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CASE REPORT

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Case report on psychogenic nonepileptic seizures: A series of unfortunate events

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Abstract

Psychogenic nonepileptic seizures, also called Functional Seizures, are a highly disabling form of Functional Neurological Disorder. The diagnosis of PNES could be missed in clinical scenarios where patients exhibit concurrent musculoskeletal/neurological findings. Characterization of convulsions and judicious use of diagnostics are equally valuable in establishing the diagnosis.

KEYWORDS

convulsions, cyanosis, dissociative, functional seizures, psychogenic

1 | INTRODUCTION

"Functional neurological disorder" (FND) is the term used to describe neurological symptoms in the absence of any structural neurological disease or injury.[1] For those with FND, although the nervous system structure appears normal, its "function" has a problem, which affects the nerve pathways that govern tasks such as movement control, pain, and attention.[2,3] The specific symptoms of FND vary and can include psychogenic nonepileptic seizures (PNES) and movement syndromes, such as limb weakness or gait problems.

Scientists agree that no single sign or symptom is a reliable diagnostic discriminator for PNES.[4] Patients experiencing PNES may exhibit a range of manifestations, including paroxysmal movements of the trunk and limbs, similar to tonic–clonic epileptic seizures (ES) or motor discrepancies such as 'give-way weakness.'[5,6] However, clinical classification systems such as that described by Maggauda et al. (2016) outline semiological differences between PNES and ES based on current research.[7,8] For instance, in a controlled study, researchers observed asynchronous jerks in 96% of PNES cases versus 5% in ES, suggesting this is an important sign and discriminating feature of PNES.[4,8,9] The International League Against Epilepsy (ILAE) Task Force (2013) suggests that in settings with video electroencephalography (vEEG), this, in combination with direct observation of abnormal signals, offers a diagnostic "gold standard."[10,11]

PNES case estimates in the literature are based on diagnoses from tertiary care epilepsy monitoring units. The prevalence range of PNES is 2–33 cases per 100,000/year.[11,12] In 2017, it was ranked among the top three neuropsychiatric problems by the ILAE.[6,13]

The risk factors described in PNES include, but are not limited to, sexual abuse, anxiety disorder, physical abuse or neglect, traumatic brain injury, and psychiatric comorbidities, with high post-traumatic stress disorder (PTSD) rates.[6,14] Additionally, evidence supports that universally felt stressors, such as mass casualties and natural disasters, may increase FNDs in adults and children.[15–18] Likewise, repeated secondary exposure to traumatic events in the media cycle, social isolation, and movement restrictions appear equally detrimental.[15] The COVID-19 pandemic is a recent example of an unprecedented global event causing individual and collective fear, stress, and uncertainty.

Emerging studies demonstrated the impact of the pandemic and the strict lockdown on mental health, especially on individuals with pre-existing psychiatric conditions, which may ultimately impact the PNES burden.[15]

Conflicting views on the course of illness and limited research on treatment response contribute to the controversy of PNES.[14] As such, current guidelines emphasize that diagnosis of PNES should rest on clear positive evidence, typically from a combination of physical signs and examination of the nature of seizures, and benefits significantly from multidisciplinary evaluation.[1,19] In this report, we present a case of PNES in a 64-year-old patient describing the challenges to diagnosis and its clinical peculiarities. We later emphasize the patient-centered approach to PNES management and discuss the implications in a COVID-19 era.

2 | PATIENT INFORMATION

Our patient was a 64-year-old Caucasian male transferred to our neurology center after developing abnormal "seizure-like" limb movements with concurrent episodes of reduced awareness and unresponsiveness. He had been hospitalized for 1 month with a new diagnosis of eosinophilic vasculitis and started on methotrexate which was discontinued before his transfer.

A typical episode, witnessed by health staff and described in detail by the patient, began with a pulsating sensation in his ears, followed by a wave of "tension and pain" down his limbs. This pain was accompanied by a facial grimace and corresponding tremors in his lower limbs, which progressed to more violent shaking while he held on to the bed barriers. He experienced no loss of consciousness (LOC) and was able to communicate throughout these events. He did not experience bowel or bladder incontinence, and there was no evidence of tongue biting. Of note, he was found to have transient peripheral cyanosis affecting his toes during his "convulsive" episodes, which lasted for a few seconds.

