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Posttraumatski stresni poremećaj i orofacijalna bol

Posttraumatic Stress Disorder and Orofacial Pain

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Sažetak
Od kronične orofacijalne boli često pate pacijenti s PTSP-om. Moramo istaknuti da se istodobno bilo koji patološki proces orofacijalnog područja može uplesti u emotivnu interpretaciju boli te kod takvih pacijenata potaknuti niz katastrofičnih reakcija povezanih sa simptomima iz skupine onih za posttraumatski stresni poremećaj. Tako izazvani bolni podražaji mogu se pojaviti i nakon što je primarno oštećenje prestalo djelovati i izazivati bol izvan anatomskog mjesta na kojemu se ozljeda dogodila. Kroničnu orofacijalnu bol i PTSP dijagnosticiramo na temelju subjektivnog iskaza, a to se redovito događa u kontekstu društvenih interakcija između pacijenata, liječnika, medicinskog osoblja ili istraživača, što otežava standardiziranje rezultata i uključuje mnoge kulturološke fenomene.

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Ključne riječi
bol lica; post-traumatski stresni poremećaj; odražena bol

Uvod

Posttraumatski stresni poremećaji povratna su, nametljiva sjećanja na teški traumatizirajući događaj koji je nastao nakon izloženosti ili svjedočenja iskustvima koja ugrožavaju život. PTSP se svrstava u skupinu anksioznih poremećaja, obilježava ga odgođeni ili produljeni odgovor na stresogeni događaj, a uključuje i neurobiološku disregulaciju i psihološku disfunkciju. Simptome PTSP-a dijelimo u tri skupine (tri klastera):

1. ponovno proživljavanje traume,
2. izbjegavanje i povlačenje
3. pojačana pobudljivost (1,2).

Kronična orofacijalna bol nastaje bez jasnoga organskog uzroka, traje dulje od tri mjeseca i, za razliku od akutne, ne upozorava, dakle, nema zaštitnu funkciju (3).

Pacijenti s PTSP-om često pate od psihogene boli, no nju ne potiču štetni podražaji ili oštećenja živčanog sustava. Istaknimo da se bilo koja ozljeda orofacijalnog područja može uplesti u emotivnu interpretaciju boli i kod pacijenata s posttraumatskim stresnim poremećajem potaknuti niz katastrofičnih reakcija povezanih sa simptomima onih iz skupine za PTSP. Tako izazvani bolni podražaji mogu se pojaviti i nakon što je primarno oštećenje prestalo djelovati i izaziva-

Introduction

Posttraumatic stress disorder (PTSD) is a recurrent, intrusive memory of an exposure to a burdensome traumatizing situation or witnessing of life-threatening events. PTSD is classified in the group of anxiety disorders, and its characteristic is a delayed or protracted response to a stressful event and it equally includes neurobiological dysregulation and psychological dysfunction. Symptoms of PTSD are divided into three groups (clusters): 1. reliving the trauma, 2. avoidance and withdrawal and 3. increased arousal (1, 2).

Chronic orofacial pain is pain without a clear organic cause, lasts more than three months and unlike acute pain it has no protective, warning function (3).

Patients with PTSD often suffer from psychogenic pain; it is not encouraged by harmful stimuli or damage of the nervous system, on the other hand, any injury of the orofacial area can get involved in emotional interpretation of pain and trigger a series of reactions associated with the PTSD group of symptoms in patients with PTSD. Painful stimuli caused in this way may occur after the primary cause ceased, and because of convergence can cause referred pain outside of the anatomical site where the primary injury occurred. Chronic orofacial pain and PTSD are diagnosed on the basis of sub-

ti bol izvan ozlijeđenoga anatomskog mjesta. Kroničnu orofacijalnu bol i PTSP dijagnosticiramo na temelju subjektivnog iskaza, a redovito se događaju u kontekstu društvenih interakcija između pacijenata, liječnika, medicinskog osoblja ili istraživača, što otežava standardiziranje rezultata i upleće mnoge kulturološke fenomene (4).

