Psychological Trauma in Chronic Pain: Implications of PTSD for Fibromyalgia and Headache Disorders

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The association of traumatic exposures with posttraumatic stress disorder (PTSD) and other mental health conditions is well known. Patients with chronic pain, particularly headache disorders and fibromyalgia (FM), associated with psychological traumas need a special management strategy. Diagnosis of headache disorders and FM in traumatized patients and collecting the clinical history of a traumatic event or diagnosing PTSD in chronic pain patients is of great importance. Psychotherapy and pharmacotherapeutic options should be started on patients with comorbid PTSD and headache disorders and/or FM.

Introduction

Exposure to traumatic stressors and psychological trauma is widespread, with a wide range of cognitive and behavioral responses/outcomes among trauma survivors [1]. The association of traumatic exposures with posttraumatic stress disorder (PTSD) and other mental health conditions is well known [2]. Although traumatic events are associated with PTSD in the literature, traumatized people do not meet DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition) criteria for PTSD in many cases and often present a range of psychoform or somatoform symptoms [3]. Considerable overlap in symptoms and disease comorbidity has been noted for medically unexplained symptoms in the primary care setting, such as chronic fatigue syndrome, low back pain, irritable bowel syndrome, primary headaches, fibromyalgia (FM), temporomandibular joint disorder, major depression, panic attacks, and PTSD [4]. It is not unusual for patients presenting with chronic pain to describe significant levels of distress, including PTSD symptomatology. One of the first studies in this field was conducted in the past decade and investigated chronic pain patterns in Vietnam veterans with PTSD. Those reporting chronic pain showed significantly higher somatization than the others [5].

Epidemiologic surveys increasingly point to a relation between exposure to traumatic events and more health care utilization, adverse health outcomes, onset of specific diseases, and premature death. Certain characteristics of traumas, particularly peritraumatic cognitive response and related cognitions, appear to heighten the risk for PTSD [6]. Data from a cross-sectional survey of 3982 twins showed that comorbidity among nine conditions (chronic fatigue syndrome, low back pain, irritable bowel syndrome, chronic tension headache, FM, temporomandibular joint disorder, major depression, panic attacks, and PTSD) far exceeded chance expectations, suggesting that these medically unexplained conditions share a common etiology [7].

The way in which people process stressors may be critical in determining whether or not trauma will be experienced, as well as the different constellation of symptoms if traumatization is characterized. More understanding of patterns of comorbidity may help clinicians care for challenging traumatized patients. This article attempts to clarify possible correlations between psychological trauma and two nosologically distinct types of chronic pain: FM and headache.

PTSD: A Single Disorder With Many Different Facets

PTSD is characterized by the emergence of three sets of symptoms after exposure to one or several traumatic events: re-experiencing trauma (nightmares, traumatic memories, intrusive thoughts); emotional avoidance/numbness (affective distance, emotional anesthesia); and autonomous hyperstimulation (irritability, insomnia, hyperarousal). It is estimated that 51.2% of women and 60.7% of men have experienced at least one potentially traumatic event during their lifetimes [8]. Intense or overwhelming experiences may trigger different

responses, and studies have shown interindividual variability in the processing of life events and basic emotions [9]. The characterization of an event as traumatic also depends on the individual's perceptual processing, which is significantly influenced by subjectivity. Rather than simply passively registering reality, acquisition of information is conceived as an intrinsically active dynamic process of deconstruction and reconstruction of the external world on the basis of patterns of stimulations exciting our sensory receptors [10].

