

Guillain-Barré Syndrome Following SARS-CoV-2 Pneumonia in a Patient Receiving Adjuvant Chemotherapy for Gastric Cancer: A Case Report

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Abstract

Guillain-Barré Syndrome (GBS) is an acute, progressive inflammatory polyneuropathy characterized by muscle weakness and distal sensory deficits. In the context of SARS-CoV-2 infection, GBS most commonly presents as a sensorimotor variant, frequently associated with facial paralysis and a demyelinating electrophysiological pattern.

We report the case of a 47-year-old female diagnosed with stage IIb (T3, N1, M0) gastric adenocarcinoma who underwent gastrectomy followed by five cycles of adjuvant chemotherapy. During the fifth cycle, she developed SARS-CoV-2-associated pneumonia involving 30% of lung parenchyma and required hospitalization. Following recovery and discharge, the patient experienced progressive lower limb weakness and sensory disturbances. Electro Neuro Myo Graphy (ENMG) demonstrated mixed sensory and motor nerve involvement in both upper and lower extremities predominantly on the left consistent with a combined axonal-demyelinating neuropathy.

This case illustrates a rare occurrence of GBS in an immunocompromised individual recovering from COVID-19. It is hypothesized that SARS-CoV-2 contributed to autoimmune activation via systemic inflammation and cytokine release, ultimately triggering GBS. Clinicians should maintain a high index of suspicion for post-infectious neuropathies such as GBS, particularly in oncology patients recovering from SARS-CoV-2 infection.

Keywords: Guillain-Barré syndrome • Covid-19 • Stomach cancer • Gastrectomy • Chemotherapy

Introduction

Guillain-Barré Syndrome (GBS) is an acute, rapidly progressive inflammatory neuropathy characterized by muscle weakness and distal sensory loss. Its etiology is frequently linked to autoimmune responses triggered by preceding infections. Common infectious agents associated with GBS include *Campylobacter jejuni*, enteric viruses, herpesviruses such as cytomegalovirus and Epstein-Barr virus and *Mycoplasma* species. In recent years, emerging pathogens like the Zika virus and more recently, SARS-CoV-2, the causative agent of COVID-19 have also been implicated in the onset of GB [1].

The COVID-19 pandemic, which began in December 2019, is caused by SARS-CoV-2, a virus primarily known for inducing acute respiratory syndrome. However, accumulating evidence suggests that SARS-CoV-2 may also affect the nervous system, with reported neurological manifestations including

headache, dizziness, anosmia and, in some cases, involvement of the peripheral nervous system [2].

SARS-CoV-2-associated GBS typically manifests as a sensorimotor variant, often accompanied by facial paralysis and is most frequently classified as a demyelinating electrophysiological subtype. The onset generally occurs within three weeks of SARS-CoV-2 infection, surgical procedures, or vaccination, supporting the hypothesis of a post-infectious autoimmune mechanism.

Case Report

On August 28, 2020, a 47-year-old female patient of Korean ethnicity was admitted to the Department of Neurology with complaints of muscle weakness affecting both the upper and lower extremities, paresthesia in the toes and paresis of the left foot.

Medical history

In March 2020, the patient was hospitalized due to gastric bleeding. Endoscopic examination revealed a suspected neoplasm located in the upper third of the stomach. A biopsy was performed and subsequent histopathological analysis confirmed the diagnosis of grade 3 gastric adenocarcinoma. Chest Computed Tomography (CT) demonstrated no pathological findings. Contrast-enhanced CT of the abdomen and pelvis revealed a right renal cyst and an accessory arterial trunk supplying the left kidney.

On April 8, 2020, the case was reviewed at a multidisciplinary team meeting, where a decision was made to proceed with surgical intervention. On April 9, 2020, the patient underwent a laparotomy with spleen-preserving gastrectomy, D2 lymph node dissection, catheter jejunostomy and abdominal cavity drainage.

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Histopathological findings

Histological specimen No. 15820-42, dated April 16, 2020, confirmed the diagnosis of signet-ring cell adenocarcinoma located in the posterior wall of the gastric body and extending to the lesser curvature. The tumor was classified as Grade 3 (G3) and demonstrated ulceration with infiltration through all layers of the gastric wall. Tumor cells were identified within the lumina of individual lymphatic vessels. No malignant elements were detected in the omental tissue and both the proximal and distal resection margins were free of tumor involvement.

