Case report

Influenza B presenting as cardiogenic shock and progressing to multiorgan failure: A case report

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A B S T R A C T

Influenza induced cardiogenic shock is rare and the majority of reported cases are a result of Influenza A myocarditis. We describe a patient with Influenza B who developed myocarditis and cardiogenic shock, with no known pre-existing heart disease. The patient’s disease progressed to include rhabdomyolysis, compartment syndrome, renal failure, and pneumonia. He was successfully managed with Oseltamivir, renal replacement therapy, antimicrobials and intubation. This case is notable due to the rarity of influenza B induced cardiogenic shock and reinforces the importance of recognition and treatment.

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Case presentation

The patient, a 53-year-old healthy man without significant past medical history, presented to a community hospital complaining of a three-day history of subjective fever, chills, diffuse myalgias, and a productive cough. Chest x-ray was not completed. The patient was clinically diagnosed with pneumonia, prescribed Levofloxacin, and discharged home. He returned the following day, complaining of dyspnea, nausea, emesis, and syncope. On presentation he was found to be tachycardic (130 beats/min), normotensive (131/88 mmHg), tachypenic (22 breaths/minute), with normal oxygen saturations (99% on room air), and a temperature of 36.5 degrees Celsius. Initial blood work was notable for a white blood cell count (WBC) of 22 × 10^9/L, troponin-t (high sensitivity) 31 ng/L, creatine kinase (CK) 7910 U/L, and by venous blood gas, a lactate of 9.7 mmol/L. His electrocardiogram showed sinus tachycardia with frequent PVCs. The patient was then approached as a suspected sepsis. He had blood cultures drawn. Treatment was undertaken with IV fluids and Piperacillin-Tazobactam. He was then transferred to the local tertiary care centre to be evaluated by General Internal Medicine.

On arrival to the tertiary care centre, the patient’s heart rate was 122 beats per minute, blood pressure was 111/88 mmHg, respiratory rate was 25 breaths per minute, and oxygen saturation was 99%, with a temperature of 36.5 degrees Celsius. Examination revealed bilateral pulmonary crackles, normal heart sounds, and a jugular venous pressure at 4 cm above sternal angle. There was no pedal edema. His skin was mottled, and his extremities were cool. Capillary refill time was approximately six seconds. Abdominal exam was benign. Chest radiograph was completed, which was unremarkable. The patient then underwent CT pulmonary angiogram, which was not corroborative for a pulmonary emboli but was notable for the presence of pulmonary edema. N-terminal pro-B-type natriuretic peptide (NT-proBNP) was completed which returned at 8146 pg/ml (normal < 125 pg/ml). He was then consulted to Cardiology, as a suspected decompenated heart failure. The Cardiology service then performed urgent cardiac transthoracic echocardiography (TTE), which demonstrated severe global hypokinesia of the left ventricle, an ejection fraction of 15–20%, and a small pericardial effusion. Based on the decreased ejection fraction, pericardial effusion, and influenza like symptoms the patient was approached as a potential myocarditis and admitted to the Cardiac Intensive Care Unit.

In the Cardiac Intensive Care Unit, the patient’s probable myocarditis was treated with Colchicine and high-dose Aspirin. In the Cardiac Intensive Care Unit his troponin peaked at 3871 ng/L (normal < 14 ng/L). He had a nasopharyngeal (NP) swab completed and sent for influenza antigen testing via Polymerase Chain Reaction (PCR). He was then started on empiric Oseltamivir. On post-admission day three, the patient’s nasopharyngeal swab returned positive for Influenza B. He remained clinically stable, until post-admission day four, when he developed increasing dyspnea and shortness of...
breath, resulting in increased oxygen requirements. Chest x-ray demonstrated a new right-sided airspace opacity. Vancomycin was then added to Piperacillin-Tazobactam to cover for the possibility of Methicillin-Resistant Staphylococcus aureus (MRSA) pneumonia. On the same date, he began to complain of pain to his forearms bilaterally. CK was again denoted to be elevated at 8321 U/L. The patient’s right forearm increased in sensitivity and became firm. Plastic surgery was called who measured a compartment pressure of 60 mmHg (normal 0–8 mmHg). The patient then underwent a bedside right forearm fasciectomy. He was then transferred to the Medical-Surgical Intensive Care Unit (ICU).

