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Untangling the Seizure Spectrum: A Case Series on Psychogenic non-epileptic Seizures in Complex Scenarios

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Abstract

Background: Dissociative neurological symptom disorder (DNSD), encompassing psychogenic non-epileptic seizures (PNES), presents a diagnostic challenge, especially when co-occurring with epilepsy. This case series explores the complex interplay of neurological and psychological factors in such presentations.

Methods: Three case reports of patients with seizure-like episodes and neurological symptoms are presented. Each case details the clinical presentation, medical history, psychological evaluation, treatment, and outcomes.

Results: Cases 1 and 2 initially presented with features suggestive of epileptic seizures, but subsequent evaluations revealed dissociative neurological symptom disorder (DNSD) due to the presence of psychological stressors, atypical seizure characteristics, and normal EEG findings during later episodes. Case 3 presented with atypical seizure-like episodes from the outset, associated with significant anxiety, depression, and a history of trauma. All cases highlight the importance of thorough clinical evaluation, including detailed history taking, neurological examination, and psychological assessment. Psycho-therapeutic interventions, such as cognitive-behavioral therapy (CBT) and eye movement desensitization and reprocessing (EMDR), proved effective in managing symptoms.

Conclusions: This case series underscores the necessity of considering psychological factors in the presence of seizure-like episodes, even in individuals with a history of epilepsy. A multidisciplinary approach, incorporating neurological and psychological assessments, is crucial for accurate diagnosis and effective treatment of DNSD.

Introduction

Dissociative neurological symptom disorder (DSND), according to the 11th revision of ICD, is defined as the involuntary disruption or discontinuation in the normal integrity of neurological functioning, including motor, sensory, or cognitive function. DSND is often triggered by psychological stress and conflicts and manifests as neurological symptoms without any identifiable organic pathology.[1] They are more common in children with adverse childhood and scholastic issues; however, they can also co-occur with established neurological conditions like epilepsy, which poses a diagnostic challenge.

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This case series explores distinct presentations of DSND seen during the time period of January 2021 to July 2021 at Brainworks neuro-psychiatry Center and emphasizes the importance of a comprehensive assessment that integrates neurological and psychological evaluations. These cases also highlight the possibility of psychological distress, especially anxiety and PTSD, presenting with non-epileptic seizures and other neurological symptoms. Given the frequent overlap with epilepsy and the potential for misdiagnosis, a nuanced approach is essential.[2]

Case Presentation

Case 1

The first case is of a 26-year-old female who presented with frequent seizure episodes occurring multiple times weekly. She has a history of febrile seizures at the age of 5 and a single epileptic seizure three years back, which was treated with sodium valproate 800mg daily for two months.

Her episodes initially were characterized by typical features of seizure like upward rolling of eyes, frothing at the mouth, and loss of consciousness lasting for 1 to 2 minutes, which were preceded by symptoms of anxiety, shortness of breath, and head tightness lasting for several hours. An EEG was done during this time which showed generalized spike wave discharges. The subsequent episodes exhibited asymmetrical jerky movements, absence of frothing, and retained consciousness, and notably, they lasted longer than the initial episodes, mostly up to 5 minutes. An EEG was taken again; however, it did not show any abnormalities. A video EEG was recommended, however, but was not completed due to patient anxiety and the non-availability of the facility in the clinic. Due to the history of seizures and the initial EEG abnormalities she was trialed with levetiracetam 500 mg and sodium valproate 600 mg for 3 months however continued to have frequent such episodes with no improvement resulting in a referral to the clinic, considering a differential diagnosis of psychogenic non-epileptic seizures (PNES).

Upon further evaluation, she disclosed a history of witnessing a traumatic accident four years prior involving a high-speed motor vehicle collision resulting in significant distress and nightmares. She also added that four days prior to the onset of her current symptoms, she sustained a mild head injury by a minor road traffic accident. She described experiencing intrusive thoughts and flashbacks related to the event, as well as hypervigilance and emotional numbing. She reported significant difficulty in sleeping and frequent nightmares related to the accident. She also reported increased anxiety in crowded places when hearing loud noises and was able to identify a combination of intense odor and loud sounds as a trigger for her seizure episodes. The patient reported, “It’s like I re-live the accident when these triggers happen.”

