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A rare complication related to H1N1 infection: Dilated cardiomyopathy

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ABSTRACT

The symptoms and findings of influenza A (H1N1) resemble the symptoms and findings of seasonal influenza and it generally emerges as an upper respiratory tract disease. Although the majority of patients with the influenza A virus recover spontaneously without complications, there have been occasional reports of myopericarditis. However, the most frequent complications have been reported as viral pneumonia and more rarely dilated cardiomyopathy. In this paper, we report a 4-month-old infant, who admitted with shortness of breath, cough, tachycardia and respiratory problems and was diagnosed as having developed dilated cardiomyopathy associated with H1N1.

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Keywords: H1N1, myocarditis, dilated cardiomyopathy

Introduction

The Influenza A (H1N1) virus was first identified in Mexico in 2009 and rapidly spread throughout the world [1]. Clinical findings resemble those of seasonal influenza, such as fever, sore throat, cough, listlessness and myalgia. In children, there may also be vomiting and diarrhea, which are not often seen in seasonal influenza. H1N1 diagnosis is made from nasal secretion or nasopharyngeal specimens samples assayed with the polymerase chain reaction (PCR) [2]. Pneumonia is the most common complication of the infection and this can occasionally lead to central nervous system findings, severe dehydration, renal failure, septic shock and multiple organ failure [1]. The influenza virus is generally self-limiting in healthy children, but even as an acute, complication free disease, it can sometimes result in death because of myocarditis and dilated cardiomyopathy [1]. In this case presentation, attention is drawn to the need to bear in mind H1N1 virus and myocarditis and dilated cardiomyopathy which may develop associated with H1N1 in patients who present with findings of viral respiratory tract infection and unlike the classic course demonstrate a long and resistant course.

Case Presentation

A 4-month-old male infant experiencing shortness

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Table 1. Laboratory findings of the patient.

Laboratory tests	Values
White blood count (K/uL)	11.87
Hemoglobin (g/dL)	12
Hematocrit (%)	30.1
Platelets (K/uL)	239.3
Glucose (mg/dL)	92
Urea (mg/dL)	28
Creatinin (mg/dL)	0.45
ALT (U/L)	16
AST (U/L)	43
Na (mmol/L)	133
K (mmol/L)	3.3
Cl (mmol/L)	106
CRP (mg/dL)	0.19
IgA (mg/dL)	14.8
IgG (mg/dL)	277
IgM (mg/dL)	77
IgE (mg/dL)	7.66
CK MB (ng/mL)	2.13
Troponin I (ng/mL)	0.03
p - ANCA	NEGATIVE
c- ANCA	NEGATIVE
ANA	NEGATIVE
Anti-ds DNA	NEGATIVE
ESR (mm/h)	6
Anti HBS(IU/L)	442.8
RF	NEGATIVE

ESR: Eritrocyte sedimentation rate, RF: Rheumatoid Factor

of breath and tachycardia was to admit another center. As cardiac arrest developed, the patient was transferred to our hospital after intubation. On the direct pulmonary radiograph, bilateral infiltrations were present. The laboratory examination results were normal (Table 1). On the electrocardiogram (ECG), 1st degree atrioventricular (AV) block was observed and there were findings of ST wave depression and left ventricular strain pattern on V3-V6 (Figure 1). Transthoracic echocardiography showed dilatation of the left cardiac chamber, moderate mitral valve regurgitation, and mild aortic valve regurgitation. The left ventricle ejection fraction (LVEF) was 32% and the shortness fraction of 15%. Tests were applied with the consideration of viral respiratory tract infection and viral myocarditis. Treatment was started of antibiotherapy, inhaled salbutamol, inotropic, furosemide and captopril. H1N1 production was determined in the nasopharynx smear taken on admittance. Oseltamivir treatment was started with the consideration that there was cardiomyopathy and the clinical manifestation could be associated with the H1N1 virus.

On the 5th day of the oseltamivir treatment, the body temperature returned to normal, the respiratory problems and clinical manifestations recovered, so the patient was extubated, and the oseltamivir treatment was terminated. On the 15th day of hospitalization, in the clinical observation, LVEF was 40% and at the 6-

