41 Headache and Facial Pain: Diagnosis, Evaluation and Management

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Introduction

Facial pain refers to any type of pain in the area bounded by the eyes and the lower mandibular margins, including the oral cavity. It is a frequent complaint that affects women more than men (female: male ratio 2:1 [1]) and has a population prevalence of around 1.9%.

Pain can be provoked by virtually all structures in the head and neck region. Whilst patients are often referred to ENT surgeons, many have already seen colleagues from other disciplines, such as dentists, ophthalmologists, psychologists, pain specialists, internal medicine physicians, neurologists and neurosurgeons. Patients may have experienced multiple consultations and treatment regimens, passing between different specialities, without perceiving any benefit.

By the time they are seen in the ENT clinic, many will have reached the firm conclusion that the cause of their facial pain lies within their sinuses, reinforced by opinions held by their primary care physician or other hospital specialists. The otolaryngologist may therefore face a significant challenge to dispel the patients' preconceived ideas that their facial pain is sinogenic, and consultations can be onerous and difficult.

Conventionally, the primary goal for the otolaryngologist was to make a distinction between sinogenic and non-sinogenic causes of headache and facial pain. However, an alternative view is for the otolaryngologist to understand pain disorders, make the correct diagnosis and instigate effective management.

Facial Pain

Facial pain can be broadly classified into primary and secondary (Table 41.1).

Primary facial pain has no obvious causative factors and includes migraine, trigeminal neuralgia, trigeminal autonomic cephalgia's, tension-type headaches, mid-facial segment pain, and atypical facial pain.

Secondary facial pain has a specific causative factor and can be subdivided into sinogenic and non-sinogenic causes.

Non-sinogenic facial pain includes pain secondary to dental pathologies, temporomandibular dysfunction and vascular disorders such as Giant cell arteritis.

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Table 41.1 Facial pain

Primary facial pain	Secondary facial pain
Migraine Mid-facial segment pain Tension headache Atypical facial pain Trigeminal neuralgia Trigeminal autonomic cephalgia	Sinusitis Orofacial/dental infections Temporomandibular dysfunction Giant cell arteritis

Approach to a Patient with Facial Pain

A structured approach to history taking remains an essential part to making a diagnosis. It may not be possible to reach a definitive diagnosis at the first visit and re-taking the history at a subsequent consultation with the patient's diary of their symptoms will aid in the diagnosis.

Systematic Approach to Facial Pain Diagnosis

History and Examination

One should elicit the following:

- Where is the pain?
  - Asking the patient to point with one finger to the site of the pain is often helpful.
- What is the type of pain? Is the pain superficial or deep?
  - Pain from the skin is sharp and localised. Deep-seated pain is dull and poorly localised.
- How long does the pain last?
  - Migraine-related pain lasts for a few hours, pain in trigeminal neuralgia and short-lasting unilateral neuralgiform headache (SUNCT) lasts for seconds to minutes.
- Is it unilateral or bilateral?
  - Migraine, cluster headaches and trigeminal neuralgia tend to be unilateral. Tension headaches and mid-segment facial pain tend to be central or bilateral.
- Is there any facial numbness?
  - If yes, these patients need full neurological assessment to exclude intracranial lesion. These patients often need an MRI brain/Sinus scan.
- Periodicity:
  - The periodicity of symptoms may be a pointer to the diagnosis, e.g. being woken in the early hours by severe facial pain which lasts about 45–120 min suggests cluster headache.
  - Monthly premenstrual headaches are typical of hormonal/menstrual migraine.
- Pattern of attacks:
  - The relentless progression of a headache, in particular if associated with nausea or effortless vomiting is worrying, and an intracranial lesion should be excluded.
- What precipitates the pain and what relieves the pain?
  - Ask whether hot, cold, eating, touching the face, weather, chocolates, wine, periods or stress precipitates the pain—more commonly seen in trigeminal neuralgia and migraine, respectively.
- Ask for any associated symptoms?
  - Nausea, vomiting, aura, nasal symptoms, clenching, bruxism habits, locking or clicking of jaw joint, altered sensation, eye symptoms and toothache.
- Any other pain conditions?
  - e.g. fibromyalgia
- Is there any impact of pain in daily routine?
  - e.g. sleep, mood, concentration, fatigue, beliefs and quality of life.
- A full drug history is important and what treatment has been tried before.
- Past medical history is important.
  - What treatment has patient tried before.
- Psychological history
- It is useful to use questionnaires to help in assessment and monitoring of effects of therapy.
  - Questionnaires such as the "*Pain self-efficacy questionnaire* and *EuroQol 5D*" [2] have been well validated and are sensitive.
  - SNOT 22 questionnaire—high symptom scores of psychosocial symptoms and ear and facial symptoms in absence of rhinosinusitis should raise the suspicion of Primary headache disorders [3, 4].