On April 20, 2020, a multidisciplinary team meeting confirmed the final diagnosis: gastric cancer, stage IIb (T3, N1, M0), status post-gastric bleeding and post-hemorrhagic anemia. Given the patient's overall condition, with an Eastern Cooperative Oncology Group (ECOG) performance status score of at least 2, adjuvant (postoperative) chemotherapy was recommended. It was further advised that contrast-enhanced CT scans of the chest and abdomen be performed following the completion of three chemotherapy cycles.

From May 6 to May 8, 2020, the patient received the first course of adjuvant chemotherapy using the FLOT regimen, which included docetaxel 80 mg administered via intravenous drip on day 1, oxaliplatin 120 mg as a 2-hour intravenous infusion on day 1, leucovorin 300 mg via intravenous drip on day 1 and a continuous infusion of 5-fluorouracil at a total dose of 4000 mg over 46 hours, along with supportive therapy. The treatment was tolerated, although the patient experienced delayed grade 2 leukopenia, febrile neutropenia and a transient fever reaching 38°C for two days, accompanied by emetic syndrome.

The second and third courses of adjuvant chemotherapy were administered from May 23 to May 25, 2020 and from June 6 to June 8, 2020, respectively. A follow-up endoscopic examination on June 19, 2020, confirmed a post-gastrectomy state due to gastric cancer and revealed erosive reflux esophagitis, with no evidence of disease recurrence.

The fourth course of adjuvant chemotherapy was administered from June 20 to June 22, 2020, followed by the fifth course, which was conducted from July 3 to July 8, 2020.

Adjuvant chemotherapy was administered on an outpatient basis, during which time the patient reported contact with individuals exhibiting respiratory symptoms. Following the fifth chemotherapy cycle, on July 19, 2020, she developed symptoms of weakness, fever and cough. Evaluation at a local clinic revealed a positive Polymerase Chain Reaction (PCR) test for SARS-CoV-2. A subsequent Computed Tomography (CT) scan of the chest demonstrated bilateral polysegmental pneumonia (CT-2) involving approximately 30% of the lung parenchyma.

From July 19 to July 30, 2020, the patient was hospitalized in an infectious disease ward with the diagnosis of U07.1 COVID-19, virus identified and J12 viral pneumonia. During hospitalization, she received comprehensive

therapy. As a result, her condition showed positive clinical and radiological improvement, with pneumonia entering the resolution stage.

Following discharge from the infectious disease ward, the patient developed progressive weakness in the lower extremities, which worsened over the subsequent month. On August 28, 2020, she was admitted to the neurology department for further evaluation and management.

Objective findings upon hospital admission

The patient's general condition was assessed as moderately severe, primarily due to pain syndrome. She was conscious, alert and oriented. Mucous membranes appeared normochromic and peripheral lymph nodes were not enlarged. The chest had a normal shape, with clear percussion sounds and vesicular breathing on auscultation. Cardiac examination revealed muffled heart sounds with a regular rhythm; heart rate was 100 beats per minute and blood pressure was 120/80 mmHg. The abdomen was soft and non-tender, with the liver palpable at the costal margin. Percussion tenderness was absent bilaterally and no peripheral edema was noted.

Neurological examination (Status localis)

The patient was oriented to time and space, fully conscious, communicative and able to follow commands. She reported cephalgia and appeared emotionally tense and asthenic.

Cranial Nerves (CN)

Palpebral fissures were symmetrical (OD=OS) and light reflexes were present. Convergence was weak bilaterally. Incomplete abduction of the eyes was noted at the extremes of gaze. Fine horizontal gaze-evoked nystagmus was observed in lateral positions. Diplopia was absent.

Motor and sensory function

Muscle tone and strength were within age-appropriate limits. Tendon reflexes in both upper and lower extremities were reduced, with decreased Achilles reflexes. Sensory examination revealed hypesthesia in a "glove and stocking" distribution.

Additional findings

Marked tenderness was noted in the Cervical (C3–C5) and Thoracic (Th3–Th5) spinal regions. The patient was unstable in the Romberg position. Limb coordination tests (L=D) showed intention tremor and mild tremor of the hands (Figure 1).

Examination and diagnostic findings

- June 9, 2020 (Chest CT): Postoperative evaluation following laparotomy and spleen-preserving gastrectomy with D2 lymph node dissection (April 9, 2020). Findings included linear areas of fibrosis and a consolidation in the lower lobe of the right lung.

