Post-concussion syndrome: Part 2. Clinical characteristics, diagnosis, and treatment

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This paper presents current data on the clinical features, diagnosis, and treatment of post-concussion syndrome (PCS) that develops after mild blast-related traumatic brain injury (mbTBI). It is emphasized that PCS is one of the most common long-term consequences of mbTBI among military personnel exposed to blast waves, which determines the clinical and social relevance of this problem. The diagnostic criteria for PCS according to the International Statistical Classification of Diseases, 10th Revision (ICD-10), and the Diagnostic and statistical manual of mental disorders, fourth edition (DSM-IV), are described, as well as the difficulty of differentiating PCS from post-traumatic stress disorder, which frequently co-occurs with PCS in combat veterans. The following symptom groups are identified: cognitive, psychoemotional, somatosensory, autonomic, and vestibular. Particular emphasis is placed on the importance of using neurophysiological methods—quantitative electroencephalography and P300 event-related potentials—to objectify the diagnosis of PCS. The therapeutic approach should be multidisciplinary and personalized, incorporating physical rehabilitation, cognitive-behavioral therapy, sleep hygiene, and pharmacological management (antidepressants, analgesics, botulinum toxin therapy, and hyperbaric oxygen therapy). Research findings indicate the importance of early physical activity.

Keywords: *post-concussion syndrome; mild blast-related traumatic brain injury; cognitive impairment; post-traumatic stress disorder; neurorehabilitation.*

Post-concussion syndrome: clinical features, diagnosis, and differential diagnosis

A blast wave may cause mild, moderate, or severe traumatic brain injury (TBI). The clinical manifestations of mild blast-related TBI (mbTBI) in the acute phase are variable and include headache, fatigue, tinnitus, irritability, as well as neuropsychiatric and cognitive disturbances. Many injured military personnel who sustained mbTBI during military operations in Afghanistan and Iraq exhibited disorders of higher nervous activity and behavior, characterized by memory and cognitive impairments (reduced attention and concentration, slowed cognitive processing, speech changes), irritability, anxiety, and fatigue. Emotional changes, including mood lability, anxiety, depression, and personality alterations, are frequently observed concurrently [1, 2].

Complete recovery after mbTBI may occur within several days or weeks; however, many symptoms persist for a prolonged period. Unlike patients with moderate or severe TBI, individuals diagnosed with mbTBI typically have no visible structural brain damage and remain conscious, presenting with characteristic symptoms such as headache, confusion, dizziness, memory impairment, and behavioral changes. These disturbances may persist long after the injury or become permanent, leading to significant functional impairment. The chronic nature of cognitive deficits may be related to the fact that blast-related TBI increases the risk of delayed development of neurodegenerative diseases, including Alzheimer's disease and chronic traumatic encephalopathy [1, 3–5].

Patients with a history of multiple mbTBIs may experience more severe and persistent symptoms. Such injuries can cause immediate and, in some cases, prolonged neuronal damage and dysfunction, axonal stretching and injury, and alterations in neuronal plasticity. Thus, mbTBI may represent not a single "event" but rather a progressive pathological "process" of brain injury sustained by secondary molecular mechanisms, including neuroinflammation, oxidative damage, excitotoxicity, and other processes [4,6]. The clinical manifestations of mbTBI should not be regarded as purely "functional" due to the limited number of abnormalities detected by conventional neuroimaging. The underlying pathology involves both structural and functional damage of varying severity, independent of the type or apparent severity of injury. These changes often remain undetectable using routine imaging techniques such as computed tomography or magnetic resonance imaging (CT/MRI) [7].

The possibility of long-term persistence of neurological and cognitive deficits after mild TBI served

as the basis for identifying a distinct nosological entity—postconcussion syndrome (PCS).

The diagnostic criteria for PCS were developed by the World Health Organization and further elaborated in the Diagnostic and statistical manual of mental disorders, fourth edition (DSM-IV). According to the International statistical classification of diseases and related health problems, Tenth Revision (ICD-10), a diagnosis of PCS may be established when a TBI is sufficiently severe to cause loss of consciousness and is subsequently (within 4 weeks after the injury) accompanied by the development of at least three of the following features: 1) complaints of unpleasant sensations (dizziness, general malaise, excessive fatigue, or noise intolerance) and headache; 2) emotional changes (irritability, emotional lability, or depression and/or anxiety of varying severity); 3) subjective complaints of difficulties with concentration and the performance of mental tasks, as well as memory problems in the absence of clear objective evidence; 4) insomnia; 5) reduced tolerance to alcohol; and 6) preoccupation with these symptoms and fear of persistent brain damage, potentially progressing to hypochondriacal concerns and adoption of the sick role [8].

