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Transient Regional Wall Motion Abnormality and Increased Wall Thickness of the Left Ventricle in Acute Myopericarditis Occurring in the Puerperium

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ABSTRACT

An unusual sequence of echocardiographic abnormalities of a 25-year-old female with acute myopericarditis was described.

She presented with shortness of breath and a high body temperature after the birth of her first child. Regional asynergy and increased thickness of the left ventricle were transiently observed by echocardiography. It is considered that these abnormalities resulted from inflammatory changes in heart muscle such as edema, which was ascribable to acute myopericarditis in the puerperium.

Acute myopericarditis is a disease which may usually result in diffuse hypokinesis of the ventricle and sudden marked compromise in previous healthy persons[6]. While, regional wall motion abnormalities classically occur in almost all patients with ischemic heart disease. However, in regional or focal myocarditis, chest pain and regional wall motion abnormalities may simulate myocardial infarction[2]. We report a case with acute myopericarditis in the puerperium, demonstrating transient regional wall motion abnormalities and increased thickness of the left ventricle echographically.

CASE REPORT

A 25-year-old woman presented suddenly with shortness of breath and a high body temperature 3 days after the birth of her first child. There was no problem in her medical history. The pregnancy was unremarkable and a healthy baby was delivered without any trouble. Physical examination and chest X-ray film revealed an enlarged heart, and auscultation of the lung disclosed basal crackles. She had a blood pressure of 120/80 mmHg, pulse 80/min, with arrhythmia and there was bilateral ankle pitting edema. ECG showed sinus tachycardia with premature ventricular contractions, low voltage and ST segment elevation in nearly all leads.

Two-dimensional and M-mode echocardiograms on the third day after admission (March 8, 1984) demonstrated "increased thickness" and "akinesis" of the infero-posterior wall and apex of the heart, contrasting with normal thickness and excellent motion of the interventricular septum (IVS=10 mm, LVPW=20 mm, Fig. 1,2). And there was a small pericardial effusion. A second echocardiogram obtained 10 days after admission (March 15) showed that contraction of the infero-posterior wall was normalized but increased wall thickness still remained (IVS=10 mm, LVPW=14 mm, Fig. 2). A third echocardiogram 2 months after (May 7) demonstrated complete normalization of both wall thickness and contrac-

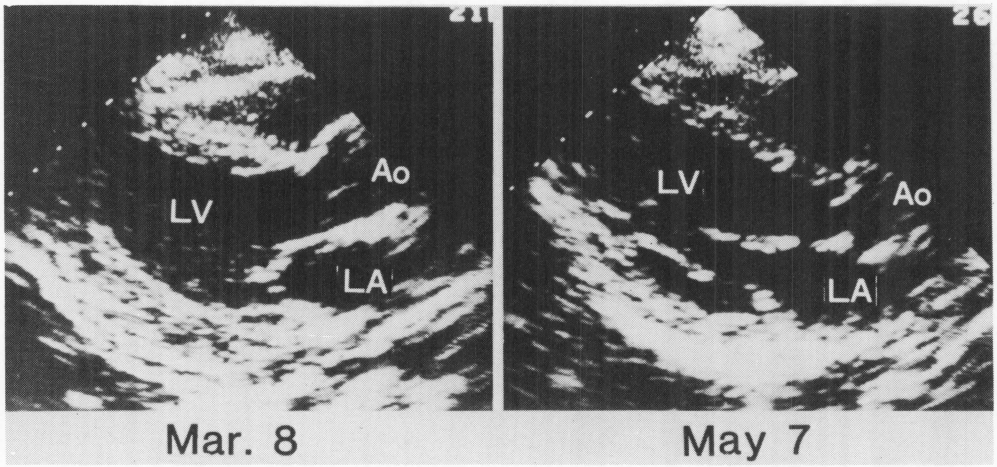

Fig. 1. Two-dimensional echocardiograms.
left; on March 8 (3 days after admission), an end-diastolic long-axis view showing "increased thickness" of the posterior wall
right; on May 7 (about 2 months later), an end-diastolic long-axis view showing complete normalization of posterior wall thickness.
Ao = aorta; LV = left ventricle; LA = left atrium

