IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 6, Issue 10, May 2026
Impact Factor: 8.2

Bickerstaff Encephalitis: Clinical Insights and Management

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Abstract: *Bickerstaff's brainstem encephalitis is marked by eye movement weakness (ophthalmoplegia), poor coordination (ataxia), and changes in consciousness. It is similar to Miller Fisher syndrome, a type of Guillain-Barré syndrome, as both conditions share symptoms like eye movement problems and loss of coordination. The difference is that people with Bickerstaff's brainstem encephalitis have reduced awareness or confusion, while those with Miller Fisher syndrome stay alert but lose their reflexes. Here, we describe the case of a 3-year-old child diagnosed with Bickerstaff's brainstem encephalitis, showing typical symptoms and notable brain scan findings. It is hard to diagnose Bickerstaff's brainstem encephalitis (BBE) during the early stage. In the emergency room, doctors may mistake it for an unknown cause of unconsciousness or confusion.*

Keywords: Guillain–Barré syndrome (GBS) spectrum, Fisher syndrome (MFS) overlap,Anti-GQ1b antibody, Immunopathogenesis, Neurological examination, Cerebrospinal fluid (CSF) findings, MRI brainstem lesions, Differential diagnosis, Electrophysiological studies, Diagnostic criteria.

I. INTRODUCTION

Historical Background:
Bickerstaff encephalitis (BE) was described in 1951 as an acute post-infectious demyelinating disease presenting with progressive ophthalmoplegia and ataxia . The original cases followed an epidemiologic pattern of clusters and outbreaks. Currently the syndrome is recognized as a sporadic, monophasic illness. To date, only one case of BE relapsing less than three years from onset has been reported.

Introduction
Bickerstaff encephalitis (BE) was first defined in 1951 as an encephalitis that causes ophthalmoplegia and ataxia. It has remained relatively scarce since the initial report, and there is limited information regarding the severity, acuity, and progression of the encephalitis. BE is an immune-mediated disorder, similar to the Guillain–Barré syndrome and Miller Fisher syndrome, which shares geographical, seasonal, clinical, immunological, and prognostic features with both entities. The immune process involved affects both the central and peripheral nervous systems, accounting for the clinical picture that can be observed in patients .

Epidemiology
Bickerstaff encephalitis (BE) is a rare neurological disorder with an incompletely characterized epidemiology . The disease develops predominantly in middle-aged adults but has an estimated annual incidence of only 0.078 per 100,000 person-years. Several case reports indicate increased risk among patients with preceding infections, particularly by Campylobacter jejuni and Haemophilus influenzae. BE does not appear to exhibit a predilection for any particular gender, and no specific race or ethnicity has been identified as being at increased risk.

DOI: 10.48175/IJARSCT-35710

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Pathophysiology

Bickerstaff encephalitis (BE) is a rare autoimmune disorder that impacts the brainstem. It presents with symptoms including ophthalmoplegia, ataxia, altered consciousness, and sometimes hyperreflexia or the Babinski sign. Neurological symptoms arise from brainstem lesions identified through neuroimaging.The pathogenesis of Bickerstaff brainstem encephalitis (BBE) remains uncertain, although it is considered a postinfectious, immune-mediated disease. BBE shares clinical features with Guillain-Barré syndrome (GBS) and Miller Fisher syndrome (MFS), which have similar adverse events following vaccination due to molecular mimicry between microbial and nerve molecules. Serum IgG anti-GQ1b antibodies in high titres are present in MFS, indicating a specific immune response against the epitope, and these antibodies are present in a comparable percentage of BBE and GBS cases with ophthalmoplegia. BE may be understood as an autoimmune disease involving the central and peripheral nervous systems.

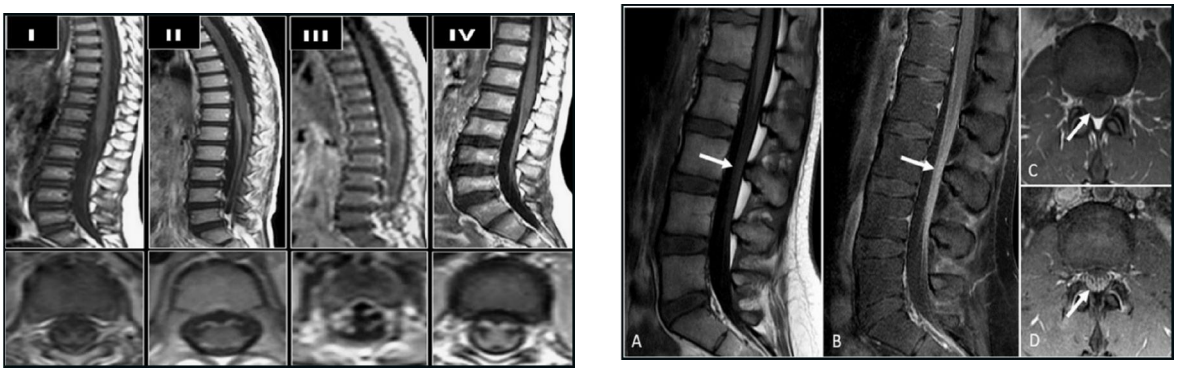

Fig. Guillain-Barr'e Syndrome

II. CLINICAL PRESENTATION

Bickerstaff encephalitis (BE) encompasses autoimmune neurological disorders triggered by an immune response post-infection. Unlike typical autoimmune conditions, postinfectious Guillain-Barré syndrome (GBS) generally lacks a chronic-exacerbating form. Since 2013, GBS classification considers patient history and diseases phases, which may be progressive or fluctuating. BE typically occurs during the complementary phase after ether exposure, presenting symptoms such as ophthalmoplegia, ataxia, and altered consciousness, often with an early fever. Permanent severe cerebral or spinal deficits may emerge, along with dysautonomias and rare extrapyramidal symptoms. Symptoms can extend from the brainstem to peripheral nerves, leading to a dysautonomia syndrome. One case reported symptoms appearing after relocation.

2.1. Neurological Symptoms

Patient presented with fever, bilateral external ophthalmoplegia, and ataxia. Bickerstaff encephalitis (BE), first described in 1951, is characterized by acute ophthalmoplegia, ataxia, and disturbance of consciousness, believed to be an immune-mediated disorder affecting the CNS. BE shares similarities with Fisher syndrome (FS) and Guillain–Barré syndrome (GBS). Typical BE cases exhibit the three main features alongside CNS involvement, such as pyramidal signs or altered consciousness. Acute ophthalmoplegia alone is a subtype of FS. Consciousness disturbance in BE often involves drowsiness or stupor, with limb weakness indicating GBS overlap. BE typically has a monophasic course, responds well to immunotherapy, and generally has a favorable prognosis. A case of chronic BE is noted.

2.2. Systemic Symptoms

In suspected Bickerstaff encephalitis (BE), systemic symptoms may occur alongside neurological features. A pediatric case presented with high fever upon hospital admission, while other vital signs, including blood pressure, heart rate, and respiratory rate, were normal. The clinical course involved persistent fever and worsening neurological

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impairments requiring intensive care. Neutrophil-predominant leukocytosis was noted, with white blood cell counts reaching about 13,000 cells/μL. Cerebrospinal fluid analysis showed pleocytosis with increased WBCs and elevated protein. Serologic tests for Enterovirus and herpesvirus returned negative. BE generally does not link to chronic systemic symptoms or cognitive decline, aiding its distinction from Bickerstaff encephalopathy.

III. DIAGNOSIS

The diagnosis of Bickerstaff encephalitis relies on clinical criteria, neuroimaging, and laboratory tests. It is characterized by acute onset within 4 weeks of ataxia, along with either significant disturbance of consciousness or hyperreflexia. Neuroimaging typically shows abnormal high intensity lesions on T2-weighted images or DWI maps, often with mild gadolinium enhancement in the brainstem, cerebellum, cerebrum, or thalamus. Laboratory tests usually indicate cerebrospinal fluid pleocytosis (10–40/mm3), normal CSF protein levels, or low titres of antiGQ1b antibodies.

