Trigeminal Neuralgia – a debilitating facial pain

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SUMMARY POINTS

- Trigeminal neuralgia (TN) is characterised by sudden usually unilateral severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve.
- Diagnosis is largely based on clinical history due to the current lack of objective investigations.
- MRI can identify those patients who have TN secondary to an underlying pathology such as multiple sclerosis.
- The first line medical management remains carbamazepine, with oxcarbazepine being the second choice medication.
- Both percutaneous techniques targeting the Gasserian ganglion and microvascular decompression can be considered effective in the management of TN. Microvascular decompression is considered to provide on average, the longest pain free period post surgery.
- There are a number of TN associations and support groups which provide a valued service to patients and clinicians.
- Due to a dearth of high quality studies in many aspects of the condition, TN requires further research to be conducted.

Introduction

Trigeminal neuralgia is a severe, incapacitating facial pain which is diagnosed and managed by a wide range of Healthcare Professionals in both primary and secondary care settings. The aims of this article are to explain the nature of trigeminal neuralgia and outline the current approaches to the diagnosis and management of this condition.

Definition and classification

Trigeminal neuralgia (TN) is defined by The International Association for The Study of Pain (IASP), as "sudden usually unilateral severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve"[1]. The International Headache Society (IHS) divides Trigeminal Neuralgia into two distinct categories: "classical" and "symptomatic" (secondary) TN[2]. Classical TN includes those patients in which no identifiable cause can be found for their TN other than a vascular compression of the trigeminal nerve. Symptomatic TN describes those patients in which an identifiable cause can be found, other than a vascular compression, such as a tumour, arteriovenous malformation or multiple sclerosis (MS). Not every patient with TN will fulfil the IHS diagnostic criteria, therefore the diagnoses of "atypical" or "type II" Trigeminal Neuralgia can be applied to these cases[3].

Epidemiology

Previously, the only robust epidemiological data on TN was from the U.S. and demonstrated an annual incidence of between 4-5 per 100,000[4]. However, recent studies from both the UK[5] and Netherlands[6] show far higher incidence rates of 26.8 and 28.9 per 100,000 respectively. The UK study[5] demonstrates a higher incidence in females of all age groups, and a peak incidence between 45 and 59 years. This age of onset appears lower than previous studies which showed the peak incidence in the over 70's[4]. The apparent rise in the incidence of TN is likely to be as a result of misdiagnosis and reporting error, rather than due to any other reasons. A study by Koopman et al[7] highlighted that misdiagnosis amongst General Medical Practitioners could be as high as 48%. Hence, many of these cases could have potentially been misdiagnosed toothaches, temporomandibular disorders or trigeminal autonomic cephalagias[8-10]. Data taken from England's NHS Health Episode Statistics database (HES)[11], suggests that for the year 2005/06, a total of 1,939 hospital admissions were as a result of TN, with 7,010 bed-

days utilised by TN patients. The mean age of TN patient admitted during this period was 64 years.

Diagnosis

Diagnostic criteria
The IHS diagnostic criteria for the diagnosis of classical TN are as follows[12]:

A. Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C

B. Pain has at least one of the following characteristics:

   1. intense, sharp, superficial or stabbing

   2. precipitated from trigger areas or by trigger factors

C. Attacks are stereotyped in the individual patient

D. There is no clinically evident neurological deficit

E. Not attributed to another disorder

The diagnostic criteria of symptomatic (secondary) TN only really vary in so far as it is attributable to an underlying cause other than a vascular compression. The current diagnostic criteria have not been validated by scientific studies using evidence based methodologies, largely due to the fact there are no objective tests available[10].

It therefore follows that the history provided by the patient is crucial in order to arrive at an accurate diagnosis of TN. Frequently, the descriptions provided by patients can be considered almost pathognomonic for TN. Below are some descriptions taken from patient narratives[13]:

- "electric shock"
- "electrical explosion"
- "shooting jolts of electricity directly into the raw nerves"
- "hot poker in my cheek"
- "a fast, jarring jolt of lightening pain"

Often the onset of TN pain is memorable and patients can often describe clearly their first attack. Pain is provoked by light touch, such as washing the face, shaving, cold wind, vibration or brushing the teeth. There is often a trigger point which invariably the patient is aware of and tries to avoid. Patients often find it difficult to appreciate that there are short breaks between the paroxysms of pain. There is often a reported "refractory period" where despite further light touch stimulus, the pain cannot be evoked. The attacks are rapid in both onset and decline, lasting at the most a couple of minutes. There can be some "after pain", a burning, dull, mild ache which gradually wears off. The painful episodes can vary in severity from the mild "twinge" up to severe and incapacitating. Often, pain can become so severe that it can impact on the patient's ability to eat, wash and maintain oral hygiene.

