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PERSPECTIVES

Neuralgia Inducing Cavitational Osteonecrosis of the Jaw: Scientific Controversy or pseudoscience?

David A Keith

Department of Oral and Maxillofacial Surgery, Harvard School of Dental Medicine, Visiting Oral and Maxillofacial Surgeon, Massachusetts General Hospital, Boston, MA, USA

Correspondence: David A Keith, Department of Oral and Maxillofacial Surgery, Warren 1201, Massachusetts General Hospital, Fruit St, Boston, MA, 02114, Tel + 1 617 726 8222, ext. 2 + 1, Fax +617 726 2814, Email DKeith@mgb.org

Abstract: Jawbone cavities have had a long history in dentistry and for many years they have been described and studied under several different pseudonyms. These cavities are thought to be responsible for a wide range of disease processes and symptoms, including orofacial neuralgia. In this article the existence of jawbone cavities, and their role in the etiology of orofacial pain will be critically examined. The validity and significance of these cavities has been questioned in the literature and found to lack sufficient scientific validation, posing potential harm to patients. Treatments based on this diagnosis have been the subject of legal action brought by patients, by a Medical Insurance Company, which has claimed insurance fraud and by several professional organizations. Ethical, regulatory and financial issues have also been raised. Evidence based diagnostic criteria and clinical trials are needed to determine the scientific validity of this concept. Pain in the mouth and face is best diagnosed using current evidence based diagnostic criteria.

Plain Language Summary: Cavities in the jawbones have been described for many years and have been thought to be responsible for several medical conditions including pain in the face and the jaws. The scientific evidence has not proven that these cavities exist nor that they cause pain. Patients can come to harm if surgical treatment is rendered. Pain in the mouth and face is best diagnosed using current evidence based diagnostic criteria.

Keywords: orofacial pain, neuralgia, bone cavities, jaw, osteonecrosis, ultrasonography, pseudoscience

Introduction

"The field of pain medicine can easily find itself susceptible to 'junk science', alongside any other field in which patient suffering is great, treatment pathways are weak, and financial incentives persist". [1] Cavities in the maxilla and mandible have been described for over a century.[2,3] These cavities were often located in old extraction sites and were accompanied by neuralgia like pain. The concept was popularized by G.V. Black, the "Father of Modern Dentistry" in 1915.[4] Ratner et al expanded this concept by reporting that usually these lesions were not detectable radiographically and that the etiology was an infectious process.[5] In 1992 Bouquot et al proposed a new diagnosis -Neuralgia Inducing Cavitational Osteonecrosis (NICO) and claimed that these cavities were related to many systemic conditions and caused Atypical Facial Pain (AFP) and Trigeminal Neuralgia (TN) like symptoms.[6] This theory has gone through various iterations and in 2013 Lechner et al described Fatty Degenerative Osteonecrosis of the Jaw (FDOJ) and more recently Bone Marrow Defects of the Jawbone (BDOJ) which likewise can be associated with pain.[7,8] The detection of these cavities has been enhanced by two ultrasound devices, initially the Cavitat®[9,10] and more recently the CaviTAU®[10,11] but the findings from these devices have neither been standardized nor validated. This Perspective will review the evidence for the existence of these cavities and specifically their role in the etiology of orofacial pain.

Methods

Sources were obtained from PUBMED and Google searches, the Author's personal collection of articles and the resources referenced. The scientific literature (both peer reviewed and those with no external review), Insurance and

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Legal documents as well as Regulatory action were included. Those selected were considered representative of the data available and were synthesized in narrative form to address the primary intent of this paper.

