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CASE REPORT

Heavy hearts: the impact of influenza on young lives, a rare case report

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ABSTRACT

Considerable information is available about the acute respiratory symptoms of influenza A and B. However, rarely, these viruses can adversely affect the cardiovascular system. Few cases of pericardial effusion and cardiac tamponade due to the Influenza virus have been reported. To the best of our knowledge, we present the first case of a 23-year-old unvaccinated woman having concurrent influenza A and B infection manifesting as pericardial effusion and cardiac tamponade. The patient was treated with oseltamivir 75 mg, resulting in significant clinical improvement. This case emphasizes the importance of considering influenza as a possible cause of cardiac tamponade.

Keywords: Cardiac tamponade; Influenza A and B virus coinfection; Pericardial effusion; Viral cardiomyopathies

INTRODUCTION

Acute pericarditis, an inflammatory condition affecting the pericardium, accounts for 0.2% of all cardiovascular hospitalizations (1, 2). In developed countries, 80-90% of acute pericarditis cases are idiopathic, suggesting a viral aetiology. Approximately 70-90% of these cases are self-limiting, with only 5% being resistant to therapy (3, 4). Among viral pathogens, enteroviruses, herpesviruses, parvovirus B19, cytomegalovirus, influenza, parainfluenza, varicella-zoster virus, HIV, and hepatitis B and C are notable causes of acute pericarditis, while bacterial, fungal, and parasitic infections being rare (5, 6).

Cardiovascular complications from Influenza infections are uncommon, though some studies have indicated a link between Influenza and increased cardiovascular mortality, primarily due to a higher incidence of coronary events such as acute coronary syndrome (ACS) and ST-segment elevation myocardial infarctions (STEMIs) (7, 8). This case report details a young woman who presented cardiac tamponade in the context of an acute coinfection with Influenza A and B.

CASE PRESENTATION

A 23-year-old woman was admitted with symptoms including fever, myalgia, arthralgia, headache, and weakness. She developed a gradual onset of non-radiating, left-sided stabbing chest pain and shortness of breath, which worsened with exertion. Upon admission, her vital signs were recorded as follows: pulse rate of 105 bpm, blood pressure of 90/60 mmHg, respiratory rate of 33 breaths per minute, and oxygen

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saturation of 94% on room air. Auscultation revealed muffled heart sounds. Notably, she was not vaccinated against influenza and had no recent travel history. The patient had no past history of any cardiovascular, central nervous system or gastrointestinal system conditions.

Baseline laboratory investigations revealed a hemoglobin level of 9.4 g/dL, normal cell counts, sodium level of 135 mEq/L, and potassium level of 4.16 mEq/L. Elevated levels of lactate dehydrogenase (LDH) at 458 units/L and D-dimer at 436 ng/mL were observed, with normal renal function tests (Table 1). An electrocardiogram showed sinus tachycardia. A chest X-ray indicated widespread bronchial wall thickening and an enlarged cardiac silhouette (Fig. 1).

Echocardiography revealed massive pericardial effusion with right ventricular collapse, necessitating the insertion of a pigtail catheter, which subsequently drained 250 ml of fluid. The patient was then transferred to the Medical Intensive Care Unit (MICU), and the Microbiology department was consulted to investigate viral etiologies. Serum sample and throat swab samples were collected and tested for cytomegalovirus (CMV), Epstein Barr Virus (EBV), severe

Table 1. Laboratory investigations during the course of hospital stay.

