International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

An Unusual Neuralgia-Like Pain Disorder Initially Presenting as Dentinal Hypersensitivity During Pregnancy: A Diagnostic Challenge

Dr. Shehna Manaf KP, BDS

Abstract: ***Dentinal hypersensitivity is generally characterized by short-lasting pain that resolves following removal of the provoking stimulus and responds to conventional management. Progression of sensitivity-like symptoms into continuous neuralgia-like pain is unusual and may create substantial diagnostic uncertainty. The challenge becomes greater during pregnancy, when physiological and hormonal changes may influence pain perception and treatment options. This report describes an atypical pain presentation that evolved from apparent dentinal hypersensitivity into persistent neuralgia-like pain of uncertain origin.***

Keywords: Carbamazepine, dentinal hypersensitivity, differential diagnosis, neuropathic pain, orofacial pain, pregnancy, vitamin b12 deficiency, vitamin d deficiency.

1. Introduction

Dentinal hypersensitivity is a common clinical condition characterized by a short, sharp pain arising from exposed dentin in response to thermal, tactile, osmotic, or evaporative stimuli. [1-3]In most patients, the pain is transient, stimulus-dependent, and responds to conventional desensitizing therapies or minimally invasive restorative treatment. Persistence of pain despite appropriate dental management, or progression beyond the typical clinical presentation, should prompt consideration of alternative pain mechanisms.

Pregnancy may further complicate the diagnostic process because physiological, hormonal, and nutritional changes can influence pain perception while simultaneously limiting diagnostic investigations and pharmacological treatment options. Although dental pain is relatively common during pregnancy, atypical neuralgia-like pain initially presenting as dentinal hypersensitivity has been rarely reported.

This report describes the clinical course of a 30-year-old pregnant dentist whose symptoms evolved from apparent dentinal hypersensitivity into continuous neuralgia-like pain that was unresponsive to conventional dental treatment and systemic analgesics but demonstrated a favorable clinical response following carbamazepine therapy. The precise diagnosis remained uncertain despite multidisciplinary evaluation. This case highlights the importance of recognizing atypical pain presentations and maintaining a broad differential diagnosis when the clinical course deviates from that of conventional dentinal hypersensitivity

2. Case Presentation

Patient Information

A 30-year-old pregnant female at 20 weeks' gestation presented with severe dentinal hypersensitivity-like pain affecting the mandibular anterior teeth, which progressively evolved into continuous sharp, electric, neuralgia-like pain involving the mandibular anterior region and later the right mandibular posterior gingival region. The symptoms significantly interfered with toothbrushing, mastication, oral intake, sleep, and routine daily activities. During the acute phase, the patient was unable to tolerate any food throughout the day because of the intensity of the pain, with temporary relief achieved only by holding cold water in the mouth

History of Presenting Illness

The patient was apparently well until 9 July 2025 (20 weeks' gestation), when she developed the sudden onset of severe sensitivity-like pain involving the mandibular anterior teeth. Initially, the pain was exclusively triggered by thermal stimuli (hot and cold), with each episode lasting approximately 10–15 minutes. Temporary symptomatic relief was achieved with repeated application of a desensitizing toothpaste and topical application of crushed clove to the sensitive teeth and adjacent gingival mucosa. Based on the initial clinical presentation, a provisional diagnosis of dentinal hypersensitivity was made, and conservative management was initiated.

Over the following days, the sensitivity-like pain gradually increased in both frequency and severity. The patient required progressively more frequent applications of the desensitizing toothpaste and crushed clove to obtain temporary relief. Despite these measures, the symptoms continued to progress. Repeated topical application of crushed clove resulted in localized gingival and mucosal irritation; however, the pain persisted.

As the symptoms failed to improve, an endodontic consultation was sought. Dentinal hypersensitivity secondary to attrition was considered, and a flowable composite restoration was placed in an attempt to reduce the sensitivity. However, no clinically meaningful improvement was observed following the procedure, and the symptoms continued to evolve.

Subsequently, the clinical pattern changed. In addition to thermal stimuli, exposure to cool air became a trigger, and the pain gradually evolved from intermittent sensitivity-like episodes to prolonged and eventually continuous sharp, electric, neuralgia-like pain. The pain also extended from the mandibular anterior region to involve the right mandibular posterior gingival region (44–46). The patient reported an itching sensation preceding the onset of pain in the affected

gingival region, where mild blanching on palpation was observed without significant swelling or erythema.

