

A possible case of encephalitis due to H1N1 infection

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Introduction

A new entrant novel H1N1 influenza A virus has already been acquired world wide attention. Globally 15174 deaths have been reported among confirmed H1N1 cases up to February 2010 (1). There were 642 confirmed cases and 48 deaths reported in Sri Lanka due to H1N1 influenza by April 2010 since first reported in June 2009 (2).

Complications expected with this novel infection is similar to seasonal influenza infection viz; exacerbations of underlying chronic infections, respiratory tract infections (otitis media, sinusitis,

pneumonia), neurological illnesses (encephalitis, encephalopathy, febrile seizures and status epilepticus), rhabdomyolysis, pericarditis, myocarditis and sepsis (3,4).

Encephalitis as a neurological sequel of H1N1 influenza was first reported in Texas during the last epidemic (5). Main causative agents of encephalitis reported in Sri Lanka are Japanese B encephalitis virus, herpes simplex virus (type 1) and rabies virus whereas seasonal influenza A and B causing fatal encephalitis are not reported.

Case Report

A 17-year old boy without any previous illness, presented to a general practitioner with cough, cold, fever and headache of two days. Fever subsided with symptomatic treatment but headache persisted. He was admitted to General Hospital Matara with generalized tonic clonic seizures lasting 45 minutes, with frothing and incontinence of urine on the 7th day of illness. He was treated with intravenous acyclovir, intravenous cefotaxime and antiepileptic drugs for six days. Cerebro-spinal fluid (CSF) revealed 03 lymphocytes but no polymorphs or red cells. CSF protein was 30 mg/dL while sugar was 82 mg/dL. CSF fluid was negative for Herpes Simplex and Japanese B encephalitis virology. Non-contrast CT scan of brain was normal and EEG showed generalized slow waves.

On 7th day he was transferred to Teaching Hospital Karapitiya for an urgent MRI scan of brain. On admission he was febrile and sedated. In the ward phenytoin sodium 18 mg/kg infusion, sodium valproate 200mg twice daily, phenobarbitone 10 mg/kg infusion and diazepam (IV) 5 mg bolus over 1 min were instituted as he developed repeated generalized tonic-clonic seizures.

Throat and nasal swabs were taken for H1N1 influenza virology on next day (15th day of illness). As real time polymerase chain reaction for H1N1 influenza virus became positive, oseltamivir 75 mg twice daily was given for 5 days. Patient developed repeated myoclonic jerks two weeks following admission to Karapitiya and clonazepam was introduced.

There was a dramatic improvement over the next few days. He was free of seizures and no focal neurological deficit was detected at discharge. He was back in school one month following discharge. During first four months of follow-up he had two attacks of generalized tonic clonic seizures, possibly due to poor compliance. Meanwhile some deterioration in his behaviour and school performances was noted.

Investigations

Full blood count was normal initially but later showed mild neutrophil leucocytosis. Serum Na, K and blood urea remained within the normal limits. Serum calcium and Mg levels were within the normal range at the beginning of illness.

C-reactive protein was between 1.2 to 3.1 mg/dL (normal < 0.8) during the illness. ECG showed sinus tachycardia while his chest radiography and echocardiography did not show any abnormalities.

EEG done during the illness showed generalised asymmetrical slow waves with occasional sharp waves which were initially thought to be due to herpes simplex encephalitis. Subsequent EEGs showed less slow waves and it became normal two months after discharge. MRI scan of the brain was normal except for the evidence of sinusitis in maxillary, ethmoid and frontal sinuses

Discussion

This 17-year old boy developed seizures and impaired level of consciousness, seven days following influenza like illness. Possible differential diagnosis included viral encephalitis, post-viral encephalopathy like acute disseminated encephalomyelitis or metabolic derangement. His CSF serology was negative for common viruses that cause encephalitis in Sri Lanka. During the early phase of illness renal and liver functions and serum electrolytes including Ca, Mg, Na and K were normal. EEG showed generalised slow waves compatible with encephalitic illness and neuro-imaging did not show any features suggestive of Herpes Simplex encephalitis.

Our patient had many similarities to four similar cases reported in Texas (5). All of them were males between 10 - 17 yrs and had influenza-like illness with seizures. In those cases encephalitis has been confirmed based on EEG changes of background slow waves. Three patients had sinusitis on imaging, similar to our case. All had positive H1N1 influenza virus in respiratory tract specimens and no other cause for encephalitis was found. However in our patient antigen test was done only on the 15th day of the illness and the possibility of nosocomial infection from H1N1 virus cannot be totally excluded. The fact that the seizures resistant to a combination of antiepileptics later subsided following anti- H1N1 therapy raises the possibility that this infection may be responsible for his clinical status.

We suggest that H1N1 influenza A infection should also be considered in patients with encephalitic illness associated with influenza-like illness, specially during an epidemic of H1N1 infections.

Case Reports

By maintaining vigilance, we hope, more cases will be detected during future epidemics to confirm this association.

References

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