Psychological evaluation using PCL-5 revealed a score consistent with PTSD, and the GAD-7 score indicated moderate anxiety. The neurological examination was unremarkable.

A diagnosis of dissociative neurological symptom disorder with non-epileptic seizures (6B60.4) was made in addition to other specified epilepsy (8A6Z), Post-traumatic stress disorder (PTSD) (6B40), and an unspecified anxiety disorder (6B0Z) considering the evolution of seizure characteristics, presence of psychological triggers, normal EEG findings during the later episodes and non-response to anti-epileptics.

Her levetiracetam and sodium valproate were discontinued due to non-response, and she was initiated on Pregabalin 75 mg to help with anxiety and considering her initial seizure episodes and escitalopram 15mg to address the PTSD and anxiety symptoms. A referred for psychotherapy including eye movement desensitization and reprocessing (EMDR) and underwent 12 sessions of EMDR, focusing on the traumatic accident and related triggers. She reported a significant reduction in her intrusive thoughts and nightmares, as well as improved emotional regulation.

She showed marked improvement within one month with a reduction in her non-epileptic seizure episodes and anxiety symptoms. She was followed up monthly for 6 months from the initial presentation and she reported only occasional anxiety and no seizure episodes.

Case 2

The second one is a complex case of a 35-year-old female presenting with a seven-year history

of epilepsy and recent behavioral changes. She describes her episodes as being different from her past episodes, being preceded by nausea, headache, and a sensation of head heaviness followed by loss of consciousness. These episodes were not characterized by jerky movements, frothing (which were present in her past episodes of seizures), tongue biting, or incontinence. However, these episodes were brief, typically lasting 2-5 minutes, followed by generalized weakness. Notably these episodes usually happened with the family members present. She also exhibited a new onset behavioral issue, which included frequent impulsive actions such as running out of the house and "feelings of lacking mental strength".

Her past MRI revealed scattered hypodensities in the right cerebellar region, and the past EEG showed spikes in the occipital area, during which she was treated with sodium valproate and levetiracetam by neurologists. She was started again on levetiracetam and sodium valproate by neurologists. However, there was no improvement, and she continued to have frequent such episodes. An EEG was taken which did not show any changes. This resulted in a query of PNES as a differential along with the underlying past history of epilepsy and was referred to a psychiatrist who started her on sertraline which was increased to 100mg, aripiprazole 5mg, clonazepam 0.25 mg and levetiracetam 500mg BD. She was later referred to the clinic for further evaluation of her symptoms.

During the consultation, she experienced an episode that involved teeth grinding and asymmetric jerky movements that lasted for 15 minutes and were preceded by nausea. She retained some level of consciousness during the episode and did not report any tiredness afterward. Her only significant past medical history was hypothyroidism. Initially, she did not report any symptoms of anxiety or depression. However, further evaluation revealed having significant familial stress concerning her father's wealth and her husband's lack of employment. She expressed frustration with her husband's dependence on her father's wealth, which she does not support, and she required to support the family financially. This contributed to elevated levels of stress and anxiety. The patient stated, "I feel completely overwhelmed and trapped".

Psychological evaluation using GAD-7 suggested a score indicating moderate anxiety, and the neurological examination was unremarkable. Treatment was initiated by increasing sertraline to 150mg and lorazepam as required and stopping aripiprazole along with a slow-reducing regimen on levetiracetam due to suspicion of levetiracetam causing behavioral changes. Her behavioral symptoms continued to persist even with this, and it was decided to add cognitive behavioral therapy (CBT) focusing on stress management and coping skills. Her behavioral symptoms started to improve with the CBT, with no seizure episodes reported. Considering her previous response to levetiracetam without any reports of behavioral episodes and improvement of such episodes with CBT queried the possibility of behavioral issues due to levetiracetam.