The presence of autonomic symptoms such as conjunctival tearing or facial flushing, are important distinguishing factors between TN and the rarer trigeminal autonomic cephalalgias. Pain localised to the first division of the trigeminal nerve needs to be more carefully assessed, as it is more likely to be a trigeminal autonomic cephalalgia than TN. Patients diagnosed as "atypical" or "type II" TN tend to present with a background, dull aching or burning type pain which can persist for many hours[14,15]. When pain is located primarily intra-orally it is often difficult to differentiate between TN and a dental cause for the pain, such as acute pulpitis. Therefore, in these instances, input from a dentally qualified practitioner can be helpful.

Investigations
Recently promulgated guidelines published by the American Academy of Neurology (AAN) and The European Federation of Neurological Societies (EFNS) have failed to find sufficient evidence to support or refute the fact that the cause of trigeminal neuralgia is related to the presence of a neurovascular compression[18,19] (Figure 1). However, neuroimaging will identify causative lesions such as MS plaques or tumours in those patients with symptomatic TN. The guidelines suggest that in TN patients (without non-trigeminal symptoms) who have routine neuroimaging, up to 15% will have causative lesions other than vascular compressions.

The guidelines also suggest that neurophysiological tests may be helpful in distinguishing classical TN from symptomatic TN. The following findings may indicate a diagnosis of symptomatic TN:

- abnormal trigeminal reflexes (specificity 94%, sensitivity 87%)
- abnormal trigeminal nerve evoked potentials
- trigeminal sensory deficits and or bilateral involvement

Pathogenesis

It is now widely accepted that TN is a neuropathic type pain. The most recognised hypothesis explaining the pathophysiology of TN is the "ignition hypothesis" as described by Devor et al[16]. The hypothesis suggests that TN is precipitated by injury to the trigeminal axons in the nerve root or ganglion. The injury in most cases is related to compression of the nerve in the root entry zone by vascular structures. Imaging has shown there to be evidence of demyelination and remyelination of the nerve in this area[17]. Therefore, these damaged neurones become hyperexcitable and exhibit the "after discharge" burst phenomenon. These bursts can be triggered by external trigger stimuli and perpetuate beyond

the duration of the stimulus. The after discharges then recruit adjacent neurones, with the help of "ephaptic cross-talk" (electrical cross-over between demyelinated neurones) which leads to the characteristic "electrical explosion" of pain. Devor et al[16] suggest the stop mechanism, or refractory period, is a consequence of post-burst potassium influx hyperpolarisation, which renders the neurone refractory to further stimuli.

Natural history

There is very little published cohort data relating to the natural history of TN. The single study that looks specifically at the progression of the disease suggests that episodes of TN pain are likely to become more frequent over time[20]. Unfortunately, the patterns of relapse and remission remain unknown and as a result no predictions can be offered about disease progression at this time.

Impact on quality of life

Trigeminal neuralgia is a severe, debilitating pain condition. Despite being eminently manageable and in many cases a curable condition, it still promotes disruption and impaired quality of life in its sufferers. There have been surprisingly few studies relating to quality of life outcomes in TN. A couple of long term cohort studies have demonstrated that many TN patients suffer depression, which can be alleviated following curative surgical procedures[21,22].

