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The Concept of Guillain–Barré Syndrome (GBS) in Unani Medicine and the Potential Role of Unani Pharmacology in Its Management

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Abstract

Introduction: *Guillain–Barré Syndrome (GBS) is an autoimmune neurological disorder characterized by progressive muscle weakness and paralysis due to immune-mediated damage to the peripheral nervous system. Conventional treatments focus on immunotherapy and supportive care, but alternative approaches, such as Unani Medicine offer promising holistic management strategies. Unani Medicine attributes GBS to humoral imbalance, particularly an excess of Balgham (Phlegm) affecting nerve function, leading to weakness and dysfunction.* ***Objective:*** *This study aims to explore the concept of GBS in Unani Medicine and evaluate the potential role of Unani pharmacology, including single and compound drugs, antioxidants, and neuroprotective agents, in its management.* ***Methodology:*** *A literature survey of classical Unani texts was conducted to identify single and compound Unani formulations relevant to GBS management. Electronic databases, such as PubMed, ScienceDirect, Wiley Online Library, and Google Scholar were explored to collect scientific evidence on the effectiveness of the recommended Unani medicines for GBS treatment. Additionally, an extensive review of both classical Unani literature and modern research was conducted to analyze the connection between Unani concepts of nerve disorders and their possible role in GBS management.* ***Conclusion:*** *Unani Medicine offers a holistic and personalized approach to GBS management through herbal therapies. However, additional clinical studies and trials are required to confirm its effectiveness and safety. Integrating Unani pharmacology with modern medical research can provide novel adjunctive therapies for GBS, improving patient outcomes in a comprehensive manner.*

Keywords: Guillain–Barré Syndrome, neurological disorder, Unani medicine, Amraz asab, GBS treatment

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INTRODUCTION

Guillain–Barré Syndrome (GBS), also called Acute Polyradiculoneuritis [1] is a rare neurological disorder that affects the peripheral nervous system, impairing nerve function outside the brain and spinal cord. It encompasses a range of immune-mediated disorders targeting the peripheral nervous system, with an estimated median incidence of 1.1 cases per 100,000 person-years [2].

Research suggests that the likelihood of developing GBS increases with age, with the relative risk rising by approximately 20% per decade, peaking between 70 and 80 years [3]. Furthermore, studies indicate that males are at a higher risk than females, with an estimated relative risk of 1.78 [3].

GBS usually occurs after an infection, where the body's immune system mistakenly attacks the protective layer of the nerves (myelin). This damage disrupts nerve signals, leading to muscle weakness, numbness, and, in severe cases, paralysis. The condition often begins with tingling and weakness in the legs, which may gradually spread to the arms and upper body. In some cases, the facial nerve (7th cranial nerve) is affected, causing difficulty in facial movements.

The condition can progress rapidly, and in severe cases, it may cause breathing problems, requiring ventilator support. Although GBS can be challenging to diagnose due to its varying symptoms, early recognition is important for effective management. Most patients recover with proper treatment, but some may experience long-term weakness, and a small percentage may not survive [1, 2].

Treatment mainly focuses on supportive care, including physiotherapy to restore movement, plasma exchange to remove harmful antibodies, and intravenous immunoglobulin (IVIG) therapy to regulate the immune response. With timely medical intervention, many patients regain normal function over time. The Process of myelin sheath to demyelination show in Figure 1.

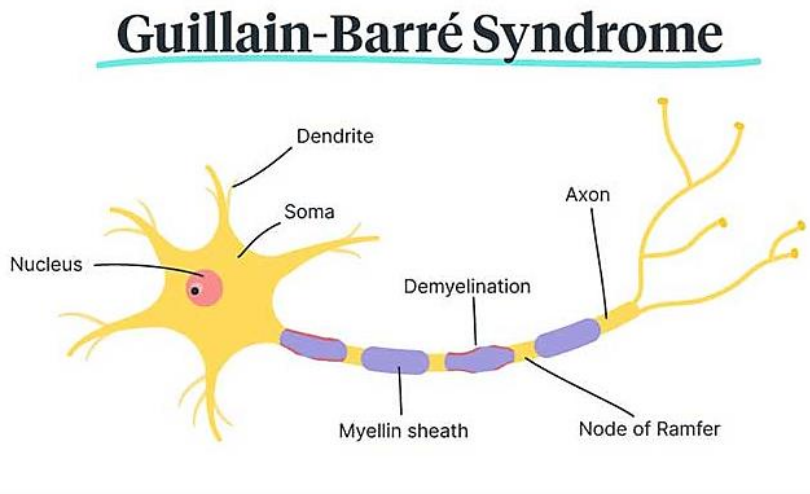

Figure 1. Process of myelin sheath to demyelination.

MODERN ETIOLOGY

In approximately 60% of Guillain–Barré Syndrome (GBS) cases, symptoms develop within 1 to 3 weeks following a mild respiratory or gastrointestinal infection or, in some instances, vaccination. The most identified causes are respiratory infections, followed by gastrointestinal infections. While many cases stem from common viral illnesses, nearly any febrile infection or immunization has been associated with the onset of GBS [1].

Campylobacter jejuni is the leading bacterial cause, detected in as many as 32% of cases. Interestingly, not all individuals with *C. jejuni*-related GBS experience digestive symptoms; in about 30% of cases, only blood tests confirm previous infection. Patients with *C. jejuni*-induced GBS often experience a more severe and rapidly progressing form of the illness, marked by high levels of anti-GM1 antibodies and a longer recovery period. This bacterial infection is strongly linked to the motor and axonal variants of GBS [1, 2].

Viral infections linked to GBS include Cytomegalovirus (CMV) and Epstein-Barr virus (EBV). CMV, the second most frequently reported cause, is detected in up to 15% of cases and primarily affects younger individuals. It can lead to serious complications, including respiratory failure, sensory disturbances, and cranial nerve dysfunction, often with elevated anti-GM2 antibodies. EBV infections precede GBS in about 10% of cases and are often linked to conditions like mononucleosis, hepatitis, or throat infections. Additionally, GBS has been observed in individuals undergoing HIV seroconversion.

Other infectious agents linked to GBS include *Mycoplasma pneumoniae*, *Haemophilus influenzae*, Lyme disease, para-influenza viruses, influenza A and B, adenoviruses, and herpes simplex virus [2, 3].

Non-infectious triggers have also been identified, including autoimmune disorders, such as systemic lupus erythematosus (SLE), certain cancers like Hodgkin's disease and lung cancer, thyroid disorders, paraproteinemia, sarcoidosis, surgical procedures, physical trauma, and the postpartum period. Older rabies vaccines derived from nervous system tissue have been implicated in cases of GBS, particularly in developing countries. Additionally, the A New Jersey (swine flu) vaccine was linked to a slight increase in GBS cases. While reports of GBS following other vaccinations exist, such occurrences are rare and inconsistent.

PATHOPHYSIOLOGY GBS

Guillain–Barré Syndrome (GBS) is a post-infectious immune disorder in which both cellular and humoral immune responses contribute to nerve damage. The most common variant, Acute Inflammatory Demyelinating Polyneuropathy (AIDP), occurs when an abnormal immune reaction causes inflammation and deterioration of the myelin sheath that insulates peripheral nerves [2, 3]. Elevated levels of cytokines, such as interleukin (IL)-2 and soluble IL-2 receptor, have been detected in the blood and cerebrospinal fluid of affected individuals, indicating immune system activation.

Most individuals experience an infection in the weeks prior to developing GBS. Various infections are believed to trigger the production of antibodies that mistakenly target gangliosides and glycolipids – key components of myelin in the peripheral nervous system. One of the most well-documented infections associated with GBS is *Campylobacter jejuni*. This bacterium has surface antigens structurally like those found in nerve tissue, causing the immune system to attack myelin through a process called molecular mimicry. In *C*. jejuni-associated GBS, ganglioside GM1 cross-reacts with bacterial lipopolysaccharide antigens, leading to immune-mediated nerve damage [3].

Pathological studies of AIDP have shown that immune cells infiltrate the blood vessels within nerves, followed by macrophage-driven destruction of myelin. This results in nerve conduction block, disrupting electrical signal transmission and leading to muscle weakness or paralysis [4–6]. In severe cases, prolonged inflammation can damage the underlying axons, resulting in slower recovery. Some patients develop a direct immune attack on nerve axons rather than myelin, producing similar clinical symptoms. GBS can affect nerves at various levels, from the spinal roots to smaller motor nerves within muscles. However, the most severe damage typically occurs in the ventral roots, proximal spinal nerves, and cranial nerves, leading to significant motor dysfunction (Figure 2) [4].

