A Rare Case Report of Guillian Barre Syndrome Presenting with Unilateal Facial Nerve Palsy

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Abstract- Gullian Barre Syndrome is an acute diffuse post infectious demyelinating disorder of spinal roots and peripheral nerves and occasionally cranial nerves. Bilateral facial nerve palsy is the most common pattern of cranial nerve involvement in GBS. However, unilateral facial palsy, although uncommon, can be seen in GBS. We report a rare case of unilateral facial palsy in a patient with Gullian Barre Syndrome.

Index Terms- Gullian Barre Syndrome, facial nerve palsy, Acute Inflammatory Demyelinating Polyneuropathy.

I. Introduction

The Guillian - Barre Syndrome is one of the commonest forms of polyneuropathy. The reported incidence rates for GBS are 1-2 per 1 00,000 population. The lifetime likely hood of any individual acquiring GBS is 1 in 1000. Available Indian literature indicates a peak incidence between June, July and September - October. In the Western Countries GBS is common in the 5th decade, but in India it occurs more commonly in younger age. GBS is equally common in men and women and can occur at any age. There is a male preponderance among the hospitalized population.[1]

Gullian Barre Syndrome also known as an Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is an acute demyelinating polyradiculopathy of uncertain aetiology which may present with facial nerve involvement in 27-50% of cases, often bilaterally[2].

Over half of Gullian Barre syndrome patients experience symptoms of viral respiratory or gastrointestinal infections during the 1-3 weeks prior to the onset of neurological symptoms. Clinical criteria, spinal fluid protein elevation, and nerve conduction abnormalities are the mainstay of diagnosis[3].

II. Case Presentation

A 70 year old male patient presented with weakness of both upper limbs and lower limbs, inability to close left eye, history of pins and needle sensation on both hands and feet of 1 week duration.

Past History: No History of trauma, Hypertension, Diabetes, CAD, Dog bite, recent vaccination and no previous significant neurological problems.

Personal History: Takes mixed diet, smoker, occasional alcoholic.

General Examination: About 70 year old Lean male with pulse rate 80/mt regular, temperature normal, no neck stiffness, Respiratory Rate 18/mt, BP – 130/80 mm Hg.

Neurological Examination: Intellectual functions normal, slurring of speech present, Bells phenomenon on left eye, deviation of angle of mouth to right while talking suggestive of left lower motor nerve type facial palsy, all other cranial nerves are normal.

Motor system: Bulk – Normal; Hypotonia of all 4 limbs, with Power 2/5 in all four limbs.

Superficial reflexes: Corneal, conjunctival, abdominals are present, plantars flexors through out the course of illness.

Deep tendon reflexes are absent in all four limbs, sensory system examination is normal, fundus is normal; No bladder and bowel involvement.

No Cerebellar signs, skull & spine are normal. No signs of meningeal irritation

Patient was treated with high dose IV steroids for 5 days with which power in all four limbs improved and power in all limbs at discharge is 4/5. Patient is advised regarding protection of eye during sleep and massage of the weakened muscles. Patient is on regular follow up. Patient is able to walk without support but unable to close the left eye is persisting.

Investigations:

Complete Urine Examination : Albumin :nil, Sugar: nil, Bile salts, Bile Pigments :nil, 1to 2 epithelial cells/hpf.

