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Case Report
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Inflammatory Polyradiculopathy Associated with Influenza B Infection

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Abstract

Influenza B is a common respiratory infection that typically presents with upper respiratory tract symptoms and systemic symptoms. Neurological complications due to influenza B infection are rare. This case report describes a 54-year-old male who presented neurological complication post viral illness. He was screened for infectious, inflammatory, neoplastic, paraneoplastic and metabolic causes all of which came back negative apart from Influenza B infection and chronic Hepatitis C infection. The radiological imaging was diagnostic of inflammatory polyradiculopathy involving multiple cranial nerves and the brachial plexus. His neurological symptoms gradually improved following a course of Tamiflu. We hereby present a case of inflammatory polyradiculopathy post Influenza B infection. This case highlights the importance of considering neurological complications in patients with influenza B infection and the need for timely diagnosis and management.

Introduction

Influenza B is a common cause for upper and lower respiratory tract infection. It mainly presents with upper respiratory tract symptoms like rhinorrhea, dry cough, and nasal congestion along with systemic symptoms like generalized fever, headache, malaise and myalgia. Neurological complication due to influenza B infection is very rare. We report a case of polyradiculopathy post influenza B infection.

Case Presentation

A 54-year-old gentleman presented to Emergency Department as he woke up with new left-hand numbness and weakness, drooping of left eye associated with double vision and right sided facial weakness. These symptoms were preceded with a two-day history of fever, cough and myalgia. He is generally fit and well with no past medical history. He denied having his annual flu vaccine.

He was initially seen by the stroke team in Emergency Department for a presumed stroke and given aspirin. Initial CT head showed no intracranial pathology and the subsequent MRI confirmed this. He was referred to neurology. Neurological examination showed he had a left cranial nerve III and VI palsy and right cranial nerve VII (LMN) palsy, with weakness of power in his left hand, there was no sensory deficit, ataxia or cerebellar signs. His deep tendon reflexes were normal and plantars were flexor.

Investigations

A nasopharyngeal swab was sent for respiratory viral screen on admission which tested positive for influenza B and was negative for all other viruses including SARS-CoV-2. MRI head and spine with contrast suggested inflammatory polyradiculopathy involving the right facial and right abducens and lower cervical nerve roots (C7 and C8). The subsequent MRI of the brachial plexus confirmed inflammatory polyradiculopathy involving the left C7 and C8 cervical nerve roots, the inferior trunk of the left brachial plexus and the right facial nerve. Nerve conduction studies further reiterated brachial plexus involvement on both sides with medial cord (C8) dysfunction that is mainly sensory on the left, with moderate left median nerve involvement and severe left radial nerve involvement, and sparing of more proximal muscles. The right radial territory (C7) was also involved. He also had a whole-body CT scan which excluded any evidence of malignancy.

His blood results on admission showed a normal white cell count of 7.9×10^9/L, thrombocytopenia with platelets of 109×10^9/L and C- reactive protein of <5 mg/L, he also was tested negative for *Borrelia*, Epstein Barr virus, Hepatitis B, *Cytomegalovirus*. However, his Hepatitis C virus antibody

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was positive, HCV RNA titer was slightly raised at 5.74 Log IU/mL. His autoimmune screen was tested negative for ANCA, ANA, complements, myeloma screen and normal ACE levels. His lumbar puncture study showed clear Cerebrospinal Fluid (CSF), CSF glucose 3.6 mmol/L and protein 0.29 g/L, CSF viral PCR was negative and CSF culture showed no growth. Furthermore, no oligoclonal bands were detected in the CSF.

Recovery

During his inpatient stay, he was started on a course of Tamiflu for 5 days. The cranial nerve palsies improved gradually as well as his left-hand strength. His neurological symptoms completely resolved by day 7 of his admission and he was discharged home.

Discussion

We report a unique case of a patient with multiple cranial neuropathies and inflammatory polyradiculopathy including brachial plexus with Influenza B infection. In view of his smoking history and sudden onset of symptoms he was suspected of stroke but following radiological study did not confirm this hence was investigated further. As he had preceding viral flu like illness, he had nasal swab test for the respiratory virus which confirmed Influenza B. The radiological study, CSF study and nerve conduction study confirmed multiple cranial neuropathy and lower cervical root involvement including brachial plexopathy.

In view of his given symptoms, he was screened for infectious, inflammatory, neoplastic/paraneoplastic and metabolic causes of polyneuropathy. All the investigations came negative except Influenza B in PCR swab test and chronic Hepatitis C infection.

Influenza viruses are negative single-stranded RNA viruses belonging to the family *Orthomyxoviridae* and cause worldwide epidemics [1]. The *Orthomyxoviridae* family is composed of five genera: Influenza virus A, Influenza virus B, Influenza virus C, *Thogotovirus*, and *Isavirus* [2]. Human influenza A and B viruses cause a self-limited acute respiratory infection. Influenza types B and C primarily infect humans; however, the severity of illness is relatively mild compared to influenza A viruses [1].

Severe complications of influenza can involve the lower respiratory tract (pneumonia), heart (myocarditis), and central nervous system (encephalitis, myelitis, meningitis, febrile and afebrile seizures, Guillain-Barré syndrome, cerebellar ataxia) and can lead to death [3-5].

Influenza A and B viruses might induce neurological complications, but most published cases are focused on influenza A viruses. Influenza B virus is known to cause mild respiratory illness with less frequent neurological complications than influenza A [1]. The earliest case report of influenza B viral encephalitis was in London, UK, in 1946 [6].

A study from UK National Health Service hospitals reporting cases of GBS from 1993 to 2002 found an association between laboratory-confirmed influenza A in any given week and GBS hospitalization in the same week. However, they were not able to link the actual influenza virus infection to the individual with GBS [7]. The influenza viruses A and B are enveloped by a lipid layer that includes various glycoproteins. Antibodies against glycolipids may be produced as a result of influenza virus infection due to the possible molecular mimicry between the glycoproteins of the influenza viruses and the glycolipids found in the human peripheral nerves [8].

Patients developing influenza A or B infections commonly experience diffuse myalgias or muscle aches and pains early in the illness [9]. The myalgias seldom interfere with muscle strength but can be associated with transient serum elevations of creatine kinase [10]. The myalgias correlate with the severity of the influenza, last several days, and resolve along with the other symptoms during recovery.

Given the rarity of neurological manifestation post influenza B infection in adults, we would like to raise awareness for polyradiculopathy post Influenza B infection and advocate for increased vigilance of clinicians worldwide.

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