RSV-induced Guillain–Barré Syndrome: A Case Report

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Abstract

A patient with a respiratory syncytial virus-induced Guillain–Barré Syndrome and acute disseminated encephalomyelitis is presented. This virus is the most common cause of upper respiratory infections, and it can become an etiology for extrapulmonary pathology with serious complications. Although rare, the possibility of adverse comorbidities makes early diagnosis and treatment of these cases critical.

Introduction

Guillain–Barré syndrome is characterized by weakness, numbness, and paresthesia in peripheral and cranial nerve-innervated musculature.[1] It often affects people following bacterial or viral infection with an autoimmune process, inducing demyelination and axonal damage.[2] The annual US incidence of Guillain–Barré syndrome is 1.2–3 per 100,000 inhabitants.[3] Approximately two-thirds of cases follow upper respiratory infections or diarrhea, 30 percent are subsequent to Campylobacter jejuni, and 10 percent are induced by cytomegalovirus.[4, 5] As of July 2021, over 100 cases of Guillain–Barré syndrome have been reported to the Vaccine Adverse Event Reporting System (VAERS) in recipients of the Ad26.COV2.S vaccine (developed by Johnson & Johnson) among 12.5 million doses administered in the US.[6]

Respiratory syncytial virus (RSV) infections commonly cause seasonal outbreaks of respiratory illness in persons of all ages. Those considered high-risk populations experience increased incidences of mortality. These high-risk populations include infants, young children, adults with chronic medical conditions, and older adults.[7, 8] Although RSV commonly affects the lungs, extrapulmonary pathologies may also occur in these high-risk populations. Extrapulmonary complications may occur in the brain, liver, heart, and kidneys.[9]

Case Description

A 44-year-old man presented to the emergency department with a one-month history of intermittent fever, cough, and congestion. After remission of his symptoms, he visited Spain and India for a few weeks; his symptoms re-emerged upon returning to the United States. Unspecified antibiotic, steroid, and bronchodilator therapies were prescribed for upper respiratory infection but were ineffective. He felt worse, experiencing fatigue, malaise, headache, and gait imbalance. A blurry vision emerged in his right eye, accompanied by pain with extraocular movements. Initially, an emergency department evaluation was inconclusive, including a normal complete blood count, metabolic panel, and erythrocyte sedimentation rate. The computerized tomographic (CT) image of the orbit and head CT angiogram were remarkable for left maxillary sinusitis. This condition was managed with antibiotics and pain medications. The patient was referred to the ophthalmology clinic, where binocular diplopia was documented.

Over the next two days, his condition worsened with slurred speech, numbness in the face and extremities, and difficulty voiding. The patient was hospitalized, where he exhibited several neurologic deficits, including orbicularis oculi and oris weakness, bilateral intrinsic hand muscle weakness, dyscoordination, and diminished sensation in the hands and feet. The presumptive diagnosis was meningitis, for which antiviral
and antibiotic drugs (acyclovir, ceftriaxone, and vancomycin) were prescribed.

Laboratory results, including complete blood count, metabolic panel, vitamin B12 level, thyroid-stimulating hormone, creatinine kinase, and blood cultures, were unremarkable. Screening for syphilis, human immunodeficiency virus, and West Nile were routine, as were a meningitis/encephalitis serum panel and an immunofixation ganglioside antibody panel. A cerebrospinal fluid study was negative for cultures, glucose concentration, and antinuclear antibodies. Testing for RSV was positive. Reimaging confirmed the left maxillary sinusitis. Magnetic resonance imaging (MRI) of the brain (Figure 1) was performed. MRI of the thoracic spine revealed leptomeningeal enhancement and degenerative changes in the lumbar region, two nonspecific findings indicating possible pathology. Since meningitis was deemed unlikely by brain MRI, antibiotic and antiviral drugs were discontinued.

A diagnosis of Guillain–Barré syndrome secondary to the respiratory syncytial virus was made at this time. The patient then received treated with intravenous immunoglobulin (IVIG), 1 g/kg. Within two days, there was an improvement—especially in ocular motor strength. However, he also developed hiccups, sinus pauses, and bradycardia, presumably secondary to autonomic nervous system involvement. He regained bladder function during the next week and improved his dyspnea and fatigue with persisting leg numbness. Still improving, he was discharged to a rehabilitation facility.

