‘Guillain-Barre Syndrome’ with some distinct dispositions (DLE, TB meningitis, Hearing Loss) – A novel presentation

Lakshmi VenkataSimhacahalam Kutikuppala¹, Dr. SanthiVadugu²,
Dr. Deepak Kumar Behra³, Dr. VenuGopala RajuSV⁴

¹Second year MBBS, Konaseema Institute of Medical Sciences & Research Foundation(KIMS&RF), Amalapuram.
²Associate Professor, Department of Physiology, RIMS, Ongole.
³Assistant Professor, Department of General Medicine, KIMS&RF.
⁴Professor, Department of Physiology, KIMS&RF.
Corresponding Author: Dr. SanthiVadugu

I. Introduction:
GuillainBarre Syndrome is an Acute Inflammatory Demyelinating Polyradiculoneuropathy(AIDP) condition¹. It is a rare but serious medical emergency characterized by rapid onset of muscle weakness caused by immune system damaging the peripheral nervous system leading to weakness, numbness and tingling eventually causing paralysis². The incidence rate is 1.2-3/100,000 persons per year globally.

II. Case Report:
Patient details and History:This is a case of 40 year old male named I. Ramesh, water works supervisor by occupation, resident of Bodasakurru, Amalapuram, Andra Pradesh. The patient was apparently alright few hours since his admission into hospital and was unveiled to hospital with fever, generalized weakness, fatigue, immobilized limbs and deafness. No history of similar episodes in the past, no history of diabetes, hypertension, diabetes, tuberculosis, asthma, seizures, etc. Patient is known DLE sufferer from his age of 12th year. Decreased appetite, Disturbed sleep, decreased bowel and bladder habit, had smoking history with 10 pack years and non-alcoholic. No similar significant history in his family. No history of drug allergy.
Examination: Patient was conscious, coherent, moderately nourished and no signs of pallor, icterus, clubbing, cyanosis, lymphadenopathy, oedema, raised temperature, and respiratory rate. On further examination, he has Hearing loss, Grade-III Dyspneoa, No loss of memory, consciousness and awareness. Generalised Weakness and Severe Fatigue with Hypotonia, Grade 0 muscle activity and Areflexia significant in peripheral parts of the body with Quadreparesis³. Symmetrical and ascending type of progressive impairment is observed⁴.

On Clinical Investigations, All general investigations including CBP, CUE, LFT, etc are normal. There is slight raise in TLC. Later, X-ray and CT scan of head were done and it was found very mild inflammatory lesion in the mid-brain portion. Later, On CSF examination, there was elevated protein level and lymphocyte level in the CSF. TB meningitis was suspected by Acid Fast staining and patient was put on Anti-TB therapy and high range antibiotics and other bactericidal drugs.

Three months after continuous follow-up, the patient condition is as usual before and do not responded for treatment. Then, as it was in our suspicion before, we tried for diagnosis of GuillainBarre Syndrome. Again, on CSF examination there was raised protein and lymphocyte level, and this time CSF showed AFB negative. On further Electromyography (EMG) and Nerve Conduction Studies (NCS), especially of the peripheral nerves, there were findings of altered nerve conduction and demyelination⁵. This confirms us the case as Acute phase of GuillainBarre Syndrome (Grade-IV). Grade- IV is as the patient is bed-ridden.

III. Discussion:
So, from the above investigations, we came to the confirmatory diagnosis of GuillainBarre Syndrome of Grade-IV and started conservative management for it. Since he was in the acute phase of the disorder, he took time but responded well with the treatment. He was managed with IV Immunoglobulins⁶, IV fluids, Neuropathic pain releivers(Pregablin, Methylcobalmin, Alpha Lipoic acid), Duloxetine, protein milk powder, other nutrient supplemtations(multi vitamins & multi minerals and regular Physiotherapy⁷. He was on continuous follow-up and recovered progressively.
IV. Conclusion:

Thereby, this is a case of 40 year old male diagnosed with Grade IV Guillain-Barre Syndrome without any facial involvement (Miller Fisher variant), having past significant history of DLE. The supporting factors for our diagnosis are symmetrical and ascending type of paresis (characteristic of GBS) and abnormal Nerve Conduction Studies and already autoimmune state of DLE. So the patient was managed conservatively, discharged with continuous physiotherapy and drug course. After 9 months of progressive treatment, he recovered and reached usual stable state.

Acknowledgement: We are very thankful to our Head of the Department, our professors, investigating departments, and other assisting staff of KIMS & RF who made this case noteworthy.

References: