A Case of Guillain Barre Syndrome Complicated By p-ANCA Positivity, Elevated CSF Sugar and Protein with Decreased 25 Hydroxy (OH) Vitamin D And B12 Levels

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Abstract: Guillain–Barré syndrome (GBS) is an acute onset, usually monophasic immune-mediated disorder of the peripheral nervous system. Etiology remains unclear but pathophysiology includes demyelination of spinal nerve roots. A patient with Guillain-Barré syndrome complicated by p-ANCA positivity is reported, who responded favourably to the Ayurvedic modalities with adequate vitamin supplements with no clinical or biochemical side effects. It helps to treat the immune attack on the nerve cells of the peripheral nervous system and their support structures leads to activation of a group of blood proteins in GBS with remyelination of nerve cells.

Keywords : Guillain-Barré syndrome; Ayurvedic modalities; demyelination; p-ANCA

I. Introduction

Guillain-Barré syndrome is a heterogeneous syndrome which may be due to a demyelinating or axonal neuropathy probably autoimmune in nature, that is clinically characterised by acute progressive and symmetric motor weakness of the limbs and of bulbar and facial musculature.^{1,2} The overall incidence of Guillain Barre syndrome (GBS) is found to be 1.1/100,00/year to 1.8/100,000/year. The incidence of GBS increases with age after 50 years from 1.7/100,00/year to 3.3/100,000/year.³ We report on a patient with Guillain Barre Syndrome complicated by p-ANCA positivity responded well with Ayurvedic treatment with adequate vitamin supplements for a review.

II. Case Report

A 23 years old male presented with the history of calf pain. He was apparently normal earlier. This was followed by weakness of both lower and upper limbs. He has difficulty in walking and getting up from the squatting position. Difficulty in holding objects in the upper limb (Left>right). Two weeks after the onset of the symptoms patient was admitted to the hospital. Neurological examinations showed that moderate weakness in proximal muscles of the hip,flexors of the neck with the weak left grip. The dorsi flexors of the feet slightly weak giving rise to mild foot drop. No cranial nerves involvement was observed.

NCS showed absent left tibial H – response, impersistent left tibial F – waves. EMG examinations showed reduced motor conduction velocity in all limbs, especially in the lower limbs (median nerve 38 m/s; ulnar nerve 36 m/s; right exterior sciatic popliteal (ESP) 32 m/s, left ESP 34m/s, posterior tibial nerve 32 m/s). The Spinal MRI showed L4 – L5 mild posterior disc bulge indenting anterior thecal sac with scalloping of lumbar end plate. Chest radiography and abdominal ultrasonography were performed to exclude neoplasms and showed no abnormalities.

Analysis of CSF showed an abnormal total protein concentration (73 mg/dl), a high level of sugar (167 mg/dl), and a cell count of 12 cells/ mm3. The deficiency of 25 Hydroxy (OH) vit D (9.2 ng/ml) and Vitamin B 12 (190 pg/ml) was noted in the blood serum analysis.Further it was noted that p-ANCA positivity (1:10) in the serum with c-ANCA negative. The Anti Nuclear Antibody by IFA was negative.The fasting plasma glucose(141mg/dl) elevation was observed with HbA1c (6.1%).The Serum Protein Electrophoresis investigation were with in normal limits with' M' band Absent.The other serological parameters such as CPK (124 U/L), CRP(03mg/L), Blood urea (17 mg/dl), Creatinine (1.0 mg/dl) HBsAg (Negative),HIV I & II Ab (Negative), Anti HCV (Negative) were normal. Decrease in HDL (35mg/dl) level was noted in the lipid profile, where as the other lipid parameters are normal.The Urine spot Porpholinogen (0.06) was also found to be normal.

The diagnosis of Guillain-Barré syndrome was made according to the Asbury diagnostic criteria.⁴ The degree of motor function was expressed on a seven point functional scale as used in earlier trials: 0=healthy; 1= minor signs or symptoms but fully capable of manual work; 2=able to walk>10 m without

assistance; 3=able to walk>10 m with a walker or support; 4=bedridden or chairbound (unable to walk 10 m with a walker or support); 5, requiring assisted ventilation for at least part of the day;and 6, dead.

