SELECTED REPORTS

Myocardial Infarction Associated with Coronary Angiography*

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A patient with acute myocardial infarction at the time of coronary angiography, and with demonstration of a thrombus occluding the circumflex artery, is described. On repeat angiography one year later, all vessels were patent. Possible mechanisms are discussed, but it appears likely that this case represents complete lysis of a coronary thrombus.

Myocardial infarction is a well recognized but infrequent complication of coronary arteriography. In most cases, it has usually been attributed to coronary artery dissection, dislodgement of an atherosclerotic plaque, sustained hypotension, thromboembolism or spasm of the vessel. Fatal thromboembolism following coronary arteriography has rarely been confirmed by postmortem examination since most of these patients survive. We describe a case of apparent nonfatal coronary thromboembolism during coronary arteriography resulting in complete occlusion of the circumflex artery and producing a true posterior wall myocardial infarction. At the time of a second angiogram one year later, the occluded vessel was found to be totally patent.

CASE REPORT

A 42-year-old man was admitted to Montefiore Hospital in February, 1972 for evaluation of atypical chest pain and paresthesias of his arms. Prior workup at another hospital had revealed only a hiatus hernia. He had had mild hypertension for three years, but did not receive therapy. Physical examination revealed blood pressure 122/70 mm Hg and normal general and cardiac findings. Routine laboratory values including serum glucose and cholesterol were normal. Electrocardiogram and chest x-ray film were also within normal limits.

Retrograde left ventriculography and selective coronary arteriography were performed using the Judkins percutaneous transfemoral technique, and right heart catheterization was performed via a percutaneous puncture of the right femoral vein. All right-sided pressures were normal. The arterial venous oxygen difference was 4.6 ml/100 ml and cardiac index calculated by the Fick method was 2.7 L/min/M². Left ventricular end-diastolic pressure was normal and there were no gradients across the mitral or aortic valves. On ventriculography there were no areas of hypokinesia. Contractions were excellent and there was no mitral regurgitation. Filling of the proximal portions of both right and left coronary vessels was also seen on the ventricular injection. After sublingual administration of 5 mg of isosorbide dinitrate and an intravenous injection of 0.4 mg atropine, the left coronary artery was selectively catheterized. A single injection of 10 ml of meglumine diatrizoate (Renografin-76) was made into the left coronary artery while the heart was being viewed in the left anterior oblique position. The main left and left anterior descending arteries were normally patent, but the circumflex artery was completely occluded 1 cm from its origin (Fig 1). Intraluminal thrombus was identified on cine angiography. Within moments of injection, the patient developed severe crushing chest pain, unlike the pain he had experienced before, and accompanied by diaphoresis and a drop in blood pressure to 90/70. The procedure was terminated and an electrocardiogram taken while the patient was still on the catheterization table revealed ST elevations in leads 1, 2, aVF, V₃₋₆ and ST depressions and T wave inversions in leads V₁ to V₄ (Fig 2B). The patient was transferred to the coronary care unit where a subendocardial and true posterior transmural myocardial infarction evolved on serial electrocardiograms (Fig 2C). Serum enzymes revealed a rise in CPK to 1755 and SGOT to 552 returning to normal in three days. The patient made an uneventful recovery and was discharged in three weeks.

He again developed atypical chest pain five months after discharge and in February, 1973 was readmitted for another angiogram. His blood pressure was 110/80 and the remainder of the physical examination was unremarkable, as were the routine laboratory and roentgenographic examinations. The electrocardiogram revealed a persistent R greater than S pattern in V₅, but was otherwise normal (Fig 2D). Repeat left ventriculography and selective coronary arteriography via the Judkins technique were performed. The left ventricular end-diastolic pressures were again normal and left ventriculogram in the right anterior oblique position showed no areas of decreased contraction and was identical to the earlier study. Coronary arteriography (Fig 3) showed completely patent right and left coronary arteries.

The previously visualized circumflex artery obstruction was no longer present. Since discharge, the patient has continued to have atypical chest pain and paresthesias.

DISCUSSION

In a cooperative series of 3312 coronary arteriograms, only five patients were reported to sustain a myocardial infarction during or after the procedure.1 This complica-

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Figure 1. Left coronary artery injection in left anterior oblique view February 14, 1972. Arrow indicates site of total obstruction of circumflex artery. Note intraluminal thrombus.
proximately two-thirds of its length. There was residual stenosis at the site of the previous occlusion. In the present case, there is evidence of spontaneous complete resolution of a catheter-induced thrombosis or embolus in a coronary vessel. Kordenat et al. recently described lysis of experimentally produced thromboses in the coronary arteries of dogs using local perfusion of fibrinolysin (Thrombolysin), but this technique of fibrinolysis has not yet been applied to man. Lysis of coronary thromboses has not been shown to occur naturally in the coronary vessels of man.

The possibility exists that the present case represents another example of coronary vessel spasm leading to myocardial infarction, similar to that recently described by Cheng.* However, even with severe spasm, total obstruction to flow, as we have demonstrated, is unlikely. Also, the sharp cutoff demonstrated at the origin of the circumflex in the present case is not usually seen with spasm, which produces a gradual tapering. Although it is possible that the occlusion was present prior to the first catheterization, this is unlikely because of the relation of a new type of pain, electrocardiographic changes and enzyme alterations that occurred with the procedure. Also, Campeau et al and others have shown that resolution of such an occlusion usually leaves behind an irregular, narrowed lumen. Therefore, we conclude that the occlusion in our case was a result of thromboembolism from the catheter tip, and the subsequent course is that of complete resolution.