Date	15/05/24	16/05/24	17/05/24	20/05/24	21/05/24	22/05/24
Haematology						
WBC	11.2	8.9	7.0	3.7		4.9
N%	74.6	74.0	67.0	39.4		64.1
L%	17.1	17.7	25.6	51.0		28.3
M%	8.0	8.0	6.7	8.5		7.2
E%	0.1	0.1	0.3	0.2		0.0
RBC	3.77	3.67	3.76	3.33		3.26
Hb g/dl	9.4	9.2	9.4	8.3		8.1
Hct	29.7	28.9	29.4	26.1		25.6
Plt*109/L	322	266	303	274		294
INR	1.21				1.14	
PT (sec)	14.6				13.7	
aPTT (sec)	28.5				30.9	
D-Dimer ng/ml	436					
Biochemistry						
Urea mg/dl	22	23	21			31
Creatinine mg/dl	0.74	0.71	0.68			0.78
Total bilirubin mg/dl	7.30	0.70	0.57			0.45
Albumin mg/dl	3.88	3.48	3.55			2.90
Total protein mg/dl	7.30	6.80	6.90			6.08
ALP U/L	83	86	94			63
ALT U/L	13	18	24			19
LDH U/L	458	242	125			137
Blood Sugar (mg/dl)	80		89			
Immunology						
AFP IU/ml			1.98	1.98		
CEA2			0.38	0.38		
OV-125 Ag			51.2	51.2		
GI 19-9 Ag			<0.8	<0.8		
Microbiology						
Influenza A and B RT-PCR						Positive
SARS CoV-2 RT-PCR						Negative
CMV IgM ELISA						Negative
EBV IgM ELISA						Negative

acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and influenza, respectively. CMV IgM ELISA kit by Biogenix INC.PVT.LTD (India), EBV-VCA IgM ELISA kit by CalBiotech (USA) and RT-PCR from NIV were employed for detection of CMV and EBV, SARS-CoV-2 and influenza respectively. The patient tested positive for both influenza A and influenza B with Cycle Threshold values of 27 and 26, respectively (Fig. 2), and negative for the other viruses. Further subtyping demonstrated influenza A subtype H1N1 pdm09 and influenza B lineage Victoria. Influenza should have been ruled out at the very onset but due to the association between influenza virus infection and cardiac manifestations being rare, it was not considered to be a differential diagnosis at earlier stages. Since the patient showed a drastic improvement after the addition of oseltamivir to the treatment regimen, we could conclude that influenza A and B coinfection was acquired before hospitalisation and was the cause of cardiac manifestations. Due to complaints of photosensitivity and rashes, systemic lupus erythematosus (SLE) was suspected but subsequently ruled out.

Treatment outcome. The patient was initially treated with azithromycin, piperacillin-tazobactam, ecospirin, sertraline and mirtazapine. Following the confirmation of influenza A and B positivity, oseltamivir was added to the treatment regimen. The patient exhibited progressive clinical improvement only after the addition of oseltamivir and was discharged in a stable and improved condition.

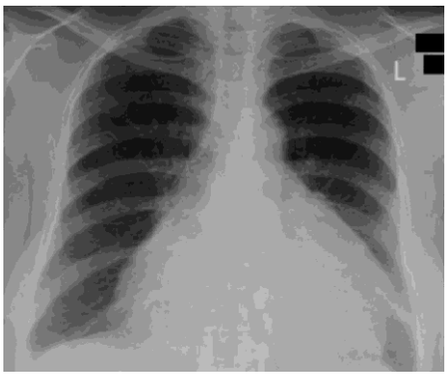

Fig. 1. Chest X Ray showing wall thickening and enlargement of the cardiac silhouette.

DISCUSSION

Influenza is caused by viruses from Orthomyxoviridae family and is categorized into influenza A and B, which are responsible for seasonal epidemics affecting 3-5 million people severely and causing around 300,000 deaths globally each year. Influenza C generally leads to milder illness (9). While influenza predominantly affects the respiratory system, it can occasionally involve the heart, either through direct viral damage or by worsening existing cardiovascular conditions (10). This cardiac involvement can range from myocarditis, which varies from as-

