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Research Article

A Chinese Clinical Guideline for the Diagnosis and Treatment of Chronic Secondary Headache and Orofacial Pain

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Abstract

Secondary headaches and orofacial pains, caused by underlying diseases or dysfunctions, are more prevalent than primary headaches and orofacial pains. To date, no clinical guidelines have been established for the diagnosis and treatment of the 11 categories of chronic secondary headaches and orofacial pains on the "International Classification of Diseases, 11th Revision (ICD-11)". To improve the recognition and management of these painful diseases and to standardize diagnostic and therapeutic practices among clinicians in China, the Expert Panel for the Development of Chinese Clinical Guidelines for Chronic Secondary Headache and Orofacial Pain (hereinafter referred to as Expert Panel) compiled the present guideline. This guideline focuses on 8 common chronic secondary headache and orofacial pain disorders that are frequently underdiagnosed or misdiagnosed, but have well-defined diagnostic and therapeutic protocols within the painology department currently. These disorders include: cervicogenic headache, trigeminal neuralgia, glossopharyngeal neuralgia, sphenopalatine neuralgia, temporomandibular joint disorders, head and facial herpes zoster-related neuralgia, post-stroke persistent headache, and cancer-related head and facial pain. The Expert Panel systematically retrieved and evaluated the available evidence on the definition, epidemiology, pathogenesis clinical manifestations, diagnostic criteria, differential diagnosis, treatment, and recent advances for each of the 8 disorders. The guideline provides recommendations for both pharmacological therapy and minimally invasive treatments based on the evidence, to serve as a practical reference for painologists, neurologists, general practitioners, and other healthcare professionals involved in the management of these chronic secondary headache and orofacial pain.

Keywords

Headache Disorders, Secondary, Facial Pain, Chronic Pain, Pharmacotherapy, Minimally Invasive Surgical Procedures

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1. Guideline Development Process and Methods

1.1. Initiating Organization and Expert Panel

This guideline was initiated and developed by the Expert Panel for the Development of Chinese Clinical Guidelines for Chronic Secondary Headache and Orofacial Pain, hereafter referred to as the Expert Panel. After three review meetings and repeated online discussions, the guideline was finalized in September 2025. All panel members completed conflict-of-interest disclosure forms, and no conflicts of interest directly related to this guideline were identified.

1.2. Guideline Registration and Protocol Development

This guideline was registered on the Practice Guideline Registration for Transparency platform (http://www.guidelines-registry.cn; registration number: PREPARE-2023CN625). Its development followed the WHO Handbook for Guideline Development published in 2014 and the Guiding Principles for the Development/Revision of Clinical Practice Guidelines in China (2022 Edition), issued by the Chinese Medical Association in 2022.

1.3. Target Users and Population

This guideline is intended as a reference for pain physicians, neurologists, general practitioners, and clinicians in other specialties at hospitals of different levels. Recommendations should be applied with consideration of each patient's individual clinical circumstances.

1.4. Evidence Retrieval

For the final included outcomes, the Expert Panel structured clinical questions according to the population, intervention, comparison, and outcome (PICO) framework. Searches were conducted in Wanfang Data, CNKI, PubMed, MEDLINE, the Cochrane Library, and other databases. Evidence was selected in the order of systematic reviews, meta-analyses, randomized controlled trials (RCTs), cohort studies, and case-control studies.

1.5. Evidence Assessment and Grading

The Expert Panel reviewed the recommendations and evidence feedback provided by the literature review group and evaluated the necessity of each item and selected recommendation during the first meeting. Recommendations based on poor-quality or conflicting evidence were removed, rephrased, or merged. During the second round of voting, the quality of evidence and strength of recommendations were graded using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system (Table 1) [1-3] and a consensus meeting method. The voting panel developed recommendations (strong or weak; for or against) according to the balance of benefits and harms of medical interventions, evidence quality, values, preferences, and cost. After repeated discussions and online voting, the final guideline was formulated.

Table 1. GRADE Evidence Quality and Strength of Recommendation.

Grade	Description
Evidence quality	
High (A)	The panel is very confident that the estimate is close to the true effect.
Moderate (B)	The panel has moderate confidence in the estimate; the estimate is likely close to the true effect, but may be substantially different.
Low (C)	Confidence in the estimate is limited; the estimate may differ substantially from the true effect.
Very low (D)	The panel has very little confidence in the estimate; the estimate is very likely to differ substantially from the true effect.
Recommendation level	
Strong (1)	Most patients would choose the recommended approach and only a few would not; most physicians should accept the intervention; more than 70% of panel members agreed.
Weak (2)	Most patients would choose the recommended approach, but many would not; clinicians should carefully review evidence or evidence summaries and discuss the evidence, patient values, and preferences; 50%-70% of panel members agreed.

Grade	Description
No clear recommendation (3)	Benefits and harms are balanced; the target population is uncertain; evidence is insufficient; fewer than 50% of panel members agreed.

2. Overview

Chronic secondary headache and orofacial pain refer to headache or orofacial pain with a defined etiology that occurs on more than half of days for 3 months or longer and lasts at least 2 hours per day [4]. Etiologies include other diseases, either focal or systemic, trauma such as physical, chemical, or radiation injury, and infection.

The epidemiology of chronic secondary headache and orofacial pain varies by population and pain type, and its pathogenesis remains incompletely understood. Pain is commonly thought to arise when mechanical, chemical, biological, or biochemical stimuli activate receptors in intracranial or extracranial pain-sensitive structures. Signals are transmitted through nociceptive pathways to the cerebral cortex, or may result from integration through descending modulatory pathways above the spinal cord [5].

Secondary headache and orofacial pain are caused by underlying disease or dysfunction. The International Classification of Diseases, 11th Revision (ICD-11), classifies chronic secondary headache and orofacial pain into 11 categories [4]. To better guide and standardize diagnosis and treatment in China, the Expert Panel developed this guideline in accordance with the Reporting Items for Practice Guidelines in Healthcare (RIGHT) and the Appraisal of Guidelines for Research and Evaluation II (AGREE II), while incorporating clinical experience in pain diagnosis and management in China. The guideline covers eight representative and relatively common chronic secondary headache and orofacial pain disorders: cervicogenic headache, trigeminal neuralgia, glossopharyngeal neuralgia, sphenopalatine neuralgia, temporomandibular joint disorders, head and facial herpes zoster-related neuralgia, post-stroke persistent headache, and cancer-related craniofacial pain. Each disorder is discussed in terms of definition, epidemiology, pathogenesis, clinical manifestations, classification and diagnosis, differential diagnosis, treatment, and recent advances, with the aim of providing a reference for pain physicians, neurologists, general practitioners, and clinicians in other specialties.