On 19 July 2025, the patient awoke with severe continuous pain that progressively intensified throughout the day [approximately 12 hours]. The pain evolved from intermittent sensitivity-like episodes to a continuous sharp, electric, shooting pain with neuralgia-like characteristics involving the mandibular anterior region and extending to the right mandibular posterior gingival region. The symptoms became so severe that she was unable to brush her teeth, chew, or tolerate any food throughout the day. Temporary symptomatic relief was achieved only by continuously holding cold water in the mouth. In an attempt to maintain oral intake, she consumed cold foods, including ice cream (falooda); however, these provoked an immediate and marked exacerbation of the pain despite the beneficial effect of cold water alone. Exposure to cool air, including air from a car air conditioner, also aggravated the symptoms.

Oral paracetamol administered during the course of the illness did not provide meaningful symptomatic relief. During gynaecological evaluation, intravenous paracetamol was also administered without significant improvement in symptoms. Because of the persistence and progression of the pain despite conservative dental treatment and analgesic therapy, a neurological consultation was obtained. Carbamazepine therapy was initiated on the night of 19 July 2025. Following the first dose, the patient's symptoms resolved completely. She remained pain-free the following morning, with only a mild recurrence later that evening that responded to the scheduled dose. Carbamazepine therapy was continued for approximately two weeks, during which no recurrence of the severe continuous pain was reported.

3. Clinical Findings

General Examination
General examination revealed a conscious, alert, oriented, and cooperative patient. The patient appeared clinically stable throughout the examination. No facial asymmetry, facial swelling, or other obvious extraoral abnormalities were observed.

Intraoral Examination
Initial intraoral examination revealed severe stimulus-evoked sensitivity involving the mandibular anterior teeth (33–43). Clinical examination demonstrated attrition involving the mandibular anterior teeth, and dentinal hypersensitivity secondary to exposed dentin was initially considered. Apart from attrition, no additional clinical findings were identified that adequately explained the severity, progression, and changing distribution of the patient's symptoms. No gingival recession was observed. Pulp vitality testing was not performed.

During the later stage of the illness, the pain extended to the right mandibular posterior gingival region corresponding to teeth 44–46. Clinical examination revealed mild blanching of the gingiva on palpation without clinically significant swelling, purulent discharge, sinus tract formation, or other signs of acute odontogenic infection. Repeated topical application of crushed clove to the affected teeth and adjacent gingival mucosa resulted in localized mucosal irritation.

Pain Characteristics
The pain demonstrated a distinct evolution during the clinical course. Initially, it presented as stimulus-dependent dentinal hypersensitivity-like pain triggered by hot and cold stimuli. Subsequently, the pain evolved into a continuous sharp, electric, shooting pain with neuralgia-like characteristics. The pain initially involved the mandibular anterior teeth (33–43) and later extended to the right mandibular posterior gingival region (44–46).

Exposure to cool air became a triggering factor during the progression of the illness. During the acute phase, temporary relief was achieved only while cold water was continuously held in the mouth. As the water gradually reached oral temperature, the pain recurred immediately, necessitating repeated replacement with fresh cold water. In contrast, cold sweet foods, including ice cream and falooda, produced immediate exacerbation of the pain. The pain was severe enough to interfere with toothbrushing, mastication, oral intake, and routine daily activities.

Clinical Photographs
Representative intraoral photographs obtained during the symptomatic phase are presented in Figures 1–4 and illustrate the clinical appearance of the affected mandibular anterior teeth and the right mandibular posterior gingival region during the course of the illness.

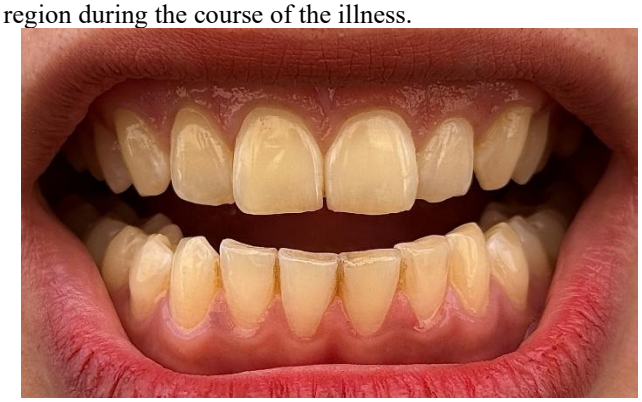

Figure 1

Figure 1. Frontal intraoral photograph demonstrating the mandibular anterior teeth (33–43). The patient's symptoms were initially localized to this region before subsequently progressing to involve the right mandibular posterior gingival region. Mild attrition of the mandibular anterior teeth is appreciable.

