



## A Case Report on Miller Fisher Syndrome, a Rare Variant of Guillain-Barre Syndrome

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### Case Presentation

**Patient information:** A 36 year old known hypertensive obese man presented with almost sudden onset gait unsteadiness for 10 days along with difficulty in micturition (Intermittency, hesitancy) & constipation.

**Physical exam:** On examination, patient was conscious, awake. Tone both limbs were measured. Upper limb tone was normal but both lower limbs had hypotonia. Though both limbs had normal power. Plantar reflex of right side was flexor & left side extensor. Heel shin test was positive & finger-nose-finger test was clumsy. There was gaze evoked nystagmus on both sides.

**Provisional diagnosis:** In this background, the patient was provisionally diagnosed to be suffering from cerebellar stroke/dorsal myelitis.

**Investigations:** Patient was advised MRI brain with Dorso-lumbar spine, Vitamin E & physiotherapy.

MRI brain didn't show any abnormality, MRI Dorso-lumbar spine showed a Mild bulge of L4-L5 disc mildly compressing the ventral thecal surface & mild degenerative changes in spine.

Serum electrolytes (Na/K) were within normal reference range.

**Assessment:** On follow up patient showed little symptomatic improvement. On the background of ataxia & areflexia with normal imaging patient was diagnosed to be suffering from GB variant of Miller Fisher syndrome. He was advised for hospital admission in a Government set up (Due to financial constraints).

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After hospital admission routine biochemical tests were performed.

MRI Cervical spine revealed early degenerative changes in cervical spine without significant posterior disc herniation, neural compression or central canal stenosis.

VDRL test was non reactive. HBsAg, Anti-HCV & ICTC test were non reactive. ANA profile was nonreactive.

**Intervention:** Lumbar puncture was done. CSF was aspirated & sent for microscopy & biochemical tests. Cells were mainly mononuclear cells; microprotein concentration was 0.5 gm/dl.

Based on the clinical & laboratory investigations the patient is diagnosed as a case of Guillain-Barre syndrome. I.V Immunoglobulin infusion was started & continued for 5 days. Patient showed gradual significant neurological improvement & was discharged after 28 days in stable condition.

### Discussion

This disease is associated with infection by *Campylobacter jejuni* and *Cytomegalovirus* and Epstein Barr Virus. The incidence of GB Syndrome is round about one in one lakh population. The diagnosis of this disease is based on clinical examination and history supported by cerebrospinal fluid analysis. The plasma exchange and immunoglobulin intravenous is effective treatment in GB Syndrome [1].

Many patients of GB syndrome have musculoskeletal complaints. Peripheral neuropathy is also commonly seen in clinical practice which may be also due to alcohol intake, long standing uncontrolled diabetes. In extremities numbness and sclerosis occur.

The most common and symptoms of GB syndrome are the weakness of the limbs associated with areflexia. One of the subtypes of the GB syndrome is Miller Fisher syndrome. It is a rarer

variant of GB syndrome and usually presents with at least two of the following features: Ataxia, areflexia, and ophthalmoplegia. This disorder is treated by the immunoglobulin and plasma exchange. It is heterogeneous autoimmune disease. Autoantibodies are present against various antigens in peripheral nerves. The Miller Fisher Syndrome is commonly associated with the involvement of the lower cranial and facial nerves and does not usually involve motor weakness of limbs. However, in some cases weakness of the respiratory system and limbs has been described [2-6].

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