

A rare case report on guillain barre syndrome and rheumatoid arthritis

Shaik Bobby Parveen¹, Bhushanam.Gayatri², Chandolu Suvarna Rani³, Bandaru Praveena^{4*}, Konda Ravi Kumar⁵

¹⁻³Student, ⁴Assistant Professor, ⁵Professor, Hindu College of Pharmacy, Amaravathi Road, Guntur. Andhra Pradesh, India

Corresponding Author: Bandaru Praveena

Email: praveenabandaru2018@gmail.com

Abstract

Guillain Barre syndrome (GBS) is an acute or subacute inflammatory demyelinating poly radiculoneuropathy. It is an acquired condition that is characterized by progressive, symmetrical, proximal, and distal tingling and weakness. We report a case of a 68 years old female who presented with complaints of quadraperisis, severe sensory motor axonal neuropathy, parasthesia, severe myalgia poly arthralgia and mononeuritis multiplex and later diagnosed as a case of guillainbarre syndrome and rheumatoid arthritis.

Keywords: GBS, Rheumatoid arthris, Immunoglobulins, Corticosteroids, Acute or subacute inflammatory demyelinating polyradiculoneuropathy, Plasma pherisis.

Introduction

Guillain- Barre syndrome (GBS) is a complicated degenerative neurological disorder, an acute or subacute monophasic, inflammatory demyelinating polyradiculoneuropathy that often follows an antecedent infection and presents by ascending progressive weakness that eventually leads to flaccid paralysis. It is an acquired condition which is characterized by progressive, symmetrical, proximal and distal tingling and weakness. Muscle stretch reflexes are decreased to absent and loss of sensation is common. This paper will discuss the causes, history, incidence and the adverse effects of 2 autoimmune disorders i.e, rheumatoid arthritis and Guillain-Barre syndrome. A 65 year female patient was admitted into neurology department.

Materials and Methods

Case Report

A 68 year female patient was admitted to the neurology department about persistent weakness in the lower extremities that later also affected the upper extremities and gradual onset with severe myalgia, polyarthralgia and paresthesia. The patient reported a previous gastroentritis infection since September 2018. Fever, on and off from September 2018. Her past medical, family and psychological history was not significant of serious neurological or musculoskeletal illness. No past medical history of hypertension, diabetes mellitus but has hypothyroidism since 10 years (on medication of L-thyroxine 50mcg).

Clinical findings, Diagnostic assessment and Therapeutic intervention

MRI brain report showed lacunar infract in the right capsulogangloinic region. MRI cervical spine report showed disc bulging from C3/C4 to C5/C6 impinging over theca. CSF exam showed compatible with CNS infection (microprotein:107.8 mg/dl) Rombergs test showed positive results. ANA control was positive. Culture sensitivity showed positive results for E.Coli. RA factor was done and the test shows positive results (27.4IU/ml). The patient was

diagnosed with 2 autoimmune disorders i.e rheumatoid arthritis and guillainbarre syndrome. The patient was immediately administered in CCU and steroids treatment was started immediately to treat rheumatoid arthritis. There is no improvement in patient condition. On further laboratory investigation it showed an increase in T4 and decrease in T3 levels, when 2D echo was done it was normal, liver function tests showed increased serum total bilirubin and serum indirect bilirubin, renal function tests showed decreased sodium, potassium, increased blood urea and serum creatinine. Complete blood picture was done and Hb value was 8gm/dl, TLC 15900 cells/cumm, neutrophils 80%, lymphocytes: 18%, PCV 20%, MCHC:30%, ESR 130mm/hr, serum protein:5.7gm/dl, serum albumin: 3gm/dl, histopathologic report showed microcytic, hypochromic anemia with neutrophilia. Single limb venous Doppler showed subcutaneous edema in the leg and foot (right lower limb) and thrombophlebitis (left lower limb). Serum uric acid was found to be 2.41mg/dl. A diagnosis of Guillain-Barre syndrome was made after utilizing the criteria that strongly support the diagnosis of Guillain Barre syndrome. Features required to rule out diagnoses other than Guillain Barre syndrome would include: no history of hexacarbon abuse, no evidence of porphyria, no history or culture evidence of diphtheria, no history or evidence of lead intoxication, symptoms not purely sensory, no evidence of poliomyelitis, botulism, toxic neuropathy, or tick paralysis.