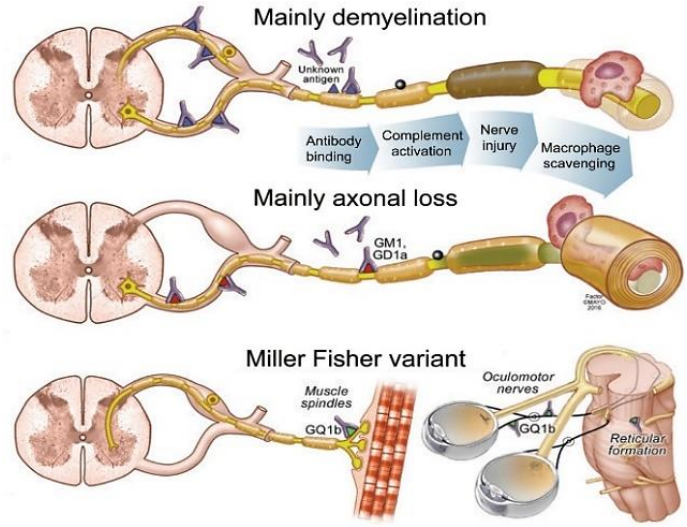

Figure 2. Pathophysiology of Guillain–Barre Syndrome (GBS).

CLINICAL MANIFESTATIONS

Guillain–Barré Syndrome (GBS) is an acute neurological disorder characterized by rapidly progressing muscle weakness, loss of reflexes (areflexia), and possible sensory disturbances. The disease typically follows an ascending pattern of paralysis, beginning in the legs and progressing upwards [4–6].

Motor Symptoms

- *Ascending Paralysis:* Weakness usually starts in the legs, described as a sensation of "rubbery legs," and spreads to the arms and upper body.
- *Progression of Weakness:* Symptoms develop over a few hours to several days. In severe cases (~5% of patients), the paralysis may progress to total motor failure and respiratory failure within a few days.

Facial and Cranial Nerve Involvement

- Facial weakness (diparesis) is observed in 50% of patients.
- Bulbar weakness (affecting the lower cranial nerves) may cause difficulty swallowing (dysphagia) and handling secretions, leading to airway obstruction.
- Ophthalmoparesis (eye movement abnormalities) occurs in up to 25% of cases, most commonly due to cranial nerve VI palsy (leading to limited lateral eye movements). Ptosis (drooping eyelids) may also occur.
- *Respiratory Involvement:* Weakness of the diaphragm and respiratory muscles may require mechanical ventilation in 30% of cases [5].

Sensory Symptoms

- Tingling or abnormal sensations (dysesthesias) in the extremities often accompany weakness.
- Mild cutaneous sensory deficits (loss of pain and temperature sensation) are common.
- Large sensory fibre involvement affects deep tendon reflexes (DTRs) and proprioception (position sense) more severely.
- Areflexia (loss of reflexes) develops within the first few days of the disease.

Pain and Early Symptoms

- Neck, shoulder, and back pain is common, occurring in ~50% of cases.
- Pain may be mistaken for lumbar disc disease, back strain, or orthopaedic disorders.

AUTONOMIC DYSFUNCTION (OCCURS IN ~65% OF HOSPITALIZED PATIENTS)

Decreased Sympathetic Activity

- Orthostatic hypotension (drop in blood pressure when standing).
- Anhidrosis (reduced sweating).
- Iridoplegia (sluggish pupil response).

Increased Sympathetic Activity

- Hypertension (high blood pressure).
- Tachycardia (rapid heart rate).
- Tachyarrhythmias (abnormal heart rhythms).
- Episodic diaphoresis (excessive sweating).
- Acral vasoconstriction (reduced blood flow to extremities).

Vagal Overactivity

- Bradycardia (slow heart rate).
- Heart block.
- Asystole (cardiac arrest) → Requires immediate medical attention.

Bladder and Bowel Dysfunction

- Urinary retention occurs in ~15% of cases, usually resolving within a few days.
- Persistent bladder dysfunction early in the disease should prompt evaluation for alternative diagnoses (e.g., spinal cord disease).

Disease Progression and Recovery

- Peak Weakness: Most patients reach maximum weakness within 4 weeks, after which further progression is rare.
- Recovery Phase: Begins 2–4 weeks after the disease stabilizes, but full neurological recovery can take months.

Prognosis

- Patients requiring ventilator support have longer hospital stays, slower recovery, and higher mortality.
- Fever and systemic symptoms are absent at disease onset; their presence suggests an alternative diagnosis [6].
- *GBS Variants:* GBS Classification Based on Electrodiagnostic and Pathological Findings [5, 6]. (Figure 3).

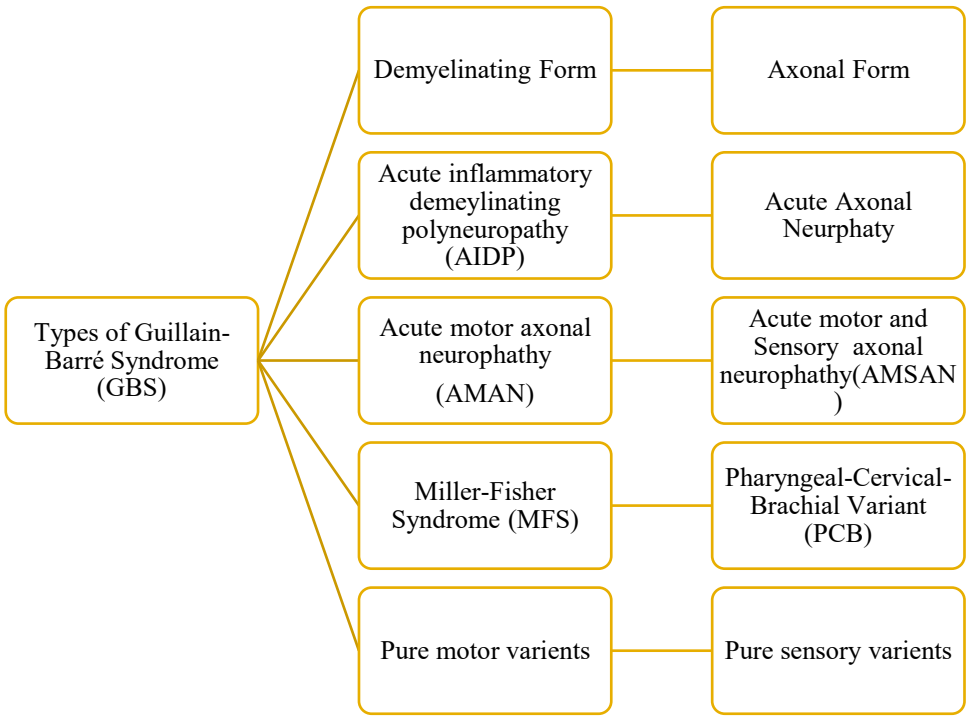

Figure 3. Types of Guillain–Barre Syndrome (GBS).

Guillain–Barré Syndrome (GBS) has different types based on how it affects the nerves. It is mainly divided into demyelinating and axonal forms. The demyelinating type, which is more common in Western countries, is called Acute Inflammatory Demyelinating Polyneuropathy (AIDP). It causes weakness in the limbs that slowly gets worse, along with loss of reflexes. A spinal fluid test usually shows high protein levels. This type responds well to treatment like intravenous immunoglobulin (IVIG) or plasma exchange, and most people recover well.

The axonal types of GBS are more common in Asia and Latin America. One of these is Acute Motor Axonal Neuropathy (AMAN**),** which mainly affects movement without causing sensory loss. It causes

sudden weakness in the arms and legs and may also affect the face and throat muscles. AMAN is often linked to *Campylobacter jejuni* infection, and patients usually have certain antibodies in their blood. Even though symptoms can be severe, recovery is often good with proper treatment. Another axonal type, Acute Motor and Sensory Axonal Neuropathy (AMSAN) is more serious. It affects both movement and sensation, leading to full-body weakness and, in some cases, difficulty in breathing. AMSAN progresses quickly, and many patients take longer to recover, sometimes with lasting nerve damage.