3.1. Clinical Criteria

Bickerstaff's encephalitis (BE) manifests as acute progressive symmetrical external ophthalmoplegia and ataxia within four weeks, accompanied by disturbance of consciousness or hyperreflexia . BBE, a related disorder, requires acute progressive ophthalmoplegia, ataxia, and impaired consciousness within four weeks, with recovery within twelve weeks; positive serum IgG anti-GQ1b antibodies; and exclusion of alternative diagnoses such as cerebrovascular disease, multiple sclerosis

3.2. Neuroimaging

Bickerstaff encephalitis, like other immune-mediated disorders such as Guillain–Barré and Miller Fisher syndromes, is primarily triggered by infections, notably Campylobacter jejuni and Haemophilus influenzae. This activates the immune response, leading to autoantibodies against gangliosides in nerve membranes and myelin proteins. The pathology includes complement activation, membrane attack complex formation, and potential peripheral nerve axon dysfunction or destruction.

Bickerstaff encephalitis features a gradual onset of ascending neurological deficits such as ophthalmoplegia, ataxia, and altered consciousness. Systemic symptoms include fever, headache, vomiting, and nausea, indicating central nervous system infiltration by a dysregulated immune response.

3.3. Laboratory Tests

Laboratory tests support the diagnosis of Bickerstaff encephalitis (BE) and assist in differential diagnosis. Routine analyses like blood counts, blood biochemistry, urine tests, and CSF analysis are typically normal or mildly abnormal. CSF analysis may show slight protein elevation with a normal cell count, though pleocytosis can occur. Intrathecal IgG synthesis is rare. Immunological investigations improve diagnostic specificity. Anti-GQ1b IgG antibodies are closely linked with the disease and its variants, like Miller–Fisher and Guillain-Barré syndromes, usually positive in BE patients. Identifying anti-GQ1b, anti-GD1a, and anti-GT1a IgG antibodies is vital, as humoral immunity significantly impacts pathogenesis. A prepontine or lumbar puncture is advised for CSF and serum analysis.

BE diagnosis remains primarily clinical with support from neuroimaging and laboratory investigations. Nevertheless, these analyses are crucial to exclude other disorders that may present with similar manifestations.

IV. DIFFERENTIAL DIAGNOSIS

The differential diagnosis of BE includes encephalitic conditions like herpes, botulism, or syphilis, differentiated by cerebrospinal fluid (CSF) findings and paraclinical characteristics. Common clinical patterns in BE, Miller Fisher syndrome (MFS), and the pharyngeal-cervicalbrachial variant of Guillain–Barré syndrome (GBS)—such as ophthalmoplegia, ataxia, and areflexia—complicate diagnosis. GBS shows elevated CSF protein with normal cell

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counts, while BE has increased CSF cell counts. Both may involve postinfectious autoimmune mechanisms, with antiganglioside antibody testing aiding diagnosis.

4.1. Other Encephalitides

Bickerstaff's brainstem encephalitis (BBE) is an autoimmune disease affecting the brainstem, often following an infection. Most patients have IgG anti-GQ1b antibodies. Symptoms include oculomotor disorders, ataxia, and consciousness impairment. BBE is a variant of Guillain–Barré syndrome (GBS) and shares features with Miller-Fisher syndrome, such as ophthalmoplegia and loss of tendon reflexes. Diagnosis requires acute external ophthalmoplegia, ataxia, and consciousness disturbance within four weeks, with supporting positive serum IgG anti-GQ1b antibodies. Other conditions like cerebrovascular disease must be excluded. Cases are "definite" when all symptoms and positive antibodies are present; "probable" if some symptoms exist with confirmed antibodies or exclusions.

4.2. Autoimmune Disorders

Two autoimmune illnesses can present with ophthalmoplegia, ataxia, and impaired consciousness: Bickerstaff's brainstem encephalitis and Fisher syndrome. Fisher syndrome, a variant of Guillain-Barré syndrome, should be dismissed if findings show peripheral neuropathy rather than central nervous system issues. Both illnesses typically reveal positive serum titres of antibodies against ganglioside GQ1b. The variation in limb weakness challenges existing views, suggesting Bickerstaff's brainstem encephalitis may evolve via autoimmune mechanisms linked to Miller–Fisher syndrome and Guillain-Barré syndrome.

