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Unique Case of Guillain-Barré Syndrome Associated with Pneumococcus in a Young Female Patient Attending Sirt Teaching Hospital, Libya

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A B S T R A C T

Guillain-Barré Syndrome (GBS) is an infrequent neurological disorder where the immune system mistakenly attacks peripheral nerves, leading to progressive weakness, sensory disturbances, and, in severe cases, paralysis. The precise cause of GBS remains unclear, though it is often preceded by an infection. Common infectious triggers include gastrointestinal pathogens such as Campyl-obacter and respiratory infections like Mycoplasma pneumoniae and influenza. In this case report, we describe a 25-year-old female who presented with acute dyspnea, bilateral limb weakness, and sensory abnormalities beginning as a "pins-and-needles" sensation in her hands. Her condition rapidly progressed, culminating in septic shock due to Pneumococcus, complicated by Acute Respir-atory Distress Syndrome (ARDS). Neurological evaluation confirmed the diag-nosis of Guillain-Barré Syndrome. This case highlights a rare but significant as-sociation between pneumococcal infection and GBS, emphasizing the complexity of its clinical presentation.

1 Introduction

Guillain-Barré Syndrome (GBS), also known as acute idiopathic polyradiculoneuritis, is an autoimmune disorder that primarily affects the peripheral nervous system (Wakerley & Yuki, 2013). The condition is triggered by various infectious and non-infectious factors, making it a complex and multifactorial syndrome (Yuki &Hartung, 2012).

The clinical presentation of GBS typically includes progressive muscle weakness, sensory disturbances, and, in severe cases, paralysis. Although the exact etiology remains unclear, numerous studies have established a strong association between GBS and prior infections, with common triggers including Campylobacter jejuni, Epstein-Barr virus, and Cytomegalovirus (Mazidi et al., 2013). Additionally, bacterial infections such as Streptococcus pneumoniae, which causes a wide range of diseases from pneumonia to meningitis, have been implicated in the development of GBS (Willison et al., 2016)

While the clinical diagnosis of Guillain-Barré Syndrome remains largely based on clinical criteria, the challenge lies in differentiating it from other similar conditions due to overlapping symptoms. Diagnosis is usually made through a thorough clinical history, neurological examination, and supportive diagnostic tests. Despite significant advances in understanding the

condition, the prognosis for GBS remains variable. Around 20% of patients experience long-term disability, and nearly 5% may succumb to the disease (Sohara et al., 2012). Furthermore, about 30% of patients experience severe complications such as respiratory failure, which necessitates intensive care and mechanical ventilation (Orlikowski et al., 2004). Despite these statistics, many still maintain a misconception that GBS has a favorable prognosis, which can delay critical interventions.

2. Materials and Methods

Patient Information: A 25-year-old previously healthy female presented to the emergency department with respiratory distress following a flu-like illness. The patient reported progressive limb weakness and shortness of breath over the past 3 days. There was no recent history of trauma, surgery, or vaccination.

Clinical Assessment: On admission, the patient was alert and oriented, though she appeared pale. No signs of anemia or jaundice were noted. Chest examination revealed decreased air entry bilaterally with coarse crepitations throughout both lung fields. Cardiovascular evaluation showed normal heart sounds (S1, S2), and the abdomen was mildly distended, soft, with normal bowel sounds. Neurologically, the patient had preserved higher mental functions, with intact speech, memory, and ocular movements. Sensory examination revealed no alterations in pain perception. However, motor examination demonstrated hypotonia and bilateral limb weakness—more severe in the lower limbs. Upper limbs showed distal weakness with a strength score of 3/5. Lower limbs were significantly weaker, with absent deep tendon reflexes and a negative Babinski reflex.

3. Results

Initial Clinical Findings

The patient presented alert and oriented, although notably pale. Chest auscultation revealed decreased air entry bilaterally with coarse crepitations. Cardiovascular assessment showed normal heart sounds (S1, S2). The abdomen was mildly distended but soft with normal bowel sounds.

Neurologically, the patient was conscious with intact speech, memory, and ocular movements. Sensory testing was unremarkable, but motor examination revealed hypotonia and weakness. Upper limb strength was reduced (3/5), more pronounced distally. Lower limb weakness was severe, with absent deep tendon reflexes and a negative Babinski reflex.

Laboratory and Radiological Investigations

Initial laboratory results (Table 1) demonstrated:

Hematology: Hemoglobin: 9 g/dL; Platelets: 83 $\times 10^3/\mu L$; WBC: 6.6 $\times 10^3/\mu L$ with lymphocytic predominance (91.7%) and neutropenia (29%).

Electrolytes: Hypocalcemia, hypomagnesemia, hypokalemia.

Renal function: Urea: 55 mg/dL; Creatinine: 1.1 mg/dL; Albumin: 2.8 g/dL.

Liver function: Elevated AST, ALT, and bilirubin.

Radiological findings: A chest radiograph revealed bilateral diffuse infiltrates with patchy opacities and consolidation, consistent with acute respiratory distress syndrome (ARDS). Chest CT confirmed bilateral lobar pneumonia, suggestive of severe pneumococcal infection. Microbiological cultures and urinary antigen testing were unfortunately not performed due to the patient's rapidly declining condition.

