Case Report

Guillain–Barre syndrome: a case report

Karan Joshi[1], Meena Patel[1], Richa Gupta[2], Naresh Bajaj[1]

[1]Department of Pediatrics, SS Medical College, Rewa, Madhya Pradesh, India.
[2]Department of ENT, SS Medical College, Rewa, Madhya Pradesh, India.
Correspondence to: Meena Patel, E-mail: meenu_grmc@yahoo.co.in

Received May 5, 2015. Accepted September 23, 2015

Abstract

Guillain–Barre syndrome (GBS) is an acute or subacute inflammatory demyelinating polyradiculoneuropathy. It is an acquired condition that is characterized by progressive, symmetrical, proximal, and distal tingling and weakness. We report a case of a 14-year-old male patient who presented with complaint of weakness in both upper and lower extremities, change in voice, and difficulty in swallowing with regurgitation of food, and later diagnosed as case of GBS.

KEY WORDS: Acute or subacute inflammatory demyelinating polyradiculoneuropathy, areflexia, Guillain–Barre syndrome, VAP

Introduction

Guillain–Barre syndrome (GBS) is an immune polyradiculoneuropathy that presents with ascending bilateral lower extremity weakness and areflexia and that affects all age groups with a slight male predisposition.[1]

Patient usually presents with ascending type of flaccid weakness. Hyporeflexia or areflexia is almost universal. Motor deficit occurs in 94% of cases, sensory paresthesia in 64%, and cranial nerve involvement in less than a third of patients. The CSF protein is elevated to more than twice of the upper limit of normal, glucose level is normal, and there is no pleocytosis.

The MRI findings include thickening of caudae equinae and intrathecal nerve roots with gadolinium enhancement.[2]

Case Report

We report a case of 14-year-old boy, who presented with complaints of unable to hold an object and stand, change in voice, and unable to swallow solid foods to PICU, Department of Pediatrics, SS Medical College, Rewa (Madhya Pradesh).

Access this article online	
Website: http://www.ijmsph.com	Quick Response Code:
DOI: 10.5455/ijmsph.2016.05052015157	

The history and CNS examination of the patient revealed absence of gag reflex, regurgitation of food, hoarseness of voice, which confirmed the palsies of 9th, 10th, and 11th nerve. The motor examination revealed grade 2 weakness in upper distal extremities and grade 3 weakness in lower limbs. Deep tendon reflexes were absent in both upper and lower limb. Babinski sign was absent. CT scan showed that the head was normal. MRI of the cervical spine showed thickening of caudae equinae.

CSF examination showed increased protein, and cell count was normal (albuminocytoplasmic dissociation). Electrolytes were normal, which ruled out possibility of hypokalemia. The CBC and urine examination were normal. Blood pressure was normal. Patient had a history of diarrhea 1 week before admission to hospital.

Patient was treated with supportive treatment initially with intravenous immunoglobulins for 5 days. After 4 days of admission, patient developed paradoxical breathing and was administered ventilatory support. X-ray of chest was done, which showed sign of aspiration pneumonia on the right side. Because of the need for prolonged ventilation, tracheostomy was done and Portex tracheostomy tube was inserted. But as the patient did not improve, second dose of intravenous immunoglobulin was administered. Patient also developed ventilator-associated pneumonia (VAP). Patient was on ventilator for 52 days. The patient later improved and thus, ventilator support was weaned off. Then he was shifted to CPAP. Two days later, CPAP was also removed. The oxygen saturation was well maintained. Chest physiotherapy was started for the improvement of lung function along with nutritional support in the form of diet chart as a regime of supportive care.

Discussion

GBS is an acute monophasic demyelinating neuropathy. The disease is characterized by progressive motor weakness of limbs with areflexia. Preceding antecedent infections, mostly viral, are seen in half of the cases. One-third of patients required ventilatory support in the past with about 10% mortality. Immunoglobulins and plasmapheresis have made a significant change in the course of the illness.[3] About 12%–20% of patients with GBS may require ventilatory support for respiratory paralysis.[4] In this case, patient required ventilator support. Within those ventilated patients, 20% may die owing to VAP, ARDS, sepsis.[5] In this case also, patient developed VAP.

Also two courses of intravenous immunoglobulins were administered, which helped in improving the patient. The risk of the flu vaccine inciting a case of GBS was one to two cases per 1,000,000 people vaccinated.[6] The past history may include vaccinations against influenza, meningitis, and tetanus toxoid, gastroenteritis, and upper respiratory infection. In this case, there was no history of influenza vaccine and upper respiratory tract infection. But the history of diarrhea 1 week before admission was positive.

Improvement in strength usually occurs in reverse order, with bulbar muscle strength returning first and lower extremity strength returning last. Deep tendon reflexes are often the last function to recover.[7] The similar series of events were observed in our case also. Bulbar and respiratory muscle involvement can lead to death if the syndrome is not recognized and not treated. Three clinical features are predictive of poor outcome with sequelae as cranial nerve involvement, intubation, and maximum disability at the time of presentation.[2] Patients with dysphagia, shoulder weakness, or cardiovascular instability may also require assisted ventilation.[8] The power of deltoid muscle was assessed for recovery in this case.

Conclusion

All the cases of GBS should be thoroughly investigated to formulate the further line of management. The power of deltoid muscle can be considered as a sign of recovery. The disorder should be diagnosed timely, to provide close follow-up and appropriate therapy and counseling. The clinician should not be afraid of administering prolonged ventilation as it can also provide favorable outcome as in our case. Repeated dose of intravenous immunoglobulin should be given to patient, if he or she is either deteriorating or not improving after first dose of intravenous immunoglobulin. In all cases, we should also focus on nutrition and physiotherapy. The parental counseling should be done in all such cases.

References

1. Jones HR Jr. Childhood Guillain–Barre syndrome: clinical presentation, diagnosis, and therapy. J Child Neurol. 1996;11(1): 4–12.
2. Sarnat HB. Guillain–Barre syndrome. In: *Nelson Textbook of Pediatrics*, 19th edn. 2011. pp. 2143–5.
3. 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.
4. Alshekhlee A, Hussain Z, Sultan B, Katirji B. Guillain–Barre syndrome: incidence and mortality rates in US hospitals. Neurology. 2008;70(18):1608–13.
5. Lawn ND, Wijdicks EF. Fatal Guillain–Barre syndrome. Neurology. 1999;52(3):635–8.
6. Newswanger DL, Warren CR. Guillain–Barre syndrome. Am Fam Physician. 2004;69(10):2405–10.
7. 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.
8. Evans OB, Vedanarayanan V. Guillain–Barre syndrome. Pediatr Rev. 1997;18(1):10–6.

How to cite this article: Joshi K, Patel M, Gupta R, Bajaj N. Guillain–Barre syndrome: a case report. Int J Med Sci Public Health 2016;5:1295-1296

Source of Support: Nil, **Conflict of Interest:** None declared.