Introduction:
- Miller Fisher Syndrome is a variant of Guillain Barre Syndrome (GBS) with features of ophthalmoplegia, ataxia and areflexia or hyporeflexia.
- GBS is usually preceded by upper respiratory symptoms or diarrhea.

Presentation:
- A 74-year-old woman presented to a community hospital with an acute onset of blurred vision and difficulty ambulating.
- She had an episode of sore throat and cough 3 weeks before presentation.
- Her cough and sore throat resolved but she developed ear fullness followed by ear discharge. She was started on Levofoxacin for acute otitis media.
- After 3 days of Levofoxacin therapy, she presented to the ED with bilateral diplopia and ataxia.
- On examination, she was noted to have bilateral conjugate gate paresis and horizontal diplopia.
- She was also ataxic and had absent reflexes in bilateral lower extremities.

Diagnostic work-up:
- CT head, MRA/MRV brain, and MRI spine were negative for stroke, aneurysm, venous thrombosis, or demyelinating plaques.
- Protein electrophoresis and myasthenia gravis panel were also negative.
- Cerebrospinal fluid (CSF) viral panel and cultures were negative.
- CSF analysis was positive for albuminocytologic dissociation.
- Serum GQ1b antibody was negative initially but repeat testing on her 4th day of admission came back positive (1:1600).

Discussion:
- GBS usually presents with symmetric limb +/- cranial motor nerve weakness.
- Recent diagnostic criteria include supportive features such as preceding infectious symptoms, presence of sensory symptoms before motor symptoms and CSF albuminocytological dissociation.
- MFS is a variant of GBS defined by ophthalmoplegia, ataxia, and areflexia/hyporeflexia.
- Unlike GBS, 76% of the MFS cases are preceded by respiratory symptoms.
- As GQ1b is strongly expressed in cranial nerves 3, 4, and 6 along with muscle spindles, it is highly associated with symptoms of ophthalmoplegia and ataxia. Anti-GQ1b antibodies can be present in up to 83% of patients with MFS.
- Once a patient is diagnosed clinically with supportive evidence, prompt treatment with IVIG should be initiated.
- Recovery is slow and patients often end up with residual deficits, as seen in our patient.

Course:
- She was treated with a 5-day course of intravenous immunoglobulin (IVIG) and her blurred vision and ataxia were partially improved.
- She followed up at the neuro-ophthalmology clinic 8-weeks after her initial presentation, where she was found to have complete resolution of her ataxia and partial resolution of her ophthalmoplegia.