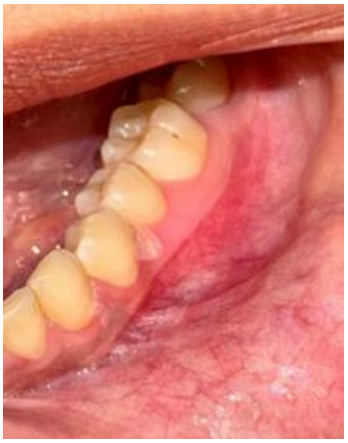

Figure 2: Intraoral photograph of the right mandibular posterior gingival and vestibular region (44–46) obtained during the symptomatic phase. A localized whitish mucosal lesion with surrounding mucosal irritation is visible. Based on the clinical history, the lesion was considered likely secondary to repeated topical application of crushed clove used by the patient for temporary symptomatic relief.

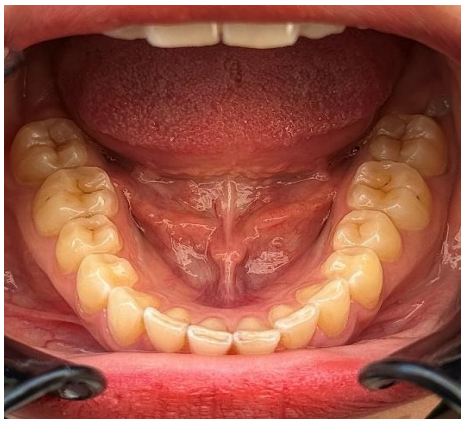

Figure 3: Frontal intraoral photograph demonstrating the mandibular anterior teeth (33–43). The patient's initial symptoms were localized to this region before progression to involve the right mandibular posterior gingival region.

Diagnostic Assessment

The patient's initial presentation was suggestive of dentinal hypersensitivity because of the presence of stimulus-evoked pain involving the mandibular anterior teeth (33–43) and the clinical finding of attrition. Based on this provisional diagnosis, conservative management with desensitizing agents and subsequent restoration of the attrited surfaces using flowable composite resin was undertaken. However, neither intervention produced significant or sustained symptomatic improvement. The pain continued to progress in severity, duration, and distribution despite these measures, prompting reconsideration of the initial diagnosis.

As the clinical course evolved, the pain demonstrated features that were not entirely consistent with isolated dentinal hypersensitivity. These included progression from stimulus-dependent pain to a continuous sharp, electric, and shooting pain with neuralgia-like characteristics, extension of symptoms beyond the initial tooth distribution to the right mandibular posterior gingival region (44–46), aggravation by exposure to cool air, inability to eat or brush because of pain, temporary relief only while cold water was continuously held in the mouth, and lack of meaningful response to both oral and intravenous paracetamol.

Clinical examination demonstrated attrition of the mandibular anterior teeth (Figures 1 and 3), which contributed to the initial consideration of dentinal hypersensitivity. During the symptomatic phase, a localized mucosal lesion was observed in the right mandibular posterior gingival region (Figure 2). Based on the clinical history, this lesion was considered likely secondary to repeated topical application of crushed clove used for temporary symptomatic relief rather than the primary disease process.

Laboratory investigations performed on 19 July 2025 revealed vitamin B12 deficiency (163 pg/mL; reference range 192–827 pg/mL) and vitamin D insufficiency (25-hydroxy vitamin D3: 27.3 ng/mL; reference range 30–100 ng/mL). HbA1c (4.7%) and estimated average glucose (88 mg/dL) were within normal limits. C-reactive protein was negative. These findings were considered potential contributors to altered neurosensory function; however, a direct causal relationship could not be definitively established.

Because of the atypical nature of the presentation and failure of conventional dental management, neurological consultation was obtained. Neurological evaluation favoured a diagnosis of mental neuralgia, and carbamazepine therapy was initiated. The rapid and substantial clinical response to carbamazepine supported consideration of a neuropathic pain mechanism. However, therapeutic response alone was not considered diagnostic.

