Case Reports

Coronary Occlusion Secondary to Blunt Chest Trauma

A First Attempt at Balloon Angioplasty

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There have been only 58 angiographically documented reports of transmural myocardial infarction due to closed-chest trauma. None of these cases has been treated by percutaneous transluminal coronary angioplasty. We report the case of a 40-year-old man who developed an anterior-wall myocardial infarction secondary to blunt chest trauma suffered in an automobile accident. Angiographic study performed 2 months after the injury revealed an isolated total obstruction of the left anterior descending coronary artery. The patient was judged a good candidate for balloon angioplasty, but total reocclusion occurred within 24 hours of the procedure and a 2nd attempt did not restore patency. Surgical revascularization was performed a week later. A year after his injury, the patient remains asymptomatic and is back at work. Despite the failure of percutaneous transluminal coronary angioplasty in its 1st application to coronary artery repair after blunt chest trauma, we believe it to be the treatment of choice in young patients and in single-vessel disease. ***(Texas Heart Institute Journal 1992;19:291-3)***

Key words: *Angioplasty, transluminal, percutaneous coronary; coronary artery bypass; coronary vessels/injuries; wounds, nonpenetrating*

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Injury to the heart after nonpenetrating chest trauma has been described rather often, but there have been only 58 angiographically documented reports of transmural myocardial infarction due to closed-chest trauma.[1-9] More common cardiac complications include septal or valvular damage, cardiac rupture, ventricular aneurysm, coronary fistula, cardiac arrhythmias, and the difficult-to-diagnose transmural lesions due to contusion.

Coronary artery damage with acute myocardial infarction is a rare complication of traffic accidents in which the chest has struck the steering wheel[1-9] or in which the whole body has struck the ground, as in motorbike accidents.[10] Severe blunt chest trauma followed by infarction has also been reported as a result of sporting activities,[9,11,12] a suicide attempt,[13] and work accidents.[14]

We report a case of total occlusion of the left anterior descending (LAD) coronary artery and consequent myocardial infarction, discovered upon investigation of severe angina following a motor-vehicle accident.

Case Report

A 40-year-old man was involved in a high-speed automobile accident on 20 October 1991. His chest struck the steering wheel. Physical examination upon admission to a local emergency room revealed a pulse rate of 80 beats per minute and a blood pressure of 120/80 mmHg. All peripheral pulses were present and there was no jugular distention. The patient had smoked 5 to 10 cigarettes per day for the preceding 5 years. He had no history of coronary artery disease, hypertension, diabetes mellitus, or hyperlipidemia. His blood glucose and lipid levels were normal. Neurologic examination was normal. Although the patient complained of a nonspecific chest pain, both his electrocardiogram on admission and a subsequent chest radiograph were normal, so he was discharged.

Three days after leaving the hospital, he felt a severe, prolonged, precordial and left brachial pain associated with nausea and sweating and was admitted to a district general hospital. Electrocardiography showed ST elevation in leads V_1 through V_4 consistent with an anterior-wall myocardial infarction. On the following day, his creatine kinase level was found to be elevated to 900 units per liter,

with a CK-MB fraction of 22%. Thereafter, the patient underwent an uneventful hospital course and was put on a waiting list for angiographic study at our center (the district general hospital had no catheterization laboratory). On the 15th day, echocardiography revealed an area of hypokinesia on the anterior apical wall but no evidence of pericardial effusion. An exercise stress test performed that same day led to angina after 10 minutes and to marked (3-mm deep) ST-segment depression in the anterior leads. A coronary arteriogram, performed 2 months after the injury, at our center in Genoa, revealed total occlusion of the proximal left LAD (Fig. 1A). The right and circumflex coronary arteries were normal. A left ventriculogram showed hypokinesia of the anterolateral segments (on the anterior and apical surfaces); the ejection fraction was 50%. The patient was judged to be a good candidate for a percutaneous transluminal coronary angioplasty (PTCA). The procedure was performed through the left femoral artery. On 1st attempt, we used a 3.5-Fr SciMed Skinny catheter with a 2.5-mm balloon (SciMed Life Systems, Inc.; Maple Grove, Minnesota, USA); dilation was attempted 3 times at 5-minute intervals, with inflation pressures of 4 atm for 30 to 60 seconds. Immediate control angiography showed a residual stenosis of about 20% (Fig. 1B). The next day, however, a control angiogram showed 100% restenosis at the same site. Repeat angioplasty was attempted with a 4.5-Fr Stack perfusion catheter (Advanced Cardiovascular Systems, Inc.; Mountain View, California, USA), chosen for the longer inflation time enabled by such a device. However, even an inflation of 15 minutes' duration, at 4 atm with a 2.5-mm balloon, was wholly unsuccessful at achieving patency.

Therefore, a week later, we performed surgical revascularization. At arteriotomy of the mid LAD, we inserted a probe with ease and moved it proximally to confirm the main stenosis, then distally to exclude unsuspected stenosis. No injury or atherosclerotic plaque was encountered along the vascular wall of the LAD, and no thrombotic material was found. Moreover, palpation revealed no evidence of plaque in the other coronary arteries. We then grafted the left internal thoracic artery to the LAD.

