CLINICAL ORAL SCIENCE AND DENTISTRY

REVIEW ARTICLE ISSN 2688-7428 Open Access

Diagnosis of Dentine Hypersensitivity: Is it time to redefine our Terminology? A Personal View

David Gillam[1]*

[1] *Institute of Dentistry, Barts and the London School of Medicine and Dentistry, Queen Mary University, London, UK

Received date: August 17, 2024, **Accepted date:** August 24, 2024, **Published date:** August 29, 2024.

Copyright: ©2024 David Gillam. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

***Corresponding Author:** Dr. David Gillam, Institute of Dentistry, Barts and the London School of Medicine and Dentistry, Queen Mary University, London, UK.

Abstract

Ideally classification systems should facilitate both accurate and reproducible definitions to avoid any misdiagnosis and/or subsequent inappropriate treatment. Historically the clinical diagnosis of Dentine Hypersensitivity has been a diagnosis of exclusion based on the Martin Addy's 1985 definition of DH as "pain derived from exposed dentine in response to chemical, thermal, tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or pathology" (Addy et al. 1985, Dowell et al. 1985, Addy 2000). This definition was subsequently modified by the Canadian Board on DH (Canadian Consensus Document 2003) which suggested that 'disease' is a more appropriate term than 'pathology'. Essentially this definition enables the clinician to have a clear picture of diagnosing pain symptoms from other dental conditions. The key words being "which cannot be explained as arising from any other dental defect or disease" which should ideally eliminate pain arises, for example from dental caries, post operative sensitivity (including bleaching sensitivity, cracked tooth syndrome), molar incisor hypomineralisation or other types of facial pain. The aim of this review, therefore, is assess whether clinicians are consistent in their diagnosis of orofacial pain (including dental pain).

Introduction

Dentine Hypersensitivity (DH) is a relatively common, yet troublesome clinical condition that may have an impact on the quality of life (QoL) of individuals who suffer from it (Gibson et al., 2010, Cuhna-Cruz & Wataha 2014, Gillam 2021a). The pain associated with the condition has been described as rapid on onset, sharp in character and transient in its duration (Addy 2000). Several surveys have indicated that clinicians struggle to identify patients with DH which may in turn lead to the underestimation of the true prevalence of the condition. Furthermore, there is some evidence that would suggest that clinicians lack of confidence in both the diagnosis and management of the condition. It should recognize that the clinical diagnosis of orofacial pain in general can be both time consuming and difficult for several reasons: 1) the difficulty in identifying areas of the mouth that may be causing the problem and 2) the highly subjective nature of pain and its variability between patients. It is therefore important for clinicians to correctly identify patients by excluding any confounding factors from other orofacial pain conditions prior to the successful management of DH or in the event of excluding DH, identify the correct diagnosis of the patient's pain (Gillam 2013). According to Gillam (2013), the importance of the definition suggested by Addy and co-workers. (1985) and Addy (2000) and the Canadian Consensus Document (2003) is that these documents provide a very useful clinical description of the condition and as such encourages the clinician to exclude other forms of tooth pain or sensitivity with similar pain features. If we accept this definition, therefore we would (by definition) exclude other clinical conditions such as dental caries, post operative sensitivity (including bleaching sensitivity, cracked tooth syndrome), molar incisor hypomineralisation or other types of facial pain.

According to Gillam (2013, 2021b) there is clear evidence that clinicians are not routinely examining their patients for DH or even considering eliminating other possible causes of dental pain that elicit similar clinical symptoms as DH (differential diagnosis) prior to any subsequent management. It is also highly likely that confusion may also be present when diagnosing other orofacial pain conditions It should be recognized, however, that the process of making a definitive diagnosis is both a complex and time consuming one and it is important for the clinician to recognise that in comparison with other dental conditions (e.g. irreversible pulpitis, dental abscess, sinusitis, periodontal disease, pericoronitis, idiopathic oral facial pain) DH is relatively less common and as such it is essential to exclude all other oral conditions with a similar presentation to that of DH prior to any further management of the patient's problem (Table 1). Patients may also experience pain from recent dental procedures (post-operative sensitivity) such as sensitivity from restorative materials, tooth whitening and bleaching (Markowitz 2010) as well as from periodontal disease and associated procedures such as non-surgical and surgical procedures (Troil et al., 2002, Sanz & Addy 2002, Gillam and Orchardson 2006, Lin & Gillam 2012). In such cases the clinician should be able to quickly identify the initiating cause by referring to the patient's notes and by asking the patient for any supplementary information. For this review selected dental conditions with their symptomology will be evaluated and compared to DH using the terminology used in the published literature to define the condition.

For most dental conditions that present in the dental office, supplementary investigations may help the clinician in correctly diagnosing the problem. For example, in the evaluation of pulp vitality [sensibility] using a pulp tester, ice stick, endo-frost, percussion, diagnostic radiographs etc.). The use of optical illumination (Fibre-optic transillumination [FOTI] with or without magnification and transillumination using a dental mirror may help in the diagnosis of dental caries and teeth identified with Cracked Tooth Syndrome [CTS]). Furthermore, the use of a Fractfinder (Denbur, Oak Brook, IL, USA) or a Tooth Slooth II (Professional Results Inc., Laguna Niguel, CA, USA), may also be helpful in diagnose CTS (a disposable plastic tip from a triple air syringe may also be suitable) (see Gillam & Koyi 2019). A diagnostic feature of CTS is the presence or absence of the 'rebound effect.' The diagnosis of conditions such as post-operative sensitivity (including both restorative (recent restorations) and periodontal

causes (non-surgical and surgical techniques) or bleaching sensitivity may be successfully diagnosed based on the patient records and associate history of the complaint which generally occurs within the first week or two of the procedure. For non-odontogenic conditions such as chronic idiopathic facial pain/persistent idiopathic pain (PIFP) (atypical odontalgia and atypical facial pain) may be very difficult to diagnose, and it is recommended that these patients should be referred to a Specialist Hospital (Oral Medicine clinics) for the appropriate management of the problem.

