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Case Report

Subacute aortic regurgitation due to traumatic tear in the aortic wall

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ABSTRACT

A 37-year-old man presented with heart failure caused by severe aortic regurgitation (AR). He had a history of being involved in a traffic accident 3 months earlier. Imaging tests at admission detected no abnormalities in the aortic valve or aortic wall; however, the left coronary cusp prolapsed slightly on transthoracic echocardiography. He underwent aortic valve replacement because of uncontrolled heart failure and severe AR. Intraoperatively, the intima of the aortic wall just above the commissure of the left and right coronary cusps was torn to the short axial direction. Local aortic tear was the final diagnosis for the subacute AR.

<**Learning objective:** Acute or subacute aortic regurgitation (AR) is comparatively rare, and it is sometimes difficult to clinically recognize. The tear in the aortic wall just above the commissure caused by a traffic accident led to the gradual progression of AR, and the diagnosis of the cause of AR was difficult despite using transesophageal echocardiography and contrast-enhanced computed tomography. We should recognize that the detection of subacute AR caused by a local aortic tear can be challenging.>

Introduction

The most common cause of aortic regurgitation (AR) is calcific valve disease, and the disease course is chronic and slowly progressive [1]. On the other hand, acute or subacute course of AR is comparatively rare, and infective endocarditis, aortic dissection, and aortic valve damage caused by trauma are known causes. Acute severe AR, if untreated, leads to advanced heart failure and early death, but it is sometimes difficult to recognize clinically [2]. We report a rare case of subacute AR caused by local aortic tear for which it was challenging to detect the aortic tear preoperatively.

Case report

A 37-year-old man was referred to our hospital for worsening dyspnea on exertion. He had a medical history of bronchial asthma and hypertension, and losartan and budesonide/formoterol had been prescribed. On presentation, his blood pressure was 151/78 mmHg, and his heart rate was 90 beats/min. A grade III/VI diastolic murmur was audible at the 4th left sternal border without jugular venous distention or peripheral edema. Chest radiographs showed bilateral pleural effusion and an enlarged cardiothoracic ratio of 60%, although chest radiographs taken 6 months earlier showed a normal cardiothoracic ratio (Fig. 1A,B). An electrocardiogram showed left ventricular (LV) hypertrophy, and two-dimensional transthoracic echocardiography showed severe AR with a wide central jet (Fig. 2A–C). Three aortic cusps were seen with no dilatation of the aortic root. The LV end-diastolic and end-systolic measurements were 65 mm and 51 mm, respectively, with a LV ejection fraction of 42.9%. Laboratory data were as follows: serum creatinine, 1.0 mg/dL; white blood cell count, 11,200/μL; C-

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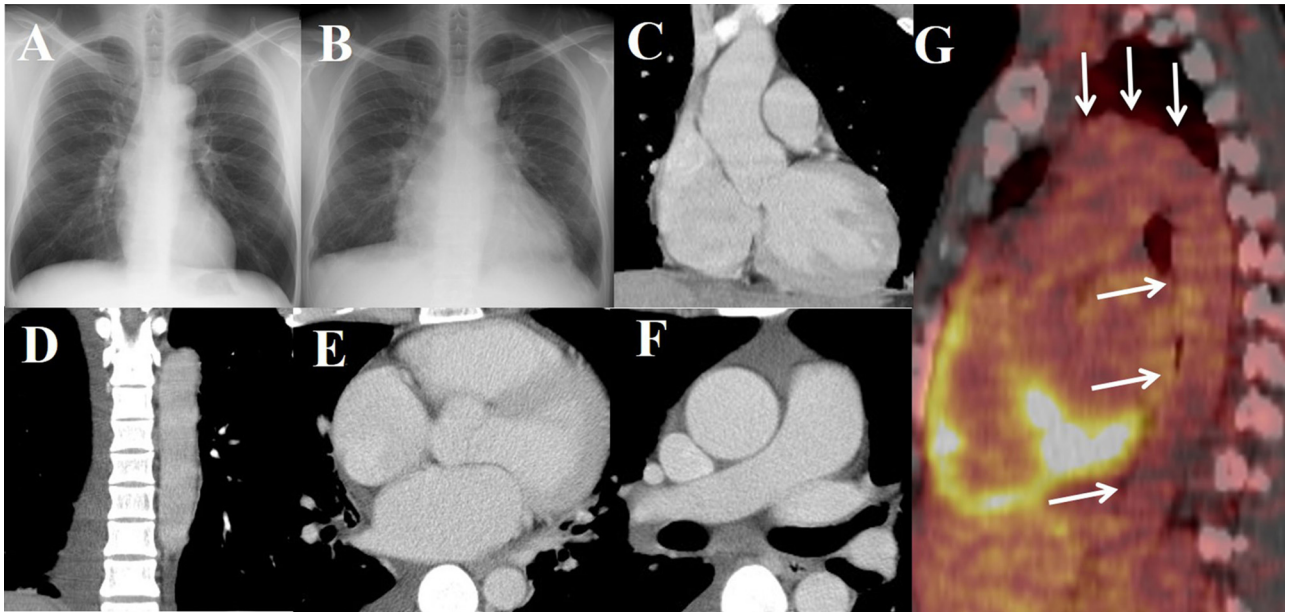

