WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

PLANT BASED HERBAL DRUG USED FOR GUILLAIN BARRE SYNDROME

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ABSTRACT:

Guillain-Barré syndrome (GBS) is an acute autoimmune polyradiculoneuropathy affecting 1–2 subjects per 100,000 every year worldwide. It causes, in its classic form, symmetric weakness in the proximal and distal limb muscles with common involvement of the cranial nerves, particularly facial weakness. Respiratory function is compromised in a case in four. Randomized controlled trials have demonstrated the benefit of therapeutic plasma exchange in hastening time to recovery. Intravenous immunoglobulin was subsequently shown to be as efficacious as plasma exchange in adult subjects. In children, few trials have shown the benefit of intravenous immunoglobulin versus supportive care. However, a subsequent trial of a second dose of immunoglobulin in such subjects failed to show improved outcome, while demonstrating a higher risk of thromboembolic side-effects. Every plant part, like leaves, stems, fruits, and roots, is used for its medicinal uses. From which Eranda and Guduchi were used for their anti-inflammatory, nerve repair, and autoimmune suppression. Herbal treatment has demonstrated promise in regulating the immune system and lowering inflammation, especially Guduchi (Tinospora cordifolia) and Eranda (Ricinus communis). Eranda has anti-inflammatory and analgesic qualities, while Guduchi is recognized for its immunomodulatory and neuroprotective activities. The therapeutic potential of these herbs in the treatment of GBS is examined in this review, along with their function in immune modulation and nerve regeneration.

KEY WORDS:plasma exchange, intravenous immunoglobulin, Tinospora cordifolia, Ricinus communis, immunomodulatory, autoimmune suppression, nerve regeneration.

INTRODUCTION:

Guillain–Baré Syndrome, or GBS, is a condition that happens quickly and affects the nerves. It occurs when the body's own immune system mistakenly attacks the nerves. This leads to sudden muscle weakness and even paralysis. GBS is a significant cause of these symptoms around the world [1]. GBS, known as Guillain-Barré Syndrome, has four main types. The first type is acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Then, there are axonal types: acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN). The last type is Miller Fisher syndrome, which mainly affects how the eyes move, balance, and reflexes. In North America and Europe, about 5% of people with GBS have axonal types. But in Central and South America, Japan, and China, these axonal types represent 30–47% of cases. Miller Fisher syndrome appears in about 5% of all GBS cases [2]. Two prominent herbs used in Ayurvedic medicine are Guduchi (Tinospora cordifolia) and Eranda (Ricinus communis). Eranda, also called castor, is useful in treating conditions related to pain and inflammation because of its analgesic and anti-inflammatory qualities. Guduchi is well known for having neuroprotective and immunomodulatory properties. These characteristics imply that Eranda and Guduchi may have therapeutic uses in the supportive treatment of GBS, especially in reducing inflammatory symptoms and regulating immune responses.

ZIKA VIRUS:

The Zika virus (ZIKV) is a flavivirus that is mainly spread by Aedes species mosquitoes, particularly Aedes aegypti and Aedes albopictus. Since its discovery in 1947 in Uganda's Zika Forest, ZIKV has been connected to serious health issues, particularly because of its link to neurological conditions and birth defects. Zika virus transmitted through blood transfusion, sexual transmission, maternal-fetal transfusion [3]. Prenatal ZIKV infection is linked to congenital Zika syndrome, which includes a variety of birth abnormalities like microcephaly. Furthermore, in adults, ZIKV has been connected to neurological issues such as Guillain-Barré syndrome. ZIKV is an icosahedral-symmetric, enveloped virus with a non-segmented, single-stranded, positive-sense RNA genome that is roughly 10 kilobases long. Three structural proteins (capsid, membrane, and envelope) and seven non-structural proteins are encoded by the viral genome. By attaching to receptors and starting endocytosis, the envelope glycoprotein makes it easier for host cells to enter. ZIKV has two main lineages: Asian and African. The strain that is causing the outbreaks in the Americas shares a great deal of genetic similarity

with the Asian lineage. The immune system may mistakenly target the host's nerves because ZIKV shares antigenic epitopes with peripheral nerve components. Furthermore, ZIKV can directly infect neural tissues due to its neurotropic properties, which may aid in neuropathogenesis [4]. Congenital Zika syndrome and neurological complications may result from ZIKV's capacity to cross the placental and blood-brain barriers due to its distinct structural characteristics, especially in the envelope glycoprotein [5].

