CASE REPORT

Skeletal Muscle Coronary Enbolism:
A Complication of Coronary Angiography

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SUMMARY A fatal case of skeletal muscle coronary embolism after transfemoral coronary angiography is described. The anatomy of the femoral triangle is such that low, oblique, lateral or transarterial punctures may deliver skeletal muscle into the vascular system to be advanced on contrast injection into the coronary circulation.

ANGIOGRAPHIC VISUALIZATION of the coronary arteries, although valuable, has risks and complications. These have been well-documented for transbrachial and transfemoral approaches.\(^1\)\(^2\) Coronary artery dissection,\(^3\)\(^4\) coronary artery thromboembolism\(^5\) and introduction of detritus into the circulation with potential coronary thromboembolism\(^6\)\(^7\) have been observed.

The transfemoral approach allows for a stable catheter position without the technical complexity of surgical arterial exposure, and allows direction of the catheter through a relatively tissue-free femoral space. However, this space is surrounded by a number of skeletal muscles (fig. 1). Adherence of skeletal muscle to the catheter tip is remotely possible, and occurred in the following case. We do not believe any such complication has been previously reported.

Case Report

A 48-year-old truck driver was hospitalized because of progressive symptoms of left ventricular failure due to mixed aortic valve disease. Three months before hospitalization he had developed angina pectoris. Cardiac catheterization was performed using a Swan-Ganz catheter into the right heart, transseptal puncture of the left atrium and retrograde aortic catheterization using a polyethylene pigtail catheter. A 75 mm Hg gradient between the left ventricle and aorta was identified. The aortic valve was heavily calcified. Cardiac index was reduced to 2.25 l/min/m\(^2\). Contrast injection into the aortic root revealed severe aortic insufficiency. Selective coronary arteriography was then performed after catheter exchange with a \#8 left Judkins polyethylene catheter. Selective injection was performed in multiple positions, and revealed evidence of virtually complete obstruction of the proximal left anterior descending coronary with some delayed distal filling. Upon removal of the catheter after the final injection, spontaneous ventricular fibrillation occurred and was terminated by DC countershock. Fifteen minutes later frequent ventricular premature beats occurred, and a second episode of ventricular fibrillation ensued with subsequent defibrillation. The Swan-Ganz catheter and an arterial line were kept in place. Frequent injections of lidocaine were given to control ventricular ectopy, and the patient was transferred in stable condition to the coronary care unit. Subsequently he developed grand mal seizures, probably resulting from lidocaine administration, and was intubated. Ventricular fibrillation occurred a third time and again was successfully reverted.

The patient stabilized overnight. The next day a pattern of acute anterior wall myocardial infarction was seen. Left axis deviation with complete right bundle branch block appeared. Transient episodes of complete heart block occurred, and a pacing catheter was inserted percutaneously and advanced to the right ventricular apex. The patient continued to deteriorate despite pacing. There was clinical and radiographic evidence of congestive heart failure and recurrent episodes of severe chest pain were experienced. The patient developed cardiogenic shock and could not be resuscitated. A pericardiocentesis was performed to exclude the possibility of left ventricular rupture.

The coronary arteriograms were reviewed. The findings were thought to be consistent with dissection or intraluminal clot in the left coronary system. Cineangiograms in the right anterior oblique projection revealed near-complete obstruction of the left anterior descending coronary with contrast material surrounding the block. Review of the aortogram previously performed did not reveal such an obstruction.

Autopsy showed fragments of skeletal muscle introduced into the main left coronary artery. There was an 80% atheromatous occlusion of the proximal anterior descending artery and a 50% occlusion of the proximal left circumflex coronary artery. The right coronary was normal. There was acute infarction of the anteroapical left ventricle and interventricular sep-

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tum and acute pulmonary edema. Fresh laminated thrombus was noted distal to the skeletal muscle fragments which had produced complete anterior descending occlusion.

**Comment**

The anatomy of the femoral triangle (fig. 1) reveals that percutaneous puncture of the femoral artery at various positions and angulations could allow for fragmentation on entry of the anterior sartorius and adductor longus muscles if the needle insertion were too low or lateral. Through-and-through arterial puncture with withdrawal into the arterial lumen could carry fragments of iliacus, psoas or pectineus muscle into the arterial stream. Fragments could lodge within the artery, on the guidewire or on the catheter tip.

The introduction of skeletal muscle into the coronary circulation after prior uncomplicated aortography is difficult to explain. It is possible that muscle fragments already were on the catheter and some embolized to remote areas subclinically after aortography. Fragments may have been attached to the guidewire on exchange or were picked up by traversing the anterior skeletal muscle on insertion of the coronary catheter at a later time. The exact time of coronary muscle embolization cannot be determined, since coronary arterial occlusion may not have been completed until the occurrence of coronary throm-

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**Figure 1.** Illustration of the skeletal muscle and vascular anatomy of the femoral triangle.

**Figure 2.** The main left coronary artery (A) contains fragments of skeletal muscle and cellular debris. Similar fragments were present in multiple sections of the same vessel. In deeper sections (B) the skeletal muscle fragments merge with an occluding, fresh thrombus.
bosis as a reaction to the presence of foreign muscle tissue.

Techniques of catheter exchange routinely include a preliminary withdrawal of blood from the catheter before its removal in order to separate any thrombogenic substance before exchange. The guidewires are also cleansed and the new catheter is flushed. However, no technique can release adherent tissue fragments at the guidewire tip.

Microscopic sections from the coronary arteries at autopsy (fig. 2) clearly demonstrated skeletal muscle fragments associated with thrombosis of the anterior descending branch of the left coronary artery. The localization corresponded to the time of the operative procedure. Artifactual “floaters” appeared unlikely, since muscle fragments appeared in multiple sections of the same vessel and merged with fresh thrombus.

Despite careful attention to techniques, such complications can occur. A technique for prevention suggests an arterial puncture just beneath the inguinal ligament with a direct penetration into the artery, not through it. Oblique, low and transarterial punctures run the risk of potentially attaching muscle or lipid substance to intravascular catheters or wires, with possibly lethal consequences as described.

References

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