Dissection of the Left Coronary Artery Complicating Retrograde Left Heart Catheterization*

Kazuto Kitamura, M.D., Frederick L. Gobel, M.D., and Yang Wang, M.D., F.C.C.P.

Dissection of the coronary artery caused by a catheter is reported. Progressive hematoma formation accounted for the delay in onset of symptoms. There was an extensive area of myocardial infarction. Microscopic examination revealed an intimal tear with dissection into the adventitia and nearly complete occlusion of the coronary artery lumen.

Although dissection of the coronary artery has been recognized recently as an infrequent complication of selective coronary arteriography,

this has not been reported during left heart catheterization. This report concerns a patient who developed acute myocardial infarction and severe left ventricular failure during diagnostic left heart catheterization. Injury of the intima and dissection of the main left coronary artery was confirmed at necropsy.

Case Report

A 53-year-old woman was admitted for evaluation of symptomatic mitral stenosis. Chest pain and exertional dyspnea had been present for several years and she had recently noted increased fatigue. The chest pain was not typical of angina pectoris. There was no history of pulmonary edema. She was receiving digitalis.

Physical examination revealed a middle-aged white woman with mild acrocyanosis. Her apical pulse was irregular at 85 beats/minute. The blood pressure was 150/95 mm Hg. There was an opening snap, loud first heart sound, apical diastolic rumble, and a soft systolic regurgitant murmur over the lower left sternal border. There was no neck vein distension, hepatomegaly or peripheral edema.

Admission laboratory data including a complete blood count, urinalysis, BUN, creatinine, serum electrolytes, SCOT, and LDH were all normal. The electrocardiogram revealed atrial fibrillation and non-specific ST-segment changes (Fig 1A). The vectorcardiogram displayed a normal QRS loop. Cardiac fluoroscopy and roentgenograms of the heart showed left atrial enlargement and minimal calcification of the mitral valve.

On September 5, 1968, she underwent right and left heart catheterization. By the Seldinger technique, a 75 cm Teflon arterial catheter with an internal diameter of 1.09 mm and an external diameter of 1.72 mm was percutaneously
Following coronary artery dissection, the electrocardiogram (right) reveals evidence of an acute anterior myocardial infarction. Sinus rhythm and left axis deviation are now present.

introduced into the right brachial artery and advanced to the left ventricle, using a flexible tip guidewire. Simultaneously, another Teflon catheter was similarly advanced from the left brachial artery to the ascending aorta and positioned 2 cm above the aortic valve. The guidewires were then withdrawn. There were no symptoms, electrocardiographic or blood pressure changes immediately after catheter positioning. Approximately 20 minutes later, however, the patient began to complain of increasing substernal chest pain, associated with T wave inversion and subsequent ST-segment depression in the precordial leads (V2-V6). This was followed by an acute drop in aortic pressure from 152/92 mm Hg (mean 111 mm Hg) to 87/49 mm Hg (mean 63 mm Hg). The left ventricular end-diastolic pressure increased from 4 to 19 mm Hg and the mean pulmonary artery wedge pressure increased from 12 to 23 mm Hg. The average end-diastolic gradient across the mitral valve decreased from 5 to 1 mm Hg, suggesting a decreased cardiac output since the heart rate decreased only by 4 beats/minute (64 to 60 beats/minute). The cardiac output at this time was 2.35 L/min with a cardiac index of 1.62 L/min/m². The patient then developed more profound hypotension and a series of ventricular arrhythmias, including ventricular tachycardia and ventricular fibrillation. She was vigorously treated sequentially with DC countershock, lidocaine, procaine amide, bretylium tosylate and diphenylhydantoin. The cardiac rhythm finally reverted to atrial fibrillation, then spontaneously changed to sinus rhythm. An electrocardiogram 12 hours later showed the development of left axis deviation and an acute anterior myocardial infarction (Fig 1B). Creatinine phosphokinase rose to a peak of 2,259 units (normal 1-6.5 Wroblewski unit in our laboratory), SCOT 14,400 units (normal: under 35 mIU), and total LDH 34,400 units (normal: under 425 mIU), with a heat stable LDH fraction of 1,295 units (normal: under 100 mIU). Despite sinus rhythm and aggressive

Figure 2A. Drawing (left) of a photomicrograph of the main left coronary artery 2 cm from its origin revealing almost complete occlusion of the coronary artery by a subadventitial hematoma (right).
therapy, she remained in shock and became anuric. Approximately 80 hours after cardiac catheterization, she developed cardiac arrest and could not be resuscitated.

At necropsy, there was an extensive area of acute myocardial infarction involving the anterolateral wall of the left ventricle, which was confirmed by microscopic examination. The left ventricular wall was 1.5 cm in thickness. The mitral valve was stenotic and calcified with an estimated orifice of 1 cm². Examination of the coronary arteries showed a subadventitial hematoma in the main left coronary artery 2 cm from its origin, causing almost complete occlusion of the lumen of the left coronary artery (Fig 2A). The point of occlusion was proximal to the origin of the left circumflex artery. A tear of the intima and the media presumably made by the catheter was demonstrated near the orifice of the left coronary artery. There was evidence of moderate left coronary atherosclerosis with narrowing of the lumen near the point of injury (Fig 2B). The right coronary artery was normal and distal to the occlusion, the left anterior descending and left circumflex arteries were patent. There was no coronary artery aneurysm.

FIGURE 3. A Teflon arterial catheter and a flexible tipped guidewire (above). A guidewire is introduced and extended beyond the tip of the Teflon arterial catheter during advancement and manipulation (below).