Although mental neuralgia was considered the leading clinical diagnosis, the prolonged continuous nature of the pain, evolution from dentinal hypersensitivity-like symptoms, and changing symptom distribution represented atypical features. Accordingly, the case was documented as an unusual pregnancy-associated neuralgia-like orofacial pain disorder initially presenting as dentinal hypersensitivity, with definitive diagnostic confirmation remaining uncertain.

Table 1: Differential Diagnosis Considered During Evaluation

Differential Diagnosis	Basis for consideration	Findings against/ Limitations
Dentinal hypersensitivity	Initial hot- and cold-induced pain; visible attrition of mandibular anterior teeth (Figures 1 and 3)	Failed desensitizing therapy and flowable composite restoration; pain became continuous and extended beyond the initial teeth
Mental neuralgia	Considered during neurological evaluation; mandibular distribution; electric and shooting pain; response to carbamazepine	Symptom distribution evolved over time and prolonged continuous pain was atypical
Classical trigeminal	Electric shock-like pain quality; cold-air	Pain persisted continuously for prolonged periods rather than

neuralgia	sensitivity; response to carbamazepine	predominantly brief paroxysmal attack
Painful trigeminal neuropathy / neuralgia-like orofacial pain	Neuropathic pain characteristics and favorable response to carbamazepine	Insufficient evidence to establish a specific neuropathic diagnosis

Therapeutic Intervention

Initial management was directed toward the provisional diagnosis of dentinal hypersensitivity. The patient was advised to use a commercially available desensitizing toothpaste (Sensodyne®) and also self-administered topical crushed clove (Garambu) to the affected teeth and adjacent gingival tissues for symptomatic relief. Both measures provided only temporary relief, and the symptoms progressively worsened over time.

As the pain persisted, an endodontic consultation was obtained. Clinical attrition of the mandibular anterior teeth prompted placement of a flowable composite restoration in an attempt to reduce dentinal sensitivity. However, the procedure failed to produce significant or sustained improvement in symptoms.

During the acute phase of the illness, oral paracetamol 650 mg was administered for pain control but did not provide meaningful symptomatic relief. Subsequently, intravenous paracetamol was administered during medical evaluation; however, the pain remained largely unchanged.

Owing to the persistence, severity, and atypical characteristics of the pain, a neurological consultation was sought. Following neurological evaluation, carbamazepine therapy was initiated on 19 July 2025 at a dose of 100 mg. Marked clinical improvement was observed following initiation of treatment. After three days, the dose was increased to 200 mg based on medical advice, and treatment was continued for a total duration of two weeks.

Laboratory investigations performed during the symptomatic phase revealed vitamin B12 deficiency and vitamin D insufficiency. Accordingly, an intramuscular vitamin B12 injection was administered on 20 July 2025, and vitamin D supplementation was initiated as part of the overall management plan

Outcome and Follow-up

Following initiation of carbamazepine therapy on 19 July 2025, the patient experienced marked improvement in symptoms. The severe continuous neuralgia-like pain that had persisted throughout the day resolved after the first dose. On the following morning, the patient reported complete absence of pain. A mild recurrence of symptoms was noted later that evening, which responded to the scheduled dose of carbamazepine without further progression.

The carbamazepine dose was subsequently increased to 200 mg based on neurological advice and continued for a total duration of two weeks. During this period, the patient remained clinically stable, with progressive resolution of symptoms. Normal oral intake, toothbrushing, and routine daily activities were gradually resumed without significant discomfort.

Following laboratory confirmation of vitamin B12 deficiency and vitamin D insufficiency, intramuscular vitamin B12 therapy and vitamin D supplementation were initiated. At the completion of the two-week treatment period, the patient reported complete resolution of symptoms. During subsequent follow-up, there was no recurrence of the neuralgia-like pain, and no further dental or neurological intervention was required.

4. Discussion

4.1 Overview of the Case

This case describes an unusual presentation of pregnancy-associated neuralgia-like orofacial pain that initially mimicked dentinal hypersensitivity, resulting in a diagnostic challenge. The patient's symptoms began as localized thermal sensitivity involving the mandibular anterior teeth in the presence of visible attrition, making an initial diagnosis of dentinal hypersensitivity clinically reasonable. However, the subsequent progression of symptoms despite appropriate conservative dental management, evolution to sharp electric and shooting pain, extension beyond the initial tooth distribution, and eventual response to carbamazepine suggested an underlying neuropathic pain mechanism. This case highlights the importance of continuous reassessment when the clinical course deviates from the expected behaviour of common odontogenic conditions.