Each episode was followed by a brief period of reduced responsiveness. Nonetheless, the patient reported being fully aware of his environment during that phase and could recall what was happening or being said around him. However, he could not move his limbs or verbalize. This reduced responsiveness lasted for about 2 min, after which he was fully mobile and responsive.

Most of these episodes occurred while he was in bed or during his clinical evaluation on the ward; they rarely occurred while alone. His vitals, especially oxygen saturation, were normal during and after each event. The attending team subsequently noted that he demonstrated a fixation on his symptoms and conducted meticulous online research into his ailment. On review of systems, he reported having palpitations with each event. He denied the occurrence of such episodes before this presentation. These episodes were unresponsive to Levetiracetam 1.5 g twice a day and low dose Diazepam 2mgs TDS as an antispasmodic, which he was started on before his transfer.

2.1 | Past medical history

Seven years prior, he was diagnosed with type 2 diabetes with peripheral neuropathy and retinal detachment, which left him with reduced vision in his left eye. His regular medications included oral hypoglycemic agents, a statin, and capsaicin cream.

2.2 | Social history

He was a self-sufficient proprietor of a printing business where he worked for long hours and was independently self-caring and mobile. He lost his wife due to ovarian cancer the year before and was the primary carer of his elderly mother after losing his father to "throat" cancer 2 years prior. His new debilitating diagnosis of eosinophilic vasculitis and a relatively extended stay in hospital were distressing to both him and his family. He repeatedly expressed concern that he had been unable to receive his COVID-19 booster shot due to his immune suppression and diminished health status.

3 | SYSTEMIC EXAMINATION

The full neurological examination yielded no objective focal abnormality representing any structural neurological events. Notably, the only abnormal findings were:

- "Give-way" weakness or a sudden loss of muscle power from a previously normal power during the assessment. However, these findings were inconsistent and non-reproducible. They mainly affected flexion of the upper limb and lower limbs to a lesser extent.
- Mental status examinations were unremarkable.

More detailed findings, including systemic examinations, are outlined in the examinations table.

Tables 1 and 2

4 | VIDEO EEG

Media File: Case Video Electroencephalogram (vEEG).

TABLE 1 Systemic examinations table

Systemic examination	Findings	
Vitals on transfer	BP: 150/96 P: 78 bpm Temp: 36.5 C Resp: 18 breaths/min SpO2: 96% RBS: 15 mmol/L	
General	Mucus membranes pink and moist Hands warm to the touch No conjunctival pallor, peripheral or central cyanosis on examination	
Cardiovascular	• No lower limb oedema • Peripheral pulses palpable bilaterally–dorsalis pedis, tibialis posterior • Good capillary refill • Radial Pulses: Symmetrical with regular rate, rhythm, and volume • Normal JVP, Nil carotid bruit • No chest heaves or thrills • Apex in the 5th left intercostal space at the midclavicular line • Normal heart sounds (S1, S2) • No heart murmurs appreciated	
Respiratory	• Central trachea • Normal chest resonance and chest wall expansion • Nil tactile vocal fremitus • Vesicular breath sounds throughout (anterior, posterior, axillae, and lateral) • No wheezing, crackles, rubs, crepitations	
Abdominal	• No visible scars, masses/nodes, or discoloration • Flat, soft, non-tender abdomen with no palpable masses • Normal liver span • Normal spleen • Normal bowel sounds in all quadrants, no bruits	
Neurological and mental status examination	**Findings**	
Glasgow Coma Scale	15/15	
Mini mental status examination: • Orientation • Registration • Attention and Calculation • Recall • Language • Copying	Well Oriented to person, place, and time (10/10) Good (3/3) Good (5/5) Normal (3/3) Follows command (8/8) Good (1/1) SCORE: 30/30 No Cognitive Impairment	
Mental status examination:	Clean, tidy appearance, normal speech and eye contact, no restlessness with full affect. Low mood, good orientation, memory, and attention. (MMSE normal) Denies hallucinations of auditory, visual, sensory nature. Nil desensitization or depersonalization. Nil suicidality, homicidality, or delusions. Cooperative with good insight and judgment. Assessment: Nil issues	
	Left	Right
Cranial nerves: • Visual Acuity • Visual fields • Pupillary reaction • Optic Discs • Extraocular movements • Light touch to face • Eyebrow raising, eye closing, smile • Hearing • Palate elevation • Trapezius and SCM strength • Tongue inspection	SLIGHTLY REDUCED NORMAL PERLA Not examined NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL	>6/18 NORMAL PERLA Not examined NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL

(Continues)

TABLE 1 (Continued)

Neurological and mental status examination	Findings	
Motor: • Pronator drift • Upper extremity • Tone • Proximal Power (Deltoid) • Proximal Power (Biceps, Triceps) • Distal Power (Hand Grip) • Lower Extremity • Tone • Proximal Power (Iliopsoas) • Distal (Foot dorsi-flexion and Plantar flexion)	NIL GOOD 5/5 5/5 with give-way weakness 5/5 with give-way weakness GOOD 5/5 with give-way weakness 5/5 with give-way weakness	NIL GOOD 5/5 5/5 with give-way weakness 5/5 with give-way weakness 5/5 with give-way weakness GOOD 5/5 with give-way weakness 5/5 with give-way weakness
Sensory: • Upper Extremity • Pinprick • Vibration (DIP and Index) • Proprioception • Lower Extremity • Pinprick • Vibration (DIP and Index) • Proprioception • Romberg	INTACT INTACT INTACT LOSS (Known Diabetic Neuropathy) INTACT INTACT NORMAL	INTACT INTACT INTACT LOSS (Known Diabetic Neuropathy) INTACT INTACT NORMAL
Reflexes: • Biceps • Brachioradialis • Triceps • Patellar • Achilles • Plantar response	NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL	NORMAL NORMAL NORMAL NORMAL NORMAL NORMAL
Coordination and Gait: • Rapid alternating movements • Coordination of Limbs • Finger-to-nose • Heel-to-shin • Casual gait • Tandem Walking • Heel and Toe walking	MODERATE SPEED GOOD INTACT INTACT NORMAL NORMAL NORMAL	

5 | TIMELINE OF CLINICAL EVENTS

6 | DIAGNOSTIC ASSESSMENT

This is a case of documented psychogenic nonepileptic seizures of psychosocial etiology. This diagnosis was confirmed using a routine video EEG. A seizure specialist confirmed that the patient manifested the typical seizure-like movement without concomitant EEG abnormalities.

7 | OTHER COMPLEMENTARY INVESTIGATIONS

8 | THERAPEUTIC INTERVENTION

We conducted a 10-min educational session in which the attending neurologist explained the diagnosis, providing him with a patient information website for further details on his condition. He was continued on low-dose Diazepam, 2 mg PO at night for anxiety and

sleep insomnia and was prescribed Gabapentin for neuropathic pain. We reduced the Levetiracetam by 250 mg per week until his transfer back to the referring center one month later.

9 | FOLLOW-UP AND OUTCOMES

Following discharge from the referring center, he reported experiencing a noticeable reduction in the number of episodes and being able to terminate the initiation of events with focused breathing techniques. However, he received no formal follow-up for PNES. He could not remember the diagnosis or explain why he had these symptoms.

10 | DISCUSSION

Early diagnosis of PNES is crucial; unfortunately, it is often missed or delayed.[20] Therefore, identifying "time-savers" and "time-wasters" in managing PNES may significantly increase service efficiency, reduce costs to the health system, and psychological costs to patients and improve continuity of patient care for more favorable long-term outcomes. For instance, multiple recent studies demonstrate that patients with an early diagnosis of PNES showed improved higher functioning at 1 year of follow-up, particularly in cognitive areas such as language and executive function.[5,6,11] On the contrary, delayed diagnosis may contribute to improper management with high doses or prolonged antiseizure therapy in PNES patients, which could lead to irreversible adverse effects.[5,20]

This case featured one of the main barriers to diagnosis, a concomitant neuromuscular pathology (eosinophilic vasculitis) which may prove a veritable red herring in PNES. Although for our patient, the onset of the episodes to diagnosis was 17 days; (Figure 1; Timeline of Clinical Events) a relatively short duration, misdiagnosis is not uncommon and comes at a high cost to patients and health systems. In 2019, one study estimated the cost of misdiagnosis of PNES at approximately $100,000 per patient.[21] On the other hand, efficient diagnosis was shown in another study to reduce total seizure-related medical charges by 84% in 6 months.[18] Although some preliminary research has shown that intensive short-term dynamic psychotherapy may be cost-effective, further studies are needed to evaluate the impact on healthcare costs of specific treatment modalities for PNES.[12,20]

Crucially, growing biopsychosocial research offers new insight into the pathophysiology of this condition to aid in its management and improve prognostic outcomes.[6] This emphasizes the need for an early diagnostic strategy and access to appropriate care pathways to minimize excessive healthcare resource utilization and social costs.