Aetiology	**Pain character and timing**	**Pain Intensity**	**Proving Factors**	**Relieving Factors**	**Associated features and Comments**
Dentine Hypersensitivity	Sharp, stabbing, stimulation evoked	Mild to moderate	Thermal, tactile, chemical, osmotic	Removal of the stimulus	Attrition, erosion, abrasion, abfraction Need to exclude dental conditions with similar pain symptoms as well as other non-odontogenic causes of pain (history and clinical/radiographic examination (where appropriate)
Atypical odontalgia (persistent dentoalveolar pain/persistent idiopathic pain)	Continuous, nonparoxysmal, dull, aching and throbbing but occasionally sharp	Mild to moderate	Touch, heat and cold	Sleep and rest Topical agents: lidocaine, capsaicin. Systemic agents: antidepressants	May have no obvious clinical features Need to exclude odontogenic (dental) pain Conduct a normal clinical neurological examination (e.g., oral medicine clinic)
Reversible pulpitis	Sharp, stimulation evoked	Mild to moderate	Hot, cold, sweet	Removal of the stimulus	Caries, restorations
Irreversible pulpitis	Sharp, throbbing, intermittent/continuous	Severe	Hot, chewing, lying flat	Cold in the late stages	Deep caries
Cracked tooth syndrome	Sharp intermittent	Moderate to severe	Biting, ‘rebound pain’		Trauma, parafunction
Periapical periodontitis	Deep, continuous boring	Moderate to severe	Biting	Removal of trauma	Periapical redness, swelling, mobility
Lateral periodontal abscess	Deep continuous aching	Moderate to severe	Biting		Deep pockets redness and swelling

Pericoronitis	Continuous	Moderate to severe	Biting	Removal of trauma	Fever, malaise, imprint of upper tooth
Dry socket (acute alveolar osteitis	Continuous 4-5 days post extraction	Moderate to severe		Irrigation	Loss of clot, exposed bone

Table 1: Differential diagnosis of selected dental pain (modified Acknowledgement Aghabeigi [2002] and Gillam 2010)

Diagnosis and differential diagnosis

According to Gillam (2013) two processes may be relevant when examining a patient complaining of dental pain namely 1) how to identify the problem (diagnosis and differential diagnosis) (Table 1) and 2) how to assess the problem (measurement) (Table 2). Diagnosis may therefore be considered as i) the art or act of identifying a disease from its signs and symptoms, ii) the decision reached by diagnosis and iii) a diagnosis made by eliminating other possible causes of disease symptoms (from the Latin diagnosis as derived from the Greek diagignōskein (Moseby 2009). It is evident, therefore that other dental conditions with similar signs and symptoms from DH may complicate a correct diagnosis of a condition and subsequent treatment. In other words, the clinician when making a diagnosis would seek to exclude or eliminate any other conflicting conditions (Table 1) (Gillam 2013). This would (to my mind) satisfy the historical definition of DH as a diagnosis of exclusion. However more recently there have been numerous papers that have associated the term 'Dentine Hypersensitivity (sensitivity or hypersensitivity)' with other dental conditions such as pain from molar incisor hypomineralisation (Inchingolo et al. 2023).

Assessment methodology used to identify dental conditions with similar pain characteristics to DH. It should be noted, however that some of the methods in Table 2 are more suited to the research environment than in a dental practice. For example, hydrostatic pressure evaluation has been reported in the literature, but may be considered impractical for use in clinical studies. It is mor likely that a clinician would use cold air from a three in one dental syringe and an explorer probe (instruments common in all dental practices) to identify DH.

Mechanical (tactile) stimuli
Explorer probe
Constant pressure probe (Yeaple)
Mechanical pressure stimulators
Scaling procedures
Single-tufted brush
Chemical (osmotic) stimuli
Hypertonic solutions, for example, sodium chloride, glucose, sucrose and calcium chloride
Electrical stimulation
Electrical pulp testers

Dental pulp stethoscope
Evaporative stimuli
Cold air blast
Yeh air thermal system
Air jet stimulator
Temptronic device (microprocessor temperature-controlled air delivery system)
Thermal stimuli
Electronic threshold measurement device
Cold water testing
Heat
Thermo-electric devices (e.g. Biomat Thermal Probe)
Ethyl chloride
Ice stick

Table 2: Methodology (Stimuli) used to assess dentine hypersensitivity in the clinical setting (acknowledgment reproduced from Gillam et al. 2000, Gillam 2013)

Selected Definitions

Dentine Hypersensitivity (DH)

As previously indicated the clinical diagnosis of Dentine Hypersensitivity has been a diagnosis of exclusion based on the Martin Addy's 1985 definition of DH as "pain derived from exposed dentine in response to chemical, thermal, tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or pathology" (Addy et al. 1985, Dowell et al. 1985, Addy 2000). This definition was subsequently modified by the Canadian Board on DH which suggested that 'disease' is a more appropriate term than 'pathology' (Canadian Consensus Document 2003).

Atypical Odontology (AO)

According to Buchanan (2019) AO or persistent dentoalveolar pain is one of several subtly different orofacial pain conditions which are included within the diagnosis of persistent idiopathic facial pain (PIFP (previously known as atypical facia pain). There appears however that there are disagreements on both the taxonomy and diagnostic criteria which presents a diagnostic challenge for both the dentist and the medical practitioner (Renton et al. 2012, Zakrzewska 2016, Malacarne et al 2018)(see below).

AO is likely neuropathic in origin, but pathophysiological mechanisms to explain the onset and persistence of the pain are still far from understood. It should also be noted that patients with AO will have additional extra-oral pain and associated depressive illnesses, stress and adverse life events which may influence the presentation of their facial pain (Harrison 2002). For these reasons, the management of AO is best undertaken in a multi-disciplinary setting (Buchanan 2019).

Selected Classifications and Definitions of PIFP and AO

• PIFP is described as a 'persistent facial and/or oral pain, with varying presentations but recurring daily for more than two hours per day over more than three months, in the absence of clinical neurologic deficit (International Headache Society Classification (ICHD) 2020).

• The International Association for the Study of Pain(IASP) has defined atypical odontalgia as a severe throbbing pain without major pathology, or it may be defined as pain of dental origin without a definitive organic cause (Woda and Pionchon 1999, Sharav 2013a, b).

• Atypical odontalgia, also known as atypical facial pain, phantom tooth pain, or neuropathic orofacial pain, is characterized by chronic pain in a tooth or teeth, or in a site where teeth have been extracted or following endodontic treatment, without an identifiable cause. Over time, the pain may spread to involve wider areas of the face or jaws. The diagnosis of atypical odontalgia is made after a thorough history, clinical examination, and radiographic assessment fail to identify a cause for the pain. Once the diagnosis is made, medications can be used to reduce the level of pain (The American Academy of Oral Medicine 2015) (selected text)

Molar Incisor Hypomineralisation (MIH)

Molar-Incisor hypomineralization (MIH) has been defined as a hypomineralization of systemic origin that affects one to all first permanent molars and is often associated with affected permanent incisors (Weerheijm et al., 2001).

