ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 9, Issue – 1, February – 2024 , Page No. : 22 – 25

Atypical Presentation of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) with Psychomotor Symptoms in a Young Male: A Case Report

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Citation this Article: Dr. Amitesh Sattineni, Dr. Manimekali P, Dr. Saketh Ramineni, Dr. Viknesh Prabhu, "Atypical Presentation of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) with Psychomotor Symptoms in a Young Male: A Case Report", IJMSIR - February - 2024, Vol – 9, Issue - 1, P. No. 22 – 25.

Type of Publication: Case Report

Conflicts of Interest: Nil

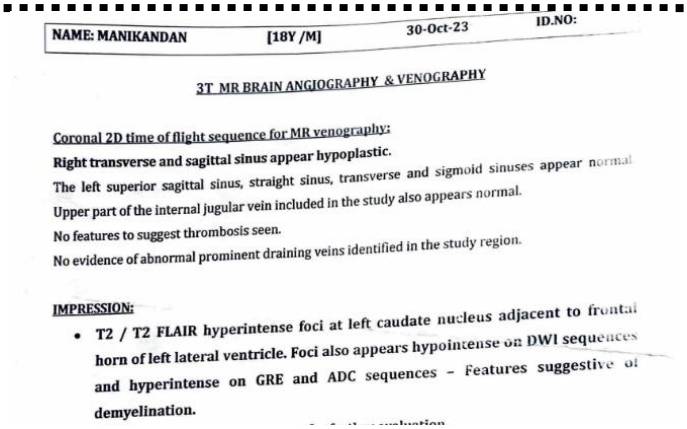
Abstract

Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is acknowledged as the most prevalent variant of Guillain-Barre syndrome, though diagnosing it can be intricate. Patients often exhibit symptoms that might be misconstrued as psychosomatic, adding complexity to the diagnostic process. This case involves a patient who presented with insomnia, tremors, and restlessness, without any associated dreams. Unlike the conventional presentation of AIDP, this atypical manifestation underscores the challenges in identifying Guillain-Barre syndrome. The case report delves into this unique presentation, elucidates the diagnostic journey for Guillain-Barre syndrome, briefly reviews the connection between psychosis and psychosomatic complaints in Guillain-Barre patients, and emphasizes the imperative of thorough evaluation, bearing in mind that psychosomatic disorders should only be considered after excluding other potential causes.

Keywords: Syndrome AIDP, EEG, NMO.

Case Report

This case involves an 18-year-old male presenting with a five-year history of bilateral upper limb tremors exacerbated over the past 10 days, accompanied by giddiness for six months, decreased sleep for three months, and diminished interest in the outside world with irritability for six months. Additionally, the patient reported a month-long history of on-and-off fever and a six-year history of smoking and alcohol use, which was discontinued a week before presentation. Following a working diagnosis of Acute Inflammatory Demyelinating Polyneuropathy (AIDP), routine investigations, including CBC, RFT, LFT, and urine routine, were normal. Serum aquaporins, done to rule out Neuromyelitis Optica Spectrum Disorder (NMO), were within the normal range. MRI brain revealed T2/FLAIR hyperintense foci at the left caudate nucleus, suggestive of demyelination, and EEG results were normal. Vitamin B12 levels were in the normal range.CSF that revealed RBC 0, WBC 2, glucose 66, and protein 73. A neurology consultation led to the initiation of intravenous steroids (methylprednisolone), followed by a 15-day oral taper. The patient demonstrated symptomatic improvement, emphasizing the importance of recognizing atypical presentations and the role of comprehensive diagnostic evaluations in managing AIDP.



NAME: MANIKANDAN [18Y /M] 30-Oct-23 ID.NO:

3T MR BRAIN ANGIOGRAPHY & VENOGRAPHY

Coronal 2D time of flight sequence for MR venography:
Right transverse and sagittal sinus appear hypoplastic.
The left superior sagittal sinus, straight sinus, transverse and sigmoid sinuses appear normal.
Upper part of the internal jugular vein included in the study also appears normal.
No features to suggest thrombosis seen.
No evidence of abnormal prominent draining veins identified in the study region.

IMPRESSION:

- T2 / T2 FLAIR hyperintense foci at left caudate nucleus adjacent to frontal horn of left lateral ventricle. Foci also appears hypointense on DWI sequences and hyperintense on GRE and ADC sequences – Features suggestive of demyelination.

Discussion

The atypical presentation of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) in the case of an 18-year-old male with psychomotor symptoms introduces a compelling narrative that challenges the traditional understanding of this neurological disorder. While AIDP typically unfolds with ascending weakness, acroparesthesia, and hyporeflexia, this particular case adds a layer of complexity by incorporating psychomotor symptoms, highlighting the importance of recognizing diverse manifestations for accurate diagnosis and prompt management. The intricate nature of AIDP often makes it challenging to diagnose, with the disorder frequently dismissed initially as psychosomatic, particularly when atypical symptoms are present. In this case, the patient's five-year history of bilateral upper limb tremors, recent exacerbation, and concurrent complaints of giddiness, decreased sleep, diminished interest in the outside world, and irritability painted a complex clinical picture. The history of on-and-off fever added another layer of complexity, prompting a comprehensive diagnostic workup. Routine investigations, including complete blood count, renal and liver function tests, and urine analysis, yielded normal results. Serum aquaporins were within the normal range, ruling out Neuromyelitis Optica Spectrum Disorder (NMO). However, the MRI brain revealed T2/FLAIR hyperintense foci at the left caudate nucleus, suggestive of demyelination. While EEG results were within normal limits, the vitamin B12 levels were also found to be normal. The culmination of these findings led to a neurological evaluation and the working diagnosis of AIDP.AIDP, as the most common variant of Guillain-Barre syndrome (GBS) in North America and Europe, typically involves lymphocytic infiltration of the myelin sheath and damage to Schwann cell membrane components. Unlike other GBS variants that entail axolemmal damage, AIDP results in segmental damage of the myelin sheath and subsequent nerve conduction abnormalities. However, what sets this case apart is the incorporation of psychomotor symptoms into the clinical presentation.Psychomotor symptoms, encompassing alterations in cognitive and motor functions, are not commonly associated with AIDP. The traditional focus has been on the ascending motor and sensory deficits, making the inclusion of psychomotor manifestations a noteworthy deviation from the norm. The case challenges preconceived notions about the clinical spectrum of AIDP, urging clinicians to broaden their understanding of potential symptomatology. The introduction of psychomotor symptoms in AIDP prompts a deeper exploration of the neural mechanisms and immunological responses involved in the disorder. While AIDP is generally considered to result from a humorally mediated immune response, the specific pathways leading to psychomotor symptoms remain elusive. The neurological consultation and subsequent initiation of intravenous steroids underscore the need for prompt intervention in the face of atypical presentations, emphasizing the significance of early diagnosis and treatment to improve outcomes. The broader discussion on AIDP and its variants, such as Miller-Fisher syndrome, AMAN, and AMSAN, involves delving into the intricate interplay between the immune system and the peripheral nervous