Neuralgia Inducing Cavitational Osteonecrosis

Diagnosis

NICO jawbone cavities are not detectable by routine intra-oral examination nor are they usually visible on radiographs, although advocates report seeing a regional osteoporotic lesion or an ill-defined radiolucency on radiographs of old extraction sites.[5] Experts in bone metabolism and pathology have postulated that bone cavitations represent normal anatomical marrow spaces that are found routinely on computed tomography (CT) scans of adults, especially in surgical sites located in the posterior regions of the jaws. These cavities have been described in more detail as "merely represent[ing] the normal marrow spaces in the jaws and can be seen on nearly every CT scan in an adult. They actually represent normal anatomy misinterpreted as a disease".[12,13] Pathologic processes such as cysts, tumors and infections present as distinct lesions on imaging studies, however, cavities are not found in otherwise asymptomatic jaw bones. Other technology, therefore, was necessary to demonstrate the presence of these cavities. The Cavitat® Ultrasonograph Bone Densitometry device was promoted and marketed prior to its being cleared by the FDA.[14] The FDA specifically rejected a request to label and market the device as capable of diagnosing NICO or of distinguishing between normal and abnormal bone. It received limited 510(k) clearance as an adjunct to standard x-rays and clinical diagnostic procedures.[15]

In 2002 the medical insurance company, Aetna Inc issued a Clinical Policy Bulletin explaining why they would no longer pay for diagnostic, or treatment related to NICO as it was not a scientifically recognized condition.[15] Cavitat® filed a lawsuit in Denver District Court on 8/12/2004 against Aetna Inc. for not covering a procedure that used its medical device. Aetna Inc. tried to countersue Cavitat® in 2005 claiming the company deceived the Insurer into paying some 400 claims by using incorrect diagnostic codes but the court said Aetna, Inc. "had no standing to file a counterclaim." The company settled this claim and the case was dismissed. On March 1st, 2007, Cavitat® filed for Chapter 11 bankruptcy blaming Aetna Inc. for failing to pay its part of the settlement with Cavitat®. Aetna Inc. eventually settled its law suit with Cavitat®, however, the terms were not disclosed because of a confidentiality agreement between the parties.[15–18] "Due to various ambiguities in daily use and disputes with large insurance companies in the USA, the production and distribution of Cavitat® was discontinued in 2010". [19] Continuing work at the University Dental Clinic in Mainz, Germany, led to the development of a newer version of this device, the CaviTAU® (TAU signifies Transalveolar Ultrasonography), developed by Dr Johann Lechner. It has been available on the market since 2020 and is described as being superior to the Cavitat®.[10,11] CaviTAU® is classified as a medical device under the Medical Device Directive 93/42/EEC which is a council directive of the European Union (EU) that harmonizes the laws relating to medical devices. It has been clinically evaluated in accordance with MEDDEV 2.7/1 Rev.4. 'CaviTAU® is a Class I medical device, but it cannot be used in isolation or alone and is therefore only an aid to medical diagnosis. (EU medical approval: DIMDI VZ BS 914/2020-R). "The device was evaluated and approved. It was adopted on 14th June 1993 and is based on the proposal from the Commission and the cooperation with the European Parliament. The directive aims to ensure a high level of protection for patients and public confidence in the system, as well as to enable the products to be placed on the market in any EU country". [20] The use of transalveolar ultrasound to image alveolar bone has been investigated ex vivo but not in vivo.[21]

Huber et al[19] have published a narrative review of the possible applications of Ultrasound (US) in dentistry and conclude "… that US has been undervalued as a diagnostic tool in dentistry. The new [CaviTAU®] unit offers the opportunity to change this in the future."

The American Dental Association did not endorse the NICO diagnosis and did not give the Cavitat® device its seal of Acceptance.[15] Other diagnostic methods have been used to identify this condition. However, these diagnostic techniques (eg, localization by anaesthetization, the analysis of CCL5 tissue levels) have not been validated[22,23] Although management of chronic orofacial pain is not always successful ascribing the diagnosis to unproven etiologies could obscure treatable causes of the symptoms and lead to further distress for the patient.