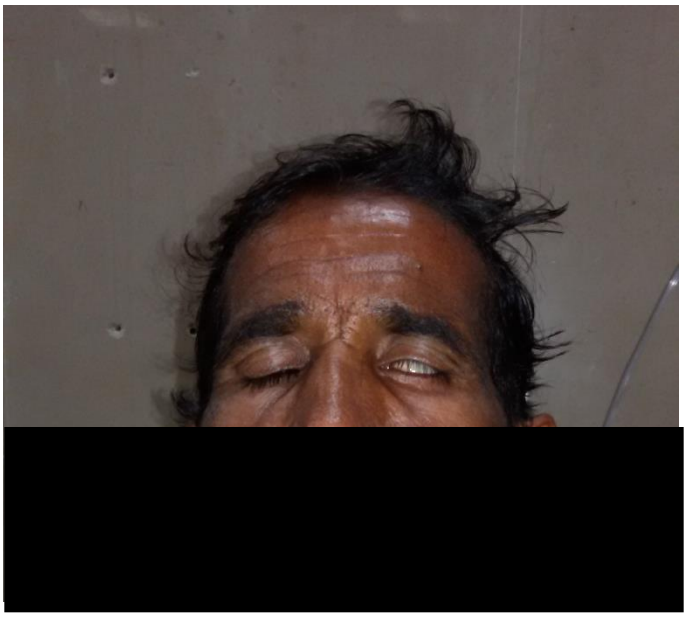
Complete Blood Picture: Hb:13.5, Wbc:7200, Neutrophils : 68, Lymphocytes:28, Monocytes:2, Eosinophils: 02, Platelets: adequate, ESR:05, RBS:90, Serum urea:20, Serum creatinine:1, Serum electrolytes: Sodium:138, Potassium:3.6

HIV and HBsAG negative

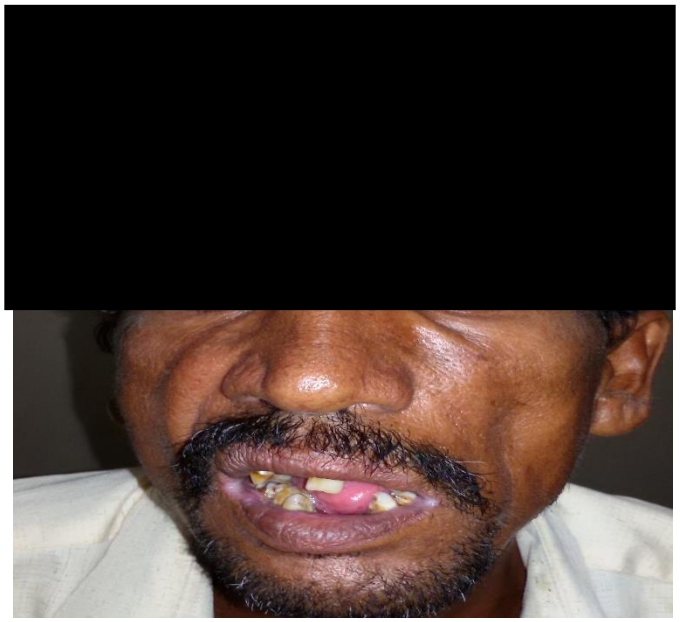
CSF analysis : Proteins – 51 mg/dL ; Sugar - 80 mg/dL ; ADA – 06 U/L ; No cells seen; culture – negative

MRI BRAIN – Normal Study.

Nerve Conduction Studies: Sensory Motor Axonal Demyelinating neuropathy with Radicular involvement

