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Case Report

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Functional neurological disorder masquerading as acute Guillain-Barre Syndrome

Abstract

Functional neurological disorders (FND) are characterized by neurologic symptoms that are inconsistent with structural neurological disease. Some FNDs can closely mimic acute neuromuscular conditions, often creating diagnostic challenges. We present a case of FND masquerading as GBS with acute flaccid paralysis. The relevant medical literature is discussed.

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

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Introduction

Guillain-Barré Syndrome (GBS), also referred to as Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), is an acute, immune-mediated demyelinating disorder of the peripheral nerves and nerve roots. It is characterized by rapidly progressive, usually symmetric limb weakness accompanied by reduced or absent deep tendon reflexes (flaccid paraplegia or quadriplegia). GBS typically follows an infectious episode, reflecting an autoimmune mechanism likely triggered by molecular mimicry. Infection with Campylobacter jejuni is the most common bacterial trigger. The syndrome can involve sensory, autonomic, and cranial nerves (the facial nerve is the common cranial nerve involved). Cerebrospinal fluid (CSF) analysis classically demonstrates albuminocytologic dissociation, with elevated protein but normal cell counts. Functional Neurological Disorders (FND) includes a varied group of conditions in which patients experience neurologic symptoms not explained by structural neurological disease. A dynamic lesion (*la lesion dynamique*) causing abnormal functioning of the nervous system is postulated to be the cause of the patient's clinical symptoms. These disorders hence fall in the border zone of neurology, psychiatry and psychology. Clinically, FND disorders present with signs that are inconsistent with known neuroanatomical pathways yet are internally recognizable and reproducible on examination. Manifestations may include motor symptoms (e.g., weakness, paralysis, tremor, gait disturbances, impaired consciousness), sensory symptoms (e.g., numbness, visual or auditory disturbances), or paroxysmal events resembling seizures. Diagnosis relies on identifying positive clinical features that demonstrate incongruence with organic disease rather than solely excluding other conditions.

Case report

A 29-year-old lady presented to the emergency department (ED) with complaints of lower limb weakness for the past 24 hours. She gave history of fever preceding the onset of leg weakness. On examination leg power was 1/5 bilaterally medical research council (MRC) grade. Ankle jerks were depressed as compared to other deep tendon reflexes which were all 2(+). She had no respiratory distress. A provisional diagnosis of acute GBS was considered. Nerve conduction study (NCV) B/L upper and lower limbs were normal (F waves were present and distal latencies were not prolonged). Cerebrospinal fluid (CSF) examination revealed 1 cell and protein of 18 mg/dl (normal 20-40 mg/dl). As the NCV study was normal and CSF examination revealed no albuminocytologic dissociation, it was decided to withhold IVIg or plasmapheresis and observe her closely in the intensive care unit setting. Next day when examined it was noticed that her leg weakness fluctuated between examinations. Hoover's sign (involuntary effort in the weak leg when the strong leg lifts) was elicited inconsistently (*varying Hoover's sign*). When assisted out of bed and made to walk, she struggled, displayed tremulousness of the hands and feet but was smiling at the same time. At other times her mood was labile and she would start to cry. Due to language barrier a detailed neuropsychological evaluation could not be carried out. Her lower limbs weakness self-resolved over 4 days and she was discharged home with a provisional diagnosis of FND masquerading as GBS.

Discussion

Guillain-Barré syndrome (GBS) represents the most frequent cause of acute flaccid paralysis and is clinically suspected in patients who present with rapidly evolving limb weakness, (flaccid paraparesis) especially when preceded by a febrile illness.[1,2,3] A number of neurological conditions can resemble GBS and create diagnostic uncertainty. Our case emphasizes the need to recognize FND as an important differential diagnosis in such presentations and highlights the role of meticulous clinical evaluation and serial neurological examinations.

In our patient, the acute onset of bilateral lower limb weakness following a febrile illness, along with reduced ankle reflexes, initially pointed towards early GBS. During the early phase of GBS, NCV study and CSF analysis may remain normal, complicating diagnostic decision making. The fluctuating lower limb weakness, normal NCV findings, including preserved F waves and normal distal latencies, together with the absence of albuminocytologic dissociation in CSF, made GBS less likely in our case. Although very early GBS cannot be entirely excluded on this basis, our findings made us consider a conservative approach with close clinical monitoring rather than initiating costly immunomodulatory therapy.

The diagnosis of FND in our patient was strengthened by the marked inconsistency and variability of weakness noted on repeated

examinations. Such fluctuating deficits that do not follow established neuroanatomical patterns are typical of FND and contrast with the steadily progressive, symmetric weakness characteristic of GBS.[4] Furthermore, the preservation of deep tendon reflexes and the absence of respiratory or bulbar involvement argued against an evolving demyelinating neuropathy. The spontaneous and complete resolution of symptoms without any treatment further favoured a functional disorder.[5]

FNDs are increasingly understood as disorders of impaired nervous system functioning rather than an underlying structural abnormality. Their diagnosis relies on the identification of positive clinical signs and internal inconsistency, rather than exclusion alone. Although psychological stressors are frequently associated with FND, their absence or the inability to assess them, as in this case due to language barriers does not rule out the diagnosis. It is important for clinicians to remember that symptoms experienced by patients with FND are involuntary and genuine, and mischaracterizing them as intentional can negatively impact patient care. Treatment involves a detailed neuropsychological evaluation to identify the underlying stressors. Referral to a mental health specialist (psychiatrist and/or psychologist), treatment with anxiolytics and selective serotonin reuptake inhibitors (SSRIs), cognitive behavioral therapy (CBT) and counselling may lead to better outcomes in patients with FND.

Accurate differentiation between FND and GBS is critical, as misdiagnosis of a FND patient as GBS may expose the patient to unnecessary, costly, and potentially harmful treatments, prolonged intensive care stays, and other invasive interventions such as central line placement. Conversely misdiagnosis of a GBS patient as FND may lead to delayed recognition and treatment of the acute demyelinating neurology resulting in significant morbidity and mortality. Our case underscores the importance of serial neurological assessments, thoughtful interpretation of investigative tests, and close observation for symptom progression in diagnostically uncertain cases.

Variants and mimics of GBS have been reported in the medical literature.[6–9] Clinicians should keep FND as a consideration in the differential diagnosis of acute limb weakness resembling GBS. Greater awareness of the distinguishing clinical features of FND and prudent use of diagnostic investigations can help prevent misdiagnosis and ensure appropriate patient management.

Conclusion

Functional neurological disorder (FND) may closely resemble acute neuromuscular disorder such as GBS, creating diagnostic challenges and the risk of unnecessary treatment. Our case highlights the importance of thorough clinical evaluation, serial neurological assessments, and careful interpretation of investigations to help distinguish FND from GBS even when features are initially suggestive of GBS. Identification of positive functional signs and symptom variability can help avoid unwarranted interventions, alleviate patient anxiety, and ensure safe, accurate, and timely care.

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Author contributions

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

Disclosures

The views expressed are those of the authors and do not necessarily reflect the views of the organizations they work for. Informed consent was obtained to publish the clinical data from the patient. Since this is a case report, IRB approval was waived off.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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