Discussion

The purpose of this review was to highlight the problems that clinicians may have in diagnosing dental conditions, particularly those conditions with similar pain symptoms. My initial concern was that both the terminology and definitions for the conditions under discussion is often confusing when reading the published literature. From a diagnostic perspective precise terminology and definitions are important if we are to be consistent with our diagnosis in resolving our patients' dental pain. In my defense it is not a question of semantics but of consistency when using terminology/definitions, and it will hopefully be able for clinicians to universally describe dental conditions and subsequently proscribe the appropriate treatment. It is recognized however, that in some disciplines (e.g. oral medicine) there is still appears disagreement and confusion in the classification of PIFP/AO (Renton et al. 2012, Zakrzewska 2016, Malacarne et al 2018). More recently there have been papers on MIH which describe the pain associated with the condition as either DH, sensitivity/hypersensitivity (Sobral et al. 2021, Bekes et al. 2022), Tooth sensitivity (Almuallem & Busuttil-Naudi 2018) and DH, sensitivity, MIH hypersensitivity (Mendonça et al. 2022, Inchingolo et al. 2023). It is evident that there should be some clarification when discussing dental pain associated with the various dental conditions which will commonly be treated by clinicians.

According to Gillam and Koyi (2019a) patients present with a variety of orofacial conditions which are complex in nature and require at times referral to a specialist service, e.g. various neuromuscular, vascular conditions as well as PIFP (as discussed in this review). For example, a study involving approximately 2500 subjects selected at random from the general population over a period of one month reported that the most commonly oral facial problems were as follows i) Temporomandibular joint (TMJ) disorders pain (1.5%), ii) Irreversible pulpitis (1.2%), iii) chronic idiopathic facial pain (0.9%) and iv), DH/ reversible pulpitis (0.3%) (Drangsholt & LeResche 2009). Results from other studies indicated that orofacial pain affects approximately 9-10% to 16.1% of the adult population with woman more affected than men (Kohlmann 2002, Horst et al. 2015, Allison et al. 2020). According to Abiko et al. (2012) AO occurs more frequently in females rather than males .Of the dental conditions outlined in Table 1, toothache (dental caries [reversible/irreversible pulpitis]) is probably the most common clinical condition clinicians

will encounter in dental practice. Pau et al (2000) reported that those individuals suffering from toothache may experience a feeling of despair (depression/sleep deprivation) or helplessness and frustration of not being able to cope with the pain that has a major impact on their quality of life (QoL) as well as the difficulty in accessing a clinician to resolve their problem. This state of despair and helplessness is also apparent in some of the other conditions outlined in Table 1 (e.g. PIFP), which may make the diagnosis of their pain difficult to accurately assess due to 1) the subjectivity of pain, 2) patient's physical and emotional state and 3) the patient's threshold of pain (pain tolerance). It is important therefore for the clinician to conduct the dental examination and recording of the patient's medical and dental history in a considerate manner. This approach is essential particularly when using special tests in the examination process, for the use of a cold air syringe (e.g., Schiff Score (SCASS) [Schiff et al. 2009]) in the evaluation of DH and MIH in children (Bekes et al. 2022).

The use of a QoL Index has also been used to evaluate the impact of various medical and dental conditions on the QoL of individuals presenting with pain or for the evaluation of the efficacy of dental procedures and treatment effects (Baiju et al. 2017, Mason et al.2019 [DHEQ], Bekes et al. 2021 [Oral health-related quality of life (OHRQoL]).Table 1 outlines the differences between the aetiology, pain characteristics and intensity, test stimuli and associated features of common dental conditions. It should be noted, however that clinicians like to have these conditions in neat diagnostic boxes, and perhaps it is time to reclassify conditions associated with the pulp, root canal and peri-radicular tissues (Abbot 2024) as well as the other conditions discussed in this review. This view appears to be supported by Allison et al. 2020 where they indicated that the traditional description of pulpitis (reversible and irreversible) does not always correlate with the histological status of the pulp. According to Närhi et al. (1992) intradental C-fibres are activated only if the external stimuli reach the pulp proper and their activation may contribute to the dull pain induced by intense thermal stimulation of the tooth and to that associated with pulpal inflammation.

DH

Several terms have been used in the literature to describe DH, for example, cervical dentine sensitivity (CDS), or cervical dentine hypersensitivity (CDH), or dentine sensitivity (DS), and more recently dentine hypersensitivity (DHS). Although the term "dentin hypersensitivity" (DH) has been preferred in the published literature due in part to its historical significance "dentin sensitivity" (DS) may be a more accurate term (Dababneh et al. 1999, Gillam 2015). As indicated above DH has been defined as "pain derived from exposed dentin in response to chemical, thermal tactile, or osmotic stimuli which cannot be explained as arising from any other dental defect or disease" which as discussed in this review is essentially a diagnosis of exclusion.

On the issue raised about the term Dentine Hypersensitivity which has been used for decades, is that it should be acknowledged that the pain associated with the condition can be an exaggerated response of the normal pulp-dentine and the patient will only be aware of the problem when an external stimulus such as cold is applied to the exposed dentine surface. Historically the hydrodynamic theory as proposed by Brännström and Åström (1972) is currently accepted as a hypothesis for explaining the mechanism of the transmission of stimuli (from the exposed dentine surface to the dental pulp). However, it is recognised that not all stimulus transmission across the dentine can be explained by the hydrodynamic theory, and as such, there may be alternative mechanisms involved (Gillam 1995, 2015).

The intradental nerve fibres associated with DH are A nerve fibres (A-β and A-δ) in nature and probably activated by a hydrodynamic mechanism (fluid dynamics) which may be dependent on whether the dentine tubules are open or blocked (Narhi et al. 1992). From a diagnostic perspective DH is differentiated from other associated tooth pain by A-δ fibres which are mainly stimulated by the application of a cold stimulus, producing sharp pain, compared to the stimulation of C fibres which produce dull aching pain (Ngassapa 1996, Abd-Elmeguid & Yu

2009). More recently the role of the odontoblast in the transmission of stimulus transmission has been revisited and there is mounting evidence that human odontoblasts express functional TRP channels and voltage-gated sodium channels may play a crucial role in mediating thermal sensation in teeth (Allard et al. 2006, El Karim et al. 2011). It may therefore be possible that several mechanisms are involved in the sensory transduction process in teeth. Currently the management of DH is based on the hydrodynamic theory that allows for both tubule occlusion and nerve desensitization products to be applied to the tooth surface.