Examination Should Include the Following

- Nasal examination including nasal endoscopy.
- Oral examination.
- Temporomandibular joint examination—looking for tenderness, crepitus, malalignment and muscle hypertrophy.
- Examination of the muscles of mastication.
- Examination of head and neck muscles for tenderness and trigger points.
- The cranial nerves examination looking for any facial numbness, paraesthesia and weakness.

Investigations

- Laboratory investigations are of limited value except in the potential diagnosis of giant cell arteritis.
- Radiographic X-ray dental imaging or orthopantomogram (OPG) is important for dental pain.
- Magnetic resonance imaging (MRIs) is indicated for suspected cancers and cranial conditions.
- Computerised tomography (CTs) is indicated for suspected chronic sinusitis.

Sinusitis and Facial Pain

Contrary to popular belief, so-called sinus headaches are uncommon and seen mostly in acute sinusitis or acute exacerbations. Acute sinus infection typically follows a bad head cold and may present as a severe throbbing pain that refers to the teeth, in association with purulent nasal discharge and pyrexia.

As per EPOS 2020 Guidelines

Sinusitis is defined as inflammation of the nose and the paranasal sinuses characterised by two or more symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip):- ± facial pain/pressure - ± reduction or loss of smell.
And/Either Endoscopic signs of: nasal polyps, and/or—mucopurulent discharge primarily from middle meatus and/or—oedema/mucosal obstruction primarily in middle meatus. And/Or
CT changes: mucosal changes within the ostiomeatal complex and/or sinuses.

Patients with facial pain secondary to sinusitis, almost invariably have coexisting symptoms of nasal obstruction, hyposmia and/or purulent nasal discharge and there are usually endoscopic signs of disease [5].

Patients with acute maxillary sinusitis complain of unilateral facial and dental pain, and endoscopy often confirms the diagnosis with findings of purulent discharge from the middle meatus. A normal nasal cavity showing no evidence of middle meatal mucopus or inflammatory changes makes a diagnosis of sinogenic pain most unlikely [6]. Key points within the medical history that are consistent with sinogenic pain are exacerbation of pain during an upper respiratory tract infection, an association with rhinological symptoms, worsening of symptoms during flying or skiing, and a good response to medical treatment.

Chronic sinusitis seldom causes facial pain except during an acute exacerbation, although this diagnosis is often made too readily. Patients with chronic rhinosinusitis will normally have endoscopic features of polypoidal mucosal change, nasal polyps or mucopurulent discharge. In a cohort of patients with suspected sinusitis, actual sinusitis was present in less than a third, and the most common cause of facial pain was migraine [8].

Whilst imaging may offer important diagnostic information interpretation must be treated with caution [7]. Approximately 30% of asymptomatic patients who undergo imaging of the head will demonstrate mucosal thickening in one or more sinuses as an incidental finding. Should patients with facial pain undergo imaging studies, mucosal thickening is not evidence of sinogenic pain or that surgery is indicated. The features should be correlated with a detailed history and endoscopic findings.

Headaches are common in the general population and care must be taken not to link headaches with unrelated nasal symptoms that lead to an erroneous diagnosis of sinusitis. Diagnostic confusion may also occur from autonomic rhinological symptoms associated with vascular pain and cephalgias.

Sinus surgery should only be considered where there is good evidence of sinogenic pain in whom

appropriate medical treatment has failed. In patients who undergo surgery for non-sinogenic pain, some experience temporary pain relief, but the pain typically recurs 2–3 months later, and a third will experience much worse pain that is difficult to manage with medical treatment [8].

Key Points

1. A careful, detailed history is pivotal.
2. Sinogenic facial pain is rarely a symptom on its own. It is most often associated with other nasal symptoms (nasal obstruction, rhinorrhoea or olfactory dysfunction).
3. A CT sinus scan is rarely indicated in the diagnosis of sinogenic facial pain.
4. Surgery should be avoided in patients with facial pain unless there are clear features of sinusitis.