Case Discussion

GBS is an acute monophasic demyelinating neuropathy. The disease is characterized by progressive motor weakness of limbs with a reflexia. Preceding antecedent infections, mostly bacterial and viral, are seen in half of the cases. Immunoglobulins and plasmapheresis have made a significant change in the course of the illness. About 12% - 20% of patients with GBS may require ventilatory support for respiratory paralysis. It is a complicated disorder which can be chronic or acute in nature. Its etiology is unclear although it has been associated with both cell - and humoral - mediated autoimmune mechanisms. Rheumatoid arthritis is an autoimmune and a chronic disease that causes pain,

stiffness, swelling and limited motion and function of many joints. While rheumatoid arthritis can affect any joint, the small joints in the hands and feet tend to be involved most often. Inflammation sometimes can affect organs as well, for instance, the eyes or lungs. It is characterized by synovial proliferation and a symmetric erosive arthritis of peripheral joints. In this case, The differentiation between guillainbarre syndrome and rheumatoid arthritis in the patient was done by evaluating the subjective evidences such as severe myalgia, quadraparesis, persistent weakness in limbs, paresthesia, dysphasia, weakness in eye muscles are reflexia, decreased plantar reflexia, poly neuropathy, increased urine frequency, sever motor sensory axonal neuropathy for guillainbarre syndrome, polyarthritis, quadraparesis, morning stiffness of Joints, mononeuritis multiplex for rheumatoid arthritis. In this patient rheumatoid arthritis was confirmed by +ve RA factor, guillainbarre syndrome was confirmed by +ve ANA blot test, electrophysiologic studies demonstrating evolving demyelination and an increased CSF protein content. The pathogenesis of Guillain-Barre syndrome shows edematous changes proximal to the spinal nerve root at the junction of the anterior and posterior roots. Researchers believe that the myelin destruction is limited to those areas of nerve trunks with intense inflammation suggesting that the inflammatory cells have a direct effect initiating the demyelination. The demyelination occurs primarily in areas infiltrated with inflammatory cells. Degeneration of the basement membrane of the Schwann cell results. This is due to macrophages in the presence of lymphocytes and not lymphocytes by themselves. This breakdown, which is unclear is associated with an autoimmune attack on a component of peripheral myelin. This attack is also mediated by T cells.

Treatment for Guillain-Barre Syndrome

Treatment for patients with Guillain-Barre syndrome depends on whether they have mildly acute, severely acute or chronic involvement. The incidence of death in one study was 1.5% to 8% of patients. Another study listed the death rate at 4.23% other common complications include ventilatory failure and cardiovascular instability for which intensive care support should be utilized. Ventilatory failure is caused by involvement of airway and respiratory muscles, particularly the diaphragm. Cardiovascular instability is due to involvement of the autonomic nervous system and results in labile blood pressure, cardiac arrhythmias, and hypovolemia. Compressive neuropathies occur in patients with protracted weakness and are an important cause of residual neurological deficits. The use of corticosteroids demonstrated no benefit. Plasmapheresis and intravenous immunoglobulins are the two main immunotherapy treatments for guillainbarre syndrome. The only well-investigated efficacious immunomodulatory therapy is plasmapheresis. Plasmapheresis has been shown to decrease ventilator dependence in severe Guillain-Barre syndrome. No irreversible complications of plasmapheresis were observed. Plasmapheresis attempts to reduce the body's

attack on the nervous system by filtering antibodies out of the bloodstream. Similarly, administration of IVIG neutralizes harmful antibodies and inflammation. These two treatments are equally effective, but a combination of the two is not significantly better than either alone. Plasmapheresis speeds recovery when used within four weeks of the onset of symptoms. IVIG works as well as plasmapheresis when started within two weeks of the onset of symptoms, and has fewer complications. IVIG is usually used first because of its ease of administration and safety. In this case IV IG are given to the patient which resulted in left ventricular dysfunction and acute interstitial nephritis as adverse drug reactions.

Treatment for Rheumatoid Arthritis

DMARDS, Anti inflammatory and analgesics are used in the treatment of rheumatoid arthritis. Glucocorticoids can be used in the short term and at the lowest dose possible for flare-ups and while waiting for slow-onset drugs to take effect. In this case methyl prednisole (250mg in 200ml NS), deflazacort (30mg) (steroids) treatment was started and the patient developed steroid induced diabetes mellitus (RBS: 283mg/dl). In all cases, we should also focus on nutrition and physiotherapy.

Conclusion

A unique complication of rheumatoid arthritis patient with GBS is discussed above and a case report is presented. Physicians (and therapists) should suspect rheumatoid arthritis in symptomatic (related to large joints) patients with GBS when risk factors exist. In this case report the patient was treated with hydroxycloquine for rheumatoid arthritis and the patient is responded to the given treatment. The patient wasn't responding to intravenous immunoglobulins and they also lead to severe adverse drug reactions like left ventricular dysfunction and acute interstitial nephritis, so the patient was referred to higher centres for advanced treatment of plasmapheresis to treat GBS.