Post operative sensitivity from restorative, periodontal and bleaching procedures

As previously mentioned, patients may also experience pain from recent dental procedures (post-operative sensitivity) such as sensitivity from restorative materials, tooth whitening and bleaching periodontal disease and associated procedures such as non-surgical and surgical procedures (Troil et al., 2002, Sanz & Addy 2002, Gillam and Orchardson 2006, Markowitz 2010, Lin & Gillam 2012). Normally patients will attend the dental practice within a very short period following the procedure and depending on the procedure the patient can be assured that in most cases that the pain will be transient in nature and will gradually diminish in a few weeks for bleaching and periodontal procedures and up to six weeks for restorative procedures. Recommendations may include the provision of an over-the-counter desensitising toothpaste or mouthrinse, modifications in dietary habits and toothbrushing habits as well as monitoring the situation (Orchardson & Gillam 2006, Gillam et al. 2013 see also Gillam & Koyi 2019b). For more severe discomfort more invasive procedures may be more appropriate.

Bleaching sensitivity (BS)

According to Markowitz (2010) vital bleaching or whitening procedures are used to improving the appearance of discoloured teeth in patients with an array of whitening products for both home and dental office use, including peroxide containing gels or solutions. One of the adverse side effects with these procedures is bleaching sensitivity (BS). It is important to acknowledge that BS occurs in healthy intact tooth without any provoking stimulus. For the whitening process to be successful and whiten the teeth, the peroxide must be able to penetrate tooth structure and oxidize coloured compounds within the dentine structure. One of the problems in dental practice is that whitening procedures may take place with patients who have gingival recession, defective or leaking restorations etc and this may complicate the diagnostic process. Although the mechanism of nociceptor(s) activation in BS is unknown, it has been hypothesized that that the mechanism of pain generation that direct activation of intradental nerve activity via a chemosensitive ion channel (TRPA1 in the pulp) which is activated by a variety of oxidizer compounds including hydrogen peroxide (Markowitz (2010). This mechanism is clearly different to that proposed by Brännström's Hydrodynamic Theory (1962) where the stimulation of the exposed dentine results in fluid shifts within the dentinal tubules, which subsequently activate mechanosensitive nerve endings in the deep dentine and pulp (Markowitz 2010). The use of potassium salts in bleaching gels and desensitising toothpastes have been recommended in the treatment of BS (Markowitz 2010).

AO

As previously described AO or persistent dentoalveolar pain is one of several subtly different orofacial pain conditions which are included within the diagnosis of persistent idiopathic facial pain (PIFP (previously known as atypical facia pain). According to Ghurye & McMillan (2017) and Malacarne et al (2018) the persistent dentoalveolar pain disorder is likely neuropathic in origin, but pathophysiological mechanisms to explain the onset and persistence of the pain are still far from understood. According to Saguchi et al. (2019) there is no gold standard diagnostic protocol for AO and existing protocols are not sufficiently reliable for diagnosis as the physiopathology is not well defined. Several investigators

have expressed their concern about the taxonomy and diagnostic criteria which presents a diagnostic challenge for both the dentist and the medical practitioner (Woda &, Pionchon 1999, Woda et al. 2005, Renton et al. 2012, Zakrzewska 2016, Malacarne et al 2018). It is also apparent that as well as with other types of chronic orofacial pain, patients with AO may have additional extra-oral pain and associated depressive illnesses, sleep deprivation, stress and adverse life events which may influence the presentation of their facial pain and complicate the diagnostic process. For these reasons, the management of AO is best undertaken in a multi-disciplinary setting based on the established biopsychosocial model (Buchanan 2019).

According to Buchanan (2019) there are several reported causes of non-odontogenic toothache in the published literature which need to be considered during the diagnostic process such as 1) 'Atypical odontalgia', 2) Myofascial pain referred to the teeth (e.g. Myofascial pain dysfunction, referred pain from the cervical pain and muscles), 3) Neuropathic toothache (Episodic-Trigeminal neuralgia; Continuous – post herpetic neuralgia, 4) Painful post-traumatic trigeminal neuropathy (PTTN), 5) Maxillary sinusitis pain referred to maxillary molar and premolar teeth, 6) Neurovascular pain - Migraine, cluster headache, temporal arteritis, trigeminal autonomic cephalgias, 7) Myocardial ischaemic pain 8) Metastasis e.g., breast carcinoma, 9) Temporomandibular pathology (Arthritic change) and 10) burning mouth syndrome (BMS). According to Tu et al (2017) although the International Headache Society in their 2013 classification AO and BMS are classified separately although they are both psychiatrically considered to be somatic symptoms disorders. These authors concluded that BMS was a comorbid oral disorder in AO patients and contributed to a more intensively painful experience (see also Abiko et al (2012).

The possibility of non-odontogenic pain should be considered when managing patients with pain of apparent odontogenic origin in the absence of any supportive investigation finding (e.g., special tests including radiographic evaluation, use of pain scales e.g. Visual Analogue Scale [VAS], McGill pain descriptors, Hospital Anxiety and Depression [HAD] scores) (Buchanan 2019). Patients complaining of pain from a non-odontogenic origin may initially attend a dental practice believing that their problem is odontogenic in nature such as toothache or following tooth extraction (Abiko et al 2012). Problems however occur when the clinician is unable to find an obvious cause of the problem, and this may lead to inappropriate dental treatment such as restorative treatment (including endodontic treatment) and extraction of teeth. Unfortunately, the treatment may prove to be ineffective and may in fact exacerbate a neuropathic pain which can impact on the patient's mood and effect their quality of life (QoL). In the absence of evidence of an odontogenic cause for a patient's toothache, the clinician should avoid attempting any irreversible dental interventions.

The role of the clinician in general practice should be as a gatekeeper identifying the non-odontogenic nature of the patient's toothache with subsequent early referral to an Orofacial pain clinic which will allow the patient to access focussed specialist multidisciplinary management (including psychiatric support in dealing depression with prescribed pharmacological support etc) (see Tu et al. 2019) and hopefully avoid the patient from seeking repeated consultations with different clinicians to find an acceptable diagnosis and effective pain management (Buchanan 2019).

Furthermore, it is evident that patients struggle with a lack of knowledge by dental and medical professionals, diagnostic delays, and unnecessary treatments. The conclusion from Malacarne et al (2018) is worth noting in that agreement on the diagnostic criteria of PIFP/AO may enable both researchers and clinicians understand the condition and reduce patient suffering.

It should be noted that both DH and PIFP (AO) have a similar definition e.g., a diagnosis of exclusion which could initially create problems in the initial diagnosis, however there are distinct differences in their presenting histories and clinical features (see Table 1).

MIH

Molar Incisor Hypomineralization (MIH) is defined "as hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors and these molars are related to major clinical problems in severe cases. In the affected teeth there are areas of demarcated, qualitative developmental defects of systemic origin of the enamel of one or more first permanent molars with or without the involvement of incisors (can affect both primary and permanent dentitions) (Weerhijm 2001, 2004 (cited by Almuallem, & Busuttil-Naudi 2018), Ortega-Luengo et al. 2024). It is worth noting that the cosmetic appearance of discoloured teeth may impact on the individual child leading to psychiatric and behavioural problems and subject to bullying from other school children (Rodd et al. 2021).

