# A Case of Panic Disorder Misdiagnosed as Epilepsy for 9 Years in a Young Male

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**Abstract** Panic disorder can be misdiagnosed as epilepsy and vice versa, which, in turn, can impact the patient, their family, and the healthcare system. Here, we describe a rare case of a 22-year-old male with a 9-year history of misdiagnosed drug-resistant epilepsy. On presentation to our hospital, the patient's physical examination and other investigations were unremarkable. The attacks were reported to last for about 5–10 minutes and were related to interfamilial distress. He reported feeling anxious about having an attack, experiencing palpitations and sweating before and during episodes, feeling chest tightness, derealization, and fearing loss of control, based on which a diagnosis of panic disorder was made. The patient was treated with 12 sessions of cognitive behavioral therapy, following which all his antiepileptic medications were stopped over 8 weeks. The dose of sertraline was increased and maintained at 200 mg once daily and was gradually stopped after 6 months of remission. This case highlights that panic disorder should be considered as a differential diagnosis of phyperventilation syndrome can be diagnosed differently by neurologists, psychiatrists, and other specialists.

**Keywords:** Clinical reasoning, cognitive bias, differential diagnosis, epilepsy, misdiagnosis, panic attacks, panic disorder

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#### **INTRODUCTION**

The rate of misdiagnosis of epilepsy is high, with about 20% of the cases being misdiagnosed.<sup>[1]</sup> Therefore, neurologists are urged to reconsider the diagnostic accuracy of epilepsy, especially when there is a poor treatment response.<sup>[1]</sup> Panic disorder (PD) being misdiagnosed as epilepsy is rare,<sup>[2,3]</sup> but its negative impact on the patients and their family can be significant. Therefore, PD, which is one of the most common psychiatric conditions, should be distinguished from epileptic disorders.<sup>[3]</sup>

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Panic attacks (PAs) are defined as a sudden intense fear response along with any 4 of the following 13 symptoms: palpitations, shaking, paresthesia, lightheadedness, sweating, nausea or abdominal cramps, chest pain/ discomfort, chills or hot flushes, shortness of breath, feeling of choking, fear of losing control or going crazy, fear of dying, and derealization or depersonalization.<sup>[4]</sup> According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), if a patient has recurrent and unexpected PAs followed by one month or more of fearing another attack or worrying about the consequences of such attacks or a significant change in

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behavior related to the attacks, a diagnosis of PD can be made.  $\ensuremath{^{[5]}}$ 

Here, we describe a rare case of a PD being misdiagnosed as epilepsy for 9 years in a young university student.

## CASE REPORT

A 22-year-old male with a prior diagnosis of epilepsy was referred to our hospital by his father for a second opinion. At the time of presentation, the patient had been under the care of a consultant neurologist and was maintained on the following medications: sodium valproate 500 mg three times daily, lamotrigine 50 mg once daily, sertraline 50 mg in the morning, clonazepam 2 mg every night (as needed), amisulpride 100 mg every night, and quetiapine 50 mg every night.

At the age of 13 years, the patient had presented to an Emergency Department with generalized shaking, rigidity, and groaning. He was evaluated by two neurologists and had multiple negative EEG recordings. A diagnosis of epilepsy was made based on the weekly occurrence of "seizure attacks," and he was started on several antiepileptic medications during these 9 years, but with negligible response.

His physical examination and other investigations at the time of presentation to our hospital were unremarkable. The first and the subsequently reported "attacks" each lasted about 5–10 minutes and appeared to be associated with interfamilial distress. He reported initially feeling anxious and worried about having an "attack." In addition, he experienced palpitations and sweating before and during the episode as well as felt chest tightness and butterflies in his stomach. He experienced feeling of derealization and fearing losing control during every attack. There was no family history of epilepsy or anxiety disorders.

Given the classical features of PD, a working diagnosis of such was made and the patient was advised to start 12 sessions of cognitive behavioral therapy (CBT) with the aim of gradually reducing his antiepileptic medications according to improvement in his condition. CBT sessions included psychoeducation about the diagnosis of PD to collaboratively switch the diagnostic conviction from epilepsy to PD. One attack was captured on video, which was presented to a neurologist, who believed that in context of the clinical findings, it represented psychogenic nonepileptic attacks (PNEA). Clark's CBT model<sup>[6]</sup> was used to familiarize the patient with how selective attention biases our interpretations of panic physical symptoms to be dangerous and life threatening and how this was not accurate. Relaxation techniques and panic induction were used to enable better control of PA-triggered hyperventilation syndrome. His PAs reduced gradually from once a week to once every 3–4 weeks and he was able to control such attacks. As the patient finished his sessions, and in consultation with the neurologist, all his maintenance medications, except sertraline, were gradually stopped over 8 weeks. The dose of sertraline was increased to 200 mg once daily, and thereafter it was gradually reduced and stopped after 6 months of remission.

# DISCUSSION

In general, PAs are more likely to be experienced during a lifetime than a diagnosis of PD. For example, in a nationally representative, non-institutionalized Spanish population, the lifetime prevalence of PA was 7.6%, while that of a diagnosis of PD was 2.4%.<sup>[7]</sup> Given the shared physical manifestations of panic and epileptic attacks, patients with PNEAs account for a large proportion of referrals to the neurology clinics, and thus are most likely to be misdiagnosed.<sup>[1,2]</sup> Similar to our case, it has been reported that patients with PNEAs who were misdiagnosed as epilepsy are likely to have received their misdiagnoses 7-10 years previously.<sup>[1]</sup> Therefore, clinicians should have a higher index of suspicion when dealing with refractory epilepsy, given that the physical, psychological, and financial impact of misdiagnosis on a patient, their family, and society are extremely high.[8]

Clinically, hyperventilation syndrome is related with PD and neurological physical symptoms such as muscle spasms, paresthesia, weakness and dizziness.<sup>[9]</sup> Tachypnoea ensues in the context of an adrenergic surge with subsequent hypocapnia, and respiratory alkalosis. Vasoconstriction in the brain results in experiencing dizziness and reduced level of consciousness, while bronchospasm causes chest tightness. Alkalotic blood, in turn, reduces the total and ionized calcium levels, while hypomagnesemia and hypokalemia lead to hyperexcitable neurons (paresthesia) and tetany (finger clawing and shaking).<sup>[10]</sup> Due to the shared physical manifestations between seizures and PAs, such syndrome can be viewed differently by psychiatrists and neurologists.

Another potential reason for such misdiagnosis can be mental illness-associated stigma, which, in Saudi Arabia, is especially high among outpatient physicians and could result in referral delays.<sup>[11,12]</sup> Similarly, cognitive bias often plays a role in the diagnostic errors of physicians. Therefore, given the commonality in misdiagnosis of epilepsy, studies could be conducted to determine cognitive biases at play during such misdiagnosis, which would increase the awareness of the same among clinicians.<sup>[13]</sup> One of the commonly known cogitative biases among clinicians is confirmation bias, wherein clinicians tend to look for supporting rather than refuting evidence to reassess clinical opinions.<sup>[14]</sup>

### CONCLUSION

PD can be treated using CBT and with or without anti-anxiety medications. A focus on the biopsychosocial aspect of the patient and across specialty referrals are potentially valuable in the differential diagnosis of PD and epilepsy.

### Declaration of patient consent

The author certifies that he has obtained all appropriate patient consent forms. In the form, the patient has given consent for his clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

#### Peer review

This case report was peer-reviewed by three independent and anonymous reviewers.

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#### **Conflicts of interest**

There are no conflicts of interest.

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