3. Cervicogenic Headache

3.1. Definition

Cervicogenic headache (CEH) is a syndrome characterized mainly by chronic head pain caused by organic or functional lesions of the cervical spine and/or cervical soft tissues. The prevalence of CEH is 4.1%-21.0%. Pain commonly occurs in the temporal, occipital, frontal, and orbital regions [6]. Proposed pathogenetic mechanisms include muscle tension, mechanical stimulation, inflammatory edema, and nerve injury [7].

Most patients present with unilateral headache, although some have bilateral headache. The pain is usually a dull, non-tearing pain. Most patients also have neck stiffness, restricted motion, trigger points in the head and neck, and, in patients with traumatic facet joint lesions, marked fixed paravertebral tenderness in the upper cervical spine. Some patients have positive vertex compression and head-lift tests. Diagnostic nerve block of the posterior medial branch of the spinal nerve innervating the relevant facet joint can confirm the diagnosis.

3.2. Diagnostic Criteria

The Cervicogenic Headache International Study Group (CHISG) described CEH as unilateral head pain accompanied by clinical features such as nausea, photophobia, phonophobia, and neck pain [8]. This differs to some extent from the revised diagnostic criteria issued by the International Headache Society in 2012 [9].

1) *History of trauma.* Patients with a history of head and neck trauma, especially traffic-related head and neck injury, should be regarded as highly suspicious for CEH.
2) *Pain originating from the posterior branch of the spinal nerve.* Patients with pain characteristics consistent with cervical posterior branch pain and obvious cervical paravertebral tenderness may undergo diagnostic nerve block, which has both diagnostic and therapeutic value. Pain relief approximately 10 minutes after local anesthetic injection, lasting longer than 2 hours, is considered a positive response. A positive test is one of the characteristic findings for early diagnosis.
3) *Radicular pain.* Cervical disc herniation is a common cause of cervical radicular pain. In later stages, uncovertebral joint hyperesthesia and vertebral osteophytes may directly compress cervical nerve roots. Involvement of C3-C6 spinal nerve roots is most common.
4) *Imaging features.* Conventional anteroposterior and lateral radiographs may show abnormal cervical curvature, osteophyte formation, intervertebral space narrowing, and ligament calcification. Cervical MRI demonstrating disc herniation helps diagnose CEH. Ultrasonography may show abnormalities in the greater occipital nerve on the painful side, including blurred nerve borders and internal fascicular imaging compared with the asymptomatic side [10].

3.3. Differential Diagnosis

CEH has complex clinical manifestations and should be differentiated from migraine and tension-type headache.

1) *Migraine* often occurs in patients aged 20-40 years, is usually unilateral and pulsatile, is associated with light and sound sensitivity, and responds to triptans.
2) *Tension-type headache* usually occurs after middle age, is often bilateral and pressing in quality, and responds to centrally acting muscle relaxants.

3.4. Treatment Recommendations

The clinical treatment principle for CEH should emphasize minimally invasive therapy. Nerve blocks can eliminate local soft tissue inflammatory lesions, with subsequent relief of CEH [11, 12].

1) General Treatment

For patients with short disease duration and mild pain, traction of the head and neck, physical therapy, extracorporeal shock wave therapy [13], Jingshu granules [14, 15], and nonsteroidal anti-inflammatory analgesics such as celecoxib may be used.

2) Trigger Point Therapy

Trigger points in pericranial and cervical muscles may be treated with shock wave therapy, acupuncture, or trigger-point injection. The sternocleidomastoid and suboccipital muscle groups are the most common muscles causing CEH; treatment is more effective when more trigger points are treated in a single session.

3) Nerve block therapy [16]

Before therapy, the lesion site should be identified and a nerve block plan developed. Common targets include the posterior branches of C2 and C3 spinal nerves, the occipital nerve [17], and the great auricular nerve. Common block drugs include local anesthetics combined with a small dose of glucocorticoid, such as ropivacaine injection plus dexamethasone palmitate injection. In addition to nerve block, Navani et al. [18] first reported pulsed radiofrequency of the greater occipital nerve for occipital neuralgia in 2006, and systematic reviews have confirmed the efficacy of radiofrequency therapy for CEH [19]. C2 dorsal root ganglion block can also achieve good therapeutic effects.

4) Minimally Invasive Interventions of the Cervical Disc and Ablation of the Cervical Dorsal Ramus

For CEH caused by cervical disc disease, minimally invasive cervical disc interventions may include cervical disc laser repair, low-temperature plasma or radiofrequency ablation [20], ozone nucleolysis, or combined cervical disc ablation and ozone nucleolysis. Under imaging guidance such as ultrasound or CT, destructive treatment of the posterior medial branch of the cervical nerve, such as radiofrequency thermocoagulation, may be performed.

The therapeutic modalities for cervicogenic headache, accompanied by their respective levels of evidence and grades of recommendation, are summarized in Table 2.

Table 2. *Treatments for Cervicogenic Headache.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	NSAIDs: celecoxib, Fenbid, ketoprofen gel patch	A	Strong
	Opioids: tramadol, oxycodone	B	Weak
	Other: Jingshu granules	A	Strong
Physical therapy	Shock wave therapy	B	Strong
Minimally invasive intervention	Trigger-point injection	B	Strong
	Greater occipital nerve block	A	Strong
	Greater occipital nerve pulsed radiofrequency	A	Strong
	Cervical disc laser repair, low-temperature plasma, or radiofrequency ablation	A	Strong

4. Trigeminal Neuralgia

4.1. Definition

Trigeminal neuralgia is a chronic painful disorder characterized by paroxysmal severe facial pain. Attacks are recurrent, usually lightning-like or electric shock-like, and begin and end abruptly. Pain is usually confined to the distribution of one or more branches of the trigeminal nerve. The prevalence is 182 per 100, 000, and the annual incidence is 3-5 per 100, 000, increasing with age. The exact etiology may remain unclear, or trigeminal neuralgia may occur secondary to another clearly defined disorder. Persistent pain of moderate or greater intensity may coexist in the affected nerve distribution.