4.2 Dentinal Hypersensitivity Versus Neuropathic Orofacial Pain

Dentinal hypersensitivity is characterized by a short, sharp pain arising from exposed dentin in response to thermal, evaporative, tactile, osmotic, or chemical stimuli. The pain typically resolves rapidly following removal of the stimulus and generally responds to conservative measures such as desensitizing toothpastes, topical desensitizing agents, and restoration of exposed dentin when indicated.

In the present case, the initial clinical presentation closely resembled dentinal hypersensitivity because of stimulus-dependent pain and the presence of mandibular anterior attrition. Consequently, management with a desensitizing toothpaste (Sensodyne®), topical crushed clove, and flowable composite restoration represented an appropriate initial therapeutic approach. However, the absence of sustained improvement despite these interventions became a pivotal diagnostic clue.

As the disease evolved, the patient's symptoms progressively diverged from the typical characteristics of dentinal hypersensitivity. The pain became continuous, assumed a sharp, electric, and shooting quality, extended beyond the initially affected teeth to involve the posterior mandibular gingival region, and significantly impaired eating, toothbrushing, and routine daily activities. Furthermore, the pain failed to respond to both oral and intravenous paracetamol. Collectively, these features

suggested that the underlying pain mechanism was unlikely to be explained by dentinal hypersensitivity alone.

This case emphasizes that persistence or progression of symptoms despite appropriate conservative dental management should prompt clinicians to reconsider the initial diagnosis. Rather than repeating restorative or endodontic procedures, dentists should maintain a broad differential diagnosis that includes neuropathic orofacial pain disorders like trigeminal neuralgia [4-6] and seek interdisciplinary consultation when indicated. Early recognition of atypical features may help prevent unnecessary dental interventions and facilitate timely referral for appropriate management.

4.3 Pregnancy-Related Hormonal Changes and Their Potential Influence on Orofacial Pain

Pregnancy is associated with profound endocrine and physiological adaptations that influence multiple organ systems, including the nervous system. Marked increases in estrogen and progesterone throughout gestation have been shown to modulate nociceptive processing, peripheral nerve excitability, neurotransmitter activity, and neuroimmune interactions. These hormonal changes may alter pain perception, although their clinical effects vary among individuals and pain conditions.

In the present case, symptom onset occurred during the second trimester, a period characterized by sustained elevations in estrogen and progesterone. While a direct causal relationship between pregnancy and the patient's neuralgia-like symptoms cannot be established, the temporal association raises the possibility that pregnancy-related hormonal changes may have influenced the clinical expression or severity of pain.

In addition, laboratory investigations demonstrated concurrent vitamin B12 deficiency and vitamin D insufficiency, both of which have been implicated in peripheral nerve dysfunction and altered pain processing. It is therefore plausible that the clinical presentation resulted from the interaction of multiple physiological factors rather than a single isolated cause.[7-9]

This case highlights the importance of considering pregnancy as a physiological context that may modify symptom presentation without assuming that pregnancy itself is the underlying cause of neuropathic pain. Further studies are needed to clarify the relationship between pregnancy-related hormonal changes and atypical orofacial pain disorders.

4.4 Neuropathic Features and Consideration of Mental Neuralgia

As the clinical course progressed, several features became increasingly suggestive of a neuropathic pain disorder rather than isolated dentinal hypersensitivity. The pain evolved from transient stimulus-evoked sensitivity to persistent sharp, electric, and shooting pain that progressively impaired eating, toothbrushing, and routine daily activities. Unlike typical dentinal hypersensitivity, the symptoms failed to respond to repeated conservative dental interventions, including desensitizing toothpaste, topical crushed clove application, flowable composite restoration, and both oral and intravenous paracetamol. These findings prompted reconsideration of the initial diagnosis and referral for neurological evaluation.

Neurological assessment favored a diagnosis of mental neuralgia based on the mandibular distribution of pain, neuropathic pain characteristics, and the subsequent favourable response to carbamazepine. Mental neuralgia is an uncommon neuropathic pain disorder involving the terminal branch of the inferior alveolar nerve and may present with pain affecting the lower lip, chin, and adjacent gingival tissues. However, the present case demonstrated several atypical features, including an initial presentation resembling dentinal hypersensitivity, progression to prolonged continuous pain, and extension of symptoms beyond the initially affected region. These findings prevented definitive diagnostic confirmation based on currently accepted clinical criteria.

