

## Case Report

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# H1N1 Associated Encephalopathy in an Adult: Response to Intravenous Immunoglobulin Supporting an Autoimmune Pathogenesis

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### Abstract

**Background:** Influenza H1N1 infection in adults is rare and neurological involvement has been reported in only a few cases.

**Case report:** We present a young woman whose initial diagnosis was viral encephalitis because she presented with headache, confusion and seizures. She was admitted and treated with acyclovir, ceftriaxone and phenytoin but however deteriorated. She was transferred to the Intensive Care Unit (ICU) where she was intubated and sedated. She was then treated with intravenous immunoglobulins (IVIg) on the presumptive diagnosis of autoimmune encephalopathy. Influenza H1N1 infection was not considered as a diagnosis but her routine viral serology obtained during admission showed a rising titre to Influenza H1N1.

**Conclusion:** Influenza H1N1 encephalopathy is rare in adults. This report supports an autoimmune pathogenesis.

### Clinical Features

Our patient is a 22 year old lady admitted with a one-week history of headache, sore throat and fever, and a day history of confusion. There is no history of recent travel, immunisations, or illicit drug use. She was agitated and not responsive to commands. She had spontaneous eye opening and was moving all her limbs and localising to pain. She developed seizures whilst on admission. Her computerised tomography (CT) head scan was normal. Lumbar puncture (LP) showed an opening pressure of 21 cm H<sub>2</sub>O, with 38 white cells per mm<sup>3</sup>, (95% lymphocytes). Cerebrospinal fluid (CSF) protein was 77.6 g/dL, glucose was 3.3 mmol/L. The CSF polymerase chain reaction (PCR) was negative for adenovirus, cytomegalovirus (CMV), herpes simplex virus (HSV), varicella zoster virus (VZV), human herpes virus 6 (HHV6), meningococcus and pneumococcus. Magnetic resonance imaging (MRI) showed a small focus of white matter edema in the splenium of the right corpus callosum (Figure 1). Anti-nuclear antibody (ANA) and anti-neutrophil cytoplasmic antibody (ANCA) were negative. Computerised tomography scan of her chest, abdomen and pelvis was normal.

### Management and Progress

She was initially treated with intravenous acyclovir, ceftriaxone and phenytoin. Her level of consciousness however deteriorated; she became stuporous and was admitted to the Intensive Care Unit (ICU) where she was sedated and intubated. Examination at this point showed papilledema with a high blood pressure of 180/110 mmHg and a relatively low pulse rate of 80 beats per minute. Repeat LP showed an opening pressure of greater than 40 cm H<sub>2</sub>O with 94 WCC (100% lymphocytes). She did not improve with the addition of intravenous methylprednisolone. On the consideration that she may have autoimmune encephalopathy she was treated with a five day course of intravenous immunoglobulins (IVIg) at a dose of 0.4 g/kilogram body weight per day.

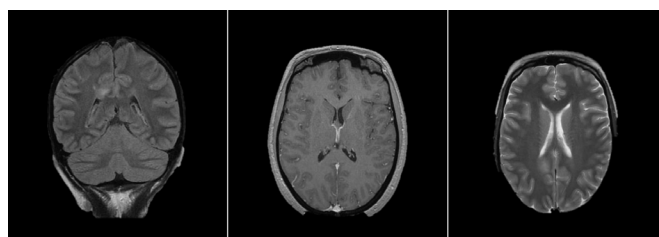
She improved over the next few days and was discharged from the ICU five days later. She made a complete recovery and was discharged home.

Virology results received after discharge showed a significant rise in H1N1 influenza titre, from undetectable to 1:64. Anti N-methyl-D-aspartate (NMDA) receptor and antinuclear (ANA) antibodies were negative. Cerebrospinal fluid (CSF) viral studies including HHV6 and varicella were negative. Blood virology and microbiology were negative.

### Discussion

Influenza H1N1 commonly affects children between the ages of 2 and 10 years. About 6% present with neurological features like seizures, encephalopathy, ataxia, movement disorders and neuropsychiatric manifestations [1]. There are only a few reports of H1N1 involving the nervous system in adults [2,3]. In a national surveillance study in Britain, four out of 25 cases of H1N1 with neurological features were adults; these had encephalopathy associated with a movement disorder or encephalitis [4]. Our patient fulfilled the Centers for Disease Control and Prevention (CDC) diagnostic criteria for neurological involvement by H1N1 infection [3]. The criteria stipulate laboratory-confirmed influenza A (H1N1) virus infection associated with seizures, encephalopathy, or encephalitis within 5 days of influenza-like illness (ILI), and without evidence of an alternative etiology [3]. The MRI findings in H1N1 encephalopathy include necrotising encephalopathy, posterior reversible encephalopathy and, as in our patient, callosal lesions typically located in the splenium [1,5-8].

Influenza H1N1 encephalopathy is considered to be the result of an autoimmune response for the following reasons: the neurological manifestations are delayed; there are no detectable viruses or antibodies in the CSF; there are very high haemagglutinating and neutralizing antibody titers; there are normal cytokine levels in serum and CSF; and there is a good response to IVIg [9-11].



**Figure 1:** Magnetic resonance imaging (MRI) of the brain showing high signal lesion in the splenium of the corpus callosum.

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Received March 17, 2015; Accepted November 15, 2015; Published November 20, 2015

**Citation:** Imam I, Turner P (2015) H1N1 Associated Encephalopathy in an Adult: Response to Intravenous Immunoglobulin Supporting an Autoimmune Pathogenesis. J Neuroinfect Dis 6: 193. doi:10.4172/2314-7326.1000193

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Our patient made a good outcome. This is against the typically poor prognosis reported in 84% of subjects with neurological involvement by H1N1 influenza infection [4]. Her favourable outcome was most probably a result of treatment with IVIg which is not usually prescribed in the previous case reports.

## Conclusion

We present a case of Influenza H1N1 encephalopathy who responded very well to immune treatment with IVIg. This case supports an immune pathogenesis of Influenza H1N1 encephalopathy. Immune treatment should be considered in all cases of suspected Influenza H1N1 associated encephalopathy.

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**Citation:** Imam I, Turner P (2015) H1N1 Associated Encephalopathy in an Adult: Response to Intravenous Immunoglobulin Supporting an Autoimmune Pathogenesis. *J Neuroinfect Dis* 6: 193. doi:[10.4172/2314-7326.1000193](https://doi.org/10.4172/2314-7326.1000193)

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