STRUCTURE OF ZIKA VIRUS:

ZIKV particle is icosahedral symmetric and has a diameter of about 50 nanometers. During viral budding, its lipid bilayer envelope is formed from the membrane of the host cell. This envelope contains 180 copies of the membrane (M) and envelope (E) proteins, which are arranged into 90 dimers to create a smooth, herringbone-like surface. Flaviviruses are known for this configuration, which is essential for host cell recognition and entry. The main antigenic determinant that promotes viral attachment and fusion with host cells is the E protein. There are three domains in it: The central β-barrel structure that acts as a hinge between the other domains is known as Domain I (DI). The fusion loop, a hydrophobic area necessary for membrane fusion during viral entry, is found in Domain II (DII). Domain III (DIII): A domain that resembles immunoglobulin and is involved in receptor binding. Interestingly, ZIKV's E protein contains a glycosylation site at Asn154 that could affect the virus's immune evasion and tropism. The main antigenic determinant, protein, promotes viral fusion and attachment to host cells. The precursor prM protein, which is cleaved during viral maturation, is the source of the M protein. The M protein helps to preserve the structural integrity of the virus by stabilizing the E protein in the mature virion. The nucleocapsid, which is made up of the C protein and contains the viral RNA genome, is located beneath the lipid envelope. Because of its abundance of basic amino acids, the C protein can interact with the negatively charged RNA more easily. The genome of ZIKV is a positive-sense, single-stranded RNA that is roughly 10.7 kilobases long. Three structural proteins (C, prM/M, and E) and seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5) are produced by co- and post-translational cleavage of the single polyprotein it encodes. These non-structural proteins play a role in host modulation, viral assembly, and replication [6].

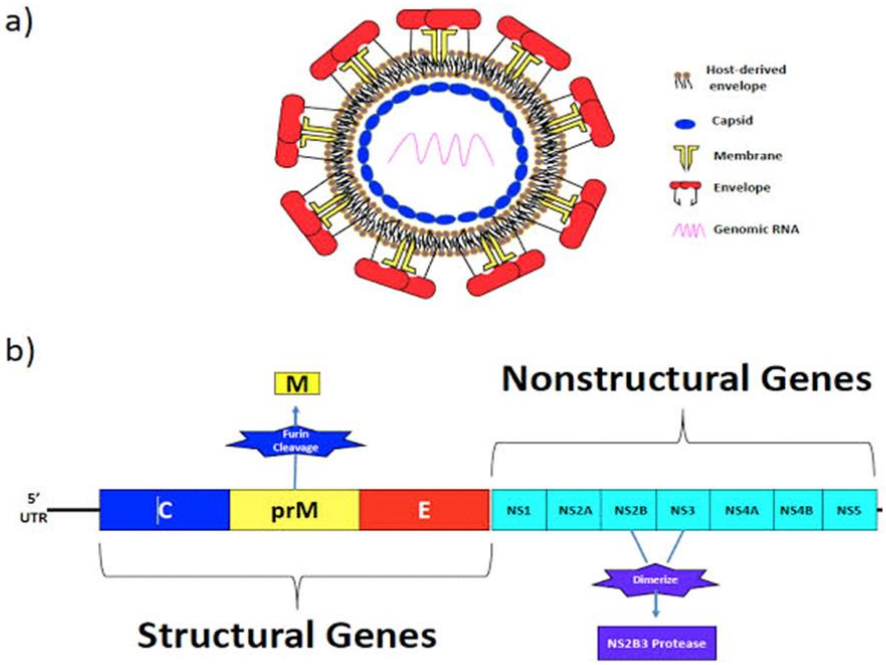


FIG 1: STRUCTURE OF ZIKA VIRUS

SIGNS AND SYMPTOMS:

Different levels of muscle weakness and, in extreme situations, paralysis can result from GBS. Early signs and symptoms such as tingling or "pins and needles" sensations in the hands and feet are frequently the first symptoms, muscle weakness usually beginning in the lower limbs, this condition can spread upward to the arms and face muscles. Ascending Paralysis, the weakness starts in the legs and moves up to the arms and upper body. Common symptoms include diminished or nonexistent reflexes, like knee jerks. Difficulty walking or maintaining balance due to muscle weakness. severe symptoms like Respiratory Issues, Ventilator support may be required if respiratory muscles are weak. Weakness in the facial muscles can cause problems with swallowing, chewing, and making facial expressions. Autonomic Dysfunction, Problems controlling the bladder and bowels, as well as abnormalities in blood pressure and heart rate. The severity of the symptoms may increase in a matter of hours, days, or even weeks. Effective management of the condition depends on early detection and timely medical intervention [7].

STATISTICAL DATA OF GBS DISEASE ACROSS SOUTHERN INDIA:

Majority of the patients were in the age group 21 - 30 Yrs i.e., 38.4% and with increasing age the prevalence of cases decreased. Male preponderance (73.78%) of GBS was seen [8].

