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Respiratory Diseases

Guillain-Barré Syndrome Complicating a Pulmonary Abscess: A Case Report

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Abstract | **Case Report**

Guillain-Barré Syndrome (GBS) is a rare inflammatory polyradiculoneuropathy often triggered by infections. We report a case of GBS in a patient with a pulmonary abscess, highlighting the link between respiratory infection and the development of this neurological syndrome. We will also discuss the pathophysiology of GBS and the clinical implications for managing patients presenting with neurological symptoms after a respiratory infection.

Keywords: Guillain-Barré Syndrome, Pulmonary Abscess, Respiratory Infection, Polyradiculoneuropathy, Autoimmunity.

I. INTRODUCTION

Guillain-Barré Syndrome (GBS) is a rare neurological disorder in which the immune system attacks the peripheral nervous system, often following an infection. The immune response can lead to inflammation of the peripheral nerves, causing weakness, paralysis, and in some cases, respiratory failure. In many instances, bacterial or viral respiratory infections are associated with the development of GBS. We report a case of GBS occurring after a pulmonary abscess in a 54-year-old patient, a rare occurrence that underscores the importance of monitoring for neurological complications following a lung infection.

II. CASE REPORT

A 54-year-old male, chronic smoker with a 40 pack-year history and no significant medical history, presented in September 2024 with dyspnea and right-sided pleuritic chest pain evolving over one week in a febrile context.

On admission, the patient had a temperature of 38.2°C, blood pressure of 130/80 mm Hg, and clinical examination revealed signs of right basal pulmonary consolidation. Neurological exam showed lower limb muscle weakness with inability to stand and absence of deep tendon reflexes.

Chest CT revealed a right lung abscess with associated ipsilateral hydropneumothorax. Laboratory tests showed an inflammatory syndrome (leukocytosis at 12,670/mm³ with neutrophil predominance, elevated CRP at 295 mg/L, and an ESR of 120 mm in the first hour). Sputum cytobacteriological exam revealed gram-positive cocci, though culture was sterile. Pleural puncture yielded foul-smelling purulent fluid with negative bacteriology. Renal, blood glucose, and viral serologies were all negative. Additional investigations to search for a portal of entry—including urinalysis, sinus CT, and abdominal ultrasound—were also negative. Flexible bronchoscopy was unremarkable. Brain CT and spinal MRI (cauda equina and plexus) performed due to motor weakness were normal. Electroneuromyography showed a non-length-dependent sensorimotor polyneuropathy with demyelinating features compatible with Guillain-Barré syndrome. Lumbar puncture revealed albuminocytologic dissociation with negative bacteriological analysis.

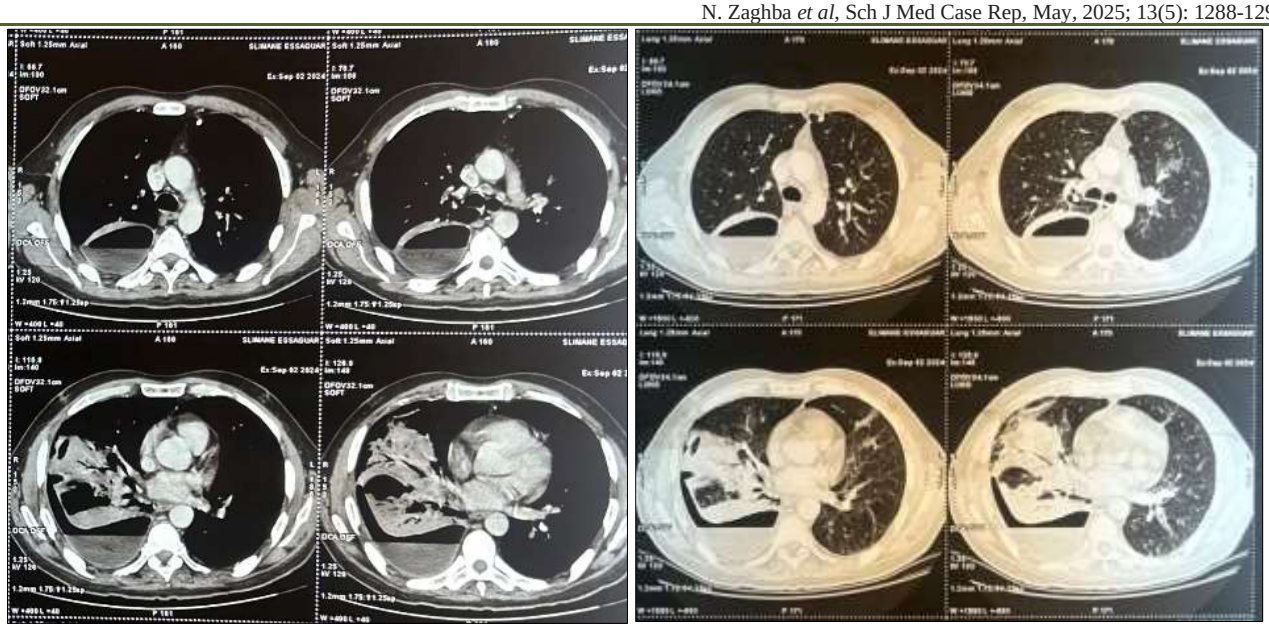

Figure 1: Thoracic CT showing a right basal pulmonary abscess ruptured into the pleural space and complicated by pyopneumothorax.

