

Psychogenic unresponsiveness: a functional neurological disorder on the border zone between neurology, clinical psychology & psychiatry

Abstract

Psychogenic unresponsiveness is a rare manifestation of functional neurological disorder (FND), presenting as apparent loss of consciousness without structural, metabolic, or toxic abnormalities. Its close resemblance to coma and related DOC places it at a challenging diagnostic intersection of neurology, clinical psychology, and psychiatry. We describe a patient with prolonged unresponsiveness who underwent extensive acute neurological evaluation. Unremarkable neuroimaging and electrophysiological studies and preserved brainstem reflexes were inconsistent with true coma. A multidisciplinary assessment ultimately led to the diagnosis of psychogenic unresponsiveness. Our case underscores the diagnostic complexity of psychogenic unresponsiveness and highlights the importance of early recognition to avoid unnecessary interventions and iatrogenic harm. Integrated care across neurology, psychiatry, and clinical psychology is essential for accurate diagnosis and optimal outcomes.

Keywords: functional neurological disorder, FND, Guillain Barre Syndrome (GBS), weakness

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Introduction

Psychogenic unresponsiveness, also known as functional unresponsiveness or dissociative stupor, is a rare and diagnostically complex manifestation of functional neurological disorder (FND). It presents as an apparent loss of consciousness or responsiveness without identifiable structural, metabolic, or toxic abnormalities, placing it at the intersection of neurology, clinical psychology, and psychiatry. Clinically, it may closely resemble coma or other disorders of consciousness (DOC), often prompting extensive neurological work-up. Conceptualized within the framework of FND as a disorder of altered brain network functioning rather than structural disease, psychogenic unresponsiveness is typically associated with preserved brainstem reflexes, normal neuroimaging and electrophysiological findings incompatible with true coma. Despite this, the striking presentation can lead to diagnostic uncertainty, delayed identification, and unnecessary medical interventions. Psychological stressors, trauma, and psychiatric comorbidities are commonly implicated, though they may not be immediately apparent at presentation. This case report describes a patient with psychogenic unresponsiveness to emphasize key diagnostic considerations and highlight the importance of early recognition and the importance of multidisciplinary approach integrating neurology, psychiatry, and clinical psychology subspecialties.

Case report

A 20-year-old female, second-year medical student with no previous medical history came to the emergency room with an alleged history of ingestion of paint thinner followed by unresponsiveness. On arrival in the emergency she was unresponsive with GCS 3/15, PR=100/min, BP=90/60 mmHg, SpO2 80% on room air. She was electively intubated to secure the airway. Routine blood investigations were unremarkable. ABG revealed normal anion gap metabolic acidosis with high lactate levels. Toxicology screening and routine blood tests came back negative. Neurology consultation was requested. Patient was unresponsive with a non-focal exam, DTRs were bilaterally brisk,

plantar reflexes were mute and there was no neck rigidity. EEG showed normal organized background with a posterior dominant rhythm of 9 Hz. There were no interictal epileptiform discharges noted. CT head without contrast and MRI brain without contrast showed no acute intracranial pathology. Psychogenic unresponsiveness was suspected. After a weaning trial, she was successfully extubated.

Discussion

Psychogenic unresponsiveness represents a rare and severe manifestation of functional neurological disorder (FND) and presents significant diagnostic and therapeutic challenges. Owing to its close clinical resemblance to coma and other disorders of consciousness, patients frequently undergo urgent neurological assessment in emergency or intensive care settings. In the present case, comprehensive investigations were required to exclude life-threatening structural, metabolic, and toxic causes before a functional diagnosis could be established as a diagnosis of exclusion. This course mirrors the typical clinical pathway in psychogenic unresponsiveness and underscores the necessity of systematically excluding organic pathology.¹⁻⁴

Diagnostic considerations and differential diagnosis

Differentiating psychogenic unresponsiveness from conditions such as true coma, akinetic mutism, catatonia, non-convulsive status epilepticus, and metabolic encephalopathy remains a clinical challenge. Detailed bedside neurological examination is essential. Preserved pupillary responses, intact corneal and gag reflexes, normal respiratory patterns, and resistance to eye opening are commonly reported features inconsistent with structural disorders of consciousness. In this case, preserved brainstem reflexes, normal electrophysiological and neuroimaging findings further supported a functional rather than organic aetiology.^{1-3,7}