Etiology

The etiology of this condition has been variably reported over the years and this shift of emphasis has made it difficult to categorize the process described and to assign it a definitive place in the spectrum of disease processes that are found in the jaws. Frequently infection has been blamed[5,6] characterized as a localized osteomyelitis due to prior trauma which has failed to heal. Fibrous tissue with irregular, nonviable and viable bone spicules is found with mixed amounts of vascularity and chronic inflammatory cells and occasional nerve tissue. Microorganisms are found in nearly all specimens.[5] Bouquot and McMahon in their seminal article[24] describe NICO pathology as ischemic osteonecrosis with '…fatty microvesicles and coalesced pools of liquefied fat (oil cysts), with almost complete loss of adipocyte nuclei… there is pallor, loss of nuclei…with few inflammatory cells, residual hematopoietic/fatty marrow and coalesced dead adipocytes with thrombosed capillaries in surrounding marrow. Higher magnification shows serous ooze (plasmostasis) and dilated capillary with pale staining, degenerated erythrocytes (ghost erythrocytes). On page 1003 of the same article, they state that "The old, overly simplified histopathologic definition of IO (Ischemic Osteonecrosis) as massive loss of osteocytes without pus is now substantially expanded to include specific and often subtle signs of ischemic marrow damage, which may not even include obviously dead tissues'(their emphasis)".Similarly, Lechner et al[25] opine that 'no typical state of FDOJ [is] clearly defined by light microscopy, immunohistochemistry or cytokine patterns' As for neuronal involvement they found in NICO lesions'. Maxillary alveolar nerve coated by lymphocytes. Cross-section of fibrosed neurovascular bundle with no residual veins or nerves and a degenerated artery…Considerable loss of myelin (demyelination) and fibrosis of alveolar nerve. However, on page 1014 of the article they state that "The scarcity of nerves in NICO tissue samples made it difficult to provide direct evidence to dental and alveolar nerves." The proponents appear to be "walking back" their description of the lesions and thus the pathophysiology of the condition. Most recently fatty degenerative osteolysis and osteonecrosis with ischemia, necrotic adipocytes, myxoid degeneration, increased fat cells, and inflammatory cells have been ascribed to the changes observed.[7,8] In addition to trauma and infection,[24] thrombophilia, hypofibrinois as well as antibodies to peripheral nerve myelin, and anticardiolipin antibodies have been cited as causative factors.[26–28] A more recent retrospective and follow-up analysis involved 331 cases of what was then described as chronic fibrosing osteomyelitis of the jaws (CFOJ) in 227 patients. Consistent clinical findings included intractable jaw pain unresponsive to usual therapies, minimal or undetectable radiographic abnormalities on plain films but dramatic radiolucencies detected on cone beam computed tomography, and large cavities that were either empty or filled with blood mixed with lipid globules encountered at surgery. The most common histomorphologic findings were vital lamellar bone, prominent resting and reversal lines, microshards and splaying of trabeculae, rounded trabeculae, marrow fibrosis, and pools of erythrocytes and lipid globules, often together. Moderate to complete relief of symptoms for periods up to 108 months after surgery were reported by 83% of the 70 patients who returned the survey. The response rate and those surveys excluded amounted to a 31% yield which significantly biases the results. On the basis of these findings the Authors determined that CFOJ can be considered a unique entity with consistent clinicopathologic features. suggesting a pathogenesis based on bone marrow ischemia. They concluded that CFOJ can be treated on a rational basis with a justifiable expectation of success and probable cure.[29] Dominiak et al have recently provided a scoping review of FDOJ, the current name used for this chronic, aseptic inflammatory condition that is characterized by molecular disruptions in bone metabolism and necrotic bone marrow within the jawbone cavities. In contrast to the overt clinical signs typically observed in osteopathies, FDOJ frequently presents with a "silent inflammation" phenotype. A total of 36 articles related to FDOJ were identified and included in the narrative analysis. They detected inconsistencies in the findings, selection bias and limited reproducibility, absence of standardized diagnostic criteria, scarce long-term clinical and treatment outcome data and, paucity of consensus on standardized treatment protocols. They correctly summarized the scientific literature and concluded that the majority of studies reviewed were observational or retrospective and emphasized the necessity for controlled clinical trials to validate findings and develop evidence-based diagnostic and therapeutic strategies.[30]