Conflict of Interest: None.

References

1. Till G, Mior SA, McGregor M. A study of chiropractic patient population in Saskatoon. Proceedings of the World Federation of Chiropractic: Toronto 1991:28.
2. Shekelle PG, Brook RH. A community based study of the use of chiropractic services. *Am J Public Health* 1991;81:439-42.
3. Oswald IH, Coulter I. Chiropractors do they help? Don Mills, Fitzhenry and Whiteside Limited, 1990:135-138.
4. Kraft GH, Freal MS, Coryell JK. Disability, disease duration and rehabilitation service needs in multiple sclerosis: patient perspectives. *Arch Phys Med Rehab* 1986;67:164.
5. Hartung HP. Immune-mediated demyelination (comment). *Ann Neurol* 1993;33(6):583-7.
6. Miller RG. Guillain Barresyndrome. *Postgrad Med* 1985;77(7):57-64.
7. Mendell JR. Chronic inflammatory demyelinating polyradiculopathy. *Annu Rev Med* 1993;44:211-9.
8. Murray DP. Impaired mobility: Gullain-Barre syndrome. *J Neurosci Nurs* 1993;25(2):100-104.

9. Hund EF, Borel CO, Comblath DR, Hanley DF, McKhann GM. Intensive management and treatment of severe Guillain-Barre syndrome. *Crit Care Med* 1993;21(3):433-46.
10. Ormerod IE, Ceekerell OC. Guillain-Barre syndrome after herpes zoster infection, a report of 2 cases. *Eur Neurol* 1993;33(2):136-318.
11. Stambough JL, Quinlan JG, Swanson JD. Guillain-Barre syndrome following spinal fusion for adult scoliosis. *Spine* 1990; 15(1):45-46.
12. Ennis JH, Bednar DA. Lumbosacral fusion in a patient with recurring Guillain-Barre syndrome and acute brachial neuritis. *J Spine Disord* 1992;5(2):217-8.
13. Ginn D. Guillain-Barre syndrome. An uncommon but severe illness. *Postgrad Med* 1991;90(5):145-416, 153-6.
14. Merelli E, Sola P, LaSpina I, Orlando A, Milanti G. Guillain-Barre polyradiculoneuritis after blood transfusion. *Ital J Neuro Sci* 1991;12(3):313-5.
15. Nadkarni N, Lisak RP. Guillain-Barre syndrome (GBS) with bilateral optic neuritis and central white matter disease. *Neurol* 1993;43(4):842-3.
16. Macleod J. Davidson's Principles and Practice of Medicine. Eleventh Edition, New York: Churchill Livingstone 1975:65-68.
17. Jones HR Jr. Childhood Guillain-Barre syndrome: clinical presentation, diagnosis, and therapy. *J Child Neurol* 1996;11(1):4-12.
18. Sarnat HB. Guillain-Barre syndrome. In: Nelson Textbook of Pediatrics, 19th edn. 2011:2143-5.
19. Koul R, Chacko A, Ahmed R, Varghese T, Javad H, Al-Lamki Z. Ten-year prospective study (clinical spectrum) of childhood Guillain-Barre syndrome in the Arabian peninsula: comparison of outcome in patients in the pre- and postintravenous immunoglobulin eras. *J Child Neurol* 2003;18(11):767-71.
20. Alsheklee A, Hussain Z, Sultan B, Katirji B. Guillain-Barre syndrome: incidence and mortality rates in US hospitals. *Neurol* 2008;70(18):1608-13.
21. Lawn ND, Wijdicks EF. Fatal Guillain-Barre syndrome. *Neurol* 1999;52(3):635-8.
22. Newswanger DL, Warren CR. Guillain-Barre syndrome. *Am Fam Physician* 2004;69(10):2405-10.
23. Haslam RH. Chapter 567, Guillain-Barre syndrome. In: Nelson Textbook of Pediatrics, 15th edn, Behrman RE, Kliegman RM, Arvin AM (Eds.). Philadelphia, PA: WB Saunders, 1996. pp. 1761-2.
24. Evans OB, Vedanarayanan V. Guillain-Barre syndrome. *Pediatr Rev* 1997;18(1):10-6.

How to cite this article: Parveen SB, Gayatri B, Rani CS, Praveena B, Kumar KR, A rare case report on guillain barre syndrome and rheumatoid arthritis. *Int J Pharm Chem Anal* 2019;6(1):24-26.