According to DSM-IV, the diagnostic criteria for PCS include: A) a history of TBI resulting in a "significant cerebral concussion"; B) deficits in cognitive functioning, specifically attention and/or memory; C) the presence of at least three of eight symptoms (fatigue, sleep disturbance, headache, dizziness, irritability, affective disturbances, personality change, apathy) that emerge after the injury and persist for ≥3 months; D) onset or worsening of symptoms following the injury; E) interference with social or role functioning; and F) exclusion of dementia due to head trauma and other disorders that better account for the symptoms. Criteria C and D define a symptom threshold requiring that the onset or exacerbation of symptoms be temporally related to the injury, differ from pre-existing symptoms, and be of sufficient duration [8,9]. It has been noted that the symptoms listed in both ICD-10 and DSM-IV occur more frequently in patients with traumatic brain injury than in those with extracranial injuries. The ICD-10 criteria encompass a broader range of symptoms than the DSM-IV criteria; however, the latter are more specific to traumatic brain injury [9].

According to V. Renga (2021) [7], the clinical manifestations of PCS can be grouped into five categories:

1) cognitive impairments: memory deficits; difficulties with attention and concentration; speech disturbances; executive function disorders; fine motor impairment;

2) psychological disturbances: depression, anxiety, irritability, personality changes, fatigue, derealization;

3) somatosensory and vestibulocochlear dysfunction: headache, nausea and vomiting, hypersensitivity to light and sound, hyperalgesia, tinnitus;

4) visual symptoms and oculomotor dysfunction: photophobia, blurred vision, convergence insufficiency, diplopia, Horner syndrome;

5) autonomic symptoms: fluctuations in heart rate and blood pressure, sweating disorders, pupillary abnormalities, impaired thermoregulation, sexual dysfunction, and sleep disturbances with reduced sleep efficiency.

The pathophysiological mechanisms underlying the delayed onset and prolonged persistence of PCS symptoms have been addressed in several literature reviews [6–8,10–13]. As noted above, the primary cause of pathological changes is damage to neuronal axons and small blood vessels, referred to as diffuse axonal injury with molecular consequences. In mild TBI, diffuse axonal injury most commonly involves the frontal and frontotemporal regions of the brain. One of the most significant consequences is disruption of functional brain networks. The human brain contains more than 100 billion neurons interconnected by trillions of synaptic connections. Neuroimaging studies (functional MRI with BOLD sequences, positron emission tomography, arterial spin labeling [ASL]) demonstrate disturbances of these functional networks following mild TBI. Such alterations persist for a prolonged period after the injury. According to functional near-infrared spectroscopy data, neurovascular coupling becomes inefficient even in the presence of normal or increased cerebral blood flow, indicating a functional mismatch between perfusion and the metabolic demands of neuronal cells. In addition to intracranial damage, the development of PCS may also be influenced by whiplash injury involving the cervical nerve roots, vestibular system, and cervical musculature. These extracranial effects contribute to headache, dizziness, visual disturbances, and balance problems. Intracranial, extracranial, and functional components of PCS pathogenesis are not mutually exclusive; they often overlap and are difficult to distinguish. The combination of these components shapes the clinical presentation of PCS and determines its individual characteristics.

Headache develops as a result of activation of the trigeminothalamic system. Activation of the trigeminal nerve begins with stimulation of the orofacial skin or cervical muscles. Whiplash injuries may initiate this process through mechanisms of convergence of sensory afferents within the trigeminocervical nucleus. In particular, stimulation of the greater occipital nerves (originating from the cervical roots) increases the excitability of nociceptive afferents of the dura mater, thereby activating the trigeminal pain system [7, 12, 13].

The vestibular system contributes to stabilization of the neck and trunk via vestibulocollic and vestibulospinal reflexes. Vestibular stem and peripheral disturbances lead to impaired cervical muscle tone and disruption of spinal balance mechanisms. Alterations of the vestibulo-ocular reflex may compromise visual target stabilization, resulting in blurred vision. Cervicovestibular dysfunction underlies the majority of symptoms associated with PCS. Cervical proprioceptors are synchronized with vestibulo-ocular and optokinetic reflexes to maintain gaze fixation on a visual target during head movements. Increased tension of the cervical muscles due to irritation of the cervical roots restricts the range of active neck movements and disrupts convergence mechanisms, leading to diplopia, dizziness, and postural instability [7, 12].

