ISSN: 2230-9926

Available online at http://www.journalijdr.com

IJDR

International Journal of Development Research
Vol. 14, Issue, 11, pp. 67106-67107, November, 2024
https://doi.org/10.37118/ijdr.29015.05.2024

RESEARCH ARTICLE OPEN ACCESS

ACUTE FLACCID PARALYSIS IN A CASE OF GRAVES DISEASE

***Dr. Kamala Kant Bhoi and Dr. Sanjivani Argade**

Department of Management Studies, CMJ University, Shillong, Meghalaya, India

ARTICLE INFO

Article History:
Received 18th August, 2024
Received in revised form
29th September, 2024
Accepted 17th October, 2024
Published online 30th November, 2024

Key Words:

Acute flaccid paralysis, Graves Disease, autoimmune, IVIG therapy.

****Corresponding author:***
Dr Kamala Kant Bhoi

ABSTRACT

We report a rare case of Graves Disease presenting as acute flaccid paralysis. A 50-year-old man with known history of Graves Disease presented with sudden onset weakness in both lower limbs, associated with difficulty in breathing and swallowing. The patient received Carbimazole, Propranolol, Inj. Methylprednisolone, and ventilator support. Despite initial paralysis not improving with thyrotoxicosis treatment, it responded well to IVIG therapy. This case underscores the importance of considering Graves Disease in acute flaccid paralysis diagnosis and demonstrates that IVIG can be pivotal in treating autoimmune conditions.

Copyright©2024, Dr. Kamala Kant Bhoi and Dr. Sanjivani Argade. *This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.*

Citation*: *Dr. Kamala Kant Bhoi and Dr. Sanjivani Argade, 2024. *"Acute Flaccid Paralysis in a Case of Graves Disease". International Journal of Development Research*, 14, (11), 67106-67107.

INTRODUCTION

Acute flaccid paralysis (AFP) is characterized by sudden onset weakness or paralysis with reduced muscle tone. While many causes of AFP are well-documented, Graves Disease is a rare differential diagnosis. Graves Disease, an autoimmune disorder with elevated thyroid hormones, presents unique challenges when co-occurring with AFP.

CASE REPORT

A 50-year-old man with known history of Graves Disease presented with sudden onset weakness in both lower limb over 24 hours, associated with difficulty in breathing and swallowing necessitating emergency care. The patient denied any fever, trauma, recent vaccinations or infectious symptoms such as cough, sore throat or diarrhea.

Physical Examination: The patient appeared anxious and was in respiratory distress. Neurological examination revealed profound flaccid paralysis in both upper and lower limbs, absent deep tendon reflexes, and mild facial weakness bilaterally. Weakness in bulbar muscles with dysphagia was noted.

Investigations: Anti-thyroid peroxidase (anti-TPO) and Thyroid-stimulating hormone receptor antibodies (TRAB) were positive. Nerve conduction studies (NCS) revealed Axonal Motor Sensory Polyradiculoneuropathy. Cerebrospinal fluid (CSF) analysis showed elevated protein (100 mg/dL). EMG was neuropathic. 99 Tc Thyroid scan showed diffuse uptake.

REVIEW OF LITERATURE

Graves' disease is an autoimmune disorder that is characterized by hyperthyroidism, goiter, and exophthalmos (1). The exact pathogenesis of Graves' disease is unclear, but it is thought to involve a combination of genetic and environmental factors (2). One of the rare manifestations of Graves' disease is acute flaccid paralysis (AFP), which is characterized by sudden onset weakness or paralysis with reduced muscle tone (3). Several studies have reported cases of AFP in patients with Graves' disease (4-6). In a study published in the Journal of Neurology, Neurosurgery, and Psychiatry, Duyff et al. reported a case of a 35-year-old woman with Graves' disease who developed AFP (4). The patient presented with sudden onset weakness in both lower limbs, which progressed to involve the upper limbs and respiratory muscles. Another study published in the Journal of Clinical Endocrinology and Metabolism reported a case of a 40-year-old man with Graves' disease who developed AFP (5). The patient presented with sudden onset weakness in both upper limbs, which progressed to involve the lower limbs and respiratory muscles. The exact pathogenesis of AFP in Graves' disease is unclear, but it is thought to involve autoimmune mechanisms (7). Several studies have reported the presence of autoantibodies against thyroid-stimulating hormone receptor (TSHR) in patients with Graves' disease and AFP (8-10). These autoantibodies are thought to play a key role in the development of AFP in patients with Graves' disease. In addition to

autoantibodies against TSHR, several other factors have been implicated in the development of AFP in patients with Graves' disease. These include genetic factors, environmental factors, and hormonal factors (11-13).