Characteristics	Frequency	Percentage
Age group		
13-20 yrs	25	15.4%
21-30 yrs	63	38.4%
31-40 yrs	40	24.3%
41-50 yrs	23	14%
51-60 yrs	11	6.7%
> 60 yrs	2	1.2%
Gender		
Male	121	73.78%
Female	43	26.22%

Table1: Demographic distribution of study participant

PATHOGENESIS OF GBS:

Globally, GBS is the leading cause of flaccid paralysis. Globally, the prevalence of GBS is still rising, and the condition's occurrence varies by geography, sex, exposure to various infections, side effects from vaccinations, and genetic susceptibilities that exist globally. When the arrival of infectious or viral agent in the body, the initiation of immune response takes place. At this stage, involvement of inflammatory and immune mediators to myelin sheaths occurs. The vesicular layer around the Schwann cell was degenerated. Finally, the complete loss of the myelin sheath that surrounds nerve fibers occurs. It causes an enhanced immunological and inflammatory response that gradually and quickly damages the myelin or axon of the body's peripheral nerves [9].

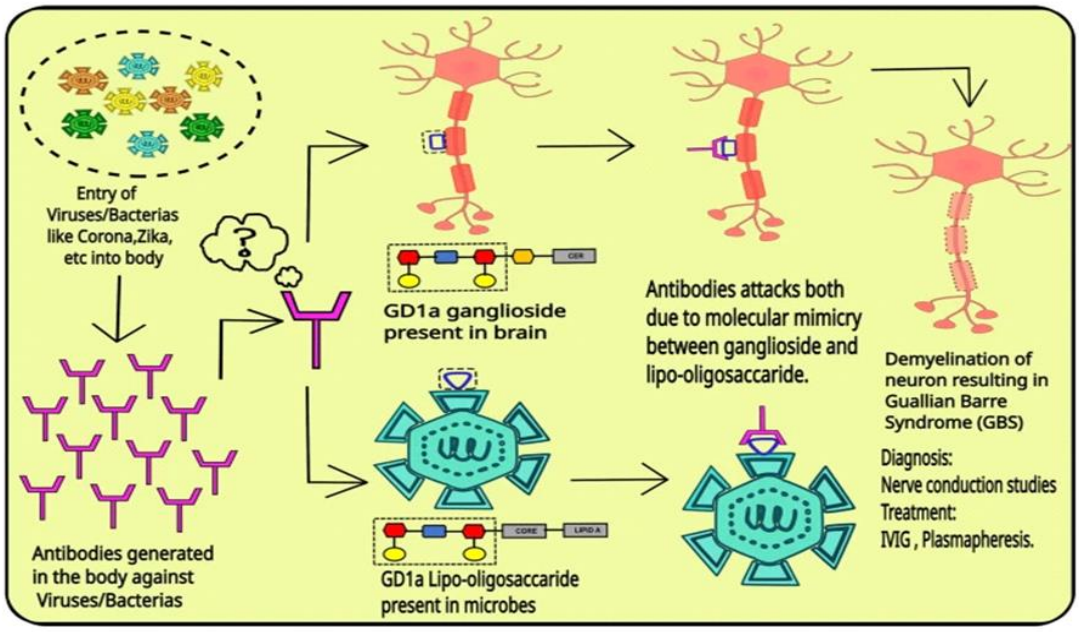


FIG 2: PATHOGENESIS OF GBS

MATHEMATICAL MODEL OF GBS:

GBS is a condition where the immune system mistakenly attacks the myelin sheath of peripheral nerves, leading to demyelination. The mathematical model of Guillain-Barré Syndrome (GBS) focuses on the rate of demyelination of neurons over time. It is expressed as,

$dM/dt = k_1 A - k_2 M$

Where, M= Rate at which myelin degrades

A= potency of autoimmune response

k_1 = Rate at which immune cells attack

k_2 = Rate of myelin repair.

The myelin sheath, a layer that envelops nerve fibers, degrades over time, as the equation illustrates. Accordingly, the expression for the rate of myelin breakdown over time is dM/dt. While the rate of myelin repair on the rate of degradation is expressed as (k2M), the degradation is essentially caused by the immune cell attack rate to the potential of an autoimmune cell, which is represented as (k1A). According to this, nerve injury is caused by immune cells attacking at a higher rate. GBS causes severe nerve damage and weakness in the muscles. GBS must be cured by reducing (immune attack reduction). Boost (myelin repair development). [10]

CONVENTIONAL TREATMENT AGAINST GBS:

Conventional therapies like plasma exchange and intravenous immunoglobulin (IVIg) are the main therapies. Plasma exchange may not be appropriate for patients with coagulopathies or those who are susceptible to bleeding complications. A history of GBS within six weeks of receiving an influenza vaccination is regarded as a preventative measure for subsequent vaccinations. In these situations, healthcare professionals may choose to delay or avoid influenza vaccination, particularly if the patient is at a high risk of developing influenza-related complications. Likewise, it is a precaution for future administration of tetanus toxoid-containing vaccines if a person has developed GBS within six weeks of receiving the vaccine. Pooled immunoglobulins from healthy donors are administered as part of IVIg, which lowers nerve inflammation by modifying the immune response. Clinical research has shown that IVIg can speed up recovery for GBS patients just as well as plasma exchange. IVIg is usually given over five days. Headaches, chills, and, in rare cases, aseptic meningitis or thromboembolic events are possible side effects. Plasmapheresis or plasma exchange is one the conventional treatment, by substituting donor plasma or a plasma substitute for the patient's blood that contains dangerous antibodies, this procedure lessens immune-mediated nerve damage. It has been demonstrated that plasma exchange helps GBS patients recover more quickly and require less mechanical ventilation. Over the course of two weeks, there are five exchanges in the typical regimen. Hypotension, infections, and complications from bleeding are among the risks [11].