V. TREATMENT APPROACHES

Several treatment approaches have been proposed for Bickerstaff encephalitis (BE).

Immunotherapy includes intravenous immunoglobulin (IVIG) and plasmapheresis. Both strategies are well tolerated overall, with relatively similar levels of efficacy, and are sometimes combined . Supportive approaches are generally required and address intracranial hypertension, respiratory support, and enteral or parenteral nutrition.

5.1. Immunotherapy

The management of Bickerstaff encephalitis (BE) relies on clinical experience, paralleling other neuroimmunological disorders, rather than trials. Key treatments include intravenous immunoglobulins (IVIG) and plasmapheresis to modulate the immune response. IVIG is preferred for its safety, while corticosteroids are generally avoided. In severe cases, rituximab may help, and acetylcholinesterase inhibitors are often used for additional clinical benefits.

5.2. Supportive Care

Supportive care is crucial for recovery from Bickerstaff encephalitis (BE). Patients with severe respiratory failure may require ICU admission for assisted ventilation. Monitoring and managing complications, maintaining hydration and electrolyte balance, and ensuring proper nutrition are vital, enhancing treatment outcomes. Patients can improve over time, even with severe disabilities.

The complex neurological syndrome of BE features progressive ophthalmoplegia, ataxia, and impaired consciousness over about 4 days. Initially linked to Guillain–Barré syndrome, BE is related to GBS and Fisher syndrome and can occur independently. Patients with both conditions show a wide range of symptoms. Improvements in critical care allow many patients needing ventilation to recover fully.

VI. PROGNOSIS

Bickerstaff encephalitis (BE) typically follows a monophasic course, with improvement occurring gradually within weeks following treatment initiation . The duration of hospitalization often extends to one month, during which clinical and immunological symptoms respectively resolve within two and six months. Although favorable response to

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treatment remains the rule, isolated instances of poor prognosis have been reported, highlighting the necessity for rigorous supportive care and, in refractory cases, consideration of immunosuppressive strategies including rituximab

VII. LONG-TERM OUTCOMES

Long-term outcomes of Bickerstaff encephalitis (BE) remain incompletely characterised. Neurological sequelae detected years after typical BE suggest possible progression towards chronic syndromes, underscoring a need for further study of prolonged disease courses . A relationship to Guillain-Barré syndrome (GBS) and related syndromes supports the contention that BE forms part of a continuous autoimmune-spectrum disorder. Most documented cases of BE establish an excellent prognosis, characterised by complete recovery without long-term impairment . Given that disease represents a 4%.

7.1. Neurological Sequelae

Neurological sequelae in Bickerstaff encephalitis (BE) are common despite an immunemediated response. Residual symptoms often include cognitive, attentional, and behavioral changes. Brain MRI may not reveal brainstem involvement, but abnormalities are detectable in severely disabled patients. Testing for serum anti-GQ1b antibodies and CSF analysis aids diagnosis; about 66% of BE cases test positive for anti-GQ1b IgG, with over 90% showing elevated CSF protein. Most respond well to immunotherapy, but chronic BE can develop, leading to persistent impairments similar to chronic inflammatory demyelinating polyneuropathy.

7.2. Quality of Life

Patients can experience long-term neurological effects after acute Bickerstaff encephalitis (BE). Chronic BE is similar to recurrent Guillain–Barré syndrome, though definitions differ. A 57year-old liver transplant patient with BE and anti-GQ1b antibodies showed relapsing symptoms but improved with treatment. Quality of life declines during the acute phase due to fatigue and cognitive issues, with many returning to work but facing reduced capacity and increased anxiety.

VIII. CURRENT RESEARCH

Studies are examining brainstem encephalitis and interventions. A prospective study (NCT05008528) at Intel IRCCS explores Bickerstaff syndrome's link to infectious agentsseeking clinical features from autopsy-confirmed cases. Another study identifies biomarkers in serum and cerebrospinal fluid for neurodegenerative diseases, including Bickerstaff's, to indicate severity.A 2017 study of 83 patients with Bickerstaff's brainstem encephalitis or Fisher syndrome evaluated adverse events and guidelines, noting improvement with plasma exchanges and high-dose intravenous immunoglobulins. A 2024 report analyzed brainstem auditory evoked responses for diagnosis and explored rituximab for severe, unresponsive cases, suggesting further research into its efficacy.