Primary Facial Pain

Migraine and Facial Pain

Migraine is a common disabling primary headache disorder predominantly affecting young and middle-aged women, affecting approximately 18.9% (18.1–19.7) women, and 9.8% (9.4–10.2) men [9].

Classical migraine presents as a unilateral throbbing headache, often associated with nausea, photophobia and phonophobia. It is frequently misdiagnosed but has strict diagnostic criteria (Table 41.2). The headache may be preceded by an aura that can be visual, sensory or motor. Migraine attacks are often induced by stress, hormonal changes and dietary triggers. About 70% of migraineurs have a positive family history.

Table 41.2 The international classification of headache disorders [11]

Migraine without aura (MO) diagnostic criteria
1. *At least five headache attacks lasting 4–72 h (untreated or unsuccessfully treated), which has at least two of the four following characteristics:*
(a) Unilateral location
(b) Pulsating quality
(c) Moderate or severe intensity (inhibits or prohibits daily activities)
(d) Aggravated by walking stairs or similar routine physical activity
2. *During headache at least one of the two following symptoms occur:*
(a) Phonophobia and photophobia
(b) Nausea and/or vomiting
Migraine with aura (MA) diagnostic criteria
1. *At least two attacks fulfilling with at least three of the following:*
(a) One or more fully reversible aura symptoms indicating focal cerebral cortical and/or brain stem functions
(b) At least one aura symptom develops gradually over more than 4 min, or two or more symptoms occur in succession
(c) No aura symptom lasts more than 60 min; if more than one aura symptom is present, accepted duration is proportionally increased
(d) Headache follows aura with free interval of at least 60 min (it may also simultaneously begin with the aura
2. *At least one of the following aura features establishes a diagnosis of migraine with typical aura:*
(a) Homonymous visual disturbance
(b) Unilateral paraesthesia and/or numbness
(c) Unilateral weakness
(d) Aphasia or unclassifiable speech difficulty

There may be a significant symptom overlap between a sinogenic headache and migraine that must be appreciated by the otolaryngologist in the quest to differentiate the two. A central lesion must be excluded in patients over the age of 50 years who present with newly diagnosed migraine.

Nasal endoscopy during an acute migraine episode may display significant mucosal inflammation due to autonomic imbalance at the time. The autonomic/cortical dysfunction is believed to induce cephalgia and the associated rhinogenic symptom complex of blockage, lacrimation and rhinorrhoea.

It is thus perfectly understandable for patients who experience increased congestion, rhinorrhoea and lacrimation during migraine episodes to be treated for sinus disease. Furthermore, patients may find that some drugs that treat rhinosinusitis-like symptoms, such as pseudoephedrine, may coincidentally alleviate their 'sinus headaches'. Pseudoephedrine is a vasoconstrictor that may prevent the downstream vasodilation effects of migraine and thus alleviate migraine pain by indirectly treating the migraine. Unfortunately, the pain relief after taking such medication further reinforces the misperception that migraines are sinus headaches.

Management includes the active treatment of acute symptoms, prophylaxis and avoidance of inducing factors, in various combinations according to the frequency and severity of the episodes.

Simple treatment in the acute phase includes aspirin and antiemetics. Serotonin agonists such as rizatriptan are frequently effective in treating acute attacks. Beta-blockers are often the first-line treatment for prophylaxis, provided there are no contraindications to their use. Prophylactic treatment is considered if symptoms occur more than three times a month with a duration of more than 48 h. Local injections of Botulinum Toxin Type A are often effective in refractory cases, but neurological referral is recommended in such patients [10].

Top Tips

- Migraine headaches can often mimic sinusitis.
- Knowledge of the presentation of facial migraine will ensure appropriate investigations, correct diagnosis and effective treatment.
- Migraine headache treatment may be commenced by ENT but refractory cases need neurological referral.

Trigeminal Neuralgia

Patients typically present with paroxysms of severe, lancinating pain, induced by a specific trigger point such as the lips and nasolabial folds. Chewing, talking, drinking hot or cold fluids, touching, shaving and brushing teeth may also precipitate the attacks in these patients. Attacks typically last from seconds to 2 min. The pain occurs in both the maxillary and mandibular divisions in more than one-third of sufferers, but the pain is confined solely to the maxillary division in one-fifth. The ophthalmic division is affected in only 3% of patients.