In PCS, dysfunction of higher central autonomic regulatory centers of the brain, as well as the peripheral autonomic nervous system, is also observed. Sympathetic innervation of the head and neck arises

from the superior, middle, and inferior cervical ganglia. The stellate ganglion gives rise to branches innervating the heart and cerebral vessels. Its involvement may result in impaired vascular regulation, leading to tachycardia and fluctuations in arterial blood pressure. In addition, disruption of central autonomic regulation also contributes to changes in heart rate and blood pressure. Ipsilateral partial Horner's syndrome is a typical sign of cervical injury. Conversely, sympathetic hyperactivity may also occur, manifesting as mydriasis, facial flushing, and hyperhidrosis [7].

A characteristic feature of mbTBI observed during recent military conflicts is the frequent coexistence of PCS and post-traumatic stress disorder (PTSD). More than one third of U.S. veterans from recent wars diagnosed with PCS following mbTBI also suffer from PTSD or depression [14, 15].

In the fifth edition of the diagnostic and statistical manual of mental disorders (DSM-5), PTSD is described as a psychiatric disorder that develops following direct or indirect exposure to life events that may result in serious injury, sexual violence, or death. PTSD comprises four symptom clusters: 1) various phenomena of re-experiencing, including recurrent, intrusive, and involuntary thoughts or images; distressing dreams; dissociative reactions such as flashbacks, during which the individual feels or acts as if the traumatic event were recurring, often involving all five sensory modalities; as well as marked emotional and physiological reactivity to external environmental or internal psychological cues associated with the traumatic experience; 2) avoidance, manifested by efforts to avoid trauma-related reminders, both internal (e.g., thoughts or memories) and external (e.g., places, conversations, films, and television programs), leading to social withdrawal and isolation; 3) negative alterations in cognition and mood, including cognitive distortions, memory deficits (inability to recall aspects of the traumatic experience), and persistent negative emotional states, as a result of which individuals with PTSD commonly experience fear, anger, guilt, or shame; hold negative beliefs and expectations about themselves (e.g., low self-esteem, self-blame, a sense of impending death) or about the world (e.g., believing that no one can be trusted or that no one is safe); exhibit diminished interest in activities, emotional detachment from others, and difficulty experiencing positive emotions; 4) marked alterations in arousal and reactivity of the central and autonomic nervous systems, manifested by irritability, verbal and/or physical outbursts of anger, reckless and/or self-destructive behavior, hypervigilance, exaggerated startle response, impaired concentration, and insomnia [16].

Epidemiological studies indicate that PTSD is a common problem among combat veterans. Between 4% and 33% of military personnel who served in Iraq and/or Afghanistan are affected by this disorder [17]. In a study conducted in the United Kingdom among active service members, 21.9% reported symptoms of mental disorders, probable PTSD was identified in 6.2%, and 10.0% reported alcohol misuse [18]. According to other data, while the prevalence of probable PTSD among active British military personnel was 4.8%, it reached 17.0% among former service members who had participated in combat operations [19]. Greater combat exposure is associated with more severe PTSD symptoms, which hinder adaptation to civilian life and adversely affect family relationships [20, 21].

When PCS and PTSD coexist, diagnostic evaluation becomes more challenging, as clinically distinguishing between these two conditions is often difficult due to substantial symptom overlap [22]. The symptoms listed in the diagnostic criteria are not specific to PCS alone; they are also observed in non-injured populations and in other non-traumatic conditions, such as depression, anxiety disorders, and chronic pain [23, 24]. In some cases, TBI and PTSD may coexist as a result of the independent effects of physical trauma and psychological stressors [22].

It has been noted that, in the absence of objective diagnostic assessments, particularly during the chronic phase of TBI, the validity of PCS as a diagnostic entity is questioned by many specialists [13]. Notably, observations indicate that the frequency of reported symptoms increases when individuals are involved in legal proceedings [25]. It has been suggested that the apparent epidemic of PCS among Western military personnel during the wars in Iraq and Afghanistan may represent an artifact resulting from a lowered diagnostic "threshold" for mbTBI, including the consideration of minor transient alterations in consciousness. Studies have shown that PCS symptomatology can be attributed solely to blast exposure only in cases involving a clear loss of consciousness, which was reported by a minority of veterans of these conflicts. A 2014 report by the U.S. Institute of Medicine concluded that there is scientific evidence indicating that, in affected individuals, most shared symptoms are better explained by comorbid PTSD rather than by the consequences of mbTBI alone [22,26].