11 | CLINICAL PECULIARITIES

11.1 | Emotional dysregulation

PNES is theorized to be a maladaptive response to stress or emotional dysregulation.[22] It, therefore, falls within the so-called neuroticism domain, which describes a tendency to experience stressful events with a perceptive hyperacuity.[5] Although arguably subjective, the patient exhibited a disproportionate emotional response to external stressors. Ekanayake et al. (2017) suggest that this "distress proneness" is exhibited more in PNES than in other psychogenic movement disorders.[5] In an analysis of his social history, multiple potential overlapping stressors could have contributed to these episodes. One such long-standing stressor appears to be the bereavement of close family members. More acutely, a new debilitating diagnosis and concerns about COVID-19 risk and vaccination seemed to compound his worry. Valente (2021) demonstrates using cross-sectional surveys that COVID-19 has potentially worsened PNES symptoms in these patients in various ways, causing more frequent aggravation and poor sleep quality.[15]

11.2 | Retained awareness with dissociation

Retained awareness during a "generalized convulsive state," as seen in this patient, is typical of nonepileptic seizures. The usual disruption in the frontoparietal region and subcortical areas of the thalamus and upper brain stem in patients with generalized seizures are not present in PNES. Blumenfeld described how generalized seizures disrupted the consciousness-preserving parts of the brain using fMRI and EEGs.[23]

The transient phase of unresponsiveness the patient experienced following his fits was of particular concern. Although he retained awareness of his environment, he could not speak or move. This phase is termed a dissociative state, and it is well described in PNES.[23,24] In a dissociative state, an individual's consciousness or self-awareness is disconnected from his emotions, memories, thoughts, and identity. His actions are not naturally in keeping with, or a product of, his somatic experiences and characteristic behavior.