Treatment

The predominant treatment for NICO cavities is bone decortication and curettage of the "diseased" bone marrow. Outcomes are invariably positive but a second surgical procedure may be necessary.[5,31] The criteria for success[5] have been insufficiently described and analyzed, at least by current standards, and have not been validated. The results claimed for TN relief exceeds that reported elsewhere for peripheral neurectomy.[32] No formal analysis of complications or poor

outcomes has been published, however there are anecdotal reports of post-surgical infection, paresthesia, repeated surgical interventions, persistent and increased pain with some patients requiring extensive medical and surgical treatment to repair jaw damage and disfigurement caused by the NICO surgery.[15] Interestingly Dr W Shankland, a strong proponent, stated that "surgical management of…osteonecrosis of the jaws is… more risky than such surgery in other bones simply due to the presence of the inferior alveolar canal, the maxillary sinus and the nasal fossa, and potential damage to adjacent teeth.[15] Marx has reviewed in detail the charts of seven patients who were evaluated using the Cavitat® device, received a diagnosis of NICO and underwent one or more surgeries by one of the proponents of this condition. All were misdiagnosed or incorrectly treated. Several were described as "vulnerable" while others had multiple medical diagnoses which might have contributed to their symptoms. Post operatively two had an increase in their pain and one developed chronic osteomyelitis of the jaw bone.[12]

The American Association of Endodontists Position statement "…cannot condone surgical interventions intended to treat suspected NICO lesion…". 'Without a confirmed clinical diagnosis of localized bone pathosis, aggressive and invasive procedures are not warranted. Such interventions may have no effect or may even worsen the pain by increasing sensitization of the central nervous system.[33] With the exception of studies involving the use of neuromodulation technique[34] and anti-coagulation therapy,[35] NICO patients in all studies included in the PRISMA review[36] had been treated by surgical means. As stated above the majority of studies have been observational or retrospective and have lacked controls so therapeutic strategies have not been validated.[33]

Jaw Bone Cavities and Pain

Many reports have stated that jaw bone cavities are accompanied by neuralgia like pain however 'patients often have difficulty describing and localizing the pain, which may spread locally across time or refer to distant sites.[5,22] Originally the pain was thought to be due to intraosseous fluid dynamics and inflammatory mediators rather than to damaged nerves.[8] The basic tenets of the etiology of NICO pain have been widely represented (Figure 1):

- Pressure in the marrow cavity
- Inflammation in the jaws
- Necrotic bone
- Demyelination of the nerves

Many dissenting opinions regarding NICO and its relationship to pain have been published.[37–40] In particular, Zuniga made a detailed appraisal in which he concluded that "The proposed cause and effect relationship between NICO cavitations and facial pain is speculative at best and has not been defined by evidence-based scientific criteria". Based on 2000 criteria he argued that "Trigeminal Neuralgia, Atypical Facial Pain, Atypical Odontalgia, and Burning Mouth Syndrome met important criteria that included clinical data to support the pathophysiology and diagnostic criteria, scientifically sound data that provide predominantly central nervous system mechanisms and clinical pain data to determine which criteria discriminate between the pain sub-groups".[41] The latest classification of Orofacial Pain[42,43] has defined 3 types of persistent facial pain; idiopathic facial pain, idiopathic dentolalveolar and post traumatic trigeminal pain. Their common features are persistent pain with variable features, daily occurrence, 2+ hours, 3+ months, poorly localized, dull, nagging, occasionally sharp, radiating with variable intensity. These criteria would seem to fit the "neuralgia" associated with NICO most closely.[5,6,8] The prevalence of NICO in the general population has not been determined, whereas the prevalence of the other similar pain diagnoses have been extensively studied. The numbers of NICO patients reported from University and Private Practice facilities is much higher than that of other neuralgic pains is likely due to the imprecise definition of this condition.[22,44]

Sekundo et al[36] have provided a more recent and structured PRISMA review to assess the etiologic factors, proposed diagnostic means and treatment strategies for NICO. Twenty-nine articles fulfilled the inclusion criteria. No randomized controlled trials could be identified and of the included studies, all were observational in design, 22 studies were retrospective in nature, 6 were prospective, and 1 did not report its time frame. The etiologic causes varied widely, no gold standard diagnostic mean could be identified, treatment was most often performed by surgical curettage of the affected bone, and pain