Two Independent Pathways for PTSD Development

In life-threatening situations, mammals tend to react in two ways: fight or flight and freezing. In light of adaptive evolutionary theory, both types of responses lead to adaptive gains for survivors. The defensive cascade animal model shows that several animals flee from or confront other predators, whereas others pretend to be dead when captured [11]. More than 100 studies have pointed to a distinction between simple and complex PTSD; therefore, some researchers have sought to include the dissociative subtype in the DSM-V [12]. Although based on recent literature, "freeze, flight, fight, fright, faint" provides a more complete description of the human acute stress response sequence than current descriptions [13]. Two main biobehavioral systems are involved in PTSD: 1) hyperstimulation of sympathetic reactivity with expressive activity of the adrenergic system typically involved in fight or flight responses, and 2) dissociation with parasympathetic reactivity involved in freeze responses [14]. Supporting these two PTSD subtypes is a model of risk factors for PTSD developed after a study with a group of acutely burned people. Two pathways to PTSD were discerned: 1) from the size of the burn and level of pain following the acute anxiety, and then to PTSD; and 2) from the size of the burn to the level of acute dissociation following the burn, and then to PTSD. Together these pathways accounted for almost 60% of variance in PTSD symptoms and constituted a model with excellent fit indices. These findings support a model of complex etiology for childhood PTSD in which two independent pathways may be mediated by different biobehavioral systems [15]. Similar results were found in a different sample of sexually abused children. Independent pathways—anxiety/arousal and dissociation—through which sexually abused children are likely to develop later PTSD symptoms, accounted for about 57% of variance in PTSD symptoms [16]. The finding that high levels of dissociative symptoms may be related to suppression of autonomic physiological responses to stress supports Bremner's conceptualization of dissociative symptoms as comprising one of two subtypes of the acute stress response, differing physiologically as well as subjectively from a predominantly hyperarousal or intrusive symptom response. The dissociative subtype may be seen in adults with a history of sexual abuse during childhood who present a consistent picture of dissociative amnesia, which occurs more often in victims of interpersonal violence during childhood than in combat soldiers and accident victims (who do not present hyperarousal symptoms).

Neuroimaging studies found distinct neural reciprocities for the two types of responses. The first pattern of sympathetic excitability involved attenuation of mid-prefrontal cortex activity and heightened amygdala activity leading to continuous autonomic arousal and state of alert [17]. The second pattern (dissociative) showed heightened activity of the mid-prefrontal cortex resulting in inhibition of amygdala activity, blunting the sympathetic response and leading to emotional numbing [18].

Subtype I

In a situation of unknown risk, heart and visceral alterations point to autonomous nervous system hyperactivity, whereas a subjective state of arousal potentiates an immediate search for syntheses and parameters for generating behavior. Peripheral and metabolic alterations (eg, tachycardia, mydriasis) reflect hyperactivity of the sympathetic nervous system and the hypothalamus-hypophysis-adrenal (HPA) axis, leading to an immediate self-preservation response [19•]. Neurofunctional studies with hyperarousal PTSD patients using symptom provocation paradigms (in most cases, the retrieval of traumatic memories) suggest that the difficulty of synthesizing, classifying, and integrating a traumatic memory in narrative form may be related to the decreased activity of the prefrontal cortex involved in reducing negative feedback from the activity of the amygdala [17]. Studies have implicated the HPA and the sympathetic-adrenal-medullar stress axes as key components of this pathogenic process [20]. The relationship between anxiety level and performance is no longer advantageous after a certain point. Self-generated information flooding into sensory pathways affects the perceptual processing of data from surroundings, thus hampering the ability to formulate new hypotheses and syntheses.

Trauma-related studies involving epinephrine (E), norepinephrine (NE), and serotonin (5-HT) suggested that alterations in NE, E, and 5-HT may have relevance for symptoms commonly seen in survivors with PTSD, including hypervigilance, exaggerated startle, irritability, impulsivity, aggression, and intrusive memories [21]. Studies related to the role of NE in arousal, orienting to novel stimuli, selective attention, and vigilance demonstrated heightened noradrenergic neuronal reactivity, increased α-2 receptor sensitivity and exaggerated arousal in organisms that have been exposed to chronic uncontrollable stress. The way an individual cognitively processes a traumatic event may trigger an anxious/arousal or a dissociative reaction. In subtype I, hyperactivity of the autonomic nervous system is observed, as in headache patients.