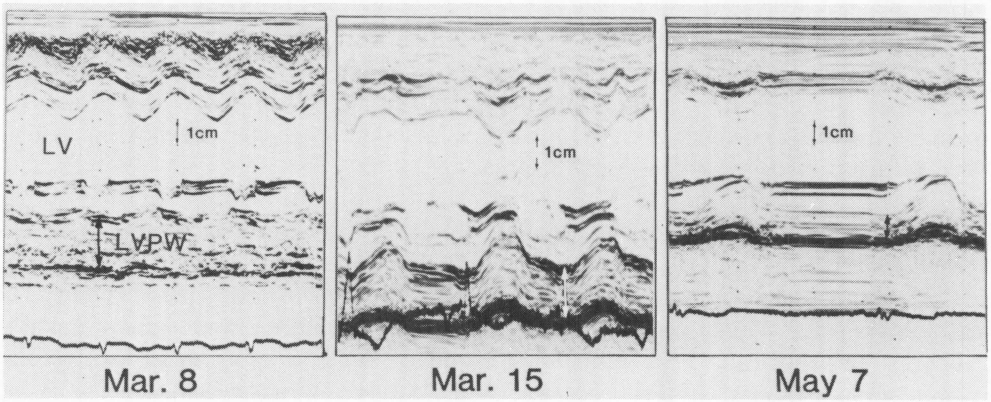

Fig. 2. M-mode left ventricular echocardiograms.
left; on March 8 (3 days after admission), this shows akinesis and "increased thickness" of the posterior wall (arrow), while the interventricular septum represents a normal thickness with excellent motion
Middle; on March 15 (10 days after admission), this indicates normalization of posterior wall motion but "increased thickness" (arrow) remains unchanged
right; on May 7 (about 2 months later), this shows normalization of both wall thickness (arrow) and motion
LVPW = left ventricular posterior wall

tion (Fig. 1,2). This case was clinically diagnosed as acute myopericarditis, occurring in the puerperium.

DISCUSSION

This case was characterized by the regional wall thickening and motion abnormalities observed transiently by echocardiography. So far, similar findings on echocardiograms have been reported only in 3 cases suspected of having acute viral myocarditis[5,7,8]. It is considered that the transient increased wall thickness in the cases reported previously, probably including this case, resulted from inflammatory edema.

On the other hand, peripartum cardiomyopathy has been well known but a relatively uncom-

mon disorder. The criteria for the diagnosis of peripartum cardiomyopathy were established by Demakis and Rahimtoola[3], those consisted of 1) development of heart failure in the last month of pregnancy or within the first 5 postpartum months. 2) absence of a determinable etiology for the cardiac failure, and 3) absence of demonstrable heart disease prior to the last month of pregnancy. There has been only one report of regional contraction abnormality in this entity[1]. But, nothing has been found in the literature concerning peripartum cardiomyopathy associated with such an abnormality of the ventricular wall as transient "increased thickness". This case does not fulfill the criteria of peripartum cardiomyopathy which includes "absence of a determinable etiology for the heart failure", since we have no data on antiviral antibody titers, endomyocardial biopsy findings, etc. to exclude myocarditis as a cause of her symptoms which appears most likely in this patient.

Although the etiologic factors leading to the development of acute myopericarditis in this patient was obscure, there has been a report suggesting enhanced myocardial sensitivity to viral infection in pregnant mice[4]. It was also described that peripartum cardiomyopathy might be in part ascribable to viral infection[9]. From the fact that our case bore a striking resemblance to the three cases reported previously as acute myocarditis, we may diagnose this patient as a variant of peripartum cardiomyopathy which viral involvement played an important role to bring about under these special circumstances.

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