Anatomija i patofiziologija

Anatomski je interna reakcija između orofacijalne boli i PTSP-a povezana s trigeminalnim živcem. Neugodne trigeminalne senzacije bitne su kao upozorenje na ozljede i potencijalno opasne tvari te pokretanje protektivnih refleksa i ponašanja. Neuroni važni za prijenos podražaja i nastajanje neurogene upale su C i A delta vlakna te u manjoj mjeri A beta vlakna. Signal se prenosi u neurone drugog reda u nukleusu kaudalisu trigeminalnih ganglija, zatim dalje u ventroposteriornu talamičku jezgru i moždanu koru. Niz je zajedničkih neuroimunosnih putova između senzoričkih putova trigeminusa i patofizioloških procesa PTSP-a (5). Najvažniji su posredovani CGRP-om (Calcitonin Gene Related Peptide), tvari P, dušikovim oksidom i receptorima TRPV1 (transitient potential vanilloid receptor) (6).

PTSP izaziva stresogeni događaj s hiperkortizolemijom kao prvim metaboličkim odgovorom koji uzrokuje plastične promjene glukokortikoidnih i mineralnokortikodnih receptora hipokampusa, izravno utječe na hipotalamno-hipofizno-adrenalnu (HHA) osovinu, s posljedičnom redukcijom dendritičnog grananja, smanjenom neurogenezom i odumiranjem neurona hipokampusa. Procesi u amigdalama, hipokampusu, hipotalamusu, periakveduktalnoj sivoj tvari i talamusu odgovorni su za niz psihičkih adaptabilnih reakcija na stres koji se sinkroniziraju s tjelesnim fiziološkim odgovorima (7,8).

Periferne stanice i stanice središnjega živčanog sustava dijele receptore neurotransmitera, neuropeptida i hormona. Tako je omogućena komunikacija povratnom spregom između periferije organizma i CNS-a, uključujući i međudjelovanje autonomnoga živčanog sustava na emocije i kognitivne funkcije. Informacije o procesima na periferiji organizma prenose se na CNS i pokreću reakcije koje se očituju prilagodbenim i metaboličkim promjenama. Obrnuto, psihičke i kognitivne reakcije izazivaju promjene na periferiji koje se mogu očitovati i kao psihosomatske bolesti (9).

Eksperimentalni modeli facijalne boli

Interakcija stresa i periferne upale može se ilustrirati modelom temporomandibularne disfunkcije, boli i stresa na štakorima. Upala masetera neće izražavati bol ako je upalni citokin interleukin-6 (IL-6) samo lokalno izražen lokalno izazvanom upalom mišića, no ako se stresom inducira njegova povišena koncentracija u serumu koji potječe iz CNS-a, pojavljuje se bol (10).

Središnji mehanizmi trigeminalnih putova mogu prouzročiti upalu i bol i izvan upaljenog područja. Smanjenjem

jective testimony and this regularly occurs in the context of social interaction between patients, doctors, medical staff or researchers making it difficult to standardize the results and introduces many cultural phenomena (4).

Anatomy and pathophysiology

Anatomically speaking, interaction between orofacial pain and PTSD is related to the trigeminal nerve. Unpleasant trigeminal sensations are important as a warning for injuries and potentially dangerous substances and for initiating protective reflexes and behavior. The neurons essential for the transmission of stimuli and the formation of neurogenic inflammation are the C and A-delta fibers, and to a lesser extent A-beta fibers. The signal is transmitted to the second order neurons located in the nucleus caudalis of the trigeminal ganglion, onward to the ventroposterior thalamic nucleus and then to the cerebral cortex. There are a number of common neuroimmunological pathways between the trigeminal sensory pathways and pathophysiological processes in PTSD (5). The most important pathways are mediated by the CGRP (Calcitonin Gene Related Peptide), substance P, nitric oxide and TRPV1 (transient receptor potential vanilloid receptor) receptors (6).

PTSD is caused by a stressogenic event, with hypercortisolemia as an early metabolic response produced by plastic changes in glucocorticoid and mineralocorticoid receptors of the hippocampus, directly affecting the hypothalamic pituitary adrenal (HPA) axis, with the consequent reduction of dendritic branching, reduced neurogenesis and the death of neurons of the hippocampus. The processes in the amygdalae, hippocampus, hypothalamus, periaqueductal grey matter and the thalamus are responsible for a series of adaptable psychological reactions to stress which sync with physical physiological responses (7, 8).

