

Guillain-Barré syndrome in remission of Ulcerative colitis: a case report

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Abstract

This case report provides a unique instance of a patient who developed Guillain-Barré syndrome (GBS) during the remission phase of ulcerative colitis (UC). GBS is a rare but severe autoimmune disorder affecting the peripheral nervous system, while UC is a chronic inflammatory bowel disease. The co-occurrence of GBS and UC is rare, and its underlying mechanisms are not well understood. The patient's medical history and the course of their treatment are discussed in detail to provide a comprehensive understanding of the condition.

The onset of GBS in this case report was characterized by lower extremity weakness and numbness of the upper extremities, which is consistent with the typical symptoms of GBS. The electromyography (EMG) results showed low limb nerve conduction and velocity with low amplitude, which supports the diagnosis of GBS. The treatment involved intravenous (IV) methylprednisolone, which is a commonly used steroid in the management of GBS. The patient showed improvement in his symptoms, with the weakness and numbness disappearing and muscle strength recovering, which is in line with the expected outcome of treatment with steroids. This case report provides valuable insight into the complex interplay between GBS and UC, and the potential impact of autoimmune diseases on each other. The findings of this report will contribute to the existing literature on GBS and UC, and help healthcare professionals to make informed decisions regarding the diagnosis and management of these complex conditions. The report concludes with a discussion of the implications of the case and the need for further research to better understand the relationship between GBS and UC.

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Introduction

Guillain-Barré syndrome (GBS) is a rare but severe autoimmune disorder affecting the peripheral nervous system (1). Ulcerative colitis (UC) is a chronic inflammatory bowel disease. In this case report, we describe a unique instance of a patient who developed GBS during the remission phase of UC (2). This case highlights the importance of considering the possibility of GBS in patients with UC and emphasizes the need for prompt diagnosis and treatment of the disorder. This case report provides valuable insight into the complex interplay between GBS and UC, and the potential impact of autoimmune diseases on each other. The co-occurrence of

GBS and UC is rare, and its underlying mechanisms are not well understood (3-5). The patient's medical history and the course of their treatment are discussed in detail to provide a comprehensive understanding of the condition. The findings of this report will contribute to the existing literature on GBS and UC, and help healthcare professionals to make informed decisions regarding the diagnosis and management of these complex conditions. The report concludes with a discussion of the implications of the case and the need for further research to better understand the relationship between GBS and UC.

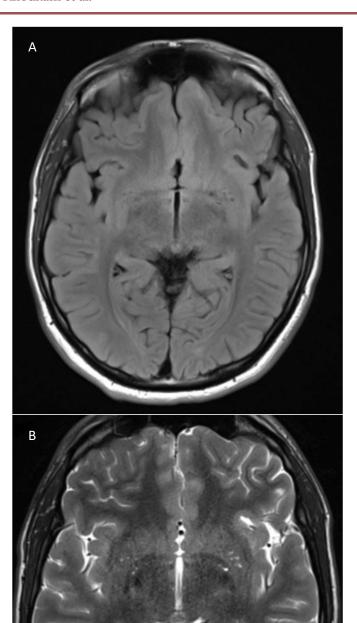


Figure 1. Axial FLAIR (A) and T2-weighted MRI

Case Presentation

A 34-year-old male with UC from two years ago was in remission for a year and had no gastrointestinal symptoms such as diarrhea, abdominal pain, fever, and no extra intestinal manifestations.

One month ago he was admitted to the Neurology department due to lower extremity weakness, numbness of upper extremities, shortness of breath, and difficulty swallowing. the patient was fully conscious. Neurological examinations showed motor deficits in both the upper and lower extremities. The eye movement was normal and there was no speech disorder. The muscle power of bilateral upper limbs was 3/5 and both legs showed 2/5. Examination of cranial nerve 2-12 was normal. He was admitted to the ICU to continue his neurological condition and ventilator in case the diaphragmatic paralysis. Brain MRI and CT scan was normal (Figure 1). Laboratory results showed normal glucose and increased albumin. Cerebrospinal fluid analysis showed no WBC and negative culture on gram stain. Electromyography (EMG) showed limb nerve conduction and velocity were low with low amplitude. The GBS was diagnosed based on the symptoms and electromyographic injury. He was managed by intravenous (IV) methylprednisolone. The weakness and numbness disappeared and muscle strength recovered. After being discharged he continued Methylprednisolone.

Discussion

The association between UC and GBS is well documented, although it is not well understood why UC patients are at increased risk of developing GBS (6-8). The onset of GBS in this case report was characterized by lower extremity weakness and numbness of the upper extremities, which is consistent with the typical symptoms of GBS. Additionally, the electromyography (EMG) results showed low limb nerve conduction and velocity with low amplitude, which supports the diagnosis of GBS.

The treatment in this case report involved intravenous (IV) methylprednisolone, which is a commonly used steroid in the management of GBS. The patient showed improvement in his symptoms, with the weakness and numbness disappearing and muscle strength recovering, which is in line with the expected outcome of treatment with steroids.

In conclusion, this case report is consistent with previous reports of GBS in patients with UC and highlights the need for close monitoring and early recognition of GBS in patients with UC. The patient's response to IV methylprednisolone is also in line with the expected outcomes of treatment for GBS.

Declarations

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Conflict of interest

The authors have no conflicts of interest to disclose.

Consent for publication

This manuscript has been approved for publication by all authors.

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