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Original Research Paper

Medicine

A RAPID PROGRESS AND SLOW RECOVERY OF G.B.S. WITH MULTIPLE COMPLICATIONS.

Dr Sumedh Agrawal* Junior Resident, Department of Medicine, Government Medical College, Aurangabad.*Corresponding Author

Dr. Vijay Gaikwad Associate Professor, Department Of Medicine, Government Medical College, Aurangabad.

Dr Nilesh Lomte D.M. Endocrinolgy, Assitant Professor , Department of Medicine, Government Medical College, Aurangabad.

Dr Meenakshi Bhattacharya Professor and Head, Department of Medicine, Government Medical College, Aurangabad.

KEYWORDS : Prolong ventilation ,bed sore, collapse lung.

INTRODUCTION:

Guillain Barre Syndrome (GBS) is acute, frequently severe and fulminant polyradiculopathy autoimmune disorder with incidence of 1-4/100000 [1]. It is characterized by areflexic Motor paralysis with or without Sensory Disturbances. Weakness typically evolves over hours to few days. Legs are more commonly involved than arms, and facial paresis is present 50% of affected individuals. Most of patients require hospitalization and upto 30% require ventilatory assistance at some time during illness. Deep tendon reflex and proprioception affected due to involvement of large sensory fibres [2, 3]. Autonomic involvement is common with fluctuation in blood pressure, postural hypotension and cardiac dysrhythmias. Pain is common feature of GBS.

CASE REPORT:

A middle age female patient admitted with complaints of sudden onset weakness of bilateral lower limbs and upper limbs. She was unable to stand and walk within 4 hours of onset of symptoms. She was not able to get up from bed and lift her upper limbs above shoulder. Her weakness was progressive over next 12 hours and she was not able to lift her upper limbs and lower limbs off the bed. She didn't have history of fever, cough, vomiting, loose motions and recent vaccination.

At the time of admission she was Conscious, oriented to time, place, and person. Vitals were stable including pulse and blood pressure. On admission her Power of lower limbs were 1/5, and upper limbs were 2/5. Hypotonia was present in all four limbs. Her sensory system was normal. All deep reflexes of upper limbs and lower limbs were absent. Her rest of central nervous system examination was within normal limits. On admission she has no respiratory distress.

Patient started complaining of breathlessness and developed respiratory distress after 26 hours of initial symptoms for which patient was intubated and shifted to M.I.C.U. and taken on mechanical ventilatory support with VCV AC Mode. Ventilatory support through endotracheal tube continued for 15 days and Tracheostomy was done. Her C.S.F. studies were performed which were within normal limits with proteins - 35 mg %, sugars – 97 % and was acellular. We have kept Provisional Diagnosis of G.B.S. with Respiratory failure. NCV studies was suggestive of AMSAN type of G.B.S. She was started on plasmapheresis after 48 hours of initial symptoms. Total 7 cycles of plasmapheresis were done over a period of 10 days. During plasmapheresis 8 liters of plasma exchange was done. Even after 7 cycles of plasmapheresis, there was no improvement in patient muscle power and patient continued on ventilatory support. After 7 days of ventilatory support she developed ventilator associated pneumonia (VAP), for that she was started on injectable Meropenem and injectable Linezolid, which was continued for next 14 days. Her fever persisted even after giving these antibiotics. Tracheal culture was suggestive of Klebsiella pneumonia and MRSA, which was resistant to injectable Meropenem and injectable Linezolid. For that she was shifted to injectable Tigecycline continued for 21 days. Later on patient was shifted to injectable Clarithromycin and injectable Amikacin on 50th day of admission for 5 days. Even after giving all above antibiotics patient has sign and symptoms of Ventilatory associated pneumonia (VAP) and chest radiograph was suggestive of right sided mid and lower zone lung consolidation. For that reason patient was shifted to Injectable Piperacillin + Tazobactum on 56th day for 21 days. After 21 days tracheal culture was done which was suggestive of Pseudomonas aeruginosa which was sensitive to collistin, patient was shifted on injectable Collistin for 7 days. Later on 85th day patient was shifted to injectable Cefoperazone + sulbactum and injectable Metronidazole which was given next for 14 days. Despite of ventilatory support and higher antibiotics she was not improving and spontaneous breathing trials were failed. Chest radiograph was repeated suggestive of right sided lung collapse with consolidation. Bronchoscopy finding were suggestive of right sided bronchial secretion with yellowish patch, mucosal injuries over secondary carina which can lead to collapse. BAL was sent for culture and sensitivity on 96th which was suggestive of pseudomonas aeruginosa sensitive to injectable piperacillin + tazobactum. And patient was started on injectable piperacillin + tazobactum for next 21 days to which patient responded well. Chest physician opinion was taken and was advised for supportive care and chest physiotherapy. Gradually patient started improving and was shifted on weaning modes of ventilation and tracheostomy tube de-cannulated after 142 days of ICU management. She also developed a Bed sore 15cm x 10 cm with depth of 3 cm. It was managed conservatively without graft surgery with debridement followed by dressing twice daily with Silver nitrate and healed with good results.

Patient was shifted to general ward on 145th day of admission, with grade 1 power in both upper limbs and lower limbs with distal > proximal muscles. She has total hospital stay of 152 days(6/7/17 to 6/12/17). At the time of discharge she was Conscious, oriented to time, place, and person. Vitals were stable including pulse and blood pressure and Power of lower limbs were 1/5, and upper limbs were 1/5.

