Case Report

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Gullian Barre Syndrome Following Excision of CP Angle Epidermoid. Importance of Early Diganosis

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Introduction

GB syndrome is an important cause of acute neuromuscular paralysis. Surgery remains one of the rare causes of the GB syndrome. Onset of symptoms of GB syndrome following neurosurgical procedures especially after spine and cranial surgery would be a diagnostic dilemma. Occurrence of such features of GB syndrome after the excision of the intracranial epidermoid would lead the surgeon to consider aseptic meningitis as possibility resulting in delay in diagnosis and treatment. GB syndrome with atypical features add further to the problems of diagnosis. The authors have described a case of Atypical GB syndrome following excision of the cerebellopontine angle epidermoid and its clinical course.

Case Report

A young female aged 28 years presented to us with episodes of on and off shock like sensation over the Rt half of the face in the trigeminal distribution for 1year. MRI brain has shown mass lesion in the Rt CP angle cistern extending across the brainstem to the opposite side with rotation of the brainstem, appearing hypointense on T1, Hyperintense on T2 and restriction on diffusion and irregular suppression on FLAIR sequences. A diagnosis of CP angle epidermoid was made. Patient was admitted for surgery and tested positive for pregnancy on urine bHcg testing. Patient opted for surgery after having been counseled regarding the risks involved in first trimester surgery and the option of postponing surgery to the second trimester in view of the benign nature of the tumor. MSOC and excision of the epidermoid was done. Rotation of the brainstem facilitated complete excision of the tumor. Until postoperative day 2 patient was conscious oriented with no cranial nerve, motor or sensory deficits. Dexamethasone was given in the postoperative period to prevent aseptic meningitis. On postoperative day 3 patient had irrelevant speech with altered sensorium along with ptosis on contralateral side of surgery. NCCT head showed no abnormality. Diffusion weighed MRI performed to look for any ischemic changes revealed no abnormality. No residual tumor could be appreciated. Chemical meningitis was suspected and patient was started on intravenous methyl prednisolone. By postoperative day 4 patient developed bilateral ptosis and became more drowsy. She started complaining of bilateral hearing loss and was only following written commands. BERA performed showed no responses on either side. CSF obtained by lumbar puncture showed high protein content (210mg/dl) with normal sugar and cells. CSF culture grew no organism. The CSF analysis was as follows: RBC-800, WBC-10, Sugar-61/98, Protein-270 and the culture showed no growth). By postoperative day 5 patient developed bilateral facial and lower cranial nerve palsy with further deterioration in sensorium. Neurologist opinion was sought and MRI brain study plain and contrast has shown no abnormality. Patient developed Rt hemiparesis on day 6 with further deterioration in the sensorium. EMG and NCV done showed no abnormality. By day 7 patient developed shallow breathing with poor respiratory efforts and pooling of secretions requiring endotracheal intubation. Subsequently patient required tracheostomy and ventilator support. Her GCS deteriorated steadily over the next 10 days and was E1VTM2 on 16th postoperative day. EMG, NCV studies repeated showeddecreased conduction velocities. Abnormality. GM 1 ganglioside antibody analysis was positive. I.V immunoglobulin theraphy was planned. Patient developed cardiac arrest probably due to cardiac arrhythmias and could not be revived. GM1 antibody was positive.

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Discussion

Guillain-Barré syndrome (GBS) is one of the acute flaccid paralysis syndromes in humans. First described in 1916 in two soldiers by French neurologists Georges Guillain, Jean-Alexandre Barré and Andre Strohl, a distinguishing feature from the then most prevalent cause of acute flaccid paralysis, poliomyelitis, was the finding of elevated cerebrospinal fluid (CSF) protein with a normal cell count, the now classic albumin-cytologic dissociation [1]. Since the original description, different subtypes producing the clinical picture of GBS have been described including acute inflammatorydemye-linatingpoly radiculo-neuropathy (AIDP), [2] acute motor axonal neuropathy (AMAN), [3, 4] acute motor and sensory neuropathy (AMSAN) [5] acute sensory neuronopathy, acute pandysautonomia and the Fisher syndrome [6]. About two-thirds of GBS cases have an antecedent infection 6 weeks prior to symptom onset, generally an upper respiratory tract infection or gastroenteritis [7]. GBS has also been reported following surgery and head trauma [8–18].The mechanisms that link GBS with surgery or trauma remain unclear. Reported after cervical spine and thoracic spine injury [19, 20]. Specifically, head trauma imparted by injury or surgery may be associated with depressed cell-mediated immunity [21] and production of antimyelin antibodies [22].

Furthermore, major stress of head trauma or surgery may result in activation of latent processes that would in turn affect the immunological system, [23, 24] as has also been documented following spinal cord injury [25]. Surprising in this regard is that GBS has not been linked to peripheral nerve injury in which one might postulate that exposure of peripheral nerve to the circulation would allow for the creation of auto-antibodies against nerve tissue and thus stimulate GBS. The occurrence of GB syndrome after surgery has well been reported after the cardiac and the facial surgeries but the diagnosis and the management is more complicated in cases occurring following intracranial surgery. GB syndrome following cranial surgery procedures is rare [9, 10, 16, 18, 26]. It has also been reported with Spinal surgery [19, 20, 27, 29, 30]. As is the problem in our case the presentation is so atypical that the diagnosis is delayed. The initial presentation resembled a surgical complication or aseptic chemical meningitis especially in case of epidermoid resulting in delay of diagnosis. Furthur confusion to the diagnosis was added by the atypical presentation like diminished sensorium and initial onset with cranial nerve palsies and also the recording of the delayed nerve conduction velocities. The present case does not have a preceding respiratory or GI tract infection as is seen in one third of the cases of GB syndrome. Cardio-respiratory arrest in GB syndrome as has occurred in this patient is rare but reported in literature in postsurgical GB syndrome cases [12].

Conclusions

GB syndrome following cranial surgery is rare. Atypical GB syndrome following the intracranial surgery can be confused with the complications of surgery. High index of suspicion should be maintained in uncomplicated postsurgical cases with unexplained neurological deteriorations for early diangnosis and treatment of GB syndrome.

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