

Facial pain: the differential diagnosis in an ENT clinic

BY BHASKAR RAM AND SANGEETA MAINI

The patient presenting with facial pain can be a heartsink. Fear not – **Bhaskar Ram** and **Sangeeta Maini** are here with a succinct overview of the common non-sinogenic causes of facial pain and headache, and how to manage them.



Facial pain is a common complaint in the ENT outpatient setting. Facial pain can be the presenting, and sometimes the only, complaint of many disorders that originate from head and neck structures. Identification of the underlying cause occasionally represents a challenge, even for an experienced clinician. Conventionally, in the evaluation of headache and facial pain, the primary goal for the otolaryngologist was to make a distinction between sinogenic and non-sinogenic causes, however we feel that otolaryngologists should try and equip themselves with the ability to diagnose and manage the common causes of primary headaches which often mimic sinusitis presentation, to shorten the patient's journey. Of course the complex and rare forms of primary headache would need referral to a neurologist.

Facial pain can be broadly divided into two types, namely sinogenic and non-sinogenic facial pain.

Sinogenic facial pain

Sinogenic facial pain includes acute and acute-on-chronic sinusitis. Facial pain is uncommon in chronic sinusitis! Facial pain related to sinus infection must have the following features [1-4]:

- Clinical, nasal endoscopic and / or radiological evidence of acute or acute-on-chronic rhinosinusitis. There should be no other features of a primary headache which could present simultaneously.
- Facial pain must develop simultaneously with the onset or acute exacerbation of rhinosinusitis.
- Facial pain must resolve within seven days of remission or successful treatment of acute or acute-on-

chronic rhinosinusitis.

- Sinonasal pain is usually deep, aching and non-pulsatile.

Non-sinogenic facial pain

Non-sinogenic facial pain can be broadly subdivided into the following categories: neuropathic facial pain – migraine, tension headache, midfacial segment pain; cranial neuralgias – trigeminal neuralgia (TN), trigeminal autonomic cephalgias; orofacial pain – dental pain, temporomandibular joint (TMJ) dysfunction, myofascial pain.

Neuropathic facial pain

Migraine

Migrainous pain is typically considered to be a throbbing, predominantly unilateral pain (although up to 40% may have bilateral symptoms) [5]. It is three times more common in women and a positive family history often exists. Classic migraine occurs in 25% of patients with associated symptoms such as nausea, aura and photophobia. Migraine symptoms can also overlap with those more characteristic of tension, cluster or so-called sinus headache. Up to one quarter of migraine sufferers develop autonomic symptoms with associated nasal congestion, nasal discharge and watery eye during acute attacks and can thus be mistaken for rhinosinusitis. In addition, facial pain may be the only presenting feature with no associated headache in up to 50%. Depending on acute or chronic presentation, migraine treatment involves abortive or prophylactic therapy.

Abortive therapy comprises simple analgesics alone or in combination with other compounds which can provide relief for mild to moderately severe headaches. For severe headaches and facial pain, 5-HT₁ agonists and / or opioid analgesics alone or in combination with

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dopamine antagonists can be used. The use of abortive medications must be limited to 2-3 days a week to prevent development of a rebound headache phenomenon.

Prophylactic therapy can be considered for the following conditions:

1. Chronic migraine-attacks lasting for >8 days per month for three months
2. Contraindication to or ineffectiveness of symptomatic medications
3. Use of abortive medications more than twice a week.

In our experience nortriptyline 10-100mg once daily and propranolol 80-140mg per day seem to be very effective for chronic facial migraine. In recent years, botulinum toxin has been used in multiple trials for the prophylaxis of migraine headaches. Findings have demonstrated that botulinum toxin is an effective prophylactic treatment for chronic migraine and has resulted in significant improvements compared with placebo in multiple headache symptom measures. Significantly reduced headache-related disability and improved functioning, vitality and overall health-related quality of life were also observed.

Tension headache

Tension-type headache (TTH) represents one of the most expensive diseases for the health service. TTH can occur at any age, but onset during adolescence or young adulthood is common [6]. Women are slightly more likely to be affected than men. TTH is the most common type of headache, and it is classified as episodic (ETTH) or chronic (CTTH) and is now believed to be due to abnormal neuronal sensitivity and pain facilitation, and not due to abnormal muscle contraction. TTH is characterised by pain that is usually mild or moderate in severity and bilateral in distribution. The headache is a constant, tight, pressing or band-like sensation mainly in the frontal

and temporal regions. Patients with TTH have normal findings on general and neurologic examinations. Some patients may have tender spots or taut bands in the cervical muscles (trigger points). Various precipitating factors may cause TTH in susceptible individuals including stress, sleep deprivation, bad posture, hunger and eye strain. Half of patients with TTH identify stress or hunger as a precipitating factor. Management of TTH consists of pharmacotherapy, physical therapy and psychophysiological therapy.

