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MANAGEMENT OF GUILLAIN BARRÉ SYNDROME IN PATIENTS WITH COMMUNITY ACQUIRED PNEUMONIA

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Abstract

Purpose*:* Gullain Barre Syndrome (GBS) is a sub-acute inflammatory demyelinating polyneuropathy which often triggered by a preceding acute infection that mainly manifests as paresthesia, progressive bilateral and relatively symmetric weakness of the limbs that progresses over days to weeks. The purpose of this study is to determining management of guillain barré syndrome in patients with community acquired pneumonia

Methodology*:* We presented a case of 41 years old male, presented with dyspnea and weakness in bilateral upper and lower limbs. The study was determining early signs of acute respiratory failure in patient, intubation with mechanical ventilation support was done and admitted in the ICU.

Findings: Mortality rate has varied between 1-18% and usually attributable to pneumonia, sepsis, adult respiratory distress syndrome, or pulmonary embolism. In the current study, after the patient was aggressively treated with broad spectrum antibiotics, plasmapheresis and was supported by mechanical ventilation in the ICU, the patient started to show improvement on second day and progressed well. The patient then was discharged from the ICU on 11th day of admission.

Unique contribution to theory, practice and policy: An aggressive management of respiratory failure with appropriate mechanical ventilation and antibiotics in *Community Acquired Pneumonia* (CAP) patients results in better patient states and faster recovery. From the current study, we recommend healthcare practitioners to perform an early aggressive management including mechanical ventilation and broad-spectrum antibiotics administration for GBS patient with respiratory failure and CAP.

Keywords: *Community Acquired Pneumonia, Gullain Barre Syndrome, Respiratory Failure*

1.0 INTRODUCTION

Gullain Barre Syndrome (GBS) is a sub-acute inflammatory demyelinating polyneuropathy which often triggered by a preceding acute infection. This autoimmune disorder mainly manifests as paresthesia, progressive bilateral and relatively symmetric weakness of the limbs that progresses over days to weeks (Aminzadeh & Rad, 2014; Kishore, Vijayabhaskar, Vishnu, Sainaresh, Sriramnaveen, Sridhar, ... & Siva Kumar, 2014; Nobile-Orazio, 2018). Most patient reports inability to move and walk few days prior onset with 20-30% needed ventilation. Mortality rate has varied between 1-18% and usually attributable to pneumonia, sepsis, adult respiratory distress syndrome, or pulmonary embolism (Bhadade, deSouza & Bawaskar, 2014).

Gullain Barre Syndrome is often associated with non-specific acute infections. The incidence of GBS cases associated with these infections are around 56% - 80%, one to four weeks before neurological symptoms such as upper respiratory tract infections or gastrointestinal infections appeared (Sawelinggi, Aryabiantara &Wiryana, 2019). The diagnosis of GBS is built upon clinical presentation (paresthesia and motoric paralysis of the limbs), examination of nerve conduction and analysis of cerebrospinal fluid (an increase in protein). Management was aimed for supportive and specific therapy (Sawelinggi, Aryabiantara & Wiryana, 2019; Prasad, et al.,2017).

Community acquired pneumonia (CAP) is an acute infection of the pulmonary parenchyma, with symptoms onset in the community. Incidence of CAP is 1.6-10.6 per 1000 adult population in Europe and 1.2-10% of them requiring ICU admission. Classic presentations of CAP included infection (fever, leukocytosis) with clinical (sputum production, tachypnea, cough, pleuritic chest pain) signs and infiltrative imaging on x-ray. The main pathogen of CAP is *Streptococcus pneumonia* and *Staphylococcus aureus*. (Morgan & Glossop, 2016; Khatib, Naous, Ghanem, Dbaibo, & Rajab, 2017).

2.0 FINDINGS

A-41-year old man presented with difficulties of breathing and general weakness of upper and lower extremities four days prior hospital admission. His symptoms began slowly following an upper respiratory tract infection he had 14 days prior. No prior past medical history was reported. Upon presentation to Emergency Department, his consciousness started to decrease, vital signs showed blood pressure 135/100 mmHg, heart rate 120x/min, temperature 37.2^0 C, rapid and shallow breathing with rate 36x/min spontaneously with cold extremities and poor perfusion. No retraction but bilateral ronchi was found on thoracic examination. Bilateral upper and lower limbs showed no power (0/0/0/0) and absent deep tendon reflexes. Endotracheal tube was inserted due to

respiratory distress. After intubated, he was supported by mechanical ventilator with SIMV mode 12, PEEP 5, PS 12 and FiO2 60%. Broad spectrum antibiotic was given. He was diagnosed as Guillain Barre Syndrome and treated in the ICU.

Chest x-ray revealed bilateral bronchopneumonia with sinus tachycardia on electrocardiogram. Laboratory results showed leukocytosis (21.740 m/L), blood gas analysis (BGA) of pH 7.381, $PaCO_2$ 35.3, PaO_2 160, HCO3 19.6, BE 4.7, SO_2 99.2%.

First day in the ICU, the patient showed stable vital signs with ventilator mode CPAP PS 14, PEEP 5 FiO2 50% and saturation 96-97%. He received Ceftriaxone 1gr/12 hours iv, leucocyte count started to decrease with BGA of pH 7.39, $PaCO_2$ 32.4, PaO_2 129, HCO3 30, BE -3.5, SO_2 98%. Plasmapheresis was planned until six times, first two days in a row and next one every other day. On the second day, the patient was febrile, temperature reached $37\text{-}39^0$ C and antibiotic was changed to meropenem. Improvement of upper limb motoric functions were seen (1/1). Ventilator mode was set to PS 12, PEEP 5 and FiO_2 45%. Patient received the fourth plasmapheresis on Day 6, all extremities' motoric function progressed to 3/3/3/3 and on Day 8 he gained function to 4/4/4/4. On day 11, he was extubated, re-imaging on chest x-ray showed improvement of bronchopneumonia.