Fig. 1. Chest radiographs 6 months before (A) and on the admission (B). The cardiothoracic ratio increased drastically during 6 months and pleural effusion was shown. (C–F) No intimal flap, false lumen, or thickening of the aortic wall are seen in contrast-enhanced computed tomographic images. (G) Positron emission tomography with 2-deoxy-2-fluoro-D-glucose integrated with computed tomography; images show no evidence of active inflammation of the aortic wall (white arrow).

reactive protein, 0.5 mg/dL; and brain natriuretic peptide, 1402.6 pg/mL. Proteinase-3 anti-neutrophil cytoplasmic antibody and myeloperoxidase anti-neutrophil cytoplasmic antibody tests were negative.

The patient was diagnosed as having congestive heart failure caused by severe AR, which was presumed to have progressed within the preceding 6 months; however, the etiology was unclear at that time. Taking into consideration that the acute or subacute

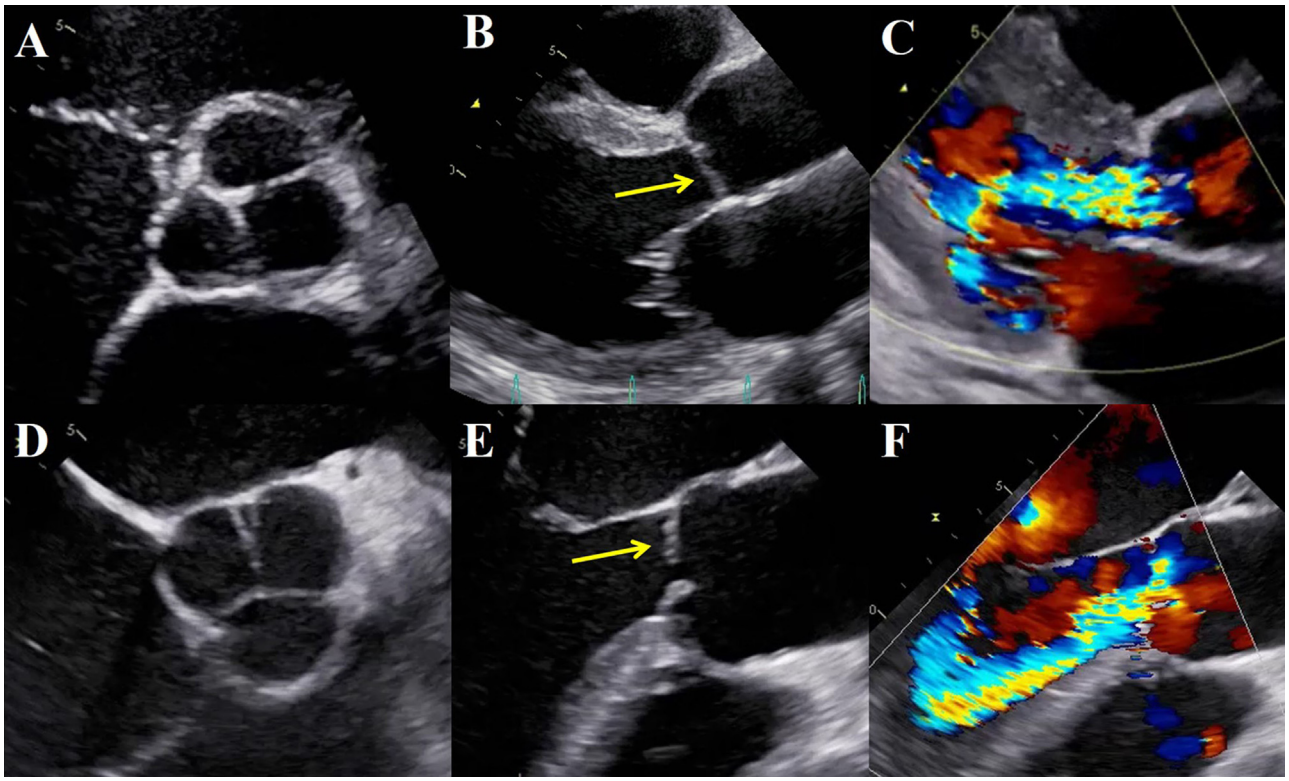

Fig. 2. Transthoracic (A–C) and transesophageal (D–F) echocardiography. Short-axis view (A, D), and long-axis view (B, E). Organic change in the aortic valve is not evident, but slight prolapse of the left coronary cusp is seen (yellow arrow). (C, F) The aortic regurgitation jet blows towards the cardiac apex from a commissure in the prolapsing left coronary cusp.