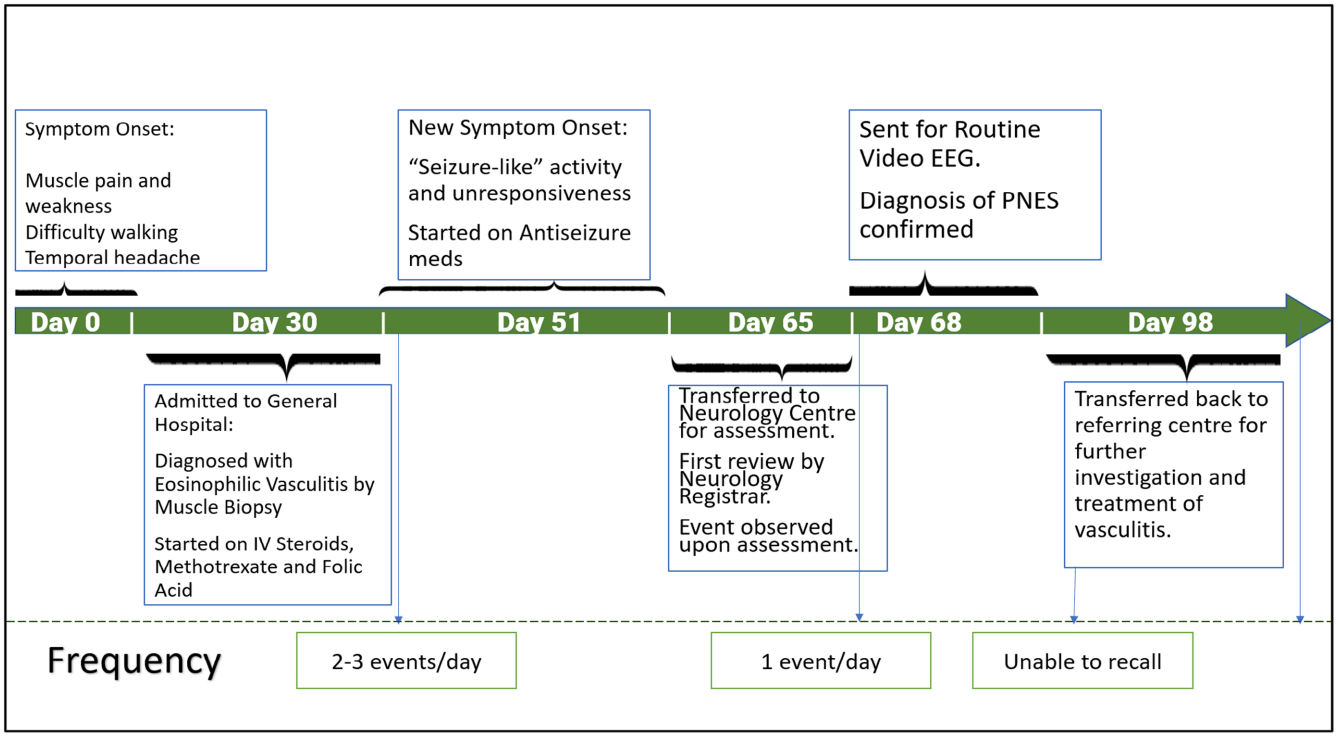

FIGURE 1 Timeline of clinical events

Van Der Krujis et al. (2012) further illustrate an interesting phenomenon in the cognitive-emotional executive control circuitry in PNES, which sheds more light on the patho-psychology underpinning these events.[24] The study highlights an aberrancy in this circuitry of a patient having a PNES, where motor function is no longer under conscious executive control but is influenced by an unstable emotional state. This aberrancy sometimes leads to involuntary limb movements, as seen in this case. This was confirmed by demonstrating a higher functional connectivity between regions of the brain associated with emotions and motor movements.[24]

11.3 | Peripheral cyanosis

Our patient suffered from peripheral cyanosis in a heightened emotional state during each convulsive event. Cyanosis is the bluish discoloration of parts of the body due to a reduction in the supply of oxygenated blood to tissues. Cyanosis has been well documented in epileptic seizures; however, in PNES, a different mechanism may be involved. Electrical disruptions can sometimes affect parts of the brain that control autonomic functions causing cardiovascular or respiratory depression, which can manifest as cyanosis.[25,26] Additionally, in 2019, a study revealed that atherosclerosis and endothelial dysfunction increase the degree of peripheral vasoconstriction in response to mental stress.[26] Therefore, the pathophysiology of this patient's cyanosis could be linked to the combination of vasomotor instability, which results from a heightened emotional state and endothelial dysfunction as a complication of the microvascular changes in diabetes and vasculitis.

Intriguingly, we observed a modest CSF protein (CSF-P) elevation without a corresponding rise in cell count, an abnormality known as albuminocytological dissociation (ACD).[27] Multiple pathophysiologies have been linked to this phenomenon.[27] For this patient, a disruption of the blood–brain or the blood-nerve barrier might seemingly explain the ACD, based on his long-standing diabetes with microvascular complications, on-going vasculitis with endothelial dysfunction, use of methotrexate, and traumatic CSF tap. However, without clinical evidence of meningeal or cerebral inflammation, the more likely explanation is a falsely raised CSF-P, as after adjusting for age, the CSF-P value falls within the range of the upper reference limit (0.62–0.66) for a 65-year-old male.[27,28] Incidentally, experts suggest that in many patients, a unilaterally elevated CSF-P cannot be linked to a diagnosis.[28]

Our team recommended numerous tests due to the non-specific clinical manifestations relayed by the referring team. We considered multiple differential diagnoses, which included cerebral vasculitis, meningoencephalitis, autoimmune/limbic encephalitis, and progressive encephalomyelitis with rigidity and myoclonus (PERM). However, we emphasize that most of these investigations are not clinically indicated when diagnosing PNES. This review underpins our previously stated