system. C. jejuni infection is a common antecedent in GBS cases, including the Miller-Fisher variant, highlighting the role of infectious triggers in the pathogenesis. The potential cross-reactivity between antibodies produced in response to C. jejuni and neural antigens underscores the complexity of the immune response in AIDP. Moreover, exploring the landscape of AIDP necessitates an understanding of diagnostic criteria and classification systems. The Brighton diagnostic case classifications provide a structured framework for categorizing GBS cases based on specific criteria levels. These criteria encompass elements such as decreased or absent deep tendon reflexes, time elapsed from onset to nadir, symmetric and flaccid weakness, cerebrospinal fluid (CSF) findings, and nerve study findings consistent with GBS variants. AIDP, falling under Level 3 criteria, emphasizes the importance of considering alternative diagnoses. The diagnostic journey further involves electrophysiological studies, with AIDP characterized by distal compound action muscle potential (dcMAP), motor conduction velocity, distal motor latency, compound muscle action potential after proximal stimulation (pCMAP)/dcMAP ratio, and F-response latency. These criteria aid in distinguishing AIDP from other GBS variants like AMAN and AMSAN, which present with more severe and rapid courses, often leading to early respiratory failure.

In the context of this atypical AIDP case, the presence of psychomotor symptoms necessitates a nuanced discussion about the potential involvement of the central nervous system (CNS). Psychotic symptoms in GBS patients have been documented in studies, with mental status changes observed in a significant percentage of cases. Vivid dreams, illusions, hallucinations, and paranoid delusions represent the spectrum of psychotic symptoms associated with GBS, adding a layer of complexity to the clinical presentation. The correlation between CSF albuminocytologic dissociation and the occurrence of psychotic symptoms further underscores the intricate relationship between the immune response and the CNS in AIDP. The case at hand aligns with existing literature that reports patients initially presenting with psychotic symptoms, mistakenly attributed to psychosomatic disorders, resulting in delayed care. From an emergency medicine perspective, the challenges in diagnosing AIDP, particularly in the presence of psychomotor symptoms, are evident. The rarity of the condition, coupled with its diverse clinical presentations, makes it crucial for emergency physicians to maintain a high index of suspicion. The diagnosis of AIDP often involves a combination of clinical evaluation, electrophysiological studies, CSF analysis, and, in some cases, diagnostic imaging. While MRI does not play a primary role in diagnosing AIDP, it can offer valuable insights, especially when other supportive studies are inconclusive. Gadolinium enhancement of the cauda equina nerve roots has shown sensitivity in diagnosing GBS in previous studies, and its utility in monitoring response to therapy further emphasizes the potential role of imaging in the comprehensive management of AIDP.

The declining cases of polio have positioned GBS as the most common cause of acute flaccid paralysis, with an annual incidence of approximately 100,000 cases. The disease typically progresses slowly over 2 to 4 weeks, but rapid onset with respiratory failure occurring within one day is not unheard of. The severity of the disease course necessitates a prompt and accurate diagnosis to prevent complications and optimize outcomes.

In conclusion, the atypical presentation of AIDP in a young male with psychomotor symptoms challenges the traditional narrative surrounding this neurological disorder. The incorporation of psychomotor

manifestations adds a layer of complexity to the clinical spectrum of AIDP, urging clinicians to consider diverse presentations for timely intervention. The case underscores the importance of maintaining a broad differential diagnosis in the emergency setting, recognizing the potential for atypical manifestations, and implementing a comprehensive diagnostic approach for optimal patient care. The intricate interplay between the immune system and the peripheral and central nervous systems in AIDP remains a subject of ongoing research, emphasizing the need for continued exploration of the underlying mechanisms contributing to the diverse clinical phenotypes observed in this disorder.

Conclusion

In conclusion, the atypical presentation of AIDP in a young male with psychomotor symptoms challenges the traditional narrative surrounding this neurological disorder. The incorporation of psychomotor manifestations adds a layer of complexity to the clinical spectrum of AIDP, urging clinicians to consider diverse presentations for timely intervention. The case underscores the importance of maintaining a broad differential diagnosis in the emergency setting, recognizing the potential for atypical manifestations, and implementing a comprehensive diagnostic approach for optimal patient care. The intricate interplay between the immune system and the peripheral and central nervous systems in AIDP remains a subject of ongoing research, emphasizing the need for continued exploration of the underlying mechanisms contributing to the diverse clinical phenotypes observed in this disorder.

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