

## Case Report

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# Rare Presentation of Intracranial Hemorrhage post Guillain Barré Syndrome: A Case Report

Sarah El Halabi<sup>1</sup>, Jaafar AL-Shami<sup>2</sup>, Ghadir Hijazi<sup>3</sup>, Zakaria Alameddine<sup>4</sup>, Maher Ghandour<sup>5</sup>, Ali Saad<sup>6</sup>

<sup>1</sup> Department of Internal Medicine, Sheikh Ragheb Harb University Hospital, Toul, Lebanon

<sup>2</sup> Department of General Surgery, Sheikh Ragheb Harb University Hospital, Toul, Lebanon

<sup>3</sup> Department of Internal Medicine, Al Zahraa University Medical Center, Beirut, Lebanon

<sup>4</sup> Department of Internal Medicine, Lebanese American University Medical Center, Beirut, Lebanon

<sup>5</sup> Department of General Surgery, Lebanese University, Beirut, Lebanon

<sup>6</sup> Pulmonary and Critical Care Department, Sheikh Ragheb Harb University Hospital, Toul, Lebanon

Corresponding Author: Jaafar Al-Shami, Beirut, Lebanon, [dr.jaafaralshami@gmail.com](mailto:dr.jaafaralshami@gmail.com)

Department of General Surgery, Sheikh Ragheb Harb University Hospital, Toul, Lebanon

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### Abstract

**Background:** Guillain Barré Syndrome (GBS) is an autoimmune disease where antibodies attack the myelin sheath of peripheral nerves. The hallmark of the disease includes symmetrical quadriparesis, respiratory distress, and failure with subsequent need for mechanical ventilation. Most cases occur after a viral or bacterial infection. Other causes, such as intracranial hemorrhage, also exist, and several case studies report an association between these two pathologies.

**Case Report:** In this report, we present the case of an elderly male patient with intracranial (IC) bleeding post-GBS. The patient was admitted to the hospital for dyspnea and diagnosed with pneumonia. When he started complaining of progressive bilateral ascending paralysis of his lower extremities, we performed a lumbar puncture, and he was diagnosed with GBS. We started him on intravenous immune globulins (IVIGs) immediately, but his weakness progressed to include his respiratory muscles, and he required mechanical ventilatory support with Intensive Care Unit (ICU) admission. We extubated him after two weeks, but he needed to be reintubated 24 hours later for a severely decreased level of consciousness. An urgent computed tomography scan of the brain showed IC bleeding. The patient developed a septic shock due to his pneumonia, which was refractory to antibiotics and vasopressors. He passed away a few weeks after that.

**Conclusion:** Our case represents a unique type of association between IC hemorrhages and GBS, where the bleeding occurred several days after, as opposed to before GBS. It also reinforces the correlation between GBS and intracranial bleeding and stresses the importance of having a high index of suspicion when facing either pathology since both have similar symptoms that may overlap or mask each other.

**Keywords:** Guillain Barré Syndrome, Intracranial Hemorrhage, IVIG, cerebral vasospasm, case report.

## Background

Guillain Barré Syndrome (GBS) is a polyneuropathy associated with autoimmune-mediated demyelination of peripheral nerves. It is a rare disease with an incidence of 0.4 to 2 per 100,000, which affects 100,000 patients worldwide per year. Men are 1.5 times more susceptible than women. Even with treatment, the associated mortality remains as high as 3.5 to 12 percent in the acute phase, with a 20 percent rate of residual disability [1-4].

Characteristically, the symptoms, both motor and sensory, occur bilaterally and in an ascending fashion. The disease initially presents with lower limb paresthesias and evolves into ascending flaccid weakness and symmetrical quadriplegia. Respiratory muscle weakness and failure occur in around 30 percent of patients and require the use of mechanical ventilation. Dysautonomia, including cardiac arrhythmias or blood pressure lability, cause the majority of morbidity and mortality. Diagnosis is usually clinical [1-4]. Ancillary tests include nerve conduction studies, electromyography, and cerebrospinal fluid (CSF) analysis. Treatment options include intravenous immunoglobulins (IVIGs), which act by an immune-modulating mechanism, and plasma exchange, which removes the pathogenic antibodies [1].

Approximately two-thirds of GBS cases occur weeks after infection, which may be bacterial or viral [5]. Other etiologies include surgery, trauma, certain vaccines, and rarely head trauma and intracranial hemorrhage (ICH) [6]. Several case studies show an association between ICH and GBS occurring in either sequence but with different pathogeneses [7].