Some other types of GBS also exist. One well-known type is Miller-Fisher Syndrome (MFS), which occurs in about 5% of GBS cases. It mainly affects eye muscles, balance, and reflexes. The first sign is often double vision, followed by trouble walking and loss of reflexes. Despite these symptoms, strength in the arms and legs is usually not affected. This type is linked to a specific antibody (anti-GQ1b) and has a good recovery rate. Another type, Pharyngeal-Cervical-Brachial (PCB) GBS, causes weakness in the throat, neck, and arm muscles, but the legs are usually not affected. A spinal fluid test shows increased protein levels, and this type sometimes overlaps with AMAN [4–6].

Some rare forms of GBS also exist. The Pure Motor Variant causes weakness in the arms and legs without affecting sensation. It is often linked to *Campylobacter jejuni* infection, and most people recover well. However, doctors must rule out other conditions like poliomyelitis and myasthenia gravis. Another rare type, the Pure Sensory Variant**,** mainly affects sensation rather than movement. Patients may experience numbness, tingling, and trouble with balance. Tests usually show changes in the nerves, and recovery is generally good, though some may need treatment like IVIG or plasma exchange [6–8].

Overall, GBS has different types, each affecting nerves in a slightly different way. Some types recover quickly with treatment, while others take longer and may leave lasting effects. Understanding these differences helps in diagnosing and treating patients effectively [9, 10].

Diagnostic tests, particularly nerve conduction studies, are essential in confirming typical Guillain-Barré Syndrome (GBS). When thoroughly examined, these studies often show similar but less pronounced abnormalities in the different GBS variants. In some cases, muscle weakness continues to worsen beyond the usual timeframe, lasting three to four weeks or more. Among these patients, some may develop Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), a long-term progressive form of neuropathy. Additionally, a subset of patients experiences symptoms that persist for four to eight weeks before gradually improving, forming an intermediate category between acute GBS and chronic neuropathy11, 12.

DIAGNOSIS

The diagnosis of Guillain–Barré Syndrome (GBS) relies on a combination of clinical evaluation, laboratory investigations, and specialized nerve studies. Physicians assess progressive muscle weakness, loss of reflexes, and sensory disturbances as key indicators of the disease. Nerve conduction studies (NCS) help detect characteristic abnormalities, differentiating GBS from other neurological conditions. Cerebrospinal fluid (CSF) analysis typically reveals elevated protein levels with a normal white blood cell count, a hallmark of GBS. Additionally, blood tests may identify specific antibodies associated with certain subtypes. In some cases, magnetic resonance imaging (MRI) aids in ruling out alternative diagnoses. A comprehensive approach integrating clinical findings and diagnostic tests is essential for accurately identifying GBS and guiding appropriate treatment [11, 12].

GBS is identified based on its characteristic pattern: rapidly progressing paralysis with absent reflexes (areflexia), without fever or systemic symptoms, and often following an infection or other triggering event.

Differential Diagnosis (Conditions That Mimic GBS) [3, 7–9, 11–12]

Several other conditions may resemble GBS and should be ruled out:

1. *Acute Myelopathy:* Often involves prolonged back pain and bladder/bowel dysfunction.
2. *Botulism:* Early loss of pupillary reflexes is a key sign.
3. *Diphtheria:* Early oropharyngeal weakness can suggest this bacterial infection.
4. *Lyme Disease & Tick Paralysis:* Can cause nerve inflammation leading to weakness.
5. *Porphyria:* Look for abdominal pain, seizures, and psychiatric symptoms.
6. *Vasculitic Neuropathy:* Vasculitic neuropathy is commonly associated with an elevated erythrocyte sedimentation rate (ESR), which serves as a marker of underlying inflammation.
7. *Poliomyelitis:* Usually presents with fever and neck stiffness (meningismus).
8. *Cytomegalovirus (CMV) Polyradiculitis:* Seen in immunocompromised patients.
9. *Critical Illness Neuropathy: Can* occur in severely ill patients in ICU.
10. *Neuromuscular Disorders (e.g., Myasthenia Gravis):* Causes muscle weakness but with preserved *reflexes.*
11. *Poisoning (e.g., Organophosphates, Thallium, Arsenic, Paralytic Shellfish Toxins):* May cause nerve damage and paralysis.
12. *Severe Hypophosphatemia (Rare):* Can lead to muscle weakness and respiratory failure [11, 12].

ROLE OF LABORATORY TESTS

Lab tests help to rule out other *conditions* rather than directly diagnosing GBS. Common tests include:

- *Lumbar Puncture (CSF Analysis):* Shows elevated protein with normal white blood cells (albumin cytologic dissociation) [8].
- *Electromyography (EMG) & Nerve Conduction Studies (NCS):* Help confirm nerve involvement and subtype of GBS [9].
- *Blood Tests:* May detect infections, autoimmune markers, or metabolic disorders that mimic GBS.

DIAGNOSTIC CRITERIA FOR GBS (ASBURY AND CORNBLATH) [12]

Features Required for Diagnosis

a. Progressive motor weakness of more than one limb. b. Areflexia.

Features Strongly Supportive of the Diagnosis Clinical Features [12, 13]

i. Progression within four weeks.
ii. Relative symmetry of symptoms.
iii. Mild sensory symptoms or signs.
iv. Cranial nerve involvement.
v. Recovery within four weeks of progression cessation.
vi. Autonomic dysfunction.
vii. Absence of fever at onset.

CSF Picture

i. Raised CSF protein after 1 week of symptoms.
ii. Cell counts of less than or equal to 10 mononuclear leucocytes per cmm of CSF.

Electro Diagnostic Studies

i. Electro diagnostic features strongly supportive of the diagnosis (nerve conduction slowing or block).

Features Casting Doubt on the Diagnosis

i. Pronounced persistent asymmetry of weakness.
ii. Persistent bladder or bowel dysfunction.
iii. Bladder or bowel dysfunction at onset.
iv. More than 50 mononuclear leucocytes/3 mm.
v. presence of polymorphonuclear leucocytes in CSF.

vi. Sharp sensory level.

Features That Rule Out the Diagnosis [13]

i. Current history of hexa carbon misuse.
ii. Abnormal porphyrin metabolism.
iii. Recent diphtheritic infection.
iv. Features clinically consistent with lead neuropathy.
v. Purely sensory syndrome.
vi. Definite diagnosis of poliomyelitis, botulism, hysterical paralysis, or toxic neuropath.

Poor Prognostic Features [13]

1. Age more than 40 yrs.
2. Rapid progression to tetraplegia.
3. Early requirement for ventilation.
4. Associated previous discharge disease due campylobacter jejuni enteritis [1].

Modern Treatment

Management consists mostly of supportive care but plasma pheresis also is important. Large multicentre controlled trial in North America and Europe have demonstrated a beneficial effect of plasmapheresis if initiated in first 2 week if illness. Iv administration of high dose immunoglobulins (2 g/kg body weight over 5 days) is as effective as plasmapheresis.

In contrast glucocorticosteroids treatment has not been shown to be effective.

- *Management:* Respiratory care: Maintain vital capacity 4 hrly. If <1 litre check accuracy if accurate consider urgent ventilation.
- *Cardiac care:* If bradycardia (<50 bpm) consider temp.
- *Prevention of thrombosis and limb deformity:* Give subcutaneous heparin and supply antiembolism stockings.

Active treatment. Iv Immunoglobulin 0.4 g/kg/day-5 days, plasma exchange (50 ml /kg given occasions over 7–10 days [12, 13].

UNANI

Unani Pathophysiology

Guillain–Barré Syndrome (GBS) is characterized by rapidly progressing muscle weakness, paralysis, loss of reflexes (areflexia), and possible sensory disturbances, such as numbness. Conditions, like paralysis (Istirkha), hemiplegia (Falij), and numbness (Khaddar), have been prevalent for centuries, and physicians have been addressing these disorders for millennia. These diseases are not new; they have been described in detail by renowned Unani physicians, such as Hippocrates, Galen, Haly Abbas, Zakariya Razi, and Ibn Sina. In their writings, they discussed the pathophysiology, etiology, clinical features, and management of these disorders [14, 15].