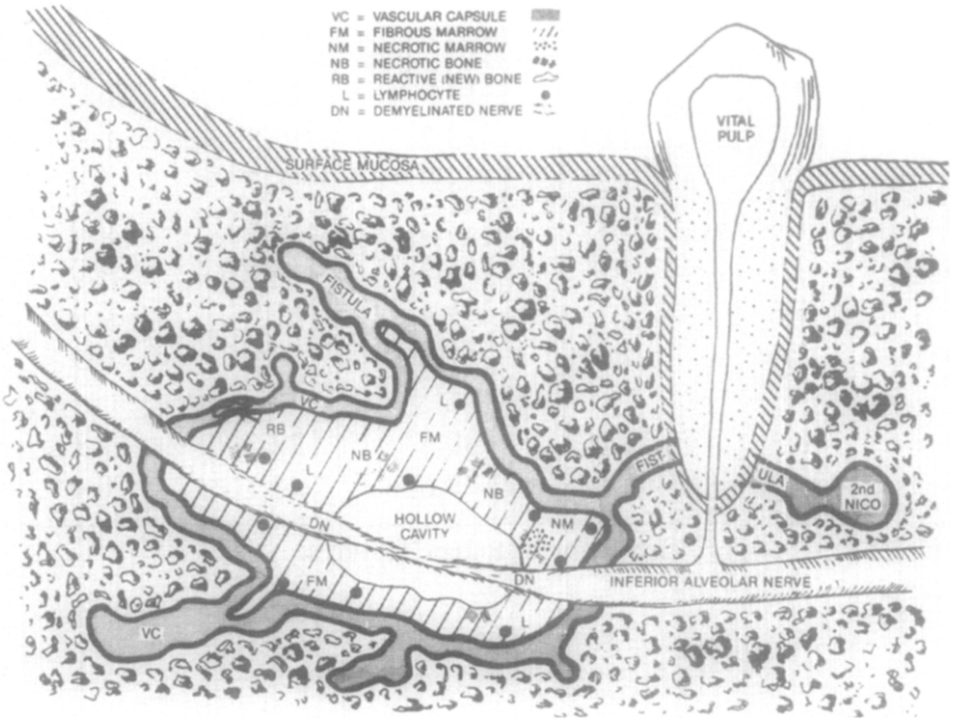

Figure 1 Depiction of the etiology of NICO lesions and their relationship to the inferior alveolar nerve. Reprinted from *Oral Surg Oral Med Oral Pathol*. Volume 73. Bouquot JE, Roberts AM, Person P, Christian J. Neuralgia-inducing cavitational osteonecrosis (NICO): osteomyelitis in 224 jawbone samples from patients with facial neuralgia. 307–319. Discussion 73:319–320, copyright 1992, with permission from Elsevier.[6] A colored copy of this image is available @ https://www.tmjdental.com/natural-holistic-dentistry/cavitations/.

outcomes varied widely in the amount and length of time of pain relief. Significant facial pain remission was reported in 66–100% for periods varying between 2 months to 18 years, whereas little or no relief and recurrences were reported in up to ⅓ of cases. All investigations were rated as poor quality because of the high risk of bias and non-transparent reporting. They concluded that evidence concerning the etiology, diagnosis and treatment of NICO is poor.[36]

The etiology of NICO pain as suggested in the Figure 1 is thus called into question;

- Pressure in marrow cavity has never been studied
- Inflammation is common in the jaws (See below)
- Necrotic bone is disputed
- Demyelination of the nerves is "difficult to provide direct evidence." Current concepts of the pathophysiology of idiopathic and traumatic trigeminal neuropathies, which most closely represent the symptoms attributed to NICO, are strongly based on CNS mechanisms.[36,41–43]

