Guillain-Barre Syndrome following Intracranial Surgery for Traumatic Intracerebral Hemorrhage

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Abstract

Background: A case of Guillain-Barre Syndrome (GBS) with respiratory failure following craniotomy for post traumatic intracerebral bleed is described. The association of Guillain-Barre syndrome with head injury is not well recognized and a possible immunological association may be existing. The objective of our study is to describe clinical and Neurophysiological findings of GBS after trauma and Surgery.

Case Presentation: On 24th August 2022, a 45 years old Bangladeshi male brought by his family to Emergency Department of Evercare Hospital Chattogram. During evaluation, family added that he sustained a head injury on 4th August 2022 after bike accident and was admitted to private hospital that day, where Computed Tomographic (CT) brain scan revealed right fronto-parietal contusions and a right subdural hematoma. He was underwent Craniotomy on the same day, followed by uneventful ICU stay, improved rapidly in cabin and was discharged on 17th August 2022. On 19th August 2022, he again become drowsy and rapidly progressive weakness of all 4 limbs. On 24th August he was intubated in hospital emergency for respiratory failure. A repeat CT scan showed no interval changes, Cerebrospinal fluid (CSF) and neurophysiology studies were suggestive of GBS. Patient was extubated after Intravenous Immunoglobulin therapy (IVIG).

Conclusion: GBS may be considered in patients with progressive muscle weakness after surgery and which improve with timely treatment with IVIG.

Key word: Craniotomy; CSF; GBS; Head injury; IVIG; Neurophysiology.

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Introduction

The Guillain-Barre syndrome (GBS) is an acute inflammatory polyneuropathy which follows a precipitating event in approximately two thirds of cases.[1] GBS has been described most frequently after non-specific viral infections, but it may also follow immunization, specific viral infections and various types of trauma, including intracranial and general surgical procedures, orthopedic operations and spinal anaesthesia.[2,3] GBS following intracranial surgery due to head trauma however, is not a well-recognized event in Bangladesh. We describe here a case of severe GBS developing after craniotomy following acute head injury.

Case Presentation

On 4th August 2022, a 45 years old Bangladeshi male sustained a head injury after bike accident. He was admitted to private hospital that day, where he was noted to be drowsy and confused but not in coma with GCS 10. There was grade 4 left sided focal limb weakness, and all limb reflexes were brisk. Plain skull X-rays showed a depressed right frontal fracture.

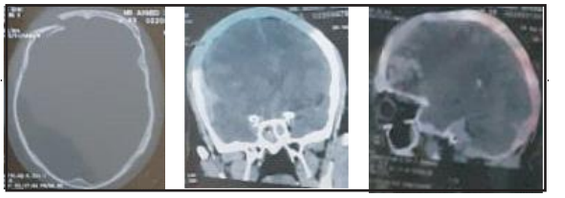

Figure 1 Pre operative Computed tomographic (CT) brain scan revealed right fronto-parietal contusions and a right subdural hematoma

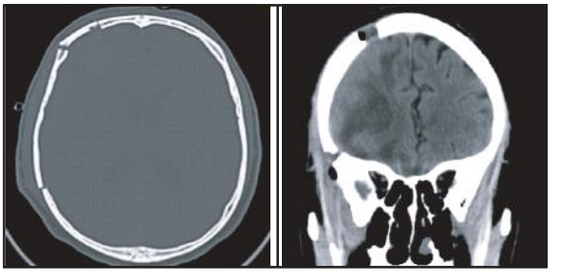

Figure 2 Post Operative CT brain showing Status post right frontal craniotomy, resolution of right subacute frontal hematoma and Early encephalomalacic changes of right temporal & left frontal lobe