Haly Abbas, in his work *Kamil-us-Sana'a*, categorizes diseases affecting the spinal cord and its nerves into five types: atony/flaccidity, paralysis of the entire body except for the face, numbness, convulsion/spasms, and tremors [15]. These conditions are typically classified under one of three categories: *sū'-i-mizāj* (dystemperament), *sue tarkīb* (diseases of arrangement), or *tafarruq ittīsāl* (loss of continuity).

Any deviation from the normal temperament can lead to dystemperament, which subsequently causes dysfunction of the affected organ. The impact of dystemperament depends on the type of cell, tissue, or organ involved. The involvement of a less significant organ, such as an auxiliary organ, typically has a lesser effect on the body. However, when dystemperament affects vital organs, like the spinal cord, its

consequences are far more significant. The effects are not limited to the local area but can be systemic. Extreme dysfunction within an integrated system, leading to severe derangement in the internal environment's temperament, may result in death [16].

Regarding dystemperament of the spinal cord and nerves, the most common type is cold and moist dystemperament, with or without matter [17]. According to the Unani System of Medicine, cold and moist *dystemperament* in the brain and spinal cord leads to the drifting of phlegmatic humor (*Rutoobat-e-Balghami*). Additionally, discarded secretions from the brain fall onto the nerves, which are responsible for sensations and movement, causing paralysis. The phlegmatic humors that drifts from the brain to the nerve origins obstructs the channels of pneuma or *ruh* (psychic spirit), hindering its passage through the obstructed nerves. This obstruction not only affects the nerves but also leads to a loss of sensation and movement [18–20].

Furthermore, cold dystemperament in the spinal cord and nerves can cause numbness, which refers to the complete or partial loss of sensation in any organ of the body. The cold dystemperament causes the psychic pneuma to become more viscous, preventing its smooth passage through the narrow channels of the nerves. This increased viscosity leads to numbness. Sometimes, nerve compression, either due to prolonged sitting or the presence of morbid matter or pneuma, may block the passages of nerves that are responsible for circulating the psychic pneuma, further contributing to numbness.

The causes of cold dystemperament include the overuse of cold-tempered foods and medications, such as opium, sudden exposure to extremely cold weather, or immersion of the feet in ice-cold water, especially in winter [21].

Moist dystemperament of the spinal cord or nerves leads to atony or flaccidity [22].

In cold and moist dystemperament, or phlegmatic dystemperament, an obstruction called *sudda* forms, which obstructs the passage of psychic pneuma [23]. The obstruction at the origin of the nerve reduces the supply of the motor faculty, leading to atony or flaccidity, and results in the loss of sensation and movement. When *sudda* is present at the origin of all nerves, sensation and movement throughout the body are lost, along with disturbances in psychic functions. This condition is referred to as *abar bilqisiya* [24].

If the obstruction occurs only on one side of the spinal cord, whether on the right or left, it causes flaccidity of the affected side [24]. Flaccidity of the facial muscles is called *laqwā* [13]. According to Hippocrates and Galen, when discarded secretions from the brain fall on the nerves that supply sensory and motor functions to the face, it leads to *l*aqwā

Fālij is a condition marked by flaccid paralysis affecting one side of the body. It may extend from the neck to the toes while sparing the head and face, or it can involve the entire half of the body from head to toe, including facial structures [25–27].

Management in Unani Medicine

Since *dystemperament* or loss of continuity in the nerves can cause neurodegeneration, the treatment approach should focus on the following:

- Avoid foods and medications with cold temperament, such as opium.
- Avoid sudden exposure to extremely cold weather.
- Use medications with properties, such as:
  - *Mukhrij-i-Balgham* (expellant of phlegm)
  - *Muqawwī-i-'Āab* (nerve tonic)
  - *Munaqi-¯ı-a'sab* (nerve cleanser)
  - *Muqawwī-i-'Ām* (general tonic)
  - *Dāfi'-i-Tashannuj* (antispasmodic)
  - *Muqawwī-i-Dimāgh* (brain tonic)

Unani Medications for the Treatment of Neurodegenerative Disorders Single Herbs

In Unani medicine, neurodegenerative disorders are predominantly treated with compound formulations, while single-herb remedies are used in only a few cases.

Single Drugs

Unani medicine uses a variety of medicinal plants to help manage neurodegenerative disorders (NDDs). Some of the commonly used herbs include Phyllanthus emblica, Ficus carica L. Anacyclus pyrethrum, Asarum europaeum. Withania Somnifera, Strychnos nux vomica, Prunus amygdalus, Nepeta Hindostana, Terminalia bellirica Roxb. Hyoscyamus niger, Myristica fragrans Houtt, Bacopa monnieri, Cinnamomum verum J. Presl, Piper longum, Piper nigrum, Gymnema sylvestre, Terminalia chebula, Delphinium denudatum wall , Juglans regi, Myristica fragans, Pipe, Phoenix dactylifera r cubeba, Alpinia galanga, Alpinia galanga, Boswellia serrata, Triana & planch, Pistacia lenticus, Commiphora wightii, Ocimum sanctum, Saussurea lappa, Punica granatum, Nigella sativa, Allium sativum , Nardostachys jatamansi, Lavandula stoechas, Acorus calamus, Crocus sativus, Olea europaea, Zingiber officinale Curcuma longa. These herbs are known for their beneficial effects on the nervous system. They help protect brain cells, improve memory, and reduce symptoms of conditions like Alzheimer's, Parkinson's, depression, anxiety, and epilepsy. Their medicinal properties include antioxidant, anti-inflammatory, neuroprotective, and mood-enhancing effects.

Table 1 provides a detailed overview of these herbs, including their Unani and scientific names, the parts used, their health benefits, recommended dosages, and commonly used Unani formulations. These natural remedies play an important role in Unani medicine for improving brain function and supporting overall neurological health.

Compound Formulations

Unani classical texts describe numerous compound formulations for various health conditions, including brain disorders. Among them, 26 polyherbal formulations have been specifically highlighted for their potential in treating neurological conditions. Detailed information about these formulations is provided in Table 2. These remedies are available in different dosage forms, such as Ḥabb (pills) and semi-solid preparations like Iṭrīfal, Khamīra, Maʻjūn, and Jawārish. Each of these formulations differs in texture and preparation techniques, but the majority are designed for oral administration.

In Unani medicine, these compound formulations are not limited to neurological disorders; they are also prescribed for various other ailments, including Waram-i-Mafāṣil (arthritis), Ḍuʻf-i-Qalb (cardiac insufficiency), Waram-i-Kabid (hepatitis), and Faqr al-Dam (anemia). However, there is an urgent need to scientifically validate both Unani single herbs and compound formulations to confirm their therapeutic efficacy and possible side effects.

A comprehensive list of Unani formulations used for brain disorders is presented in Table 2, while Table 3 outlines studies conducted so far to evaluate their medicinal properties. Research records available on Google Scholar have been referenced to analyze their effectiveness and pharmacological impact. Further scientific validation is crucial to establish their role in modern medicine [28].

Neuroprotection in Unani Medicine: Traditional Insights and Modern Perspective

Unani medicine attributes the effectiveness of herbs and compound formulations in treating Neurodegenerative Disorders (NDDs) to their properties, such as nerve tonic, brain tonic, memory enhancer, nervine stimulant, brain cleanser, and remover of morbid matter. From an allopathic perspective, these treatments are understood through mechanisms including antioxidant effects, memory enhancement, anti-Alzheimer's and anti-Parkinson's activities, antidepressant and anti-anxiety effects, as well as neuroprotective properties. In Unani System of Medicine (USM), NDDs are often associated with dryness in the body leading to organ weakness and dysfunction, akin to oxidative stress-related degenerative changes seen in conventional medicine during aging. Therefore, neuroprotective herbal antioxidants are considered crucial for slowing disease progression and reducing neuronal cell death.

Table 1. Brief description of Single herbs recommended for Neurodegenerative disorders.