8.1. Trials

Numerous trials have focused on Bickerstaff encephalitis (BE), an immune-mediated disorder causing acute ophthalmoplegia, ataxia, and altered consciousness, targeting autoantibodies against nerve antigens. A pediatric patient treated with intravenous immunoglobulin and corticosteroids showed significant recovery. A 44-year-old man with chronic BE improved after repeated treatments. Controlled trials are essential for optimal treatment regimens for BE.

8.2. Emerging Therapies

Plasma exchange and high-dose immunosuppressive treatments are second-line options for Bickerstaff encephalitis patients who have suboptimal responses to IVIG or corticosteroids. Current evidence mainly consists of isolated case reports, with plasma exchange being the preferred choice. IVIG combined with corticosteroids doesn't offer extra benefits compared to using either treatment alone.

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Human immunoglobulins play a crucial role in antiviral and immunomodulatory effects, being the sole treatment with potential benefits. This highlights the need for clinical trials to evaluate IVIG for Japanese encephalitis (JE). Previous small studies have shown inconclusive results, but ongoing trials seek to assess IVIG derived from pooled plasma, which may contain neutralizing antibodies to JEV from vaccinated donors.

IX. CASE STUDIES

Notable cases illustrate the management of a rare disorder. A healthy 39-month-old boy had stupor, fever, pharyngeal injection, bilateral ptosis, brisk reflexes, Babinski sign, and ataxia. CSF analysis revealed elevated white blood cells and protein, with negative viral tests. Initial treatments failed, leading to ICU transfer. MRI showed high signal intensity in the pons and cerebellum, while EEG indicated encephalopathy. Immunoglobulin and steroids improved his condition, leading to a diagnosis of Bickerstaff's brainstem encephalitis. In another case, a 44year-old man had cognitive impairment and weakness post-diarrhea. Neuropsychological evaluation showed memory deficits, with CSF indicating albuminocytological dissociation. Immunological tests were positive for anti-ganglioside antibodies. Although initial immunoglobulin therapy was effective, clinical worsening required more treatments, suggesting chronic Bickerstaff's encephalitis. This underscores the need for ongoing research and early diagnosis for effective therapy and rehabilitation.

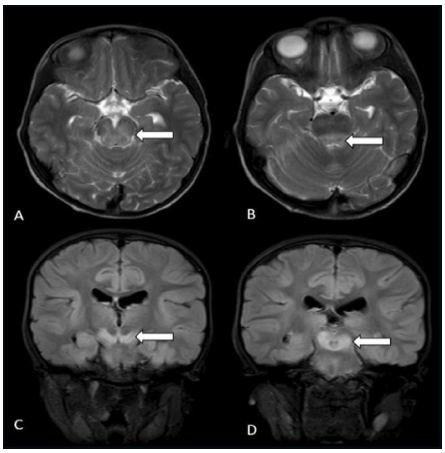

Fig. Bickerstaff's Encephalitis

9.1. Notable Cases

Bickerstaff encephalitis (BE) is a rare immune-mediated neuropathy impacting the central nervous system, defined by acute ophthalmoplegia, ataxia, and altered consciousness. A 34-yearold woman displayed diplopia, balance issues, fever, somnolence, and brisk reflexes. Positive for IgG anti-GQ1b antibody and influenza, she had an MRI revealing abnormal signals. CSF analysis showed pleocytosis and elevated protein. Treatment with corticosteroids and IVIG resulted in significant recovery and resolution of imaging abnormalities over two months, marking the first report of BE concurrent with influenza and highlighting molecular mimicry's role.

9.2. Learnings

Clinical awareness of Bickerstaff encephalitis (BE) is of importance. Since the aetiology is considered to be immune-mediated, early treatment with immunotherapies aims to reduce the antibody load and the severity of the inflammatory damage. Signs and symptoms of the disease are mild and resolve rapidly in most cases, so supporting a positive outcome . However, in certain circumstances neurological complications due to demyelination may remain .

