

## Review

# Anthropometric, Microscopic and Neuroimaging Markers of Somatization in Psychogenic Non-Epileptic Seizures: A Narrative Review

**Aisha Abdulrauf<sup>1,2\*</sup>, Abdulmuiz Oluwatobi Adebisi<sup>1,3</sup>, Ajayi Praise-God<sup>1,4</sup>,  
Tooba Noor ul Ieman<sup>2</sup> and Wahab Kehinde Idris<sup>2</sup>**

<sup>1</sup>Department of Anatomy,  
College of Health Sciences, University of Ilorin, Ilorin, Nigeria.

<sup>2</sup>Department of Research and Development,  
Awibi Health, Ilorin, Nigeria.

<sup>3</sup>Department of Human Anatomy,  
College of Health Sciences, Al-Hikmah University, Ilorin, Nigeria.

<sup>4</sup>Department of Human Anatomy,  
College of Health Sciences, Federal University Lokoja, Lokoja, Nigeria.

**\*Corresponding Author Email:** aishaabdulrauf05@gmail.com

## ABSTRACT

*Paroxysmal episodes similar to epileptic seizures are called psychogenic non-epileptic seizures (PNES) however, they do not have a corresponding epileptiform correlate on video electroencephalography (vEEG) which is unarguably the current diagnostic gold standard. PNES are well known to be characterized by psychological distress regarding somatic expression but the biological somatization of PNES remains incompletely defined. This narrative review probes evidences on anthropometric (body mass index and waist circumference), structural/volumetric ("microscopic"), and neuroimaging correlates of somatization in PNES. PubMed, Google Scholar, ScienceDirect and ResearchGate were used to generate literature using series of key terms "psychogenic non-epileptic seizures", "somatization", "anthropometric markers", "microscopic markers" and "neuroimaging markers". Various evidences show that patients with PNES carry more than psychological dysfunction but a disproportionately high burden of obesity similar to epileptic cases. Also, volumetric imaging revealed reduced volume of amygdala, hippocampus and altered uncinate fasciculus microstructure. Although, some findings across structural and functional neuroimaging studies are inconsistent but they converge on the limbic-prefrontal emotion-regulation networks dysfunction rather than a single discrete lesion. vEEG therefore remains indispensable, while anthropometric and neuroimaging findings offer adjunctive, biologically informative but not diagnostic, insight into the somatization construct that underlies PNES.*

**KEYWORDS:** Psychogenic non-epileptic seizures; Somatization; Anthropometry; Amygdala; Neuroimaging; Conversion disorder.

## INTRODUCTION

Many conditions similar seizure are very common in emergency departments leading to admission to intensive care units for a neurological complaint<sup>1</sup>. Psychogenic non-epileptic seizures (PNES) are encountered frequently by clinicians as they strongly mimic epileptic seizures due to their sudden, dramatic presentation posing a recurring challenge in diagnosis for emergency physicians, neurologists and psychiatrists in-charge. It is now classified as one of the most common manifestations of functional neurological symptom disorder (FND), popularly known as conversion disorder<sup>2</sup>. However, with the adoption of video electroencephalography (vEEG), clinicians' confidence in diagnosing PNES has improved substantially which allows clinicians to critically differentiate epileptic seizures (ES) from PNES<sup>3</sup>.

PNES are neurobehavioral conditions that rely on the convergence between neurology and psychiatry; the DSM-5 classifies them within the conversion (functional neurological symptom) disorders, while the ICD-10 places them among the dissociative disorders. Epidemiologically, the incidence and prevalence of PNES are 1.4 - 4.9 per 100,000 per year and 2 - 33 per 100,000, with a significant proportion of patients carrying a dual diagnosis of epilepsy and PNES<sup>4</sup>. Usually, the onset of PNES occurs during adolescence and early adulthood which disproportionately affects females. This pattern is characterized by the combination of neurobiological, psychosocial and trauma-related vulnerability factors<sup>5</sup>.