Unani Name	Scientific Name	Family	Part Used	Action in Unani	Therapeutic Used in Unani	Dose	Dosage Form	Reference
Amla	Phyllanthus emblica L.	Phyllanthaceae	Fresh and dired fruits, seed, nuts, flowers, Leaves and bark	Muqawwī-Dimagh (Braintonic), Muqawwī-i-āfizā (Memoryenhancer)	u‘f-i-Dimāgh (Cerebrasthenia)	3–5 g	Jawarish Anoshdārū, Murabbā, Jawārish-i-Āmla Sāda	[29, 30]
Anjeer	Ficus carica L.	Moracea	Fruits	Mughadhdhi (Nutritive)	u‘f-i-arārat Gharīziyya (innate heat insufficiency) u‘f-i-‘Ām (General debility)	Safūf Safūf-i-Bara		[29, 30]
Aqar qarha	Anacyclus pyrethrum (L.)	Asteraceae	Roots	Muqawwī-i-‘Ām (General tonic), Mukhrij-i-Balgham (Expellant of phlegm)	u‘f-i-‘Ām (General debility), Amrā-iBārida Balghamiyya (Cold phlegmatic diseases)	2–3 g	Jawārish Jawārish-i-Zar‘ūnī, Falūniya Rūmī, Barsha’sha	[29, 30]
Asaroon	Asarum europaeum L.	Aristolochiaceae	Rhizome	Muharrik-i-A‘sab (nervinestimulant)	Sar‘ (epilepsy), Falij (paralysis), Istirkh¯a (flaccidity), Khadr (numbness), Ra‘sha (Tremor), Nisyān (Dementia)	3 g	Jawārish Jawārish-i Jalinūs	[29, 30, 31]
Asgand	Withania Somnifera (L)	Solanaceae	Dried root	Muqawwī-i-‘Ām (General tonic), Muqawwī-i-‘Āab (Nervina tonic), Munaqi-¯ı-a‘sab (nerve cleanser)	u’f-i-‘Ām (General debility), u’f-i-Āab (Nervine weakness), Nisyān (Dementia)	5-10g	Habb, Ma‘jun Habb-i-Asgand, Safūf-iAsgand, Ma‘jūn, Muqawwi Raim	[30, 31]
Azaraqi	Strychnos nux vomica L.	Loganiaceae	seed	Muqawwī-i-‘Āāb (Nervine tonic), Muarrik-i-Ā’āb (Nervine stimulant), Dāfi’-i-Amrā ‘Abāniya (Useful in nervine diseases)	u’f-i-Āab (Nervine weakness)	60–250 mg	Habb, Ma‘jun Habb-i-Azārāqī, Ma‘jun Azārāqī, Ma‘jun Lanā	[30, 31]
Badam shirin	Prunus amygdalus Batsch.var.dulces	Rosaceae	seed	Muqawwī-i-Dimagh (Brain tonic), Muqawwī-i-‘Āāb (Nervine tonic)	u’f-i-Dimāgh (Cerebrasthenia), u’fiĀab (Nervine weakness)	7–11	Seeds Rawghan, Lubūb Rawghan Bādām Shīrīn, Lubūb Kabīr, Lubūb aghīr	[30, 31, 32]
Badranjboya	Nepeta Hindostana (Roth) Haines	Lamiaceae	Leaf	Muqawwī-i-arārat Gharīziyya (Tonic for innate heat)	u’f-i-arārat Gharīziyya (innate heat insufficiency)	5–7 g	Ma‘jun Ma‘jun-i-Khadar	[30, 31]

Balela	Terminalia bellirica Roxb.	Combretaceae	Fruits	Muqawwī-i-Dimāgh (Brain tonic)	u'f-i-Dimāgh (Cerebrasthenia)	5–7 g 11, 98	Itrīfal Itrīfal Ustūkhudūs, Itrīfal aghīr, Itrīfal Muqil	[31]
Bazr-ul-Banj	Hyoscyamus niger	Solaneceae	seed	Dāfi'-i-Tashannuj (Antispasmodic), Mukhaddir (Anaesthetic), Munawwim (Hypnotic), Musakkin (Sedative)	Amrā 'Abāniya (Nervine diseases), Dard wa Alam (Pain), Sahar (Insomnia), Junūn (Mania)	0.5–1 g 11, 12	La'ūq Barsha'sha, La'ūq Bazr-ulBanj, Banādiqul Buzūr	[30, 31]
Bisbasa	Myristica fragrans Houtt.	Myristicaceae	Arill	Muqawwī-i-Dimāgh (Brain tonic)	u'f-i-Dimāgh (Cerebrasthenia)	2–5 g	Itrīfal, Jawārish Anoshdārū, Itrīfal Kabīr, Jawārish Bisbāsa	[30, 31, 32]
Brahmi	Bacopa monnieri (Linn)	Scrophulariaceae	Whole plant	Muqawwī-i-Dimāgh (Brain tonic), Muqawwī-i-'Āab (Nervine tonic	u'f-i-Dimāgh (Cerebrasthenia), u'f-iĀab (Nervine weakness)	5 g	Ma'jun Ma'jun Brahmi	[31, 32]
Darchini	Cinnamomum verum J.Presl	Lauraceae	Bark	Muqawwī-i-arārat Gharīziyya (Tonic for innate heat)	u'f-i-arārat Gharīziyya (innate heat insufficiency)	1–2g	Jawārish, Rawghan Jawārish Jālīnūs, Rawghan Dār Chīnī, Jawārish Bisbāsā	[30, 31, 32]
Filfil Daraz	Piper longum L.	Piperaceae	Fruits	Muqawwī-i-'Ām (General tonic), Muqawwī-i-arārat Gharīziyya (Tonic for innate heat)	u'f-i-'Ām (General debility), u'f-iarārat Gharīziyya (Innate heat insufficiency)	500mg –1 g 11, 12, 98	Jawārish, Ma'jun Jawārish Jālīnūs, Jawārish Bisbāsā, Ma'jun Falāsifā	[30, 31, 32]
Filfil Siyah	Piper nigrumL.	Piperaceae	Fruits	Muqawwī-i-'Āab (Nervine tonic)	u'f-i-Āab (Nervine weakness	1–2 g 12, 91, 98	Jawārish, Jawārish Kamūni, Dawā-ulShifā, Jawārish Jālīnūs	[30, 31, 32]
Gurmar	Gymnema sylvestre	Asclepiadaceae	Leaf and Root	Musakkin-i- 'Aāb (Nervine sedative), Muqawwī-i-'Āab (Nervine tonic	) u'f-i-Āab (Nervine weakness)	5–7 g 12, 91	7g Qur Qur Dhayābīus Khā	[30, 31, 32]
Halela Zard	Terminalia chebula	Combretacea	Fruits	Muqawwi-e-Dimagh (Brain tonic), Muqawwī-i-Hafiza (Memory enhancer)	u'f-i-Dimāgh (Cerebrasthenia), u'f-iāfizā (Poor memory)	3–5 g 11, 12, 98	Itrīfal Itrīfal Kishnīzī, Itrīfal Ustūkhudūs, Itrīfal Mulayyin	[30, 31, 32]
Jadwar	Delphinium denudatum wall	Ranunculaceae	Root	Muqawwī-i-'Āab (Nervina tonic)	u'f-i-Āab (Nervine weakness), Istirkhā (Atony/flaccidity)	0.5–1g	Habb, Khamīra Habb-i-Jadwār, Khamīra	[30, 31, 32]

