs	11/15 (73.3%)
Propranolol	1/6 (16.7%)
Antipsychotics	2/11 (18.2%)
AEDs	0/13 (0)
Lithium carbonate	1/1
Other antihypertensive drugs	0/2 (0)
Antihistamine	0/2 (0)
Total	24/67 (35.8%)

[a]Moderate and great improvement of headache (intensity, frequency, duration) are regarded as effective.
AEDs: Anti-epileptic drugs; NSAIDs: Non-steroidal anti-inflammatory drugs.

for secondary CH have a higher chance of being published. Our findings must be interpreted within the context of the limitations and further widely prospective studies need to be conducted to verify the results.

CONCLUSION

As in all primary headaches, the diagnosis of primary CH must be excluded to rule out underlying causal conditions. This review provides a summary of clinical features of secondary CH cases and highlight imaging examinations in the diagnostic process. There are important implications for the application of these findings into clinical practice to achieve accurate and efficient diagnosis of CH.

ARTICLE HIGHLIGHTS

Research background

Among the primary headaches, cluster headache (CH) presents very particular features allowing a relatively easy diagnosis based on criteria listed in Chapter 3 of the International Classification of Headache Disorders. However, as in all primary headaches, possible underlying causal conditions must be excluded to rule out a secondary cluster-like headache (CLH).

Research motivation

The review aims to contribute to the knowledge on CLH caused by structural lesions and to provide better guides in the distinguishing process.

Research objectives
We analysed the published cases of symptomatic CH and CLH in PubMed and Google Scholar databases.

Research methods
We conducted a systematic review of published cases of symptomatic CH and CLH and analysed the features of the patients.

Research results
Structural pathologies associated with CH were vascular (37.7%), tumoural (32.5%) and inflammatory (27.2%). Patients with atypical clinical features require careful evaluation for structural origins. CLH patients may be responsive to typical CH treatments; the treatment response is not specific. CLH can be triggered by contralateral structural pathologies. CLH-associated sinusitis and cerebral venous thrombosis are worth paying attention to.

Research conclusions
Secondary headaches could perfectly mimick primary CH, hence neuroimaging should be conducted in patients in whom primary and secondary headaches are suspected. Cerebral magnetic resonance imaging scans are the diagnostic management of choice, and further examinations include vessel imaging with contrast agents and dedicated scans focusing on specific cerebral areas (sinuses, ocular and sellar regions). Neuroimaging is as necessary at follow-up visits as at the first observation.

Research perspectives
We investigated the clinical features of CLH patients in published cases and tried to summarize their distinctive characteristics by comparing to patients with primary CH.

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