Early Occlusion and Late Stricture of Normal Coronary Arteries Following Blunt Chest Trauma*

R. J. Wainwright, M.B.; A. C. Edwards, M.B.; M. N. Malsey, M.D.; and E. Soutton, M.D.

A 27-year-old man had inferior myocardial infarction following superficial chest trauma. Coronary arteriography documented an isolated total occlusion of the right coronary artery, probably caused by localized extracoronary compression, and no other evidence of intrinsic coronary disease. The left anterior descending artery later developed a localized proximal stenosis, most likely due to mural thrombus, which progressed rapidly to cause anterior myocardial infarction despite coronary artery surgery. The etiology of this lesion is discussed and support given to the “encrustation” hypothesis of human atherosclerosis. Exercise thallium scintigraphy proved helpful in the management of this case.

Minor myocardial damage following blunt chest trauma is now recognized fairly frequently,1 but frank myocardial infarction is an uncommon sequel usually associated with preexisting coronary artery disease,2 rarely with entirely normal coronary arteries.3-4 This report documents right coronary artery (RCA) occlusion and inferior myocardial infarction following superficial chest trauma in a young man who was presumed to have previously normal coronary arteries. A mural stricture later developed in the left anterior descending (LAD) coronary artery and underwent rapid progression. Observations are made on the pathogenesis of human atherosclerosis in the light of this sequence.

CASE REPORT

On Sept 25, 1976, a 27-year-old telephone engineer presented at Guy’s Hospital with severe crushing central chest pain of three hours’ duration. Two weeks previously, switching gear (9.13 kg in weight) had fallen from a ladder and struck his lower sternum. He was winded but recovered quickly and resumed work. On arrival, he was afebrile with a regular pulse of 84 beats per minute and blood pressure, 110/70 mm Hg (14.6/9.3 kPa). There was a small tender abrasion over the lower sternum but no ecchymoses. Inspection and palpation of the cardiovascular system was normal. On auscultation, a fourth heart sound and a loud pericardial friction rub were heard with normal first and second heart sounds. The lung fields were clear. The chest roentgenogram was normal with no bony fractures. Serial ECGs showed acute transmural inferior myocardial infarction with pathologic Q waves in leads 2, 3, and aVF. The initial serum CK was 155 units/L (normal < 90 units/L) which rose to 2,057 units/L in 24 hours. There was a leukocytosis of 15,800 cells/μl with a rise in the erythrocyte sedimentation rate (ESR) to 21 mm hr⁻¹. Urinalysis and routine biochemical screening were normal including fasting blood glucose, cholesterol, and triglyceride levels. He made an uneventful recovery. Exercise thallium-201 (²⁰¹Tl) myocardial scintigraphy performed three weeks later showed severe persistent tracer deficit in the inferior myocardial wall attributable to the recent necrosis. The rest of the scintigram was normal (Fig 1A [left]).

A coronary arteriogram was performed on Oct 20, 1976, despite the absence of symptoms. The left ventriculogram showed regional hypokinesis in the inferior wall, and the left ventricular end-diastolic pressure was 12 mm Hg. The left coronary artery was entirely normal but the RCA was totally occluded at the acute cardiac margin with retrograde opacification of the distal vessels. The patient remained asymptomatic receiving no therapy and returned to work.

In August 1977, he developed typical angina pectoris and an exercise test provoked angina at a total work load of 5,850 kpm with no abnormal ECG changes. Repeat exercise ²⁰¹Tl scintigraphy demonstrated severe tracer deficit in the ventricular septum in addition to the previous inferior wall defect (Fig 1B [center]). This septal defect was inapparent on re-imaging the myocardium six hours later due to myocardial redistribution of tracer (Fig 1C [right]), suggesting severe ischemia of the LAD coronary artery. Another coronary arteriogram performed on Aug 22, 1977, confirmed this interpretation. The left main coronary artery remained normal, but a new severe stenosis was seen in the proximal portion of the LAD coronary artery (Fig 2). Sublingual nitroglycerin caused no alteration in its appearance. However, the RCA was no longer obstructed and appeared normal apart from some persistent irregularity of the proximal segment (Fig 3). There was no clinical evidence of arteritis, and all laboratory investigations, including repeated ESR and serum lipid estimations, were normal.