							Gāozabān Ambarī Jadwār ʻŪd Salīb Wālā	
Jawz	Juglans regi L.	Juglandaceae	Kernels	Muqawwī-i-Aʻā' Raʾīsa (Tonic for vital organs), Musakkin-i- ʻAāb (Nervine sedative)	u'f-i-Dimāgh (Cerebrasthenia), u'f-iĀab (Nervine weakness), u'f-i-āfizā (Poor memory)	10–20 g	Lubūb Lubūb-ī-Kabīr, Lubūb-i-aghīr	[30, 31, 32]
Jawzbuwa	Myristica fragans Houtt.	Myristicaceae	Fruits	Muqawwī-i-ʻĀab (Nervina ton'ic)	u'f-i-Āab (Nervine weakness), Amrāi-Bārida (Diseases of cold temperament), Istirkhā' (Atony), Khadar (Numbness)	1–2 g	Jawārish, Maʻjun Anoshdārū, Jawarish Ood Shirin, Maʻjun -i Chob Chini	[30, 31, 32]
Kababa	Piper cubeba L.	Piperaceae	Fruits	Muqawwī-i-arārat Gharīziyya (Tonic for innate heat), Muarrik (Stimulant)	u'f-i-arārat Gharīziyya (Innate heat insufficiency)	1–3 g	Maʻjun Maʻjun-i-Anakī, Labūb-iSaghīr	[30, 31, 32]
Khulanjan	Alpinia galanga L.Wild.	Zingiberaceae	Rhizome	Muqawwī-i-ʻĀab (Nervine tonic), Musakkin (Sedative	Amrā-i-Aʻāb (Nervine diseases	2–3 g	Jawārish, Habb Jawārish Jālīnūs, Habb-i-ʻĀmbar	[30, 31]
Kundur	Boswellia serrata Triana & planch	Burseraceae	Gum/exudate	Muqawwī-i-Dimagh (Brain tonic)	u'f-i-Dimāgh (Cerebrasthenia)	3–4 g	Maʻjun, Jawārish Maʻjun Kundur, Jawārish Kundur	[30, 31]
Mastagi	Pistacia lenticus L.	Anacardiaceae	Resin	Muqawwī-i-arārat Gharīziyya (Tonic for innate heat)	u'f-i-arārat Gharīziyya (Innate heat insufficiency)	1–2 g	Jawārish Jawārish Maagī, Jawārish Jālīnūs	[30, 31]
Muqil	Commiphora wightii (Arn)	Burseraceae	Gum/exudate	Muqawwī-i-ʻĀab (Nervine tonic)	u'f-i-Āab (Nervine weakness)	1–15 g	Habb, Maʻjun Habb-i-Muqil, Maʻjun Muqil	[30, 31]
Rehan	Ocimum sanctum Linn.	Lamiaceae	Leaf	Munaffith-i-Balgham (Expectorant), Muallil-i-Waram (Anti-inflammatory)	Tap-i-Balghami (Phlegmatic fever)	5–7 g	Khamīra Khamīra Ābresham ʻŪd Maagiwālā	[30, 31, 32]
Ratab	Phoenix dactylifera L.	Arecaceae	Fruits	Muqawwī-i-ʻĀab (Nervine tonic), Muqawwī-i-ʻĀm (General tonic),	uʻf-i-Āab (Nervine weakness), uʻf-i-ʻĀm (General debility)	10–15 g	Maʻjun Maʻjun Aarad Khurmā, Maʻjun Supārīpāk	[30, 31, 32]

Qust Shirin	Saussurea lappa CB Clarke	Asteraceae	Root	Muqawwī-i-‘Āab (Nervine tonic), Mohallil-i-waram (Antiinflammatory	Falij (paralysis), Ra‘sha	2–3 g	Jawārish Jawārish-i-Jalinūs	[30, 31, 32]
Rumman	Punica granatum L.	Lythraceae	Fruit	Muwallid-i-Dam (Haemopoietic), Muqawwī-i-arārat Gharīziyya (Tonic for innate heat)	Faqruddam (Anaemia), u’f-i-arārat Gharīziyya (Innate heat insufficiency)	5–10 g	Jawārish, Sharbat Jawārish Anāayn, Sharbat Anār, Jawārish Pudīna	[30, 31, 32]
Shoneez	Nigella sativa L.	Ranunculaceae	Seed	Muqawwī-i-‘Āab (Nervine tonic)	u‘f-i-Āab (Nervine weakness)	1–2 g	Ma‘jun Ma‘jun Kalkalānaj, Ma‘jun Fanjnosh	[30, 31, 32]
Seer	Allium sativum L.	Liliaceae	Bulb	Muqawwī-i-arārat Gharīziyya (Tonic for innate heat), Qāti‘ Akhlāt-i-Ghalīza (Breaking agent for viscous humour)	u’f-i-arārat Gharīziyya (Innate heat insufficiency), Amrā-i-Bārida Balghamiya (Cold Phlegmatic disorders)	2–3 g	Ma‘jun Ma‘jun Sīr, Ma‘jun Sīr Alvi Khān	[30, 31, 32]
Sumbul-Ut-Teeb	Nardostachys jatamansi DC.	Caprifoliaceae	Rhizome	Muqawwī-i-Dimāgh (Brain tonic)	u’f-i-Dimāgh (Cerebrasthenia)	3–5 g	Jawārish Jawārish Fanjnosh, Barsh‘sha, Anoshdārū	[30, 31, 32]
Ustukhuddus	Lavandula stoechas L.	Lamiaceae	Leaves, flowers, whole plant	Muqawwī-i-‘Āab (Nervine tonic), Muqawwī-i-Dimāgh (Brain tonic), Muharrik-i-A’sab (nervine stimulant)	u‘f-i-Āab (Nervine weakness), u’f-iDimāgh (Cerebrasthenia)	7–10 g	Itrīfal Itrīfal Ustukhuddūs	[30, 31, 32]
Waj turki	Acorus calamus L.	Araceae	Rhizome	Muqawwī-i-‘Āab (Nervine tonic), Muqawwī-i-Dimāgh (Brain tonic)	u‘f-i-Āab (Nervine weakness), u’f-iDimāgh (Cerebrasthenia)	1–3 g	Ma‘jun Ma‘jun Vaj	[30, 31, 32]
Zafran	Crocus sativus L.	Iridaceae	Style & Stigma	Muqawwī-i-Dimāgh (Brain tonic)	u’f-i-Dimāgh (Cerebrasthenia), Nisyān (dementia)	25–50 mg	Dawa Dawa ul Kurkum, Daw aul Misk Mo‘tadil Sada	[30, 31, 32]
Zaitoon	Olea europaea L.	Oleaceae	Fruit oil & Leaf Fruit oil & Leaf	Muqawwī-i-‘Āab (Nervine tonic)	u’f-i-‘Ām (General debility), Nisyān (Forgetfulness)	50 ml	Rawghan Rawghan Zaitūn	[11, 31]
Zanjabeel	Zingiber officinale Rosc	Zingiberaceae	Rhizome	Muqawwī-i-‘Āab (Nervine tonic	u‘f-i-Āab (Nervine weakness), Nisyān (Forgetfulness)	1–1.5 g	Jawārish Jawārish Zanjabīl, Murabbā Zanjabīl, Ma‘jun Zanjabīl	[30, 31, 32]
Zard Chob	Curcuma longa L.	Zingiberaceae	Rhizome	Musakkin (Soothing agent), Mufatti-i-Sudad (Deobstruent)	u’f-i-‘Ām (General debility)	5–7 g	Sanūn, Safūf Sanūn-i-Zard, Safūf-i-ihāl, Rawghan Aurāq Qawī	[30, 31, 32]

Table 2. Compound formulations in Unani Medicine for Neurodegenerative disorders.

