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Cognitive Dysfunction In Conversion Disorder Patients Brief Review

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Introduction

Conversion disorder (CD), classified under somatic symptom and related disorders (SSRD) in the DSM-5 [1], involves deficits in voluntary motor or sensory functions that cause significant distress and impact multiple areas of daily life. CD symptoms include motor weakness, abnormal muscle contractions, sensory loss, and non-epileptic seizures [2]. These symptoms cannot be explained by neurological or other medical conditions [1]. Risk factors include depression, psychological or physical trauma, and low education levels [3]. Recent studies highlight that CD is also linked to cognitive impairments in domains like attention, learning, auditory-verbal memory, memory, executive functioning, and visuospatial skills [4].

Conversion Disorder

Introduction

Conversion disorder (CD), also called functional neurological disorder, involves physical and sensory symptoms—such as paralysis, numbness, blindness, deafness, or seizures—that lack any identifiable neurologic pathology. These symptoms severely impact essential functions like academics, social life, and family relationships [5]. In children, CD symptoms are genuine but not due to medical issues, often arising from poorly expressed distress, which is alleviated by the symptoms' presence [6, 7].

Etiology

CD may stem from psychological, social, and biological factors, often following trauma, life stress, or chronic adversity. Many patients report a history of childhood abuse (emotional or sexual) [8].

- **Psychological Factors**: CD is linked to poor coping skills and psychological conflicts. It commonly coexists with psychiatric conditions such as depression, anxiety, and personality disorders [5]. Patients with CD often report multiple somatic complaints like fatigue, weakness, or unexplained pain. Physical injuries or neurologic issues like

strokes can trigger symptoms, with higher prevalence in individuals with lower education, socioeconomic status, or rural backgrounds [9].

- **Biological Factors**: Biological contributors may include impaired cerebral communication, excessive cortical arousal reducing body awareness, and mild neuropsychological impairments [10].

Theoretical Models of CD

- **Psychodynamic Model**: Emotional conflict, repressed into the unconscious, may manifest as physical symptoms, functioning as a defense mechanism [11].
- **Cognitive-Behavioral Model**: CD symptoms emerge when an individual's worry activates memory representations of symptoms. For example, a person who saw a seizure in a film might later experience similar episodes triggered by anxiety, a process driven by unconscious psychological influences [12][13]. Hypotheses also suggest that neuroimmune mechanisms, such as altered synaptic plasticity, may contribute to motor dysfunctions in CD [14].

Risk Factors

Risk factors for CD include having a neurological disorder (e.g., epilepsy, migraines), significant recent stress or trauma, a mental health condition, a family member with a neurological disorder, and a history of childhood abuse. Women may be more prone to CD than men [15].

Epidemiology

CD has an incidence of 4–12 per 100,000 people annually (4–5 for motor CD; 1.5–4.9 for psychogenic non-epileptic seizures [PNES]) and a prevalence of 50 per 100,000. In neurology clinics, CD was a primary diagnosis in 5.4% of 3,781 outpatients, with 30% presenting symptoms not fully explained by disease. Women constitute 60–75% of cases, though functional myoclonus and parkinsonism affect men equally or more often. Misdiagnosis rates are low (4% after five years of follow-up). In a study of 1,030 patients, only 0.4% were later diagnosed with a neurological condition after 18 months, affirming the accuracy of CD diagnosis by neurologists [16-18].

Egyptian Prevalence Study

A national survey in Egypt involved 14,640 adults across five regions, diagnosing mental disorders using the MINI-Plus. The overall prevalence was 16.93%, with 6.43% experiencing mood disorders, 4.75% anxiety disorders, and 4.72% multiple disorders. Prevalence correlated with factors like gender, employment status, marital status, and chronic illness (e.g., heart disease, kidney disease) [19].

Pathophysiology

Neurobiological models suggest that conversion disorder (CD) involves altered cortical processing. Emotional stress may activate the frontal and subcortical brain regions, inhibiting basal ganglia-thalamocortical circuits, which impacts conscious sensory and motor functions. Although studies with large samples are scarce, functional neuroimaging has provided insights. For instance, Spence et al. found that CD patients showed decreased left dorsolateral prefrontal cortex activity, differing from controls feigning weakness, suggesting unique brain involvement in CD [20-21]. Similarly, Voon et al. reported abnormal amygdala and supplementary motor area activation in CD patients performing emotional tasks [22].