After two months, the patient was re-hospitalized due to more urinary retention, paresthesia, ataxia, and hiccups. On physical examination, a decreased sensation was noted in all extremities. Additionally, dermatomal thoracic pain and sensory loss across the abdomen and both arms were also noted. A chronic inflammatory demyelinating polyradiculoneuropathy was among the differential considerations, and IVIG therapy continued. MRI of the brain (Figure 2) demonstrated an expansive abnormality of the medial left temporal lobe, bilateral caudate nuclei, bilateral thalami, left basal ganglia, and left ventral midbrain; these findings were not visualized in previous imaging. These abnormalities were consistent with acute disseminated encephalomyelitis. The patient responded to treatment with IVIG as his hiccups, urinary retention, and paresthesia continued to resolve.

Further workup was performed. Electromyography was normal. Numerous markers of autoimmune processes were within normal limits, such as neuromyelitis optica (NMO) Aquaporin-4 Receptor Antibody (AQP4-Abs), myelin-associated glycoprotein immunoglobulin (MAG(Ig)M), myelin-oligodendrocyte glycoprotein an-
tibodies (MOG-Abs), B-lymphocyte antigen CD20 (CD 20), thiopurine methyltransferase tests, antinuclear antibodies, and antithyroid antibodies. However, the glutamic acid decarboxylase 65, a common marker of type 1 diabetes mellitus, was elevated to 0.07 nmol/L. Moreover, the anti-GD1a antibody, a marker of numerous motor syndromes, was elevated to 64 u/L.

At two and five months, MRIs were performed and demonstrated complete resolution of the spinal and brain lesions (Figure 3). Clinically, the patient has remained asymptomatic for over a year. The definitive diagnosis was GD1a antibody–associated RSV-induced Guillain–Barré syndrome with post-infectious acute disseminated encephalomyelitis.

Discussion

RSV infection activates cytokines, arachidonic acid, interleukins, tumor necrosis factor, and platelet-activating factors. The etiology of neural disruption remains unclear; however, it has been proposed that these inflammatory mediators may play a significant role in this process.[10] Associations between RSV and Guillain–Barré syndrome with neurologic involvement usually occur among children.[11]

Typical features of Guillain–Barré syndrome include symmetrical weakness in the legs with absent or weak deep tendon reflexes. Up to 90 percent of patients experience paresthesia, and nearly 70 percent demonstrate dysautonomia with urinary retention. About 10 percent report weakness of extremities and up to 30 percent in respiratory musculature. Other manifestations can include facial nerve palsy, oropharyngeal or oculomotor weakness, diarrhea or constipation, and tachycardia; hyponatremia is also observed.[12, 13] Guillain–Barré syndrome reportedly also appears in other variants, such as acute inflammatory demyelinating polyradiculoneuropathy, Miller Fisher syndrome, acute motor axonal neuropathy, and acute motor and sensory axonal neuropathy.[14]

Cerebrospinal fluid testing often helps to exclude other etiologies rather than to confirm Guillain–Barré syndrome. Brain imaging is less valuable to assist in diagnosis. Glycolipid antibodies are not reliably diagnostic for Guillain–Barré syndrome.[12] There are various criteria for recognizing Guillain–Barré syndrome and its variants.[15, 16] Currently, nerve conduction studies are the “gold standard” for diagnosing Guillain–Barré syndrome.[17] One case report documented Guillain–Barré syndrome caused by RSV in an 81-year-old immunocompromised woman.[18] Her illness began with weakness and required respiratory assistance; she died despite treatment with plasmapheresis.
Identifying Guillain–Barré syndrome and its variants is difficult in patients with atypical presentations. Clinical features help to confirm diagnoses; there are no diagnostic biomarkers. Early treatment is vital to minimize neurological complications, including dangerous, quickly developing apneas. Some affected individuals require respiratory assistance. Diagnostic precision is paramount.

References


