*Case report*

# Influenza B infection, an underestimated killer – an experience at a Tertiary care Hospital – Sri Lanka

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**Key words:** Influenza B infection; severe viral pneumonia; viral myocarditis

## Abstract

Influenza B virus causes outbreaks that are generally less extensive and are associated with less severe disease than those caused by influenza A virus. Influenza B outbreaks are generally seen in schools, military camps and elderly homes. Even though influenza B causes mild disease, severe outbreaks have also been reported and can be associated with significant morbidity and mortality. We report here an outbreak of a rare severe influenza B virus infection which started in a family home causing death of one patient due to severe pneumonia and another needing ventilation due to severe myocarditis which then spread to involve the health care workers who were looking after the patient diagnosed as having influenza B. Eventually the outbreak was successfully contained preventing further spread. This illustrates the importance of having a high index of suspicion for the presence of influenza B virus infection as early initiation of treatment can prevent complications and death. It is also a reminder of the importance of taking good hygienic measures including hand washing and isolation of highly infectious patients.

## Introduction

Influenza B virus is a genus in the virus family Orthomyxoviridae. Influenza B viruses are only known to infect humans and seals,[1] giving them mild influenza. This limited host range is apparently responsible for the lack of Influenza B virus causing influenza pandemics in contrast with those caused by the morphologically similar Influenza A viruses as both mutate by both genetic drift and reassortment.[2] Further diminishing the impact of this virus in man, influenza B viruses evolve slower than influenza A viruses and faster than influenza C viruses.[3] Influenza B virus mutates at a rate 2 to 3 times lower than influenza A[4] but mutates at a rate that does not allow a long lasting immunity.

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Influenza B virus causes outbreaks that are generally less extensive and are associated with less severe disease than those caused by influenza A virus. Influenza B outbreaks are generally seen in schools, military camps and homes for the elderly.[5,6] It generally gives rise to a minor acute respiratory tract infection but rarely can give rise to severe pneumonia.[7] Many neurologic complications such as Reye's syndrome, transverse myelitis, Guillain-Barre syndrome and encephalitis[8], myocarditis[9] and arthritis[10] are well recognized. Even though influenza B virus causes mild disease, severe outbreaks have also been reported which can be associated with significant morbidity and mortality.[11]

We report here an outbreak of a rare severe influenza B virus infection which started at a home and then spread to involve the health care personnel who were looking after the sick patients.

**Case 1**

A previously healthy 60 year old male patient presented with a sore throat, productive cough with white sputum for 3 days and fever with shortness of breath of 1 day duration. His illness had started following a mild upper respiratory tract infection which was present in everyone in his house. On admission he was ill, febrile and dyspnoeic. Room air oxygen saturation was 92% with a respiratory rate of 40 breaths per minute. Auscultation of lungs revealed bilateral diffuse coarse crepitations. Blood pressure was 130 / 70 mmHg and pulse rate was 108 beats per minute (bpm). Abdomen and neurological system were clinically normal. His initial investigation results revealed a WBC of 9.1 x $10^3$/ µL (N–67%, L-29%), Hb of 16.9 g/dL and platelet count of 120 x $10^3$/µL. CRP was 48 mg/L (normal <6 mg/L ) and the urine full report (UFR) showed albumin 2+. ECG, blood sugar and electrolytes were normal. CXR revealed multi-lobar pneumonia.

Treatment was initiated for a severe bronchopneumonia with broad spectrum antibiotics ( IV vancomycin, meropenem, clarithromycin) and oseltamivir (Tamiflu) after obtaining samples for microbiological investigation, which included blood and urine for bacterial culture and nasopharyngeal swabs which were sent to the Reference Laboratory for virological studies. Within 24 hours of admission his clinical condition deteriorated rapidly necessitating intubation, ventilation and ICU care. Within a few hours his condition gradually deteriorated and the patient died. Blood culture had no growth and urine cultures showed no significant growth. r.t.PCR performed on nasopharyngeal swabs were positive for Influenza B virus.

**Case 2**

3 days following the death of the first patient (case 1), his relative who was living in the same house, a 32 year old lady, was admitted with sore throat and a dry cough for 3 days with upper respiratory tract symptoms. Apart from that she had no other significant symptoms. She was afebrile and the rest of the clinical examination was completely normal. On the 2$^{nd}$ day following admission, she developed a mild fever with no other significant new symptoms. On the 4$^{th}$ day after admission, she developed sudden chest pain and shortness of breath. On examination she was dyspnoiec, sweating and febrile. Her pulse rate was 42 bpm with a blood pressure of 110/60 mmHg. ECG revealed deep T inversions in the anterior leads ($V_2$ – $V_6$ ) and $L_2$ which were significant. A clinical diagnosis of acute viral myocarditis was made and patient transferred to ICU for further care. The patient had to be intubated and ventilated while in the ICU. Over the

next 3 days, the patient developed left sided lower lobe pneumonia with a pleural effusion. IV levofloxacin and oseltamivir was initiated after obtaining blood, endotracheal tube and urine samples for bacterial culture and nasopharyngeal aspirate ( NPA ) for virological studies. An intercostal tube was inserted to drain the large effusion and pleural fluid also sent for bacterial culture.