4.2. Diagnostic Criteria

According to the International Classification of Headache Disorders, 3rd edition (ICHD-3), diagnostic criteria [21] are as follows:

1) Recurrent paroxysms of unilateral facial pain in the distribution of one or more divisions of the trigeminal nerve, without radiation beyond the trigeminal distribution, fulfilling criteria 2 & 3.
2) Pain has at least three of the following features: 1) duration from a fraction of a second to 2 minutes; 2) severe intensity; 3) electric shock-like, shooting, stabbing, or sharp quality; 4) precipitation by innocuous stimuli to the affected side of the face such as washing, brushing teeth, speaking, or light touch.
3) No clinically evident neurological deficit attributable to causes other than vascular compression.
4) Not better accounted for by another ICHD-3 diagnosis.

4.3. Clinical Classification

1) Classic trigeminal neuralgia is confirmed by MRI or intraoperative evidence of neurovascular compression, not simple contact, with morphological changes in the trigeminal root.
2) Secondary trigeminal neuralgia may last longer than 30 minutes and is caused by an underlying lesion that explains the neuralgia, including multiple sclerosis, space-occupying lesions, herpes zoster, and other causes.
3) Idiopathic trigeminal neuralgia is diagnosed when MRI or electrophysiological examination excludes classic and secondary trigeminal neuralgia.

4.4. Differential Diagnosis

Trigeminal neuralgia should be differentiated from glossopharyngeal neuralgia, toothache, cluster headache, and temporomandibular joint disorders.

1) *Glossopharyngeal neuralgia* is deeper, involving the tongue root, pharynx, and deep ear canal, and is mainly triggered by swallowing rather than face washing or tooth brushing.
2) *Toothache* is usually continuous dull or throbbing pain, sensitive to cold or heat, and lacks abrupt electric shock-like attacks and trigger points.
3) *Cluster headache* lasts longer per attack, from 15 minutes to 3 hours, and is often accompanied by ipsilateral lacrimation, nasal congestion, conjunctival injection, and other autonomic symptoms.
4) *Temporomandibular joint dysfunction* presents as chronic aching pain in the preauricular joint area, often with limited mouth opening or joint clicking.

4.5. Treatment Recommendations

1) Pharmacotherapy

a) Carbamazepine [22], a sodium channel blocker, is the first-line drug for primary trigeminal neuralgia and has demonstrated efficacy. Secondary trigeminal neuralgia should be managed primarily by removing the cause, but carbamazepine can also relieve symptoms. Adverse reactions include gastrointestinal irritation, ataxia, dizziness, somnolence, and liver dysfunction. Severe skin reactions, including Stevens-Johnson syndrome and toxic epidermal necrolysis, are the most serious adverse reactions. Carbamazepine may also cause aplastic anemia and agranulocytosis; discontinuation should be considered if marked bone marrow suppression occurs [23].

b) Oxcarbazepine [24], a derivative of carbamazepine, may be used clinically.

c) Alternative Medications: Gabapentin [25], pregabalin [26], lamotrigine, and other drugs may be used as adjunctive therapy; major adverse reactions are dizziness and somnolence. Cobratide injection, whose main component is cobra venom neurotoxin, may be used for refractory pain such as trigeminal neuralgia.

2) Minimally Invasive Surgical Interventions

a) Microvascular decompression [27] currently provides the best efficacy and longest duration of relief for classic trigeminal neuralgia. Complications such as facial numbness and chewing weakness are uncommon; complete postoperative pain relief exceeds 90%, and reaches 71% at 10 years [28]. Surgical risks include facial sensory loss, hearing loss, aseptic meningitis, cerebrospinal fluid leak, and cerebellar hematoma [29, 30].

b) Percutaneous balloon compression [27, 31] is safe, simple, and effective, relieving approximately 90% of pain [32], with a 5-year recurrence rate of 15.1%-19.2% [33].

c) Percutaneous radiofrequency thermocoagulation [31] of the trigeminal ganglion and peripheral branches is commonly used and offers good efficacy, minimal trauma, low risk, and repeatability [34, 35].

d) Gamma knife therapy. Few randomized controlled trials are available for gamma knife therapy. The probabilities of remaining pain-free without medication at 3, 5, 7, and 10 years after treatment were reported as 77.9%, 73.8%, 68% and 51.5%, with mean onset of effect beginning approximately 1 month after treatment and facial sensory disturbance occurring in 9%-37% of patients [36-38].

e) Other treatments include chemical lesioning by injecting agents into the trigeminal ganglion, trigeminal cistern, or peripheral branches. Agents include absolute

ethanol, phenol glycerol, botulinum toxin A, and doxorubicin. Chemical lesioning is recommended for peripheral branches as an adjunct to other methods; intracranial trigeminal ganglion lesioning should be used cautiously [24, 38, 39].

The therapeutic modalities for trigeminal neuralgia, along with their respective levels of evidence and grades of recommendation, are presented in Table 3.

Table 3. Treatments for Trigeminal Neuralgia.

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	Sodium channel drugs: carbamazepine, oxcarbazepine	A	Strong
	Calcium channel drugs: pregabalin, crisugabalin, and mirogabalin	A	Weak
	Calcium channel drug: gabapentin	B	Weak
Minimally invasive intervention	Percutaneous balloon compression	A	Strong
	Radiofrequency thermocoagulation	A	Strong
Surgery	Microvascular decompression	A	Strong
Other treatment	Cobratide injection; other chemical lesioning	B	Strong

5. Glossopharyngeal Neuralgia

5.1. Definition

Glossopharyngeal neuralgia, also called vagoglossopharyngeal neuralgia, is a chronic painful disorder in the distribution of the glossopharyngeal nerve and the auricular and pharyngeal branches of the vagus nerve, including the posterior tongue, tonsillar fossa, pharynx, mandibular angle, and/or ear. It usually presents as brief, recurrent, unilateral stabbing pain with abrupt onset and termination, often triggered by swallowing, speaking, or coughing [21]. It may be accompanied by vagal dysfunction, such as cough, hoarseness, syncope, and/or bradycardia. Its incidence is much lower than that of trigeminal neuralgia, accounting for only 0.2%-1.3% of orofacial pain; incidence is 0.2-0.8 per 100, 000, and patients older than 50 years are more commonly affected.

5.2. Pathogenesis

The pathogenesis is complex. It may be related to vascular compression at the nerve root entry zone, most often by a tortuous or sclerotic posterior inferior cerebellar artery or vertebral artery. Other mechanisms include chronic inflammatory stimulation in the cerebellopontine angle causing arachnoid inflammatory thickening and close contact between vessels and nerve roots; degenerative changes of the medullary root leading to demyelination and ephaptic transmission; or inflammation, tumor, and elongated styloid process.

