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Case Report

Multiple Ruptured Chordae in an Amphetamine-Addicted Woman: A Rare Manifestation of Illicit Drugs

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ABSTRACT

A 48-year-old woman presented to our hospital with exacerbating cyanosis, edema, lethargy, and dyspnea. The patient declared amphetamine usage until 3 days earlier. Her respiratory function deteriorated gradually, resulting in intubation. During the hospitalization course, she became febrile with a positive blood culture due to an infected central venous catheter. Transesophageal echocardiography revealed a normal left ventricular systolic function, severe left ventricular hypertrophy, and a severe pulmonary arterial hypertension. We found multiple ruptured chordae, resulting in a flail mitral valve and severe mitral regurgitation. To the best of our knowledge, this is the first report of multiple ruptured chordae in the setting of amphetamine intoxication. ***(Iranian Heart Journal 2019; 20(2): 81-84)***

KEYWORDS: Amphetamine, Flail mitral valve, Ruptured chordate

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Amphetamine is a synthetic stimulant with a strong impact on the cardiovascular system.[1] A spectrum of the cardiovascular manifestations of amphetamine intoxication such as palpitation, hypertension, arrhythmias, acute coronary syndrome, aortic dissection, sudden cardiac death, and methamphetamine-associated cardiomyopathy has been previously reported.[2] Hereby, we report multiple ruptured chordae in a case with a long-term amphetamine addiction.

CASE PRESENTATION

A 48-year-old woman referred to us with exacerbating cyanosis, extremity edema, lethargy, and dyspnea both on exertion and at rest. The patient was under treatment with furosemide, metoprolol, lisinopril, and hydralazine for systemic and severe pulmonary arterial hypertension. She declared amphetamine usage until 3 days prior to the admission. On admission, she was confused with skin erythema, pallor of the sclera, remarkable cyanosis, and edema of the face and

the extremities. On physical examination, muffled heart sounds, coarse crackles of the lungs, and extremity edema were noticeable. Electrocardiography demonstrated a poor R progression and evidence of elevated pulmonary artery pressure (Fig. 1). Ultrasonography demonstrated enlarged mediastinum, bilateral pleural effusion with collapsed underlying lung, and infiltration. The oxygen saturation at rest was 72%, and the respiratory function deteriorated gradually and ultimately necessitated intubation. During her hospitalization course, the patient had prolonged hyponatremia, leukopenia, neutrophilia, an elevated erythrocyte sedimentation rate (90 mg/dL), positive highly sensitive C-reactive protein (58 mg/dL), a negative urine culture, and a positive blood culture for coagulase-negative *Staphylococcus;* the source of infection was the central venous catheter. She became febrile, which was subsequently subsided by antibiotic treatment. The treating physician requested echocardiography to rule out infectious endocarditis.Transesophageal echocardiography revealed the following data: a normal left ventricular size and systolic function, severe concentric left ventricular hypertrophy, moderate diastolic dysfunction, moderate right ventricular enlargement and systolic dysfunction, a malapposed tricuspid valve, severe pulmonary arterial hypertension, moderate circumferential pericardial effusion without compressive effects, and a plethoric inferior vena cava. Interestingly, there were multiple ruptured chordae, resulting in a flail mitral valve and severe mitral regurgitation without any evidence of endocarditis. Diffuse atherosclerotic plaques were also noticeable in the descending aorta (Fig. 2). Unfortunately, the patient expired a week later.