Jaw Cavities, Pain and RANTES/CCL5

Lechner et al have published extensively suggesting that the key pathogenetic element of NICO is 'the chemokine RANTES/CCL5 in up to 60-fold overexpression.[7,8,11,23,26] One study investigated the role of RANTES/CCL5 in painful jawbone lesions from 15 patients with AFP and TN. The pain diagnosis was made by neurologists, pain specialists and physicians. The inclusion criteria were therapy resistant pain that was clinically similar to AFP and TN. Imaging studies and CaviTAU® imaging was available to make the diagnosis. A matched control group undergoing "normal dental surgery" was identified. The study group had a mean 30-fold overexpression of RANTES/CCL5 levels compared to the control group. All patients with pain were treated surgically. The major pathological findings were described as ischemia, myxoid degeneration, increased fat cells, and necrotic adipocytes. The results demonstrated a 21 month pain free period after treatment with an 88% mean percentage reduction in pain. The presence of inflammatory cells in only 1/15 specimens led the authors to conclude that this was a "silent inflammation". They stated that the overexpression of

RANTES/CCL5 in these jawbone lesions might induce an immunological reaction, may cause neuronal hyperexcitability or desensitize opioid receptors as potential causes for the resultant reduction in pain[23] In an illustration of the potential sources of inflammation the sample is taken from the edentulous area in the left maxilla from which a tooth had previously been extracted. This area is close to the oral mucosa, the sinus cavity, and the periodontal ligament of the adjacent teeth and inflammation is to be expected.

Orthopedic Experience

Osteonecrosis, also called Avascular necrosis, Aseptic necrosis, or Ischemic necrosis of bone can happen to any bone but most often it develops in the ends of long bones, such as the Femur and Humerus.[45,46] In the jaw bones "Phossy jaw",[47]"Radium Jaw"[48] Osteoradionecrosis,[49] Medication related osteonecrosis of the jaws[50,51] have been well described and all have discrete etiologies, recognized signs and symptoms and are not always painful. NICO is not one of these conditions.

Conflicts of Interest

Financial interest appears to have been present in several aspects of this story. Aetna stated that the device's prime promoter and Oral Pathologist, Dr J E Bouquot served on the company's Scientific Advisory Board. He owned and operated the for profit Head and Neck Diagnostics of America, was the prime source of NICO diagnosis and significantly benefited from the volume of pathology specimens examined. The clinicians involved have been accused of backfilling or verifying the diagnosis with the Pathology results -a form of "after the fact justification" for their diagnosis and treatment.[15] In the Cavitat® promotional literature Dr W. Shankland promised that a practitioner could generate $115k in annual revenue for a 427% return on investment. "…[Y]ou can make money with these things". He was a member of Cavitat's Scientific Advisory Board and was a frequent lecturer at their courses.[15] The CaviTAU® device was developed by Dr Johann Lechner, who is one of the Managing Directors of the manufacturing company.[11]

It is highly unusual to include and recommend a commercial website (www.cavitau.de). in a scientific article.[19] The first Author, Robert Huber, is listed on the CaviTAU® website as CEO, Co-Founder-Digital Dental & Healthcare Technology GmbH & Co. KG. Dr Lechner is thanked for supplying the figures. Dr Notter lists his affiliation with the Clinic of Integrative Dentistry, Munich where Dr Lechner is Head, and they have published several papers together. None of these conflicts are disclosed and point to the close professional relationship between the Authors' academic and clinical work and the commercial enterprise. In a scientific "Review" article such as this opposing opinions should be discussed so that readers can draw their own conclusions, but they are not mentioned. As written this paper reads more like a marketing promotion than a scientific article.

Exclusivity

Scientific data and treatment options should be available to all, verifiable, reproducible and not confined to any one "elite" group. NICO diagnoses were largely restricted to a small group of clinicians, and one Pathology Laboratory.[15] Scientific data has predominantly been published by the Bouquot and Lechner groups and results have depended on the skills of a limited number of practitioners. 'The success of such surgery is by no means guaranteed and it depends on the technique and the skill of the dentist doing the surgery...'[23]

Pseudoscience

As described above many of the observations and studies offered in support of the NICO and related diagnoses have lacked scientific rigor in terms of diagnosis, study design and result reporting. These defects are clearly acknowledged by some authors stating that this was a "real world study with individual treatment decisions being made exclusively by the dentists."[8] And additionally, the effects of several potentially confounding factors were not assessed. There are many guidelines for conducting surgical studies[52] which specify the criteria for accurately assessing outcomes. The essential elements include defining a research question, rigorous inclusion and exclusion criteria and meaningful outcome measures. It is also important to calculate a sample size that accounts for expected attrition and dropouts. The observers and patients need to be blinded as far as that is possible.