We present a case of ICH following GBS. The patient needed to be admitted to the ICU and mechanically ventilated due to respiratory failure. In recent years many case studies detailing this complication have been published. We present this case to shed light on this subject, which deserves greater attention in clinical practice to reduce patients' morbidity and mortality.

## Case Report

We present the case of a 69-year-old male patient who presented at our hospital complaining of dyspnea at rest, subjective fever, and productive cough of yellowish sputum. He denied the presence of chest pain, abdominal pain, bowel, or urinary habit

changes. The patient had a history of diabetes mellitus on Galvus met (vildagliptin/ metformin hydrochloride) 50mg/850mg per os twice daily, but no previous surgical or family history. Vitals on presentation were as follows: blood pressure 130/70 mmHg, heart rate 93 bpm, respiratory rate 20 breaths per minute, temperature 39.1 Celsius, oxygen saturation 97%. Physical examination revealed decreased air entry on the right side associated with end-inspiratory crackles. Cardiovascular, abdominal, and neurological exams were within normal.

Routine laboratory tests on admission were also within normal except for an increase in neutrophil percent to 80% (Reference Range [RR] 40 to 70%) and a CRP level of 221 mg/L (RR 0 to 10 mg/dL). Urine analysis was negative for nitrite but positive for leukocyte esterase with numerous red and white blood cells; urine culture was negative. Chest X-ray showed right basal infiltrates. A diagnosis of pneumonia was made and confirmed with computed tomography (CT) scan of the chest without intravenous (IV) contrast, which reported consolidation of the right lower lobe with early pneumonia in the left lower lobe. The patient was started on Levofloxacin 500 mg intravenously once per day.

The following day, the patient started to complain of bilateral lower limb numbness that evolved into a weakness of ascending nature. On examination, he was alert and oriented. His lower limbs had absent deep tendon reflexes and reduced motor power bilaterally (Grade 3). His upper limbs were normal, and there was no neck stiffness. The sensation was intact.

CT scan of the brain was performed and showed chronic arteriosclerotic disease with no intracranial hemorrhage (ICH). Magnetic Resonance Imaging (MRI) without gadolinium of the whole spine showed degenerative disc disease, as well as bladder distention with no significant neural impingement or spinal canal stenosis.

Lumbar puncture (LP) revealed the following: protein fluid 51 mg/dl (RR 15 to 45 mg/dL), red blood cell fluid 1 (RR <0), glucose fluid 138 mg/dL (RR 40 to 70 mg/dL), white blood cell body fluid 1 (RR <5), polymorphonuclear count 1, lactate dehydrogenase fluid 21 (RR ≤ 40 units/L for adults).

Blood tests revealed the following: white blood cell count  $13.6 \times 10^3/\mu\text{L}$  (RR 4.5 to  $10 \times 10^3/\mu\text{L}$ ), neutrophil percent 85% (RR 40 to 70%), CRP

level 244 mg/L (RR 0 to 10 mg/dL) which showed mild elevation compared to the lab results at admission. Vitamin B12 level 74 pg/mL (RR 180 to 914 pg/mL).

Based on lumbar puncture results and the ascending paralysis of the patient, a diagnosis of Guillain Barré Syndrome was made. We started the patient on Intravenous Immune Globulin (IVIg) 0.4 grams per kilogram per day for four days. He also received an intravenous formulation of vitamin B12 for replenishment.

On his first day of IVIg, the patient's respiratory status worsened. He had tachypnea reaching 32 breaths per minute and desaturation with a SpO<sub>2</sub> of 83%. He became obtunded with a decreased level of consciousness, so we transferred him to the intensive care unit (ICU) for close observation. Levofloxacin was switched to piperacillin/tazobactam 4.5 g intravenously every 8 hours.

After finishing the course of IVIg, the patient had a septic shock and respiratory failure and needed vasopressor support with noradrenaline and intubation. We also broadened the spectrum of antibiotics choosing Meropenem 1 g intravenously every 8 hours instead of Piperacillin/tazobactam.

We extubated the patient after attaining both respiratory and hemodynamic stability. However, he deteriorated again in the following 24 hours and needed a reintubation.

We repeated the brain CT scan, and it showed diffuse intraparenchymal bleeding in the cortico-medullary junction, mainly in the frontal, parietal, and temporal lobes (Fig. 1).

A CT of the chest with IV contrast was performed after few days due to increased oxygen requirement and revealed a progression of his pneumonia, manifested by bilateral lower lobe ground-glass pattern as well as upper lobe diffuse opacities. These findings suggest a radiographic appearance of acute respiratory distress syndrome (ARDS).