Her investigations revealed a WBC of 5.6 x $10^3$/µL which subsequently increased up to 19 x$10^3$/µL (N=61% , L=26%). Hb and platelet counts were 12.6 g/dL and 296 x $10^3$/µL respectively. CRP was 24 mg/L. CXR later revealed left sided lower lobe pneumonia with pleural effusion. Electrolytes, liver enzymes, renal functions, sputum AFB were all negative. Cultures obtained for bacteriological studies from urine and endotracheal aspirate revealed mixed growths suggestive of probable colonization. Blood and pleural fluid cultures had no growth. Subsequently her nasopharyngeal aspirate r.t.PCR was reported as positive for Influenza B by the Reference Laboratory. Over the next few days she improved, was extubated and managed to have a complete recovery. She was discharged 3 weeks following admission.

**Cases 3 & 4**

One week later, the two nursing officers who took part in resuscitating the 1st patient (case 1) were admitted with fever and cough of 3 days duration. One nursing officer had fever associated with productive cough and had developed mild haemoptysis that morning. After admission he developed severe nausea and vomiting. Towards the evening, his pulse oxymetry saturation gradually started dropping and was 90% on air. He was transferred to the ICU for monitoring and immediately started on oseltamivir after obtaining samples for cultures and NPA. Over the next few days he had an uneventful recovery. The other nursing officer was also immediately started on oseltamivir and her recovery was also uneventful. Both had normal investigation results on admission. All cultures were negative. Subsequently NPA from both patients were reported as r.t. PCR positive for Influenza B by the Reference Laboratory**.**

**Case 5**

10 days following the resuscitation of the 1st patient, the medical officer who resuscitated the patient also developed a febrile illness with upper respiratory tract symptoms. She needed no hospitalization but was started on oseltamivir early in the illness. Within 3 days she made a complete recovery. Her NPA which were obtained prior to treatment was also reported as r.t.PCR positive for Influenza B virus.

**Discussion**

The above outbreak started following a minor respiratory tract infection in a household. Two of the household personnel developed severe infection where one died of severe pneumonia and the other had to be given intensive care with ventilation due to severe myocarditis and pneumonia. All healthcare personnel who took part in the resuscitation of the patient with pneumonia developed a respiratory tract infection with influenza B virus. This indicates the severity and the transmissibility of the virus which was present in that patient. Also patients infected with presumably the same virus showed a different clinical manifestation (i.e. severe pneumonia,

severe myocarditis, lower respiratory tract infection with desaturation, simple upper respiratory tract infection) which is well described. The small outbreak of this influenza B virus infection which could have easily infected a lot of patients and health care workers in the hospital was successfully contained within the hospital due to proper infection control measures which included patient isolation, hand washing, adhering to universal precautions and the wearing of protective gloves and face masks by all health care personnel and family attending to the patient.

The pathogenesis of fatal influenza B virus infection is inconclusive. Some studies have suggested that bacterial pneumonia and cardiac injury contribute to fatal outcomes after infection with influenza B virus.[12] Patient 2 had myocarditis and pneumonia. Although she had a neutrophil leukocytosis and high CRP suggestive of bacteria aetiology, cultures of blood, sputum and pleural aspirate were negative.

The disease pattern of influenza B virus infection in Sri Lanka is not well studied and documented, except for a handful of reported cases.[13,14] The recent pandemics due to influenza A infection caused a lot of panic and discussion among local health care officials and as a result oseltamivir was introduced into the market for treatment. Vaccination was also started for high risk groups. Treatment is very similar for influenza A and B. Therefore early recognition of outbreaks is critical for successful outbreak control and mitigation of its adverse effects. Oseltamivir has been shown to be effective against influenza B infection[15] provided the drug is given early in the disease. Vaccination also helps to reduce the number of infections and reduce disease severity. Despite these measures, large outbreaks of influenza B with high attack rates (35.9%) are reported despite high resident vaccination rates (93%), even when the vaccine strain was matched to the circulating strain.[11]

Influenza B virus infection can cause significant mortality and morbidity in people. Therefore a high index of suspicion in the early phase when examining patients is needed as early treatment can prevent complications and death. Also proper infection control measures including proper hand hygiene and isolation of the suspected patient/s are of utmost importance to prevent spread of the infection in a given setting.

Informed consent was taken from the subjects and the relatives of the deceased prior to submission.

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