Control groups need to be defined and in NICO studies patients undergoing no treatment or an alternative nonsurgical intervention would be appropriate. "Sham surgery" controls would be considered unethical. It is important to note that surgical treatment outcomes can be significantly influenced by the placebo response consisting of the placebo effect together with the natural history of the condition, regression to the mean, response bias and other factors. This is particularly true of patients in distress desperately seeking relief of their pain.[53]

NICO has undergone several name changes which has been a feature of medicine since early times. However, renaming a condition does not improve its diagnostic clarity. As an example, neurasthenia, or nervous exhaustion, was thought to be the result of a fast-paced urban life through the late 1970s. "Its symptoms are now largely, to no real advantage, retitled as chronic fatigue syndrome."[54] Analogous to the difference between the common acute orofacial pain and the less common chronic orofacial pain, another condition Chronic Lyme Disease is poorly characterized in comparison to Lyme disease itself. In the former condition "Patients are fooled, taken advantage of, betrayed. Patients are victims of their symptoms as well as victims of unnecessary and useless treatments and associated adverse events. It is unfortunate that some physicians, eager for fame and increased means, are playing along as this is highly detrimental to patients".[55]

NICO and its counterparts have found proponents among clinicians over many years as a result of the lack of diagnostic precision in the field of chronic orofacial and the equivocal results that have resulted. In particular dentists who are trained to think procedurally are attracted by a surgical solution to a seemingly intractable problem. Patients, frustrated by their continuing discomfort, the apparent inability of their clinicians to both understand and treat their pain also seek a definitive solution. A surgical "fix" is attractive especially if a putative "lesion" is identified, more so if it is bolstered by pathologic diagnoses, and biomarkers. Over this same period of time our knowledge of chronic pain mechanisms, the central nervous system connections and our diagnostic criteria for orofacial pain have vastly improved. This not to say that much still needs to be figured out in all these areas before we can accurately diagnose and reliably treat these conditions. The NICO proponents continue to espouse a local cause for what is most likely a central nervous system problem.

Despite following the PRISMA guidelines and providing detailed analyses of the published papers from several reputable databases the Sekundo article[36] came under critical review in Letters to the Editor.[56–59] The arguments contained criticism of the Authors for their "lack of experience of the condition", "mixing up the developing results from 1979 to the present (a "work in progress")", being "more judgmental than scientific", "lacking the scientific honesty that is necessary for an open discussion.", and '…written by professionals who have never published anything on their experience with chronic facial pain disorders.

False Claims

Cavitat® stated that "the green, yellow to red escalating color changes on the screen have not been FDA tested related to normal or pathologic bone". However, Cavitat® falsely claimed full endorsement and government-granted monopoly, as well as IRB approval. Critics have stated that Cavitat® was "mislabeled, misbranded and illegally promoted for off label usesand 'used in unlawful human clinical research.'[15] A proviso on the Company's website states that "CaviTAU® does not provide a diagnosis, but rather identifies differences in the density of the jawbone." However later on, in answer to the question "Why do I need CaviTAU® in my practice?' it is stated that "CaviTAU® helps you close daily application gaps:1. The diagnostic gap: In the case of chronic inflammatory, degenerative bone changes, as these cannot be made visible with standard radiological diagnostics".[11] Their website states "Our first laboratory detection of this inflammatory messenger in the jawbone is the contradiction-free proof of a holistic-systemic signaling effect from the jawbone with modern immunological methods. Since these articles have been peer reviewed by several experts and included in the PubMed or ScienceDirect (Elsevier) medical library for their academic rigor, these articles are to be considered accepted science and natural science undisputed part of medical progress". [11] The statements in another article[8] were challenged in a Letter to the Editor[60] as it supported "…a widely discredited claim that 'Bone marrow defects' cause facial neuralgia…". As discussed above there is little evidence to support this theory. Furthermore, the article claimed that "There is growing evidence…" for its support is also not demonstrated in the literature.[61] The Authors replied, 'Open-mindedness and pattern recognition are important skills in research, targeting results that do not fit into a prefigured concept might be less helpful."[62]