Below is a patient narrative which clearly demonstrates the severity and impact of TN pain. Moreover, it clearly highlights the commonly found anxiety within TN patients relating to their fear of the return of pain, or their worry that the drugs or procedures will cease to be efficacious over time:

> *"Even as the spasm eases, you know that the relief is only because your nerves have been overcome by the pain. It will come back tomorrow if it doesn't come again today. You know that the demon is never going to go away. It does not care if you are a nice human being. It does not care if you pray hourly. It's only mission in your life is your pain. And it is DEDICATED!"*[13]

Management of Classical Trigeminal Neuralgia

Pharmacological

Local anaesthesia

There have been a few publications relating to the use of local anaesthesia in the management of TN, demonstrating variable degrees of efficacy and practicality[23-26]. It is generally accepted that TN patients with a specific trigger area, particularly those intra-orally, can gain benefit, albeit short term relief from local anaesthesia blocks such as those provided by dentists. It is advisable to first place a short acting local anaesthetic with a vasoconstrictor, such as lidocaine with adrenaline, and then add a long acting agent, such as bupivicaine. Often these blocks will provide several hours of complete analgesia, which is particularly useful in acute exacerbations of TN while awaiting systemic drugs to become therapeutic. Lemos et al have shown that regular injections of ropivicaine into trigger areas in conjunction with oral gabapentin were more effective than taking gabapentin alone[27].

Pharmacological management

Despite local anaesthesia being a useful treatment in certain cases, systemic drug treatment remains the most commonly used first line treatment modality. There have been several Cochrane collaborations, systematic literature reviews and published guidelines on the medical management of TN[18,19,28-32]. However, these studies are derived from a surprising lack of quality scientific research in this area, with a paucity of high quality randomised controlled trials (RCTs). Table 1 describes the main medicines used in the management of TN. The table was supplemented by data from the AAN/EFNS guidelines[18,19] and the BNF[33]. The evidence scoring was taken from the AAN definitions for classification of evidence[34].

Oral medicine therapy

Carbamazepine is a well established and effective drug in managing TN and currently remains the medicine of choice[18,19]. It is highly effective and likely to provide complete pain relief with a few days. Although efficacious, carbamazepine is compromised by it's poor tolerability and "numbers needed to harm" (NNH) of 3.4 for minor and 24 for serious adverse events[19]. There are reported risks of severe mucocutaneous reactions such as Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis (SJS/TEN) in patients taking carbamazepine. This risk is higher in some oriental and Asian populations than in other ethnic groups, with the presence of HLA allele B*1502 believed to be the significant factor involved. As a result, some advocate genotyping at risk patients before prescribing carbamazepine[35]. Currently, the second line medicine of choice is oxcarbazepine[18,19], a prodrug structural derivative of carbamazepine. Unlike carbamazepine, oxacarbazepine doesn't utilise the liver cytochrome system, hence medicine interactions and unwanted side effects are less likely. Oxcarbazepine used for TN is an off license use in the UK, but is recommended as second line in the BNF[33]. Lamotrigine and baclofen are other second line drugs that could be considered in TN[18,19]. Pregabalin has shown promise in a prospective open labelled study[36]. Medical treatment may fail for a variety of reasons, such as reduced drug efficacy or poor tolerability. It is often sensible to reconsider the accuracy of the diagnosis should pharmacotherapy fail to provide adequate pain relief. There are a couple of long term medicine studies that suggest pharmacotherapy fails over time independent of the drug plasma concentrations[37,38].

Parenteral therapy

The acute management of TN using parenterally administered medicines is an area that currently lacks adequate evidence in order to provide any recommendations. There are a few case reports on the use of intravenous fosphenytoin to manage TN in the acute setting[18,19].