He was underwent craniotomy on the same day, followed by uneventful ICU stay, improved rapidly in cabin and was discharged on 17th August, able to walk and talk normally. 2 days later he again become drowsy, developed cramps in both legs and rapidly progressive weakness of all 4 limbs, as well as inability to swallow or breath. There had been no recent viral illness. He was attended emergency department of Evercare Hospital where he suffered type II respiratory failure for which he was resuscitated, and was then readmitted to ICU. On examination he was apyrexial, with a tachycardia of 134/minute, blood pressure of 160/100mmHg and a respiratory rate off the ventilator of 45/minute. Air entry to the chest was generally reduced. He was fully alert and oriented. The fundi were normal. There was bilateral lower motor neuron facial palsies. There was severe flaccid symmetrical weakness of all 4 limbs, more severe proximally. All tendon reflexes were absent and the plantar responses were absent. The sensation in all 4 limbs were intact. Investigations revealed normal routine hematology and biochemistry. ABG was showing PH 7.05, PCO2 78.2, SPO2 75% before intubation. Serum lead & Urinary porphyrins were not done. A repeat CT scan showed Status post right frontal craniotomy and late subacute right frontal hematoma. Electrodiagnostic studies revealed Mixed Sensory Motor Symmetrical Demyelinating Secondary Axonal polyradiculo-neuropathy suggestive of a diagnosis of GBS (Table I-V).

Cerebrospinal Fluid (CSF) examination showed glucose 8.4 mmol/L, Protein 145 mg/dL, WBC- 2 (100% Lymphocyte). During the first 5 days of his admission his respiratory function worsened, due to a right lobar pneumonia and his limb weakness and bulbar function deteriorated further and over the week, he had five days Intravenous Immunoglobulin (IVIG) course. His condition then began to improve rapidly hereafter from second dose. Respiratory and bulbar function as well as his cranial nerve palsies recovered completely within a few days. Improvement in limb power occurred very slowly but continued to show recovery when he was transferred to rehabilitation unit.

Require permission taken from authorities for this study.

Table I Motor Nerve Conduction Study showing increased distal latency of both median, Ulnar nerve and Right Tibial. No response (NR) of both Tibial and Left Peroneal nerve

Nerve / Sites	Muscle	Latency ms	Amplitude mV	Dist. mm	Lat Diff ms	Velocity m/s
L Median - APB						
Wrist	APB	7.10	2.2	80		
Elbow	APB	12.33	2.1	240	5.23	45.9
R Median - APB						
Wrist	APB	7.98	2.3	80		
Elbow	APB	12.40	2.4	240	4.42	54.3
L Ulnar - ADM						
Wrist	ADM	5.27	1.7	80		
B.Elbow	ADM	10.60	1.8	250	5.33	46.9
A.Elbow	ADM	12.50	1.7	100	1.90	52.7
Axilla	ADM	15.02	1.1	90	2.52	35.7
R Ulnar - ADM						
Wrist	ADM	4.44	1.3	80		
B.Elbow	ADM	9.52	1.3	250	5.08	49.2
A.Elbow	ADM	12.08	1.0	100	2.56	39.0
Axilla	ADM	14.04	1.0	90	1.96	46.0
R Peroneal - EDB						
Ankle	EDB	NR	NR	80		
B. Fib Head	EDB	NR	NR	320	NR	NR
A. Fib Head	EDB	NR	NR	100	NR	NR
L Peroneal - EDB						
Ankle	EDB	NR	NR	80		
B. Fib Head	EDB	NR	NR	320	NR	NR
A. Fib Head	EDB	NR	NR	100	NR	NR
R Tibial - AH						
Ankle	AH	9.10	1.3	80		
Knee	AH	17.90	0.8	350	8.79	39.8
L Tibial - AH						
Ankle	AH	NR	NR	80		
Knee	AH	NR	NR	350	NR	NR
L Median, Ulnar - Lumbrical-Interossei						
Median Wrist	Lumb II	NR	NR	100		
Ulnar Wrist	Interos	NR	NR	100		
					NR	
R Median, Ulnar - Lumbrical-Interossei						
Median Wrist	Lumb II				100	
Ulnar Wrist	Interos	NR	NR		100	
					NR	
R Peroneal - Tib Ant						
Fib Head	Tib Ant	6.96	0.7			
Pop fossa	Tib Ant	8.75	2.0	100	1.79	55.8
L Peroneal - Tib Ant						
Fib Head	Tib Ant					
Pop fossa	Tib Ant	NR	NR	100	NR	NR
L Radial - EIP						
Forearm	EIP	3.85	2.1			
Elbow	EIP	7.50	1.6	100	3.65	27.4
R Radial - EIP						
Forearm	EIP	10.31	0.5			
Elbow	EIP	8.69	0.5	100	-1.62	61.5