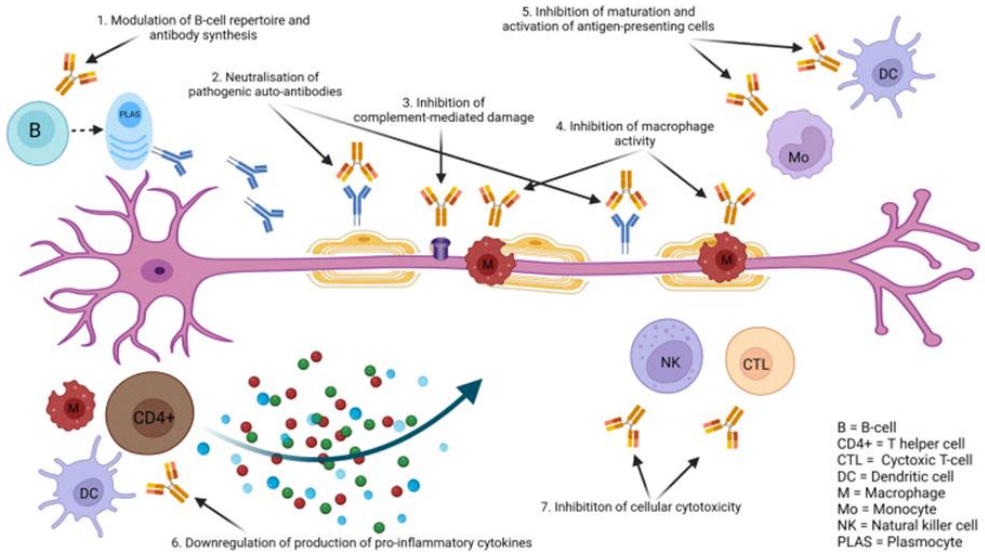


FIG 3: CONVENTIONAL TREATMENT AGAINST GBS

ROLE OF PLANT BASED HERBAL DRUG AGAINST GBS:

ERANDA (RICINUS COMMUNIS):

Ricinus communis, also known as the castor oil plant, is a member of the Euphorbiaceae spurge family [12]. The roots of the Eranda are also used in several formulations for the treatment of rheumatic and neurological disorders. Traditionally, Ricinus communis, also referred to as Eranda or the castor oil plant, has been utilized for its therapeutic qualities, especially its ability to reduce inflammation. The active ingredients in Eranda include triterpenes, flavonoids (such as quercetin and rutin), alkaloids that contain ricinine, and 85–90% ricinoleic acid, which serves primarily for its anti-inflammatory properties. Extractions from this plant have been found to modulate inflammatory pathways through the inhibition of important cytokines, including the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) signaling pathway, interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). The NF-κB pathway and cytokines such as TNF-α and IL-6 are important contributors to the inflammatory processes linked to GBS. IL-6 and TNF-α are pro-inflammatory cytokines that are increased in GBS patients and lead to nerve damage and inflammation. The transcription factor known as the NF-κB pathway controls the expression of several inflammatory cytokines, such as TNF-α and IL-6. An increased inflammatory response brought on by NF-κB activation is linked to the pathophysiology of GBS [13].