Table 1 Commonly used medicines in TN

Medicine	Daily dose range	Important side effects	Recommendations on use	Comments
Carbamazepine (CBZ) (Evidence rating A – Effective / Should be used)	200 -1600mg	Contraindicated with unpaced AV conduction abnormalities. Neurological side effects (dose related). Hyponatraemia / blood disorders - monitor bloods at regular intervals. SJS/TEN rarely	Begin with small doses, depending on tolerability. Increase and decrease slowly.	Adverse drug interactions.e.g warfarin. HLA-B*1502 allele in individuals of Han Chinese or Thai origin – increased risk of SJS/TEN.
Oxcarbazepine (Evidence rating B – Probably effective / Should be considered)	300-1200mg	Neurological side effects, hyponatraemia with higher doses. Very rarely blood disorders, SJS/TEN	Use on a four times a day basis	Generally better tolerated than CBZ.
Baclofen (Evidence rating C – Possibly effective / may be considered)	50-80mg	Neurological side effects	Begin very slowly, divided doses.	Withdraw drug slowly to avoid side effects. Useful in patients with MS
Lamotrigine (Evidence rating C – Possibly effective / may be considered)	200-400mg	Neurological side effects, blood disorders, rarely SJS/TEN.	Initially very slow escalation. Can use in combination with CBZ	Cutaneous reactions common if increase dose too quickly
Gabapentin with ropivacaine[27] (Evidence rating C – Possibly effective / may be considered)	1800-3600mg (RCT utilised up to 900mg) + 2ml of mg/ml Ropivicaine	Neurological side effects	Ropivacaine injected weekly into trigger spots	Use of ropivacaine reduced dose of gabapentin required. (Small RCT with newly diagnosed patients likely to go into remission)
Medicines not evaluated in Randomised Controlled Trials				
Phenytoin	200-300mg	Neurological side effects, blood disorders, rarely SJS/TEN.	Can use with CBZ. HLA-B*1502 cross reactivity with CBZ.	>300mg can lead to severe side effects
Sodium valproate	600-1200mg	Neurological side effects, blood disorders, rarely hepatic dysfunction.	Monitor liver function for first 6/12.	Often used by Neurologists
Pregabalin	150-600mg	Neurological side effects - dose dependent. Peripheral oedema with higher doses.	Use twice daily, avoid abrupt withdrawal.	Long term cohort study shows promise

CBZ – carbamazepine

HLA-B*1502 – Human Leukocyte Antigen- B*1502

AV – Atrioventricular

SJS/TEN – Stevens Johnson Syndrome / Toxic Epidermal Necrolysis

Surgical management

At what point in the TN disease process should a surgical option be considered? This question was addressed by the AAN/EFNS guideline group who found that there was currently insufficient evidence to determine at which point surgery should be offered. 18,19 A recent study investigating how patients make treatment decisions, suggested that given a hypothetical scenario, patients would opt for surgical intervention ahead of medical treatment[39]. Neurosurgeon should be involved early in the patient's care pathway. This allows patients to broach the subject of surgery earlier and ideally while their symptoms are well controlled. This strengthens the patient's ability to make fully informed decisions about future treatment options.

There are a variety of surgical options available for the management of TN. They can be classified according to the principal target site:

- peripheral i.e. distal to the Gasserian ganglion
- Gasserian ganglion level
- posterior fossa root entry zone

With the exception of microvascular decompression, all of the surgical techniques can be described as destructive or ablative procedures as they are concerned with reduction in sensory input, hence involve varying degrees of nerve damage. Microvascular decompression (MVD) is the only procedure at present which aims to maintain the integrity of the trigeminal nerve following surgery.

The various surgical techniques are summarised in Table 2. The data is based on the AAN/EFNS guidelines[18,19] with the evidence scoring taken from the AAN definitions for classification of evidence[34].

Peripheral techniques

Numerous peripheral surgical techniques have been studied, including: neurectomies, acupuncture, cryotherapy, peripheral radiofrequency thermocoagulation (RFT) and various injections such as phenol and alcohol. All of the studies were case series, which didn't employ independent outcome observers. Consequently, there is insufficient evidence to advocate the use of peripheral techniques[18,19].

Gasserian ganglion percutaneous interventions

These techniques are performed under a short general anaesthetic or deep sedation and usually can be provided on a day-case inpatient basis. A cannula is inserted into the trigeminal ganglion via the foramen ovale. At this point during RFT, many operators will reduce the level of the patient's sedation and confirm correct placement of the cannula by stimulating the nerve in order to evoke the TN like pain. The nerve is lesioned using heat (RFT), glycerol injection or mechanical balloon compression. Pulsed radiofrequency