5.3. Diagnostic Criteria [21]

1) Paroxysmal attacks of pain lasting from fractions of a second to 2 minutes, which can also be greater than 30 minutes, fulfilling criteria 2 and 3.
2) Pain fulfills the following 4 characteristics: a) Unilateral; b) Distributed along the posterior tongue, tonsillar fossa, pharynx, beneath the angle of the mandible, and/or ear; c) Pain character can present as electric shock-like, tearing/shooting, stabbing, or sharp; d) Precipitated by swallowing, coughing, speaking, or yawning.
3) Attacks with trigger points, usually on the posterior pharyngeal wall, tonsil, or tongue root.
4) No obvious neurological dysfunction clinically.
5) No attribution to another disease after exclusion by history, physical examination, and/or special investigations. Swallowing often triggers pain, leading to fear of eating, weight loss, dehydration, laryngeal spasm, arrhythmia, and hypotensive syncope. Imaging of the glossopharyngeal region, high-resolution MRI of the skull base, and styloid-position radiographs may show inflammation, tumor, or elongated styloid process.
6) The superior laryngeal nerve is a branch of the vagus nerve. Superior laryngeal neuralgia may resemble glossopharyngeal neuralgia and can be difficult to distinguish clinically. Imaging may show vascular compression of the glossopharyngeal nerve. Some authors recommend the diagnosis of vagoglossopharyngeal neuralgia when pain is accompanied by cardiac arrest, convulsion, or syncope [40].

7) High-resolution MRI of the glossopharyngeal region and skull base, as well as X-rays of the cranial styloid process, may show imaging evidence of inflammation, tumors, and elongated styloid processes in the corresponding areas.

Sensory changes are usually absent on clinical examination; marked sensory change or reduced/absent pharyngeal reflex suggests the need for etiological evaluation.

5.4. Differential Diagnosis

Pain in the third division of the trigeminal nerve often resembles glossopharyngeal neuralgia. Trigeminal neuralgia is usually electric shock-like, triggered by facial mechanical stimulation such as washing, eating, and tooth brushing, and is often responsive to carbamazepine. Glossopharyngeal neuralgia is usually located at the tongue root or related regions, worsens during swallowing, and with only a modest or inconsistent response to carbamazepine.

Elongated styloid process, neck trauma, multiple sclerosis, tonsillar or nasopharyngeal tumor, cerebellopontine angle tumor, lesions around the jugular foramen, retropharyngeal or retromandibular/parotid space lesions, and Arnold-Chiari malformation can cause recurrent pain in the glossopharyngeal distribution and should be differentiated [41, 42].

5.5. Treatment Recommendations

1) Pharmacotherapy

When glossopharyngeal neuralgia is secondary to inflammation, tumor, or elongated styloid process, removal of the cause should be preferred. Pharmacotherapy may include carbamazepine or oxcarbazepine. Local anesthetic application to the tonsil and pharyngeal wall, such as lidocaine, can temporarily relieve pain for several hours [41, 42].

2) Glossopharyngeal Nerve Block [43]

Is usually performed at the midpoint between the mastoid process and mandibular angle, advancing toward the styloid process. At a depth of 3-4 cm the styloid process is located, and local anesthetic or neurolytic agent is injected after the needle tip passes approximately 0.5 cm beyond it. CT or ultrasound guidance improves accuracy and reduces adverse events. A nerve stimulator may be used; paresthesia in the pharynx or ear canal indicates proximity to the nerve. Because the nerve is adjacent to the facial and accessory nerves, neurolytic injection should avoid injury to neighboring nerves.

3) Glossopharyngeal Nerve Radiofrequency [43]

Pulsed radiofrequency or radiofrequency thermocoagulation may be applied to the glossopharyngeal nerve trunk around the styloid process. The puncture point is usually the midpoint between the mastoid process and mandibular angle. Under imaging guidance, the needle tip is adjusted to reach and pass approximately 0.5 cm beyond the styloid process. Sensory and motor stimulation tests are then performed to elicit paresthesia in the glossopharyngeal distribution before radiofrequency treatment [44].

4) Surgical Treatments

Surgical options include microvascular decompression of the glossopharyngeal nerve [45], glossopharyngeal rhizotomy, and partial vagal root section.

The treatment methods for glossopharyngeal neuralgia, along with their levels of evidence and grades of recommendation, are presented in Table 4.

Table 4. *Treatments for Glossopharyngeal Neuralgia.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	Sodium channel drugs: carbamazepine, oxcarbazepine	A	Strong
	Local anesthetic: lidocaine	B	Strong
Minimally invasive intervention	Glossopharyngeal nerve block; glossopharyngeal nerve radiofrequency	A	Strong
Surgery	Glossopharyngeal microvascular decompression	B	Strong

6. Sphenopalatine Neuralgia

6.1. Definition

Sphenopalatine neuralgia (SN), also called Sluder syndrome, was first identified and named by Sluder in 1908. It is a rare atypical facial neuralgia [46]. ICHD-3 regards SN as a former term for cluster headache, but SN is currently recognized as a form of secondary craniofacial pain. SN may result from injury or irritation of the sphenopalatine ganglion, including demyelination, infection, trauma, and tumor compression [47]. No epidemiological data are currently available for SN in the general population. Its clinical manifestations are complex and atypical, making diagnosis difficult.

SN mainly presents as unilateral craniofacial pain accompanied by ipsilateral autonomic symptoms. Pain often involves the lower half of one side of the face, usually beginning behind the nasal root, eye, and maxilla, possibly involving the mandible and gums, and radiating to the frontal, temporal, occipital, and auricular regions. It may be electric shock-like or burning, lasting from minutes to hours, or may be persistent. Attacks may be accompanied by autonomic symptoms including facial flushing, conjunctival injection, photophobia, lacrimation, nasal congestion, and rhinorrhea, and may also include dizziness, nausea, precordial pain, and tinnitus. Physical examination often reveals no obvious positive signs.

6.2. Diagnostic Criteria

He et al. [47] proposed diagnostic criteria distinguishing idiopathic from secondary SN.

1) Idiopathic SN: a) Begins at the nasal root, orbit, maxilla, teeth, and frontotemporal region and may radiate to the mastoid and neck. It presents as unilateral moderate to severe headache lasting minutes to hours or as persistent pain. b) At least one ipsilateral autonomic symptom or sign is present, such as lacrimation and/or conjunctival injection, or nasal congestion and/or rhinorrhea. c) Pain is sporadic or continuous without cluster attacks. d) Oxygen or indomethacin is ineffective or poorly effective. e) the condition is not better explained by another ICHD-3 diagnosis.
2) Secondary SN: a) Symptoms meet the criteria for idiopathic SN. b) Has imaging evidence or history of structural abnormality or disease around the sphenopalatine ganglion. c) With significant pain relief after etiological treatment.