Diagnosis

Clinical Presentation

Conversion disorder symptoms vary based on affected brain regions. Common symptoms include psychogenic non-epileptic seizures (PNES), sensory disruptions (vision, hearing, touch), muscle weakness, tremors, difficulty swallowing, dizziness, fainting, and chronic fatigue [23].

Common Misdiagnoses

The symptoms of CD can lead to misdiagnosis, with an estimated error rate of 4%. Misdiagnoses often include Guillain-Barré syndrome, HIV/AIDS, health anxiety, lupus, myasthenia gravis, and various neurological disorders [24][25]. CD patients may exhibit "la belle indifference," appearing unconcerned about their symptoms, although this is not always present [15].

Diagnostic Criteria

A CD diagnosis requires evidence of altered motor or sensory function not attributable to a medical condition or other mental disorder, with symptoms causing significant distress or impairment. Diagnostic tools like EEG and neuropsychological assessments have improved diagnosis accuracy. According to DSM-5 criteria, CD can present with weakness, paralysis, abnormal movement, swallowing or speech issues, sensory loss, and seizures. Diagnosis is generally performed by neurologists and mental health professionals [26][27].

Co-occurring Conditions

CD commonly coexists with mood, panic, generalized anxiety, PTSD, dissociative, OCD, somatic, and personality disorders. Depression is frequently found among CD patients experiencing non-epileptic seizures [28].

Diagnostic Challenges and Tools

Diagnosing CD can be complex due to its symptom overlap with other conditions. Diagnosis should not rely on negative test results for other disorders. Often, neurologists and psychiatrists collaborate, with neurologists ruling out neurological conditions and psychiatrists confirming CD. Tests may include:

- **Health Assessment and Physical Exam**: Symptom history, life events, and functional tests assess movement and reflexes.
- **Imaging Tests**: X-rays, MRI, and EEG help detect brain abnormalities or electrical issues, such as epilepsy.
- **DSM-5 Criteria**: Diagnosis requires symptoms outside conscious control, unlinked to drugs or other conditions, causing distress in social or work life [29-31].

Treatment Approach

Multidisciplinary Involvement

Neurologists and mental health professionals should align on diagnosing functional neurological disorders (FND) for effective treatment. Studies indicate discrepancies in belief about diagnostic methods, such as video EEG's accuracy for psychogenic non-epileptic seizures, especially among psychiatrists versus neurologists. Patient adherence improves with initial referrals to mental health professionals, which facilitates diagnostic acceptance. Identifying susceptibility traits (e.g., avoidance tendencies, alexithymia) aids in creating a treatment framework [32-33].

Medication Management

Patients with FND frequently have psychiatric comorbidities, including depressive and anxiety disorders, PTSD, and hypochondriasis. Evidence for antidepressants in FND is limited to a few uncontrolled studies, with no placebo-controlled trials proving efficacy for specific medications [34]. Medication choices should be guided by the treatment needs of comorbidities, with clinical discretion determining the timing and suitability of psychiatric medications [34].

Psychotherapy

Therapies like psychotherapy and physical therapy (PT) target brain information processing changes to minimize symptom manifestation. Psychotherapy helps patients alter distress responses through cognitive and behavioral adjustments. A structured approach is recommended, involving symptom diaries, chain analysis, and coping strategies [26].

Physical Therapy

PT is beneficial for motor and gait issues in FND. A randomized crossover study of 60 patients with psychogenic gait disorders found that a 3-week inpatient PT program showed substantial improvements sustained for a year post-treatment [35]. Consensus guidelines support PT for motor FND, emphasizing both education and active patient participation. Clinicians should provide PT guidelines to therapists unfamiliar with FND for consistency in treatment [36].

Cognitive-Behavioral Therapy (CBT)

CBT effectively manages FND symptoms by educating patients on FND and stress responses, training in stress management, and restructuring harmful thought patterns. Randomized pilot

studies have demonstrated that self-guided CBT workbooks reduce symptom burden at 3 and 6 months compared to standard care, achieving significant results. CBT is particularly effective for episodic symptoms, such as psychogenic seizures [37].

Alternative Treatments

Hypnosis shows efficacy in randomized trials for FND. Short-term psychodynamic psychotherapy, initiated immediately or after 3 months, reduces psychogenic movements. Other treatments include psychodynamic therapy, prolonged exposure for comorbid PTSD [38], mindfulness-based therapy, psychoeducational interventions, and transcranial magnetic stimulation targeting the motor cortex [39].

