Angiographic Evidence of Myocardial Squeezing Accompanying Tachyarrhythmia as a Possible Cause of Myocardial Infarction*

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The coronary arteriogram of a 52-year-old man with Basedow's disease and who was suffering from myocardial infarction following rapid atrial fibrillation, showed severe myocardial squeezing without organic stenosis. Angiographically, the functional obstructive lesion was always observed both at systole and diastole during atrial pacing, 150 beats per minute, and suggests that myocardial squeezing may be the cause of myocardial infarction following tachyarrhythmia.

A number of studies have shown that the main cause of myocardial infarction is coronary atherosclerosis, but there are reports of myocardial infarction without obstructive lesion observable on coronary arteriograms. Without conclusive explanation of the phenomenon1 or with misinterpretation as coronary occlusion, coronary spasm,2 coronary embolism followed by subsequent clot lysis,3 retraction or recanalization, each might result in a normal angiographic picture.

Angiographically, the coronary artery may sometimes be compressed from outside, especially in the systolic phase, to produce a coronary occlusion pattern. This is so-called myocardial squeezing, and is thought to be caused by myocardial bridges.8 Because this phenomenon usually produces stenosis or obstruction only in the systolic phase, it does not significantly affect coronary circulation and therefore has not been attributed of itself as producing myocardial infarction.4

Noble et al.,8 however, have recently reported that myocardial ischemia during atrial pacing may be caused by myocardial bridges of the coronary artery.

This report describes one case of myocardial infarction seen at our institute which did not show any significant organic stenosis in coronary arteries angiographically, and was thought to have generated myocardial infarction only by reason of functional stenosis arising from myocardial squeezing. We accordingly examined the influence of myocardial squeezing on coronary circulation.

CASE REPORT

A 52-year-old Japanese man, an office employee, who had received methimazole (Mercazole) for Basedow's disease from the ages of 38 to 42 years and again at age 52 years, for recurrent hyperthyroidism, was admitted to Yamanashi Central Hospital on October 29, 1975 because of sudden and progressively stronger chest pain lasting several hours, palpitations, and vomiting following drinking 275 ml of an alcoholic beverage the night prior to admission.

An electrocardiogram made earlier on May 10, 1975, had showed no abnormal Q wave (Fig 1A), but the electrocardiogram on admission showed atrial fibrillation, ST segment elevation of leads V1,2 and QS pattern of V1,3, which led to a diagnosis of anterosetal myocardial infarction (Fig 1B). The value of serum enzyme determinations on admission showed levels of SGOT of 192 units, SCPT 64 units, and LDH 456 units, and on the next day SGOT levels of 71 units, SCPT 52 units and LDH 751 units. The clinical course after admission was satisfactory and the patient was discharged on December 20, 1975.

On March 8, 1976 the patient was admitted to our institute in Tokyo for the purpose of taking a coronary arteriogram and at that time the electrocardiogram was as in Fig 1C. The patient at this second admission did not complain of any chest pain, dyspnea and palpitation, and there were no apparent objective findings of arrhythmia and cardiac failure; exophthalmus was observed. Results of Master two-step test

![Figure 1A. Control (10 MAY, 1975), no abnormal Q wave. 1B. At admission (30 NOV, 1975), complicated with atrial fibrillation. ST segment elevation and abnormal Q wave are observed in leads V1,2. 1C. (8 MAR, 1975), QS pattern is observed in leads V1,3.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21004/ on 06/24/2017)
were negative. Thyroid gland function values were also normal.

The aortic pressure, left ventricular systolic pressure and left ventricular end-diastolic pressure, measured by cardiac catheterization, were, respectively, 120/80 mm Hg, 120 mm Hg and 14 mm Hg, and the cineangiogram of the left ventricle showed hypokinesis of the anterior wall and dyskinesis of the apical wall. No significant abnormal findings were observed in the right coronary arteriogram.

Severe myocardial squeezing, indicated by arrow in Figure 2A, was observed at the proximal portion of the left anterior descending artery. This finding was observed in both right anterior oblique and left anterior oblique views.