6.3. Differential Diagnosis

SN should be differentiated from trigeminal neuralgia, glossopharyngeal neuralgia, migraine, and tumor-related craniofacial pain.

1) *Trigeminal neuralgia* is electric shock-like, triggered by facial mechanical stimuli such as washing, eating, and tooth brushing, and usually responds to carbamazepine.
2) *Glossopharyngeal neuralgia* is commonly located at the tongue root and worsens during swallowing, with only a modest or inconsistent response to carbamazepine.
3) *Migraine* commonly begins in young women; attacks often start in the unilateral frontotemporal, retro-orbital, or periorbital region, then spread to one side or the entire head, with nausea and/or vomiting and phonophobia and/or photophobia.
4) *Tumor-related craniofacial pain* presents as persistent unrelieved headache that may radiate to the orbit or occipitocervical region when severe and may be accompanied by nausea, vomiting, papilledema, and blurred vision; imaging shows a tumor mass.

6.4. Treatment Recommendations

Treatment includes removal of factors that may irritate the sphenopalatine ganglion and reduction or elimination of the ganglion's response to noxious stimuli.

1) Conservative Treatment

In patients with an identified cause, all causes of ganglion irritation, such as infection or trauma [48] should first be treated. For patients without an identified cause, no evidence-based recommended drug treatment is currently available. One study suggested that, based on effective etiological treatment, carbamazepine and oxcarbazepine can effectively relieve pain; alternatives include gabapentin and pregabalin [47].

2) Minimally Invasive Interventions

Include sphenopalatine ganglion block, pulsed radiofrequency, and neurolytic procedures [49, 50]. Block and/or pulsed radiofrequency is particularly suitable for patients concerned about neurolytic damage. Adverse reactions of block are usually local, including infection, epistaxis, and local hematoma [51]. Neurolysis may be performed by chemical lesioning or radiofrequency thermocoagulation. Radiofrequency thermocoagulation is now widely used because it is minimally invasive and has demonstrated efficacy, but limitations include a small lesion range, recurrence, and facial numbness. Facial numbness after thermocoagulation is very common; other complications include infection, internal bleeding or epistaxis, hemodynamic instability, hyperesthesia and/or sensory disturbance of the upper teeth, hard palate, or pharynx, and decreased tear or nasal mucus secretion.

3) Other Treatments

Include iontophoresis, laser irradiation, stereotactic radiotherapy, acupuncture of the sphenopalatine ganglion, and sphenopalatine ganglionectomy [49].

The treatment methods for sphenopalatine neuralgia, along with their levels of evidence and grades of recommendation, are presented in Table 5.

Table 5. *Treatments for Sphenopalatine Neuralgia.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	Sodium channel drugs: carbamazepine, oxcarbazepine	B	Weak

Treatment modality	Treatment	Evidence level	Recommendation
	Calcium channel drugs: pregabalin, gabapentin	B	Strong
Minimally invasive intervention	Sphenopalatine ganglion block and pulsed radiofrequency; vagus nerve stimulation	B	Weak
	Radiofrequency thermocoagulation	A	Strong

7. Temporomandibular Joint Disorders

7.1. Definition

Temporomandibular joint disorders (TMD) most often begin between 20 and 30 years of age and are common around 45 years of age. Prevalence in women is 3-4 times that in men [52]. The course is long, usually years or decades, and recurrences are common. Disease progression includes an early functional stage, an intermediate structural-change stage, and a late stage of organic joint destruction. TMD is mainly divided into pain-related and joint-related categories; clinical symptoms include pain, joint sounds, and abnormal mandibular movement [52, 53].

7.2. Diagnostic Criteria

The 2014 International Association for Dental Research guideline classifies common TMD into pain-related and joint-related categories [52].

1) Pain-related TMD: a) myalgia, consisting of localized myalgia, myofascial pain, and referred myofascial pain; b) arthralgia, presenting as unilateral or bilateral pain in the face, temporomandibular region, ear, or preauricular area, aggravated by jaw movement; c) headache, presenting as temporal pain aggravated by jaw movement, with clinical examination confirming temporal headache elicited by temporalis palpation or jaw movement.
2) Joint-related TMD: a) disc displacement with reduction, with or without intermittent locking; b) disc displacement without reduction, with or without limited mouth opening; and degenerative joint disease or subluxation. History may include joint noises, transient locking, current locking with limited mouth opening affecting eating, passive mouth opening less than 40 mm when limited or at least 40 mm when unrestricted; c) degenerative joint disease and joint subluxation, history or chief complaint of mandibular locking or catching after wide mouth opening (including brief locking or catching), and the patient is unable to close the mouth without manual assistance. If joint subluxation occurs during the clinical examination, manual assistance is required for reduction.

7.3. Differential Diagnosis

TMD should be differentiated from tumors, suppurative arthritis, and traumatic arthritis.

1) *Tumors* include benign or malignant tumors of the temporomandibular joint, infratemporal fossa tumors, posterior maxillary sinus wall tumors, and nasopharyngeal carcinoma.
2) *Suppurative arthritis* has acute onset, joint pain with swelling, marked tenderness, distinguishing imaging findings, and purulent effusion on joint aspiration. Acute traumatic arthritis presents with joint swelling, pain, and limited mouth opening.
3) *Traumatic arthritis* may present with masticatory muscle soreness, intra-articular noise, limited mouth opening, and joint or facial pain.

7.4. Treatment Recommendations

Treatment aims to relieve pain in the temporomandibular joint region and improve mouth opening and joint function [52, 54]. Noninvasive methods should be used whenever possible, including Pharmacotherapy, physical therapy, and minimally invasive interventions.

Common noninvasive drug treatments include oral NSAIDs [55], glucocorticoids, centrally acting muscle relaxants, antidepressants and anxiolytics, topical NSAID patches, and traditional Chinese medicine patches such as Qufeng Gutong gel plaster [56]. Physical treatments may include heat, ultrasound iontophoresis, electrical stimulation, acupuncture, high-energy laser irradiation, magnetic therapy, low-frequency therapy, and extracorporeal shock wave therapy [57, 58]. Occlusal splint therapy and mouth-opening training aim to improve occlusal function [54, 59]. Patients should be advised to consume a soft diet, take small bites, and chew slowly, and targeted cognitive education and behavioral treatment should be provided when appropriate.

Table 6. Treatments for Temporomandibular Joint Disorders.