Ancillary investigations, including neuroimaging and electroencephalography, are indispensable for excluding organic disease and are typically unremarkable in psychogenic

unresponsiveness. Normal investigations alone are insufficient to establish a functional diagnosis, their integration with non-anatomical or internally inconsistent clinical findings increases diagnostic confidence. Early identification of these features is crucial to prevent repeated testing and avoid iatrogenic harm.¹⁻³

Pathophysiological perspectives

Current conceptual models view FND as a disorder of altered brain network functioning, characterized by impaired integration across systems governing attention, self-agency, emotional processing, and motor control. In psychogenic unresponsiveness, arousal mechanisms appear preserved, while voluntary responsiveness is functionally inhibited. Proposed neurobiological mechanisms include abnormal top-down modulation of consciousness-related and sensorimotor networks, potentially driven by heightened threat processing or dissociative responses. This framework provides a unifying explanation that bridges neurological and psychological perspectives⁴⁻⁶

Psychological and psychiatric dimensions

Psychological stressors, trauma, affective disorders, dissociative phenomena, and maladaptive coping strategies are frequently associated with psychogenic unresponsiveness, although they may not be immediately evident during acute presentation. In this case, a multidisciplinary approach facilitated the identification of relevant psychological contributors that were not apparent during initial neurological assessment.

Diagnostic overlap with psychiatric conditions such as dissociative disorders and catatonia further complicates evaluation. Unlike catatonia, psychogenic unresponsiveness generally lacks features such as rigidity, waxy flexibility, or echophenomena and does not respond to benzodiazepine challenge. Recognition of these distinctions is essential to ensure appropriate diagnosis and management.⁴⁻⁶

Management and communication

Management priorities include early diagnostic clarification, avoidance of unnecessary or invasive medical interventions, and effective communication with the patient and family. Providing a clear, non-stigmatising explanation of the condition's functional nature is a key therapeutic intervention and may contribute to clinical improvement. Emphasizing that symptoms are genuine and potentially reversible helps promote patient understanding and engagement^{3,4,8}

Long-term management typically involves psychological interventions, treatment of comorbid psychiatric conditions, and functional rehabilitation. Close collaboration among neurology, psychiatry, and clinical psychology is essential to address the disorder's multifactorial nature and reduce the risk of symptom persistence or recurrence.^{3-5,8} Once the diagnosis of psychogenic unresponsiveness was established, our patient was started on paroxetine (SSRI drug). She was referred to a psychiatrist as well as a clinical psychologist.

Implications for clinical practice

This case illustrates psychogenic unresponsiveness as a prototypical example of the diagnostic border zone between neurology, clinical psychology, and psychiatry. It highlights the importance of interdisciplinary assessment in patients presenting with unexplained DOC and reinforces the value of integrating neurological examination with psychological formulation. Improved awareness among acute care clinicians may facilitate earlier recognition, minimize iatrogenic harm, and ultimately enhance patient outcomes^{1,3,4}.

Conclusion

Psychogenic unresponsiveness is an uncommon and striking manifestation of FND that closely resembles coma and other DOC, situating it at the complex interface of neurology, clinical psychology, and psychiatry. This case highlights the need for careful exclusion of organic pathology while remaining alert to functional etiologies when clinical findings are inconsistent with structural disease. Timely recognition through meticulous bedside assessment, targeted investigations, and multidisciplinary collaboration is crucial to prevent unnecessary interventions and iatrogenic harm. Clear, non-stigmatizing communication and coordinated management addressing neurological, psychological, and psychiatric aspects are essential for optimal patient care and outcome. Enhanced awareness of psychogenic unresponsiveness among clinicians helps earlier diagnosis and improves clinical outcomes.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

Author contributions

GN, NKS, AM and SS drafted the manuscript. All authors contributed equally to the manuscript and share first authorship.

Disclosures

None. The views expressed are those of the authors and do not necessarily reflect the views of the organizations they work for. Written informed consent was obtained from the patient for the publication of the clinical data. A standard single-patient case report is generally not considered "research" under federal regulations (like the Common Rule) because it lacks a hypothesis and the intent to produce generalizable knowledge; thus, our manuscript did not require IRB review or a formal waiver at all.

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