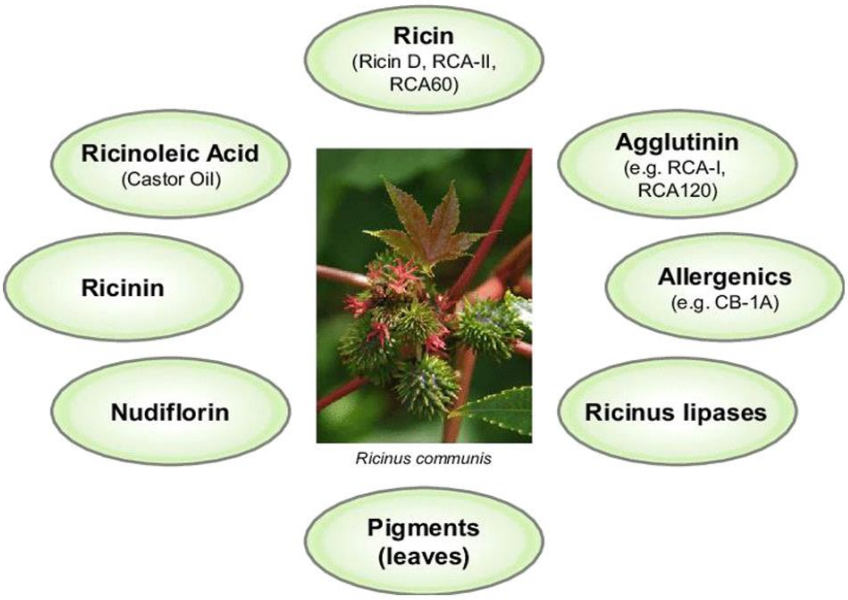


FIG 4: ACTIVE COMPONENTS OF ERANDA

MATHEMATICAL MODEL OF ERANDA:

The anti-inflammatory qualities of Eranda may have an impact on cytokine levels. In order to mitigate inflammation, Eranda could effectively lower cytokine levels by increasing the suppression coefficient. The mathematical model demonstrates the concentration of cytokine in presence of Eranda as,

$dC/dt = k_3 - k_4 E$

Where C= concentration of cytokine

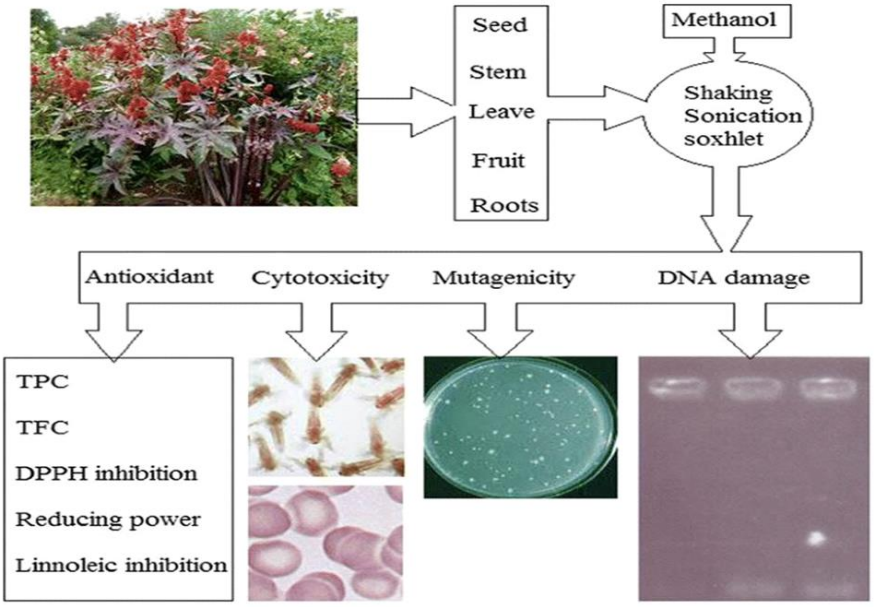
E= effect of Eranda (anti-inflammatory factor)

k_3= rate of cytokine production

k_4= Eranda suppression coefficient

The equation illustrates that concentration of cytokine produced over time which is inhibited by Eranda. Consequently, the cytokine production concentration over time is expressed as dC/dt. The anti-inflammatory factor (Eranda) required is represented by E. The innate rate at which the body produces cytokines, frequently in response to inflammatory stimuli, is represented by the symbol k3. Eranda's anti-inflammatory effect causes cytokine levels to drop at a rate indicated by k4 E. In this case, EE stands for Eranda's concentration or effect level, and k4k4 measures the extent to which Eranda suppresses cytokine production. Eranda increases K4, decreases cytokine levels, and lessens inflammation and nerve damage [14].

FIG 5: EXTRACTION & USES OF ERANDA



REGENERATION OF MYELIN:

Eranda is used in the remyelination process, which is essential for regaining nerve function adhering to damage. Schwann cells, which form the myelin sheath in the peripheral nervous system, proliferate and differentiate during this process. Eranda (anti-inflammatory qualities) could foster a condition that is favorable for nerve healing. Reducing inflammation may indirectly promote Schwann cell function and myelin regeneration because chronic inflammation can obstruct the remyelination process. In order to comprehend the dynamics of myelin regeneration, the remyelination process can be mathematically described as,

$R=S\cdot(1-e^{-k_5 t})$

Where, R= regenerated myelin

S= Schwann cell (activation factor)

k_5= growth rate constant

Through this, remyelination process was expressed by R. The regeneration rate of the Schwann cell was expressed by S. Over time, as Schwann cells activate and multiply, the amount of regenerated myelin increase. The rate of remyelination and extent are influenced by the parameters S and k_5 [15].