TABLE 2 Other complementary test results

Investigation	Result	Normal Range (If applicable)
Serum:		
Hb (g/L)	124	130–180
White Blood Cell count ($\times 10^9$/L)	10.7	4–11.0
Red Blood Cell count ($\times 10^{12}$/L)	3.93	4.50–6.00
Hematocrit (L/L)	0.39	0.4–0.52
Platelet count ($\times 10^9$/L)	221	150–400
Mean Cell Volume (fL)	98	80–100
Mean Cell Hemoglobin (pg)	31.6	27–33
Red Cell Distribution width (%)	10.9	11–14.8
Neutrophil count ($\times 10^9$/L)	6.2	1.7–7.5
Lymphocyte count ($\times 10^9$/L)	3.5	1.0–4.5
Monocyte count ($\times 10^9$/L)	0.8	0.2–0.8
Eosinophil count ($\times 10^9$/L)	0.3	0.0–0.4
Basophil count ($\times 10^9$/L)	0.0	0.0–0.1
Nucleated red blood cell count ($\times 10^9$/L)	0.0	0.0–0.1
Sodium (mmol/L)	142	133–146
Potassium (mmol/L)	4.7	3.5–5.3
Urea (mmol/L)	10.7	2.5–7.8
Creatinine (umol/L)	78	58–110
Estimated GFR (ml/min/1.73 m^2)	>90	
IgG (g/L)	3.70	(6–16)
IgA (g/L)	1.54	(0.8–4)
IgM (g/L)	0.16	(0.5–2)
IgG Anti-Cardiolipin antibodies (U/ml)	<2.6	(0–20)
IgM Anti-Cardiolipin antibodies (U/ml)	<1	(0–20)
IgG Anti-B2-GP1 antibodies (U/ml)	<6.4	(0–20)
IgM Anti-B2-GP1 antibodies (U/ml)	<1.1	(0–20)
Anti-ENA antibody screen	Negative	Negative
Anti-Ds-DNA antibody	Negative	Negative
Erythrocyte sedimentation rate (mm/h)	9	0–15
C-reactive protein (mg/L)	<5	<5
Creatinine kinase (U/L)	31	40–320
TSH (mU/L)	0.73	0.27–4.20
Free T4 (pmol/L)	16.3	11.0–25.0
Bilirubin (umol/L)	4	<21
Protein (g/L)	55	60–80
Albumin (g/L)	37	35–50
Globulin (g/L)	18	19–47
Alkaline phosphatase (U/L)	73	30–130
Alanine transaminase (U/L)	164	<41
Anti-GABAb receptor	Negative	Negative
Anti-AMPA1 antibody	Negative	Negative
Anti-AMPA2 antibody	Negative	Negative
Glycine receptor antibody	Negative	Negative
Anti-GAD antibody	Negative	Negative
Anti-CASPR2 antibody	Negative	Negative
Anti-LGI1 antibody	Negative	Negative

(Continues)

TABLE 2 (Continued)

Investigation	Result	Normal Range (If applicable)
Anti-NDMA receptor antibodies	Negative	Negative
Anti-Yo antibodies	Negative	Negative
Anti-Hu antibodies	Negative	Negative
Anti-Ri antibodies	Negative	Negative
QuantiFERON TB ELISA	Negative	Negative
Investigation	**Result**	**Normal Range (If applicable)**
CSF Microbiology:		
White Blood Cell count ($\times 10^6$/L) Microbiology	87	
Red Blood Cell count ($\times 10^{12}$/L) Microbiology	5	
Gram Stain	No organisms seen	No organisms seen
Culture	No growth seen	No growth seen
CSF Polymerase Chain Reaction (PCR) Panel		
Herpes simplex Type 1 PCR	DNA not detected	DNA not detected
Herpes simplex Type 2 PCR	DNA not detected	DNA not detected
Varicella-zoster PCR	DNA not detected	DNA not detected
Meningococcal PCR	DNA not detected	DNA not detected
Pneumococcal PCR	DNA not detected	DNA not detected
Enterovirus PCR	DNA not detected	DNA not detected
Investigation	**Result**	
Electromyography (EMG)	No convincing EMG evidence of myopathy or myositis	
MRI (Cervical, Thoracic, Lumbar, Sacral Vertebrae)	No acute abnormal findings. No cord edema. No diffuse changes noted	
MRI Brain	The brain parenchyma intensities appear unremarkable. No suggestion of focal edema or vasculitis. No restricted diffusion or recent infarction. No mass effect was noted. No bleeding. Other structures appear unremarkable.	
CT Head × 2	Nil acute abnormal findings	

caution in dissuading over-investigation and encourages evidence-based diagnostic stewardship in PNES.

The anomalies above demonstrate the diversity of so-called "red herrings" in the diagnostic process. A high index of suspicion is therefore essential when differentiating epileptic seizures from functional seizures, especially in the presence of confounding comorbidities. The next challenge is ensuring patients are managed appropriately according to their unique needs.

12 | WHAT DO PATIENTS NEED TO KNOW?