Subtype II

Tonic immobility (TI) is a possible component of the fear response characterized by freezing or immobility in situations involving extreme fear coupled with physical

restraint, and is observed in 30% to 40% of rape victims [16]. A study of TI in victims of childhood sexual abuse (CSA)—female undergraduates ($n = 39$) and female psychiatric inpatients ($n = 41$)—showed that more than 52% of all participants reported TI in response to CSA. TI reports were associated with greater current psychological impairment [22] and may be typical not only of rape and sexual abuse victims, but other kinds of directly experienced traumas as well [23].

Emotional factors play a role in many reflex TI manifestations referred to as *feigning death*. Whether orienting and defense responses provide a valid model in humans has yet to be proven, but the dissociative response in certain trauma cases resembles animal TI. Bovin et al. [24] asked whether TI mediates relations between perceived inescapability, peritraumatic fear, and PTSD symptom severity among sexual assault survivors. Their findings indicated that TI fully mediated relations between perceived inescapability and overall PTSD symptom severity, as well as re-experiencing and avoidance/numbing symptom clusters. Beyond the hyperarousal PTSD model, TI may be a pathway through which trauma survivors develop severe PTSD symptoms. Supporting this hypothesis, psychophysiological changes associated with peritraumatic dissociation in female recent-rape victims were studied, as well as the relation between these changes and PTSD symptoms. Individuals in the high peritraumatic dissociation group showed a significantly different pattern of physiological responses from those in the low dissociation group. There was suppression of autonomic physiological responses in the former group, which also showed a discrepancy between self-reports of distress and objective physiological indicators in the laboratory setting (low heart rate and skin conductance), whereas high measures of subjective suffering were observed while volunteers talked about rape. On the other hand, the low dissociation group showed increased sympathetic system response (heart rate and skin conductance) during the same task. These findings support the hypothesis that in addition to the anxiety/arousal subtype, there is a dissociative subtype of persons with PTSD symptoms who exhibit diminished physiological reactivity [25]. Another study found that female rape victims with acute PTSD who scored high on the Peritraumatic Dissociative Experiences Questionnaire exhibited suppression of physiological responses during exposure to trauma-related stimuli [26].

Routes to traumatic amnesia from dissociative detachment (loss of emotional content leading to loss of factual content) and from dissociative compartmentalization (failure in integration) are frequent in PTSD subtype II patients. Women with high disability pain were more likely to have experienced child abuse, adult sexual assault, more severe spousal abuse, lifetime abuse-related injuries, and PTSD symptoms. Chronic pain persisted long after leaving abusive partners and extended beyond usual locations (back, pelvic, gastrointestinal, headache) to include swollen/painful joints. Understanding patterns of chronic pain in abuse survivors and their associations with abuse history, mental health symptoms, health service use, and medication is important for clinical assessment and intervention.

Fibromyalgia

FM, a common chronic pain condition that affects at least 2% of the US adult population, is characterized by chronic widespread pain, tenderness, and associated symptoms such as fatigue, sleep disturbance, mood disorder, and cognitive dysfunction. Although the etiology of FM is not completely understood, the syndrome is thought to arise from acute stress, medical illness, and various pain conditions in some, but not all, patients, in conjunction with a variety of neurotransmitter and neuroendocrine disturbances. These include HPA-axis dysregulation, excessive pronociceptive input, and deficient modulatory signaling via noradrenergic and serotonergic pathways [27].

Trauma and dissociation tend to be interrelated, and many FM features resemble PTSD. Individuals with PTSD symptoms often present with co-occurring pain problems, and vice versa [4]. Chronic pain and PTSD are mutually maintaining conditions, and several pathways exist through which both disorders may be involved in escalation of symptoms and distress following trauma [28]. Over the past decade, PTSD literature has gradually become more sophisticated, resulting in well-supported theories and treatments [29]. However, to date there has been minimal attention devoted to concurrence of the two disorders, even though high comorbidity has been noted.