At entrance our patient was assigned a 3 disability grade. After obtaining informed consent from the patient, he was treated orally with the indigenous medicines (Ayurveda) like a polyherbal decoction namely Maharasnadhi Kashayam 20 ml twice daily before food, capsule Dhanwantharam 101 two capsules twice daily after food for fifteen days, Aswagandhadi Lehyam (Herbal Linctus) 5 gms twice daily after food. Massage with Mahamasha Thailam (Herbal oil) followed by fomentation of the affected parts with medicated milk and Rice.(Navara Kizhi). He was advised to take Cholecalciferol 60,000 I.U (DRISE Softgel capsule,USV, India) weekly once for twelve weeks and methycobalamine 1000 mcg (Capsule Renerve plus, Strides Arcolab,India) once daily after food for one month. He was also given rehabilitative treatment during his stay.

After three weeks, he was able to walk (grade 2) and presented a progressive improvement in the motor deficits of the arms. The patient was discharged at grade 1 after 8 weeks of treatment and advised to continue the medication with physiotherapy. He was instructed to keep off of from heavy physical endeavors including sports. His weakness gradually improved over one month. Therapeutic management was well tolerated and no clinical, biochemical, or haematological side effects were noticed.

At clinical follow up, performed after 6months, the patient showed no modification of the clinical grade. All deep tendon reflexes were normal. A neurophysiological study showed an improvement of the motor conduction velocity (median nerve 50 m/s; right ESP 46 m/s and left ESP 46 m/s). Neurological examination at the 1 year follow up showed a slightly distal muscular strength deficit of the left arm, but all the deep tendon reflexes were normal. He reported occasional numbness and tingling in both lower limbs. Motor conduction velocities were normal. The patient was able to perform normal daily activities.

III. Discussion

Guillain-Barre syndrome (GBS) is also known as polyradiculoneuritis, and chronic inflammatory demyelinating polyradiculopathy (CIDP).⁵ It is the most common cause of acute weakness in patients under 40 years of age. ⁶ It's etiology is unclear although it has been associated with both cell and humoral mediated autoimmune mechanisms.⁷ Mostly GBS occurs as sporadic cases. It has been reported to follow an infection by several organisms including Cytomegalovirus (CMV), Ebstien Barr virus (EBV) and influenza vaccine.⁸ The commonest epidemiological association has been described with a preceding infection by *Campylobacter jejuni*.⁹ Although most patients spontaneously recover from Guillain-Barré syndrome, the course of the disease might be severe, leading to severe tetraparesis which requires artificial ventilation in about 20% of the patients, with a long lasting and costly stay in intensive care units, and with residual deficits occurring in 5%-10% of the patients.¹⁰ The course of the disease is favourably modulated by plasmapheresis¹¹ and high dose intravenous gammaglobulin.¹² However, these approaches are expensive, so alternative therapies are much needed.

Ayurveda, the traditional Indian medicinal system remains the most ancient yet living traditions with sound philosophical and experimental background, helps to treat the immune attack on the nerve cells of the peripheral nervous system and their support structures leads to activation of a group of blood proteins in GBS.

IV. Conclusion

In the demyelinating forms of GBS, the basis for flaccid paralysis and sensory disturbance is conduction block. Degeneration of the basement membrane of the Schwann cell results in GBS.⁷ Hence, recovery can take place rapidly as remyelination occurs. The medicines such as Aswagandhadi Lehyam (Herbal Linctus) and Dhanwantharam 101capsules are well known for the remyelination of the nerves. The medicines used are well known anti-inflammatory and antioxidant drugs used in Ayurveda. Along with proper supplements of Vitamin complex in case of deficiency, the Ayurvedic treatment can be effectively employed to combat GBS without any clinical, biochemical, or haematological side effects.

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Conflicts of interest

The authors declare that they have no conflicts of interest relating to this short report.

Nerves	Side	BT NCV (m/s)	AT NCV (m/s)	
Peronial Nerve	Right	30	39	
	Left	31	41	
Post Tibial Nerve	Right	32	38	
	Left			
Median Nerve	Right			
	Left	38	50	
Ulnar Nerve	Right			
	Left	36	48	
AT : After Treatmen	t, BT : Befo	ore Treatment		

Table 1 Summary of Nerve conduction velocities before and after treatment