From the literature it is apparent that there is a range of enamel disruption depending on the severity of the problem, for example, with the exposure of the underlying dentine the teeth may be very sensitive to external stimuli and if they are subject to post-eruptive tissue breakdown which may in turn lead to the development of caries (Rodd et al. 2021). Previous nomenclature included non-fluoride enamel opacities, internal enamel hypoplasia, non-endemic mottling of enamel, idiopathic enamel opacities and cheese molars (Weerhijm 2004 cited by Almuallem, & Busuttil-Naudi 2018). According to Lygidaki et al. (2022) MIH has a multifactorial aetiology where factors such as the duration, strength and timing of occurrence of the condition are responsible for the variable clinical characteristics on the affected teeth such as childhood diseases.

Other factors also appear to increase the risk of having MIH such as perinatal hypoxia, prematurity and other hypoxia related perinatal problems, including caesarean section (Lygidaki et al. (2022). There is also a consensus that genetic predisposition as well as the role of epigenetic influences (evidence from twin studies and genome and single-nucleotide polymorphisms analyses in patients and families (Bezamat et al. 2021, Lygidaki et al. 2022, da Silva Figueira et al. 2023). From a differential diagnostic perspective MIH should be distinguished from other abnormalities (defects in enamel formation such as: Fluorosis, Enamel hypoplasia, Amelogenesis imperfecta, White spot lesion and traumatic hypomineralisation) (Almuallem & Busuttil-Naudi 2018).

The treatment of MIH will depend on the severity of the problem and the tooth type in question (e.g., incisor or molar). For example, in incisors, the recommended treatment can include: microabrasion, Tooth bleaching, Etch-bleach-seal technique, Resin infiltration, composite restorations or porcelain veneers (for patients >18 years of age) and for molars: resin infiltration (e.g., Icon system consists of: Icon-Etch (15% hydrochloric acid), Icon-Dry (99% ethanol) and Icon-Infiltrant (Methacrylate-based resin), Restorations (e.g., Glass ionomer cement (GIC) or resin modified GIC restorations, full or partial coverage restorations (e.g., preformed metal crowns (PMCs), indirect onlays and extraction in severe cases. (Almuallem & Busuttil-Naudi 2018, Rodd et al. 2021). For the early remineralisation of the enamel in less affected cases the application of fluoride containing products such as Tooth Mousse (GC Corporation, Tokyo, Japan) and MI Paste Plus (GC Corporation, Tokyo, Japan) which contain calcium, phosphate, plus or minus fluoride) (Almuallem & Busuttil-Naudi 2018).

Several authors, however, have reported on the difficulties in managing MIH particularly in younger patients such as behavioural issues (due to fear and dental anxiety), the difficulty in anaesthetising the affected teeth, tooth sensitivity, failure of bonding when using adhesive materials etc (Almuallem & Busuttil-Naudi 2018, Rodd et al 2021). It should also be acknowledged that treatment of MIH will be a lengthy process which may result in multiple visits involving several medical (e.g., psychiatric management) and dental (e.g., paediatric and orthodontic) disciplines, absences from school as well as financial costs over time and impact on their QoL (Almuallem & Busuttil-Naudi 2018, Rodd et al 2021).

There have several issues raised over the years regarding the aetiology and idiopathic nature of MIH and the lack

of consensus regarding protocols, standardisation and limited understanding regarding the exact pathogenesis, as well as the lack of clarity in the diagnosis and management of MIH, particularly with the recommended criteria used to evaluate the extent of the problem (Lygidakis et al, 2010, 2022, Weerheijm 2015, Rodd et al 2021, Ortega-Luengo et al. (2024). Rodd et al (2021) also make a very good point in that although expert groups (e.g., European Academy of Paediatric Dentistry [EAPD]) have established MIH guidelines over the last two decades, health professionals from different countries and cultures are exposed to factors that may influence different treatment approaches. One of the issues raised by Weerheijm in 2015 was the lack of standardisation in the reporting of MIH which appears to have been addressed in the updated guidelines of the EAPD (Lygidakis et al, 2022). The question remains however is how MIH is being managed in general practice given the complexities of the condition and the extensive time required in both diagnosis and in short- and long-term management procedures. To address this issue, the British Society of Paediatric Dentistry (BSPD) has published a briefing paper on MIH to build up awareness of the condition and support the dental profession in both the diagnosis and management of MIH (BSPD 2020). Other developments of interest were the introduction of an easy-to-use clinical index and treatment plan (Based on the Würzburg concept) to aid clinicians in general dental practice (Bekes et al 2016a, b). A recent update of the Würzburg concept was published by the same group in 2023 (Bekes et al. 2023).

As previously mentioned in this review, the term DH has been used by investigators and clinician to describe the pain experienced by a patient affected by MIH, the objection to this description is not a question of semantics but reliance on the traditional definition of DH as proposed by Addy et al. (1985) namely “pain derived from exposed dentine in response to chemical, thermal, tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or pathology (disease).” It is essentially a diagnosis of exclusion and as such describing pain arising from MIH as DH does not fit in with this definition since it is a recognised clinical condition as with dental caries and AO as described in this review. Furthermore, as with the other conditions they clearly have defining characteristics that can be distinguish from DH, for example MIH arising from an abnormality in tooth development and in severe cases affected by the caries process. Another defining feature would be the age of the population with MIH compared with those individuals suffering with DH. Perhaps a reasonable compromise would be MIH hypersensitivity as described by Mendonça et al. (2022) and Inchingolo et al. (2023) or re-evaluating the traditional definition of DH. It is of note in this discussion that both BS and MIH were not considered to be linked with DH by the Committee providing DH guidelines for the management of a common oral health problem (Gillam et al. 2013)

Summary

A recurring theme with the various dental conditions discussed in the review was the lack of clarity, confusion and disagreement with the current definitions, for example, concerns were raised about a so-called gold standard diagnostic protocol, the taxonomy and diagnostic criteria or the lack of clarity in the diagnosis and management process. It was also evident that all the specialist bodies recognised that their guidelines needed to be updated but often this was conducted within the specific professional body rather than collaboration with all the various professional bodies to agree on the terminology to be used when describing pain arising from these conditions. A final observation would be the need to provide updates on the condition as well as guidelines to be used in daily dental practice to enable clinicians to correctly identify these conditions and where necessary refer to the relevant speciality.

Acknowledgements

Gillam D & B Koyi B (2019) Dentin Hypersensitivity in Clinical Practice. JP Medical, India. ISBN: 1909836478.