After many failed trials of extubation, the patient underwent a tracheostomy. Thus, we stopped sedation, and the patient was alert, conscious, and oriented. However, there was a reduction in upper limbs and neck motor power. Deep tendon reflexes were absent on lower limbs and reduced on upper limbs. The sensation was intact. Unfortunately, a few days later, the patient died from septic shock due to his pneumonia.

## Discussion

We presented the case of an elderly patient with ICH post-GBS. In the literature, there are only cases of GBS occurring after or concomitantly with ischemic stroke, IC bleeding, hemorrhagic transformation of ischemic stroke, and traumatic brain injury. However, several hypotheses explain ICH after GBS, as occurred in our patient.

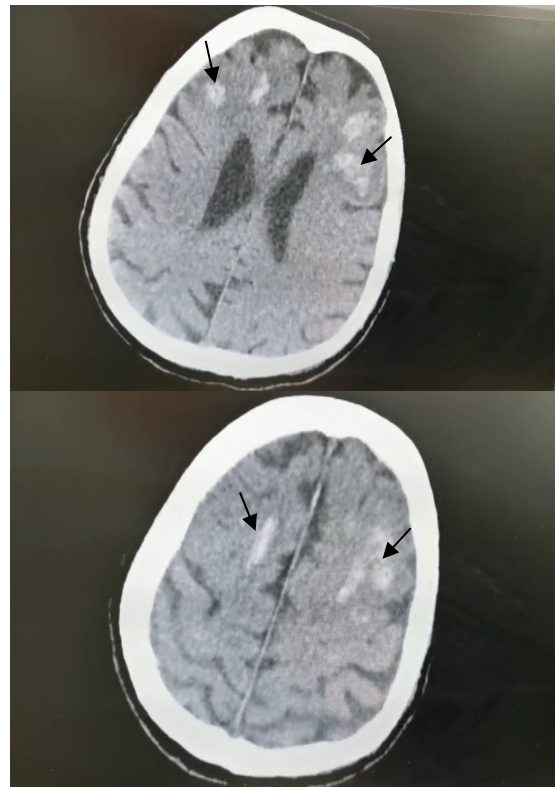


Figure 1: CT scan of the brain done after the patient was extubated and needed a reintubation, showing intracranial bleeding in the cortico-medullary junction, mainly in the frontal, parietal and temporal lobe (black arrows)

As to why GBS happens in patients with IC bleeding/trauma, one of the most commonly postulated mechanisms is an immune activation after intracranial insult. This occurs due to neuronal injury caused by the bleed/trauma, leading to blood-brain-barrier disruption. T-lymphocytes and macrophages accumulate and induce the transformation of microglial cells into antigen-presenting cells. As such, myelin-associated proteins released from neuronal debris are presented to the immune system, and anti-myelin antibodies are formed, precipitating GBS [6,8].

On the other hand, the mechanism for intracranial bleeding after GBS includes the

administration of IVIGs. Gamma globulins have several adverse effects, including hyperviscosity, thrombosis, vasculitis (microangiopathies), and cerebral vasospasm leading to subarachnoid hemorrhage [7]. Increased intracranial pressure (ICP), which has been reported by one case report to occur in GBS, can also cause IC hemorrhage. This report stated that increased cerebrospinal fluid (CSF) protein leads to increased intracranial pressure (ICP), although not all those cases had elevated CSF proteins [5].

Regarding the case of our patient, we did not measure the CSF pressure, and there was no marked increased CSF protein (51 mg/dl with a RR: 15-45mg/dl). So, by exclusion, the most probable explanation is that the bleeding occurred after IVIG treatment, due to either cerebral vasospasm or a possible vasculitis.

This case report shows the increasingly relevant association between GBS and IC bleeding. It is also unique because no previous case reports mention ICH after GBS. While we could not prove that bleeding occurred due to other causes, the symptoms of ICH and GBS may mask each other or even overlap, which emphasizes the importance of a high index of clinical suspicion to diagnose IC bleeding in this neurological disease. As such, an exacerbation of the neurological symptoms in a patient with confirmed GBS might not only be caused by the syndrome itself but also, by a concomitant intracranial pathology, such as intracranial bleeding, as in the case of our patient. Further investigation by means of imaging is warranted in case of neurological symptom exacerbation.

## Conclusion

What was remarkable in our case is that we could not find any case reports of ICH occurring several days after GBS as opposed to GBS following IC bleeding. This might represent a new type of association between these two pathologies. However, more studies and case reports are needed to corroborate a significant and clinically relevant association between these two pathologies, in this order.

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