Conversely, complaints of cognitive and behavioral impairments often do not emerge immediately after the injury but rather 1–3 months thereafter. This delay may lead to their misinterpretation as being caused not by TBI but by affective disorders [27,28].

Despite its apparent simplicity, the diagnosis of PCS remains a subject of debate for several reasons, including insufficient symptom specificity, the influence of premorbid status on injury outcomes, "recall bias", and the impact of psychosocial factors [13]. "Recall bias", also referred to as the "good old days" phenomenon, occurs both in healthy individuals and in various pathological conditions. It reflects a tendency to underestimate the severity and extent of prior symptoms, particularly during the premorbid period [29]. A study involving children and adolescents with PCS during the post-traumatic period demonstrated that patients and their parents, when retrospectively assessing premorbid symptomatology one month after injury, underestimated its severity by approximately 80%. This level of underestimation persisted until the end of follow-up (three months post-injury) [30]. These findings indicate that the "good old days" phenomenon may lead to underestimation of premorbid symptoms and overestimation of PCS severity. Investigation of this phenomenon and the development of methods to mitigate "recall bias" may help reduce patients' anxiety regarding the severity of their condition and promote a more accurate subjective perception of TBI severity and its impact on quality of life [13, 31].

The diagnosis of PCS following mbTBI may also be complicated by additional factors. Clinicians may find it difficult to accurately assess the true severity of TBI due to limited information about the circumstances of the injury, which may rely on the injured individual's subjective recollection months or even years after the event(s). Furthermore, the absence of certain objective characteristics of the injury circumstances may compromise the accuracy of "blast self-assessment," particularly regarding distance from the explosion and the severity of concussive forces—data that are often missing from primary medical records. The lack of reliable information on injury characteristics complicates determination of cognitive recovery trajectories and rehabilitation planning. Typically, the presence and severity of PCS are assessed based on the current state of neuropsychological and cognitive functioning using screening scales, which are not necessarily specific to TBI and may instead reflect the severity of comorbid PTSD or depression [10, 32].

Thus, the symptom complexes of PCS and PTSD are largely similar and may substantially "overlap" within an individual patient. In patients with PCS, the likelihood of comorbid PTSD is high due to its considerable prevalence among combatants. The presence of PTSD significantly complicates the diagnosis of PCS because of the similarity of clinical manifestations. However, TBI represents an organic injury to neural tissue, whereas PTSD is a psychological response to a stressor and is not accompanied by structural changes. This fundamental distinction necessitates different therapeutic approaches to these conditions. While the treatment of PTSD primarily focuses on normalization of stress responses through psychological and pharmacological interventions, the management of TBI must account for structural and molecular-biological alterations in order to prevent their impact on the severity and duration of neurological deficits [22, 33].

An important objective is to improve the diagnosis of PCS following mbTBI by refining objective criteria for structural and functional disturbances of the central nervous system. This would enhance prognostic assessment of mbTBI in injured individuals and facilitate optimization of treatment strategies, including personalized approaches aimed at preventing the development of persistent neurological deficits. Neurophysiological investigations hold substantial potential for the objective verification of PCS following mbTBI. According to previously published results of our study, quantitative electroencephalography (qEEG) in patients with PCS reveals alterations in the frequency and topography of the α-rhythm, reduced α amplitude, frequency–spatial inversion, and signs of dysfunction of nonspecific brain structures; spectral analysis demonstrates decreased α power, increased β power, and elevated activity in the θ- and δ-frequency bands. These characteristics may persist into the long-term period after mbTBI and should therefore be taken into account in the differential diagnosis with PTSD [34]. The analysis of event-related cognitive potentials (P300) has shown that these parameters are statistically significantly associated with the severity of cognitive impairment and may serve as an effective tool for objective assessment of the degree of cognitive dysfunction in patients with PCS [35].

