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An Integrative Review on Autoimmune Neurological Disorder

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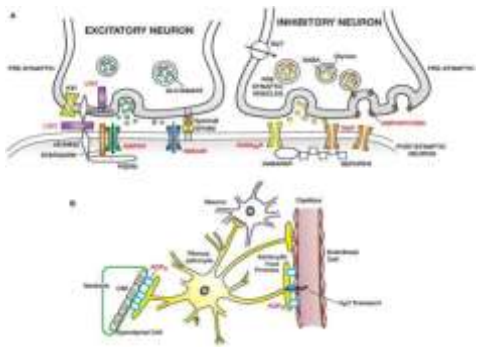
ABSTRACT:
Autoimmune diseases are conditions in which our immune system mistakenly damages healthy cells in the body and it occurs when the body's immune system mistakenly attacks and damages its own tissues, instead of fighting off foreign invaders like bacteria or viruses. This happens because the immune system loses the ability to distinguish between healthy cells and those that are foreign, leading to inflammation and damage. In the past few decades, autoimmune and non-infectious inflammatory disorders of the nervous system have become increasingly recognized as major sources of disease and disability. Autoimmune disorders of the nervous system are generally diagnosed at a younger age than other acquired disorders of the nervous system, such as infections or vascular injuries. However, all age groups can be affected. The main aim of this investigation is understand the mechanisms by which the immune system mistakenly attacks components of the nervous system, leading to neurological disorders. By exploring the specific auto - antibodies involved, the affected neural pathways, and potential treatment strategies to shed light on the causes, pathology, and management of neurological conditions arising from autoimmune responses within the central and peripheral nervous systems. By identifying the specific antibodies that target components of the nervous system in different autoimmune neurological diseases, can help with diagnosis and treatment development. By understanding the disease mechanism to know how these auto antibodies interact with neural tissues and disrupt normal nervous system function is crucial for developing targeted therapies.

KEYWORDS: Auto immunity, CNS, PNS, Brain cells, Inflammation.

I. INTRODUCTION

An Autoimmune disorder of the nervous system may affect any part of the nervous system, including the brain and spinal cord (central nervous system, CNS) and also the peripheral nerves, neuromuscular junction and skeletal muscle (peripheral nervous system, PNS). Autoimmune diseases in nervous system are mediated by self-invasive lymphocytes or anti-neuronal antibodies (Abs), anti-glial Abs. These immune cells and Abs were recognized as antigens by the immune system, and because these antigens could not be entirely eradicated, their presence caused the protein components of the neurological system to constantly be attacked by the immune system.

When this abnormal immune attack occurred in the nervous system or the neuro-muscular connections, it could cause in neuronal damage, axon injury and demyelinating or neuromuscular junction lesion. In the central nervous system, multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD),and acute disseminated encephalomyelitis (ANEM)were represented. In the peripheral nervous system, it is represented by Guillain-Barre syndrome (GBS), and neuromuscular junction lesions are represented by myasthenia gravis (MG) [1].



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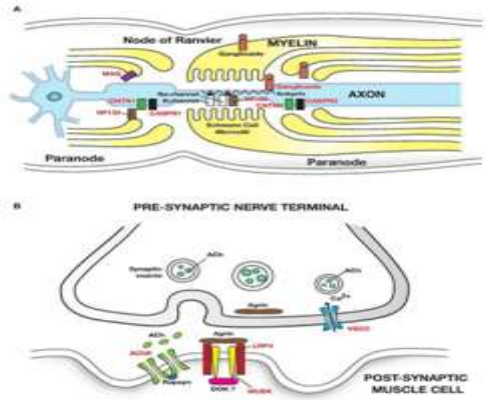

Fig. 1: Pathophysiology in Central Nervous System & Peripheral Nervous System

Depends upon the pathophysiology, the autoimmune disease can be categorized under three major divisions.