The patient received triple antibiotic therapy with amoxicillin-clavulanic acid 1g three times daily, ciprofloxacin 500 mg daily, and metronidazole 500 mg three times daily, along with bronchial drainage physiotherapy, resulting in good clinical and biological outcomes. The pneumothorax improved following thoracic drainage. Motor weakness improved without the need for immunoglobulin therapy due to the mild form of GBS and favorable progression.

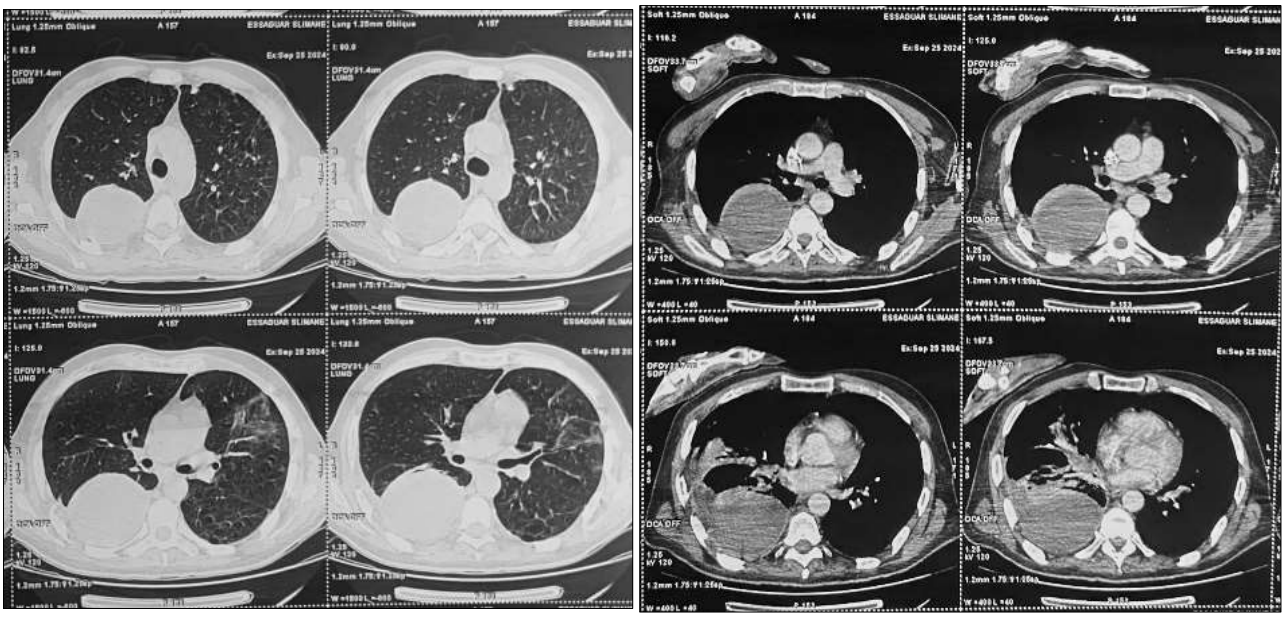

Figure 2: Follow-up thoracic CT showing resolution of the pyopneumothorax.

III. DISCUSSION

Guillain-Barré Syndrome (GBS) is an inflammatory polyradiculoneuropathy characterized by rapid limb weakness, often with sensory and cranial nerve involvement. Recent studies have shown that GBS is frequently preceded by infection, particularly respiratory infections.

A retrospective study at Saint-Luc University Clinics found that 53.7% of GBS patients had suffered an infection within 30 days prior to symptom onset, with respiratory infections being the most common, followed by gastrointestinal infections [1]. Another study by McGrogan and al. confirmed that 60–70% of GBS cases follow prior infections, with respiratory infections accounting for a significant portion [2].

The role of respiratory infections in the pathogenesis of GBS is well documented, often involving molecular mimicry. The infection triggers an immune response against microbial carbohydrate antigens, leading to the formation of anti-ganglioside antibodies that may cross-react with host nerve structures, causing conduction failure or demyelination [3].

Mycoplasma pneumoniae is a cause of atypical pneumonia and has been identified as a potential GBS trigger due to this molecular mimicry mechanism [4]. Other pathogens such as Campylobacter Jejuni and respiratory viruses have also been shown to be major GBS triggers [5].

The COVID-19 pandemic further emphasized the link between viral respiratory infections and GBS. Several GBS cases were reported following SARS-CoV-2 infection [6, 7]. The pathophysiology involves exaggerated immune responses, elevated pro-inflammatory cytokines, and the presence of anti-neuronal ganglioside autoantibodies [8].

GBS clinical presentations vary from mild limb weakness to respiratory muscle involvement requiring mechanical ventilation. The severity often correlates with the infectious trigger; for instance, C. jejuni is linked to more severe axonal forms [5]. It is essential to differentiate neuromuscular weakness due to GBS from generalized tiredness from infection. Key diagnostic elements include albuminocytologic dissociation on lumbar puncture and conduction block on electroneuromyography [9]. Anti-ganglioside antibody testing can also support diagnosis, especially in post-infectious cases [10].

Treatment relies on intravenous immunoglobulin (IVIG) or plasmapheresis, both of which have proven effective in improving neurological deficits [10]. Severe cases with respiratory involvement require intensive care due to the risk of acute respiratory failure [11].

IV. CONCLUSION

GBS is a rare but serious complication of respiratory infections. Early recognition is essential for prompt treatment and improved prognosis. Complex immunopathological mechanisms, including molecular mimicry, explain the association between respiratory infections and GBS. This case highlights the need for careful neurological examination in patients with severe respiratory infections.

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