Early empathetic patient education is key to ensuring patients understand their condition and trust their on-going treatment. One suggestion of how this condition could be expressed to patients is as follows:

In psychogenic nonepileptic seizures, a "stress attack" that affects the function of the brain triggers physical symptoms like irregular muscle contractions, shaking, pain and weakness. Although triggered by stressors in a large percentage of patients, in a few cases, the stressor is unknown. Despite the absence of a structural or physical problem, it is just as limiting and does require treatment.

Explaining the condition in layman's tongue, contextualizing to patient experience, and pausing for consideration, are all ways of ensuring the right message is conveyed in an appropriate tenor.

13 | WHAT CAN THEY DO?

Upon telephone follow-up, the patient could not explain his condition; a critical reminder that confirmation of understanding is the next crucial step following patient education. Encouraging self-guided learning by providing tools such as leaflets and reliable health websites and introducing patients to a community of others dealing with PNES may decrease truancy to care.[11] Medical

professionals are encouraged to embrace and advocate for "eHealth literacy" by providing web-based tools such as videos, graphics, and social media community pages.[15]

14 | WHAT CAN WE DO FOR THEM?

Occasionally, family members and even health professionals may accuse patients with PNES of faking their symptoms.[21] In these instances, physicians thereby embark on rebuilding trust following a breakdown in communication, disappointment, and delays in treatment. While not elicited during this presentation, the patient would benefit from a detailed, systematic psychiatric evaluation assessing family, social, financial, and employment challenges.[1,11,16] Psychotherapy remains a validated treatment modality in PNES.[10] In a large, multicenter randomized controlled trial in the UK (CODES, 2020)[29], researchers observed more favorable clinically relevant secondary outcomes in patients receiving cognitive-behavioral therapy (CBT), such as coping with PNES diagnosis and overall quality of life in addition to specialist care.[1]Generally, the involvement of a multidisciplinary team consisting of a specialist neurologist, psychologist/psychiatrist, social worker, community health worker, and others in holistic care is ideal.[17] Other current recommendations include stress management through therapeutic mindfulness, physiotherapy and rehabilitation, taught coping skills, and therapeutic massages.[18,19]

Therapeutic management of PNES requires careful revision and, at times, discontinuation of antiseizure medications. It is worth repeating that overtreatment with these medicines, which could carry severe adverse effects, is not uncommon. In a few cases, while withdrawing antiseizure medications, unmasked seizure disorders have been described; therefore, tapering was done under neurologist supervision to monitor for these potential events. Furthermore, Oto (2005) suggests that tapering can be achieved safely following clear instructions to GPs and patients in the outpatient setting.[30] However, in the same study, the small proportion of patients who unilaterally stopped antiseizure drugs without supervised weaning did not experience adverse effects.[30] It could be argued then that abruptly discontinuing these medications may benefit patient independence with few negative consequences; however, more research is needed.

15 | CONCLUSION

Although there are numerous pitfalls to PNES diagnosis, it remains a common presentation in in- and outpatient settings. As we have underscored in this review, patients with PNES are, in fact, frequently misdiagnosed and treated for epilepsy. This difficulty may be attributable to a well-described knowledge gap in healthcare workers managing these patients presenting with diverse symptoms outlined in our case. We have highlighted the etiology of the maladaptive stress response, cyanosis, and dissociation as crucial clinical peculiarities in the diagnosis addressed sparingly in the literature. Clarifying doubts while providing tools for self-guided learning is paramount to trust-building. Patients may be more receptive to a care plan that emerges organically through open, honest conversation in which they can raise concerns about their new diagnosis. Finally, we have yet to understand the true impact of a global 'stressor' such as the current COVID-19 pandemic related to PNES burden. There is a gap for fully powered, robust studies evaluating this and the efficacy of multidisciplinary treatment modalities. We question what this could mean for imminent global catastrophes if we fail to address this correlation now.

AUTHOR CONTRIBUTIONS

Both AU and TR made equal contributions to this paper's write up and iterative editing. AU's role was case identification, developing patient case summary and obtaining patient consent. TR contributed to the literature review, graphic design, and case summary. Both authors made equal contributions to case discussion, recommendations, and conclusion. Furthermore, we acknowledge Constantinos Demetriou, a graduate entry medical student who contributed to data collection and patient communication. All authors have read and approved the final manuscript for submission. Both authors have agreed to be accountable for all aspects of the work. We will ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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CONFLICT OF INTEREST

All authors declare that they have no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

CONSENT

Throughout this process, we have taken great care to protect the confidentiality of the patient, who has provided his consent without coercion. Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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