Table 1: Key Laboratory findings

Table 1. Key Laboratory Findings

Test	Result	
Leucocytes ($\times 10^3/\mu L$)	6.6	
Neutrophils (%)	29	
Lymphocytes (%)	91.7	
Hemoglobin (g/dL)	9	
Platelets ($\times 10^3/\mu L$)	83	
Electrolytes	Hypocalcemia, Hypomagnesemia, Hypokalemia	
Renal Function	Urea (mg/dL)	55
	Creatinine (mg/dL)	1.1
	Albumin (mg)	2.8
Liver Function	AST, ALT, Bilirubin	Elevated

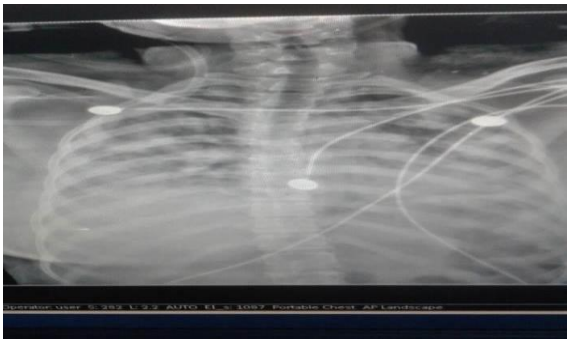

Figure 1. Chest X-ray showing diffuse bilateral pulmonary infiltrates

Subsequent Management and Outcome:

The patient was promptly transferred to the intensive care unit (ICU) for in-tensive monitoring and supportive care. Upon arrival, she remained alert and oriented but exhibited signs of severe distress, including pallor, dyspnea, tach-ypnea, and fever. Her vital signs were as follows: temperature 38.4°C, pulse rate 140 bpm in sinus rhythm, blood pressure recorded

at 70 mmHg systolic with undetectable diastolic pressure, and oxygen saturation levels ranging between 75% and 80% on room air.

Despite initial management, her condition worsened progressively, with clear signs of septic shock. To address the hypotension, intravenous fluids were administered, but blood pressure remained critically low at 70/40 mmHg. In re-sponse, a noradrenaline infusion was started, along with Meronam (1g IV every 8 hours) and Flagyl (500mg IV every 8 hours). Laboratory findings indicated thrombocytopenia, and due to the patient's unstable cardiorespiratory status, lumbar puncture was deemed contraindicated.

Unfortunately, the patient's condition continued to deteriorate. Despite ef-forts to stabilize her, respiratory support was required, and she was intubated six hours after admission. Tragically, six hours later, the patient succumbed to her condition.

4. Discussion

Guillain-Barré Syndrome (GBS) remains a significant cause of acute flaccid pa-ralysis. Although the exact etiology is not fully understood, numerous infectious and non-infectious triggers have been implicated in its pathogenesis. Among the infectious agents most commonly associated with GBS are Campylobacter jejuni, cytomegalovirus (White et al., 2011), Epstein-Barr virus, Mycoplasma pneumoniae, and vari-ous influenza-like viruses and rarely streptococcus pneumoniae. Non-infectious factors, including vaccinations, trauma, surgical interventions, and bone marrow transplantation, have also been documented. These triggers may initiate an aber-rant immune response against peripheral nerve components through mecha-nisms such as molecular mimicry (Bianchi & Domenighetti, 2006).

In the case of our patient, the clinical presentation was highly suggestive of GBS, characterized by ascending paralysis starting in the lower limbs and progressing to involve the upper limbs and respiratory muscles. The development of respira-tory distress and areflexia further supported this diagnosis. Typically, GBS diagnosis is confirmed through cerebrospinal fluid (CSF) analysis—demonstrating albuminocytologic dissociation—and electrodiagnostic studies (Mandell et al., 2007). However, in our patient, both procedures were contraindicated due to severe coagulopathy and cardiorespiratory instability.

Although microbiological confirmation was not obtained, the patient's clinical presentation—including acute febrile illness, respiratory distress, and hypotension—along with characteristic chest radiograph findings of bilateral consolida-tion and rapid deterioration, strongly suggest Streptococcus pneumoniae as the probable causative pathogen. This is consistent with the typical clinical and ra-diographic features of pneumococcal pneumonia, which remains the most com-mon cause of community-acquired pneumonia (CAP) worldwide, particularly in cases progressing to sepsis and acute respiratory distress syndrome (ARDS) ((Musher & Thorner, 2014, (Jain et al., 2015).

Interestingly, pneumococcal infection has been rarely reported as a potential precursor to GBS. A notable case involved a 68-year-old woman who developed severe S. pneumoniae bacteremia and Austrian's triad—pneumonia, meningitis, and endocarditis—subsequently presenting with an unusual GBS variant not previously associated with pneumococcal disease (7). Similarly, a 78-year-old male with pneumonia and pleural effusion later developed acute flaccid tetraparesis. Culture from the effusion confirmed S. pneumoniae, and electrophysiologi-cal studies revealed a mixed motor axonal and demyelinating GBS variant, along with positive anti-GM1 and anti-GD1 antibodies—autoantibodies known to be associated with motor forms of GBS (11).

These rare but informative reports highlight the potential of S. pneumoniae to serve as a trigger for GBS and underscore the importance of clinical vigilance in atypical presentations, especially when confirmatory testing is not feasible.

5. Conclusions

The diagnosis of GBS is primarily clinical, with early recognition and prompt intervention being key factors for a better prognosis. However, cases involving rapid progression or autonomic dysfunction often have a poor outcome, under-scoring the need for timely and aggressive management.

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