Treatment modality	Treatment	Evidence level	Recommendation
Oral Pharmacotherapy	NSAIDs	A	Strong
	Glucocorticoids	B	Weak
	Centrally acting muscle relaxants	B	Weak
	Antidepressants and anxiolytics	B	Weak
Topical Pharmacotherapy	Qufeng Gutong gel plaster	B	Weak
	Yunnan Baiyao	B	Weak
Physical therapy	High-energy laser	A	Strong
	Shock wave therapy	A	Strong
Intra-articular injection	Dexamethasone	A	Strong
	Dexamethasone palmitate injection	A	Strong
	Platelet-rich plasma	B	Strong
	Botulinum toxin	B	Weak
	Ozone	B	Weak

Minimally invasive interventions include temporomandibular joint cavity injection, such as dexamethasone palmitate injection [60], with joint lavage when necessary to relieve pain, lubricate the joint, and promote structural remodeling. If clicking is present, lateral pterygoid muscle block [61] may be performed. Platelet-rich plasma [62] and botulinum toxin injections [63-65] may also be used.

The treatment methods for temporomandibular joint dysfunction syndrome, along with their levels of evidence and grades of recommendation, are presented in Table 6.

8. Head and Facial Herpes Zoster-Related Neuralgia

8.1. Definition

Head and facial herpes zoster (HZ) is caused by reactivation and massive replication of latent varicella-zoster virus (VZV) in cranial ganglia when immune function declines. The virus spreads along sensory nerves to the innervated skin and induces rash and pain.

8.2. Clinical Manifestations

1) Rash usually appears on one side of the head and face. The affected area first develops erythema, followed by clustered papules the size of millet to soybeans, which then become vesicles. Lesions correspond to the affected neural segment. Pain is variable, commonly stabbing, burning, electric shock-like, cutting, or tearing.
2) Pain may appear before or after the rash, or concurrently with it.
3) Postherpetic neuralgia (PHN) is defined as pain persisting for 1 month or longer after HZ rash healing [66]. PHN is the most common complication of HZ. Patients with HZ in the trigeminal distribution of the head and face, especially the ophthalmic branch, often have severe pain and a higher PHN risk [67, 68].
4) Ramsay Hunt syndrome (RHS) commonly occurs when VZV invades the geniculate ganglion and may coexist with infection of adjacent ganglia, such as the trigeminal, spiral, and vestibular ganglia and corresponding nerves. It mainly presents as ear canal rash, pain, and peripheral facial paralysis [69].

8.3. Differential Diagnosis

Head and facial HZ should be differentiated from primary trigeminal neuralgia, glossopharyngeal neuralgia, occipital neuralgia, and other neuropathic pain disorders.

1) *Primary trigeminal neuralgia* presents with abrupt electric shock-like pain and specific trigger points, whereas HZ more often presents as continuous burning pain with cutaneous hyperalgesia and subsequent vesicles.
2) *Glossopharyngeal neuralgia* is mainly triggered by swallowing and lacks skin lesions, whereas HZ involving this region, such as RHS, often has vesicles in the external auditory canal and peripheral facial palsy.
3) *Occipital neuralgia* usually presents as paroxysmal throbbing pain arising from cervical muscle tension or cervical spondylosis, with radiating pain on compression

of the greater occipital nerve exit; HZ presents with burning of the occipital scalp and clustered erythematous vesicles.

8.4. Treatment Recommendations

1) Pharmacotherapy

Antiviral drugs such as acyclovir [70-72] should be added early in patients with head and facial HZ. Because these patients, especially those with trigeminal ophthalmic branch infection, are at high risk for PHN, early combined use of the calcium channel modulator pregabalin [73] is recommended to prevent PHN.

When PHN develops, recommended first-line drugs include calcium channel modulators [73-75] such as pregabalin and gabapentin, antidepressants, and lidocaine patches [76]. Second-line drugs mainly include opioids, tramadol [77], and neurotropin.

Pregabalin and gabapentin have similar adverse reactions, including somnolence, dizziness, peripheral edema, and blurred vision. Calcium channel drugs should be titrated gradually upward or downward [78].

2) Physical Therapy

Common physical treatments include ultrashort wave therapy, high-energy laser, super lizer irradiation, and red-light irradiation [79]. These treatments mainly improve inflammation at skin lesions, reduce nerve edema, and promote local blood circulation, thereby providing anti-inflammatory and analgesic effects and promoting nerve recovery and regeneration.

3) Minimally Invasive Interventions

Nerve trunk, plexus, and peripheral terminal blocks are commonly used for HZ-related craniofacial pain. During head and facial injection therapy, suspension-type particulate glucocorticoids should be avoided as much as possible to prevent severe embolic complications if particles enter blood vessels, such as blindness and stroke. Nonparticulate water-soluble glucocorticoids such as dexamethasone are recommended. Clinically, minimally invasive treatments for PHN mainly include neuromodulation, such as pulsed radiofrequency and nerve stimulation [80-82].

The treatment methods for craniofacial herpes zoster neuralgia, along with their levels of evidence and grades of recommendation, are presented in Table 7.

Table 7. *Treatments for Head and Facial Herpes Zoster-Related Neuralgia.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	Antiviral drugs: acyclovir, valacyclovir	A	Strong
	Calcium channel drugs: gabapentin, pregabalin	A	Strong
	Antidepressants and anxiolytics: duloxetine	A	Strong
	Topical drugs: lidocaine preparations (patch, ointment, etc.)	A	Strong
	Opioids: tramadol, oxycodone sustained-release tablets	B	Weak
Physical therapy	High-energy laser	A	Strong
Minimally invasive intervention	Pulsed radiofrequency	A	Strong
	nerve stimulation	A	Strong

9. Post-Stroke Persistent Headache

9.1. Definition

Post-stroke persistent headache (PSPH) is a new headache closely temporally related to cerebral infarction or nontraumatic cerebral hemorrhage and clearly different from any prior headache in location, severity, quality, or attack frequency. It usually persists after the stroke has been adequately treated or has spontaneously stabilized for 3 months [21]. The prevalence of PSPH is 10.8%-23.4% [83-87]. Female sex, young stroke, posterior circulation involvement, larger infarct/hemorrhage area, and pre-existing chronic headache are risk factors [88-91].

Post-stroke neuroinflammation [92], involvement of pain-modulating brain regions or nuclei such as trigeminal nuclei, thalamus, brainstem, and insula [93, 94], central sensitization of nociceptive pathways, and weakened descending inhibition may underlie PSPH [95]. Clinically, PSPH can be tension-type-like or migraine-type-like.