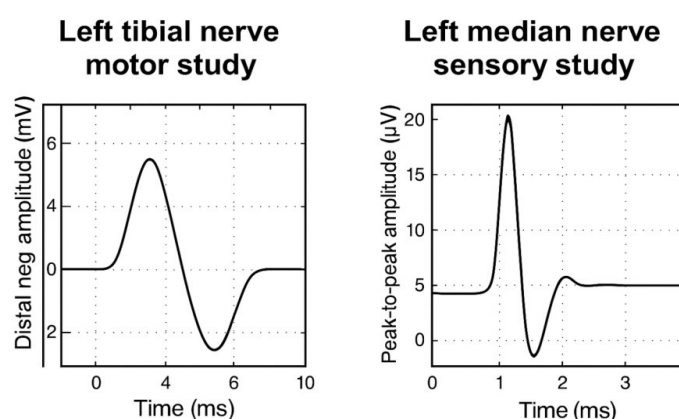


Figure 1. Electro Neuro Myo Graphy (ENMG) findings based on the patient's original neurophysiological data, reconstructed using AI-assisted image processing. **A)** Upper limb study demonstrating reduced compound muscle action potential amplitudes and slowed conduction velocities, consistent with mixed axonal–demyelinating polyneuropathy. **B)** Lower limb study showing similar abnormalities with more pronounced sensory involvement.

- July 16, 2020 (Chest CT): Revealed polysegmental pneumonia of the right lung, affecting approximately 30% of lung parenchyma.
- July 28, 2020 (Chest CT, post-discharge): Showed resolution phase pneumonia with only 5% lung involvement.
- July 11, 2020 (PCR test): SARS-CoV-2 detected.
- July 28, 2020 (Control PCR): SARS-CoV-2 not detected.
- May 20, 2020 (Brain MRI): Revealed hypoplasia of the right vertebral artery.
- August 13, 2020 (Cervical Spine MRI): Showed degenerative changes, spondyloarthrosis and disc protrusions at levels C3–C4, C4–C5, C5–C6 and C6–C7.
- August 13, 2020 (Thoracic Spine MRI): Demonstrated degenerative changes in the thoracic spine.
- August 28, 2020 (ECG): Sinus rhythm, heart rate 75 bpm, normal electrical axis.
- August 24, 2020 (ENMG): Detected sensory and motor nerve impairments in both upper and lower extremities, more pronounced on the left. Findings were consistent with an axonal-demyelinating polyneuropathy: reduced amplitude of muscle responses and slowed conduction velocity in both motor and sensory fibers.
- August 7, 2020 (Complete Blood Count): Leukocytes $3.2 \times 10^9/L$, erythrocytes $3.95 \times 10^{12}/L$, hemoglobin 114 g/L, platelets $202 \times 10^9/L$, ESR 15 mm/h.
- August 7, 2020 (Urinalysis): Volume 40 ml; light yellow, cloudy; specific gravity 1030; protein 0.28 g/L; glucose negative; epithelium 5–6; leukocytes 1.
- August 7, 2020 (Biochemistry): Total protein 67.9 g/L, glucose 7.4 mmol/L, urea 5.1 mmol/L, creatinine 66.8 $\mu\text{mol/L}$, ALT 21.7 U/L, AST 23.6 U/L, bilirubin 12.4 $\mu\text{mol/L}$, calcium 1.09 mmol/L, potassium 4.4 mmol/L, glycated hemoglobin 5.2%.

Chest CT scan of the patient, obtained on April, 18, 2022 demonstrating an area of pulmonary fibrosis (circled) in the right lung (Figure 2).

Final diagnosis

- G61.0 – Guillain-Barré Syndrome. G61.8 – Other inflammatory polyneuropathies following SARS-CoV-2-associated pneumonia

Symptomatic therapy

- Infusion therapy with 0.9% sodium chloride, 400 mL daily
- Neuromidin (B vitamins), 15 mg in 1.0 mL administered intramuscularly once daily for 10 days

- Pentoxifylline 100 mg in 250 mL of 0.9% sodium chloride administered intravenously once daily
- Therapeutic physical exercise regimen

The patient was discharged in improved condition. Gastric cancer remains in remission, with no evidence of recurrence over a five-year follow-up period. However, as of 2025, the patient continues to experience persistent weakness in the lower extremities.

Discussion

This case highlights the development of Guillain-Barré Syndrome (GBS) in an immunocompromised patient following SARS-CoV-2-associated pneumonia. The patient had recently completed the fifth cycle of adjuvant chemotherapy after undergoing gastrectomy for gastric cancer. The diagnosis of GBS was confirmed through Electro Neuro Myo Graphy (ENMG), which revealed a mixed axonal and demyelinating sensorimotor neuropathy, with features consistent with Acute Motor And Sensory Axonal Neuropathy (AMSAN).