Community acquired pneumonia is defined as pulmonary parenchyma infection with symptoms onset in the community (Morgan & Glossop, 2016). Criteria of CAP included new infiltrates in lungs (less than 24 hours prior admission), and one of the following: current cough symptoms with or without sputum, fever (>37.8C) or hypothermia (<36.5), leukocytosis or leukopenia, without hospitalization 14 days prior admission (Orlikowski, et al., 2006). The main pathogen causing CAP is *Streptococcus pneumoniae* and *Staphylococcus aureus* (Slupsky, Cheypesh, Chao, Fu, Rankin, Marrie & Lacy, 2009). Severity of pneumonia is determined by Pneumonia Severity Index for mild symptoms and SMART-COP for patients requiring respiratory support or vasopressor. (Mohamed & Abd Allah, 2019).

Aggressive early diagnosis and therapy aimed to prevent multiorgan worsening (Orlikowski, et al., 2006). Non-invasive ventilation might be used for supportive therapy and patients with severe CAP might need intubation with mechanical ventilation support (Brochard,2003). Antibiotics are adjusted to the severity of disease (Ott, Hauptmeier, Ernen, Lepper, Nüesch, Pletz, ... & Bauer, 2012).

In this case, patient had prior upper respiratory tract infection for 14 days prior hospital admission. The symptoms followed by an increase in leukocyte and rhonchi in both lungs. Therapy was done supportively with mechanical ventilation and Ceftriaxone as antibiotic. On the second day, a rise in temperature and leukocyte was shown, ceftriaxone

was replaced by meropenem. After eight days intensive care, physical examination rhonchi disappeared and chest x-ray shown improvement of bronchopneumonia.

Guillain Barre Syndrome (GBS) is a peripheral neuropathy syndrome that causes acute neuromuscular failure. Incidence of GBS is 1.2-1.6 per 100,000 adult population (Winer, 2008). Clinical presentation is an acute neuropathy such as progressive weakness of the limbs worsening in four weeks, with or without loss of sensory function. Decrease tendon reflexes and an increase of protein in CSF examination might be found (Khatib, et al., 2017; Prasad, et al., 2017).

GBS is divided into several subtypes, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome. Its incidence is between 1-2 events per 100,000 people. (Dimachkie & Barohn, 2013). The subtype of GBS incidence varies from one country to another. Europe and North America predominantly had AIDP in 90% of cases, where 5% of cases were axonal subtypes. The safest subtype was found in China and Japan. The United States, Central America, Japan and China had axonal subtypes incidence of 37–47%; the incidence of the Miller Fisher subtype is around 5%. The incidence of AIDP and AMAN is almost the same in Indians, although SAFE is more common in young patients. Two thirds of patients had symptoms of infection within three weeks before the onset of weakness. Associated symptoms include fever, cough, runny nose and diarrhea. Most of the symptoms preceding GBS infection were upper respiratory and digestive tract infections. The most commonly found pathogen is *C. Jejuni* (Meena, Khadilkar & Murthy, 2011).

Infection induce aberrant immune response has 4 processes; antiganglioside antibodies, molecular mimicry and cross reactivity, activation of the complier and host factor (Robert, Usuki & Ariga, 2006). Progressive weakness of the limb is a major feature. Maximum time of weakness to occurs is at week four, but the initial symptoms may start at week 2. Patient then experiences a plateau period, varying from several days, weeks to months (Van Doorn, et al., 2010).

Treatment for GBS includes specific therapy and supportive therapy. Supportive therapy with mechanical ventilation is important in patients with quite severe conditions. The initial consideration of mechanical ventilation use is important. Patients with a vital lung capacity of less than 20 ml / kg are at high risk. Specific treatment consists of plasma exchange (PE) and intravenous immunoglobulin (IVIG). Administration of PE is considered most effective if given before 7 days of onset. PE is recommended as much as 4 times in 1-2 weeks (Kishore, et al., 2014; Borse, Avate, & Palasdeokar, 2016). IVIG administration is carried out at a dose of 0.4 g / kg for 5 consecutive days. Both of these

treatments have the same effectiveness (Kerr, et al., 2014). Some therapies take place in the ICU such as physical therapy, chest physiotherapy, eye care, gastrointestinal and bladder care, position and beds that can prevent the formation of wounds, adequate nutrition and psychological support.

This patient was diagnosed with GBS due to upper respiratory infection 2 weeks prior hospital admission, weakness of the 4 limbs that was ascending, and motor strength decreased until zero. Respiratory failure was also found due respiratory muscles involvement. Supportive therapy was given by inserting ETT and mechanical ventilation.

Administration of PE is carried out on the 2nd and 3rd day in a row, followed by an interval of 6 times a day. On the 8thday of care, respiratory assistance was minimal, the motoric strength in both limbs slowly improved to 3 and on the 10th day of treatment, the patient was extubated with motor strength 4. On the 11th day the patient was moved to the ward.

3.0 CONCLUSION AND RECOMMENDATION

Conclusion

Aggressive management of respiratory failure with appropriate mechanical ventilation and antibiotics in CAP patients results in better patient states and faster recovery.

Management of aggressive respiratory failure with mechanical ventilation and administration of 6 times plasmapheresis therapy provides a good and rapid cure in patients with Guillain Barre Syndrome.

Recommendation

From the current study, we recommend healthcare practitioners to perform an aggressive management including mechanical ventilation and broad-spectrum antibiotics administration for GBS patient with respiratory failure and CAP.

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