Coronary Embolism Following Repair of a Ventricular Septal Defect

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Coronary embolism, in contrast to coronary atherosclerosis or thrombosis, is a rare cause of myocardial infarction. Virchow, in 1856, was the first to describe this accident and also to trace its source to bacterial endocarditis. Saphir and Hamman found dislodgment of bacterial vegetations from the aortic or mitral valve to be responsible for the majority of cases. Other causes were embolic or atherosclerotic material in a coronary artery, thrombi covering atherosclerotic plaques at the root of the aorta, intracardiac mural thrombi, thrombi in pulmonary veins and paradoxical emboli from peripheral veins. To these causes should have been added emboli of neoplastic origin. Later, a unique instance of calcific embolus originating in a nodular calcified aortic valve free of bacterial endocarditis was reported. The introduction of cardiac valvular surgery has made this lesion a not uncommon one.

We report herein another unique cause of coronary embolism, open-heart surgery for repair of a ventricular defect, a cause which may not remain unique for long.

Case Report

C. A., a 17-year-old boy, was first admitted to Temple University Hospital on October 8, 1953, for evaluation of a cardiac murmur present since birth. He had been asymptomatic except for moderate dyspnea, fleeting sharp precordial pain, and cyanosis following strenuous exercise.

Examination revealed retarded physical development, deformity of the anterior chest wall, and findings consistent with a ventricular septal defect. The blood pressure was 100/78. The roentgenogram revealed an enlarged cardiac silhouette and pulmonary artery segment with marked pulsations of its branches. The electrocardiogram was consistent with left ventricular hypertrophy.

Catheterization disclosed pressures of 14/0.5 mm. Hg in the right atrium, 98/17 in the right ventricle, and 98/40 in the pulmonary artery. The oxygen content of the superior vena cava was 10.9 volumes per cent, right atrium 11.1, mid-right ventricle 12.9, high right ventricle 14.6, pulmonary artery 14.4, and radial artery 16.2. The oxygen capacity in the radial artery was 16.9, and its saturation was 96 per cent.

The patient was readmitted on November 13, 1958. He had an episode of syncope, dyspnea, and cyanosis 1 year previously. He complained of recurrent substernal pain lasting 10 to 15 minutes with dyspnea produced by exertion.

Physical examination was unchanged. The roentgenogram showed increased cardiac enlargement. The electrocardiogram was now consistent with right ventricular hypertrophy as well as left. Catheterization disclosed no significant change.

Respiratory functional studies revealed abnormalities considered to be a mixture of a restrictive and obstructive ventilatory problem, consistent with moderate emphysema. All blood studies were normal.

Operation was performed on January 14, 1959. A high ventricular septal defect approximately the size of the aorta was closed with silk. The heart was arrested for 14 minutes. The total time on the pump oxygenator was 25 minutes. The course throughout surgery and for the first 24 hours was uneventful. Then pulmonary congestion and retained secretions increased. Right heart failure developed and the patient died 63 hours after operation.

The postoperative electrocardiogram revealed right bundle-branch block (fig. 1). The subsequent tracings showed abnormal T waves in leads I and II, and on the day of death elevation of the S-T takeoff that was interpreted as due to pericardial reaction. Unfortunately, pericardial leads could not be taken because of the surgical dressings.

At autopsy, the heart weighed 670 Gm. The right side of the heart was voluminous; the chambers were dilated. The ventricular wall was hypertrophied to 1.1 cm. in thickness. The left ventricle

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was dilated and hypertrophied; the wall was 1.6 cm. thick. From this chamber, the high ventricular defect was outlined by a ridge of firm gray-white fibrous tissue. There were minute remnants of adherent thrombus over the defect, directly opposite and a little below the orifice of the left coronary artery. An opening of approximately 0.8 cm. remained at the anterosuperior end of the defect, permitting residual interventricular shunt. The endocardium below the defect was opaque, gray-white, and thickened. In the lower two thirds of the anterior ventricular wall, the inner one third to one half of the myocardium was dull and pale, with yellow, gray, and red mottling. The area extended to the apex and included the anterior two thirds of the interventricular system. The gross appearance was that of a recent subendocardial infarct supplied by the anterior descending branch of the left coronary artery (fig. 2).

The coronary arteries were free of atherosclerosis. However, in the anterior descending branch, 2.5 cm. from its orifice, thrombus material was found occluding the artery for a distance of 3 cm. Beyond was propagated clot. The lungs were congested.

Microscopic sections of the anterior left ventricular wall exhibited extensive subendocardial necrosis. The inner one third of the myocardium was reduced to clumps of deeply eosinophilic material, representing necrotic myocardial fibers. There was unusual edema of the interstitial tissue with infiltration of numerous red blood cells and disintegrating neutrophils. The histologic appearance was that of an infarct, 48 or more hours old (fig. 3). Adherent to the endothelium of the anterior descending branch of the left coronary artery was a minute remnant of thrombus material. The pulmonary arteries showed lipid-rich atherosclerosis and reduction of their lumina.

**Discussion**

The finding at necropsy of an embolus in an anterior descending coronary artery with infarction of the subendocardial region of the anterior left ventricular wall was a complete
surprise to us. From the anatomic relation of the orifice of this vessel and that of the defect and its overlying thrombus, it would appear that coronary emboli could arise at least as frequently from this site as from other regions within the cardiac chambers or from fracture of the aortic or mitral valves. Because the re-institution of anticoagulant therapy (in the absence of contraindications) may be beneficial, it is important to attempt to diagnose this complication at its onset.

It is unlikely that clinical symptoms or signs will be definitive because pain, pulmonary congestion, pulmonary edema, shock, and arrhythmias are common to many complications. The electrocardiogram is the most likely aid in establishing a diagnosis.

Abnormal Q waves would be most definitive, indicating infarction due either to a major coronary embolism or to inadvertent injury to a coronary artery. A current of injury however is a common finding after cardiac surgery due to traumatic pericarditis. Pericarditis was present in our case. This finding alone is therefore of no value in establishing a diagnosis of coronary embolism after cardiac surgery.

On the other hand, we have not encountered the peculiar T-wave changes present in this case in any other postoperative pericarditis. We attributed these T-wave changes to non-specific myocardial abnormality. Perhaps a detailed study of daily electrocardiograms after open-heart surgery will define the usual changes and unmask patterns characteristic of specific complications.

Summary

A unique cause of coronary embolism is described: open-heart surgery for correction of a ventricular septal defect.

Summarino in Interlingua

Es describite un causa unie de embolismo coronari: chirurgia a corde aperte pro corriger un defecto ventriculo-septal.

References


A 38-year-old man suddenly developed signs of heart failure with a left-to-right shunt and a diamond-shaped systolic murmur with a decrecendo diastolic murmur. The electrocardiogram showed wide diphasic P waves in leads V1-V4 and right bundle-branch block with diphasic T in V5. Autopsy disclosed an aneurysm about 5 cm. long originating immediately above the posterior aortic valve with rupture into the right ventricle.

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