Patients do not usually have any sensory deficits, but when present, magnetic resonance imaging (MRI) is required to exclude secondary causes such as multiple sclerosis or posterior [12] fossa tumours (present in 2% and 4% of patients, respectively). MRI imaging is also indicated in young patients presenting with unilateral or bilateral trigeminal neuralgia to exclude disseminating sclerosis (DS), as trigeminal neuralgia can be the first manifestation of DS in some patients.

Carbamazepine remains the first-line medical treatment. In cases refractory to medical treatment, referral to specialist centres for consideration of other treatment modalities such as microvascular decompression, glycerol gangliolysis, balloon compression or stereotactic radiotherapy may be appropriate.

Table 41.3 Common key features of trigeminal cranial neuralgias

- Periorbital or ocular pain
- Unilateral pain
- Excruciating severity
- Accompanied by at least one of these ipsilateral autonomic phenomena/signs:
  - Conjunctival injection/lacrimation
  - Nasal congestion/rhinorrhoea
  - Eyelid oedema
  - Forehead/facial sweating
  - Miosis and ptosis

Trigeminal Autonomic Cephalgias (TACs)

Trigeminal autonomic cephalgias (TACs) are primary headaches with a common clinical phenotype consisting of trigeminal pain with autonomic signs such as lacrimation, rhinorrhoea and miosis (Table 41.3).

Three main types are recognised. These are cluster headache, paroxysmal hemicrania, and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT).

Cluster Headaches

Young adults with periodic unilateral orbital pain associated with autonomic symptoms should raise a suspicion of cluster headache, especially if precipitated by alcohol. The classical symptoms include a unilateral, severe stabbing pain over the periorbital region that disturbs sleep. Patients may have 2–3 attacks per day, lasting from 30 min to 2 h, characteristically at a similar time daily, with phases over several weeks, followed by months of remission.

In contrast to migrainous pain where patients prefer to lie down and rest in a dark room, cluster headache induces restlessness without associated nausea or vomiting.

Associated symptoms include rhinorrhoea, lacrimation and conjunctival injections, facial flushing and sometimes miosis during these episodes. As patients experience pain and nasal symptoms, they assume that they have severe sinusitis and seek ENT opinion. Clinician often finds normal Nasal endoscopy and CT scan is usually normal. In such situations, one must have a high index of suspicion to diagnose this condition.

Acute episodes respond well to nasal or subcutaneous sumatriptan or oxygen inhalation. Verapamil or topiramate provide effective prophylaxis if required. A neurological opinion should be considered.

Paroxysmal Hemicrania

Paroxysmal hemicrania is an excruciating unilateral pain occurring almost exclusively in women. It can occur at any time of day or night, with 2–40 attacks per day over several episodes.

Similar to cluster headaches, patients with this condition present with unilateral facial pain/headache, affecting the frontal, ocular, cheek or temporal regions, with associated autonomous symptoms [13]. However, in contrast to cluster headaches, where attacks last for hours, paroxysmal hemicrania headaches are shorter, lasting from 15 min to 30 min.

Patients and clinicians will often attribute symptoms to recurrent sinusitis, and some may occasionally undergo endoscopic sinus surgery, but nasal endoscopy is invariably normal.

Indomethacin, 25 mg orally thrice daily, is the drug of choice, typically inducing a dramatic response that is characteristically diagnostic. In contrast to migraine and cluster headache, paroxysmal hemicrania does not usually respond to triptans. A high index of suspicion should be maintained with a neurological referral if in doubt.

SUNCT

Short-Lasting, Unilateral, Neuralgiform Headache Attacks with Conjunctival Injection and Tearing (SUNCT)

This condition bears many similarities to trigeminal neuralgia but affects the ophthalmic division of the trigeminal nerve with associated autonomic symptoms.

Attacks of sharp, shooting periorbital pains lasts for seconds only, that may total 250 attacks per day. Lamotrigine is the drug of choice and a neurological opinion should be considered.

Table 41.4 Tips: summary points of trigeminal cephalic cephalgias

	Cluster headache	Paroxysmal hemicrania	SUNCT
Attack frequency (daily)	2–3	2–40	250
Duration of attack	30 min to 2 h	15–30 min	5–240 s
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

Table 41.4 summarises the key differences between these three conditions.