I also acknowledge the expertise of the investigators in the papers I have quoted particularly in the areas outside my own expertise.

References

1. Abbott PV. Pulp, Root Canal and Peri-radicular Conditions: The Need for Re-classification. Iran Endod J. 2024;19(3):158-175. doi: 10.22037/iej. v19i3.44394. PMID: 39086712; PMCID: PMC11287045.
2. Abd-Elmeguid A, Yu DC. Dental pulp neurophysiology: part 1. Clinical and diagnostic implications. J Can Dent Assoc. 2009; 75:55–9.
3. Abiko Y, Matsuoka H, Chiba I, Toyofuku A. Current evidence on atypical odontalgia: diagnosis and clinical management. Int J Dent. 2012; 2012:518548. doi: 10.1155/2012/518548. Epub 2012 Jul 9. PMID: 22844283; PMCID: PMC3400349.
4. Addy M, Mostafa P, Absi EG, Adams D (1985) Cervical dentin hypersensitivity. Etiology and management with particular reference to dentifrices. In: Rowe NH (ed) Proceedings of symposium on hypersensitive dentin—origin and management. University of Michigan, pp 147–167
5. Addy M. Dentin hypersensitivity: definition, prevalence distribution and etiology. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. pp. 239–248.
6. Aghabeigi B. Dental pain. In: Zakrzewska JM, editor. Pain research and clinical management: assessment and management of orofacial pain, vol. 4. Amsterdam: Elsevier; 2002. pp. 181–189.
7. Allard B, Magloire H, Couble ML, Maurin JC, Bleicher F. Voltage-gated sodium channels confer excitability to human odontoblasts: possible role in tooth pain transmission. J Biol Chem. 2006 Sep 29; 281 (39):29002-10. doi:10.1074/jbc.M601020200. Epub 2006 Jul 10. PMID: 16831873.
8. Allison JR, Stone SJ & Pigg M (2020) The painful tooth: mechanisms, presentation and differential diagnosis of odontogenic pain. Oral Surgery Special Issue: Oral facial Pain, 13: 4; 309-320
9. Almuallem, Z., Busuttil-Naudi, A. Molar incisor hypomineralisation (MIH) – an overview. Br Dent J 225, 601–609 (2018). https://doi.org/10.1038/sj.bdj.2018 .814
10. American Academy of Oral Medicine. www.aaom.com › index Atypical Odontalgia - AAOM (2015) Accessed 10th August 2024.
11. Baiju RM, Peter E, Varghese NO, Sivaram R. Oral Health and Quality of Life: Current Concepts. J Clin Diagn Res. 2017 Jun;11(6): ZE21-ZE26. doi:10.7860/JCDR/2017/25866.10 110. Epub 2017 Jun 1. PMID: 28764312;PMCID: PMC5535498.
12. Bekes K, Amend S, Priller J, Zamek C, Stamm T, Krämer N.

Changes in oral health-related quality of life after treatment of hypersensitive molar incisor hypomineralization-affected molars with a sealing. Clin Oral Investig. 2021 Nov;25(11):6449-6454. doi: 10.1007/s00784-021-03947-z. Epub 2021 Apr 20. PMID: 33876317; PMCID: PMC8531117

13. Bekes K, Amend S, Priller J, Zamek C, Stamm T, Krämer N. Hypersensitivity relief of MIH-affected molars using two sealing techniques: a 12-week follow-up. Clin Oral Investig. 2022 Feb;26(2):1879-1888. doi:10.1007/s00784-021-04163-5. Epub 2021 Sep 1. PMID: 34468888;PMCID: PMC8816787.

14. Bekes K, Steffen R. The Wuerzburg MIH concept: part 1. The MIH treatment need index (MIH TNI). A new index to assess and plan the treatment in patients with molar incisior hypomineralization (MIH) Oralprophylaxe Kinderzahnheilkunde. 2016a; 38:165–170. doi: 10.3238/OPKZH.2016.0165-0170

15. Bekes K, Krämer N, van Waes H, Steffen R. The Wuerzburg MIH concept: part 2. The treatment plan. Oralprophylaxe Kinderzahnheilkunde. 2016b; 38:171–175. doi: 10.3238/OPKZH.2016.0171-0175.

16. Bekes K, Steffen R, Krämer N. Update of the molar incisor hypomineralization: Würzburg concept. Eur Arch Paediatr Dent. 2023 Dec;24(6):807-813. doi: 10.1007/s40368-023-00848-5. Epub 2023 Oct 19. PMID: 37856065; PMCID: PMC10657291.

17. Bezamat M, Souza JF, Silva FMF, Corrêa EG, Fatturi AL, et al. Gene-environment interaction in molar-incisor hypomineralization. PLoS One. 2021 Jan 6;16(1):e0241898. doi:10.1371/journal.pone.0241898 PMID: 33406080; PMCID: PMC7787379.

18. Brännström M, (1962). The elicitation of pain in human dentine and pulp by chemical stimuli. Arch Oral Biol 7: 59-62.

19. Brännström M, Åström A. The hydrodynamics of the dentin; its possible relationship to dentinal pain. Int Dent J. 1972;22(2):219–27.

20. BSPD publishes MIH paper. Br Dent J 228, 144 (2020). https://doi.org/10.1038/s41415-020-1295-2

21. Buchanan J (2019) Atypical odontalgia Section 2 Case 6: In Dentin Hypersensitivity in Clinical Practice pp 91-104. Gillam D & B Koyi B eds JP Medical, India. ISBN: 1909836478.

22. Canadian Advisory Board on Dentin Hypersensitivity Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity. J Can

Dent Assoc. 2003; 69:221–226.

23. Cunha-Cruz J and Wataha JC (2014) The burden of dentine hypersensitivity. In Dentine Hypersensitivity: Developing a Person-centred Approach to Oral Health. Robinson PG (ed), pp 34-44.

24. Dababneh RH, Khouri AT, Addy M. Dentine hypersensitivity - an enigma? A review of terminology, mechanisms, aetiology and management. Br Dent J. 1999 Dec 11;187(11):606-11; discussion 603. doi: 10.1038/sj.bdj.4800345. PMID: 16163281

25. da Silva Figueira R, Mustafa Gomes Muniz FW, Costa LC, Silva de Moura M, Moura LFAD, Mello de Oliveira B, Lima CCB, Rösing CK, de Lima MDM. Association between genetic factors and molar-incisor hypomineralisation or hypomineralised second primary molar: A systematic review. Arch Oral Biol. 2023 Aug; 152:105716. doi:10.1016/j.archoralbio.2023.105716. Epub 2023 May 7. PMID: 37210809.

