CRCP Case Reports in Clinical Practice

Case Report

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A Missed Diagnosis of Guillain-Barre Syndrome Masquerading as Stroke in an Acute Medical Unit: A Case Report

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Citation Madu A, Moqsith M, Raoof A. A Missed Diagnosis of Guillain-Barre Syndrome Masquerading as Stroke in an Acute Medical Unit: A Case Report. Case Reports in Clinical Practice. 2021; 6(6):235-238.

Running Title Guillain-Barre Syndrome Masquerading as Stroke

Article info:
Received: 18 Nov 2021
Revised: 28 Nov 2021
Accepted: 22 Dec 2021

Keywords:
Guillain-Barre syndrome; Autoimmune inflammatory demyelinating polyneuropathy; Acute autoimmune neuropathy; Acute infectious polyneuritis; Acute stroke; Cerebrovascular accident

ABSTRACT

An elderly woman aged 66 presented to a general hospital with left-sided facial paresis, bilateral lower limb weakness, and back pain. After undergoing clinical assessments and investigations, she was diagnosed and treated as a case of acute stroke. Two weeks later, she was repatriated to our hospital for neuro-rehabilitation. Unconvinced, we revisited her clinical history and, after a thorough physical examination, made a clinical diagnosis of Guillain-Barre Syndrome (GBS), i.e., confirmed by electrophysiological studies. She was treated with intravenous immunoglobulin and made a complete recovery 6 weeks later.

This case highlights an infrequent presentation of GBS as a stroke-mimic. It re-emphasizes the need to maintain a high index of clinical suspicion for similar expressions, especially in busy acute medical units. Such cases are easily missed if basic clinical skills, such as good history-taking and thorough physical examination, are glossed over. Despite time constraints, these skills are indispensable in clinical practice.

Introduction

Guillain-Barre Syndrome (GBS) is an autoimmune inflammatory disease of the peripheral nervous system. GBS is characterized by an acute ascending weakness, paraesthesia, and diminished or absent deep tendon reflexes, usually after an acute infectious illness [1]. Although relatively rare, it is the commonest cause of acute paralytic neuropathy worldwide, with a global annual incidence of 1-2/100 000 person-year [1, 2].

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GBS diagnosis is mainly clinical and can be made after a detailed history and thorough physical examination. Laboratory and neurophysiological investigations are mainly supportive and should not delay the commencement of treatment [1, 2].

Various presentations of GBS have been described in the literature. However, making the correct diagnosis GBS promptly, remains a challenge for primary and acute care doctors as many cases are still misdiagnosed on the first presentation. In this report, we present a case of GBS that was missed on two separate presentations to the hospital and treated as acute stroke for two weeks.

Case Presentation

A 66-year-old Caucasian female presented to the emergency department with a mild-moderate unspecific lower back pain which had been ongoing for one week. She was prescribed simple analgesics and discharged home. Her symptoms deteriorated over the next few days, and she gradually became more lethargic. Subsequently, she reported progressively worsening bilateral lower limb weakness and paraesthesia in her upper limbs. This was initially overlooked and managed at home with simple remedies such as massage and analgesics purchased over the counter. She became worried when her gait became wobbly, requiring assistance with her mobility. A week later, her speech became slurred, her vision blurred and the left half of her face flaccid, causing lagophthalmos. She collapsed the next day at home and was immediately driven to the regional stroke center by the ambulance service as she was FAST positive.

A quick physical examination revealed signs of lower motor neuron facial palsy at the stroke center and reduced power in both lower limbs. Her blood tests and neuroimaging studies were normal, including Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) scans of the brain and spine. A presumptive diagnosis of acute stroke was made. Besides, she was administered a loading dose of aspirin (300mg). Other differential diagnoses were Bell's palsy due to her facial symptoms and lumbar radiculopathy due to her lower back pain and paraparesis. She has commenced treatment for all three with aspirin, prednisolone, valaciclovir, analgesics, and physiotherapy.

Her symptoms did not improve but rather deteriorated further. She encountered three more falls in the hospital. These were presumed to be a sequela of poor mobility from her stroke. She was repatriated two weeks later to our hospital for ongoing medical rehabilitation. She was admitted into a general medical ward on presentation to our hospital. We found no evidence of a detailed clinical assessment and neurological review from her referral letter and notes. Thus, a neurologist visited her, who took a detailed history and conducted a thorough neurological examination. The only cardiovascular risk factor in her past medical history was hyperlipidemia. She was neither hypertensive nor diabetic. Her mother also had a positive family history of ischaemic heart disease. She neither smoked tobacco nor drank alcohol. There was no recent history of a respiratory tract or gastrointestinal infection. She was previously normally independent and walked unaided. Her examination findings include a bilateral lower motor neuron facial paresis, complete eye movements, areflexia, distal sensory loss, moderate tetra-paresis (power grade 2/5 in all limbs), and a downward plantar reflex.

Clinical suspicion of GBS was made, and she was immediately commenced on intravenous immunoglobulin (IVIG) at 0.4 g/kg/day. Other supportive treatments were also instituted, including blood pressure monitoring, proper hydration, thromboprophylaxis, and physiotherapy. A lumbar puncture for Cerebrospinal Fluid (CSF) analysis was attempted but was unsuccessful. A nerve conduction study revealed absent sensory responses in all limbs, markedly prolonged distal motor latencies, low amplitude dispersed potentials and presented conduction velocities in keeping with a diagnosis of severe demyelinating sensory-motor polyneuropathy, consistent with GBS.