DISCUSSION

The retrograde technique of left heart catheterization is now widely used. Since the tip of the Teflon catheter has a sharp rigid edge, our practice is to advance the flexible tip of the guidewire 2–3 cm beyond the end of the catheter while advancing or manipulating the catheter (Fig 3). On occasion, incidental entrance of a guidewire and catheter into a coronary artery has been noted, although by forming a loop with the flexible spring-tip of the guidewire, an attempt is specifically made to avoid this. Slipping into the right coronary artery is frequent but can easily be recognized by the course of the guidewire under fluoroscopy. Entrance into the left coronary artery is sometimes mistaken for having crossed the aortic valve into the left ventricular cavity at fluoroscopy, but the site can be confirmed by observing the pressure contour.

Coronary artery dissection may occur spontaneously,¹ has been reported as a rare complication of selective coronary arteriography,²–⁴ and may occur during cannulation for direct coronary artery perfusion at open heart surgery.⁵ Coronary artery dissection has not previously been reported during retrograde left heart catheterization. Fortunately, this complication must be extremely rare but it should be emphasized that once any instrument, catheter or flexible tipped guidewire is introduced into the coronary artery, it can produce the complication of arterial dissection.

In the present case, the lumen of the left main coronary artery apparently became rapidly occluded by a subadventitial hematoma after the guidewire or the catheter made a tear in the intima. There was a time lag between catheter manipulation and onset of symptoms, accounted for by a growing hematoma, in contrast to complete occlusion of a
coronary artery by the catheter itself, which usually causes immediate appearance of symptoms and electrocardiographic changes. Rapid development of severe left ventricular failure was quite striking and was due to an extensive myocardial infarction involving the left ventricle.

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REFERENCES

Reprint requests: Dr. Kitamura, Department of Medicine, University of Minnesota Medical School, Minneapolis 55455

Tylosis and Intrathoracic Neoplasms*

Walter D. Schwindt, M.D.,** Louis C. Bernhardt, M.D.,† and Sture A.M. Johnson, M.D.†

The relationship of tylosis (hyperkeratosis palmaris et plantaris) and intrathoracic neoplasms has been cited previously. Two cases are presented, one of whom exhibited the skin manifestations of tylosis coexisting with an extensive squamous cell carcinoma of the esophagus. The second patient demonstrated the skin lesions coexisting with bronchogenic carcinoma. Tylosis has been considered an hereditary cutaneous disorder which seems to be controlled by a single autosomal gene with high penetration and heterozygous effect. The physical findings of tylosis should alert the physician to the possibility of intrathoracic neoplasms as this association is now well documented.

Skin lesions such as the adult type of acanthosis nigricans and dermatomyositis quite often coexist with a malignant tumor. The report by Howell-Evans and associates1 brought to light the possible association of hyperkeratosis palmaris et plantaris (tylosis) and carcinoma of the esophagus. In another report from England, there was a family with tylosis, sliding hiatus hernia, and one member with carcinoma of the esophagus.2 This is a report of two patients with tylosis and intrathoracic neoplasms.

CASE 1

Case Reports

This 58-year-old white man entered the University of Wisconsin Medical Center with a six-month history of progressive dysphagia and recent hoarseness. A barium esophagogram demonstrated an obstructing lesion in the upper thoracic esophagus. At esophagoscopy, a fungating tumor was found. Biopsy was performed and a hard palpable scalene node was interpreted as grade 4 squamous cell carcinoma. The patient received 7000 rads during a five-week period with subsequent improvement in his dysphagia. Six months later he developed pain in his left buttock. A gluteal mass was biopsied and found to be metastatic carcinoma. On this admission, it was observed that he had extremely hyperkeratotic dry palmar and plantar skin. Clinically, this was tylosis. Although this patient had been a farmer, he had not worked for one year prior to admission due to the chronic illness.

This man expired seven months after his initial admission. At postmortem examination, there was an extensive squamous cell carcinoma of the esophagus with metastasis to the cervical, mediastinal, and paraesophageal lymph nodes, in addition to the liver, adrenals, lumbar spine and gluteal muscle.

CASE 2

This 49-year-old white woman was admitted to the dermatology service for evaluation of thickness and dryness of the skin of the hands and feet. She also complained of recent weakness of her right arm and leg. For at least ten years, she had dry and rough skin of her elbows, but preceding her admission, the palmar and plantar skin had become thick and dry. A diagnosis of tylosis was made, with probably associated neoplasm. This clinical impression was confirmed by biopsy of the skin of the palm which revealed marked thickening of the corium and moderate acanthosis. The rete pegs were broadened and moderately elongated, and a few chronic inflammatory cells were noted in the upper dermis, consistent with the diagnosis of hyperkeratosis palmaris et plantaris.

Admission chest x-ray examination showed a right peritracheal mass suggestive of a pulmonary tumor. A palpable right supraclavicular lymph node was removed and it contained metastatic squamous cell carcinoma. A search was made for the primary tumor, but despite bronchoscopy and esophagoscopy with cytology, gastrointestinal radiographs, an intravenous pyelogram and thorough ENT examination, no definite focus could be found. Skull films and brain scan were interpreted as normal, but the EEG demonstrated an abnormal focus in the left frontal area. With carotid arteriograms, a left frontoparietal mass was seen. The clinical impression was squamous cell carcinoma of the lung with cervical lymph node and cerebral metastasis.

She was given systemic 5-fluorouracil and radiation therapy to the "primary" tumor and metastasis. A short but definite remission was obtained following which there was progressive neurologic deterioration. Despite supportive treatment which included diphenylhydantoin (Dilantin) and

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SCHWINDT, BERNHARDT AND JOHNSON