1. Paraneoplastic disorders
2. Post infectious conditions
3. Primary autoimmune disorders

In Paraneoplastic disorder which occurs in the context of a neoplasm, the immune response is directed against neuronal antigens that are ectopically expressed by the tumor.
In Post infectious conditions are mediated by an abnormal immune response to a foreign antigen that results in the elaboration of an immune response targeting a similar- appearing self-antigen (eg, Guillain-Barre syndrome following an infection with Campylobacter jejuni, resulting in the production of anti-ganglioside antibodies).
In Primary autoimmune disorders, the abnormal immune response develops in the absence of any clear inciting trigger; however, patients may have a personal or family history of autoimmunity, suggesting an underlying inherited predisposition.

Table 1: Neurological Disorders Under Nervous System

S.NO	Autoimmune disease in CNS	Auto immune disease in PNS
1.	Multiple sclerosis	Guillian barre syndrome
2.	Neuro myelitis optica spectrum disorder	Systemic lupus erythmatosus (lupus)
3.	Transverse Myelitis	Sjogren's syndrome:
4.	Acute disseminated encephalomyelitis	Chronic Inflammatory, DemyelinatingPolyneuropathy
5.	Hashimoto's encephalopathy	Rheumatoid arthritis:

II. MATERIALS AND METHODS:

GUILLIAN BARRE SYNDROME:

Guillain Barre Syndrome (GBS) is an auto immune disorder of the peripheral nervous system and immune mediated poly neuropathy, the body immune system attacks the peripheral nerves leading to varying degree of motar dysfunction and sensory impairment. GBS occurs more frequently in males than in females and all age will be affected. Patients with GBS can rapidly develop progressive weakness and sensory deficit that leads to complete paralysis and requires mechanical ventilation. The GBS can be influenced by various geographical factors, seasonality and specific infectious agents such as Campylobacter jejuni, Cyto megalo virus (CMV), Epstein-Barr virus (EBV) and Zika virus. Recent researches suggest that COVID- 19 became onset of GBS[3].

Common Symptoms of GBS [5]:

- Tingling feel in the fingers, toes, ankles or wrists.
- Weakness in the legs that spreading to the upper body leads to paralysis.
- Unable to walk or climb stairs, Trouble in breathing
- Feeling trouble with facial movement, such as during speaking, chewing, or swallowing
- Having double vision or being unable to move eyes
- May feel severe pain like achy, shooting or cramp like and may be worse at night.
- Trouble with bladder control or bowel function.
- Increased heart rate and BP may low or high

The symptoms of Gullian Barre syndrome is varies from their subtypes and GBS can be classified as many types, some important subtypes are as

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follows.

Classification of GBS Subtypes and its Symptoms:

The auto immune mechanism underlying various types such as,

- Acute inflammatory demyelinating polyneuropathy (AIDP) - The most common sign of AIDP is muscle weakness that starts in the lower part of the body and spreads upward.
- Acute motar axonal neuropathy (AMAN) – The symptoms include sensory impairment such asfacial movements, speaking, chewing etc.
- Acute motar and sensory axonal neuropathy (AMSAN) – Common sign of sensory impairment such asfacial movements, speaking, chewing etc.
- Fisher Muller Syndrome (MFS)[7] – Paralysis starts in eyes and associated with unsteady walk which is more common in Asia.

Diagnosis of GBS:

The diagnosis of GBS can be difficult; especially in the first days because many disorders may mimic the delay of treatment for GBS. The different diagnosis factors for GBS are as follows,

Antecedent Event:

In patients with suspected GBS, the diagnosis of GBS is more likely if there is a history of recent (within the previous 6 weeks) diarrhea, Campylobacter infection, respiratory infection, fever or influenza-like illness. Case–control studies showed that GBS is associated with infections (C. Jejuni, cytomegalovirus (CMV), Epstein–Barr virus, Hepatitis E virus, Zika virus), and Mycoplasma pneumonia in children[8].

CSF Examination:

CSF examination is often considered helpful to support the diagnosis of GBS, CSF examination in patients suspected to have GBS shows an increased CSF protein (normal range = 0.18-0.58g/l) and normal CSF white cell count of >50cells/µL should raise suspicion for alternative diagnoses.