Tension Headache

This is another common condition which is commonly seen in young adults. It is usually stress related and anxiety, depression or agitated depression will often co-exist. Patients describe a feeling of tightness, pressure or constriction over their forehead, retro-orbital or temple and cheeks. Hyperaesthesia of the skin or muscles of the forehead often leads patients to assume they have rhinosinusitis, as they know their sinuses lie under the forehead [14]. Patients will often take large quantities of various analgesics without deriving much benefit. Patients are often convinced that their headaches are sinus related, often reinforced by family/friends and sometimes even other healthcare providers.

Treatment should involve good counselling. Low-dose amitriptyline is often effective [15], but propranolol, sodium valproate, gabapentin or a change in lifestyle may also bring successful relief of symptoms. Amitriptyline should be given for at least 6 weeks before judging its effect and should be continued for 6 months if effective. The starting dose is 10 mg/day, and gradually increased up to 50 mg/day over 6 weeks if necessary, according to the clinical response. Patients need to be warned of the sedative effects, even at this low dose, but they can be reassured that tolerance usually develops after the first few days. Symptoms will return after stopping amitriptyline in 20% of patients, and they should re-start medication should this occur.

It is good practice to inform patients that amitriptyline is also used in higher doses for other conditions such as depression, but that it is not being given for this reason. It is often reassuring for patients to know that the dose used for depression is some seven or more times the dose used in tension-type headache. Cognitive behaviour therapy is another option that could be considered.

Mid-Facial Segment Pain

Mid-facial segment pain has all the characteristics of tension-type headache with the exception that it affects the midface. Patients describe a feeling of pressure, heaviness or tightness and they often complain of a blocked stuffy nose despite a normal nasal examination. The symptoms are symmetrical and may involve the nasion, the bridge of the nose, either side of the nose, the periorbital region, retro-orbitally or across the cheeks. The forehead and occipital region may also be simultaneously affected in about 60% of patients. There are no consistent exacerbating or relieving factors. Patients often take a range of analgesics but with negligible or minimal effect, other than ibuprofen that may occasionally help to a minor extent. The symptoms are typically episodic initially but become persistent as time progresses. Whilst patients will often complain of episodic pain, it is quite common to reveal that they effectively have constant pain or discomfort with episodic exacerbations. Patients may be convinced that their symptoms are due to sinusitis as they know that their sinuses lie in the area of the pain.

Patients may have undergone treatment over long periods with antibiotics and topical nasal steroids. Some patients have a transient but inconsistent response that may be related to the placebo effect or cognitive dissonance.

Routine of physical activity and the ability to go to sleep are rarely affected. To make matters more complex, the stimulus of a genuine acute sinus infection may exacerbate symptoms, with a return to the background headache on resolution of the infection. It is therefore hardly surprising that both patients and medical practitioners will interpret their symptom complex as being related to their sinuses. Patients often describe tenderness on touching the areas of the forehead or cheeks, reinforcing the concept of underlying inflammation of the bone. Examination will often demonstrate hyperesthesia of the skin and soft tissues over the face and gently touching certain areas is enough to cause discomfort, but there is no evidence of underlying bony disease. This is similar to the tender areas over the forehead and scalp seen with tension-type headache. It appears that this is an organic disorder, in line with an increase in the pain sensitivity in the central nervous system described in tension-type headache.

Interestingly, nasal endoscopy is typically normal. A CT sinus scan may confuse the picture, as approximately 1 in 3 asymptomatic people have incidental findings. However, patients who undergo maximal nasal medical treatment with oral or nasal steroids and a broad-spectrum antibiotic with anaerobic cover fail to improve. The most effective treatment is low-dose amitriptyline as described above. If amitriptyline is not tolerated or fails, then relief may be obtained from gabapentin, pregabalin, propranolol, carbamazepine and, occasionally, sodium valproate. Cognitive behaviour therapy is another option that could be considered. It seems likely that the underlying pathophysiology in mid-facial segment pain is similar to tension-type headache. The aetiology of this type of pain is uncertain. It is of interest that if surgery is performed in the mistaken hope that pain will resolve, the pain may abate temporarily, only to return after several weeks to months.

Atypical Facial Pain

This condition is a diagnosis of exclusion after a careful clinical assessment even when backed up by previous opinions and no evidence of identifiable pathology. The history is often vague and inconsistent. Pain may be widespread, extending from the face to other areas of the head and neck and may move from one part of the face to another between different consultations. Additional odd symptoms, such as ‘mucus moving’ in the sinuses, are often described.