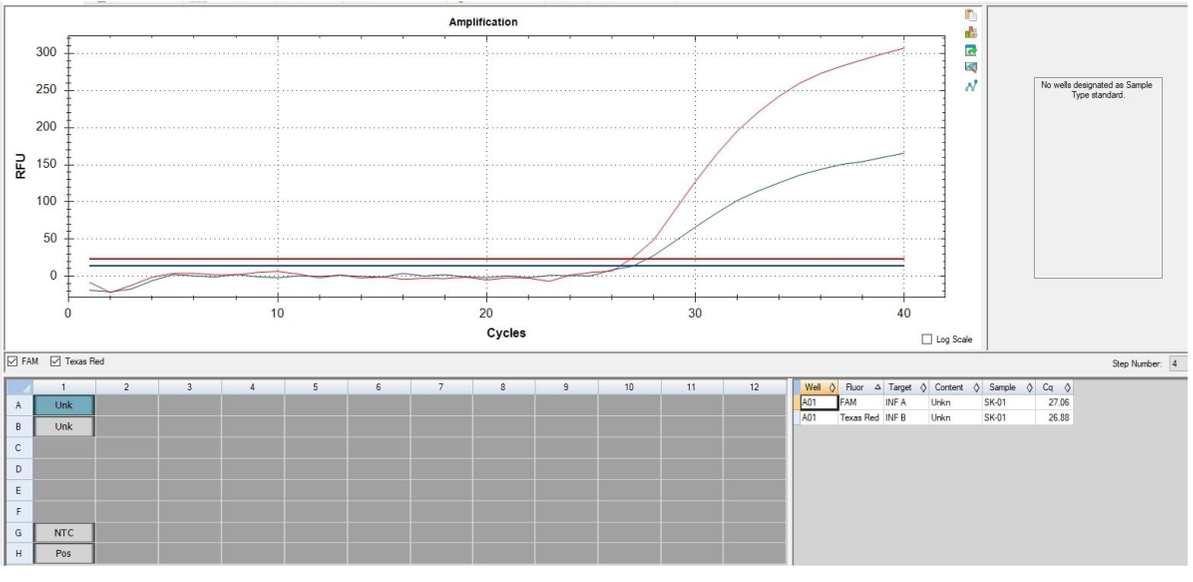

Fig. 2. RT-PCR graph showing positive results for both influenza A and B.

ymptomatic cases to severe myocarditis resulting in cardiogenic shock and death, to pericardial issues like pericarditis, pericardial effusion and cardiac tamponade (10-13).

The precise incidence of pericardial effusion and cardiac tamponade in influenza patients is not well-documented, potentially due to misdiagnosis or underreporting (14). Few cases have been reported to date (15-17). Interestingly, a study was conducted by Chughtai et al. in 2020 in which they observed that creatinine kinase isoenzyme MB (CK-MB) levels were significantly higher among the unvaccinated group compared to the vaccinated group (p value < 0.05). Troponin levels were also higher among the unvaccinated group than the vaccinated group, however, it was not statistically significant (18).

In this case, the patient presented pericardial chest pain and shortness of breath, developing pericardial effusion and cardiac tamponade within a day due to a coinfection with influenza A and B. The patient did not respond to NSAIDs and antibiotics but showed significant improvement with the introduction of oseltamivir. Timely diagnosis and specific antiviral treatment averted the need for pericardiocentesis, reduced the risk of recurrence and simplified the patient management. This is to the best of our knowledge the first reported case of such severe cardiac involvement due to influenza A and B coinfection.

Clinicians should consider specific molecular testing for influenza viruses in nasopharyngeal swabs or tracheal aspirates during the diagnostic evaluation of pericarditis. Additionally, pericarditis should be recognized as a possible complication of influenza-like illnesses, particularly in patients with hemodynamic or respiratory failure. While specific antiviral treatment combined with standard therapy can lead to recovery, vaccination remains the most effective preventive measure, with effectiveness rates of 50-60%, especially in high-risk groups such as the elderly, young children, those with comorbidities and the immunocompromised individuals.

CONCLUSION

Clinicians should consider influenza virus infection as a potential cause of pericarditis and significant pericardial effusion. Prompt diagnostic evaluation and targeted therapy can substantially improve patient outcomes. This is the first reported case of a coinfection with influenza A and B leading to severe pericardial effusion and cardiac tamponade. This case underscores the importance of vaccination in preventing such severe complications.

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