Antibody testing:

Serum antibodies against gangliosides and other antigens have been found in GBS, particularly in motor GBS and MFS. The test accuracy varies depending on GBS subtype, tested antigen and control group. Anti-GM1 IgG antibody sensitivity was found in most cases of GBS patients. Anti-GM1 specificity is reported to be high in GBS compared with other neurological diseases. For MFS, sensitivity for anti-GQ1b antibodies is present.

Electromyopathy:

In this type of diagnosis, thin-needle electrodes are inserted in to the muscles to measure nerve activity.

- Sensory and/or motor conduction abnormalities consistent with a poly neuropathy.
- Absent H-reflexes.
- Facial nerve direct responses showing either increased distal motor latency.
- Blink responses either absent or showing prolonged R1and R2 responses and contra lateral R2 response.
- Sural sparing pattern.

Nerve Conduction Studies:

Electrodes are taped to the skin above your nerves. A small shock is passed through the nerve to measure the speed of nerve signals [9].

Treatment of GBS:

Treatment for GBS focuses on managing the symptoms obtained and speeds up recovery, as there is no cure for the condition. The primary treatments involve immunotherapy, with Intravenous Immunoglobulin (IVIG) and Plasma Exchange (plasmapheresis) is the most common and effective treatment choice. Then, supportive care is necessary which including pain management with medications, physiotherapy and prevention of complications like breathing trouble with ventilation.

Intravenous Immunoglobulin (IVIG):

This involves administering healthy antibodies from blood donors to block the harmful antibodies attacking the nerves. IVIG is often the first-line treatment and is commonly used.

Plasma Exchange (Plasmapheresis):

This procedure removes the plasma (liquid part of the blood) and filters out harmful antibodies before returning the blood cells and replacement fluids to the body. It can be an alternative to IVIG, especially in severe case [10].

MULTIPLE SCLEROSIS:

Multiple sclerosis (MS) is an autoimmune inflammatory illness that affects the central

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nervous system (CNS) including the brain, spinal cord, and optic nerves. It is characterized by demyelination and varying degrees of axonal loss. Generally MS is detected between the ages of 20 and 40 years, but less than 1% can occur in childhood and approximately 2-10% after 50 years of age [11].

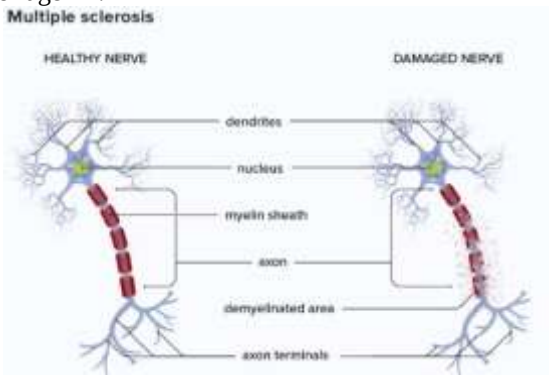

Fig. 2: Damaged nerve on Multiple Sclerosis

Pathogenesis of Multiple Sclerosis:

Pathogenesis of MS occurs in two main steps:

1. Destruction of myelin sheath:

Destruction of myelin sheath followed by lesions formation in the central nervous system (brain and spinal cord): MS primarily involves lesions in the white matter of the visual neuron, basal ganglia, brain stem, and spinal cord. The white matter tracts extremely close to the lateral ventricles may include the lesions. While no lesions form in the peripheral nervous system, white matter cells carry neural signals from the grey matter region where information is gathered throughout the entire body. In MS, oligodendro cytes (the cells that build and maintain the myelin coating of the neuron that transmits neural signals) are damaged, and as a result, the myelin sheath's deterioration has ultimately resulted in the breakage of the nerve axon. No electrical signals could be sent because the myelin sheath was destroyed.