Compound Dose Dosage Form	Formulation Main	Indication Actions	Dose	Dosage Form	Reference
Dawa-ul-Misk	Motadil Sada Ḍu‘f-i A‘dā’ Ra’īsa (General debility), Malikhuliya (Melancholia)	Muqawwī-i- Dimāgh (Brain tonic), Muqawwī-i- Āam (General tonic)	5–10 gm	Semi-solid preparation	[32, 33]
Habb-e-Azaraqi	Fālij (Hemiplegia), Laqwa (Bell’s palsy), Khadar (Numbness)	Muqawwī-i- A‘sāb (Nervine tonic), Muharrik-i- A‘sāb (Nervine stimulant)	250–500 mg Pills 13,17	Pills	[32, 33]
Habb-e-Beesh	Ḍu’f-i-Āṣab (Nervine weakness)	Muqawwī-i- A‘sāb (Nervine tonic)	150 gm	Semi-solid preparation	[33]
Habb-e-Bolas	Ḍu’f-i-Āṣab (Nervine weakness), Nisyān (Dementia)	Muqawwī-i- Dimāgh (Brain tonic)	5–10 gm	Semi-solid preparation	[33]
Habb-e-Jadwar 250–500 mg	Ḍu’f-i-Āṣab (Nervine weakness)	Muharrik-i- A‘sāb (Nervine stimulant)	250–500 mg	Pills	[32, 33, 34]
Habb-e-Jalinoos	Ḍu’f-i-Āṣab (Nervine weakness)	Muqawwī-i- A‘sāb (Nervine tonic)	500–1 gm	Pills	[32, 33, 34]
Habb-e-Jawahar	Ḍu‘f-i A‘dā’ Ra’īsa (General debility), Ḍu’f-i-Āṣab (Nervine weakness)	Muqawwī-i-A‘dā’ Ra’īsa (General tonic)	150–250 gm	pills	[32, 33]
Habbe-e-Sara	Ṣar‘ (Epilepsy)	Muqawwī-i- A‘sāb (Nervine tonic)	250 gm	Semi-solid preparation	[33]
Itrifal Aftimoon Semi-solid preparation 13, 17	Malikhuliya (Melancholia)	Muqawwī-i- Dimāgh (Brain tonic)	10 gm	Semi-solid preparation	[32, 33]
Itrifal Muqawwi Dimagh 5–10 gm Semi-solid preparation	Ḍu’f-i-Dimāgh (Cerebrasthenia)	Muqawwī-i- Dimāgh (Brain tonic)	5–10 gm	Semi-solid preparation	[32, 33, 34]
Itrifal Sagheer 10–15 gm Semi-solid preparation 1	Ḍu’f-i-Dimāgh (Cerebrasthenia), Nisyān (Dementia)	Muqawwī-i- Dimāgh (Brain tonic)	10–15 gm	Semi-solid preparation	[13, 17, 33, 34]
Itrifal Zabeeb	Ṣar‘ (Epilepsy)	Mufattiḥ-i- Sudad (Deobstruent) 10–15 gm Semi-solid preparation	10–15 gm	Semi-solid preparation	[33, 34]
Jawarish Shahanshahi Ambari 5–10 gm Semi-solid preparation	Ḍu’f-i-Dimāgh (Cerebrasthenia)	Muqawwī-i- Dimāgh (Brain tonic)	5–10 gm	Semi-solid preparation	[33, 34]
Jawarish Shahi Ḍu’f-i-Dimāgh (Cerebrasthenia) Muqawwī-i-Dimāgh (Brain tonic)	Ḍu’f-i-Dimāgh (Cerebrasthenia)	Muqawwī-i- Dimāgh (Brain tonic)	5–10 gm	Semi-solid preparation	[33]
Jawarish Zarooni sada 5–10 gm Semi-solid preparation	Ḍu’f-i-Dimāgh (Cerebrasthenia)	Muqawwī-i- Dimāgh (Brain tonic)	5–10 gm	Semi-solid preparation	[33]
Khamira Abresham Hakim Arshad Wala, Muqawwī-i- Āam (General tonic)	Ḍu‘f-i A‘dā’ Ra’īsa (General debility), Ḍu‘f-i-Umūmi (General weakness)	Muqawwī-i-A‘dā’ Ra’īsa (General tonic)	3–6 gm	Semi-solid preparation	[33, 34]
Khamira Abresham Sheera Unnab wala 3–6 gm Semi-solid preparation	Ḍu’f-i-Dimāgh (Cerebrasthenia), Ḍu ‘f-i-Baṣar (Poor eyesight)	Muqawwī-i- Dimāgh (Brain tonic), Muqawwi Basar (Eye tonic)	3–6 gm	Semi-solid preparation	[33, 34]
Khamira Gaozaban Ambari Jawahar wala 3–6	Ḍu’f-i-Dimāgh (Cerebrasthenia), Malikhuliya	Muqawwī-i- Āam (General tonic)	3–5 gm	Semi-solid preparation	[33, 34]

gm Semi-solid preparation	(Melancholia), Ḍu'f-iĀṣab (Nervine weakness)				
Khamira Gaozaban Sada	Ḍu'f-i-Dimāgh (Cerebrasthenia), Malikhuliya (Melancholia)	Muqawwī-i- Dimāgh (Brain tonic), Muqawwī-i- Āam (General tonic)	5–10 gm	Semi-solid preparation	[32, 33, 34]
Khamira Marwareed Muqawwī-i- Āam (General tonic) 3–5gm Semi-solid preparation	Ḍu'f-i-Āṣab (Nervine weakness), Ḍu'f-i-Dimāgh (Cerebrasthenia)	Muqawwī-i- Āam (General tonic)	3–5 gm	Semi-solid preparation	[33, 34]
Majoon Asal-e-Baladur Muqawwī-i- A'sāb (Nervine tonic) 3–5 gm Semi-solid preparation	Ḍu'f-i-Āṣab (Nervine weakness)	Muqawwī-i- A'sāb (Nervine tonic)	3–5 gm	Semi-solid preparation	[33, 34]
Majoon Azaraqi	Ḍu'f-i-Āṣab (Nervine weakness), Ṣar' (Epilepsy)	Muqawwī-i- A'sāb (Nervine tonic)	3–5 gm	Semi-solid preparation	[33, 34]
Majoon Baladur	Ḍu'f-i-Āṣab (Nervine weakness), Nisyān (Dementia) Muqawwī-i-A'sāb (Nervine tonic), Muqawwī-i- Dimāgh (Brain tonic)	Muqawwī-i- Āam (General tonic)	5–10 gm	Semi-solid preparation	[34, 35]
Majoon Falasafa	Nisyān (Dementia))	Muqawwī-i- Āam (General tonic	5–10 gm	Semi-solid preparation	[33, 35]
Majoon Jograj	Ḍu'f-i-Āṣab (Nervine weakness), Ra'sha (Tremor)	Muqawwī-i- A'sāb (Nervine tonic)	5–10 gm	Semi-solid preparation	[33, 34, 35]
Majoon Khadar	Ḍu'f-i-Dimāgh (Cerebrasthenia), Ḍu'f-i-Āṣab (Nervine weakness), Waram-i-Āṣab (Neuritis)	Muqawwī-i- A'sāb (Nervine tonic), Muharrik-i- A'sāb (Nervine stimulant)	5–10 gm	Semi-solid preparation	[33, 34]
Majoon Lana	Ḍu'f-i-Āṣab (Nervine weakness), Ra'sha (Tremor), Ṣar	Muqawwī-i- A'sāb (Nervine tonic)	3–5 gm	Semi-solid preparation	[34]
Majoon Mujarrab	Malikhuliya (Melancholia)	Munaqqi-i- Dimāgh (Braun cleanser)	5–10 gm Semi-solid preparation 17	Semi-solid preparation	[34]
Majoon Muluki	Ḍu'f-i-Āṣab (Nervine weakness)	Muqawwī-i- A'sāb (Nervine tonic)	5–10 gm	Semi-solid preparation	[34]
Majoon Najah	Malikhuliya (Melancholia) Muqawwī-i- A'sāb (Nervine tonic	Muqawwī-i- A'sāb (Nervine tonic	5–10 gm	Semi-solid preparation	[33, 34]
Majoon Nisyan	Nisyān (Dementia) Muqawwi hafiza,	Muqawwī-i- A'sāb (Nervine tonic)	5 gm	Semi-solid preparation	[34]
Majoon Seer Alvi Khan	Fālij (Hemiplegia), Ra'sha (Tremor)	Muqawwī-i- A'sāb (Nervine tonic)	5–10 gm	Semi-solid preparation	[33, 34]
Majoon Talkh	Fālij (Hemiplegia), Ṣar' (Epilepsy)	Muqawwī-i- A'sāb (Nervine tonic)	5–10 gm	Semi-solid preparation	[34]
Mufarreh Barid	Ḍu'f-i-Āṣab (Nervine weakness)	Muqawwī-i- A'sāb (Nervine tonic)	5–10 gm	Semi-solid preparation	[33, 34]
Sharbat Ahmad	Shahi Malikhuliya (Melancholia)	Muqawwī-i- A'sāb (Nervine tonic)	25–35 ml	Syrup	[34]
Tiryaq Samania	Ra'sha (Tremor), Ṣar ' (Epilepsy), Laqwa (Bell's palsy)	Muqawwī-i- A'sāb (Nervine tonic)	3–5 gm	Semi-solid preparation	[34]

Table 3. Antioxidant/Memory enhancing/Anti depression/ Immunomodulatory activities of the proposed herbs.