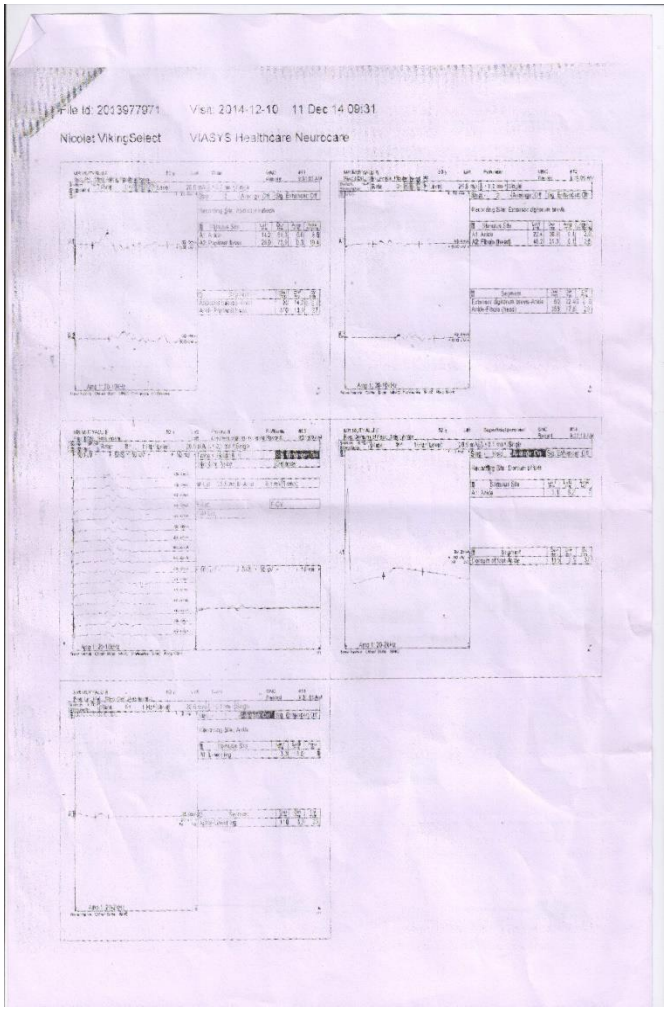


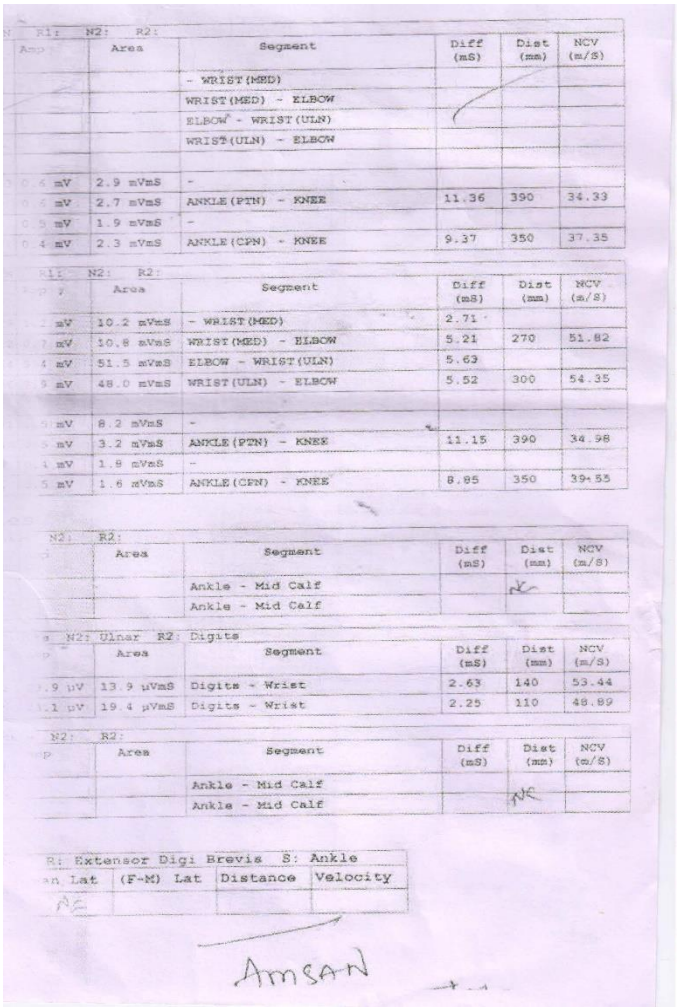
Bells Phenomenon of left eye



Deviation of Angle of Mouth to Right & loss of Nasolabial fold on left side

Electro physiological Studies (Nerve Conduction Report)