Pharmacotherapy consists of abortive therapy and long-term preventive therapy. These headaches (especially ETTH) generally respond to simple over-the-counter (OTC) analgesics. Patients must be discouraged from overusing analgesics because of the risk of dependence, abuse, and development of chronic daily headache.

Preventive drugs are the main therapy for CTTH. Preventive medications may be considered if headaches are frequent (>2 attacks per week), of long duration (>3-4 h), severe enough to cause significant disability. Nortriptyline in low dosage (10-100 mg) is the most frequently used tricyclic antidepressant for prophylaxis. The following various minimally invasive techniques may also provide pain relief: "trigger point" injections, greater or lesser occipital nerve blocks and auriculotemporal nerve block.

Physical therapy techniques include hot or cold applications, positioning, stretching exercises, traction, massage, ultrasound therapy, transcutaneous electrical nerve stimulation (TENS) and manipulations.

Psychophysiological therapy includes reassurance, counselling, relaxation therapy, stress management programmes and biofeedback techniques.

Midfacial segment pain

This is a form of TTH of the mid-face, with a symmetric pressure sensation in the nasion, nasal dorsum, periorbital and / or

malar regions. Hyperaesthesia of the skin and soft tissues is also found. Treatment consists of low-dose nortriptyline (10-50 mg) for six months, which may take up to six weeks to show effect.

Cranial neuralgias [7]

Cranial neuralgias are a group of disorders characterised by sudden lancinating pains that are unilateral and limited to the distribution of the affected cranial nerves. These include trigeminal neuralgia, cluster headaches, paroxysmal neuralgias, SUNCT (short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing) and SUNA (short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms).

Trigeminal neuralgia

This is the most common cranial neuralgia, with an incidence of up to 5 per 100,000. It is a brief, paroxysmal unilateral stabbing pain in the distribution of one or more of the branches of the fifth cranial nerve. The mandibular branch is most commonly affected. Pain is triggered by minimal stimulation of the affected area, often in the location of a "trigger zone." Patients seldom have any associated sensory loss or background pain. Most cases are idiopathic, but secondary TN can be caused by vascular or neoplastic compression of the trigeminal ganglion or infiltrating lesions, hence MRI is indicated. Treatment initially consists of carbamazepine or other antiepileptic drugs. Surgical microvascular decompression and gamma knife radiation have been shown to treat cases caused by compression of the trigeminal nerve from pontine vessels. Other less invasive techniques include balloon dilatation of the trigeminal ganglion and glycerol ganglion lysis. Microvascular decompression may provide the best long term remission of over 10-15 years when compared to other techniques.

Table 1: Trigeminal autonomic cephalgias

| | Cluster headache | Paroxysmal hemicrania | SUNCT |
|--------------------------|------------------|-----------------------|-------------------|
| Attack frequency (daily) | 1-8 | 1-40 | 3-200 |
| Duration of attack | ¼-3 hrs. | 2-30 mins. | 5-240 secs. |
| Pain quality | Sharp, throbbing | Sharp, throbbing | Stabbing, burning |
| Pain intensity | Very severe | Very severe | Very severe |
| Treatment | Sumatriptan | Indomethacin | Lamotrigine |
| Circadian periodicity | 70% | 45% | Absent |

Trigeminal autonomic cephalgias

These are a group of neuralgias which typically cause recurrent lancinating pain over the ophthalmic branch of the trigeminal nerve and almost always are associated with autonomic symptoms including ipsilateral tearing, nasal discharge, ptosis and miosis. They consist of three types: cluster headaches, paroxysmal hemicranias, SUNCT and SUNA (Table 1).

Cluster headache, also known as histamine headache, is a form of neurovascular headache. Attacks are usually severe, unilateral and located at the temple and periorbital region. The pain is typically associated with ipsilateral lacrimation, nasal congestion, conjunctival injection, miosis, ptosis and lid oedema. Each headache is brief in duration, lasting a few moments to two hours. Cluster headaches most commonly occur in men aged 30-40 years. Treatment of an acute cluster headache is with oxygen (8 L/min or 100% by mask for 10 min), which may abort the headache if used early. Subcutaneous injections of sumatriptan (6 mg) can be effective, in large part due to the rapidity of onset. Studies have indicated that intranasal administration is more effective than placebo but not as effective as injections. No evidence suggests that they are effective orally.

Paroxysmal hemicrania is characterised by recurrent attacks of severe unilateral periorbital pain lasting 2-30 minutes. Patients typically have 5-40 attacks per day and, similar to cluster headache, also have autonomic symptoms such as ipsilateral nasal congestion, rhinorrhoea and tearing. Approximately 65% of untreated patients go on to develop hemicrania continua with chronic headaches. Diagnosis is made based on history and response to indomethacin.

25mg-75mg thrice daily administration of indomethacin during acute spells typically resolves the pain within 72 hours.

SUNCT / SUNA are a form of periorbital pain / neurovascular headache characterised by recurrent severe sharp, electric shock-like pain, lasting seconds and centred around the periorbital region. Patients typically have 30-200 attacks daily and almost always have associated ipsilateral autonomic symptoms. Lamatrogine 100-400 mg per day remains the main drug of choice for this condition.