The patient may have a completely fixed idea about their condition and will not be convinced otherwise, whatever the weight of evidence to the contrary. Pain is often described in dramatic terms in conjunction with an excess of other unpleasant life events. Many will have a history of other pain syndromes and their extensive records show minimal progress, despite various medications. Previous sinus or dental surgery is not uncommon, and pain might be attributed to interventions, but the true onset of pain usually precedes any of these procedures, thus and differentiating it from postsurgical neurogenic pain.

Many patients with atypical facial pain exhibit significant psychological disturbance or a history of depression and are unable to function normally as a result of their pain. Some project a pessimistic view of treatment, almost giving the impression they do not wish to be rid of the pain that plays such a central role in their lives.

The management of such patients is challenging, and confrontation is easily induced but counterproductive. A comprehensive examination (including nasendoscopy) is essential to identify significant pathology before the patient is labelled as having atypical pain.

A good management strategy is to reassure the patient that you accept that their pain is genuine and maintain an empathetic persona. Reassure

them that you will continue to treat their condition and maintain a level of hope.

Medication revolves around a gradual introduction of high levels of analgesia and antidepressant with amitriptyline, 75–100 mg at night. Patients should be sympathetically counselled that psychological factors may play a role in their condition and referral to a clinical psychologist may well be helpful.

Giant Cell Arteritis (Temporal Arteritis)

This is a rare cause of facial pain that requires a rapid diagnosis to avoid disease progression affecting the ophthalmic artery leading to visual loss. Patients are typically women, aged over 50 years, who present with fever, malaise and severe temporal or retroauricular pain. On examination, the temporal artery is thickened and exquisitely tender. Investigation reveals an elevated erythrocyte sedimentation rate (ESR). The diagnosis is confirmed by histology of a temporal artery biopsy showing intimal hyperplasia and fragmentation of the internal elastic lamina.

Other causes of facial pain that can mimic temporal arteritis include dental infections and deep neck space infections involving the maxillary space/pterygoid spaces. An urgent CT scan of the sinuses and head neck will exclude these pathologies.

If there is a strong clinical suspicion that temporal arteritis is present, high-dose systemic steroid (60 mg prednisolone daily) should be rapidly commenced, prior to a diagnostic biopsy. An urgent medical referral to a rheumatologist should be arranged.

Dental and Oral Disorders Causing Facial Pain

Dental pathology is an important cause of facial pain that can be mistaken for sinusitis, but the diagnosis can be easily missed. Patients typically present with unilateral facial pain, sometimes radiating to the teeth. Examination may show bite problems. Whilst some patients may have sought a dental opinion, this does not always exclude dental pathology, and vigilance is required.

Some patients will present with unilateral facial pain associated with a purulent, dentally induced sinusitis. This is often accompanied by a foul-smelling unilateral nasal discharge and pus in the middle meatus seen on nasal endoscopy.

Pertinent investigations include an OPG and/or a CT sinus scan that should include all of the paranasal sinuses as well as the lower maxilla and dentition. Cone-beam CT is becoming more popular and is improving with time but may not provide a full assessment of the paranasal sinuses. Key radiological features include periapical lucency with unilateral maxillary sinusitis.

The patient should be referred to a dentist or maxillo-facial colleague should a dental cause be identified.

Temporomandibular (TMJ) Dysfunction

Patients usually present with unilateral dull facial pain across the face which may radiate to the temple, ear and jaw. As it radiates across the face, patients will often assume that they have sinusitis, but a careful history will elicit minimal no associated nasal symptoms. The patient may complain of pain made worse with chewing, locking of the jaw on mouth opening, bruxism or teeth clenching. Examination may show some limitation of jaw opening, tenderness over the masticatory muscles, clicking or tenderness of the TMJ and lateral deviation of the mandible. Most patients will respond to simple analgesics, moist heat and massage of the masticatory muscles and a soft dental appliance. Should the history include jaw locking and lateral deviation of the mandible, an MRI scan of

the temporomandibular joint should be requested and patient referred on to an oro-maxillo-facial colleague.

Sluder's Neuralgia

In 1908, Sluder described 'sphenopalatine neuralgia' as a cause of an ipsilateral, boring and burning facial pain beginning along the lateral side of the nose associated with lacrimation, rhinorrhoea, injected conjunctiva and sometimes involving the cheek [16]. Sluder's definition did not describe a single entity but a diverse symptom complex. The term Sluder's syndrome is often used loosely and it is best avoided as his description differs from most clinical entities.