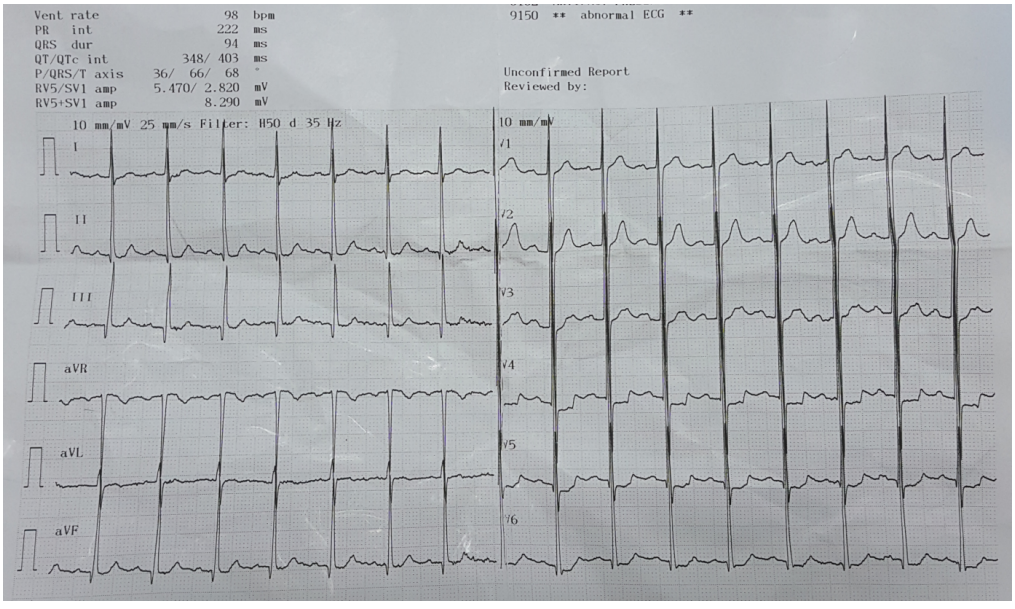

Figure 1. Electrocardiogram shows 1st degree atrioventricular block and findings of ST wave depression and left ventricular strain pattern on V3-V6.

month follow-up examination, LVEF was 50% (Figure 2). The echocardiography findings of the follow-up period are shown in Table 2.

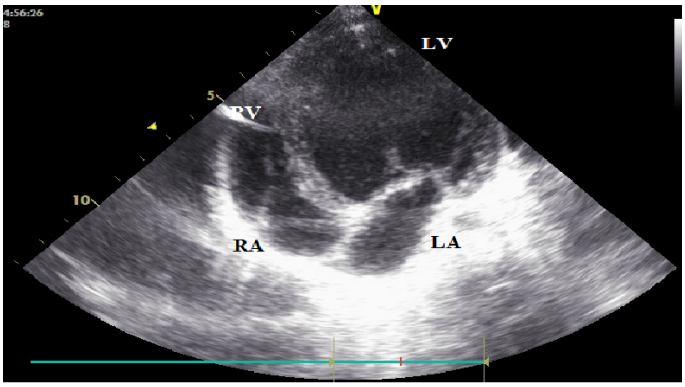

Figure 2. The 6-month follow-up echocardiographic examination. LV = left ventricle, RV = right ventricle, LA = left atrium, RA = right atrium

Discussion

In dilated cardiomyopathy, which is the most frequently seen cardiomyopathy, the left ventricle or both ventricles are dilated, and the heart contractions are reduced. The etiology is most commonly idiopathic followed by viral (coxsackie virus type B, adenovirus, echovirus, influenza types A and B), immune, genetic and toxic causes. In a previous study of 24 patients diagnosed below the age of 2 years, myocarditis was determined in the etiology of 45% of the cases. Influenza type A has been reported to be among the causes of myocarditis [3, 4]. As in some myocarditis cases, the current patient presented with upper respiratory tract symptoms. Tachycardia, tachypnea and cardiac failure, not consistent with fever, are the common findings seen in myocarditis patients. Sinus tachycardia, ST, T-wave changes are seen on ECG, cardiomegaly on telecardiogram, impaired left ventricle function on echocardiography, and elevated CKMB-Troponin levels [5, 6]. Although a definitive diagnosis of myocarditis can be made from myocardial biopsy, there must be an awareness of the risks of taking a biopsy from a patient with myocarditis [7]. While supportive treatment is generally sufficient in myocarditis, in patients presenting with dilated cardiomyopathy, as in the current case, treatment must be directed to congestive heart failure. H1N1, which often presents with influenza-like findings, is more severe in patients aged below 2 years [8]. Although very rare, this may be due to dilated cardiomyopathy, as in the current case.

The pathophysiology of cardiomyopathy associated with influenza is not yet fully understood. In severe influenza infection, vascular permeability increases with cytokine mediation and endothelial cell damage contributes to cardiac dysfunction [9]. Martin *et al.* [10] reported that there was systolic dysfunction in approximately 5% of patients and a return to basal values in 60% of these. This showed that although systolic dysfunction is rare, it can be corrected. As in the current patient, severe complications such as dilated cardiomyopathy thought to be related to H1N1, can be reduced with the use of oseltamivir and similar antiviral agents [11]. In patients presenting with symptoms of upper respiratory tract infection, who are followed up because of the subsequent development of respiratory problems and lower respiratory tract infection, H1N1 virus should be considered in the etiology.

Conclusions

Although cardiac complications associated with H1N1 virus are rare, myocarditis and dilated cardiomyopathy should be kept in mind, especially for patients with a complicated course. The necessary interventions and treatments should be applied to the

Table 2. The echocardiographic findings at follow-up period

	EF (%)	SF (%)	LVEDd (mm)	LVESd (mm)
1st day	32	15	59	50
15th day	40	19	50	40
3rd month	42	21	47	37
6th month	50	26	45	33

EF = ejection fraction, SF = left ventricular shortness fraction, LVEDd = left ventricular end-diastolic diameter, LVESd = left ventricular end-systolic diameter

patient with a multidisciplinary approach.

Informed consent

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

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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