Jafari et al. (2020) characterized the neurobiological parameters of PNES similar to other literatures, however, less attention has been paid to other classes of biological markers, such as anthropometric indices, structural or volumetric brain correlates, that might further establish the somatization process underlying the PNES disorder<sup>6,7</sup>. This narrative review explores current evidence on anthropometric, structural/volumetric, and neuroimaging correlates of somatization in PNES to collectively examine and consider what the findings further imply about the biology of the disorder.

## METHODS

Literature was identified through scientific search engines such as PubMed, Google Scholar, ScienceDirect and ResearchGate. Key terms including "psychogenic non-epileptic seizures" or "PNES", "somatization", "anthropometric markers", "body mass index", "waist circumference", "histopathology", "amygdala", "hippocampus", "magnetic resonance imaging", "diffusion tensor imaging", "functional MRI" and "video electroencephalography" were used to extract literatures. Studies were restricted to English-language publications involving adult patients with PNES diagnosed by clinical assessment or vEEG, with case reports and single-author commentaries excluded; key systematic reviews and meta-analyses were also retained.

## RESULTS

### Anthropometric Markers of Somatization

One of the most accessible and low-cost indices that might reflect the somatic burden of PNES is body habitus. The study conducted by Marquez et al. compared body mass index (BMI) in 46 patients with PNES and 46 age- and sex-matched patients with epilepsy. This study found significantly higher BMI in the PNES group (30.5 vs 26.1 kg/m<sup>2</sup>,  $p = 0.006$ ) with a difference that persisted after accounting for the weight-promoting effects of antiepileptic drugs<sup>8</sup>. Abubakr and Wambacq's study further supported this finding by reporting that 69.3% of a PNES cohort were overweight or obese, this rate is statistically comparable to the 55.2 - 72% range reported in epilepsy populations and higher than general-population estimates<sup>9</sup>. Because anti-seizure medication and reduced physical activity can significantly contribute to obesity in epilepsy, and because a comparable rate is observed in PNES, these findings corroborate the interpretation that excess adiposity in PNES reflects a more generalized pattern of psychosocial and behavioral dysregulation, comorbid depression, reduced physical activity secondary to disability, psychotropic medication use, and possible hypothalamic-pituitary-adrenal axis dysregulation rather than a direct neurophysiological consequence of seizure activity itself despite the absence of true ictal cortical discharge<sup>8,9</sup>.

Studies regarding other anthropometric indices such as waist circumference (WC), waist-hip ratio (WHR), Body mass index (BMI) and skinfold measures in relation to PNES are significantly limited, and existing reports rarely apply standardized anthropometric protocols or population-specific reference ranges. This heterogeneity limits comparison across studies and represents a clear gap for future prospective work.

### Structural and Microscopic Correlates

Epileptogenic structural lesions and brain biopsy are usually neither indicated nor ethically justified in these PNES patients. This is why true histopathological data on brain tissue in PNES are essentially very limited. Consequently, "microscopic" evidence in PNES is usually derived indirectly and chiefly by depending on high-resolution volumetric MRI morphometry rather than from basic tissue histology. However, for epilepsy, studies have documented its histological characterization by hippocampal sclerosis, neuronal loss and astrogliosis<sup>6</sup>. Nonetheless, morphometric correlates are obtained from volumetric studies of PNES provided that they plausibly reflect the same limbic circuitry implicated in somatization.

Using a semi-automated segmentation approach, the study by Tatekawa et al. revealed significantly smaller left amygdala and right hippocampus in patients with dissociative seizures than in healthy patients (controls), this is independent of psychiatric comorbidity; however, this difference was not statistically significant. With the ipsilateral structure volume obtained, it is suggestive of atrophy which may reflect broader

network-level change rather than a focal lesion<sup>10</sup>. This finding is consistent with a wider cross-sectional MRI study reporting a significantly higher-than-expected prevalence of structural brain abnormalities in patients with PNES<sup>14</sup>. The study by Hernando *et al.* using diffusion tensor imaging (DTI) demonstrated a marked rightward asymmetry in uncinate fasciculus connectivity which is the principal white-matter tract linking the amygdalohippocampal complex to the medial prefrontal cortex at the white-matter level in patients with PNES compared with healthy patients (controls)<sup>11</sup>. The study by Lee *et al.*, via complementary tract-based spatial statistics, found increased fractional anisotropy in the left corona radiata, internal and external capsules, superior temporal gyrus and uncinate fasciculus of patients with PNES<sup>12</sup>, while more recent neurite-orientation imaging has confirmed that white-matter and neurite microstructure differ between patients with PNES and healthy patients (controls)<sup>13</sup>. These structural findings implicate the amygdala-hippocampal-prefrontal emotion-regulation network as a recurring, if inconsistent, correlate of PNES, rather than pointing to a single discrete or histologically defined lesion.