9.2. Diagnostic Criteria [21]

1) Occurrence of a stroke event with definitive clinical or neuroimaging evidence.
2) A new headache, different in quality, frequency, or duration from previous headache, closely temporally related to the stroke, usually from 72 hours before stroke symptoms to 7 days after symptom onset.
3) The headache severity during: 1) the acute phase varies with changes in stroke symptoms, signs, or imaging findings; 2) headache persists after the stroke has been adequately treated or has spontaneously stabilized for 3 months; 3) for hemorrhagic stroke, headache is ipsilateral to the hemorrhage.
4) The headache is not better explained by another ICHD-3 headache type.

9.3. Differential Diagnosis

PSPH should be differentiated from pre-existing headache, headache related to abnormal intracranial pressure, post-cerebral angiography headache, medication-overuse headache, and substance-withdrawal headache.

1) *Pre-existing headache* has the same quality, location, and accompanying symptoms as before stroke, whereas PSPH must be a new headache type or show significant changes in frequency or intensity.
2) *Headache due to abnormal intracranial* pressure has prominent postural features and imaging changes in the ventricles, whereas PSPH usually has no obvious postural pattern.
3) *Post-cerebral angiography headache* occurs within 24 hours after the procedure and usually resolves within 72 hours, whereas PSPH lasts longer than 3 months.
4) *Medication-overuse headache* involves long-term high-frequency analgesic use, more than 10-15 days per month, with headache changes or relief after withdrawal, whereas PSPH is not directly caused by drug abuse.
5) *Substance-withdrawal headache* occurs shortly after cessation of caffeine, alcohol, or other substances and is self-limited, resolving within days, whereas PSPH is a long-term pathological process caused by brain tissue injury.

9.4. Treatment Recommendations

1) Pharmacotherapy

For PSPH with migraine features, similar medications may be used, such as NSAIDs [96] or compound preparations in the acute phase, including ibuprofen and ketoprofen [97-99]. Triptans and dihydroergotamine are prescription migraine treatments, but are not recommended for ischemic stroke patients because of vasoconstrictive effects. Maintenance therapies that may be considered include antiepileptic drugs such as topiramate [100] and valproate, the beta-blocker metoprolol, antidepressants such as amitriptyline [101] and duloxetine, and the centrally acting muscle relaxant tizanidine [102].

2) Physical Therapy

Repetitive transcranial magnetic stimulation [103-105], used to prevent migraine attacks, may benefit PSPH, but may induce seizures and is contraindicated in patients with intracranial metal implants. Shock wave therapy [106, 107] is effective for post-stroke neck, shoulder, limb muscle stiffness, and spasticity.

3) Minimally Invasive Interventions [108-110]

These mainly include pulsed radiofrequency or block of the greater occipital nerve, C2 and C3 posterior medial branches, and the sphenopalatine ganglion. Stellate ganglion block is also commonly used. Adverse reactions include local numbness and pain. Contraindications include coagulation dysfunction and allergy to anesthetic drugs.

4) Traditional Chinese Medicine and Others [111-113]

Acupuncture [114], cognitive behavioral therapy, biofeedback, and other approaches may be used as adjunctive therapy, but acupuncture is contraindicated in patients with coagulation dysfunction, skin infection, or ulceration.

Table 8. *Treatments for Post-Stroke Persistent Headache.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	NSAIDs or compound preparations	B	Weak
	Antiepileptic drugs: topiramate, sodium valproate	B	Weak
	Calcium channel drug: pregabalin	A	Strong
	Antidepressants: amitriptyline, duloxetine	A	Strong
	Centrally acting muscle relaxant: tizanidine	B	Weak
Physical therapy	Repetitive transcranial magnetic stimulation	A	Strong
	Shock wave therapy	B	Weak

Treatment modality	Treatment	Evidence level	Recommendation
Minimally invasive intervention	Pulsed radiofrequency or block of greater occipital nerve, C2-C3 posterior medial branches, sphenopalatine ganglion; stellate ganglion block; nerve stimulation (SCS, MCS, DBS)	B	Strong
Traditional Chinese medicine and others	Acupuncture	B	Weak

9.5. Prevention and Rehabilitation

For patients with frequent PSPH attacks, defined by reference to migraine as at least 6 days per month, preventive treatment during asymptomatic periods may reduce attack frequency. Exercise, relaxation training, biofeedback, traditional Chinese medicine, and physical therapy are low-risk interventions for stroke patients and may promote recovery from stroke symptoms.

The treatment methods for post-stroke persistent headache, along with their levels of evidence and grades of recommendation, are presented in Table 8.

10. Cancer-Related Craniofacial Pain

10.1. Definition

Chronic cancer-related craniofacial pain refers to pain caused by the primary tumor itself or by metastasis, namely chronic cancer pain, or pain caused by cancer treatment, namely chronic post-cancer-treatment pain [115]. It mainly includes pain caused by brain tumors, nasopharyngeal tumors, oral mucosal tumors, facial or auricular tumors, or tumors metastatic to other head and facial sites. Specific incidence data are lacking and the condition is closely related to cancer progression and cancer-related treatment.

10.2. Pathogenesis

The mechanisms of head and facial cancer-related pain are mainly associated with compression or infiltration of adjacent nerves by the tumor itself or by distant metastatic tumors or lymph nodes. Pain is also closely related to chemotherapy, radiotherapy, and surgery performed for tumors, such as meningioma, skull base tumors, and nasopharyngeal carcinoma [116].

10.3. Clinical Manifestations

1) Headache is a common manifestation of brain tumors. Brain tumor-related headache is usually persistent dull pain, but may occasionally be throbbing. The most common headache phenotype is tension-type headache, accounting for 40%-80%, followed by migraine at 10.0% [117]. Headache is often bifrontal and more severe on the tumor side, and symptoms usually worsen gradually.
2) In nasopharyngeal carcinoma, the most common initial symptom is a cervical mass caused by regional lymph node metastasis, seen in nearly 90.0% of patients. Symptoms from the primary tumor include hearing loss related to serous otitis media, tinnitus, nasal congestion and pain, muscle involvement from invasion of adjacent structures, and dysfunction of cranial nerves II-VI [118].
3) Oral tumors such as tongue cancer and lip cancer may present with oral pain or persistent oral ulceration, loose teeth, ill-fitting dentures, dysphagia, odynophagia, weight loss, bleeding, or referred otalgia.
4) Any solid or hematologic tumor may involve the leptomeninges, but common tumors include lung cancer, breast cancer, lymphoma, and leukemia [119, 120]. Some patients have headache with or without increased intracranial pressure, occasionally accompanied by nausea and vomiting. Metastases involving the skull base or leptomeninges, or soft-tissue tumors of the head, neck, and sinuses, may cause malignant tumor-related cranial neuralgia, such as glossopharyngeal neuralgia or trigeminal neuralgia. Head and neck radiotherapy may also cause oral mucositis or ulceration; pain is mainly burning or discomfort and may later affect swallowing and speech [121, 122].