Given the atypical seizure presentation, lack of improvement with anti-epileptic medications, presence of significant psychosocial stressors, and behavioral changes, she was diagnosed with DNSD along with the diagnosis of epilepsy and an unspecified anxiety disorder.

During four months of regular follow up, she was seizure free, after which she reported an episode of seizure following the death of a neighbor. She attributed this seizure to the stress from the event and reported stopping levetiracetam. She was advised to resume the medication due to her past history of epilepsy and to continue regular follow-up. She is currently symptom-free and has made substantial progress in managing her behavioral changes.

Case 3

The third case is of a 29-year-old female presenting with a history of seizure episodes followed by a subjective experience of unilateral body paralysis and numbness. The seizure episodes were typically abrupt in onset, often triggered by loud sounds without any preceding aura or prodrome, characterized by involuntary jerky movements that lasted for 5-10 minutes, followed by unilateral numbness or paralysis lasting for approximately 30 minutes. She described this experience as a sudden, overwhelming sense of her body becoming unresponsive, as if "frozen" or "locked ."She reported that during the

episodes, she felt a disconnect between the mind and her body, experiencing a sense of detachment from the affected side. The seizure episodes were not associated with tongue biting, frothing, incontinence, or loss of consciousness. She experienced 2-3 such episodes per month, which she attributed to feelings of overwhelming stress and anxiety and having features of panic attacks like feelings of impending doom, palpitations, chest tightness, and shortness of breath.

Her EEG and MRI results were normal. She has a previous history of seizures at the age of 13 which were associated with incontinence and loss of consciousness. She recently consulted a psychiatrist for her anxiety symptoms and was prescribed escitalopram 10mg, mirtazapine 15mg, olanzapine 5mg, sodium valproate 500mg BD and Clonazepam 0.5mg. She experienced some improvement initially; however, later, she started experiencing a cluster of such episodes over a period of 2 weeks. She was referred to the clinic for further evaluation with the query of DNSD.

Upon psychiatric evaluation in the clinic, she displayed pervasive sadness, anxiety, and insomnia with heightened sensitivity to loud sounds. Psychological evaluation using GAD-7 revealed a score indicating severe anxiety and HAM-D score indicating moderate depression. No formal PTSD evaluation was done. The patient reported significant stress related to interpersonal conflicts and financial strain. She stated, "I feel like my body just shuts down when I get overwhelmed". Neurological examination during the consultation was unremarkable. During the episodes of subjective weakness, she reports having decreased motor strength, but no clear objective findings of motor weakness could be demonstrated.

Differential diagnoses considered included recurrent epileptic seizures, PNES, and a combination of both. Given the atypical presentation of seizures (lack of loss of consciousness, lack of post-ictal confusion, and the subjective sense of paralysis) and no response to the anti-epileptics with normal EEG and MRI findings and a strong association with stress and anxiety a diagnosis of PNES was made with co-morbid anxiety, panic disorder (6B01) and depression. The past history of seizures at the age of 13 was considered to be a separate event, not directly related to the current presentation.

Treatment was adjusted to a reducing regimen of sodium valproate while continuing the other medications. The patient also began CBT, focusing on stress management, coping skills, and challenging maladaptive thought patterns.

Throughout treatment, she showed significant improvement and is currently free of episodes with a marked reduction in anxiety and depressive symptoms. She was also slowly weaned off from Olanzapine and mirtazapine, stabilizing her on escitalopram 20mg. She was followed up monthly and is currently symptom-free with better managed anxiety and depression.