Peripheral cells and central nervous system cells share the neurotransmitter receptors, neuropeptides and hormones. This enables feedback communication between the periphery of the organism and the CNS, including the interaction between the autonomic nervous system and emotions and cognitive functions. Information about the processes at the periphery of the body is transmitted to the CNS and triggers reactions that are manifested in adaptive and metabolic changes. Conversely, psychological and cognitive reactions cause changes in the periphery, which can manifest themselves as psychosomatic diseases (9).

Experimental models of facial pain

Interaction of stress and peripheral inflammation can be illustrated with a model of temporomandibular dysfunction, pain and stress in rats. Masseter inflammation will not express pain if the inflammatory cytokine interleukin-6 (IL-6) is only locally pronounced, by locally induced inflammation of the muscle, but if the stress induces an increased concentration of IL-6 in the serum originating from the CNS, pain occurs (10).

Central mechanisms of trigeminal pathways can cause inflammation and pain outside of the inflamed area. The re-

koncentracije CGRP-a u stražnjem rogu produžene moždine trigeminalnog puta, smanjuje se i pojačana osjetljivost na bol u upalnoj i neuropatskoj boli. Dalje centralni putovi neurona CGRP ulaze u amigdale gdje bol dobiva asocijativnu komponentu, bitnu za procese učenja i straha. Projekcije u jezgre talamičkog trakta i inzularni kompleks potiču autonomne, motorne i emocionalne odgovore vezane za bol. Blokada receptora CGRP-a u navedenim područjima blokira i reakcije vezane za emotivnu nadogradnju boli (11). TRPV1 (transitient potential vanilloid receptor) receptori su neurona koji oslobađaju CGRP i zato su važni u upalnim i neupalnim bolnim stanjima. Jednostrano izazvanom upalom i hiperalgezijom maseteričnog mišića povećava se broj receptora TRPV1 i CGRP mRNA u upaljenom području. Hiperalgezija bez upale i povišenja koncentracije TRPV1 i CGRP mRNA pojavljuje i u simetričnom kontralateralnom području, te se kontralateralna bolnost može se spriječiti blokiranjem funkcije TRPV1 u hipokampusu, što upućuje na ulogu hipokampalnih receptora TRPV1 u etiopatogenezi upalom uzrokovane facijalne boli i izvan upalnog područja (12). CGRP trigeminusa uključen je tako u patofiziologiju boli na svim razinama – od periferije do centara u mozgu, te ponovno prema periferiji preko silaznih inhibitornih mehanizama. Tako su integrirane amigdale, hipotalamus i inzularni kompleks koji procesuira emotivnu strukturu boli koja uključuje i reakciju na strah i stres. Daljnjom obradom svjesnoga u kortikalnim centrima, bolni podražaj postaje potpuno obrađen (13).

PTSP i facijalna bol kao klinički entiteti

Psihoimunoneurologija osnova je vrlo kompliciranih evolucijskih i interpersonalnih odnosa. Bol i psihotrauma sastavni su dio ljudske evolucije. Bol je primarni osjet koji se teško zaboravlja i njegovom manipulacijom stvaraju se novi svjesni ili nesvjesni kognitivni obrasci. Za razumijevanje boli i PTSP-a nužna je spoznaja da se pod utjecajem kulture bol može upletati u biološke procese (14, 15).

Orofacijalna bol jedan je od najčešćih oblika boli glave i vrata i smatra se da u SAD-u i Europi čini 40 posto svih kroničnih bolnih stanja (12).

Bolesnici s PTSP-om – ratni i mirnodopski, osam puta češće boluju od nekog oblika kronične boli i dva puta češće nego bolesnici s dijagnosticiranom tjeskobom. Obrazac reakcije na bol u slučaju PTSP-a drukčiji je negoli u zdravoj populaciji, a čine ga povišen prag boli, povećana osjetljivost na bol iznad praga boli, nepromijenjena osjetljivost na sumaciju više uzastopnih nebolnih podražaja i nepromijenjen prag za osjet toplo/hladno. Takav obrazac boli PTSP-a upućuje na očuvane putove senzitizacije drugoga i trećeg neurona kralježnične moždine i središnjega živčanog sustava te izostanak senzorne polineuropatije. Navedeni uzorak dokazuje da je glavnina doživljaja boli kod pacijenata s PTSP-om uvjetovana emotivnim obrascima doživljaja boli (16).