### Neuroimaging Markers

Although individual findings are heterogeneous, neuroimaging studies in PNES have shown structural and functional brain abnormalities affecting sensorimotor, emotion-regulation and executive-control networks in patients with PNES<sup>15</sup>. Altered connectivity between regions implicated in dissociation and emotional dysregulation has been revealed variably by functional MRI studies, including the amygdala, orbitofrontal cortex, anterior cingulate cortex and default-mode network nodes, again supporting a network-based rather than localizationist model of the disorder<sup>6,7</sup>. Despite these advances, there are yet to be structural or functional neuroimaging indices that possess sufficient sensitivity or specificity to be used diagnostically at the individual-patient level; vEEG remains the only tool that directly demonstrates the absence of epileptiform activity during a typical event in PNES<sup>3</sup>.

Basal hyper-cortisolism, blunted post-ictal prolactin rise, and altered post-ictal ghrelin and nesfatin levels are non-neuroimaging biomarkers that have also been proposed as adjuncts to differentiate PNES from ES, further illustrating the broader biopsychosocial character of somatization in this disorder rather than a single unifying biological signature<sup>6</sup>.

### DISCUSSION

This review narrates available evidence that establishes a distributed pattern of dysfunction in PNES. This includes a psychosocial disability plausibly defined by a body habitus skewed toward overweight and obesity and reduced activity rather than a direct neurophysiological effect of seizures<sup>8,9</sup>. Furthermore, the subtle, inconsistent reductions in amygdala and hippocampal volume together with altered uncinate

fasciculus and broader white-matter microstructure<sup>10-13</sup> corroborate these findings structurally; while functional connectivity changes within limbic-prefrontal networks governing emotion regulation, interoception and executive control further support this pattern<sup>15</sup>. This pattern is compatible with contemporary biopsychosocial and network-based models of functional neurological disorder, in which symptoms arise from altered communication between limbic, salience and motor-control networks rather than from a discrete structural lesion<sup>2,6</sup>. However, these parameters do not converge on any single reliable biomarker of PNES or of the somatization construct that underlies it.

Due to the limited rate of PNES presentations, there are several limitations recurring across PNES studies which include typically small sample sizes (often fewer than 30 patients per group). Additionally, comparator groups (healthy controls versus patients with epilepsy versus psychiatric controls) vary considerably, while few studies adequately control for psychotropic medication use, comorbid depression, anxiety or post-traumatic stress disorder, or illness duration, all of which can independently influence body composition and brain structure. Most neuroimaging studies involved are predominantly cross-sectional, precluding any causal inference as to whether the observed anthropometric and structural changes precede, accompany, or result from the onset of PNES.

From a clinical standpoint, these findings strongly reinforce rather than displace the central role of vEEG and structured psychiatric assessment in diagnosis. However, anthropometric screening and neuroimaging findings should be regarded as adjunctive contextual information used to potentially characterize the somatic burden of illness and guide multidisciplinary management rather than as stand-alone diagnostic tools.

### CONCLUSION

This narrative review confirms that video electroencephalography remains the gold standard for diagnosis, and comprehensive psychological and behavioral evaluation continues to be the most valuable tool for understanding and managing somatization in PNES. Anthropometric data is critical as a major highlight of disproportionate burden of overweight and obesity among patients with PNES; structural and diffusion imaging point toward subtle limbic and white-matter alterations rather than a discrete lesion; and functional neuroimaging implicates disrupted emotion-regulation networks. No single anthropometric, structural, or neuroimaging marker currently provides definitive diagnostic evidence for psychogenic non-epileptic seizures. Larger, prospective, multicenter studies with standardized anthropometric protocols and harmonized imaging methodology are needed to clarify whether these biological

correlates can ultimately be translated into clinically useful markers.