Treatment of post-concussion syndrome resulting from mild blast-related traumatic brain injury

The management of PCS differs from therapeutic approaches used in the acute and subacute phases of mild TBI. Rather than emphasizing rest and inactivity, the primary goals are to improve overall functioning, restore daily activities, and facilitate a return to normal life. This is achieved gradually, with active involvement of the patient and their social environment. Once the clinician has a clear understanding of the clinical presentation, attention should be focused on one or two of the most problematic symptoms, as other symptoms often regress during the course of treatment. Patients with persistent dizziness, balance disturbances, or ongoing visual complaints should be referred to appropriate specialists. Peripheral vestibular dysfunction should initially be managed with specialized physical therapy. Migraine-associated dizziness may be treated with preventive migraine therapies. Convergence insufficiency and persistent reading dysfunction may respond to oculomotor neurorehabilitation. The most common problems typically include sleep disturbances, headaches, as well as cognitive and mood disorders [13].

Rest and sleep. According to polysomnographic data, sleep disturbances in PCS are characterized by reduced sleep efficiency, prolonged sleep latency, and an increased number of nocturnal awakenings. Their development correlates with structural changes observed on magnetic resonance imaging, psychological disorders (particularly anxiety), and decreased melatonin production [7,36]. Adequate sleep and rest are of crucial importance for both the prevention and treatment of PCS. Rest should include both physical and cognitive components; however, prolonged rest is not recommended. Routine daily activities are necessary to maintain basic functioning. The optimal duration of rest varies between individuals. For physically active persons, complete cessation of activity may be unnatural; in such cases, maintaining a certain level of daily activity may be beneficial. Individual characteristics related to age, physical condition, and psychological factors should be taken into account.

Brain recovery depends on sufficient sleep. Sleep quality and rest are particularly important, as deeper stages of sleep promote the reorganization of neural connections. Waking up feeling refreshed is an indicator of good sleep quality. In patients with PCS, sleep quality is often impaired. Adjunctive measures such as melatonin supplementation, sleep music, or white noise may be helpful. Cyclobenzaprine provides a dual effect by relaxing muscles and improving sleep. It is especially important for patients with PCS to fall asleep at the same time each night, as this may help stabilize disrupted circadian rhythms [7, 13]. For the management of sleep disturbances, amitriptyline may be used; it can also be beneficial for headache treatment, short courses of non-benzodiazepine hypnotics (e.g., zopiclone) and cognitive-behavioral therapy may also be considered [13].

Physical therapy. It has been noted that restriction of physical activity after mild TBI leads to delayed recovery and greater severity of symptoms within the spectrum of post-concussion syndrome (PCS) [38]. Prolonged physical inactivity may exacerbate the development of secondary symptoms, such as depression, anxiety, and fatigue [39]. In contrast, early rather than delayed initiation of moderate physical exercise improves clinical outcomes in patients with PCS [40]. Improvements have been demonstrated in patients who received physical therapy compared with those who did not [12]. In the study by [39], the PCS treatment program included physical therapy in the form of jogging, cycling, joint and soft tissue repositioning or mobilization procedures, as well as vestibular and oculomotor rehabilitation techniques. Physical therapy contributed to a greater regression of post-traumatic symptoms. The authors note that the absence of spontaneous recovery after mild TBI may be partly attributable to concomitant injuries of the spine and other body regions caused by the traumatic physical force.

The cornerstone of PCS therapy is physical therapy targeting the vestibular system and the cervical spine. Vestibular therapy includes exercises aimed at training the vestibular apparatus through oculomotor exercises, as well as head and trunk exercises designed to improve balance and visual stability. Visual and oculomotor therapy may help restore smooth pursuit eye movements and fixation. A normal range of cervical motion is essential for balance reflexes that prevent falls. Most patients with PCS exhibit components of cervicogenic dizziness and balance impairment. Therefore, cervical physical therapy should be an integral part of PCS rehabilitation. Manual therapy and relaxation of the cervical musculature using therapeutic exercises, massage, warm compresses, and topical applications may also be beneficial. Severe vestibular dysfunction, particularly in the acute phase, can be difficult to manage because of pronounced dizziness and nausea. In such cases, the use of vestibular sedative agents, such as benzodiazepines, flunarizine, or betahistine, may be considered. Gabapentin and amitriptyline may also help reduce sensitization phenomena, while cyclobenzaprine is useful for muscle relaxation. All of these medications also have sedative effects [7].

