

GUILLAIN BARRÉ SYNDROME- AN INFLAMMATORY PARALYTIC NEUROPATHY

Neurology

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KEYWORDS

INTRODUCTION

Guillain Barre Syndrome also known as landry paralysis was first recognised by the french physician Jean Baptiste Octave Landry, who described it as ascending paralysis.(1)

Later in 1916, Georges Guillian, Jean Alexandre Barre and Andre Strohl diagnosed two soldiers with this illness, in which they observed high levels of protein in the cerebrospinal fluid, which drew the attention of clinician, researchers and patients worldwide.(2)

GBS- Acute inflammatory polyradiculoneuropathy- An immune mediated condition of peripheral nerves usually followed by infections leading to demyelinating or axonal damage of peripheral nerves.(2)

And causes acute flaccid ascending paralysis which eventually leads to respiratory muscle paralysis causing to acute respiratory failure resulting in death of the patient.

Epidemiology And Etiology

Eventhough it can affect individual of 0-59 age, it has bimodal distribution with people occuring in young adults and older people.

Recent estimates suggest a global annual incidence ranging from 0.89 to 1.89 cases per 100,000 person-years, though variations exist across regions and populations.(3)

On January 9 2025, there was an outbreak of GBS in India, Maharashtra- Pune where 170 suspected cases GBS were reported from which atleast 20 patients were on ventilator support and 61 patients were in intensive care unit and there were 6 fatalities, these patients suffered sudden numbness and severe muscle weakness.

In which a patient of 40 years died who suffered severe breathlessness, weakness in lower limb and diarrhoea was kept on ventilator support but eventually died due to GBS.

Another patient of 63 years suffered from fever, diarrhoea, weakness in lower limb with comorbidities of uncontrolled diabetes died due to acute ischemic stroke could be due to systemic inflammation, autoimmune dysfunction and hyperviscosity of IVlg.

Its multifactorial with preceding infection notably with *Campylobacter jejuni*, Cytomegalovirus, Epstein Barr Virus and Severe Acute Respiratory Syndrome- Coronavirus 2 (SARS-CoV2)- they are implicated as triggering events in majority of the cases.(4)

Pathophysiology

GBS is believed to induce autoimmune processes due to cross-reactivity of neuronal and pathogenic proteins- this is known as molecular mimicry- as it emerges from selection pressure on pathogens to avoid the host immune system.

Organisms causing infections or trauma activates the immune system which stimulates the dendritic cells which presents bacterial antigen to both T cell and B cell which further initiates complex immune response, as there is decreased production of antibodies by B cells causing limited clearing of the infection which actuate molecular mimicry between bacterial antigen and the host cell nerve tissue leading to cross reactivity to antibodies, then to nerve gangliosides, the ganglioside antibody then activates the complement system which recruit macrophage to the nodes of ranvier, cause axonal degeneration and block the conduction, leads to the stoppage or slowing of the nerve impulses resulting in flaccid paralysis with muscle denervation and atrophy.

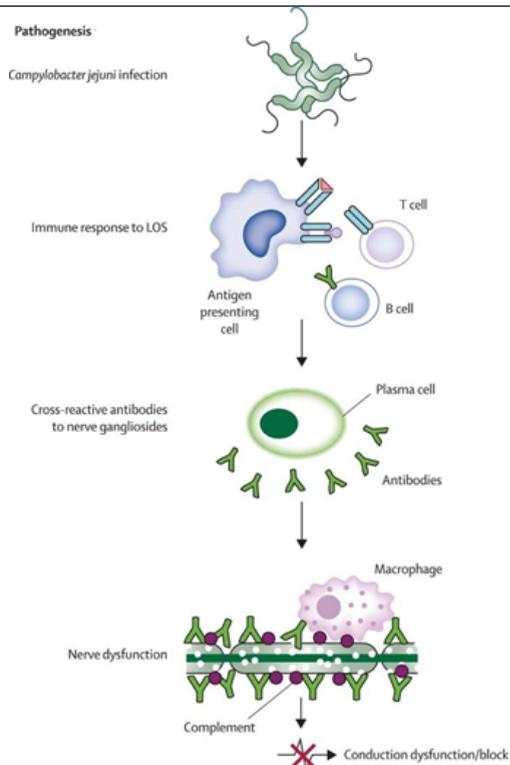


Fig-1(5)

Symptoms

Early Symptoms

Weakness-begins rapidly and worsens over hours to days. There is ascending weakness leading to paralysis- first feet are affected it is noticed when the patient has unexpected difficulty to climb stairs or to walk. Then eventually weakness impacts muscles of arms and legs, respiratory muscles.