Kod 88 posto takvih bolesnika glavobolja je najčešći simptom, a ako je uzročnoj traumi bila pridružena i fizič-

duction in concentration of CGRP in the dorsal horn of the medulla oblongata of the trigeminal pathway reduces the increased sensitivity to inflammatory and neuropathic pain. Further the central pathways of the CGRP neurons go in the amygdalae where the pain gets the associative component, which is essential for learning processes and fear. Projections in the thalamic tract nucleus and insular complex encourage autonomous, motor and emotional responses related to pain. Blockade of the CGRP receptors in these areas also blocks the reactions related to the emotional upgrade of pain (11). TRPV1 (transient receptor potential vanilloid receptor) receptors are neurons that release the CGRP and as such play a key role in inflammatory and non-inflammatory pain conditions. Unilateral induced inflammation and hyperalgesia of the masseter muscle increase the number of TRPV1 receptors and CGRP mRNA in the inflamed area. Hyperalgesia without inflammation and without increasing the concentration of TRPV1 and CGRP mRNA occurs in a symmetrical contralateral area, and this contralateral pain can be prevented by blocking the function of the TRPV1 in hippocampus indicating a role of the hippocampal TRPV1 receptors in the etiopathogenesis of the inflammation-induced facial pain outside of the inflammatory area (12). Trigeminal CGRP is involved both in the pathophysiology of pain at all levels from the periphery to the center in the brain, and back to the periphery via the descending inhibitory mechanisms. Thus, amygdalae, hypothalamus and insular complex are integrated in processing emotional pain structure that includes a reaction to fear and stress. Further conscious processing in cortical centers fully processes the pain stimulus (13).

PTSD and facial pain as clinical entities

Psychoneuroimmunology is the basis of very complicated evolutionary and interpersonal relationships. Pain and psychological traumas are an integral part of human evolution. Pain is the primary sensation that is hard to forget and its manipulation creates new conscious or unconscious cognitive patterns. For the understanding of pain and PTSD it is necessary to realize that influenced by culture pain can interfere with biological processes (14, 15).

Orofacial pain is one of the most common forms of head and neck pain and it accounts for 40% of all the chronic pain conditions in US and Europe (12).

Patients with PTSD, war and peacetime, are eight times more likely to suffer from some form of chronic pain, and two times more often than patients diagnosed with anxiety. The pattern of the reaction to pain in patients with PTSD is different than in the normal population, and it consists from an elevated pain threshold, increased sensitivity to pain above the pain threshold, unchanged sensitivity to summation of multiple consecutive non-painful stimuli and unchanged threshold for sensing hot/cold. This kind of pain pattern in patients with PTSD suggests preserved sensitization pathways of the second and third neuron of the spinal cord and the central nervous system and the absence of sensory polyneuropathy. This sample proves that the majority of pain perception in PTSD is conditioned by emotional patterns of pain perception (16).

ka, samo devet posto takvih pacijenata s PTSP-om smješta kroničnu bol na ozlijeđeno mjesto, što sugerira da primarna ozljeda nije presudna za pojavu kronične boli, nego se ona stječe naknadno (4). Glavobolja je i najčešći bolni sindrom bračnih partnera pacijenata oboljelih od PTSP-a (17).

Traumatski događaj u anamnezi navelo je oko 50 posto pacijenata s orofacijalnom boli, a od 15 do 24 posto njih zadovoljava kriterije za PTSP (18). Istraživanja o komorbiditetu otežana su zbog niza simptoma sadržanih u klasterima PTSP-a i jer je komorbiditet posttraumatskoga stresnog poremećaja s drugim psihičkim bolestima i do 80 posto (2). Pri raščlanjivanju simptoma PTSP-a sadržanih u glavnim skupinama simptoma i povezivanja s orofacijalnom boli, uočava se da su glavni simptomi PTSP-a koji pojačavaju probleme vezane za kroničnu orofacijalnu bol izbjegavanje suočavanja s problemima, tupost i disforija, a najmanji utjecaj imaju uznemirenost i ponovno proživljavanje traumatskih sjećanja. Suprotno uvriježenom mišljenju, poremećaji spavanja ne utječu izravno na kroničnu orofacijalnu bol kod pacijenata s PTSP-om, nego je ta povezanost nađena samo preko uznemirenosti kao glavnog simptoma (18).