Cognitive Impairment

Introduction

Cognitive impairment refers to challenges in memory, attention, language, and recognition of people or places, which can cause difficulties in new or complex settings [40]. Cognitive function involves processes like memory, decision-making, perception, and language. The term "cognitive deficit" broadly describes impairments across these areas [41].

Etiology

Cognitive deficits can be congenital or result from factors like brain injury, mental illness, or neurological disorders. Though more common in the elderly, not all aging individuals experience cognitive decline [42-43].

Short-term or reversible cognitive impairment causes include infections (e.g., UTIs or pneumonia), vitamin deficiencies, dehydration, medication reactions, or falls [44]. Long-term causes include stroke, delirium (acute confusion), dementia (progressive decline), depression, schizophrenia, chronic alcohol use, brain metastasis, head injury, and brain infections. Brain pathologies like Alzheimer's, Parkinson’s, Lewy body dementia, and Huntington's also contribute to cognitive impairment. In younger populations, causes can include cancer therapy effects, heavy metal poisoning, malnutrition, metabolic conditions, and autism [45].

Risk Factors

Major risk factors for mild cognitive impairment (MCI) include advanced age and the APOE e4 gene, associated with Alzheimer's disease. However, possessing the gene does not guarantee cognitive decline [46]. Additional risk factors are diabetes, smoking, high blood pressure, high cholesterol, obesity, depression, sleep apnea, lack of physical activity, low education, and limited social or mental engagement [47].

Epidemiology

While cognitive deficits are challenging to predict precisely, aging is the strongest associated factor. Alzheimer’s disease is a significant contributor to cognitive impairment, affecting around 5.5 million in the U.S. and over 24 million globally [48]. Among African Americans, Alzheimer’s prevalence is nearly double compared to European Americans. After age 60,

dementia incidence is expected to double every decade, with Alzheimer's rates rising from under 1% per year before 65 to about 6% per year after 85 [49].

Pathophysiology

Cognitive deficits stem from neuronal damage, including harm to grey matter areas (e.g., cortex, thalamus, basal ganglia) and white matter (e.g., axonal coverings). Specific areas of damage cause particular deficits: parietal lobe injury can impair visuospatial abilities, frontal lobe injury affects planning and abstraction, and temporal lobe injury impacts language and memory [50]. Causes of damage include toxins (e.g., heavy metals, metabolic disorders, toluene), ischemic injury from strokes, head trauma, cancer, or degenerative diseases like Alzheimer's, Parkinson's, and Huntington's, which involve abnormal protein-induced immune responses [51].

Diagnosis

Signs and Symptoms of Mild Cognitive Impairment (MCI): MCI manifests as mild declines in memory, language, attention, reasoning, and complex planning. Signs include recent memory loss, language issues, reduced focus, problem-solving difficulties, and impaired depth perception. While individuals may need help with some daily tasks, MCI does not typically impact everyday functioning severely. Greater impairment suggests dementia rather than MCI [52-54].

Diagnostic Criteria:

MCI is diagnosed based on international expert panel criteria:

1. Memory or mental function issues (memory, planning, decision-making), often corroborated by a close contact [57-58].
2. Decline over time, confirmed via personal history and third-party input [59].
3. Intact daily activities despite symptoms [60].
4. Mild impairment noted in mental testing (e.g., MoCA, MMSE) [61].
5. Symptoms not severe enough for dementia diagnosis [62].

Neurological Exam: Basic neurological tests (e.g., reflexes, eye movement, balance) can reveal conditions affecting cognition like Parkinson's, strokes, and tumors [63-64].

Laboratory and Imaging Tests: Blood tests can rule out deficiencies like B-12 or thyroid hormone issues, while MRI or CT scans detect structural issues such as tumors or strokes [65-66].

Mental Status Testing: Brief tests (10 minutes) assess cognitive status, while detailed assessments compare function to age and education norms and help identify cause-specific symptom patterns [43, 67].

Treatment

MCI treatment varies by cause:

- No pharmacologic treatment exists specifically for MCI, with a focus on maintaining function [68].
- Supportive therapies include physiotherapy, exercise, sleep hygiene, cognitive training, and relaxation.
- A Mediterranean diet, physical/occupational therapy, and environmental adjustments (e.g., noise reduction, familiar surroundings) may aid function [69].
- Psychotherapy and support for patients and families improve symptom management and quality of life [40].