Orofacial pain [8]

Dental pain

Pain of dental origin can be referred to many areas of the head and face. Facial pain of dental origin is often caused by caries which progresses to infection of the pulp, apical abscess or periodontal disease.

Temporomandibular joint (TMJ)

disorders are known to cause facial pain and headache. Females with TMJ disorders outnumber males, and the onset is usually in those aged 30-50 years. The three main categories of TMJ syndrome are chronic myofascial pain, internal derangement and degenerative joint disease (DJD).

Chronic myofascial pain is most common and is similar in nature to fibromyalgia. The pain is unilateral, dull in character and localised to the pre-auricular region. Pain is exacerbated by chewing, yawning or the stimulation of certain trigger points that are usually located with palpable bands of muscle. Treatment consists of soft diet, analgesics, corticosteroids, local anaesthetic blocks, muscle stretching and treatment of psychological factors.

Internal derangement usually consists of an anterior displacement of the disc.

A dull pre-auricular pain with joint tenderness and an audible or palpable joint "click" upon examination assists diagnosis. Treatment consists of a soft diet, orthotic appliances and physical therapy. Surgical management can be used in refractory cases, but this is becoming less common.

DJD is essentially osteoarthritis of the joint and should be treated with a soft diet and NSAIDs.

Clinical approach to facial pain

History taking should include the points mentioned in Table 2. A thorough head and neck examination should be performed including testing of the cranial nerves, palpation for points of tenderness and trigger points, jaw clicks and dental pain. A neurologic examination should also be performed. If a rhinogenic source is a concern, nasal endoscopy looking for purulence, oedema, inflammation, trauma and tumour should be performed.

Routine requests for computed tomography (CT) scan of the sinuses is discouraged as a diagnostic tool. Magnetic resonance imaging (MRI) is sometimes useful for the evaluation of TMJ disorders, specifically if internal derangement is suspected and symptoms persist. MRI and / or MR angiography (MRA) of the brain is useful to evaluate for intracranial pathology (tumour, hydrocephalus) and vascular sources of headache. It also is used to assess for microvascular compression of cranial nerve roots.

We believe that equipping ourselves with the knowledge of common causes of headaches based on history, clinical features and clinical examination can reduce the need for investigations and unnecessary imaging studies. This will also reduce costs and the length of the patient journey.

Table 2: Clinical features of facial pain

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|--|
| Localisation - unilateral/bilateral |
| Time pattern |
| Onset - sudden, gradual |
| Circadian distribution - day, night, random |
| Course and progression - constant, paroxysmal, recurrent |
| Duration - seconds, minutes, hours |
| Quality - dull, throbbing, pulsating, aching |
| Intensity - sharp |
| Precipitating and alleviating factors - stress, exercise, dehydration; rest, dark room |
| Autonomic symptoms - watery nose / eyes, droopy eyes, facial swelling |
| Associated symptoms and signs - photophobia, phonophobia, visual field defects |

References

1. The Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgia and facial pain; 2nd edition. *Cephalalgia* 2004;**24**(51):24-136.
2. Rebeiz EE, Rastani K. Sinonasal facial pain. *Otolaryngol Clin North Am* 2003;**36**(6):1119-26.
3. Fahy C, Jones NS. Nasal polyposis and facial pain. *Clin Otolaryngol Allied Sci* 2001;**26**(6):510-13.
4. Jones NS. Sinogenic facial pain: diagnosis and management. *Otolaryngol Clin North Am* 2005;**38**(6):1311-25.
5. Blanda M. Headache, Migraine. *eMedicine from WebMD* [serial online]. Available at <http://emedicine.medscape.com/article/792267/overview>.
6. Jensen R. Tension-type headache. *Curr Treat Option Neurol* 2001;**3**(2):169-80.
7. Chavin JM. Cranial neuralgias and headaches associated with cranial vascular disorders. *Otolaryngol Clin North Am* 2003;**36**(6):1079-93.
8. Anion DJ Jar, Gouge LA. Pain from the oral cavity. *Otolaryngol Clin North Am* 2003;**36**(6):1127-35.

LIFETIME PREVALENCE IN GENERAL POPULATION:

Headache: > 90%
 Migraine: 11-15%
 Temporomandibular joint dysfunction: 12-88%
 Sinusitis: 5-15%
 Tension headache: 1-3%
 Trigeminal neuralgia: < 1%



Bhaskar Ram

Department of Otolaryngology,
 Aberdeen Royal Infirmary,
 Foresterhill Rd, Aberdeen, AB25 2ZN, UK.

E: b.ram@nhs.net



Sangeeta Maini

Department of Otolaryngology,
 Aberdeen Royal Infirmary,
 Foresterhill Rd, Aberdeen, AB25 2ZN, UK.

E: sangeetamaini@nhs.net

Declaration of Competing Interests:
 None declared.