Naring et al. [27] examined the frequency of traumatic experiences and somatoform dissociation in FM patients reporting significantly higher levels of various forms of traumatization and dissociation, and found a significant correlation between traumatization and somatoform dissociation. Another study investigated FM–PTSD comorbidity in a cohort of men following an intensive, initial, defined traumatic event. Of the PTSD patients, 49% fulfilled the American College of Rheumatology criteria for FM ($P < 0.0001$), suggesting that PTSD is highly associated with FM, and that the degree and impact of these disorders are also closely related [30]. FM patients reported significantly higher levels of various forms of traumatization and dissociation than patients with other types of chronic pain. For instance, in FM patients, but not in patients with rheumatoid arthritis, there was a significant correlation between traumatization and dissociative symptoms [30]. A study aimed to determine whether PTSD symptoms are related to FM in a genetically informative community (based on a sample of twin pairs containing 1042 monozygotic and 828 dizygotic twin pairs), and if so, to ascertain whether the association is due to familial or genetic factors. The Impact of Events Scale (IES) was used to assess PTSD symptoms, and scores were strongly associated with FM ($P < 0.0001$). Although IES scores were more closely associated with FM among dizygotic than

among monozygotic twins, the difference was not significant. The findings suggest that PTSD symptoms, as measured by the IES, are strongly related to FM, but this association is not explained by a common familial or genetic vulnerability to both conditions [31].

Adults with FM report high rates of childhood trauma. Neuroendocrine abnormalities also have been noted in this population. Exploratory analyses tested relationships between retrospective reports of childhood trauma and diurnal salivary cortisol patterns among 85 women with FM. Childhood physical abuse predicted flattened diurnal cortisol rhythms and greater cortisol responses on awakening. Some findings suggest that severe traumatic experiences in childhood may be a factor of adult neuroendocrine dysregulation among FM sufferers [32]. Another study showed that women with FM are more likely to report a history of sexual and/or physical abuse than women without FM, and that chronic stress in the form of PTSD may mediate the relationship between rape and FM [33].

In contrast to a default "risk factor" hypothesis, a study of women in the New York/New Jersey metropolitan area (n = 1312) posed three alternative explanations for FM–PTSD comorbidity: 1) the association is due to sampling bias introduced by the study of care-seeking individuals; 2) FM is an additional burden that strains coping resources when confronting life stress; and 3) PTSD and FM arousal symptoms are confounded. The probability of PTSD was more than three times greater for women with FM-like symptoms, and this ratio was not reduced by controlling for FM-like symptoms or for the potentially confounded symptoms of PTSD specifically related to arousal. These findings rejected alternative explanations for FM–PTSD comorbidity; therefore, speculations that FM and PTSD share psychobiological risk factors remain plausible [34].

A study of the frequency of PTSD in FM patients and the influence of gender on PTSD measures in FM showed that 57% of the FM sample had clinically significant levels of PTSD symptoms [35]. FM patients with PTSD reported significantly higher levels of avoidance, hyperarousal, re-experiencing, anxiety, and depression than patients without clinically significant levels of PTSD symptoms. The prevalence of PTSD among FM patients was significantly higher than in the general population. Women with FM and PTSD reported more past traumatic events than their male counterparts. These findings suggest a significant overlap between FM and PTSD, according to the currently accepted diagnostic criteria for each. Many patients meet the criteria for diagnosis of PTSD and FM, and each disorder appears to be often induced by relatively short-term stress followed by chronic pathology, suggesting that stress may induce a self-perpetuating vicious cycle [36].