Behavioral interventions. Recovery involves relearning previously acquired skills. Adherence to a structured daily routine represents the most important behavioral modification for restoring normal brain function. Waking and going to sleep at consistent times may help entrain the brain to a regular schedule. In the evening, the use of stimulants, including caffeine and alcohol, as well as substances that depress the central nervous system, should be avoided. It is important to avoid public gatherings, excessive social media engagement, loud sounds, and rock music. Screen time involving television, computers, or mobile phones should be reduced. Beneficial activities include quiet walks in nature at dawn or sunset, listening to calming instrumental music, and reading printed books [7].

Hyperbaric oxygen therapy. This modality involves placing the patient in a chamber in which the pressure of pure oxygen is gradually increased to levels 1.5–3.0 times higher than normal atmospheric pressure. The mechanism of action is thought to involve stimulation of angiogenesis, potentially leading to increased cerebral perfusion. Enhanced blood supply to brain regions that have become ischemic or hypoxic due to vascular injury may accelerate recovery processes. A study evaluating the neurotherapeutic effects of hyperbaric oxygen therapy in PCS using cerebral perfusion imaging and clinical assessment of cognitive function demonstrated positive outcomes. Patients showed significant improvements in cognitive domains such as information processing, processing speed, and visuospatial processing. In addition, cerebral blood flow and cerebral blood volume increased in brain regions responsible for visual, sensorimotor, memory, and attentional functions [41].

Pharmacotherapy. Headache management. Treatment of pain syndromes, including headache, typically begins with the administration of anti-inflammatory and analgesic agents such as acetylsalicylic acid, ibuprofen, and paracetamol. Due to the similarity between post-traumatic headache and migraine, migraine-specific medications, such as triptans, are often prescribed. However, frequent use of large quantities of medications increases the risk of medication-overuse headache. To prevent this condition, preventive therapy with antidepressants, β-blockers, anticonvulsants, and agents for neuropathic pain is recommended. More invasive procedures, including botulinum toxin therapy and nerve blocks, may also be effective. Botulinum toxin therapy is an effective modality approved by the U.S. FDA for the treatment of chronic migraine. The neuromuscular blocker botulinum toxin type A is believed to inhibit peripheral signal transmission to the central nervous system, thereby preventing central sensitization and reducing pain perception in both migraine and post-traumatic headache. A common cause of headache in PCS is irritation of the cervical nerve roots. Irritation of the occipital nerves resulting from whiplash injury often leads to migraine attacks, dizziness, and cervical muscle spasm. Occipital nerve blocks may help interrupt the headache pathway between the cervical nerve roots and the spinal trigeminal nucleus [7, 12, 42, 43].

Antidepressants – are among the most commonly prescribed medications for the symptomatic treatment of post-concussion syndrome (PCS). Patients with depression exhibit deficits in cerebral monoaminergic neurotransmitters (norepinephrine, serotonin, and/or dopamine). The balance and levels of these neurotransmitters play a crucial role in many behavioral symptoms, including mood, fatigue, and psychomotor activity. The etiology of comorbid depression in patients with PCS is associated with post-traumatic chemical imbalance of these neurotransmitters in the brain. Certain selective serotonin reuptake inhibitors have been widely used in the management of PCS. Correction of post-traumatic depression and anxiety has been achieved with sertraline at daily doses of 25–100 mg and fluoxetine at daily doses of 20–60 mg [12]. Cannabinoids have also been considered a potentially effective option for the treatment of PCS symptoms [44].

Cognitive rehabilitation. Cognitive rehabilitation begins with a baseline assessment using a battery of tests, such as the Montreal Cognitive Assessment. In cases of chronic persistent cognitive difficulties with

symptoms resembling attention-deficit/hyperactivity disorder, central nervous system stimulants, including methylphenidate, may be considered. However, methylphenidate should not be prescribed in patients at risk of dependence or seizures, or in those with sleep disturbances. Donepezil has been evaluated for short-term memory impairments associated with PCS. Daytime sleep-related problems may be addressed with modafinil. Cognitive-behavioral therapy is beneficial for mood disorders associated with PCS. Breathing and meditation exercises help improve autonomic regulation and enhance attention, serving as an anchor around which recovery processes may occur [7].

Thus, the therapeutic range for PCS includes a broad range of non-pharmacological and pharmacological interventions. The selection of treatment modalities is determined by the individual clinical characteristics of the patient, with clinical presentation often being highly variable. This underscores the need for an individualized approach to management and, in many clinical situations, for the adoption of personalized clinical decision-making.

Disclosure of Information

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