Table II Sympathetic Skin Response (SSR) shows No Response (NR)

Nerve / Sites	Latency s	P-P Amp mV	Stim Type
L Sympathetic - Palm Palm	NR	NR	

Table III Sensory nerve conduction study shows no response of both sural nerve

Nerve / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	NP Amp µV	PP Amp µV	Distance mm	Velocity m/s
L Median - Dig II (Antidromic) Wrist	Index	3.07	3.44	2.4	1.8	140	46
R Sural - (Antidromic) Calf	Ankle	NR	NR	NR	NR	140	NR
L Sural - (Antidromic) Calf	Ankle	NR	NR	NR	NR	140	NR

Table IV F waves show no response in studied motor nerve

Nerve	F Latency ms	M Latency ms	F - M Lat ms
L Median - APB	NR	NR	NR
L Ulnar - ADM	NR	NR	NR
R Median - APB	NR	NR	NR
R Ulnar - ADM	NR	NR	NR
R Tibial - AH	NR	NR	NR
R Peroneal - EDB	NR	NR	NR
L Tibial - AH	NR	NR	NR
L Peroneal - EDB	NR	NR	NR

Table V H Reflex shows no response in both side

Nerve	H Latency ms
R Tibial - Soleus	NR
L Tibial - Soleus	NR

Discussion

This patient showed the typical features of GBS with the rapid development of symmetrical flaccid limb paralysis, bilateral lower motor neuron facial weakness, respiratory and bulbar involvement, areflexia with normal sensory. The diagnosis was supported by the electrophysiological and CSF findings of a severe demyelinating neuropathy. Now the question may arise here why a patient with head injury and craniotomy has developed GBS within 2-3 weeks and what could be the precipitating factors Or really is there any relation between this two or something else? IVIG has been shown to be effective in the treatment of GBS.[3] The theoretical basis of the treatment consists of the finding of demyelinating factors in the plasma of GBS patients and study shows Myelin Basic Protein (MBP) can induce experimental allergic encephalomyelitis in a variety of animal species.[4-6] This protein is raised in head injury and or post neurosurgical cases.[7] So it is suggestive that anti myelin antibody induced against traumatized nervous tissue causes demyelinating neuropathy. But still question remain, why it is not that common feature following all head injury or brain surgery. May be immunogenic factor originates in the central nervous system due to surgery and trauma, is usually only weakly immunopathogenic for peripheral nervous tissue.

Limitation

Lack of laboratory evidence which is supporting immunogenic factor like Myelin Basic Protein (MBP) were not done. Patient is under our supervision & Follow Up to observe clinical course.

Conclusion

GBS can occur after intracranial trauma or surgery. So, GBS may be considered in suspected patient any patients with rapidly progressive muscle weakness after surgery. Its diagnosis can be missed since the symptoms may be incognito by the presentations from the primary illness. Therefore, clinicians should rule out disease when patients develop new neurological symptoms or no improvement after the appropriate treatments for the primary disorder.

Recommendation

Bedside Nerve Conduction Study (NCS) can be crucial as CSF study should be done once raised CSF pressure is excluded. IVIG and plasma exchange are the available treatments for GBS and should be initiated once the diagnosis of GBS is confirmed.

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Contribution of authors

NU- Conception, citing references. drafting & final approval.
FA- Design, critical revision & final approval.
SD- Citing references, drafting & final approval.

Disclosure

All the authors declared no competing interest.

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