Coronary Embolism: Review of the Literature and Report of a Unique Case

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This is a brief review of the literature on coronary embolism and a case report of a very unique type of coronary embolus—a piece of calcium from a calcified aortic valve. Microscopic sections revealed that the calcium plaque was covered by endothelium which had grown out from the vessel wall, thus attaching it to the coronary artery. The patient lived approximately two months after the embolism occurred.

Embolic occlusion of the coronary arteries is a very rare occurrence, but probably not as rare as the few reports in the literature would lead one to believe. Saphir,1 in 1932, reviewed 16 cases of coronary embolism from the literature, and reported 3 cases of his own observation. Garvin and Work,2 in 1932, added 3 cases which had occurred in patients with bacterial endocarditis. Porter and Vaughan,3 in 1940, reported 3 more cases occurring in patients with syphilitic aortitis associated with mural thrombi of the aortic wall. In 1941, Hamman4 was able to refer to 40 cases, 30 from the literature and 10 from the records of the Johns Hopkins Hospital. In recent years, individual cases have been reported by Ivanov,5 Park,6 Greenstein,7 Pratt-Thomas,8 and Ramos9; thus there is a total of some 45 cases of coronary embolism referred to in the literature.

From these reports there appear to be six possible sources for emboli which may occlude the coronary arteries: (1) Bacterial vegetations on the mitral or aortic valves (20 cases in the literature). (2) A mural thrombus on an arteriosclerotic or syphilitic lesion in the ascending aorta (10 cases). (3) Intracardiac mural thrombi (5 cases). (4) Thrombi in the peripheral veins (paradoxic embolism) (4 cases). (5) A thrombus or atheromatous material in a coronary artery (2 cases). (6) Thrombi in the pulmonary veins (2 cases).

We present here a case of coronary embolism in which the source of the embolus is unique.

Case Report

A 43 year old white man was found to have a systolic aortic murmur fifteen years prior to hospitalization. No history of rheumatic fever was elicited. The patient had been in good health except for occasional attacks of precordial pain which had become more severe and had occurred oftener within the last two years. On December 14, 1947, while doing physically laborious work, the patient developed shortness of breath, anginal pain and a feeling of tightness in his chest; dyspnea was so marked that he had to sit up most of the night. The next morning the patient was deeply cyanotic and was coughing up some bloody sputum. He lapsed into unconsciousness in the process of hospitalization. Oxygen, atropin and Metrazol were given, and the patient was digitalized. His condition improved after digitalization and he was discharged after a hospital stay of one week, on a maintenance dose of 0.1 mg. digitoxin.

The patient returned to work and had no complaints, except for a dull ache on the left side of the chest. About February 1, 1948 he developed a sharp precordial pain radiating to his left arm and marked dyspnea; he was rehospitalized.

The patient was tall, slender, malnourished and ashen pale in color. He spoke with great effort because of dyspnea. The apex impulse was in the seventh intercostal space in midaxillary line. The right border of the heart was 4 cm. from mid sternal line in the fifth intercostal space. A mitral systolic murmur and a harsh aortic systolic murmur were heard over the second intercostal space to the right of the sternum transmitted toward the vessels of the neck. A diastolic aortic murmur was also present but heard best in the third intercostal space to the left of the sternum. At times there was a suggestion of a systolic thrill over the aortic area. The pulse was faint and weak, the rhythm was regular, and the pulse rate was 100 per minute. The blood pressure was 90/80 in both arms. There were many moist rales in the lung bases. The liver was palpable 9 cm. below the right costal margin; it was tender and presented a faint pulsation. There was mild clubbing of

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the fingers. Laboratory studies were not significant. An electrocardiogram revealed inversion of the T wave in Leads I and II, depression of the S-T segment in Lead I, and elevation of the S-T segment in CF. Two previous electrocardiograms taken during January showed essentially the same picture. Radiographic examination of the chest revealed a pronounced enlargement of the cardiac shadow both to the right and to the left. The aortic shadow was very broad and there was calcification in the area of the aortic valve.