## DECLARATIONS

**Funding:** None Declared.

**Conflict of Interests Disclosure:** The author(s) have declared that no competing interests exist.

**Declaration of Generative AI and AI-Assisted Technologies:** The author(s) declare that no generative AI or AI-assisted technologies were used in the preparation of this manuscript.

## ARTICLE INFORMATION

**Accepted for Publication:** June 06, 2026

**Published:** June 27, 2026

**Peer Review:** Externally Peer Reviewed.

**Open Access:** This is an open access article distributed in accordance with the Creative Commons Attribution Non-Commercial (CC BY- NC 4.0) license, which permits others unrestricted use, distribution, and reproduction in any medium, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial.

**Copyright:** © 2026 By The Authors

## REFERENCES

- Martindale JL, Goldstein JN, Pallin DJ. Emergency department seizure epidemiology. *Emerg Med Clin North Am.* 2011;29(1):15-27.
- Stone J. Functional neurological symptoms. *J R Coll Physicians Edinb.* 2011;41(1):38-41.
- LaFrance WC Jr, Reuber M, Goldstein LH. Management of psychogenic nonepileptic seizures. *Epilepsia.* 2013;54(Suppl 1):53-67.
- Anzellotti F, Dono F, Evangelista G, Di Pietro M, Carrarini C, Russo M, et al. Psychogenic non-epileptic seizures and pseudo-refractory epilepsy, a management challenge. *Front Neurol.* 2020; 11:461.
- Asadi-Pooya AA, Emami M, Emami Y. Gender differences in manifestations of psychogenic non-epileptic seizures in Iran. *J Neurol Sci.* 2013;332(1-2):66-68.
- Jafari A, Rezaei Tavirani M, Parvareshi Hamrah M, Ahmadi Karvigh S, Bashi Zadeh Fakhari H. Psychogenic non-epileptic seizures; a narrative review. *Arch Acad Emerg Med.* 2020;8(1): e10.
- Sundararajan T, Tesar G, Jimenez XF. Biomarkers in the diagnosis and study of psychogenic nonepileptic seizures: a systematic review. *Seizure.* 2016; 35:11-22.
- Marquez AV, Farias ST, Apperson M, Koopmans S, Jorgensen J, Shatzel A, et al. Psychogenic nonepileptic seizures are associated with an increased risk of obesity. *Epilepsy Behav.* 2004;5(1):88-93.
- Abubakr A, Wambacq I. The rate of obesity in psychogenic non-epileptic seizures: a retrospective study. *J Neurol Res.* 2018;8(1-2):1-3.
- Tatekawa H, Kerr WT, Savic I, Engel J Jr, Salamon N. Reduced left amygdala volume in patients with dissociative seizures (psychogenic nonepileptic seizures). *Seizure.* 2020; 75:43-48.
- Hernando KA, Szaflarski JP, Ver Hoef LW, Lee S, Allendorfer JB. Uncinate fasciculus connectivity in patients with psychogenic nonepileptic seizures: a preliminary diffusion tensor tractography study. *Epilepsy Behav.* 2015; 45:68-73.
- Lee S, Allendorfer JB, Gaston TE, Griffis JC, Hernando KA, Knowlton RC, et al. White matter diffusion abnormalities in patients with psychogenic non-epileptic seizures. *Brain Res.* 2015; 1620:169-176.
- Goodman AM, Allendorfer JB, Blum AS, Bolding MS, Correia S, Ver Hoef LW, et al. White matter and neurite morphology differ in psychogenic nonepileptic seizures. *Ann Clin Transl Neurol.* 2020;7(10):1973-1984.
- Asadi-Pooya AA, Homayoun M. Structural brain abnormalities in patients with psychogenic nonepileptic seizures. *Neurol Sci.* 2020;41(3):555-559.
- McSweeney M, Reuber M, Levita L. Neuroimaging studies in patients with psychogenic non-epileptic seizures: a systematic meta-review. *Neuroimaging Clin.* 2017; 16:210-221.