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A rare presentation of miller fisher syndrome- a case report

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ABSTRACT

In this report we describe a rare presentation of Miller Fisher syndrome, a variant of Guillain Barre Syndrome. This distinctive syndrome is an acute immune mediated neuropathies usually preceded by an infectious illness characterized by total external ophthalmoplegia, severe ataxia and tendon areflexia. The presence of the IgG anti-GQ1b antibody in serum is an excellent diagnostic marker for MFS. This antibody often cross reacts with GT1a and is patho physiologically associated with ophthalmoplegia or ataxia in MFS and GBS.

Key words: IgG anti-GQ1b antibody, ptosis, diplopia, miller fisher syndrome.

CASE REPORT

We report case of 50 year old female who presented with complaints drooping of both upper eyelids, diplopia, and swaying of body on either side while walking for last 7 days. Patient also gave history of nasal intonation of voice since 3 days. On general physical examination patient had Normal vitals.[1] Her central nervous system showed normal higher mental functions and fundus examination was normal.Patient had bilateral ptosis; bilateral dilated pupils with absent light, consensual and accommodation reflex. She also had loss of extra ocular movements in all directions(Fig1)

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Figure 1: Patient with bilateral ptosis; bilateral dilated pupils with absent light, consensual and accommodation reflex, loss of extra ocular movements in all directions

Power of muscles at various joints in both upper and lower limb was normal but she had hypotonia and areflexia in all limbs. Plantars were bilaterally mute. Rest systemic examination was normal.

Investigations: Routine baseline investigation like renal function test, liver function test and serum electrolytes were normal. She was retroviral and VDRL Negative. ECG and 2D Echo, CT scan head were normal. CSF analysis was normal. Nerve conduction study:

Motor – Normal; Sensory – All nerve has absent SNAP`s. Anti Gq1b antibody was present (Fig 2)

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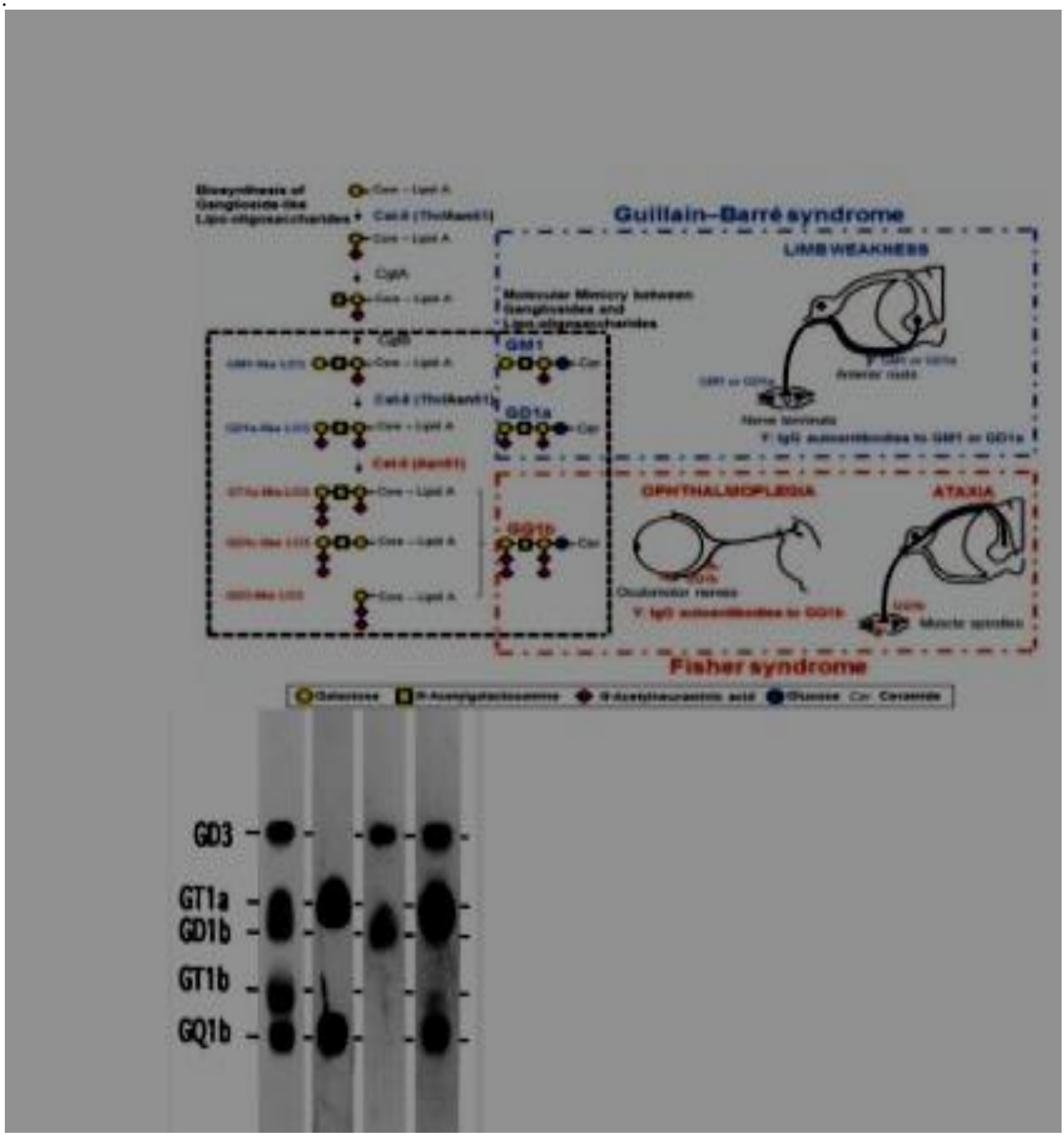

Figure 2:Representation of Miller Fisher syndrome

As per the history, examination and investigation findings the diagnosis of Miller Fisher Syndrome was made and treatments started with –5 cycle of Plasmapheresis which were given every alternate day for a period of 12 days. Patient gradually improved, ptosis slightly decreased and started walking after 2nd plasma pheresis with no swaying.

DISCUSSION

In miller fisher syndrome Serial neurophysiologic studies have shown involvement of peripheral nerve, with prolonged peripheral conduction in the blink reflex arc[2]. MFS has emerged as the archetypal anti ganglioside antibody mediated immune neuropathy.[3]It is associated with acute phase IgG antibodies to Gq1B, Gt1A in over 90% of cases which are highly disease specific. In MFS serum anti ganglioside titres are at their peak at clinical presentation and decay rapidly, where ELISA test with tryptophan based chromatography shows a corrected optical density of > 0.1 which is considered as positive[4].MFS follows a wide variety of infections including campylobacter jejuni and upper respiratory viral tract infections. It follows the principle mimicry between Gq1B, Gt1A and C.Jejuni lipopolysaccharide and lipoligosaccharide core oligosaccharides as central to induction of this response, hence also termed as Anti Gq1B IgG Antibody syndrome. 5- 10 % of cases do not have any symptoms of any preceding infections such as C.Jejuni eneteritis[5].The sera with Anti Gq1B antibodies causes a reversible failure of Ach release from presynaptic motor nerve terminals. Since only 5% incidences of MFS with Anti Gq1B positive cases reported in india, proper evaluation helps in diagnosing and treating this condition[6].

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Conflict of Interest: None

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