Somatoform dissociation in patients with PTSD and FM requires a broad approach to treatment with a special role of the psychologist or psychiatrist. Trauma history should be evaluated, and psychosocial intervention may be indicated as a component of FM treatment [32]. An emphasis on an integrated treatment plan in which chronic pain and PTSD must be strengthened, and treatment for these patients should focus on both physical and emotional dimensions of dysfunction [37].

Coping styles were analyzed with specific emphasis on differences between FM patients with and without PTSD [38]. FM patients showed significantly higher levels of suppression (P < 0.00001), and FM patients with PTSD presented higher suppression scores than FM patients without PTSD. Thus, suppression was identified as the only coping style significantly more common among "PTSD plus FM" patients than among patients with FM alone. These results may serve to further characterize cognitive and behavioral aspects of FM patients and subsequently guide therapeutic interventions [38]. Thought suppression—the process of deliberately trying to prevent certain thoughts—is a coping style often associated with dissociative PTSD symptoms. Engelhard et al. [39] examined 1) predictors for peritraumatic dissociation, 2) their relations with acute and chronic PTSD symptoms, and 3) pathways for these relations in response to pregnancy loss. Peritraumatic dissociation was related to acute PTSD symptoms and self-reported memory fragmentation and thought suppression of pregnancy loss mediated this relation. It also predicted chronic PTSD symptoms, and this relation was mediated by acute PTSD symptoms [39]. A prospective longitudinal study assessed 967 patients attending emergency clinics shortly after motor vehicle accidents, at 3 months, and at 1 year. PTSD prevalence was 23.1% at 3 months and 16.5% at 1 year. Chronic PTSD was related to certain objective measures of trauma severity, perceived threat, and dissociation during the accident. Maintaining psychological factors (ie, negative interpretation of intrusions, rumination, and thought suppression) enhanced the accuracy of predictions [40]. Another study explored whether the cognitive factors specified in the Ehlers et al. [40] study predict chronic PTSD in children who had been in a road traffic accident, who were tested 2 weeks, 3 months, and 6 months after the accident. Data-driven processing during the accident, negative interpretation of intrusive memories, alienation from other people, anger, rumination, thought suppression, and persistent dissociation at initial assessment predicted PTSD symptom severity at 3 and 6 months [41]. A 3-year follow-up of a prospective longitudinal study of PTSD after motor vehicle accidents found that perceived threat and dissociation during the accident and maintaining thought suppression were important predictors of PTSD persistence [42]. The relationship between engagement in physical activity, the psychometric traits of PTSD, and the future development of FM was studied in men suffering from combat-related PTSD; it was found that engaging in physical exercise was also associated with less severe disease. Physical exercise in male patients with combat-related PTSD provides protection from future development of FM. Activation of the adrenergic sympathetic system is probably related with better outcomes for PTSD patients with FM due to the negative feedback of the suppression–dissociation coping style [43].