She made a remarkable improvement in her symptoms soon after IVIG was commenced. By the fifth day of her treatment, she had regained full power in both upper limbs and grade 4/5 power in the lower limbs. Seven days after her treatment, her facial symptoms had completely resolved, and with full power in all limbs now, she could walk with the aid of a Zimmer frame. She was discharged to a medical rehabilitation facility for neuro-rehabilitation and could walk unaided 6 weeks later. She is now back to her baseline (premorbid) state.

Discussion

Though well described in the literature, GBS remains a medical enigma, especially for acute care physicians, often resulting in medico-legal issues like the index case. Only a quarter of patients are correctly diagnosed on the first presentation to the emergency department, with some patients reporting as many as 4 physician visits before reaching the correct diagnosis [3]. Myasthenia

gravis and disc herniation syndrome are the most frequent conditions misdiagnosed as GBS [3].

The chief complaint in most patients is weakness, which is often (but not always) symmetrical and ascending [3]. Several subtypes of GBS such as Acute Motor Axonal Neuropathy (AMAN), Acute Motor and Sensory Axonal Neuropathy (AMSAN), Acute Inflammatory Demyelination Polyradiculoneuropathy (AIDP), and Miller-Fisher Syndrome (MFS) are recognized [4]. Some patients may present with paraparesis or cranial nerve palsies (facial, oculomotor, or bulbar) and may be misdiagnosed as a stroke or Bell's palsy, as observed in the index case. It is also essential to note that there may be an overlap between the different clinical syndromes of GBS, e.g., AMSAN and MFS, as may have been the case in our patient. A few patients may present with predominant sensory symptoms, such as paraesthesia and ataxia, while autonomic dysfunction may predominate (pandysautonomia). In some patients, muscle or radicular pain that precedes weakness will present [2]. Our patient sees non-specific back pain as a joint presentation in GBS patients but is often overlooked because much emphasis is placed on ascending limb paralysis.

The history of initial infection is not always evident and should not be used to exclude the diagnosis of GBS. Par- ticular attention should be paid to autonomic function as ascending paralysis can affect the muscles of respi- ration and deglutition, leading to breathing and swal- lowing difficulties, respectively. If not recognized and treated immediately, these cohorts of patients account for a large portion of the mortality burden from GBS.

The diagnosis of GBS is primarily clinical. Investigations are supportive and should not delay treatment [2] [5]. The National Institute of Neurological Disorders and Stroke (NINDS) and the Brighton Collaboration are the two most common diagnostic criteria used to diagnose GBS (Table 1).

An acute care physician should not always expect to encounter the typical and classical presentations of symmetrical ascending weakness and hypo or areflexia. Numerous patients will present with various combinations of symptoms mentioned above. Therefore, a high index of suspicion, in addition to detailed history-taking and thorough neurological examination, must be deployed to make the correct diagnosis of GBS early, as we later did in our case. Prompt diagnosis and early treatment lead to faster recovery and fewer complications. This lesson is essential in emergency departments and acute medical wards with a rapid turnover of acutely unwell patients and will likely be the first presentation for these patients. Amid busy schedules, physicians are advised to examine patients thoroughly and adequately.

The treatment of GBS is with IVIG or plasmapheresis. Both are effective and have similar clinical outcomes [6]. Approximately 20%-30% of patients will have respiratory failure requiring intubation in the intensive care unit. The prognosis after treatment is good, with most patients regaining full strength by 6 months. However, 20% of sufferers may have permanent neurological sequelae, with 3%-10% death [1, 6].

Conclusion

This case highlights a frequent occurrence in acute medical settings where clinical diagnoses are missed due to uncommon or atypical presentations of otherwise well-documented diseases, especially for junior

Table 1. Brighton collaboration criterion for the diagnosis of Guillain-Barre syndrome [5]

Criteria	Diagnostic Certainty		
	Level 1	Level 2	Level 3
Bilateral and flaccid weakness of the limbs	+	+	+
Decreased or absent deep tendon reflexes in weak limbs	+	+	+
Monophasic illness pattern; and the interval between onset and nadir of weakness between 12h and 28 days; and subsequent clinical plateau	+	+	+
Absence of identified alternative diagnosis for weakness	+	+	+
Cytoalbuminologic dissociation (i.e., the elevation of CSF protein level above normal laboratory value and CSF total white cell count	+	+	
Electrophysiological findings consistent with GBS	+		

doctors without much experience yet, and poor clinical practice due to rushed physical examinations. There are two crucial learning points from this case. Firstly, the diagnosis of GBS may not always be evident on the first presentation. Therefore, acute care physicians should always have a high index of suspicion, especially in patients presenting with back pain and neurological symptoms. Secondly, the role of the primary clinical practices of good history-taking and detailed physical examination in clinical practice cannot be over-emphasized. Despite the dynamic nature of acute medical wards and the increased availability of more sophisticated laboratory and imaging studies. Unfortunately, these essential practices are gradually being eroded in modern clinical practice.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles are considered in this article.

Funding

This research did not receive any grant from funding agencies in the public, commercial, or non-profit sectors.

Authors' contributions

All authors equally contributed in preparing this article.

Conflict of interest

The authors declared no conflict of interest.

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