Paresthesia- Feeling of tingling, formication (a sense of insects crawling under the skin) and deep muscular pain.

Late Symptoms

Difficulty with eye muscles and vision, difficulty swallowing, speaking or chewing, abnormal heart rate and blood pressure, problems with digestion and bladder control.(6)

Diagnosis

Medical history and Physical examination To assess how the patients muscles and nerves are coordinating and working. Whether the symptoms involved are unilateral or bilateral.

Electromyography And Nerve Conduction Velocity Test

To assess muscles and nerves activity and ability to send a signal and the responses are recorded. In GBS- the signals travelling along the damaged nerves are slowed down due to damage to the myelin sheath.

Cerebrospinal Fluid Analysis

In GBS- more protein(>400mg/l) and few immune cells can seen.

Imaging

MRI Scan of Spinal cord or Brain to find any other potential causes of muscle weakness.

Treatment

Plasmapheresis- In this process some blood is removed through a catheter and plasma is separated from blood cells, then the cells along with replacement fluid is returned to the body- it removes antibodies and other potentially injurious factors from the blood stream.

Intravenous Immunoglobulin Therapy- Injections of immunoglobulins are administered, it can lessen the immune attack on the nervous system and shorten the recovery time.(7)

REFERENCES

- 1) Skalski, P., Owecki, M.K. & Magowska, A.M. Jean Baptiste Octave Landry (1866–1940). *J Neurol* 266, 2341–2343 (2019). <https://doi.org/10.1007/s00415-018-9120-4> <https://pubmed.ncbi.nlm.nih.gov/articles/PMC6687682/>
- 2) Clinical presentation and symptomatology of Guillain-Barré syndrome: A literature review Elendu, Chukwuka BSc, MDA,*; Osamuyi, Emmanuella I. MBBSb; Afolayan, Ikeoluwa A. MBBSc; Opara, Nnamdi C. MBBSd; Chinedu-Anunaso, Nkeiruka A. HND, MBBS, FWACP, MBA, MSc; Okoro, Chinonso B. MBBS; Nwankwo, Augustine U. MBBSa; Ezidiegwu, Dianne O. MBBSf; Anunaso, Chinweike A. MBBSg; Ogbu, Collins C. MBBS; Aghahowa, Samuel O. MBBS; Atuchukwu, Chibuzor S. MBBSf; Akpa, Everister U. MBBSj; Peterson, Jesse C. MBBSa *Medicine* 103(30):p e38890, July 26, 2024. | DOI: 10.1097/MD.00000000000038890 https://journals.lww.com/md-journal/fulltext/2024/07260/clinical_presentation_and_symptomatology_of.28.aspx#JCL-P-10
- 3) Yuki N, Kokubun N, Kuwabara S, Sekiguchi Y, Ito M, Odaka M, Hirata K, Notturmo F, Uncini A. Guillain-Barré syndrome associated with normal or exaggerated tendon reflexes. *J Neurol*. 2012 Jun;259(6):1181-90. doi: 10.1007/s00415-011-6330-4. Epub 2011 Dec 6. PMID: 22143612. <https://pubmed.ncbi.nlm.nih.gov/22143612/>
- 4) Criteria for diagnosis of Guillain-Barré syndrome. *Ann Neurol*. 1978 Jun;3(6):565-6 <https://pubmed.ncbi.nlm.nih.gov/677829/>
- 5) Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome Pieter A van Doorn, PhDa p.a.vandoom@erasmusmc.nl · Liselotte Ruts, MDA · Bart C Jacobs, PhDa,b, Volume 7, Issue 10P939-950 October 2008 [https://doi.org/10.1016/S1474-4422\(08\)70215-1](https://doi.org/10.1016/S1474-4422(08)70215-1) [https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422\(08\)70215-1/abstract](https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422(08)70215-1/abstract)
- 6) Clinical Intelligence Guillain-Barré syndrome in general practice: clinical features suggestive of early diagnosis Virginia A Head and Benjamin R Wakerley *British Journal of General Practice* 2016; 66(645): 218-219. DOI: <https://doi.org/10.3399/bjgp16X684733> <https://bjgp.org/content/66/645/218>
- 7) Sprenger-Svačina, A., Svačina, M. K. R., Gao, T., Zhang, G., & Sheikh, K. A. (2024). Emerging treatment landscape for Guillain-Barré Syndrome (GBS): what's new? *Expert Opinion on Investigational Drugs*, 33(9), 881–886. <https://doi.org/10.1080/13543784.2024.2377323> <https://www.tandfonline.com/doi/full/10.1080/13543784.2024.2377323>