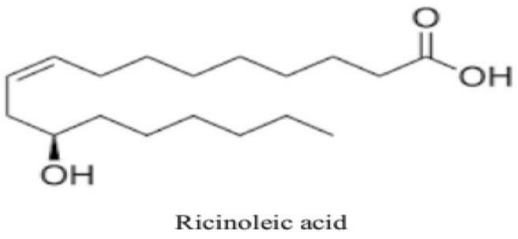
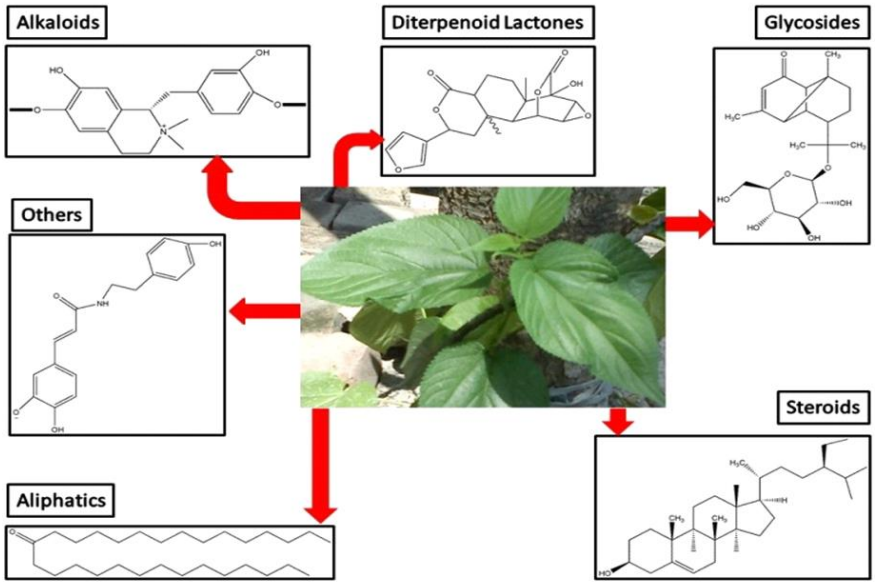


FIG 6: STRUCTURE OF RICINOLEIC ACID

GUDUCHI (TINOSPORA CORDIFOLIA):

Tinospora cordifolia, also referred to as Guduchi or giloy, is a member of the Menispermaceae family. Tinospora cordifolia (Giloy) has a wide range of bioactive substances, all of which support its medicinal properties. Alkaloids, glycosides, steroids, flavonoids, and polysaccharides are the main groups of compounds. Among these, the alkaloids berberine and magnoflorine stand out due to their antimicrobial and anti-inflammatory properties [16]. Glycosides such as cordifolioside A and syringin have been linked to the plant's immunomodulatory effects, making them central to its use in traditional medicine. Guduchi has shown promise in regulating the immune system, which may help to balance the hyperactive immune responses seen in autoimmune diseases. According to a thorough analysis, it has the 0 ability to modulate both innate and adaptive immunity by increasing the activity of natural killer cells, macrophages, and cytokine production. This herb has strong anti-inflammatory properties, which may be helpful in diseases like GBS where inflammation is a major factor. Research has indicated that Tinospora cordifolia has anti- inflammatory properties due to the presence of bioactive substances like glycosides, steroids, and alkaloid [17]. It has been shown in studies to boost cytokine production, activate macrophages, and improve the body's immune response in general. T. cordifolia's polysaccharides boost phagocytosis and activate white blood cells, bolstering immunity and fostering resistance to infections. Furthermore, bioactive substances like glycosides and alkaloids enhance its immunostimulatory effects by modifying cell-mediated immune responses and stimulating lymphocytes, indicating possible uses in immunocompromised environments [18].

FIG 7: ACTIVE COMPONENTS OF GUDUCHI



AUTOIMMUNE SUPPRESSION MODEL:

Guduchi is well known for its immunomodulatory qualities, particularly in controlling B-cell activation and T-regulatory (T-reg) cells. These processes are essential for preserving immunological homeostasis and averting autoimmune reactions. Guduchi can lessen unwarranted B-cell activation and stop the production of autoantibodies that could harm the body's own tissues by controlling cytokine production and signaling pathways. The rate of immune over action over the can be expressed as,

$dI/dt = -k_6 \cdot G + k_7$

Where, I = level of immune overreaction, particularly excessive B-cell activation.

G = Guduchi intake, acting as an immunomodulatory factor.

K_6 = rate of Guduchi-induced immune suppression.

K_7 = beginning of immune factor.

The immune overreaction is reduced more significantly when Guduchi intake (G) is increased because this results in a higher value of the term $-k_6 \cdot G$. By reducing excessive B-cell activation, Guduchi's immunomodulatory action preserves immunological balance and averts possible autoimmune attacks on nerve tissues. Tinospora Cordifolia's immunomodulatory activity demonstrated its capacity to stimulate macrophages and increase nitric oxide production, suggesting a function in regulating immune responses. Consuming Guduchi raises the immune suppression rate, which reduces the immune overreaction (averts an excessive nerve attack) [19].