DISCUSSION:

Guillain Barre Syndrome is acute, frequently severe and fulminant polyradiculopathy autoimmune disorder, Characterized by Areflexic Motor paralysis with or without Sensory Disturbances (2). Diagnosis of GBS is usually made based on history and examination alone. Clinical features includes Antecedent infectious symptoms

,Presence of distal paresthesia at or before the onset of weakness ,Symmetrical weakness, Monophasic disease course with interval between onset and nadir of weakness of 12 hours to 28 days, followed by clinical plateau (2,3). GBS has 4 subtypes recognized primarily by electrodiagnosis and pathologic distinction.

Types	Features	Electrodiagnosis
Acute inflammatory demyelinating polyneuropathy (AIDP)	Adults affected more than children, recovery rapid, anti-GM1 antibodies (<50%)	Demyelinating
Acute motor axonal neuropathy (AMAN)	Children and young adults, recovery rapid, anti-GD1a antibodies.	Axonal
Acute motor sensory axonal neuropathy (AMSAN)	Mostly adults, uncommon, recovery slow, closely related to AMAN.	Axonal
Miller Fisher syndrome MFS	Adults and children, ophthalmoplegia, ataxia and areflexia ,anti-GQ1b antibodies	Axonal or demyelinating

In our case of guillian barre syndrome (AMSAN) patient has acute onset of weakness in lower limbs followed by bilateral upper limbs within a period of 4 hours (ascending paralysis).

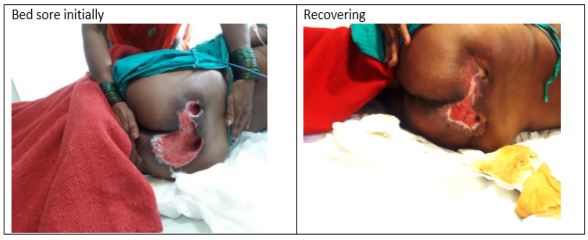
In Guillain Barre Syndrome there is Weakness and areflexia in all four limbs which can be with or without cranial nerve involvement and respiratory depression. Sensory symptoms frequently appear before or at the onset of weakness and many patients complain of a tingling or pricking sensation (paresthesia) in their hands and feet. Respiratory depression and cranial neuropathy often occur later (3). In our case she has areflexia including all joints of upper limbs and lower limbs. There was no involvement of autonomic nervous system. There was no cranial nerves and sensory involvement. After 26 hours of presentation she developed weakness of respiratory muscle and developed respiratory distress for which she was taken on ventilatory support.

Laboratory feature of G.B.S. include C.S.F protein 1-10 g/L without pleocytosis and earliest feature in Nerve Conduction Velocity (NCV) studies are prolonged f waves latencies, prolonged distal latencies, reduced amplitude of compound muscle action potentials (CMAPs). In our case NCV studies were suggestive of AMSAN type of G.B.S. and C.S.F studies were with in normal limits.

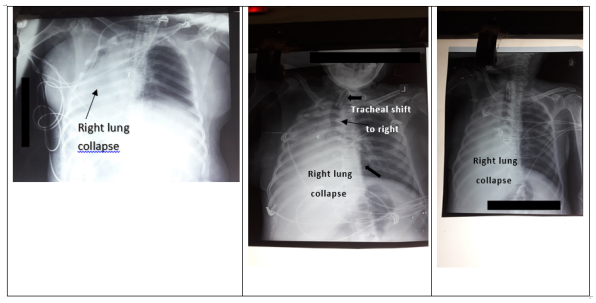
Not all patients require treatment, but in most centres intravenous immunoglobulin or plasma exchange are initiated if weakness is rapidly progressive or if there is significant bulbar or respiratory muscle compromise (4). Our patient was started on plasmapheresis of which 7 cycles were given. During hospital stay she develop VAP for which she was given multiple higher antibiotics, she developed bed sore treated by silver nitrate dressing, developed left lung collapse for which ventilatory support was given. She required ventilatory assistance for a period of 142 days.

Despite current treatment, GBS remains a severe disease, as about 30 % of patients require artificial ventilation during a period of days to months, about 20% of patients are still unable to walk after 6 months and 3-10% of patients die. Additionally, many patients have pain, fatigue or other residual complaints that may persist for months or years. Our patient was discharged after 152 days of in patient management.

Images of Bed sore and Chest radiographs



Radiograph suggestive of Right lung collapse



References:-

1. Ann Indian Acad Neurol. 2011 Apr-Jun; 14(2): 98 102., doi: 10.4103/09722327. 82793PMCID: PMC3141496, Clinical features and prognosis with Guillain-Barré syndrome Sinan Akbayram, Murat Doğan, Cihangir Akgün, Erdal Peker, Refah Sayın,1 Fesih Aktar, Mehmet-Selçuk Bektaş, andHüseyin Çaksen[2]
2. Wakerley BR, Uncini A, Yuki N. Guillain-Barré and Miller Fisher syndromes: new diagnostic classification. Nat Rev Neurol. 2014;10(9):537–544.
3. Br J Gen Pract. 2016 Apr; 66(645): 218–219.,doi: 10.3399/bjgp16X684733 PMCID: PMC4809704 ,Guillain–Barré syndrome in general practice: clinical features suggestive of early diagnosis, Virginia A Head, MRCP ,Benjamin R Wakerley, PhD, MRCP
4. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med. 2012;366(24):2294–2304
5. Presse Med. 2013 Jun;42(6 Pt 2):e193-201. doi: 10.1016/j.lpm.2013.02.328. Epub 2013 Apr 28. Diagnosis, treatment and prognosis of Guillain-Barré syndrome (GBS). van Doorn Pa1. prognosis of Guillain-Barré syndrome (GBS). van Doorn PA[1].