He continued to experience angina on slight exertion, and another coronary arteriogram on Sept 23, 1977, showed progression of the LAD lesion to 99 percent luminal narrowing, and urgent saphenous vein coronary bypass graft surgery was performed. At operation, there was no apparent abnormality of the coronary arteries, but retrograde probing of the

*From the Department of Cardiology and Nuclear Medicine, Guy’s Hospital, London, England.
Reprint requests: Dr. Wainwright, Guy’s Hospital, Cardiac Department, London SE1 9RT, England.

Figure 1. Exercise thallium-201 myocardial scintigrams in left anterior oblique view. A (left), Sixteen days after trauma. An uptake defect is seen in inferior wall of left ventricle with normal tracer uptake in septum and posterolateral wall, appearances consistent with an isolated dominant right coronary artery occlusion. B (center), Eleven months after trauma. In addition to inferior wall defect seen previously, new severe uptake defect has appeared in septum which undergoes reperfusion several hours later. C (right), Suggests ischemia in distribution of left anterior descending coronary artery.
time (two weeks) between trauma and presentation, this causal relationship must remain speculative. Coronary thrombosis was an unlikely cause of this appearance, particularly as the distal RCA was still patent at the time of study. This conclusion is supported by the absence of coronary thrombosis in any of the 105 cases studied by Parmley et al. Resolution of obstruction with the return of a relatively normal right coronary arteriogram further supports this suggestion. Extracoronary compression from hemorrhage and edema has not been previously reported as a cause of myocardial infarction.

In the case reported by Oren et al, it was suggested that coronary occlusion had occurred in normal coronary arteries following a blow upon the chest, while Harthorne et al also report a case of traumatic myocardial infarction with entirely normal coronary arteries. In both cases, inferior myocardial infarction had occurred but coronary arteriography was not undertaken until some months after the event, and by then, trauma to the coronary vessels may not be evident. It was interesting that our case also sustained inferior myocardial infarction, but early coronary arteriography showed occlusion of the RCA which was not present in the arteriogram 11 months later. The conclusion drawn by Harthorne et al that myocardial infarction had occurred purely on the basis of traumatic myocardial contusion must therefore be regarded with caution.

The lesion which developed in the proximal LAD coronary artery was almost certainly a mural thrombus. This interpretation is suggested by its eccentric position, smooth surface with long tails, and its rapid later progression. It is more difficult, however, to relate this lesion to the time of chest trauma when the left coronary artery was angiographically normal. Endothelial damage in the proximal LAD coronary artery may have been caused by altered shear stress following trauma and would not necessarily be detected at angiography. Slow net accumulation of lipid at a site of defective endothelialization may then have initiated mural thrombosis which became incorporated into a plaque.

The natural history of a mural thrombus in the coronary arteries of man has not been previously reported. Salimi et al have demonstrated that experimental coronary artery thrombi grow for at least 72 hours with a balance between their dissolution and reformation. Our case supports the concept that mural thrombosis occurs in vivo and may exhibit progress growth over one month. It is likely that massive myocardial infarction would have occurred in the absence of surgical intervention. Thus, the natural history of this coronary lesion does not support the conclusions of Erhadt et al that coronary artery thrombi develop only after infarction.

In conclusion, this case emphasizes the need for early coronary arteriography in the investigation of patients with suspected myocardial trauma and that further arteriography may be necessary to document the resolution or evolution of coronary disease even in the absence of symptoms. Myocardial perfusion imaging is a valuable noninvasive indicator of coronary constriction and can also be used to monitor progress.

Figure 2: Eleven months after trauma, repeat left coronary arteriogram in left anterior oblique view shows a severe stenosis of proximal left anterior descending coronary artery which was previously normal.

LAD artery revealed an obvious stenosis. No other evidence of arteriosclerosis or inflammation was found. Biopsy done on the internal mammary artery revealed no abnormality. Recovery was complicated by a perioperative anteroseptal myocardial infarction, but he then became free of angina and was discharged from hospital receiving no therapy and has remained well subsequently.

**DISCUSSION**

Unequivocal myocardial infarction after superficial chest trauma is rare, and few cases have been fully documented by coronary angiography or post-mortem examination. No report has included 201Tl myocardial scintigraphy.

The RCA occlusion illustrated in this case possibly followed intramyocardial hemorrhage and edema caused by trauma. This mechanism was suggested by extravasation of dye around the occluded portion of the RCA and by unusual beat-to-beat oscillation of contrast medium at the site of occlusion. However, in view of the length of

Figure 3: Eleven months after trauma, repeat right coronary arteriogram in right anterior oblique view shows resolution of previous total occlusion with opacification of normal distal lumen.
REFERENCES