Danas se komorbiditet PTSP-a i orofacijalne boli opisuje modelom uzajamnog odnosa boli i PTSP-a koji je prvi postavio Sharp. Model se grafički prikazuje kao niz povezanih i uzajamnih simptoma i obrazaca bolesti. Komponente tako prikazanog modela – prekomjerno usmjeravanje pozornosti, anksiozna osjetljivost, podsjećanje na traumu, izbjegavanje, depresija, pogrešna percepcija boli i manjak kognitivnih sposobnosti za adaptivnu strategiju – uzajamno su ovisni sa stresom, kroničnom boli, posttraumatskim stresnim poremećajem i invalidnošću (u smislu imobilizacije). Svaka od njih može se pogoršati ili potaknuti pogoršanje i PTSP-a i kronične boli (19).

Emocionalna interpretacija boli pacijenata s PTSP-om objašnjava se dvama mehanizmima: disocijacijom i anksioznom osjetljivošću. Disocijacija je psihološki obrambeni mehanizam pri kojem su identitet, sjećanja, ideje, osjećaji ili percepcije odvojeni od svjesne spoznaje i ne mogu se dozvati u sjećanje ili voljno doživjeti. Disocijativni mehanizmi povezani su s poremećajima pažnje kako u mirnom tako i u emocijama nabijenom stanju. Anksiozna osjetljivost strah je da će promjene povezane s tjeskobom, poput lupanja srca, znojenje, derealizacije, mišićne napetosti i glavobolje, potaknuti negativne socijalne, tjelesne ili mentalne posljedice. Može se pojaviti i strah od anksioznosti ili boli, strah da su navedeni simptomi siguran put u ludilo. Povećani intenzitet boli kod pacijenata s PTSP-om dio je emotivnog obrasca prema kojemu se bol shvaća kao nerealna opasnost i/ili je podsjećanje na traumu koja je izazvala posttraumatski stresni poremećaj. Tako bolni podražaj služi kao *okidač* za uznemirenost i poticanje katastrofičnog odnosa prema boli, pa samim time i za strah od boli. Rezultat je mnogo jača bol nego u kontrolnoj populaciji (4).

Dvije trećine pacijenata s PTSP-om ima još jednu psihijatrijsku dijagnozu, a najčešće su to depresivni, anksiozni i somatoformni poremećaji te bolesti ovisnosti (2). Komorbiditet umnogome povećava mogućnost povezivanja s drugim simptomima iz klastera simptoma i ti mnogostruki mehaniz-

In 88% of patients with PTSD headache is the leading symptom of pain, and if the causal trauma of PTSD was associated with a physical trauma, only 9% of such patients referred to chronic pain in previously injured area, which suggests that the primary injury is not crucial for the occurrence of chronic pain but it appears subsequently (4). Headache is the most common pain syndrome of marriage partners of patients with PTSD (17).

About 50% of patients with orofacial pain have a history of a traumatic event and 15-24% of them meet the criteria for PTSD (18). Research on comorbidity is more difficult due to a number of symptoms contained in PTSD clusters and because comorbidity of PTSD with other mental illnesses is up to 80% (2). When analyzing the symptoms of PTSD contained in the main groups of PTSD symptoms and connecting them with orofacial pain, it can be seen that the main symptoms of PTSD that enhance the problems associated with chronic orofacial pain are avoidance of dealing with problems, numbness and dysphoria, and that anxiety and relieving of traumatic memories have the lowest impact. Contrary to popular belief, sleep disorders do not have a direct impact on chronic orofacial pain in PTSD, instead this association was found only through anxiety as the main symptom (18).

Today, the comorbidity of PTSD and orofacial pain is described by a model of a mutual maintenance of pain and PTSD that was first defined by Sharp. The model is graphically displayed as a variety of interconnected and mutually reciprocal symptoms, diseases and patterns. Components of such a model: excessive concentration of attention, anxiety sensitivity, remembering the trauma, avoidance, depression, misperception of pain and lack of cognitive abilities for adaptive strategy are interdependent with stress, chronic pain, post-traumatic stress disorder and disability (in terms of immobilization). Each component of this model can aggravate or cause deterioration of PTSD and chronic pain (19).