The marked improvement following carbamazepine therapy supports the presence of a neuropathic pain mechanism, as carbamazepine is a first-line treatment for several neuralgic disorders because of its ability to reduce neuronal hyperexcitability through blockade of voltage-gated sodium channels. Nevertheless, therapeutic response alone should not be regarded as diagnostic, since carbamazepine may provide symptomatic benefit in a variety of neuropathic pain conditions. Consequently, the diagnosis in the present case was based on the overall clinical presentation, symptom evolution, exclusion of common odontogenic causes, and multidisciplinary assessment rather than on drug response alone

4.5 The Cold-Water Phenomenon and Possible Neurophysiological Mechanisms

One of the most distinctive clinical observations in the present case was the paradoxical effect of cold stimuli on pain perception. During the acute phase, the patient experienced temporary relief only while cold water was continuously held in the oral cavity. As the water gradually reached oral temperature, the pain recurred immediately, necessitating repeated replacement with fresh cold water over several hours. In contrast, ingestion of cold foods such as ice cream and falooda precipitated severe pain rather than providing relief. This apparent discrepancy suggests that different cold stimuli may influence pain through distinct physiological mechanisms.

A possible explanation for the transient analgesic effect of cold water is the reduction in peripheral nerve excitability induced by tissue cooling. Experimental studies have demonstrated that cooling decreases nerve conduction velocity and reduces the excitability of sensory nerve fibers, thereby temporarily suppressing nociceptive transmission. In neuropathic pain conditions, cooling has also been reported to reduce spontaneous ectopic neural discharge by modulating voltage-gated sodium channel activity. These mechanisms may explain why pain subsided while the oral

tissues remained cooled but rapidly recurred as tissue temperature normalized.

The contrasting response to ice cream and falooda may be explained by the complex combination of thermal, mechanical, and osmotic stimulation associated with these foods. Unlike cold water, which provides relatively uniform cooling without significant mechanical stimulation, frozen foods require contact pressure, mastication, and often contain high concentrations of sugar, all of which may stimulate exposed dentinal tubules or sensitized peripheral nerves. Therefore, the patient's response suggests that thermal modulation alone was insufficient to determine pain perception and that multiple sensory mechanisms were likely involved.

Interestingly, carbamazepine, which produced sustained clinical improvement in this patient, acts primarily by stabilizing hyperexcitable neuronal membranes through inhibition of voltage-gated sodium channels. Although the mechanism of cooling differs from that of carbamazepine, both interventions may transiently reduce peripheral nerve hyperexcitability. This observation raises the hypothesis that the temporary analgesic effect of cold water and the sustained response to carbamazepine may reflect modulation of a common neuropathic pain pathway. However, this proposed mechanism remains speculative and requires further experimental investigation.

Reports describing prolonged dependence on continuous cold-water retention for relief of pregnancy-associated neuralgia-like orofacial pain appear to be limited in the available literature. This unusual clinical observation may represent a useful diagnostic clue in selected patients presenting with atypical orofacial pain that is refractory to conventional dental management. Further clinical studies are needed to determine whether this phenomenon is reproducible in other neuropathic orofacial pain disorders.

4.6 Vitamin B12 Deficiency and Vitamin D Insufficiency: Potential Contributing Factors

An additional noteworthy finding in the present case was the presence of biochemical abnormalities, including vitamin B12 deficiency (163 pg/mL) and vitamin D insufficiency (25-hydroxyvitamin D: 27.3 ng/mL). [10]. Both vitamins play important roles in maintaining normal nervous system function, and deficiencies have been associated with peripheral neuropathy, altered nociceptive processing, and neuropathic pain in both experimental and clinical studies.

Vitamin B12 is essential for DNA synthesis, myelin formation, and neuronal repair. Deficiency has been associated with impaired peripheral nerve function and may manifest as paresthesia, dysesthesia, or neuropathic pain. Similarly, vitamin D has been increasingly recognized for its role in neuroimmune regulation, neuronal function, and pain modulation. Although vitamin D insufficiency has been linked to chronic pain and certain neuropathic pain conditions, the available evidence remains heterogeneous, and a direct causal relationship has not been established.