Discussion

The cases presented above highlight the importance of recognizing NES as a manifestation of psychological distress, even in individuals with a history of epilepsy. Differentiating between epileptic seizures and NES requires a meticulous clinical evaluation, including detailed history taking, neurological examination, and EEG monitoring, ideally combined with video EEG.[3] The various factors that can help distinguish epileptic seizures from PNES include:

The ICD-11's classification of DNSD provides a framework for understanding these presentations, emphasizing the role of psychological factors in the etiology of neurological symptoms. Cases 1 and 2 initially presented with features suggestive of epileptic seizures, including EEG abnormalities. However, no improvements with anti-epileptics, the normal subsequent EEG queried a possibility of PNES along with underlying epileptic seizures. In Case 3 as well the history of epileptic seizures contributed to diagnostic uncertainty, highlighting the challenge of differentiating epileptic and non-epileptic seizures when a history of epilepsy is present.

Across all three cases, the presence of significant psychological stressors, including trauma, familial stress, and anxiety, played a pivotal role in the manifestation of seizure-like episodes. This underscores the importance of a comprehensive assessment that integrates neurological and psy-

Table 1: Clinical profile of Psychogenic non-epileptic Seizures in the included cases

Psychogenic non-epileptic seizures	*Epileptic seizures*	*Case 1*	*Case 2*	*Case 3*
May or may not have a history of seizures	History of seizure	Previous history of seizures	Previous history of seizures.	No previous history
Anxiety and absence of aura	Aura	Shortness of breath and head tightness	Aura is seen	No aura
Induced by stress	Not induced by stress	Induced by stress and has features of PTSD	Induced by stress from family and sometimes without any reason	Induced by stress
Conscious during seizures	Unconscious during seizures	Conscious during seizures	Loss of consciousness seen	Conscious during seizure
Occurs in front of a witness to seek attention	May not have witness	Not seen	Usually but happens when she is alone too	Not seen
Asymmetrical body movements during seizures	Symmetrical body movements during seizures	Asymmetrical body movements	No body movements were noted during a seizure.	Asymmetrical body movements
No delirium	Post-ictal confusion	No confusion seen	No post-ictal confusion.	No confusion post-seizure, but weakness of the body was noted
No increase in prolactin	Raised prolactin after seizure if blood is taken within 30 minutes	-	-	-
No injury because of seizures	Injury because of seizures	No injuries seen	No injuries	No injuries
No incontinence	Incontinence after seizure	Not seen	Not seen	Not seen
Normal EEG and normal MRI	Abnormal EEG and some changes in MRI	Initial EEG showed generalized spike-wave change; however, subsequent ones did not show any changes. Normal MRI	Initial EEG showed spikes in the occipital region and hypodense areas in the right cerebellar region in MRI. Later, EEG showed no changes.	Normal EEG and normal MRI

chological evaluation, and a high index of suspicion is crucial for accurate diagnosis of PNES.[4] The successful implementation of psycho-therapeutic interventions such as CBT and EMDR highlights the efficacy of addressing the underlying psychological distress.[5] By alleviating comorbid anxiety and mood instability through pharmacotherapy, patients can achieve greater emotional stability, which, in turn, supports the progress of psychotherapy. In our cases, a combined approach of pharmacotherapy and psychotherapy—particularly cognitive behavioral therapy—proved highly effective, resulting in the complete resolution of seizure episodes and significant improvement in the patients' psychological well-being.

Conclusion

Studies indicate that the prevalence of dissociative disorders in inpatient settings ranges from 5% to 21%, while outpatient prevalence is between 12% and 29%, underscoring the complexities involved in achieving an accurate diagnosis.[6] The frequent comorbidity of PNES and epilepsy emphasizes the

need for careful evaluation to avoid misdiagnosis and ensure appropriate management. The initial presentation of cases 1 and 2 accentuates the need for clinicians to be vigilant in the evaluation of seizure characteristics and the importance of repeated EEG testing, possibly video EEG, when the initial findings are not congruent with the clinical picture. The presence of both epileptic and non-epileptic seizures in the same individual necessitates a nuanced approach to diagnosis and management, requiring a thorough understanding of both neurological and psychological factors.[7] This can be achieved by the involvement of a multidisciplinary team collaborating across specialties such as psychiatry, neurology, and psychology, resulting in early recognition and effective management of PNES.

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