2. Inflammation:

T cells play a significant role in both inflammation and the demyelination process. However, T cells can directly enter the brain through a breach in the blood-brain barrier, where they perceive the myelin sheath as a foreign object and start attacking it. The demyelination of the neuron sheath increases the activation of inflammatory processes, and as a result, immune cells start to release more cytokines and antibodies, further damaging the blood-brain barrier and triggering the activation of macrophages, cytokines, and other detrimental proteins. The increased production of cytokines and antibodies causes complete axon destruction. Together, the two processes work synergistically to damage the neuronal tissue and cause MS. [13]

Symptoms [14]:

Symptoms of MS can be different from person to person. MS can affect any part of the central nervous system which includes,

- Vision problems
- Difficulty walking or keeping balance and thinking clearly
- Numbness or weakness especially in the arms and legs
- Muscle stiffness
- Depression
- Problems with sexual function or urination
- Feeling very tired

Classification [15, 16]:

It can be classified according to progression of disease over time as follows,

1. Progressive-Relapsing MS (PRMS):

It is characterized by continuous decrease in the neurological function since the onset of the disease and also by later superimposed acute attacks. As long as the relapses happen it is impossible to differentiate PPMS and PRMS

2. Secondary progressive MS (SPMS):

Primarily characterized by initial relapses and followed by progressive declination of neurological function totally unrelated to acute attack.

3. Primary Progressive MS (PPMS):

It is characterized by loss of neurological function gradually at the onset of disease. Relapse is not seen.

4. Relapsing-Remitting MS (RRMS):

Almost 85% of MS cases belong to this type. It is characterized by preliminary isolated attacks developing over a period of days to weeks and recovery over a period of weeks to months. No decline in neurological function is experienced by the patient in between episodes.

Diagnosis [17]:

- MRI scans of the brain and spinal cord, which may reveal lesions.

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- Spinal fluid analysis, which may identify antibodies that suggest a previous infection or proteins consistent with a diagnosis of MS
- An Evoked potential test, which measures electrical activity in response to stimuli.

Treatment:

The treatment plan mainly focuses on disease-modifying therapies (DMTs), which have three main objectives such as controlling inflammation, reducing the rate of relapse, and delaying the course of the illness.

These treatments includes injectable drugs like Glatiramer acetate (synthetic polypeptide mixture) and Interferon beta (Immuno- modulator), oral therapy like Fingolimod (Immuno- modulator) and Dimethyl Fumarate , and injection-or infusion-based monoclonal antibodies like Natalizumab (Humanized monoclonal antibody), Ocrelizumab, and Alemtuzumab.[18]

NEURO MYELITIS OPTICA SPECTRUM DISORDER:

Neuro myelitis optica spectrum disorder (NMOSD) also known as Devic's disease is an autoimmune, inflammatory and demyelinating disorder of the central nervous system with a predilection for the optic nerves and spinal cord. NMOSD is caused by a pathogenic serum IgG antibody against the water channel aquoporin 4 (AQP4) in the majority of patients. AQP4-antibody (AQP4-ab) presence is highly specific, and differentiates NMOSD from multiple sclerosis [20]

Neuromyelitis optica spectrum is disease distributed worldwide. It affects preferably adults and females, with a gender ratio of up to 9:14, approximately 5 to 10% of NMOSD cases start before 18 years old [21].

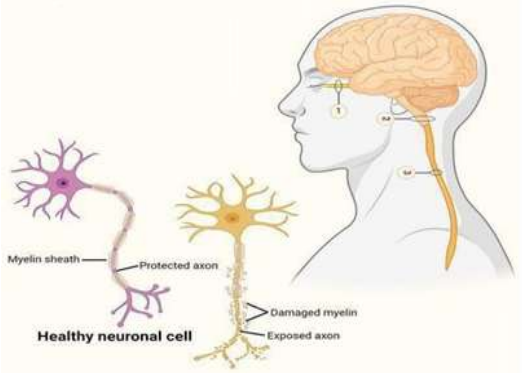

Fig. 3: Site of Damaged Neuronal Cells in Neuromylitis Optica Spectrum Disorder

Pathophysiology [22]

Proposed model for the mechanisms by which AQP4- and MOG-specific antibodies target two different cell types in the CNS, astrocytes (A) and oligodendrocytes (Oligo) respectively. The upper panel shows that MOG- specific effector T cells may initiate CNS inflammation, which is characterized by the accumulation of lymphocytes.