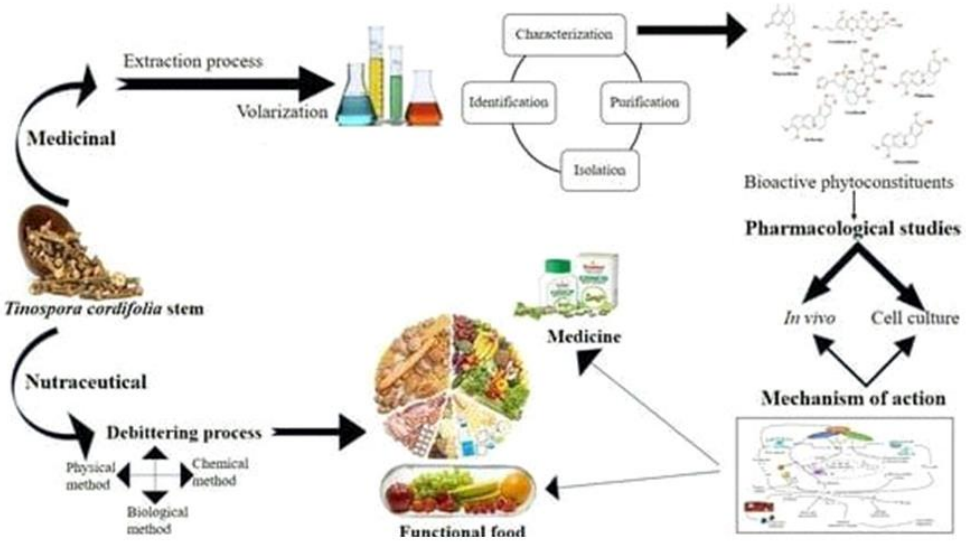


FIG 8: EXTRACTION OF GUDUCHI

REMYELINATION PATHWAY:

Due to its neuroprotective and neuroregenerative qualities, Guduchi is highly valued in traditional medicine. by increasing the expression of neurotrophic factors like Brain-Derived Neurotrophic Factor (BDNF) and Nerve Growth Factor (NGF), it may aid in nerve repair [20]. NGF, or nerve growth factor, is essential for the survival, upkeep, and regeneration of neurons. Nerve repair processes can be aided by substances that increase NGF levels. BDNF, or brain-derived neurotrophic factor, is essential for neuroplasticity and the healing process after neural injury. It promotes the survival of existing neurons and the development and differentiation of new neurons and synapses. The process of remyelination can be facilitated by Guduchi was expressed as,

$M = M_0 \cdot e k_8 t$

Where, M= refers to myelin growth.

M_0= denotes initial myelin level.

K_8 = Guduchi induced myelin growth rate.

t = signifies time.

Guduchi's elevation of NGF and BDNF levels causes an increase in the rate constant k_8 in this model, which over time accelerates myelin regrowth and aids in nerve recovery. In primary cerebellar neuronal cultures, Tinospora cordifolia's neuroprotective and neuroregenerative potential against glutamate-induced excitotoxicity revealed that Guduchi extract enhanced cerebellar neuron migration, regeneration, and plasticity. These results imply that Guduchi may stimulate neurotrophic factor expression, which would aid in myelin regeneration and neuronal repair [21].

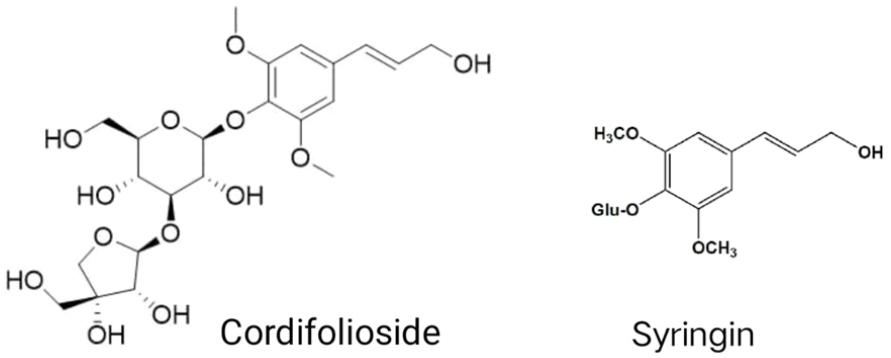


FIG 9: STRUCTURE OF CORDIFOLIOSIDE AND SYRINGIN

COMBINED THERAPEUTIC EFFECT OF GRANDA AND GUDUCHI ON GBS:

The pathophysiology includes impaired nerve regeneration and demyelination brought on by inflammation. There may be synergistic benefits to treating GBS by incorporating traditional medicinal plants like Guduchi (Tinospora cordifolia) and Eranda (Ricinus communis) into medical regimens. Together, Eranda's anti-inflammatory qualities and Guduchi's capacity to regulate immune responses may lessen the abnormal immune activity that GBS patients experience that attacks peripheral nerves. Immune homeostasis may be restored and additional nerve damage may be avoided by means of guduchi's stimulation of immune responses (22). The combined therapeutic effect of Eranda and Guduchi on GBS can expressed by the healing factor over time as,

$H(t) = (k_4 \cdot E + k_6 \cdot G) / k_1 \cdot e\,(k_5 + k_8) \cdot t$

Where, H(t) = represents healing factor over time.

k_4 and k_6= illustrating anti-inflammatory and immunosuppressive properties of Eranda and Guduchi.

k_5 and k_8 = denotes myelin repair and nerve regrowth facilitated by Eranda and Guduchi.