![Image of heart and lungs]

**Fig. 1.**—Nodular calcification of the aortic valve and the myocardial infarct at the apex with mural thrombosis.

Although the patient was irritable and anxious, he improved after being placed at rest and put on a low sodium diet and digitalis. On the twelfth hospital day, while resting in bed talking to his wife, he suddenly became deeply cyanotic and expired.

**Pathologic Examination.** The body showed cyanosis and enlargement of the superficial veins. All the serous cavities contained fluid transudates.

The heart weighed 760 grams and showed some dilatation of all chambers. The left ventricular myocardium measured 20 mm. in thickness and the right 6 mm. In the anterior wall of the left ventricle, and near the apex of the heart there was an area of infarction about 5 cm. in diameter involving the anterior wall of the left ventricle and a portion of the interventricular septum. Some intraventricular mural thrombi were found attached to the infarcted area. The aortic valve was severely stenosed and distorted by nodular masses of calcium (fig. 1). The coronary ostia were patent and the coronary arteries showed very little atherosclerosis. A small fragment of calcific material 3 mm. long was found occluding the anterior descending branch of the left coronary artery 2.5 cm. from the aortic opening of the vessel (fig. 2).

Both lungs showed moderate congestion and edema, and the lower lobes revealed small hemorrhagic infarcts. The liver weighed 1300 grams, had round margins and was severely congested. No permission was obtained for examination of the head.

Sections from the area of infarction in the left ventricle revealed the muscle to be largely replaced by granulation tissue infiltrated with chronic inflammatory cells. On the endocardial surface, there were organizing thrombi. The small coronary branches were well preserved and patent. The anterior descending branch of the left coronary artery showed a large mass of amorphous basophilic material attached to the intimal lining of the vessel by several thin pedicles of connective tissue covered by endothelium. The entire calcific mass was also covered by endothelium. The wall of the vessel was well preserved and showed no arteriosclerosis (fig. 3). The
Fig. 2.—Calcific embolus impacted in the anterior descending branch of the left coronary artery.

Fig. 3.—Section of the coronary artery at the site of the embolus. Notice the normal arterial wall and the large calcific mass completely lined by vascular endothelium.
aortic valve revealed amorphous masses of basophilic, calcific material. No bacteria were seen. The ascending aorta presented an occasional small accumulation of lipid material in the intima.

The lungs showed many heart failure cells and areas with much hemorrhage into the alveoli and bronchi. A number of organizing thrombi were seen in the small branches of the pulmonary artery. The liver revealed marked central congestion and necrosis of the central portions of the lobules.

**DISCUSSION**

It was noted in reviewing the literature that about one half of the cases of coronary embolism were associated with bacterial endocarditis. In such cases the emboli consisted of fragments of vegetations and usually contained bacteria. Saphir\(^1\) reported the presence of myocardial granulomas in 4 cases of subacute bacterial endocarditis which had been treated with antibiotics. These granulomas presented a calcific center, the result of microscopic emboli from healed and calcified vegetations of the aortic valve.

Our case is unique in that the large calcific embolus originated in a nodular, calcified aortic valve without bacterial endocarditis.

We believe that the patient here presented suffered an acute embolic coronary occlusion in December 1947. The date of this accident is confirmed by the clinical history.

The amount of organization and fibrosis of the infarcted area of the myocardium and the complete attachment of the embolus by a covering of endothelium to the vessel wall would substantiate the fact that the accident occurred several months prior to death. The rigidity and irregular shape of this calcific embolus would not permit a complete occlusion of the vessel even though the embolus was firmly impacted in the artery. These conditions would permit some blood flow through the involved vessel. This fact, we believe, accounts for the survival of this patient at the time of the initial acute embolic accident.

**SUMMARY**

The literature on coronary embolism is reviewed. The findings in an additional case are reported. This case is somewhat unique in that the source of the embolus was a calcific mass which became detached from a calcified aortic valve.

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