In the present case, these biochemical abnormalities may have lowered the threshold for neural sensitization or influenced the severity of the patient's symptoms. However, given the coexistence of pregnancy-related physiological changes and the absence of definitive evidence linking these deficiencies to the observed clinical presentation, they should be regarded as potential contributing factors rather than primary etiological causes. Correction of these deficiencies formed part of the patient's comprehensive management, although the marked clinical improvement following carbamazepine suggests that treatment of the underlying neuropathic pain mechanism played a central role in symptom resolution.

This case highlights the importance of assessing nutritional status in patients presenting with atypical orofacial pain, particularly when symptoms are persistent, progressive, or refractory to conventional dental management. Recognition and correction of potentially reversible deficiencies may contribute to comprehensive patient care, even when they are not considered the primary cause of the pain disorder.

4.7 Failure of Conventional Dental Therapy and Diagnostic Implications

The present case highlights the importance of reassessing the diagnosis when symptoms fail to respond to evidence-based conservative management. The initial presentation was consistent with dentinal hypersensitivity, and treatment with a desensitizing toothpaste (Sensodyne®), topical crushed clove (Garambu), and restoration of the attrited surfaces using flowable composite resin was clinically appropriate. Despite these interventions, the patient's symptoms progressively worsened, ultimately interfering with eating, oral hygiene, and daily activities.

Similarly, both oral and intravenous paracetamol failed to provide meaningful pain relief, suggesting that the underlying pain mechanism was unlikely to be predominantly inflammatory or nociceptive. Persistent pain despite appropriate dental treatment should prompt clinicians to reconsider the initial diagnosis and broaden the differential diagnosis to include neuropathic orofacial pain disorders. Rather than repeating restorative procedures, timely multidisciplinary evaluation may facilitate earlier diagnosis and help avoid unnecessary dental interventions

4.8 Therapeutic Response to Pharmacotherapy

The patient's favourable response to carbamazepine represented an important component of the overall diagnostic assessment. While the diagnosis was established based on the clinical history, symptom evolution, exclusion of common odontogenic causes, and neurological evaluation, the substantial improvement following carbamazepine further supported the presence of a neuropathic pain mechanism. [11]. Nevertheless, therapeutic response alone should not be regarded as diagnostic, as carbamazepine may provide symptomatic benefit in several neuropathic pain disorders.

This case underscores the importance of interpreting treatment response within the broader clinical context rather

than relying solely on pharmacological effectiveness. A comprehensive assessment incorporating symptom characteristics, disease progression, clinical examination, laboratory investigations, and multidisciplinary evaluation remains essential for accurate diagnosis.

4.9 Clinical Implications

This case provides several important clinical lessons for dental practitioners. First, dentinal hypersensitivity should remain a diagnosis of exclusion when symptoms become progressive, persistent, or inconsistent with the expected clinical course. Second, failure of appropriate conservative therapy should prompt careful reassessment rather than repeated dental intervention. Third, the development of neuropathic pain characteristics, including sharp electric pain, evolving triggering factors, and prolonged symptom duration, should raise suspicion for non-odontogenic orofacial pain. Finally, early collaboration between dentists, oral medicine specialists, and neurologists may facilitate timely diagnosis, reduce patient morbidity, and prevent unnecessary dental procedures.

4.10 Strengths and Limitations of the Present Case

The strengths of this case include detailed chronological documentation of symptom progression, comprehensive clinical evaluation, laboratory investigations demonstrating vitamin B12 deficiency and vitamin D insufficiency, serial intraoral photographic documentation, and longitudinal follow-up demonstrating complete clinical resolution following neurological management. These findings provide a comprehensive description of an uncommon diagnostic presentation.

However, several limitations should be acknowledged. This report describes a single patient, limiting the generalizability of the findings. Advanced neurophysiological investigations specific to the mental nerve were not performed, and the proposed pathophysiological mechanisms remain hypothetical. Furthermore, although pregnancy-related hormonal changes and nutritional deficiencies may have contributed to the clinical presentation, causal relationships cannot be established from an individual case. Additional studies are required to determine whether similar clinical presentations occur in other patients with neuropathic orofacial pain.