Table 2 Surgical interventions for TN

Procedure	Pain relief duration (Kaplan-Meier estimate)	Mortality	Morbidity	Comments
Peripheral i.e. cryotherapy, neurectomy, laser ablation, acupuncture, thermocoagulation, injections of alcohol / phenol (Evidence rating U – data inadequate / treatment unproven)	50% at 12 months	Nil	Localised sensory loss, haematoma formation, infection	Can be performed under local anaesthetic. Suitable for medically unfit for GA
Gasserian ganglion i.e. radiofrequency thermocoagulation, glycerol rhizolysis, balloon compression (Evidence rating C – Possibly effective / may be considered)	50% at 5 years	Very low	Sensory loss >50% dysaesthesia <6% anaesthesia dolorosa 4% eye complications 4% meningitis 0.2%, up to 50% have masticatory deficit following balloon compression	Can be performed under heavy sedation or short GA. Often suitable alternative for patients unfit for MVD. Glycerol rhizolysis provides shortest pain relief duration
Gamma knife (Evidence rating C – Possibly effective / may be considered)	52% at 3 years	Nil	Problematic sensory loss 6-13% often six months later Anaesthesia dolorosa practically absent	The only non-invasive technique Pain relief can be delayed up to 6 months
Microvascular decompression (Evidence rating C – Possibly effective / may be considered)	73% at 5 years	0.2 - 0.5%	Major post-operative morbidity 4% Ipsilateral hearing loss up to 10% Diplopia transiently Sensory loss 7%	Highest improvement in quality of life

thermocoagulation is a procedure whereby the RFT is applied in pulses rather than a continuous current. The presumed benefit of pulsed over regular RFT is a reduction in post operative sensory loss. However, current evidence suggests the pain relief outcome following pulsed RFT is inferior when compared to traditional RFT. 40 There is only limited evidence to support the use of these percutaneous techniques. The chief outcome measures are relating to pain relief, with no RCTs and only scant evidence from prospective case series. Around 90% of patients will be pain free immediately following the procedure, but this drops to around 50% after 5 years[18,19]. Although mortality is very low, unwelcome side effects can occur, such as sensory loss (almost 50% of cases), dysaesthesias (less than 6% of cases) and anaesthesia dolorosa (around 4% of cases).

Gamma knife® surgery

This procedure utilises a cobalt-60 gamma emission source focused at the trigeminal root entry zone. This stereotactic radiosurgical procedure is ablative and the only non-invasive technique employed to treat TN. Although no complications have been reported outside the trigeminal nerve, problematic sensory deficits have been reported in 6 – 13% of cases, albeit anaesthesia dolorosa is almost absent[18,19]. Pain relief can often be delayed for a mean duration of 1 month[41]. Complete pain relief without medication is present in up to 69% of patients at 1 year, but this falls to 52% after 3 years[18,19]. One study which utilised a non-validated questionnaire, suggested this procedure is associated with a marked improvement in quality of life, with 88% of patients being satisfied with their outcomes[42].

Microvascular decompression

This is the most invasive procedure used in the treatment of TN. It requires a craniotomy to be performed in the post auricular region, thus exposing the trigeminal root entry zone in the posterior fossa. Any vessels impinging on the trigeminal nerve are identified before being moved out of contact with the nerve and secured using teflon tape or a vascular sling. Once again, there are no RCTs available to evaluate this procedure and only a small number of studies with independent outcome measures[18,19]. Due to the invasive nature of the procedure it follows that there will be a mortality rate associated, the average being 0.2% rising to 0.5% in some studies[18,19]. The majority of operative complications occur early in the post operative period and serious problems occur in around 4% of patients. Complications include issues such as: haematoma, infarcts, CSF leak, transient diplopia and rarely facial muscle weakness. The most common complication is that of aseptic meningitis, which occurs in roughly 11% of patients[18,19]. Sensory loss occurs in around 7% of patients[43]. The most significant long term complication is that of ipsilateral hearing loss which occurs in as many as 10% of patients[18,19]. Evidence suggests that neurosurgical units which conduct high numbers of these procedures suffer less with perioperative mortality and morbidity[44]. MVD will continue to provide pain relief for 73% of patients after 5 years[18,19].

Comparison of different surgical techniques

When considering the previous evidence, it is apparent that the percutaneous procedures at the Gasserian ganglion, Gamma knife® surgery and microvascular decompression are all relatively effective in the management of TN. However, when considering the AAN/EFNS guidelines, the only direct comparison one could draw between the different techniques is that MVD provides the longest duration of pain free period following treatment[18,19]. There is an apparent paucity of high quality, directly comparative studies between the different surgical techniques.