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REFERENCES

**FIGURE 2.** Electrocardiograms recorded: A) prior to coronary arteriography February 13, 1972; B) immediately following left coronary artery injection February 14, 1972; C) time of hospital discharge February 28; D) prior to repeat coronary arteriography February 28, 1973.

Massive Thrombosis Associated with Use of the Swan-Ganz Catheter*

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A patient with severe respiratory failure due to overwhelming pneumonia was monitored with a Swan-Ganz catheter. Postmortem examination revealed massive antemortem thrombosis surrounding the Swan-Ganz catheter tip. The presence of the thrombus about the catheter tip was not suspected before death, but retrospective analysis of a variety of parameters suggested massive pulmonary vascular obstruction.

Recently the use of a flow-directed balloon-tipped catheter has been introduced to safely measure and record right atrial, right ventricular, pulmonary arterial, and pulmonary capillary wedge pressures in patients with acute myocardial infarction. At our institution, use of this catheter has been extended to the differential diagnosis and management of other cardiopulmonary disorders, including certain acute respiratory distress syndromes in the adult, such as those recently reviewed by Petty and Ashbaugh. Infrequent complications have been reported with the use of this catheter, including rare instances of thrombosis about the catheter tip.

The purpose of this report is to describe a patient who presented with an acute respiratory distress syndrome and in whom placement of a flow-directed balloon-tipped catheter in the pulmonary artery was associated with the acute development of massive thrombosis about the catheter tip, an event that was probably contributory to the patient's death. Certain clinical and physiologic features suggestive of massive pulmonary vascular occlusion, which may be valuable in the diagnosis of this serious complication were noted retrospectively.

CASE REPORT

A 63-year-old man was admitted to the hospital because of the onset of acute progressive dyspnea over the previous five days. Four years previously, peptic ulcer disease had been demonstrated. For several months prior to his hospitalization the patient had taken up to 18 tablets of a mixture of oxycodeone terephthalate, aspirin, phenacetin and caffeine (Percodan) a day for discogenic back pain and had used mineral oil nightly for several years prior to his hospitalization to relieve constipation. The onset of dyspnea was associated only with a dry cough without fever, chills, upper respiratory symptomatology, sputum production, chest pain, or gastrointestinal complaints. The patient related no history of aspiration or prior cardiac or pulmonary disease.

Physical examination revealed an acutely ill appearing man in severe respiratory distress. His temperature was 38.8°C, blood pressure 200/100 mm Hg in the supine position, pulse rate 100 per minute, and respiratory rate 30 per minute. There was dullness at the right posterior base and basal inspiratory rales bilaterally. Chest expansion was symmetric.

Supraventricular retraction as well as tracheal descent were present with each inspiration. No neck vein distention, gallop rhythm, heart murmurs, abdominal tenderness, organomegaly, or ankle edema was present.

The hematocrit level was 33 percent and the white blood cell count was 7,500/mm³, with a normal differential. Urinalysis, serum electrolyte, and creatinine values were normal. No blood was present in the stool. Gram stain of the sputum revealed moderate numbers of polymorphonuclear cells, but only scant Gram-positive and Gram-negative diplococci. Arterial blood gas measurement done while the patient was breathing room air showed a pH of 7.52, arterial carbon dioxide pressure (PaCO₂) of 30.5 mm Hg, and an arterial oxygen pressure (PaO₂) of 42 mm Hg. The electrocardiogram was unremarkable except for a QRS axis of −30°. Studies performed later in this patient's course included a normal prothrombin time, partial thromboplastin time, fibrinogen level and platelet count. The chest x-ray film on admission revealed extensive bilateral diffuse parenchymal infiltrates extending from the hilum to the peripheral lung fields. A chest x-ray film made on the second hospital day revealed further extension of the infiltrates, with nearly complete opacification of both lung fields.

At the time of admission, the diagnostic considerations included extensive aspiration pneumonia or a diffuse viral pneumonitis. The patient was initially treated with supplemental oxygen therapy by face mask and begun on intravenous penicillin. Because of continuing hypoxemia, which could not be adequately corrected with low concentrations of oxygen, ie, less than 50 percent inspired oxygen concentration (FiO₂), as well as progressive respiratory distress, nasotracheal intubation was performed and intermittent positive pressure breathing was instituted with a volume-cycled respirator (Bennett, MA-1). Hypoxemia worsened despite an FiO₂ of 100 percent oxygen and about 24 hours after the patient was admitted, continuous positive pressure breathing (CPPB) with an FiO₂ of 100 percent was instituted, with an end-expiratory pressure of 12 cm H₂O. It was necessary to sedate the patient heavily with barbiturates to prevent him from fighting against the respirator. His minute ventilation was then maintained with a fixed rate and tidal volume. He tolerated this without any systemic hypotension. The PaO₂ rose from 45 mm Hg to 65-70 mm Hg within 15 to 30 minutes after the institution of CPPB. To aid in the assessment of fluid administration as well as to determine whether