Once entering the CNS, MOG-specific IgG may bind to myelin and myelin-forming oligodendrocytes that express MOG. This binding promotes demyelination and damage to oligodendrocytes or myelin. The lower panel shows that the production of AQP4-specific IgG requires B cells to differentiate into plasma cells expressing AQP4-IgG with the assistance of antigen-specific T-helper cells.

CNS inflammation may be initiated byAQP4-specific effector T cells, which is characterized by the accumulation of neutrophils and eosinophils. Along with complement, AQP4-IgG1 binds to AQP4 water channels (abundantly expressed on astrocytes end-feet processes) and exacerbates astrocytes injury [23].

Symptoms:

Generally, NMOSD and MS show proximity and are misdiagnosed often, as their symptoms look similar. NMOSD is more severe attacks than MS conditions. NMOSD affects the optic nerves and spinal cord, while MS affects other brain regions [24,25].

- CNS lesions
- Trouble walking
- Fatigue
- Loss of color vision
- Intractable hiccups
- Loss of sensation
- Spasticity
- Bladder dysfunction
- Anxiety and depression

Diagnosis:

NMOSD should be suspected in patients who have experienced clinical involvement of at least one of the following structures: optic nerve, spinal cord, area postrema, brainstem, or diencephalon.

Serological diagnosis of NMOSD [26]:

Serological testing should be performed in patients before steroid treatment and plasma exchange. The samples should be secured for

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testing before treatment. In the case of negative serological test results for AQP4-IgG in a patient with typical NMOSD symptoms, the test should be repeated 3–6 months after the first determination.

Neuroimaging diagnosis of NMOSD [27]:

An MRI scan of the spinal cord should be performed in patients with suspected NMOSD before and after contrast administration. This should include at least two segments of the spinal cord (i.e. cervical and thoracic).

- Spinal cord MRI
- Optic nerve MRI
- Cerebral MRI

Treatment Methods of NMOSD

Treatment of the acute phase of NMOSD usually involves intravenous steroids such as methyl-prednisolone, often used in high doses (500-100 mg daily) for 5to 10days. Other studies advocate plasmapheresis treatment (55 mL/kg) and intravenous immunoglobulin.

Chronic immuno suppressive therapy can be accomplished with azathioprine or rituximab as first-line agents. Second-line agents may include mycophenolate or methotrexate. The second- line agents may be advantageous due to their infrequent dosing. Newer biologics are directed against specific immune mediators such as anti-IL-6, anti-complement, or anti-AQP4-IgG.A retrospective multicenter analysis showed reductions in relapse rate with Rituximab, Mycophenolate, Andazathioprine (with Prednisone)of 88.2%, 87.4%, and 72.1% respectively[28].

III. CONCLUSION

Autoimmune is the mechanism where the system fails to recognize its own constituent parts as a ‘self’, which results in an immune response against its own cells and tissues. Any disease that results from such an aberrant immune response is termed an autoimmune disease. An autoimmune disease affecting the nervous system occurs when the body's immune system mistakenly attacks healthy nerve cells in the brain and spinal cord (central nervous system) or peripheral nerves, leading to a range of neurological symptoms depending on the affected area, potentially causing conditions like muscle weakness, numbness, sensory disturbances, cognitive impairment, and even seizures, highlighting the need for specialized medical care due to the complex nature of these disorders and the ongoing research into new treatments.

This article summarizes that autoimmune disease on nervous system such as Guillain barre Syndrome, Multiple sclerosis and Neuromyelitis optica spectrum disorder from diagnosis to treatment. Most of the auto immune diseases are not completely curable because prolonged periods of recovery complicated by behavioral dysfunction and neuronal dysfunction symptoms in the months to years after the diagnosis. Due to lack of knowledge and awareness on autoimmune disease it causes great impact on health of millions of population, on worldwide. The ultimate goal is to prompt the early diagnosis of this autoimmune disease helps to manage symptoms, prevent complications and improve the quality of life.

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