E and G = denote the dosages of Eranda and Guduchi.

k_1 = related to disease severity and progression rate.

Guduchi and Eranda have anti-inflammatory qualities. Improving k4 and k6 with the right dosage of these herbs may relieve nerve damage by lowering inflammation and regulating immune function. Eranda and Guduchi's ability to support myelin repair and nerve regeneration is represented by the numbers k_5 and k_8. These herbs may improve remyelination and functional recovery by promoting Schwann cell proliferation and stimulating neurotrophic factors. The core severity and rate of progression of GBS is represented as k_1. A milder disease course is indicated by a lower k_1 value, which can be impacted by early detection and efficient management techniques. Eranda and Guduchi dosages that are specifically designed to maximize therapeutic effects (k_4, k_6, k_5, and k_8) while minimizing disease severity (k_1) could accelerate GBS patients recovery. By addressing the disease's neurological and immunological components, a comprehensive approach to managing GBS may be possible by combining these herbal interventions with traditional therapies [23].

CONTRAINDICATION:

On December 26, 2018, the patient was admitted to the IPD, and treatment began immediately thereafter. Ajmodadi + Gokshur Churna 3GM B.D., Sunthi Siddha Eranda Tail 10 mL B.D., Sunthi and Aamlaki Churna 3GM BD, and 150 mg of Agnitundi vati were given for five days. Shastika Shali Pinda Sweda and Lasunadi Vati 150 mg B.D. were introduced on the seventh day. Still, the symptoms grew worse. Lasunadi Vati was therefore terminated because it raised Pitta Dosha. The 30-day course of medicinal enema (Basti) was started with 750 mL of Erandmuladi herb decoction and 70 mL of sesame oil. The patient continued to complain of weakness, and nothing changed. Taking into account Vatakaphahar Chikitsa, the treatment was completed until January 10, 2018. The treatment plan was modified in accordance with Vata Pittahar Chikitsa as the patient's symptoms showed no signs of improvement.

With the exception of Pinda Sweda, all prior therapies were stopped. Pitta Dosha is raised by Erandmuladi Basti and Lasunadi Vati. It is therefore contraindicated in GBS. The medication prescribed by Nakanekar et al and Manspachak Vati 250 mg twice daily were initiated. It involves rectal administration of milk processed with pacifying Pitta Dosha [(Pittaghna Gana Siddha Basti: Sariva (Hemidesmus indicus L.), Manjistha (Rubia cordifolia L.), Yashtimadhu (Glycyrrhia glabra L.)] for 30 days along with Shirodhara (Tila + Brahmi Tail), and Bruhatvatchintamani Kalpa (a combination of Bruhatvatchintamani Ras 10 tab +Rajat Bhasma 5 gm +Guduchi Satva 30 gm + Sutshekharras 10 tab. [24]

CONCLUSION:

Acute immune-mediated polyneuropathy is an indication of Guillain-Barré Syndrome (GBS), a dangerous neurological condition that causes muscle weakness and, in extreme situations, paralysis. Guduchi (Tinospora cordifolia) and Eranda (Ricinus communis) are valued for their immunomodulatory and anti-inflammatory qualities in Ayurvedic medicine, respectively. These characteristics highlight possible advantages in the treatment of autoimmune diseases like GBS. Thorough scientific validation is required before incorporating these Ayurvedic herbs into GBS treatment regimens. Clinical studies evaluating Eranda and Guduchi's safety and effectiveness specifically for GBS patients are currently lacking. To guarantee safe and well-coordinated care, people thinking about implementing such herbal interventions must speak with medical professionals. In order to improve Eranda's efficacy and safety profile, future research will focus on comprehending its bioactive components and creating targeted delivery formulations. In order to improve

guduchi's effectiveness in integrative and preventive healthcare, future research will concentrate on increasing its bioavailability using techniques like nanoparticle encapsulation, examining synergistic effects with other medicinal herbs, and carrying out comprehensive clinical trials. Furthermore, studies are looking into how it might improve immune responses and manage autoimmune diseases. By increasing their bioavailability, comprehending their mechanisms of action, and incorporating them into contemporary medical procedures using evidence-based methods, current and upcoming research attempts to fully realize these potentials.

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