Emotional interpretation of pain in PTSD is explained by two mechanisms: dissociation and anxious sensitivity. Dissociation is a psychological defense mechanism in which the identity, memories, ideas, feelings, or perceptions are separated from conscious cognition and cannot be recalled or willingly experienced. Dissociative mechanisms are associated with disorders of attention both in peaceful and in emotionally charged state. Anxiety sensitivity is the fear that the symptoms such as palpitations, sweating, derealization, muscle tension and headaches will have adverse social, physical or mental effects. The fear of anxiety or pain itself can also occur, a fear that these symptoms are a sure way to insanity. The increased intensity of pain that occurs in PTSD is part of the emotional pattern according to which the pain is perceived as an unrealistic threat and/or associated with the trauma that caused the PTSD. Therefore, painful stimulus serves as a trigger of anxiety and encourages the catastrophic attitude towards pain and thus the fear of pain. The result is a much stronger pain than in the control population (4).

Two-thirds of patients with PTSD have another psychiatric diagnosis; the most common are depressive disorders, anxiety disorders, substance abuse and somatoform disorders (2). Comorbidity greatly increases the ability to con-

mi mogu pogoršavati bol i PTSP. Bol u slučaju PTSP-a može uzrokovati nelagodu, pojačati stres, bezvoljnost i nemoć te može biti podsjetnik na traumu. Pacijenti s PTSP-om prema boli imaju pristranost pažnje, što može voditi u amplifikaciju boli. Sve to opet može rezultirati uznemirenošću, pokušajem izbjegavanja traume i svega što na nju podsjeća, čime se širi uzorak mogućih uznemirujućih situacija. Tako je stvorena pozitivna povratna sprega između osjetnih, afektivnih i kognitivnih sastavnica psihogene i kronične boli, pa bilo koja komponenta može potaknuti i održavati kroničnu bol (18).

Zaključak

Kompliciranost odnosa i međuovisnosti simptoma PTSP-a i kronične boli komplicira i terapijsku strategiju da se pokušaju raskinuti uzročno posljedične spone međusobno podržavajućeg modela komorbiditeta. Farmakoterpija nije prvi izbor u liječenju, nego kognitivne i bihevioralne strategije suočavanja s problemom kako bi pacijent shvatio povezanost simptoma koji vode u katastrofične situacije s kroničnom boli i pogoršavanjem PTSP-a kao posljedicom. Ovakav način rada zahtijeva ne samo liječničku (stomatološku, psihijatrijsku, otorinolaringološku) skrb, nego i šire socijalno djelovanje.

nect with other symptoms from the cluster of symptoms and these multiple mechanisms can worsen the pain and PTSD. Pain in PTSD can cause discomfort, boost stress, apathy and helplessness and may be a reminder of the trauma. PTSD patients have an attentional bias to pain that can lead to amplification of pain. All this in turn can lead to anxiety, attempt to avoid the trauma itself and all that is reminiscent of the trauma, which extends the pattern of possible disturbing situations. This has created a positive feedback loop between sensory, affective and cognitive components of psychogenic and chronic pain, where any component can induce and maintain chronic pain (18).

Conclusion

The complexity of the relationship and interdependence of the symptoms of PTSD and chronic pain complicate the therapeutic strategy: we have to try and terminate the causal bonds of the mutual maintenance model of comorbidity. The model of choice in treatment is pharmacotherapy combined with other methods of treatment both psychiatric concerning PTSD such as psychotherapy and local treatment, concerning orofacial pain which include: massage, laser therapy, transcutaneous electrical nerve stimulation, exercise protocols for muscles and ultrasound. This principle of action requires not only medical (dental, psychiatric, otorhinolaryngological) care, but falls under the domain of broader social action.

Abstract
Chronic orofacial pain occurs frequently in patients with posttraumatic stress disorder (PTSD) and at the same time any pathological process involving orofacial area can be reflected in emotional interpretation of pain and can trigger a series of reactions associated with the PTSD group of symptoms in patients with PTSD. Painful stimuli caused in this way may occur after the primary cause ceased, and because of convergence can cause referred pain outside of the anatomical site where the primary injury occurred. Chronic orofacial pain and PTSD are diagnosed on the basis of subjective testimony and this regularly occurs in the context of social interaction between patients, doctors, medical staff or researchers making it difficult to standardize the results and introduces many cultural phenomena.

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