Unani Name (Scientific Name)	Common Name	Family	Active Constituent	Pharmacological Activity	Reference
(Phyllanthus emblica L.)	Amla	Amla Phyllanthaceae	Emblicannin A & B, Alkaloid (Phyllantine), Phenol (Gallic acid)	Antioxidant	[36]
Anjeer (Ficus carica L.)	Fig	Moraceae	Phenols	Antioxidant	[37]
Aqar qarha (Anacyclus pyrethrum (L.) Lag.)	Pellitory	Asteraceae	Anacyclin	Antioxidant, Memory enhancer, Anti-depressant	[38, 39]
Azārāqi (Strychnos nux-vomica L.)	Kuchla	Loganiaceae	Strychnine, Brucine	Antioxidant, Anti-amnesic, Anti-convulsant	[40, 41]
Balela (Terminalia bellirica Roxb.)	Bahera	Combretaceae	Beta-sitosterol, gallic acid, ellagic acid, bellaricanin	Antioxidant, Anti-Alzheimer's, Anti-depressant	[42, 43, 44]
Bazr-ul-Banj (Hyoscyamus niger L.)	Henbane	Solanaceae	Hyoscyamine, hyoscine, Scopolamine	Antioxidant, Anti-depressant, Anti-parkinsonism	[45, 46]
Bisbāsa (Myristica fragrans Houtt.)	Mace, Javitri	Myristicaceae	Terpenoids phenol	Antioxidant, Memory-enhancer	[47, 48]
Brahmi (Bacopa monnieri (Linn.) Pennel.)	Water hyssop	Scrophulariaceae	Bacoside, brahmin, herpestine, d-mannitol, luteolin, apigenin	Antioxidant, Memory-enhancer, Anti-depressant, Anti-Alzheimer's, Antiparkinsonism, Neuroprotective	[47, 48]
Filfil Siyah (Piper nigrum L.)	Black & White Pepper	Piperaceae	Piperine, Piperidine	Antioxidant, Neuroprotective	[49, 50]
Jadwar (Delphinium denudatum wall.)	Blood veined sage	Ranunculaceae	Flavonoids, Triterpenoids, Alkaloids	Antioxidant, Anti-Alzheimer's, Anti-depressant, Anti-anxiety, Anti-convulsant	[51, 52, 53]
Jawz (Juglans regia L.)	Walnut	Juglandaceae	Juglandic acid, Juglonone, Juglone	Antioxidant, Memory enhancer, Anti-depressant	[54]
Jawzbuwa (Myristica fragrans Houtt.)	Nutmeg	Myristicaceae	Fixed oil, Volatile oil, lignan	Antioxidant, Anti-depressant, Anti-convulsant	[55]
Kundur (Boswellia serrata Triana & Planch.)	Olibanum	Burseraceae	Essential oil, Terpenoids, Boswellic acids	Anti-Alzheimer's, Neuroprotective	[56]
Ratab (Phoenix dactylifera L.)	Dates	Arecaceae	Phenols, Flavonoids, tannins	Antioxidant, Prevention of peripheral neuropathy activity	[57]
Rehan (Ocimum tenuiflorum L.)	Holy Basil	Lamiaceae	3,4-dimethoxycinnamic acid, Pedunculin	Antioxidant, Memory enhancer, Anti-anxiety, Anti-depressant, Anti-convulsant	[58, 59, 60]
Sumbul ut-teeb (Nardostachys jatamansi)	Muskroot	Caprifoliaceae	Sesquiterpenes, coumarins, valeranone, jatamansone	Antioxidant, Stress-relieving activity	[34, 40]
Zafran (Crocus sativus L.)	Saffron	Iridaceae	Crocin, picrocrocin, crocetin	Antioxidant, Anti-Alzheimer's, Memory enhancer	[41, 50, 61]
Zanjabeel (Zingiber officinale Roscoe)	Ginger	Zingiberaceae	Terpenes, Phenols, Essential oils, Oleoresin (Gingerol, shogaol, zingerone)	Antioxidant, Neuroprotective, Cognitive enhancer activity	[60]
Zard Chob (Curcuma longa L.)	Turmeric	Zingiberaceae	Curcumin	Antioxidant, Anti-depressant	[44]

METHODOLOGY

This study examines the pathophysiology and management of Guillain–Barré Syndrome (GBS) from both modern and Unani perspectives. A detailed literature review was conducted to understand the Unani interpretation of GBS and identify herbal and compound formulations traditionally used for neurodegenerative disorders.sKey Unani texts, including *Kamiluṣ Sana'a*, *Al-Qanoon Fit-Tib*, *Zakhira Khwarazm Shahi*, Kitab Al-Adwiyya wal Aghziyya, Kitab al-Mukhtarat Fit-Tib, Muheet-e-Azam, Bayaze-Kabeer, Qarabadeen Qadri, Makhzanul Mufradat, and Khazainul Advia, were reviewed along with their translations, research theses, dissertations, journals, and conference proceedings. Additionally, a manual survey was conducted using reference books to match traditional Unani medicinal plants with their scientific names. This process was carried out over three months, from January to March 2025.

To assess the therapeutic potential of Unani medicines for neurodegenerative disorders, electronic databases, such as ScienceDirect, PubMed, Wiley Online Library, and Google Scholar were systematically searched. The search strategy incorporated MeSH (Medical Subject Headings) terms and relevant keywords, including "Neurodegenerative disorders," "Alzheimer's disease," "Parkinson's disease," "Dementia," and "Cognitive impairment," along with traditional Unani terms, such as Nisyā*n*, Ra'sha, Mālankhūliya, *Sehar*, and *Saktā*. Each recommended herb and formulation were analyzed for its pharmacological properties, including antioxidant, memory-enhancing, anti-Alzheimer's, antiparkinsonian, anticonvulsant, anxiolytic, cognitive-enhancing, neuroprotective, and antidepressant effects.

LITERATURE FINDINGS

The key insights from the literature review are outlined in Tables 1–3:

- Table 1 provides a list of individual herbs recommended by Unani scholars for managing neurodegenerative disorders.
- Table 2 presents details of compound formulations used in Unani medicine for treating neurodegenerative conditions.
- Table 3 summarizes the pharmacological properties of these formulations and their therapeutic effects.

This systematic approach ensures a thorough review of Unani medicine's potential contributions to managing neurodegenerative disorders, particularly in the context of Guillain–Barré Syndrome.

DISCUSSION

Guillain–Barré Syndrome (GBS) and other neurodegenerative disorders (NDDs) have long been recognized in Unani medicine under conditions, such as Istirkha (paralysis), Falij (hemiplegia), and Khaddar (numbness). Classical Unani scholars, including Hippocrates, Galen, Haly Abbas, Zakariya Razi, and Ibn Sina, provided detailed descriptions of these disorders, their pathophysiology, and management strategies. According to the Unani System of Medicine (USM), these conditions arise due to disturbances in temperament, particularly cold and moist dystemperament, which leads to obstruction (sudda) in the passage of psychic pneuma (ruh), resulting in impaired nerve function.

The Unani approach to managing neurodegenerative disorders focuses on restoring the balance of temperament, removing morbid matter, and strengthening the nervous system. Treatment strategies involve dietary modifications, lifestyle adjustments, and the use of single herbs and compound formulations with properties, such as *Mukhrij-i-Balgham* (phlegm expellant), *Muqawwī-i-'Āsab* (nerve tonic), and *Muqawwī-i-Dimāgh* (brain tonic). Several Unani medicinal plants, including *Bacopa* monnieri, Withania somnifera*, Acorus calamus*, and *Nigella sativa*, have shown neuroprotective, antioxidant, anti-Alzheimer's, and anti-Parkinsonian activities. Moreover, Unani polyherbal formulations, prepared in dosage forms, such as Itrifal, Khameera*,* Ma'jun*,* and Jawārish, have traditionally been used for neurological health and warrant further scientific validation.

From a modern biomedical perspective, the efficacy of these Unani interventions can be attributed to their antioxidant, anti-inflammatory, neuroprotective, and memory-enhancing properties. The overlap between Unani principles and contemporary neuropharmacology highlights the potential of traditional medicine in managing neurodegenerative conditions. However, rigorous clinical studies are required to establish the safety, efficacy, and pharmacological mechanisms of these treatments. The integration of Unani medicine with modern research methodologies could provide new therapeutic insights into the management of neurodegenerative disorders, including GBS, and contribute to holistic healthcare approaches.

CONCLUSIONS

Guillain–Barré Syndrome (GBS) and other neurodegenerative disorders (NDDs) have been recognized in Unani medicine for centuries, with classical scholars detailing their causes, symptoms, and treatments. The Unani approach attributes these conditions to disturbances in temperament, particularly cold and moist dystemperament, leading to obstruction in nerve function. Management involves restoring balance through dietary and lifestyle modifications, along with herbal and polyherbal formulations that exhibit neuroprotective, antioxidant, and anti-inflammatory properties. Although these traditional therapies show potential, comprehensive clinical research is crucial to establish their scientific validity. Integrating Unani medicine with modern research could offer valuable insights for the holistic management of neurodegenerative disorders, including GBS.

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