

Atheromatous Embolization during Angiography (A Case Report)

Sang Yong Song and Je G. Chi

Department of Pathology, Seoul National University College of Medicine
Seoul 110-744, Korea

= Abstract =Cholesterol embolization is one of the serious complications of ulcerative atheromatous plaque. Ischemic changes in the lower extremity, brain and other organs including the kidney, pancreas and gastrointestinal tract due to atherosclerotic microemboli have been well documented in autopsy cases in the western world. However, it has been seldom reported in the Korean literature. We report a case of cholesterol embolization of a foot that happened during coronary angiography with resultant gangrene necessitating amputation.

A 60-year-old man visited hospital for the evaluation of angina pectoris. He felt a sudden severe abdominal pain during the coronary angiography followed by loss of pulsation of the dorsalis pedis arteries. Acute renal failure, glucose intolerance and gangrene in both feet developed almost simultaneously, and Syme operation was done. Dissection of the dorsalis pedis artery demonstrated segmental occlusion by cholesterol emboli with various stages of organization.

Key Words: *Atheromatous embolization, Cholesterol emboli, Coronary angiography, Gangrene.*

INTRODUCTION

Cholesterol embolization is a serious complication of ulcerative atheromatous plaque (Cotran *et al.* 1989). Ulceration of atheromatous plaque is induced either spontaneously or secondarily (Schwartz and McDonald 1987). Angiographic procedures, cardiovascular surgery and treatment with anticoagulants are favorite causes (Drost *et al.* 1984; Rosansky and Deschamps 1984). Clinically, it is manifested by hypertension, pain, confusion and skin discoloration or loss of pulsation occurred during or shortly after angiographic

procedure (Rosansky and Deschamps 1984). But it is not specifically described as a complication of arteriography in radiologic or pathologic texts.

Atheromatous emboli are also called, "cholesterol emboli", because cholesterol clefts intermingled with granulation tissue are the striking histologic feature. The diagnosis is established by microscopic examination of affected tissue, usually skin (Schwartz and McDonald 1987), but its demonstration in amputation specimen which is complicated by coronary angiography is rarely reported. This paper deals with a patient with multiple cholesterol embolization of the foot, that occurred during coronary angiography.

Received June 1992, and in final form September 1992.

서울대학교 의과대학 병리학교실 : 송상용, 지제근

CASE REPORT

A 60-year-old man underwent elective coronary angiography(CAG) through a percutaneous femoral artery approach. He was admitted to the hospital for the evaluation of unstable angina pectoris which had first developed 10 years ago. The chest pain had been intermittent, squeezing in nature, and radiated to both shoulders. From five months prior to admission, frequency, duration and severity of the angina had been increasing. There was no previous history of hypertension, diabetes

mellitus and vasculitis. He had smoked 2packs for 20 years and had taken a small amount of alcohol every day. His younger brother died of a cerebrovascular accident. Physical examination and initial laboratory data were within normal limits except for the elevated levels of

Table. 1. Pulsations of arteries after coronary angiography

Side	Femoral	Popliteal	Posterior tibial	Dorsalis Pedis
Right	++	+	+	-
Left	+	+	(- → +)	(- → +)