Headache Disorders

Primary headache disorders are common in the general population [44,45•,46]. Recent data suggest that PTSD may be more common in headache sufferers than in the general population [47]. To evaluate PTSD prevalence in migraine patients and assess its relation to migraine severity, a study evaluated 92 patients fulfilling the International Headache Society criteria for migraine with and without aura using the Clinician-Administered PTSD Scale and headache severity scales. The prevalence of specific traumatic events in migraine patients was 16.3% (n = 15). Six of the 92 patients (6.5%) met the DSM-IV criteria for PTSD. The study emphasized that migraine patients do not suffer from PTSD more than the general population. However, when they do suffer from PTSD they report high levels of disability. On the other hand, the relative frequency of PTSD in episodic migraine (EM) and chronic daily headache (CDH) sufferers and the impact on headache-related disability were also evaluated, and results showed that the frequency of PTSD in migraineurs, whether episodic or chronic, is higher than historically reported PTSD prevalence in the general population. In the subset of migraineurs with depression, PTSD frequency was greater in patients with CDH than in EM patients [48]. Another study assessed the relative frequency of self-reported PTSD in patients with EM and chronic/transformed migraine. PTSD was more common in patients with chronic migraine than in EM. Thus, the researchers suggested that PTSD may be a risk factor for headache chronification, pending longitudinal studies to test this hypothesis [47]. A previous study looked at the prevalence of significant traumatic stressors and PTSD symptoms in a headache population. Almost 64% of headache patients reported one or more major traumatic stressors. This percentage was not significantly different from that of the comparison group (chronic masticatory muscle pain) and fell within the broad range reported for exposure to traumatic stressors in epidemiologic studies with nonpatient populations. One of six patients in the total headache sample, and one of four of those reporting a traumatic stressor, reported symptoms suggestive of current PTSD. The prevalence of current PTSD-like symptomatology reported by the headache patients was comparable to that of the comparison group of the present study, but higher than that reported for the general population. Traumatic stressors most often reported were not related to direct physical trauma, but rather associated with the loss or serious illness of a loved one. Symptoms consistent with a diagnosis of current PTSD appear to be more frequent in patients with recurrent headaches than reported in the scientific literature for nonpatient populations; therefore, screening for PTSD symptomatology is recommended as part of routine clinical evaluation for headaches [49].

Treatment

PTSD is an increasingly recognized and potentially preventable condition. However, ensuring physical safety by providing a safe environment is a primary goal that supersedes any therapeutic work. Psychological treatments are presently considered the first-line intervention of choice for PTSD patients with traumatic memories [50]. According to the Expert Consensus Guideline Series for treatment of PTSD [51], exposure-based therapy was indicated as psychological treatment of choice for flashbacks, intrusive thoughts, trauma-related fears, and avoidance. Clinical studies suggest that abnormalities in interpretation, synthesis, and integration of emotionally salient episodes play a crucial role in their being received as traumatic. Neuroimaging researches support the nonverbal nature of traumatic memories in PTSD volunteers and a more narrative-type expression of traumatic memories in non-PTSD volunteers [12]. The lower level of prefrontal cortical activity involved in reduced negative feedback from amygdala activity may obstruct processing of cognitive syntheses and may represent a defective extinction of responses to fear and emotional deregulation in PTSD [52]. On the other hand, the narrative structure of memory is an indicator of prefrontal-dependent cognitive processing in traumatized subtype I patients after psychotherapy [29]. Building new narratives based on healthy perceptions may facilitate the integration of traumatic mnemonic traces and sensorial fragments in a new cognitive synthesis, thus decreasing PTSD symptoms [29].

Whenever an event is retrieved, it may undergo cognitive and emotional change. In this respect, psychotherapy should facilitate a new framing of the traumatic experience by reviving and strengthening memories of successful coping and self-effectiveness prior to the trauma. These memories, with their respective emotional valences and states of consciousness, may be recognized and interconnected with memory of trauma during a restructuring session. All the multicomponent treatments that include cognitive interventions have exposure as one of their key elements [53]. Revisiting traumatic memories may have therapeutic benefits, as long as a well-structured process of restructuring of the emotional content is employed. We found that each time a patient narrated a traumatic episode, the narrative could be structured with new cognitive and emotional elements extracted from reinforced memories of successful coping. Therefore, the reinterpretation and reconstruction of traumatic memories may lead to changes in the neural networks involved and may relieve symptoms. Just confronting traumatic memories does not appear to be sufficient to provide a therapeutic effect; their restructuring and integration is required too. In many cases, the trauma per se must be accessed before mourning can proceed. In this respect, Pierre Janet's hypnotherapy and its approach based on a dissociation model has been used satisfactorily for cases in which traumatic grief occurs when psychological trauma obstructs mourning [54].

The two PTSD-plus-comorbid chronic pain subtypes also deserve special attention for psychotherapeutic treatment. Patients with high dissociation scores showed worse responses to exposure-based cognitive restructuring therapy.

