

Atypical Presentation of Guillain Barre Syndrome as Urinary Retention & Constipation.

Dr.Onkar Kakare*,Dr.Piyush Patel*,Dr.Isha Bansal*,Dr.Shailendra Mane**,Dr.Vinay Wagh**.

*Resident , Dept. of Medicine, D.Y. Patil Medical College & Hospital, Kadamwadi, Kolhapur.

**Professor , Dept. of Medicine, D.Y. Patil Medical College & Hospital, Kadamwadi, Kolhapur.

Corresponding Author: Dr.Onkar Kakare

Abstract- GB syndrome is an acute inflammatory demyelinating polyneuropathy, an autoimmune disease affecting the peripheral nervous system which is triggered by acute infection. As it is a disease of peripheral nerves, if autoimmune involvement like bladder & bowel dysfunction occurs early then this raises suspicion for other diagnosis because this involvement occurs late in the disease course. Normally in 25% cases bladder involvement is seen. Hence early bladder involvement in GB syndrome is considered as atypical presentation of disease which is not commonly seen. Here we are reporting a case of GB syndrome who presented as urinary retention with constipation and progressive lower limb weakness. This led us to suspect other diagnosis ,but it is a atypical presentation confirmed by NCV test and when treated with plasma pheresis, patient recovered.

Keywords- Demyelinating, Guillain Barry syndrome, Polyneuropathy

Date of Submission: 09-02-2018

Date of acceptance: 26-02-2018

I. Introduction

GB syndrome is an acute onset post infectious polyneuropathy involving mainly motor but sometimes also sensory and autonomic nerves. It is an acquired condition which is characterized by progressive symmetrical distal and proximal tingling and weakness. If bladder dysfunction is a prominent feature & occurs early then this raises suspicion for other diagnosis.(1). GB syndrome can affect anybody. It can strike at any age & both sexes are equally prone to the disorder. The syndrome is rare, afflicting only about one person in one lakh(2). In GB syndrome, cardiovascular dysfunction is seen in 60% patients while urinary dysfunction is seen in 25% patients(3).

II. Case report

A 51 year old male, an auto rickshaw driver, came with complaints of fullness of abdomen, difficulty in passing urine since two days & weakness in the both lower limbs since two days. Two days back, when he woke up in the morning, he went to the washroom but was unable to pass urine and stools. He gives history of straining but was not able to defecate. On the next morning, he developed weakness in both lower limbs followed by weakness of both upper limbs which he developed on the same evening. This weakness went on gradually progressing to such an extent that he couldn't stand without support nor eat food with his own hands ; after which he was brought to our OPD .On examination, patient was uncomfortable, talking properly, obeying commands . His speech was clear & normal. He was averagely built & averagely nourished. There was hypotonia in all four limbs with 0/5 power in both lower limbs & 3/5 in both upper limbs. All deep tendon reflexes were absent and all sensations were intact all over the body . Plantars were mute. His single breath count was 14. On abdominal examination, there was distension in the lower abdomen suggestive of bladder retention. The patient was admitted to the ICU and his MRI brain & MRI spine was done which came normal. Now his lumbar puncture was done and till CSF reports were awaited, he was started on high dose of Glucocorticoids. The next morning, on examination –He had power 5/5 in upper limbs & 2/5 in lower limbs. CSF report findings- sugar- 71, protein – 179, TLC- 1, Polymorph- 0, Lymphocytes – 100. Nerve conduction studies of all the four limbs were suggestive of severe demyelinating plus axonal motor neuropathy. Glucocorticoids were omitted and patient was shifted on plasmapheresis of which five cycles were given. After the third cycle, patient started improving in his power. After fifth cycle, his upper limb power regained to 5/5 and lower limb power 3/5. His ultrasound of abdomen did not show any prostatomegaly . The patient was discharged on seventh day with Foleys catheter for his bladder retention. Patient was followed for three weeks and he showed improvement in walking but his bladder voiding did not improve till third week so he was maintained on Silicon Foleys catheter.

III. Discussion

Acute ascending weakness was first described by Landry in 1859 but the full extent of disease was described by Guillain, Barre, Strohl in 1916. The disease gained international notoriety under the name that remains today, Guillain-Barre syndrome. GB syndrome is now the world's most common cause of acute neuromuscular paralysis. It affects 0.4 to 2.4/ 1 Lakh people annually with a bimodal incidence in early and late adulthood, however affects young adults more than elderly(4). It consists of atleast four subtypes of acute peripheral neuropathy. The histological appearance of acute inflammatory demyelinating polyradiculopathy subtypes resembles experimental autoimmune neuritis which is predominantly caused by T cells directed against peptides from the myelin proteins P0, P2, and PMP22. The role of T-cell-mediated immunity in AIDP remains unclear and there is evidence for the involvement of antibodies and complement. Strong evidence now exists that axonal subtypes of Guillain-Barré syndrome, acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN), are caused by antibodies to gangliosides on the axolemma that target macrophages to invade the axon at the node of Ranvier. About a quarter of patients with GB syndrome have had a recent *Campylobacter jejuni* infection, and axonal forms of the disease are especially common in these people. The lipo-oligosaccharide from the *C jejuni* bacterial wall contains ganglioside-like structures and its injection into rabbits induces a neuropathy that resembles acute motor axonal neuropathy. Antibodies to GM1, GM1b, GD1a, and GalNac-GD1a are in particular implicated in acute motor axonal neuropathy and, with the exception of GalNacGD1a, in acute motor and sensory axonal neuropathy. The Fisher's syndrome subtype is especially associated with antibodies to GQ1b, and similar cross-reactivity with ganglioside structures in the wall of *C jejuni* have been discovered. Anti-GQ1b antibodies have been shown to damage the motor nerve terminal in vitro by a complement-mediated mechanism(5) As it is acute disease of peripheral nerves bowel and bladder involvement takes place late in the 2 to 3rd week of disease progression but bladder & bowel involvement in the initial presentation as chief complaint makes it as atypical presentation of GB syndrome[6].

IV. Conclusion

This atypical presentation of GB syndrome , leads the physician to think of other demyelinating diseases of spinal cord but ; careful observation of the clinical course and awareness and suspicion of a possibility of GB syndrome is essential to diagnose GB syndrome. Diagnosis must be confirmed by doing nerve conduction tests and CSF analysis and further treatment should be planned.

References –

- [1]. Stephen L. Hauser , Anthony A. Amato, Guillain Barry Syndrome & other immune mediated Neuropathies, Harrisons principle of internal medicine, 19th edition, McGraw –Hill Education 2015;2694
- [2]. Guillain-Barre syndrome fact sheet , NINDS , july 2011
- [3]. R. Sakakibara, T. Uchiyama et.al , urinary retention & sympathetic sphincter obstruction in axonal Guillain- Barre syndrome, Muscle Nerve 35: 111-115,2007
- [4]. Sheridan, Josh M, Smith, Dustin, Atypical Guillain- Barre in the emergency department, Western journal of emergency medicine: integrating emergency care with population health 11(1): 80-82, Jan 2010
- [5]. Prof. Richard AC Hughes, David R Corbinath, Guillain- Barre syndrome ,The Lancet: p1653-1666, Vol 366, No.9497, 5 November 2005
- [6]. Arup Kumar Kundu, Bedside clinics in Medicine part 1, 7th edition , 169

Dr. Onkar Kakare "Atypical Presentation of Guillain Barre Syndrome as Urinary Retention & Constipation.". "IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), Volume 17, Issue 2 (2018), PP 01-02.