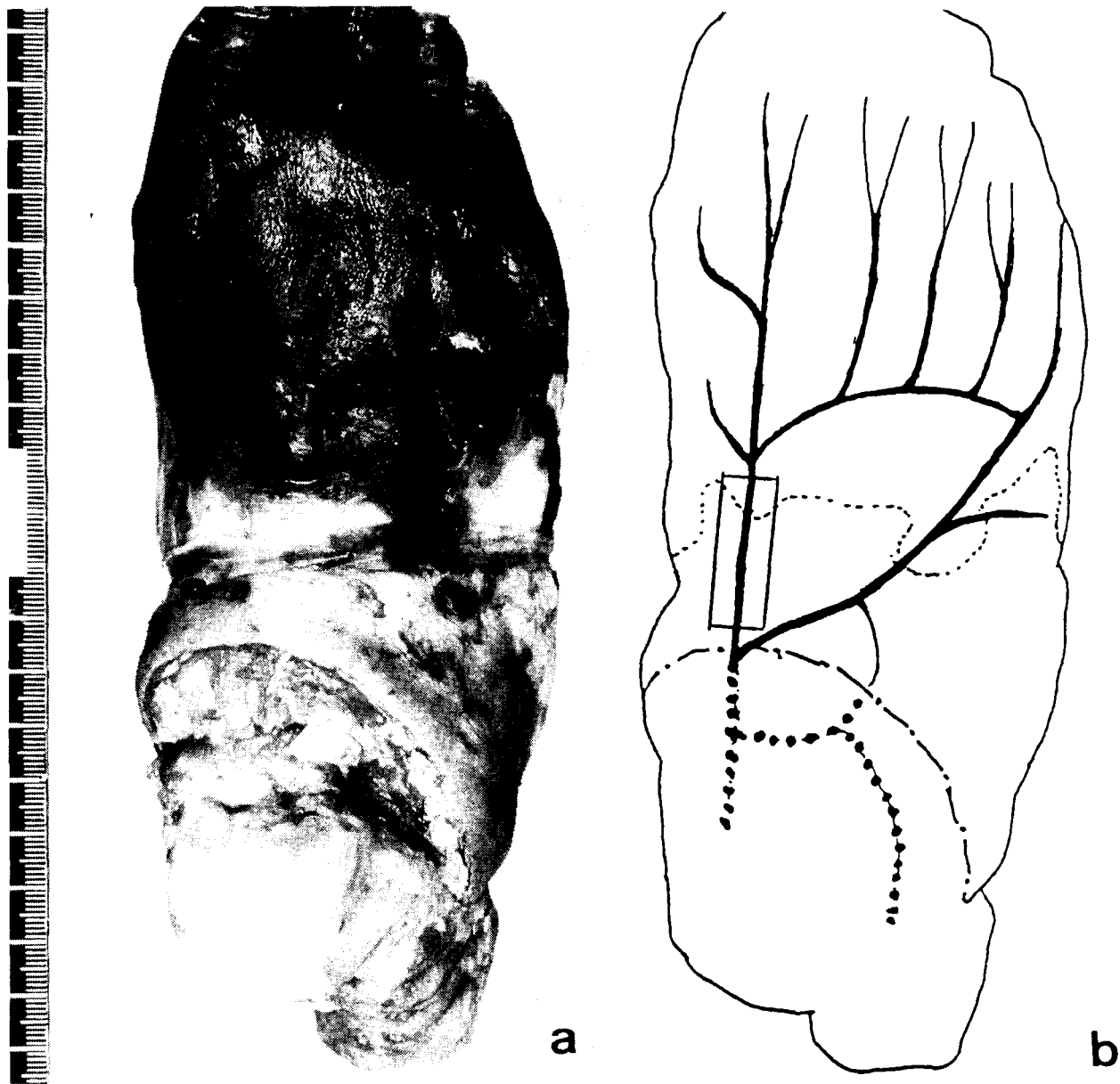


Fig. 1. a) Right foot amputated by Syme operation shows dry gangrene in the territory of the dorsalis pedis artery. b) Schematic illustration of the arterial supply of the foot dorsum. Sampling is done from the proximal segment of the dorsalis pedis(box).