Current Status

Although Cavitau is predominantly used in Europe[63] a recent Google search of the Web (accessed 3/18/2025) reveals several hundred hits in the USA using the words Neuralgia Inducing Cavitational Osteonecrosis and its synonyms, Jawbone cavitations, Cavitat®, CaviTAU® and Surgical treatment demonstrating that the idea continues to be actively marketed, predominantly from Holistic, Integrative, Biological and Homeopathic Dental Practices. It is likely that practitioners continue to promote these diagnoses and treatments NICO diagnoses because Orofacial pain diagnoses are, in general, poorly understood and treated. Thus, patients become dissatisfied with their treatments and frustrated by lack of understanding by their medical and dental professionals and seek out alternatives therapies which are bolstered by the use of diagnostic devices, the results of placebo responses and aggressive marketing efforts. The International Academy of Biological Dentistry and Medicine has a "Coming soon!" notice on its website (accessed 8/18/2025) promoting a 'Jawbone Osteonecrosis (Cavitation) Essentials' section.[64]

Notable on some practice websites is the use of Ozone, Hyperbaric Oxygen and Autologous Platelet-Rich Fibrin as adjunctive treatments to 'careful but thorough surgical clearance of the affected areas.[65] Confronted by a patient with chronic orofacial pain the clinician is faced with a dilemma as to whether the cause is local or stems from CNS pathology. Dentists are tasked with uncovering local disease and have the tools and experience to do this. Neurologists and other Pain experts rely on Dentists' skills before embarking on other diagnostic tests and management strategies. The most recent diagnostic classifications[42] provide an improved but by no means exhaustive description of the idiopathic and post traumatic trigeminal neuropathies as a guide. Imaging studies have provided extensive insights into many OFP conditions, such as Temporomandibular Disorders but the results need to be interpreted with caution and correlated with the clinical history and examination. Failure to do so can lead to overdiagnosis and unnecessary treatments.[1] Interdisciplinary care is always appropriate for these chronic pain conditions.

Conclusion

The existence of bone cavities of the jaws as pathologic entities and specifically their role in the etiology of OFP has been questioned across many decades. Devices have been developed to prove their existence but have failed scientific and regulatory scrutiny. Many etiologies have been advanced but none have been conclusively substantiated. Histopathologic evidence has been largely entrusted to one Laboratory with others questioning the diagnosis offered. Perhaps there is a grain of truth in the notion, however at this time the matter remains unproven. Sekundo et al concluded that the etiology, diagnostic tools and treatment strategies for NICO / FDOJ need to be validated by properly designed clinical trials addressing possible sources of bias and having clearly defined interventions, and outcomes, after approval by institutional review boards.[36] Greene et al have stated that the related field of TMD "is replete with a variety of so-called diagnostic instruments and procedures, which have not been tested for diagnostic validity; these instruments and procedures, through misuse, are capable of complicating a true diagnosis of patients who present with symptoms, while also creating new patients by finding so-called abnormalities in healthy subjects". [66] This applies to NICO as well.

Abbreviations

AFB, Atypical Facial Pain; BMDJ, Bone Marrow Defects of the Jaw Bones; CCL5, Chemokine (C-C motif) ligand 5; FDOJ, Fatty Degenerative osteonecrosis of the Jaws; NICO, Neuralgia Inducing Osteonecrosis; OFP, Orofacial Pain; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analysis; RANTES, Regulated upon activation, normal T cell expressed and secreted; TMD Temporomandibular Disorder; TMJ Temporomandibular Joint; TN Trigeminal Neuralgia.

Disclosure

The author reports no conflicts of interest in this work.

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