Case Report

A Rare Case Report of Guillain–Barré Syndrome Presenting as Unilateral Facial Palsy with Isolated acute Bulbar Palsy

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Guillain–Barré syndrome (GBS) is an immune-mediated acute inflammatory polyradiculoneuropathy and it is the most common cause of acute flaccid paralysis worldwide. There are some rare variants of GBS, which may be easily missed unless suspected. Here we present a case of GBS presenting as isolated acute bulbar palsy. A 10-month-old infant, known case of tricuspid atresia with pulmonary stenosis, presented with left-sided lower motor neuron type of facial palsy and palsy of bilateral glossopharyngeal and vagus nerve of 2 weeks’ duration. On detailed neurological examination, motor and sensory system were normal. Nerve conduction study showed demyelinating motor neuropathy and hence the diagnosis of GBS was made. To the best of our knowledge, no case of isolated bulbar palsy due to GBS in infancy has been reported.

Keywords: Case report, demyelinating motor neuropathy, isolated bulbar palsy, nerve conduction study

Introduction

Guillain–Barré syndrome (GBS) is the most common cause of childhood acute flaccid paralysis throughout the world. The term includes a variety of clinical syndromes with a common pathophysiological basis, which is thought to be autoimmune, acute inflammatory demyelinating polyneuropathy being the most common variant. There are several other localized subtypes of GBS. Although it was recognized centuries ago, there are many unanswered questions about GBS and its florid presentation. We describe a case of GBS presenting as acute bulbar palsy with unilateral facial nerve palsy, which is a rare presentation both in adults and children. Treating physician must be aware of these atypical presentations of GBS for early diagnosis so that intervention can be planned in the form of plasmapheresis or intravenous (IV) immunoglobulin in progressive form of the disease.

Case Report

A 10-month-old male child, known case of tricuspid atresia, presented to our outpatient department (OPD) with complaints of facial deviation, dysphagia, nasal regurgitation of food, and nasal intonation of voice for the last 15 days. Symptoms were acute in onset, progressed initially for 5–6 days, followed by a static course. He did not have weakness of any limb. He was admitted in another hospital in view of pneumonia since last 20 days and he developed these complaints in the hospital duration only and was referred to our hospital for further workup. There was no history of any intramuscular injection, insect bite, head injury, or ear discharge. His immunization status was complete and has received three doses of diptheria pertusis tetanus and oral polio vaccine.

On examination, the child was fully conscious, alert, and responding. Physical examination showed facial deviation to the left along with drooling of saliva. Respiratory rate was 34 breaths/min, heart rate was 110 beats/min, SpO2 was 76%, and blood pressure was

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How to cite this article: Yadav P, Dhaka S, Chaudhary R, Damke S, Lohiya S. A rare case report of Guillain–Barré syndrome presenting as unilateral facial palsy with isolated acute bulbar palsy. J Pediatr Neurosci 2020;15:157-9.
90/60 mm Hg. On central nervous system examination, left-sided lower motor neuron type of facial nerve palsy was noted along with 9th and 10th cranial nerve (CN) palsy. Rest all CNs were normal. Limb muscle’s power was normal, deep tendon reflexes were 1+, and plantar response was down going bilaterally. Sensory system examination was within normal limits. Fundus examination was normal. Nerve conduction study was carried out, which showed reduced amplitude and decreased conduction velocity in bilateral peroneal motor nerves and left tibial nerve. Study also showed reduced amplitude in bilateral sural nerves along with reduced amplitude but normal onset latency and conduction velocity in right tibial nerve. Routine blood investigations including complete hemogram were within normal limits. Workup for poliomyelitis was negative. Throat and nasal swab were sent, which showed no growth. As regurgitation of feeds was present and child vomited every meal, he was started on ryels tube (RT) feeding. Relatives were taught how to give RT feeding and once they were confident child was discharged on RT feeding after 1 month of hospital stay. Patient regularly visited OPD for changing feeding tube and detailed neurological examination at each visit [Figure 1]. There was no progression of the disease and slowly child improved with first recovery of facial nerve and complete recovery after 4 months of follow-up. Now patient is taking meals normally. Serological tests for antibodies were not carried out due to financial constraints. The diagnosis was kept as acute bulbar palsy plus syndrome—a localized variety of GBS on the basis of clinical course and nerve conduction study and negative workup for poliomyelitis.

**Discussion**

GBS may rarely present as bulbar palsy in isolation or as a part of pharyngeal–cervical–brachial (PCB) variant (with mild proximal upper limb involvement). Only 5% of the patients with GBS present with multiple cranial neuropathies.[1] In such cases, GBS is not kept in the differential because of noninvolvement of limbs and it may be missed. To the best of our knowledge, no case with pure bulbar palsy with unilateral facial palsy as a presentation of GBS in infancy has not been reported earlier. Similar cases have been reported from Malaysia[2] in a 19-year-old girl and from New Delhi[3] in a 13-year-old girl. Both of them had spontaneous improvement without any intervention. Unilateral facial palsy is extremely rare in GBS and only few cases have been reported. Verma *et al.*[4] mentioned a case report of adult male who presented with unilateral facial palsy. Kim *et al.*[5] studied 11 adult cases of acute bulbar palsy plus syndrome and concluded it as a variant of GBS. O’Leary *et al.*[6] described cases of acute oropharyngeal palsy as a regional variant of GBS. Ondera *et al.*[7] reported the case of a patient with acute bulbar palsy as an isolated neurological symptom, whose serum had an elevated level of antiganglioside antibodies immunoglobulin (anti anti-GT1a IgG) antibody. Similarly, Hamidon[2] reported a case of GBS presenting with isolated bulbar palsy. Our patient also presented with isolated acute bulbar palsy with areflexia and nerve conduction velocity (NCV) was suggestive of GBS. Nerve conduction study provides strong supportive evidence in the atypical cases of GBS. After a period of 5–6 days from the onset of disease, abnormality in the conduction studies in the form of demyelinating neuropathy or axonal variety is almost universally detected[8,9] and hence NCV should be performed in all cases of suspected GBS especially the atypical ones.

**Conclusion**

This case highlights the fact that GBS should be considered in cases of isolated multiple cranial palsies for early intervention and close monitoring should be carried out for progression or complications. IV

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**Figure 1:** Photograph of the patient after recovery from facial palsy but still on RT feeding as palatal palsy was not recovered fully.
immunoglobulin should be considered in case of progressive disease.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their 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.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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