26. Dowell P, Addy M, Dummer P. Dentine hypersensitivity: aetiology, differential diagnosis and management. Br Dent J. 1985 Feb 9;158(3):92-6. doi:10.1038/sj.bdj.4805542. PMID: 3857921.

27. Drangsholt M, Leresche L. Epidemiology of Orofacial pain. In: Zakrzewska JM, (Ed). Orofacial Pain, Oxford University Press, Oxford, UK 2009.

28. El Karim IA, Linden GJ, Curtis TM, About I, McGahon MK, Irwin CR, Lundy FT. Human odontoblasts express functional thermo-sensitive TRP channels: implications for dentin sensitivity. Pain. 2011 Oct;152(10):2211-2223. doi: 10.1016/j.pain.2010.10.016. Epub 2010 Dec 17. PMID: 21168271.

29. Ghurye, S., McMillan, R. Orofacial pain – an update on diagnosis and management. Br Dent J 223, 639–647 (2017). https://doi.org/10.1038/sj.bdj.2017.879

30. Gibson B, Boiko OV, Baker S, PG Robinson PG, A Barlow A, Player T, Locker D (2010) The everyday impact of dentin sensitivity: personal and functional aspects. Social Sciences and Dentistry. 1(1) 11–20.

31. Gillam DG. Mechanisms of stimulus transmission across dentin—a review. J West Soc Periodontol Periodontal Abstr. 1995;43(2):53–65. Review.

32. Gillam DG. Dentin hypersensitivity: an introduction to differential diagnosis. Dent Pract. 2010;48(32):34–35.

33. Gillam DG (2013) Current diagnosis of dentin hypersensitivity in the dental office: an overview. Clin Oral Investig. 17 Suppl 1(Suppl 1): S21-S29.

34. Gillam DG. Management of Dentin Hypersensitivity. Curr Oral Health Rep 2, 87–94 (2015).

https://doi.org/10.1007/s40496-015-0047-x

35. Gillam DG, Orchardson R, Närhi MVO, Kontturi-Närhi V. Present and future methods for the evaluation of pain associated with dentin hypersensitivity. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. pp. 283–297

36. Gillam D, Orchardson R. Advances in the treatment of root dentin sensitivity: mechanisms and treatment principles. Endod Top. 2006; 13:13–33. doi: 10.1111/j.1601-1546.2006.00209.x.

37. Gillam D, Chesters R, Attrill D, Brunton P, Slater M, Strand P, Whelton H, Bartlett D. Dentine hypersensitivity--guidelines for the management of a common oral health problem. Dent Update. 2013 Sep;40(7):514-6, 518-20, 523-4. doi: 10.12968/denu.2013.40.7.514. PMID: 24147382.

38. Gillam D & B Koyi B (2019a) Examination and Diagnosis Section 1 Chapter 2: In Dentin Hypersensitivity in Clinical Practice pp13-23. Gillam D & B Koyi B eds JP Medical, India. ISBN: 1909836478.

39. Gillam D & B Koyi B (2019b) Management Section 1 Chapter 3 Dentin Hypersensitivity in Clinical Practice. Pp 25-42. Gillam D & B Koyi B eds JP Medical, India. ISBN: 1909836478.

40. Gillam DG (2021a). The Impact of Dentine Hypersensitivity on the Quality of Life: An Overview Clin Oral Sci Dent (2021), 4:1.

41. Gillam D (2021b). Clinician's awareness of the Management of Dentine Hypersensitivity. An Overview. Advanced Journal of Dentistry and Stomatology vol. 2021, (1)

42. Harrison SD. Atypical facial pain and atypical odontalgia. Chapter 14; 255-266. In Assessment and Management of Orofacial Pain. Pain Research and Clinical Management Eds Zakrzewska JM and Harrison SD: Vol 14 2002

43. Horst OV, Cunha-Cruz J, Zhou L, Manning W, Mancl L, DeRouen TA. Prevalence of pain in the orofacial regions in patients visiting general dentists in the Northwest Practice-based REsearch Collaborative in Evidence-based DENTistry research network. J Am Dent Assoc. 2015 Oct;146(10):721-8.e3. doi: 10.1016/j.adaj.2015.04.001. Erratum in: J Am Dent Assoc. 2015 Dec; 146(12):874. PMID: 26409981;PMCID: PMC7289192.

44. Inchingolo AM, Inchingolo AD, Viapiano F, Ciocia AM, Ferrara I, Netti A, Dipalma G, Palermo A, Inchingolo F. Treatment Approaches to Molar Incisor Hypomineralization: A Systematic Review. J Clin Med. 2023 Nov 20;12(22):7194. doi:10.3390/jcm12227194. PMID: 38002806;PMCID:

PMC10671994.

45. The International Association for the Study of Pain (IASP) (cited by Woda and Pionchon 1999).
46. International Headache Society. The international classification of headache disorders, 3rd edition (beta version). Cephalalgia. 2013; 33:629–808
47. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia. 2020; 40(2):129-221. doi:10.1177/0333102419893823International Headache Society Classification (ICHD) 2020).
48. Kohlmann T. Epidemiologie orofazialer Schmerzen [Epidemiology of orofacial pain]. Schmerz. 2002 Sep;16(5):339-45. German. doi:10.1007/s004820200000. PMID: 12235497.
49. Lin YH, Gillam DG (2012). The Prevalence of Root Sensitivity following Periodontal Therapy: A Systematic Review. Int J Dent vol. 2012
50. Lygidakis, N.A., Wong, F., Jälevik, B. et al. Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralisation (MIH). Eur Arch Paediatr Dent 11, 75–81 (2010). https://doi.org/10.1007/BF03262716
51. Lygidakis NA, Garot E, Somani C, Taylor GD, Rouas P, Wong FSL. Best clinical practice guidance for clinicians dealing with children presenting with molar-incisor-hypomineralisation (MIH): an updated European Academy of Paediatric Dentistry policy document. Eur Arch Paediatr Dent. 2022 Feb;23(1):3-21. doi: 10.1007/s40368-021-00668-5. Epub 2021 Oct 20. PMID: 34669177; PMCID: PMC8926988.
52. Malacarne A, Spierings ELH, Lu C, Maloney GE. Persistent Dentoalveolar Pain Disorder: A Comprehensive Review. J Endod. 2018 Feb;44(2):206-211. doi: 10.1016/j.joen.2017.09.009. Epub 2017 Nov 23. PMID: 29174443.
53. Markowitz K. Pretty painful: why does tooth bleaching hurt? Med Hypotheses. 2010 May; 74(5):835-40. doi: 10.1016/j.mehy.2009.11.044. Epub 2009 Dec 31. PMID: 20045265.
54. Mason S, Burnett GR, Patel N, Patil A, Maclure R. Impact of toothpaste on oral health-related quality of life in people with dentine hypersensitivity. BMC Oral Health. 2019 Oct 22; 19(1):226. doi: 10.1186/s12903-019-0919-x. PMID: 31640741; PMCID: PMC6805594.
55. Mendonça FL, Regnault FGDC, Di Leone CCL, Grizzo IC, Bisaia A, Fragelli C, Oliveira TM, Magalhães AC, Rios D. Sensitivity Treatments for Teeth with Molar Incisor Hypomineralization: Protocol for a Randomized Controlled Trial.