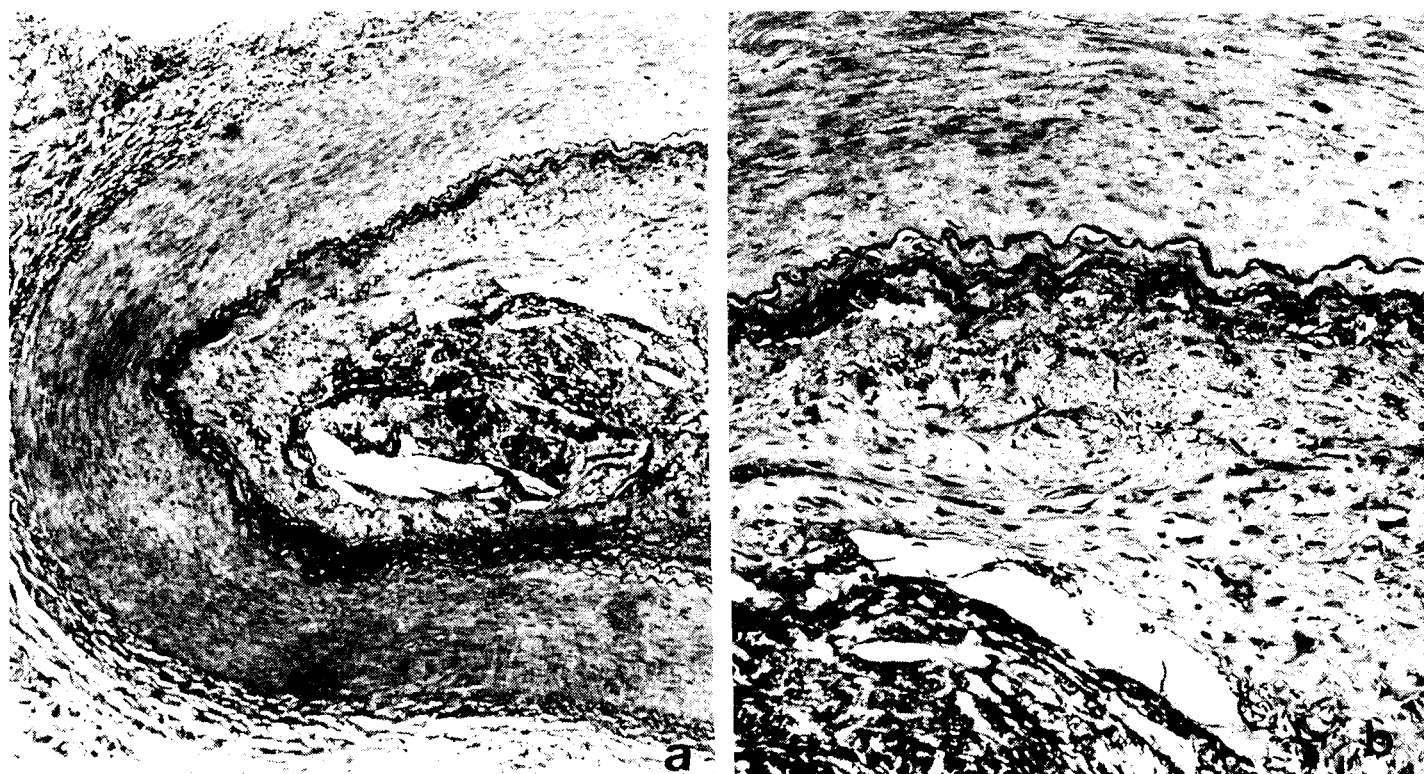


Fig. 2. a) Low power photomicrograph of proximal dorsalis pedis occluded by loose granulation tissue containing several cholesterol clefts. There is no lammellation or fibrous cap. b) High power view shows relatively well preserved media and adventitia of the involved artery. Note preserved lamina elastica interna(Verhoeff's elastic stain, x40, x100).



Fig. 3. High power view of cholesterol embolus showing many biconvex clefts intermingled with hyalinized and fibrous connective tissue(H&E, x100).

creatinine phosphokinase and lactic dehydrogenase. Electrocardiogram was not remarkable. At the end of CAG, the patient felt a sudden severe abdominal pain followed by loss of pulsation in bilateral feet(Table 1). Oliguria and hypertension(250/130mmHg) were also associa-

ted in that day. Laboratory data after CAG were as follows ; serum total protein 5.4g/dl, albumin 2.6g/dl, blood urea nitrogen 47mg/dl, creatinine 4.4mg/dl. creatine phosphokinase 26,620IU/l, lactic dehydrogenase 1,356IU/l, aspartate aminotransferase 235IU/l, alanine aminotransferase 117IU/l, fast blood sugar 336mg/dl, white blood cell count 17,800/ μ l, erythrocyte sedimentation rate 53mm/hr, and urine glucose 3+. CAG revealed 90-99% occlusion of the left anterior descending and circumflex arteries. General supportive care improved renal function and blood glucose level 3-4days later. A normal pulsation of the dorsalis pedis artery returned to the left foot after hyperbaric oxygen therapy. However the right foot failed to improve, and Syme operation was done one month after CAG. At seventh postoperative day, pulmonary embolism and localized infarction were complicated but successfully managed. Two months after CAG, he showed several episodes of altered mentality.