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X. PUBLIC HEALTH IMPLICATIONS

Awareness and education programs are important for the early detection, diagnosis, and timely treatment of Bickerstaff encephalitis (BE).Preventive strategies through the prevention of infectious diseases could help reduce cases of BE.

10.1. Awareness and Education

Bickerstaff encephalitis (BE) is an uncommon but serious neurological syndrome characterized by an impaired level of consciousness, ataxia with an inability to coordinate muscle movements, and ophthalmoplegia or paralysis of the movements of the eyes Awareness of this potentially life-threatening illness is crucial for early diagnosis and timely treatment, which include intravenous immunoglobulin, corticosteroids and plasmapheresis. This paper provides an overview of the clinical features, diagnostic criteria and therapeutic approaches to BE in order to raise awareness among the clinical and scientific community

10.2. Preventive Strategies

Preventive measures for Bickerstaff encephalitis (BE) are currently unclear due to the limited understanding of its exact causes and risk factors. BE is a rare, immune-mediated disorder that affects the brainstem, characterized by symptoms of ophthalmoplegia, ataxia, and altered consciousness. It is closely related to Guillain–Barré syndrome and its variant, Miller Fisher syndrome. Reported outcomes vary widely, ranging from complete recovery to severe neurological impairment and death.

Because BE is an immune-mediated disease, people with abnormal immune responses may be more susceptible. Further research is needed to fully appreciate the multiple facets of BE and devise strategies that might mitigate infection risk.

XI. FUTURE DIRECTIONS

New strategies must be envisaged for the unexpected chronic form of Bickerstaff encephalitis (BE) reported in 2014, which differs from the monophasic pattern often observed . BE generally requires no long-term maintenance therapy though low-dose corticosteroids or immunosuppressant medication is sometimes necessary.

XI.1. Research Gaps

A comprehensive understanding of Bickerstaff encephalitis (BE) still eludes the medical community, necessitating further research. The potential for a chronic form marked by cognitive impairment warrants examination . Standard diagnostic tools may not capture all disease aspects, as cases have demonstrated, pointing to the diagnostic value of auditory brainstem response (ABR) analysis for lesion detection and prognosis . Epidemiological data remain sparse, impeding a full grasp of incidence and risk factors. Detailed characterization of the pathophysiological mechanisms by which anti-GQ1b antibodies disrupt neural conduction is also incomplete. Moreover, a systematic appraisal of long-term neurological sequelae and quality-oflife outcomes after BE episodes is lacking. Emerging immunotherapeutic agents require rigorous evaluation to establish efficacy relative to conventional immunoglobulin and plasmapheresis approaches. Attention to these gaps will inform clinical practice and guide patient care improvements.

XI.2. Potential Innovations

Although Bickerstaff encephalitis (BE) is extremely rare, it is a critical construct to consider for patients with brainstem involvement and a preceding infection; appropriate treatment can lead to a good prognosis. However, when anti-GQ1b antibodies are detected, the diagnosis may lean toward Miller Fisher syndrome, as encephalopathy generally excludes the latter despite the continuous spectrum between the syndromes. To validate the proposed clinical approach, larger prospective studies confirming the association of CNS features with prognosis and clarifying the pathological mechanism of BE are necessary. Should future studies demonstrate that central nervous system (CNS) involvement

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leads to a more severe clinical picture, these findings would significantly influence treatment strategies and prophylaxis.

XII. CONCLUSION

In summary, Bickerstaff encephalitis is a rare, immune-mediated inflammatory disorder of the central nervous system characterized by acute brainstem dysfunction, consciousness disturbances, and ophthalmoplegia. Because of its overlapping clinical features with Miller Fisher syndrome and Guillain–Barré syndrome, Bickerstaff encephalitis is often considered part of a continuous clinical and pathophysiological spectrum that connects these syndromes. Despite its rarity, the illness usually follows a monophasic and self-limiting course. Although some patients reach full recovery, the majority continue to experience mild neurological symptoms, while severe sequelae remain uncommon. Effective treatment with corticosteroids, plasmapheresis, or intravenous immunoglobulin is associated with reduced mortality rates.

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