Cognitive Dysfunction in Patients with Conversion Disorder

Introduction

Neurocognitive symptoms are prevalent in individuals with somatic symptom and related disorders (SSRD), yet specific neurocognitive impairments in conversion disorder (CD) or functional neurological disorder (FND) remain poorly understood [70].

Neurocognitive Functioning in CD/FND

According to the DSM-5, CD/FND involves deficits in voluntary motor or sensory functions that cannot be attributed to neurological or medical conditions. Symptoms can include motor weakness, abnormal muscle contractions, loss of sensory functions, and non-epileptic seizures. The prevalence of CD/FND ranges from 0.7% to 5.0%, with typical onset between ages 10 and 35. Women are more affected than men, and risk factors include depression, trauma, and low education levels [71]. Patients with CD/FND often have lower education levels compared to healthy individuals and exhibit high rates of psychiatric comorbidities such as depression and personality disorders. Physical stressors, such as limb injuries, can also trigger symptoms [72].

Neurocognitive impairments in CD/FND affect attention, learning, memory, executive functioning, and visuospatial abilities. However, methodological limitations in existing studies, such as varied neuropsychological assessments and inadequate consideration of psychiatric comorbidities, necessitate caution in interpreting results. Notably, conditions like chronic pain and chronic fatigue syndrome also lead to cognitive deficits in attention, processing speed, and memory [73][74][75]. Studies comparing neurocognitive functioning between CD/FND and other SSRDs are limited, making it unclear if cognitive deficits in CD/FND are more severe.

A Unifying Theory for Cognitive Abnormalities

Reports of non-dementia cognitive symptoms are rising, with 5% to 32% of healthy young adults experiencing benign cognitive symptoms. The term functional cognitive disorder (FCD)

describes subjective cognitive dysfunction without underlying brain pathology [77][78]. Symptoms include task forgetfulness and absent-mindedness, despite neuropsychological assessments showing no objective deficits, indicating a discrepancy between subjective experience and objective performance [79][80].

FCD is linked to psychological factors such as memory perfectionism and heightened self-monitoring, though empirical support is limited. It falls under the FND spectrum, characterized by both internal and external inconsistencies in cognitive symptoms [82][83].

Patients with FND often present with movement control abnormalities and report cognitive difficulties. Ongoing discussions explore how the pathophysiological understanding of FND relates to chronic pain and fatigue syndromes, such as fibromyalgia (FM) and chronic fatigue syndrome (CFS), which share overlapping symptoms and cognitive difficulties referred to as "brain fog" [84][85].

The relationship between FCD and cognitive symptoms in FM, CFS, and FND is not well-defined. However, the extensive literature on cognitive symptomatology in these conditions provides a basis for comparison and understanding of FCD [86].

Transdiagnostic Impairment of Cognitive Control in Mental Illness

Introduction to Cognitive Control

Cognitive control, or executive functions, encompass processes that enable the flexible and adaptive use of cognitive resources in response to changing circumstances. This capability is crucial for self-regulation and successful adaptation to daily life demands. Cognitive control predicts success in education, occupational stability, health promotion, and overall quality of life among healthy individuals. Its influence suggests a vital role in the development, maintenance, and treatment of psychopathology [87].

Neurocircuitry and Cognitive Control in Mental Health

In the context of mental illness, cognitive control neurocircuits are activated to regulate symptoms during distress. Meta-analyses of functional neuroimaging show that adaptive emotion regulation recruits cognitive control networks, including the frontoparietal network (dorsolateral prefrontal and posterior parietal cortices) and the cingulo-opercular network (dorsal anterior cingulate cortex, anterior insula, and anterior prefrontal cortex) [88].

Behavioral and Structural Patterns

Intact cognitive control manifests in specific behavioral patterns and functional neurocircuitry. Cognitive control disruptions in psychopathology are pronounced across various disorders, indicating transdiagnostic characteristics. A meta-analysis reveals gray matter reductions in a dorsal cingulate-anterior insula network, which is linked to impaired cognitive control performance [89].

Implications for Treatment

Functional neuroimaging studies highlight cognitive dyscontrol across disorders such as psychotic, bipolar, unipolar depressive, anxiety, and substance use disorders. There is potential to utilize cognitive control and its neurocircuitry in developing effective transdiagnostic interventions aimed at reducing distress and enhancing real-world functioning [90].

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