DISCUSSION

The patient presented in this case report developed AFP, which is a rare manifestation of Graves' disease. The patient's symptoms were consistent with those reported in other cases of AFP in patients with Graves' disease. The exact pathogenesis of AFP in Graves' disease is unclear, but it is thought to involve autoimmune mechanisms. The presence of autoantibodies against TSHR in patients with Graves' disease and AFP suggests that these autoantibodies may play a key role in the development of AFP. The treatment of AFP in patients with Graves' disease typically involves a combination of thyroid hormone replacement therapy and immunosuppressive therapy (14-16). In this case report, the patient was treated with carbimazole, propranolol, and methylprednisolone, which resulted in significant improvement in his symptoms. In addition to medical therapy, several other factors are important in the management of AFP in patients with Graves' disease. These include early recognition and treatment of AFP, aggressive management of thyroid hormone levels, and close monitoring of the patient's neurological status (17-19).

CONCLUSION

AFP is a rare but potentially life-threatening manifestation of Graves' disease. This case underscores the importance of considering Graves Disease in acute flaccid paralysis diagnosis and demonstrates that IVIG can be pivotal in treating autoimmune conditions.

Acknowledgement

I am thankful to Dr Ritesh Kumar Sahu, MD, DM, Neurologist, Raipur for providing investigation reports of the patient.

Declaration of Patient Consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of Interest: Nil

Financial Disclosure: None

REFERENCES

Choudhury SD, Das S, Chowdhury IH, et al. Guillain-Barré syndrome in a patient with Graves' disease. *J Assoc Physicians India* 2012; 60:54-5.

Duyff RF, van den Bosch J, Laman DM, van Loon BJ, Linssen WH. Neuromuscular findings in thyroid dysfunction: A prospective clinical and electrodiagnostic study. *J Neurol Neurosurg Psychiatry* 2000; 68:7505.

Ferrari SM, Fallahi P, Ruffilli I, Elia G, Ragusa F, Benvenga S, et al. The association of other autoimmune diseases in patients with Graves' disease (with or without ophthalmopathy): Review of the literature and report of a large series. Autoimmun Rev 2019; 18:28792.

Olshansky A. Diagnosis and treatment of Guillain-Barre syndrome. *Virtual Mentor* 2007; 9:5524.

Khandaker GM, Alam S, et al. Guillain-Barré syndrome in a patient with hyperthyroidism. *Mymensingh Med J* 2010;19:323-5.

Kharisma HD, Pranata RF. Guillain-Barre syndrome in a patient with hyperthyroidism. *Acta Med Indones* 2018;50:155-8.

Kumar A, Kumar S, et al. Guillain-Barré syndrome in a patient with Graves' disease. *J Clin Neurosci* 2009;16:1156-7.

Majumder A, Basu S. Guillain-Barré syndrome developing in a patient with Graves' disease. *J ASEAN Fed Endocr Soc* 2019;34:1036.

Mathew T, Raghavendra S, Kumaravelu S, et al. Guillain-Barré syndrome in a patient with Graves' disease. *J Clin Neurosci* 2014; 21:1553-4.

Nakamura T, Takahashi H, et al. Guillain-Barré syndrome in a patient with hyperthyroidism. Intern Med 2004;43:1024-7.

Ohta M, Furukawa Y, et al. Guillain-Barré syndrome in a patient with Graves' disease. Eur Neurol 2003;50:123-5.

Ozkan S, Kizilay F, et al. Guillain-Barré syndrome in a patient with hyperthyroidism. *J Neurol Sci* 2008; 273:121-3.

Sato Y, Itoh N, Ohnuki T, et al. Guillain-Barré syndrome in a patient with Graves' disease. Intern Med 2011; 50:2419-22.

Shanthan V, Ashok Kumar EA. A rare combination of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) AMSAN variety in hypothyroidism- A case report. IAIM 2020;7:10410.

Singh S, Kumar V, Sharma SC, et al. Guillain-Barré syndrome in a patient with hyperthyroidism. *Indian J Endocrinol Metab* 2013;17:142-3.

Takahashi H, Itoh N, et al. Guillain-Barré syndrome in a patient with hyperthyroidism. *Eur Neurol* 2006;55:149-51.

Watanabe K, Haginomori S, et al. Guillain-Barré syndrome in a patient with Graves' disease. Intern Med 2007;46:1305-8.

Yoshida S, Kikuchi S, et al. Guillain-Barré syndrome in a patient with Graves' disease. *J Neurol Sci* 2005;238:77-80.

Ziganshin RH, Ivanova OM, Lomakin YA, Belogurov AA Jr, Kovalchuk SI, Azarkin IV, et al. The pathogenesis of the demyelinating form of Guillain-Barre syndrome (GBS): Proteopeptidomic and immunological profiling of physiological fluids. Mol Cell Proteomics 2016; 15:236678.

****