	Area	Segment	Diff (mS)	Dist (mm)	NCV (m/S)
		- WRIST (MED)			
		WRIST (MED) - ELBOW			
		ELBOW - WRIST (ULN)			
		WRIST (ULN) - ELBOW			
mV	2.9 mVmS	-			
mV	2.7 mVmS	ANKLE (PTN) - KNEE	11.36	390	34.33
mV	1.9 mVmS	-			
mV	2.3 mVmS	ANKLE (CPN) - KNEE	9.37	350	37.35

	Area	Segment	Diff (mS)	Dist (mm)	NCV (m/S)
mV	10.2 mVmS	- WRIST (MED)	2.71		
mV	10.8 mVmS	WRIST (MED) - ELBOW	5.21	270	51.82
mV	51.5 mVmS	ELBOW - WRIST (ULN)	5.63		
mV	48.0 mVmS	WRIST (ULN) - ELBOW	5.52	300	54.35
mV	8.2 mVmS	-			
mV	3.2 mVmS	ANKLE (PTN) - KNEE	11.15	390	34.98
mV	1.9 mVmS	-			
mV	1.6 mVmS	ANKLE (CPN) - KNEE	8.85	350	39.55

	Area	Segment	Diff (mS)	Dist (mm)	NCV (m/S)
		Ankle - Mid Calf			
		Ankle - Mid Calf			

	Area	Segment	Diff (mS)	Dist (mm)	NCV (m/S)
µV	13.9 µVmS	Digits - Wrist	2.63	140	53.44
µV	19.4 µVmS	Digits - Wrist	2.25	110	48.89

	Area	Segment	Diff (mS)	Dist (mm)	NCV (m/S)
		Ankle - Mid Calf			
		Ankle - Mid Calf			

Lat	(F-M) Lat	Distance	Velocity

Amsan

III. Discussion

The Guillian Barre Syndrome produces a relatively symmetrical areflexic tetraparesis. In three quarters of patients, the first neurological symptom is of paraesthesiae in the toes, less often in the finger3.

Muscle weakness usually starts in the legs and ascends to the arms. Proximal muscle weakness may be prominent from the onset. The weakness is fairly symmetrical and usually involves the trunk musculature. Maximal weakness generally develops within 12-14 days of the onset of neurological symptoms. Although cessation of symptom progression within 4 weeks is often regarded as a necessary criterion for the diagnosis of Guillain – Barre Syndrome (Asbury and Cornblath 1990).

Tendon reflexes are usually lost early in the disease. Total areflexia occurs in over 80 per cent of patients at some stage of the illness. Approximately half the patients develop cranial – nerve palsies, usually in the wake of severe ascending limb weakness ***(Loffel Rossi, Mumenthaler, et al 1977 ; Winer, Hughes, and Osmond 1988)***. Isolated unilateral or bilateral facial palsy is the commonest cranial – nerve lesion in Guillain – Barre syndrome.

Bulbar palsy and weakness of the muscles of mastication are the next commonest cranial nerve abnormalities. Ocular palsy only occurs in about 10 per cent of patients[3].

Laboratory Findings: The most important laboratory aids are the electro diagnostic studies and the CSF examination. The CSF is under normal pressure and is a cellular or contains only a few lymphocyte in all. In few patients (10 percent of less), the CSF protein values are normal throughout the illness.

Nerve conduction studies are a dependable and early diagnostic indicator of GBS, and in instances with a typical clinical and EMG presentation, one can dispense with the CSF analysis. The most frequent early findings are a reduction in the amplitudes of muscle action potentials, slowed conduction velocity, or conduction block in motor nerves. Prolonged distal latencies (reflecting distal conduction block) and prolonged or absent F response (indicating affection of proximal parts of nerves) are other important diagnostic findings, all reflecting demyelination[4].

Most patients with the Guillian Barre Syndrome will make a good spontaneous recovery if they receive competent supportive treatment.

GBS treatment should be initiated as soon after diagnosis as possible. Either high-dose intravenous immune globulin (IVIg) or plasmapheresis can be initiated. IVIg is administered as five daily infusions for a total dose of 2 g/kg body weight[5].

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