Although there has been no research to date, our clinical experience is that patients with PTSD and FM also present worse response to exposure.

A systematic overview of studies of dissociation in children and adolescents published over a 10-year period involved a total of 1019 references, and aspects of assessment and/or treatment were studied in detail [55]. Combinations of individual psychotherapy, pharmacotherapy, and family therapy were often required to treat these disorders. Hypnotherapy, eye movement desensitization and reprocessing (EMDR), psychodynamic therapy, and an integrated approach were the main psychotherapeutic approaches described. Chronic traumatization from childhood may result in a number of additional problems not included in a DSM-IV–based diagnosis of PTSD. Upon examining the efficacy of a three-stage psychodynamically oriented inpatient treatment program and the role of chronic childhood threat for treatment outcomes, researchers showed that psychodynamic trauma therapy supports the stabilization of patients with childhood experiences of chronic threat [56]. For individuals who develop persistent PTSD, trauma-focused cognitive behavioral therapy (TFCBT) within a few months of the trauma may be beneficial. For those who develop chronic PTSD, TFCBT and EMDR are best supported by the current evidence [57].

The numerous functions of serotonin in the central nervous system include regulating sleep, aggression, appetite, cardiovascular and respiratory activity, motor output, anxiety, mood, and neuroendocrine secretion. Evidence of serotonergic dysregulation in PTSD includes frequent anxiety, aggression, and impulsivity symptoms [21]. The selective serotonin reuptake inhibitors (SSRIs), in particular sertraline and paroxetine, have emerged as the first-line pharmacological treatment for trauma victims experiencing hyperarousal symptom clusters, yet their effects are modest compared with psychological treatment. However, patients with complex subtype II PTSD—extreme stress disorder with symptoms such as dissociation and somatization—must be recognized as suffering from a trauma-related disorder that is qualitatively different from the one presently captured by the DSM-IV [58]. In addition to their being the most exhaustively studied and effective drugs for PTSD, SSRIs have a favorable adverse effect profile, making them the first-line treatment for PTSD in general. If SSRIs are not tolerated or ineffective, non-SSRIs should be considered [59]. Our clinical evaluations of headache patients point to behavioral patterns covered by the anxiety/arousal subtype, whereas FM patients presented a depressive pattern of autonomic suppression as well as dissociative symptoms.

The efficacy of the serotonin/norepinephrine reuptake inhibitor venlafaxine has been tested in PTSD patients. Venlafaxine showed significantly more improvement than placebo in cluster scores for re-experiencing ($P = 0.008$) and avoidance/numbing ($P = 0.006$), but not for hyperarousal [60].

Further controlled clinical trials are needed to investigate effective pharmacological alternatives. Patients with PTSD and chronic pain (headache and/or FM) may benefit from SSRIs or adapt better with serotonin-potentiating non-SSRIs, such as venlafaxine, nefazodone, trazodone, and mirtazapine. Presently, there is no consistent empirical support for using benzodiazepines in PTSD prevention or treatment, although these drugs may alleviate certain associated symptoms (eg, insomnia or anxiety) that are also seen in headache patients. Buspirone, a nonbenzodiazepine anxiolytic, was found to be effective in PTSD only in open-label studies [59]. The use of adrenergic β-blockers such as propranolol to treat PTSD may also lead to treatment of a coexistent migraine headache [21].

Conclusions

In light of the data reviewed here and our clinical experience, we suggest that patients with chronic pain, particularly headache disorders and FM, associated with psychological traumas certainly need a special management strategy. Diagnosis of headache disorders and FM in traumatized patients and collecting the clinical history of a traumatic event or diagnosing PTSD in chronic pain patients is of great importance. Psychotherapy and pharmacotherapeutic options should be started on patients with comorbid PTSD and headache disorders and/or FM.

Disclosure

No potential conflicts of interest relevant to this article were reported.

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have been highlighted as:
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••   Of major importance

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