4.11 Evolution of Therapeutic Response: A Diagnostic Clue

An important clinical observation in the present case was the evolution of the patient's response to conservative therapy. During the initial phase of the illness, both desensitizing toothpaste (Sensodyne®) and topical application of crushed clove (Garambu) consistently produced temporary symptomatic relief lasting several seconds to minutes during painful episodes. This transient response reinforced the provisional diagnosis of dentinal hypersensitivity and supported the initial conservative management approach.

However, as the disease progressed, the pain underwent a marked qualitative change, evolving into persistent sharp, electric, and shooting pain. Simultaneously, the previously effective conservative measures gradually lost their therapeutic benefit and eventually failed to provide meaningful relief. This change in treatment response paralleled the evolution of the clinical features and suggested that the underlying pain mechanism had progressed beyond uncomplicated dentinal hypersensitivity.

The present case illustrates that an initial favorable response to desensitizing agents or topical analgesic measures should not be regarded as definitive confirmation of dentinal hypersensitivity. Instead, clinicians should continuously reassess the diagnosis when symptom characteristics change or when previously effective therapies become ineffective. Dynamic changes in therapeutic response may themselves represent valuable diagnostic information, particularly in patients with evolving orofacial pain syndrome.

5. Conclusion

This case highlights the diagnostic challenges associated with atypical orofacial pain presenting initially as dentinal hypersensitivity during pregnancy. Progressive evolution of pain characteristics, failure of evidence-based conservative dental management, emergence of neuropathic features, and favourable response to carbamazepine emphasized the importance of reconsidering the initial diagnosis when the clinical course becomes atypical. The coexistence of vitamin B12 deficiency and vitamin D insufficiency may have contributed to the clinical presentation; however, their precise role remains uncertain. This report underscores the value of comprehensive clinical assessment, multidisciplinary collaboration, and careful longitudinal follow…

Learning Points

- Persistent dentinal hypersensitivity that fails appropriate conservative treatment warrants reassessment of the diagnosis.
- Progressive evolution from stimulus-dependent sensitivity to sharp, electric, continuous pain should raise suspicion for neuropathic orofacial pain.
- The emergence of new triggering factors, such as cold-air exposure, should be interpreted in the context of the overall clinical progression rather than in isolation.
- Nutritional assessment, including vitamin B12 and vitamin D status, may be valuable in selected patients with persistent atypical orofacial pain.
- Early multidisciplinary collaboration between dental clinicians and neurologists may facilitate timely diagnosis and reduce unnecessary dental procedures.

References

[1] West NX, Lussi A, Seong J, Hellwig E. Dentin hypersensitivity: pain mechanisms and aetiology of exposed cervical dentin. *Clin Oral Investig.* 2013;17(Suppl 1): S9-S19.
[2] Liu XX, Tenenbaum HC, Wilder RS, et al. Pathogenesis, diagnosis and management of dentin hypersensitivity: an evidence-based overview for dental practitioners. *BMC Oral Health.* 2020; 20: 220.

[3] Aminoshariae A, Kulild JC. Current concepts of dentinal hypersensitivity. *J Endod.* 2021;47(11):1696-1702.
[4] Benoliel R, Gaul C. Persistent idiopathic facial pain. *Cephalalgia.* 2017;37(7):680-691.
[5] Zakrzewska JM, Linskey ME. Trigeminal neuralgia. *BMJ.* 2014; 348: g474.
[6] Craft RM. Modulation of pain by estrogens. *Pain.* 2007;132(Suppl 1): S3-S12.
[7] Fillingim RB, King CD, Ribeiro-Dasilva MC, Rahim-Williams B, Riley JL III. Sex, gender, and pain: a review of recent clinical and experimental findings. *J Pain.* 2009;10(5):447-485.
[8] Julian T, Syeed R, Glascow N, Angelopoulou E, Zis P. B12 as a treatment for peripheral neuropathic pain: A systematic review. *Nutrients.* 2020;12(8):2221.
[9] Stabler SP. Vitamin B12 deficiency. *N Engl J Med.* 2013; 368: 149-160.
[10] Shipton EA, Shipton EE. Vitamin D and pain: Vitamin D and its role in the aetiology and maintenance of chronic pain states and associated comorbidities. *Pain Res Treat.* 2015; 2015: 904967.
[11] Wu Z, Malihi Z, Stewart AW, Lawes CMM, Scragg R. Effect of vitamin D supplementation on pain: A systematic review and meta-analysis. *Pain Physician.* 2016.