The size of lumen of the anterior descending artery was normal in the diastolic phase, but there was total obstruction at the systolic phase. The obstructive lesion was always observed in the lumen of the left anterior descending artery either at systole or diastole by the artificially (electric stimulation) induced atrial pacing, 150 beats per minute (Fig 2C, D).

The patient’s clinical course afterward was satisfactory, and he performed his usual office work tasks without suffering from either anginal attack or atrial fibrillation after discharge.

**DISCUSSION**

The cause of the myocardial squeezing seen on coronary arteriograms has been experimentally proved in dogs to be myocardial bridges. The inference was therefore made that myocardial squeezing observed in human coronary arteriograms is also induced by myocardial bridges.4,5

Geiringer reported that atheromatous conditions are less frequently found in the coronary artery running intramurally than in the coronary artery running extramurally. Edwards et al 10 reported opposite findings, giving the impression that there remain unknown points with respect to the pathologic significance of myocardial bridges.

Sones states that the clinical significance of myocardial bridges can be neglected insofar as they are not complicated by organic stenosis, but, even when not complicated with significant organic stenosis, if marked tachycardia occurs, the diastolic phase becomes extremely short, and comprises an adverse influence on coronary circulation. In fact, it has been shown that the diastolic phase becomes extremely short, and that the coronary artery in the diastolic phase is hardly dilated at all.

Our findings lead us to the conclusion that myocardial squeezing may possibly be the cause of myocardial infarction under specific circumstances and conditions.

**REFERENCES**

Echocardiographic Evidence of Left Ventricular Tumor in a Neonate*

Allan H. Rees, M.D.; Francisco E. Elbl, M.D.; Kareem V. Minhas, M.D.; and Robert E. Solinger, M.D.

We present the case of a ten-hour-old infant with clinical findings suggestive of hypoplastic left-heart syndrome. A diagnosis of left ventricular tumor was made by echocardiographic examination. This diagnosis was confirmed at autopsy, when a large rhabdomyoma was found in the interventricular septum, obliterating the major portion of the left ventricular cavity. The echocardiographic findings are discussed.

A neonate initially had clinical findings compatible with a diagnosis of hypoplastic left-heart syndrome. The results of echocardiographic studies performed on this patient were highly suggestive of the presence of a left ventricular tumor. This was later confirmed at autopsy. Our purpose is to report this case and to discuss the echocardiographic features found in the study performed on this infant.

**CASE REPORT**

A white male infant who weighed 3.6 kg (7 lb 15 oz) at birth showed signs of respiratory distress shortly after delivery. A cardiac murmur was first detected when he was 4 hours of age, and a chest x-ray film at that time showed moderate cardiomegaly. For this reason the infant was referred to Norton-Children's Hospital, Louisville, for further evaluation.

On admission the infant was in severe cardiorespiratory distress, with weak and irregular peripheral pulses. There was a grade 3/6 ejection systolic murmur heard best at the lower left sternal border. The heart sounds were difficult to evaluate, due to the presence of an irregular rhythm. On auscultation, rales were noted over both pulmonary fields. The liver was palpated 5 cm below the right costal margin.

The chest x-ray film (Fig 1) showed marked cardiomegaly. The electrocardiogram revealed ventricular fibrillation. This was successfully converted to sinus rhythm by direct-current shock. The patient continued in critical condition, with persistent weak peripheral pulses. Echocardiographic studies were then performed.

**Echocardiogram**

The echocardiograms shown in Figure 2 were obtained in sequence as the ultrasonic beam was swept from the aortic root (Fig 2A) through the upper (Fig 2B), middle (Fig 2C), and apical (Fig 2D) areas of the left ventricle. The echocardiogram of the aortic valve was normal. The right ventricular outflow tract, aortic root, and left atrial dimen-

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**Figure 1.** Posteroanterior (A) and lateral (B) chest x-ray films showing marked cardiomegaly.