10.4. Diagnostic criteria

Diagnosis requires head or facial pain, including nociceptive, neuropathic, or mixed pain; definite tumor of the head and face or another site, with or without metastasis, supported by imaging or pathology; and confirmation that clinical manifestations such as pain are directly related to the tumor itself or tumor-related treatment, including radiotherapy, chemotherapy, or surgery. All three conditions are required.

10.5. Differential Diagnosis

Differential diagnosis is mainly from non-cancer-related craniofacial pain disorders.

1) *Primary trigeminal neuralgia* presents as abrupt electric shock-like severe pain with normal neurological examination, whereas cancer-related pain may mimic it but often includes persistent background pain and early neurological deficits such as facial numbness and loss of corneal reflex.
2) *Atypical facial pain* is usually chronic aching pain with vague localization, not following neuroanatomical distribution and with normal imaging, whereas cancer-related pain has clear anatomical localization, progressive worsening, and organic signs such as diplopia or nasal congestion.
3) *Burning mouth syndrome* presents as bilateral symmetric burning with normal-appearing oral mucosa, often milder in the morning and worse in the evening, whereas cancer-related pain is almost always unilateral and fixed, with local mass, ulcer, or lymphadenopathy on examination.

10.6. Treatment Recommendations

1) Pharmacotherapy

Treatment principles include active treatment of the primary tumor, multidisciplinary collaboration, and longitudinal pain management by the department of pain medicine.

a) For mild to moderate pain, NSAIDs and compound opioid analgesics [123-127] may be used early, with attention to ceiling effects and avoidance of prolonged use. Examples include diclofenac sodium, loxoprofen sodium, hydrocodone/acetaminophen, and tramadol/acetaminophen; gastrointestinal and cardiovascular adverse reactions should be monitored.
b) Opioids [128, 129], such as morphine and different oxycodone preparations, are used for moderate to severe pain. During use, pain should be dynamically assessed and combination therapy considered. Adverse reactions include nausea, vomiting, pruritus, and constipation.
c) Calcium channel drugs [130, 131], mainly pregabalin and gabapentin, should be used early in patients with neuropathic pain. Dizziness should be monitored at initiation, and long-term use may cause lower-limb edema.
d) Bone metabolism drugs [132, 133], such as zoledronic acid and denosumab, may be used once bone destruction is identified.
e) Centrally acting muscle relaxants, including tizanidine and baclofen, may be used early in combination therapy; adverse reactions include dizziness and fatigue.
f) Other agents may be combined according to the patient's condition, including cobratide injection, lappaconitine, sedatives, anxiolytics and antidepressants [130, 134], dehydrating agents, and Chinese patent medicines.

2) Minimally Invasive Interventions

When conservative treatment is ineffective, minimally invasive interventions may be performed early and at an appropriate time [135]. Relevant examinations should be completed and contraindications excluded. Pulsed radiofrequency [43, 136] or peripheral nerve stimulation [137-139] may be considered for responsible nerves such as the trigeminal or occipital nerve, and intrathecal drug delivery systems [140, 141] may also be used. However, RCT evidence for these methods is generally limited.

3) Surgical Treatments

Surgical indications should be strictly controlled and treatment should be carried out through multidisciplinary collaboration.

The treatment methods for tumor-related craniofacial pain, along with their levels of evidence and grades of recommendation, are presented in Table 9.

Table 9. *Treatments for Cancer-Related Craniofacial Pain.*

Treatment modality	Treatment	Evidence level	Recommendation
Pharmacotherapy	NSAIDs and compound opioid analgesics: diclofenac sodium, loxoprofen sodium, hydrocodone/acetaminophen, tramadol/acetaminophen	A	Strong
	Opioids: morphine, oxycodone	A	Strong
	Calcium channel drugs: pregabalin, gabapentin	A	Weak
	Bone metabolism drugs: zoledronic acid, denosumab	A	Weak
	Anxiolytics and antidepressants: duloxetine	B	Weak
Minimally invasive intervention	Intrathecal drug delivery	B	Strong
	Pulsed radiofrequency	B	Weak
	Peripheral nerve stimulation	B	Weak

Treatment modality	Treatment	Evidence level	Recommendation
Other drugs	Cobratide enteric-coated capsules/injection	B	Strong

11. Conclusion

The eight disorders described above are highly representative of craniofacial pain, and their clinical manifestations often overlap; careful differential diagnosis is therefore required. Treatment should prioritize effective pain relief and functional improvement. For patients in the acute attack phase or during diagnostic treatment, comprehensive therapy combining nerve block with integrated Chinese and Western medicine is recommended. For refractory or intractable cases, short-term or long-term implanted peripheral nerve stimulation and other neuromodulation techniques may be considered to expand treatment options and overcome limitations of traditional therapy. In addition, because ideal disease-specific drugs remain lacking and some treatments still lack high-level evidence, more high-quality clinical studies are urgently needed.

Abbreviations

AGREE II	Appraisal of Guidelines for Research and Evaluation II
CEH	Cervicogenic Headache
CHISG	Cervicogenic Headache International Study Group
CT	Computed Tomography
DBS	Deep Brain Stimulation
GRADE	Grading of Recommendations Assessment, Development and Evaluation
HZ	Herpes Zoster
ICD-11	International Classification of Diseases, 11th Revision
ICHD-3	The International Classification of Headache Disorders, 3rd edition
MCS	Motor Cortex Stimulation
MRI	Magnetic Resonance Imaging
NSAIDs	Nonsteroidal Antiinflammatory Drugs
PNH	Postherpetic Neuralgia
PICO	Population, Intervention, Comparison and Outcome
PSPH	Post-Stroke Persistent Headache
RIGHT	Reporting Items for Practice Guidelines in Healthcare
RHS	Ramsay Hunt Syndrome
RCTs	Randomized Controlled Trials
SCS	Spinal Cord Stimulation
SN	Sphenopalatine Neuralgia
VZV	Varicella-Zoster Virus

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Conflicts of Interest

The authors declare no conflicts of interest.

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