Contact Point Pain

McAuliffe described stimulating various points within the nasal cavity and paranasal sinuses in five individuals and said that both touch and faradic current caused referred pain to areas of the face [17]. These findings have been used to support theories which state that mucosal contact points within the nasal cavity can cause facial pain. McAuliffe's work has recently been repeated in a controlled study and was found not to produce the referred pain that he described [18]. The prevalence of a contact point has been found to be the same in an asymptomatic population as in a symptomatic population and when they were present in symptomatic patients with unilateral pain, they were present in the contralateral side to the pain in 50% of these patients.

Current Developments

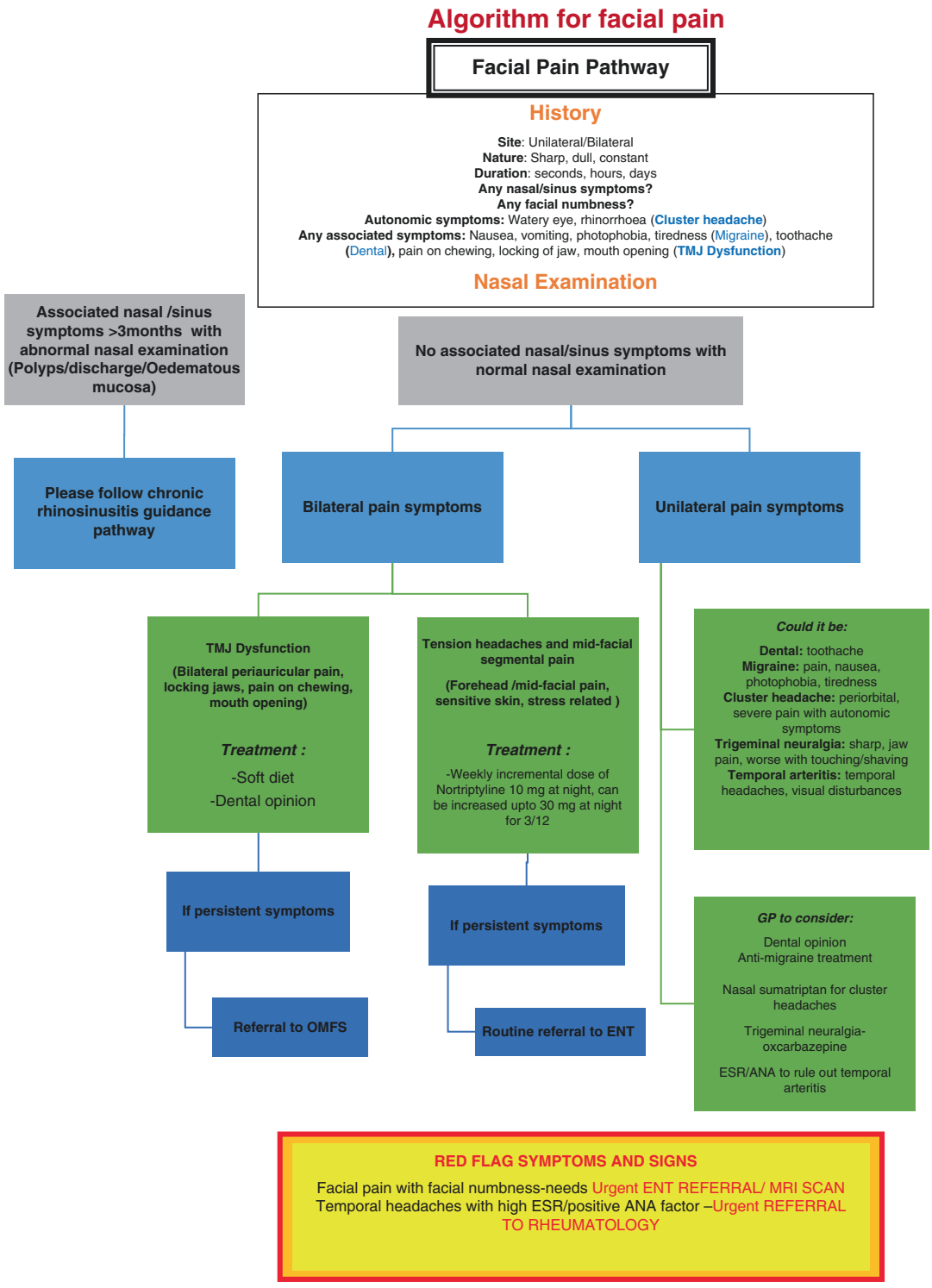
There have been new developments in the management of patients with refractory facial pain:

1. *Sphenopalatine ganglion: block, radiofrequency ablation and neurostimulation*

   The Sphenopalatine ganglion is a promising target for treating cluster headache using blocks, radiofrequency ablation and neurostimulation. Blocking activity in the Sphenopalatine ganglion also has some supporting evidence for use in several other conditions. However, most of the controlled studies were small and without replications. Further controlled studies are warranted to replicate and expand on these previous findings.
2. *Role of botulinum toxin type A in pain management*

   The neurotoxin, botulinum toxin type A, has been used successfully, in some patients, as an analgesic for myofascial pain syndromes, migraine, and other headache types. The toxin inhibits the release of the neurotransmitter, acetylcholine, at the neuromuscular junction thereby inhibiting striated muscle contractions. In the majority of pain syndromes where botulinum toxin type A is effective, inhibiting muscle spasms is an important component of its activity [19].
3. *An update on botulinum toxin A injections of trigger points for myofascial pain*

   Myofascial pain syndrome (MPS) is a common chronic pain condition that is characterised by distinct 'trigger points'. Despite current treatments with physical therapy, analgesics, anti-depressants and trigger-point injections, myofascial pain remains a challenging chronic pain condition in clinical practice. Botulinum toxin A (BTX-A) can cause prolonged muscle relaxation through inhibition of acetylcholine release. It may offer some advantages over the current treatments for MPS by providing a longer sustained period of pain relief [20].


Algorithm for facial pain
Facial Pain Pathway
History
Site: Unilateral/Bilateral
Nature: Sharp, dull, constant
Duration: seconds, hours, days
Any nasal/sinus symptoms?
Any facial numbness?
Autonomic symptoms: Watery eye, rhinorrhoea (Cluster headache)
Any associated symptoms: Nausea, vomiting, photophobia, tiredness (Migraine), toothache (Dental), pain on chewing, locking of jaw, mouth opening (TMJ Dysfunction)
Nasal Examination
Associated nasal /sinus symptoms >3months with abnormal nasal examination (Polyps/discharge/Oedematous mucosa)
No associated nasal/sinus symptoms with normal nasal examination
Please follow chronic rhinosinusitis guidance pathway
Bilateral pain symptoms
Unilateral pain symptoms
TMJ Dysfunction
(Bilateral periauricular pain, locking jaws, pain on chewing, mouth opening)
Treatment :
-Soft diet
-Dental opinion
Tension headaches and mid-facial segmental pain
(Forehead /mid-facial pain, sensitive skin, stress related)
Treatment :
-Weekly incremental dose of Nortriptyline 10 mg at night, can be increased upto 30 mg at night for 3/12
Could it be:
Dental: toothache
Migraine: pain, nausea, photophobia, tiredness
Cluster headache: periorbital, severe pain with autonomic symptoms
Trigeminal neuralgia: sharp, jaw pain, worse with touching/shaving
Temporal arteritis: temporal headaches, visual disturbances
If persistent symptoms
If persistent symptoms
GP to consider:
Dental opinion
Anti-migraine treatment
Nasal sumatriptan for cluster headaches
Trigeminal neuralgia- oxcarbazepine
ESR/ANA to rule out temporal arteritis
Referral to OMFS
Routine referral to ENT
RED FLAG SYMPTOMS AND SIGNS
Facial pain with facial numbness-needs Urgent ENT REFERRAL/ MRI SCAN
Temporal headaches with high ESR/positive ANA factor –Urgent REFERRAL TO RHEUMATOLOGY

Key Learning Points

- A careful structured history of facial pain, examination, supplemented in some cases by diagnostic tests, will permit a definitive diagnosis in most patients.
- Imaging should be kept to the minimum and only done for appropriate cases. Very rarely a CT scan may be indicated to reassure an extremely anxious patient and may have a positive psychological impact on him.
- Laboratory studies are indicated only when a systemic cause is suspected, such as Giant cell arteritis.
- Neuroimaging is indicated regarding the diagnosis of primary headache when the clinical features suggest a secondary cause.
- Should a precise diagnosis not be possible, a multidisciplinary approach should be considered.

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