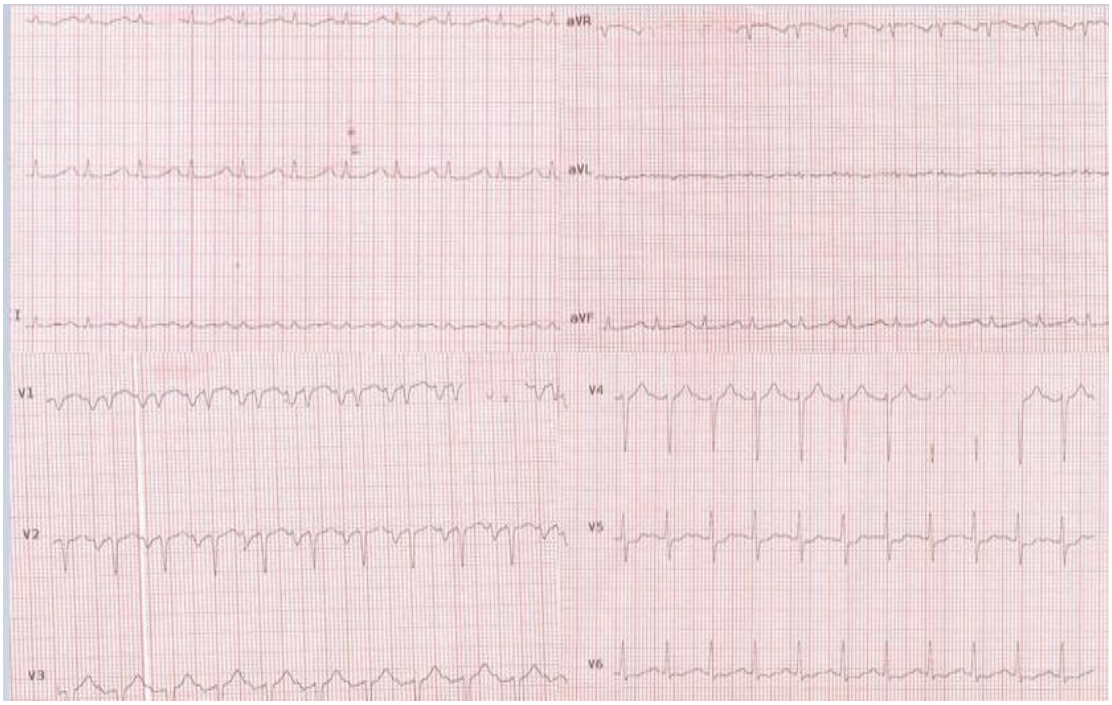

Figure 1. Electrocardiogram, demonstrating poor R progression and evidence of elevated pulmonary artery pressure

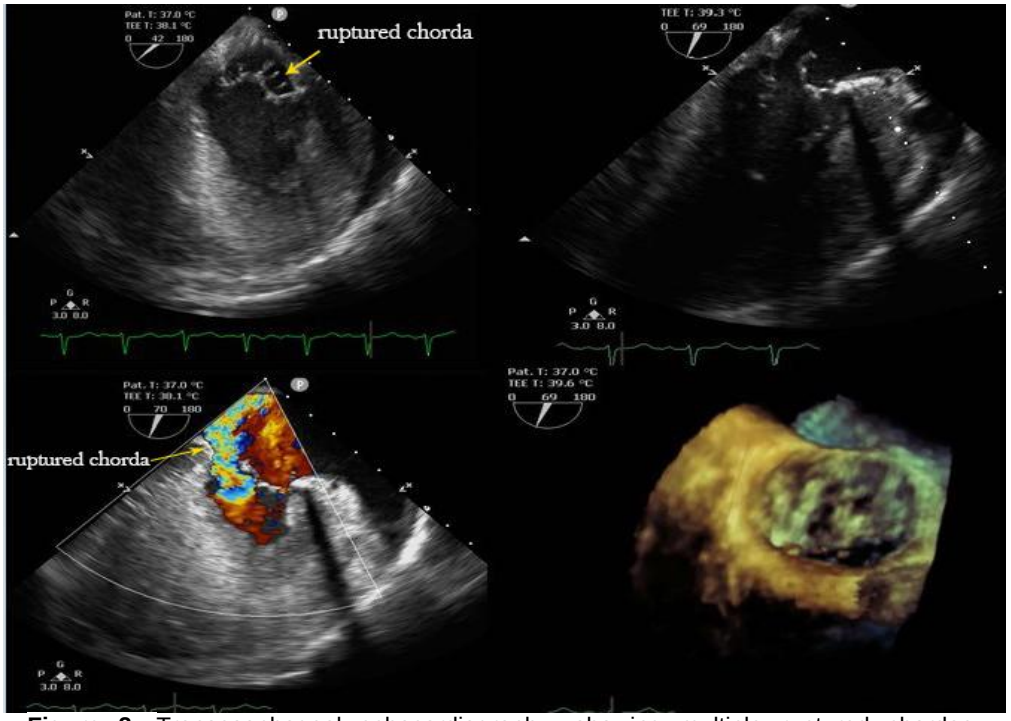

Figure 2. Transesophageal echocardiography, showing multiple ruptured chordae, resulting in a flail mitral valve and severe mitral regurgitation without any evidence of endocarditis

DISCUSSION

The most common cardiotoxic complications of amphetamine abuse are associated with excess peripheral catecholamine levels and coronary vasoconstriction.[3] Amphetamine abuse could lead to the development of various valvular heart diseases, including mitral regurgitation.[4] The most common reported mechanism for mitral regurgitation is annular dilatation due to a dilated left ventricle with the consequent tethering of the posterior mitral leaflet and the mitral leaflet thickening.[5-9] Nonetheless, multiple ruptured chordae as the pathologic mechanism for mitral regurgitation have not been reported in the setting of amphetamine abuse.

CONCLUSIONS

The mechanism of mitral regurgitation in the setting of amphetamine intoxication could be a flail mitral valve. To the best of our knowledge, our report is the first of its kind to report multiple ruptured chordae in the setting of amphetamine intoxication.

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