JMIR Res Protoc. 2022 Jan 6;11(1):e27843. doi: 10.2196/27843. PMID: 34989687;PMCID: PMC8778566.

56. Mosby's Medical Dictionary, 8th edition. © 2009, Elsevier http://www.merriam-webster.com/dictionary/diagnosis. Accessed 10th August 2024.

57. Narhi M, Kontturi-Narhi V, Hirvonen T, Ngassapa D. Neurophysiological mechanisms of dentin hypersensitivity. Proc Finn Dent Soc. 1992;88 Suppl 1:15–22.

58. Ngassapa D. Correlation of clinical pain symptoms with histopathological changes of the dental pulp: a review. East Afr Med J. 1996; 73:779–81.

59. Orchardson R, Gillam DG. Managing dentin hypersensitivity. J Am Dent Assoc. 2006 Jul;137(7):990-8; quiz 1028-9. doi: 10.14219/jada.archive.2006.0321. PMID: 16803826.

60. Ortega-Luengo, S., Feijóo-Garcia, G., Miegimolle-Herrero, M. et al. Prevalence and clinical presentation of molar incisor hypomineralisation among a population of children in the community of Madrid. BMC Oral Health 24, 229 (2024). https://doi.org/10.1186/s12903-024-04003-4

61. Pau AKH, Croucher R, Marcenes W. Perceived inability to cope and care-seeking in patients with toothache: a qualitative study. Brit Dent J. 2000; 189:503–506.

62. Renton T, Durham J, Aggarwal VR. The classification and differential diagnosis of orofacial pain. Expert Rev Neurother. 2012 May; 12(5):569-76. doi:10.1586/ern.12.40. PMID: 22550985.

63. Rodd HD, Graham A, Tajmehr N, Timms L, Hasmun N. Molar Incisor Hypomineralisation: Current Knowledge and Practice. Int Dent J. 2021 Aug;71(4):285-291. doi: 10.1111/idj.12624. Epub 2021 Jan 27. PMID: 34286697; PMCID: PMC9275314.

64. Saguchi A-H, Toshie A, Yamamoto A, de Almeida Baldini Cardoso C, de Oliveira Lira Ortega A. Atypical odontalgia: pathophysiology, diagnosis and management Odontalgia atípica: fisiopatologia, diagnóstico e tratamento. BrJP. São Paulo, 2019 oct-dec;2(4): 368-73

65. Sanz M, Addy M. Group D summary. J Clin Periodontol. 2002;29 (Suppl 3):195–196. doi: 10.1034/j.1600-051X.29.s3.19.x.

66. Schiff T, Delgado E, Zhang YP, Cummins D, DeVizio W, Mateo LR (2009) Clinical evaluation of the efficacy of an in-office desensitizing paste containing 8% arginine and calcium carbonate in providing instant and lasting relief of dentin hypersensitivity. Am J Dent 22 Spec No A:8A-15A.

67. Sharav, Y. (2013a). Atypical Odontalgia. In: Gebhart, G.F., Schmidt, R.F. (eds) Encyclopedia of Pain. Springer, Berlin, Heidelberg.

https://doi.org/10.1007/978-3-642-28753-4_322

68. Sharav, Y. (2013b). Atypical Facial Pain: Etiology, Pathogenesis, and Management. In: Gebhart, G.F., Schmidt, R.F. (eds) Encyclopedia of Pain. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-642-28753-4_321

69. Sobral APT, Santos EM, Aranha AC, et al. The control of pain due to dentin hypersensitivity in individuals with molar–incisor hypomineralisation: a protocol for a randomised controlled clinical trial. BMJ Open 2021;11: e044653. doi:10.1136/bmjopen-2020-044653

70. Troil BV, Needleman I, Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. J Clin Periodontol. 2002;29(Suppl 3):173–177. doi: 10.1034/j.1600-051X.29.s3.10.x.

71. Tu, T.T.H., Miura, A., Shinohara, Y., Mikuzuki, L., Kawasaki, K., et al. (2018), Evaluating Burning Mouth Syndrome as a Comorbidity of Atypical Odontalgia: The Impact on Pain Experiences. Pain Pract, 18: 580-586. https://doi.org/10.1111/papr.12647

72. Tu TTH, Miura A, Shinohara Y, Mikuzuki L, Kawasaki K, et al. Pharmacotherapeutic outcomes in atypical odontalgia: determinants of pain relief. J Pain Res. 2019 Feb 27; 12:831-839. doi: 10.2147/JPR.S188362. PMID: 30881094; PMCID:

73. Weerheijm KL, Jalevik B, Alaluusua S (2001) Molar-incisor hypomineralisation. Caries Res 35:390–391 47479

74. Weerheijm, K. The European Academy of Paediatric Dentistry and Molar Incisor Hypomineralisation. Eur Arch Paediatr Dent 16, 233–234 (2015). https://doi.org/10.1007/s40368-015-0189-5.

75. Woda A, Pionchon P. A unified concept of idiopathic orofacial pain: clinical features. J Orofac Pain. 1999 Summer;13(3):172-84; discussion 185-95. PMID: 10823031.

76. Woda A, Tubert-Jeannin S, Bouhassira D, Attal N, Fleiter B, Goulet JP, Gremeau-Richard C, Navez ML, Picard P, Pionchon P, Albuisson E. Towards a new taxonomy of idiopathic orofacial pain. Pain. 2005 Aug;116(3):396-406. doi: 10.1016/j.pain.2005.05.009. PMID: 15979796.

77. Zakzrewska JM. Chronic/ Persistent Idiopathic Facial Pain. Neurosurg Clin N Am 27 (2016); 345-351.

© The Author(s) 2024. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.

Ready to submit your research? Choose RN and benefit from:

- Fast, convenient online submission.
- Thorough peer review by experienced researchers in your field.
- Rapid publication on acceptance.
- Support for research data, including large and complex data types.
- Global attainment for your research.
- **At RN, research is always in progress.**
- **Learn more:** researchnovelty.com/submission.php