GBS is an acute autoimmune polyradiculoneuropathy characterized by rapidly progressive limb weakness, distal sensory disturbances, cranial nerve involvement and the absence of deep tendon reflexes. Cerebrospinal fluid analysis typically demonstrates albuminocytologic dissociation. The three primary electrophysiological subtypes of GBS include Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN) and Acute Motor And Sensory Axonal Neuropathy (AMSAN) [3,4].

In a 2021 systematic review by Aladawi M, et al. [5], 109 cases of SARS-CoV-2-associated GBS were analyzed. The average patient age was 56 years and neurological symptoms typically developed approximately 12 days after the onset of COVID-19 symptoms. While most cases were classified as AIDP, axonal variants, including AMSAN as observed in our patient were also reported.

During the COVID-19 pandemic, the incidence of GBS reportedly increased from the pre-pandemic baseline of 0.6–4 cases per 100,000 individuals annually to as high as 15 per 100,000 among patients infected with SARS-CoV-2 [6-8].

The pathogenesis of GBS in the context of SARS-CoV-2 infection is believed to involve post-infectious immune dysregulation. Elevated levels of pro-inflammatory cytokines, including Tumor Necrosis Factor-Alpha (TNF- α) and Inter Leukin-17 (IL-17), have been associated with GBS and correlate with disease severity [9-11]. COVID-19 often induces a hyperinflammatory state commonly referred to as a cytokine storm which may provoke autoimmune responses targeting components of the peripheral nervous system [4,12]. Additionally, the possibility of direct viral neuroinvasion is supported by case reports such as one involving SARS-CoV-2 RNA detected in the cerebrospinal fluid of a child with GBS [9].

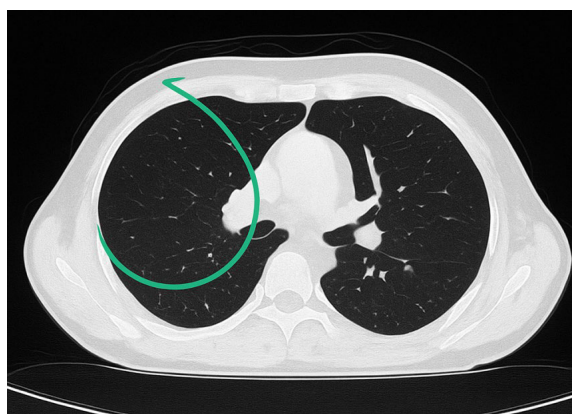


Figure 2. Chest CT scan April 18, 2022.

Another proposed mechanism is molecular mimicry, wherein viral antigens share structural similarities with peripheral nerve components, resulting in the generation of autoantibodies. In COVID-19-associated GBS, antiganglioside antibodies commonly linked to axonal variants have been detected in some cases [5]. Although antiganglioside antibodies were not tested in our patient, the ENMG findings of axonal damage suggest that such immune-mediated mechanisms may have contributed to disease development.

In oncology patients, particularly those undergoing cytotoxic chemotherapy, immunosuppression may enhance susceptibility to viral reactivation and dysregulated immune responses. In this case, the patient's immunocompromised state due to both underlying malignancy and recent chemotherapy likely predisposed her to post-infectious autoimmune complications. The onset of GBS following SARS-CoV-2 infection, characterized by 30% pulmonary involvement, underscores the potential neurotropic and immunomodulatory properties of the virus.

This case emphasizes the importance of maintaining clinical vigilance for post-infectious neurological complications such as GBS in immunocompromised populations. Prompt diagnosis through clinical evaluation and ENMG, coupled with early initiation of therapy, is essential to minimizing long-term morbidity.

Conclusion

In this case, Guillain-Barré Syndrome developed in an immunocompromised patient following SARS-CoV-2-associated pneumonia after undergoing gastrectomy and multiple courses of adjuvant chemotherapy for gastric cancer. It is plausible that the COVID-19 infection, through a combination of direct neuroinvasive potential, systemic inflammation and cytokine-mediated immune activation, triggered autoimmune mechanisms culminating in axonal neuropathy.

This case underscores the importance of recognizing neurological complications such as GBS in cancer patients recovering from COVID-19, particularly those with chemotherapy-induced immunosuppression. Early neurological evaluation and timely initiation of supportive therapy are critical to improving outcomes in this high-risk population.

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Human Subjects

Consent for treatment and open access publication was obtained or waived by all participants in this study.

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All authors have declared that no financial support was received from any organization for the submitted work.

Financial Relationships

All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

Other Relationships

All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Patient Consent Statement

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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None.

Conflict of Interest

Authors of this work have nothing to disclose.

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