The patient's postoperative course was uneventful. At 5-month follow-up, he was free of angina and had negative electrocardiographic results upon undertaking an exercise stress test at maximum settings. As of October 1992, the patient remains asymptomatic and is back at work.

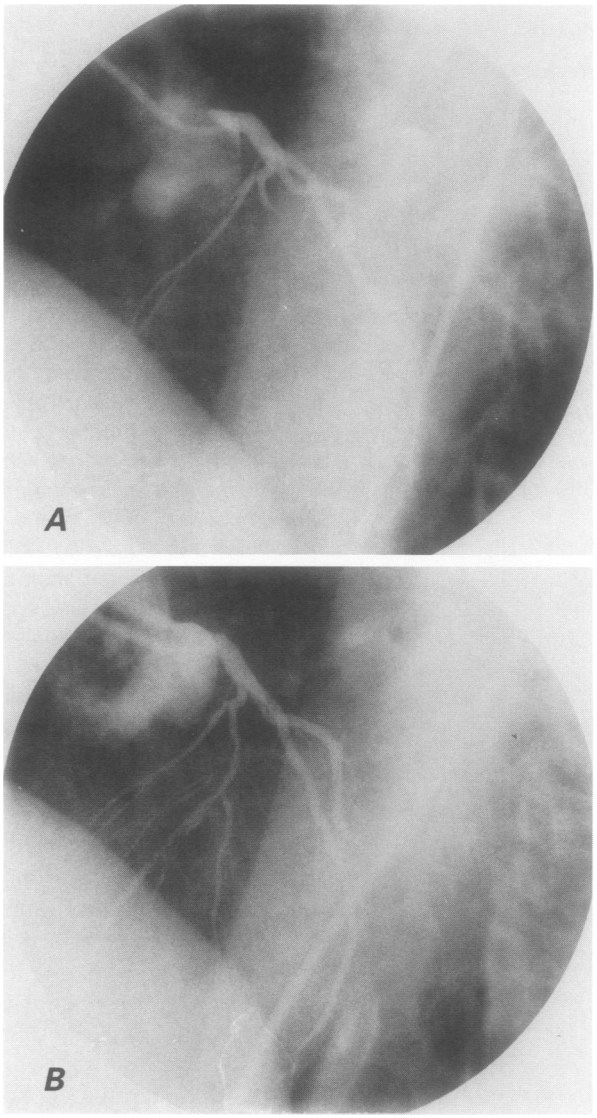

Fig. 1 ***A****) Selective arteriography of the left coronary artery. The totally occluded LAD is seen before PTCA.* ***B****) Immediate control angiography after the 1st attempt at recanalization shows residual obstruction of the LAD at about 20%. Total reocclusion occurred within 24 hours, however, and a 2nd attempt at balloon angioplasty failed.*

LAD = left anterior descending coronary artery;
PTCA = percutaneous transluminal coronary angioplasty

Discussion

The exact mechanism of coronary occlusion after nonpenetrating chest trauma remains an object of speculation: coronary artery dissection, intraluminal thrombosis, subintimal hemorrhage, vascular rupture, coronary embolism, and vascular spasm at the site of injury have been suggested as possible pathophysiologic mechanisms.[5] Probably because of its anterior location, the LAD coronary artery is the vessel most frequently involved, followed by the right and the circumflex coronary arteries.[3,6]

In our judgment, intimal lesion with subsequent thrombotic occlusion should be the main suspect in the pathogenesis of posttraumatic coronary occlusion. In our patient, we suggest a traumatic origin of

the occlusion, because his history as a smoker was his only risk factor, because he had no history of angina, and because we found no signs of atherosclerosis in his coronary arteries. It is logical to assume that a small intimal lesion would have repaired spontaneously 2 months after blunt trauma, so that only the residual stenosis associated with fibrotic stiffening of the vascular wall would have been in evidence upon surgical exploration.

We believe that all patients with closed-chest trauma should have electrocardiography, chest radiography, and a CK-MB laboratory test as part of the initial assessment. If all these suggest myocardial injury, we recommend further assessment by echocardiography and coronary angiography.

Treatment by percutaneous transluminal coronary angioplasty in this situation has not been described before. We attribute the failure of PTCA to vessel-wall stiffness, probably caused by the trauma.

Recommendations

In cases of blunt chest trauma, we recommend cardiologic assessment (electrocardiography, CK-MB laboratory test, and chest radiography) whenever there is a high index of suspicion of cardiac damage.

Management of such patients has to be individualized. Definitive therapy, of course, depends on the angiographic findings. Despite its failure here, we believe that the 1st line of treatment should be PTCA, especially in young patients and in single-vessel disease. Because we consider the stent to be an emergency tool for bridging to surgical revascularization, rather than a long-lasting treatment, we consider surgery preferable to stent implantation when reocclusion occurs after PTCA.

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