Despite the utilization of non-invasive respiratory support, the patient’s hypoxemia worsened on post-admission day four, resulting in intubation. On admission to the ICU, the patient’s heart rate was 111 beats per minute, blood pressure was 86/57 mmHg, with a MAP of 67 mmHg. He was saturating 97%, via a pressure support of 8 cmH2O, fraction of inspired oxygen of 45%, a positive end expiratory pressure of 10 cmH2O, with a respiratory rate of 19 breaths per minute. The patient had a temperature of 37.1 degrees Celsius. Examination revealed diffuse bilateral crackles in the lungs, normal heart sounds, and an elevated jugular venous pressure at 6 cm above the sternal angle. There was 3 + pitting edema to his sacrum. His skin was mottled, and his extremities were cool. Norepinephrine was again started to support the patient’s blood pressure. As the patient remained volume overloaded, a Furosemide infusion was initiated.

The Norepinephrine was discontinued by post-admission day five. He had a repeat TTE which demonstrated complete recovery of his ejection fraction at 60–65%. As the patient remained intubated on post-admission day seven, the Oseltamivir was continued. On the same date, both sets of the patient’s anaerobic and aerobic blood cultures returned negative for growth. A repeat NP PCR test was sent for Influenza, which returned positive for Influenza B on post-admission day ten. On the same day, the patient became oliguric, with a creatinine of 416 umol/L. His renal failure was felt to be multifactorial, including from both medications and rhabdomyolysis. He then underwent two days of continuous renal replacement therapy before recovery of renal function. Post admission day twelve the patient was successfully extubated. Following three days of observation, he was transferred to home hospital for rehabilitation. Following twelve days of admission there, he was discharged to his place of residence.

Discussion

In pediatrics, Influenza B has been commonly reported as a cause of myocarditis [2]. However, in adult medicine it has been rarely reported [3]. In fact, a literature review of reported clinical cases of Influenza B induced myocarditis, revealed only twenty three cases, with two associated presentations. The associated presentations, include two cases of peripartum cardiomyopathy, both described by Muroya in 2010, which were associated with elevated Influenza B antibody titers [4].

This patient’s presentation of cardiogenic shock, with a pericardial effusion, decreased left ventricular function, and positive testing for Influenza B, supports the diagnosis of viral myocarditis. While coronary catheterization was not undertaken, the patient’s subsequent normal echocardiogram, and lack of ischemic electrocardiographic changes, suggest against an ischemic component. Additionally, while right ventricular biopsy is a known diagnostic option in this setting, it was forgone in this patient secondary to its risk, limited sensitivity, and because the treatment team had a working diagnosis for his presentation [5,6].

Secondary to the patient’s critical state and in keeping with current Center for Disease Control and Prevention guidelines, the treatment team utilized an extended course of Oseltamivir [7]. While this patient had rapid resolution of his left ventricular dysfunction, other serious consequences of his infection remained, some beyond the five days for which Oseltamivir is typically prescribed. In 2006 Kawai published a study of 3303 Influenza patients in Japan treated with a 4–6 day course of Oseltamivir, which demonstrated an influenza B virus reisolation rate of 51.6%, compared to 15.9% in Influenza A [8]. While this patient’s critical illness necessitated the continuation of Oseltamivir, some of his complications could have been theoretically worsened if the typical treatment course was undertaken. This example, combined with Kawai’s evidence, presents the argument that a longer recommended duration for Oseltamivir in Influenza B, may be of benefit.

The reason behind the severity of this patient’s presentation is unclear. However, early intervention of antiviral therapies may have prevented his severe clinical course or otherwise prevented his clinical decline. He was otherwise healthy, and his past medical history was notable only for femoral and scapular fracture. Interestingly, the patient had not received influenza immunization and had a family history of congenital heart disease. The effect that either of these had on his risk of cardiogenic shock remains to be seen.

Conclusions

This case report draws attention to the potentially serious cardiogenic and systemic effects of Influenza B. The experience of this patient serves as a reminder for the need to be suspicious of influenza infection and reinforces the importance of prevention, early, and if needed, extended, antiviral treatment.

Consent

Written informed consent was obtained from the patient for the publication of this case report.

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