Pathologic findings : The right foot, separ-

ated at the ankle joint, measured 21×8×7cm. The distal two-thirds of the foot showed a dark bluish discoloration with soft friable consistency(Fig. 1a). Cut sections revealed subcutaneous pus collections with foul odor. The dorsalis pedis artery was traced for a length of 8cm. Serial sections of this artery revealed luminal occlusion that involved a proximal 2cm segment(Fig. 1b). There was some soft, gray-white material lodged in the arterial lumen. Microscopically, a segment of the proximal dorsalis pedis artery was occluded by loose granulation tissue containing several biconvex clefts, cholesterol microemboli, that were in various stages of organization(Fig. 2 & 3). Several foci of recanalization were noted. The intimal portion was also involved in this organization process, but there was no lamellar organization or fibrous cap to suggest an in situ origin of the occlusive or atheromatous lesions. The media, adventitia and intima of the remaining segment were devoid of arteriosclerotic change. Adjacent soft tissue showed varying degrees of chronic nonspecific inflammation and ischemic neuropathy.

DISCUSSION

Although thromboembolism after angiography is a well known complication of cardiovascular catheterization, the precise pathophysiology of this serious complication is not specifically described in radiologic or pathologic texts(Rosansky and Deschamps 1984). This may be partly due to the paucity of published data on this complication. In 1963, Lang reported nine cases of postangiographic embolism out of 11,402 cases of percutaneous angiography using the Seldinger technique. In 1987, Ramirez *et al.* detected cholesterol embolization in 25.5% of 71 autopsy specimens who had a history of angiography, in comparison with 4.3% in an age and disease-matched control population. Antemortem diagnosis may be extremely difficult because it may either be asymptomatic or only mildly symptomatic. Furthermore, cholesterol emboli can spread into

almost any organ and cause a diverse clinical picture. Our case also showed the involvement of multiple organs including the pancreas, kidney and peripheral small arteries of the lower legs. Although the general outcome of cholesterol embolization is poor and progressive, immediate and careful supportive care would be effective in treatment as in our case.

Pathologically, primary atherosclerotic lesion and other types of vasculitis such as periarteritis nodosa, B rger's disease and granulomatous arteritis should be differentiated in diagnosis(Soh 1957; Hoyer *et al.* 1959). Generally, primary atherosclerosis is a disease of large and medium-sized muscular arteries and elastic arteries. The basic lesion is an atheromatous plaque usually showing zonation consisting of a superficial fibrous cap and deeper necrotic core. And the distribution is rather diffuse or patchy than segmental. But in the case of cholesterol embolization, it could involve the small artery or arterioles and usually with an intact arterial wall. Although microemboli are seen in multiple organs, their distribution is quite segmental. Microscopically, differential diagnosis from the other vasculitides is relatively easy because of the usual presence of cholesterol clefts.

Other approaches such as a brachial approach may be considered for the purpose of decreasing the cholesterol embolization, because most microemboli originate from the abdominal aorta and iliofemoral arteries(Perdue and Smith 1969). But the transbrachial approach also has several complications including thrombosis, hematoma and brachial plexus injury(Rosansky and Deschamps 1984). Thus, gentle maneuver and careful observation of the patient during or after catheterization are necessary. Amputation specimens or autopsy material that were associated with previous angiography should be carefully dissected, and the proximal segment of the artery must be serially sectioned.

REFERENCES

- Cotran RS, Kumar V, Robbins SL. Robbins pathologic basis of disease. 4th ed. , WB Saunders, Philadelphia, 1989
- Drost H, Buis B, Haan D, Hillers JA. Cholesterol embolism as a complication of left heart catheterization. Report of seven cases. *Br heart J* 1984 ; 52 : 339-42
- Hoye J, Teitelkaum S, Gore I, Warren R. Atheromatous embolization. A factor in peripheral gangrene. *New Eng J Med* 1959 ; 261 : 128-31
- Lang EK. A survey of the complications of percutaneous retrograde arteriography. *Radiology* 1963 ; 81 : 257-63
- Perdue GD, Smith RB. Atheromatous microemboli. *Ann Surg* 1969 ; 169 : 954-9
- Ramirez G, O'Neill, Jr WM, Lambert R, Bloomer A. Cholesterol embolization. A complication of angiography. *Arch Intern Med* 1978 ; 138 : 1430-2
- Schwartz MW, McDonald GB. Cholesterol embolization syndrome. Occurrence after intravenous streptokinase therapy for myocardial infarction. *JAMA* 1987 ; 285 : 1934-5
- Soh MS. Atheromatous embolism. A report of 12 autopsied cases. *Korean J Pathol* 1957 ; 1 : 37-40
- Rosansky SJ, Deschamps EG. Multiple cholesterol emboli syndrome after angiography. *Am J Med Sci* 1984 ; 288 : 45-8