An Adult Case of Cardiac Fibroma

Nariaki Kanemoto, Kazutane Usui and Yuichi Fusegawa

The patient, a 48-year-old woman with cardiac fibroma, is the second oldest patient with this disease in Japan. Her electrocardiogram showed findings compatible with old high lateral, posterior and possibly lateral myocardial infarction, regions which corresponded to the tumor site. In patients whose electrocardiogram suggests a previous myocardial infarction (pseudo myocardial infarction), the possibility of intramyocardial tumor should be taken into consideration. (Internal Medicine 33: 10–12, 1994)

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Introduction

The incidence of primary cardiac tumors in autopsy series is reported to range from 0.0017% to 0.28% (1–3). About 75% of all cardiac tumors are benign histologically with fibromas being the fifth most common and having a relative incidence of 4% (1–3). Approximately 90% of cardiac fibroma cases occur in infants and children (4). We report a 48-year-old patient with cardiac fibroma, the third adult case and the second oldest patient in Japan, to our knowledge (5–7).

Case Report

A 48-year-old woman was admitted to our hospital for further evaluation of her abnormal electrocardiogram (ECG). Three years ago, the patient was told to have an abnormal ECG at an annual physical check up at another hospital. However, because an exercise ECG was negative, no further studies were done. Although she enjoyed her usual daily life as a housewife, she noted slight restriction of exercise over the past three years. Other past medical history includes pneumonia 10 years earlier.

Physical examination on admission showed her height was 154 cm, weight was 51kg. Blood pressure was 120/80 mmHg and pulse was 80/min and regular. She was not anemic nor icteric. Heart and lungs were clear. Abdomen was soft and flat. No peripheral edema was noted.

Chest x-ray film showed slight cardiomegaly with a prominent 4th left cardiac border, minute accentuation of upper peripheral vascular markings and only mild cephalization without apparent pulmonary congestion or abnormal calcification. The cardiothoracic ratio was 0.53. ECG showed a normal sinus rhythm with a heart rate of 75/min (Fig. 1). Abnormal Q wave in lead aVL and small q wave in lead I together with coronary T waves suggested previous high lateral myocardial infarction. Tall R waves, in lead V1 and V2 with a duration of 40 msec with positive coronary T waves, and the presence of small q waves with a duration of 30 msec with coronary T waves in lead V4–6 suggested the presence of previous posterior and possible lateral myocardial infarction.

Two-dimensional echocardiography revealed an echogenic space-occupying lesion within and extending from the lateral wall to the apex with almost complete akinesis (Fig. 2). The left ventricular diastolic dimension was 55 mm and the systolic dimension was 36 mm. Computed tomography of the heart showed markedly thickened and inhomogenously enhanced lateral and posterolateral left ventricular wall with bulging of the lateral wall. Magnetic resonance imaging showed prominent thickening of the lateral to posterolateral segment of the left ventricle. The center of this region showed a slightly higher intensity than the surrounding myocardium (Fig. 3). Bilateral cardiac catheterization showed normal intracavitary pressures. Although coronary angiography showed normal findings, the left anterior descending artery was markedly deviated anteriorly due to the prominent mass. In the late phase, the mass was blushed faintly via an obtuse marginal branch of the left circumflex coronary artery. Left ventriculography showed a defect of the anterolateral wall due to the mass effect. The wall motion and left ventricular dimension were within normal limits. The diagnosis of a benign left ventricular tumor, probably fibroma, was made. Surgical operation was performed based on the following reasons; a large tumor with obstruction of the left ventricular cavity, and fear of future congestive heart failure and sudden death due to ventricular arrhythmias (7).

The excised tumor was white and hard, and 4x5x6 cm in size (Fig. 4). The tumor was composed of collagen fibers and markedly elongated fibroblasts. Hyalinized foci were occasion-
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Fig. 1. A 12-lead electrocardiogram on admission.

Fig. 2. A two-dimensional echocardiogram on admission.

Fig. 3. A magnetic resonance image on admission.

ally seen. In the peripheral area entrapped bundles of myocardium were noted. The pathological findings were consistent with cardiac fibroma (Fig. 5). The post-operative course was stated elsewhere (8).

Discussion

Cardiac fibroma is a benign tumor mostly occurring in the free wall of the left ventricle or ventricular septum but rarely in the right ventricular wall or annulus of the aortic valve (9). In the English language literature, we have found 80 reported cases to date (5, 10, 11). The age of these patients with cardiac fibroma ranged from 42 hours to 65 years. Approximately 90% of the case were infants and children less than 12 years of age, of which 5% were less than 2 years (12). In Japan 17 cases have been reported, of which 15 patients were 15 years of age or less, with 8 of these being 3 years or less (6, 7, 9). Only two adult cases were reported and their ages were 22 and 76 years. Our 48-year-old patient which is now the 18th reported case is the second oldest case in Japan.

Symptoms and signs are found only in 50% of the cases; congestive heart failure (31%) being the most frequent, followed by heart murmurs (27%), sudden death (13%), syncope (6%), and chest pain (3%) (5, 9, 10). When patients were divided into anteroseptal and postero-lateral groups according to the cardiac sites involved, those with the former lesion presented with congestive heart failure much more frequently. In addition, the patient with the latter lesion showed chest
ventricular hypertrophy, right ventricular hypertrophy, right bundle branch block, first degree A–V block, and inverted T waves (5). ECGs of primary or metastatic cardiac tumors rarely show transient or persistent ST elevation with or without abnormal Q waves (13–15). Proposed mechanisms of ST elevation include progressive involvement of previously unaffected muscle, change in transverse Na⁺-K⁺ gradient, and inflammatory reaction of the pericardium (15). Mechanisms of Q waves include coronary artery occlusion secondary to tumor emboli or direct invasion (14–16), and electrical silence of the involved myocardium, that is, pseudomyocardial infarction (17). Our patient’s ECG showed a previous Q wave myocardial infarction, the mechanism of which is ascribed to the latter as the ECG changes were compatible with the tumor site. In light of these findings, in patients with ECG changes consistent with silent Q wave myocardial infarction, the possibility of cardiac tumor should be considered.

### References