Management of Symptomatic (secondary) Trigeminal Neuralgia

There is currently insufficient evidence to support or refute the effectiveness of drugs in symptomatic (secondary) TN. There are no high quality trials investigating which drugs should be employed in MS patients suffering from TN[18,19]. This patient group remains difficult to manage with pharmacotherapy, as often the side effects of the drugs compound the symptoms of the MS.

There is insufficient evidence to give clear recommendations on surgical interventions in TN patients who suffer from MS[18,19]. However, the AAN/EFNS did suggest that due to the uncertainty of surgical outcome in this cohort, surgical procedures should only be considered if there is compelling evidence of drug resistance[18,19].

Psychological management approaches

As mentioned previously, there is a surprising lack of evidence relating to the impact of TN on quality of life, in particular on the psychological wellbeing of sufferers. Despite this, it is recognised that psychological and psychiatric comorbidity is found within this

Table 3 TN Support groups

Support groups		
Trigeminal Neuralgia Association UK PO Box 234, Oxted, Surrey, RH8 8BE, England Phone: 01883 370 214 Website: www.tna.org.uk	**The Facial Pain Association** (Formerly the Trigeminal Neuralgia Association) Suite C, 925 NW 56th Terrace, Gainesville, Florida, 32605-6402 USA. Website: www.endthepain.org (This association also now caters for sufferers of trigeminal neuropathic pain.)	**Trigeminal Neuralgia Association Australia** PO Box 1611 Castle Hill, NSW, 1765, Australia Website: www.tnaaustralia.org.au

cohort of patients[45]. Therefore, it should be considered necessary to manage TN within a multi-disciplinary team which includes specialists such as psychologists and liaison psychiatrists[46].

Patient support groups and associations

Historically, TN has been managed using a biomedical approach, with very little credence and thought being given to the psychological and social impact that this condition has on its sufferers. However, as in most chronic medical conditions there are increasing numbers of TN associations and support groups now available (Table 3). These groups help to engender confidence in patients, encourage them to take a leading role in their care, challenge the experts and share experiences with fellow sufferers[47]. A review of events organised by TN associations suggest that patients not only attend in order to glean more knowledge, but also to liaise with experts within the field of TN; with satisfaction from the conferences is reported as high[48].

Research questions

As identified by this review, there is a dearth of high quality evidence with regards to many aspects of TN. Some of the key areas for future research are as follows:

- Population / cohort based studies of TN in order to clarify the epidemiology, natural history and identify associated risk factors
- Studies to determine what objective investigations can confirm the clinical diagnosis, possibly including functional MRI
- RCTs in order to compare surgery / medical treatments including quality of life outcome measures, and timing of surgical referral
- RCTs in order to compare different surgical techniques including quality of life outcome measures
- RCTs of newer medicines, other than carbamazepine

Conclusions

Although relatively rare, trigeminal neuralgia is a debilitating and complex facial pain condition which requires a patient centred, multi-disciplinary approach to its management. Currently, the guidelines and associated evidence are insufficient to be able to recommend definitive management, and further high quality research is required.

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Orofacial Pain Multiple Choice Questions

Dental (odontogenic) pain

More than one answer may be correct. Select all that apply.

1. Risk factors for acute necrotizing ulcerative gingivitis include:

a) Smoking
b) Poor oral hygiene
c) Immunosuppression
d) Stress
e) All of the above

2. The most appropriate analgesic for pericoronitis providing it is not contraindicated is:

a) paracetamol
b) co-codamol
c) codeine phosphate
d) ibuprofen
e) diclofenac

3. The osmolality theory of dental pain elicits an action potential in which pulpal fibres?

a) A delta fibres
b) A beta fibres
c) A gamma fibres
d) C fibres
e) D fibres

4. Which of the following conditions is more commonly known as a 'dry socket'?

a) acute pulpitis
b) periapical periodontitis
c) alveolar osteitis
d) pericoronitis
e) acute necrotizing ulcerative gingivitis

5. Select from the following list materials suitable as temporary restorative materials for carious teeth:

a) amalgam
b) composite
c) glass ionomer cement
d) zinc oxide eugenol
e) compomer

6. Pericoronitis can be managed with:

a) antibiotics
b) alvogel
c) analgesia
d) tooth extraction
e) root canal treatment