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Spontaneous Plaque Rupture and Thrombus Formation in the Left Main Coronary Artery Documented by Intravascular Ultrasound

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Key words: thrombus; plaque rupture; intravascular ultrasound

INTRODUCTION

The development of acute coronary syndromes is thought to be the result of spontaneous rupture of the fibrous capsule with release of thrombogenic components from the lipid core of the plaque [1]. Evidence for this hypothesis has come from: 1) angiography during acute infarction, which demonstrates a meniscus consistent with thrombus [2]; 2) pathologic specimens obtained at autopsy [3]; and 3) angioscopy during unstable angina or acute myocardial infarctions [4]. Although our attention is drawn to acute clinical syndromes, it has been hypothesized that spontaneous rupture with thrombus formation and resolution occurs at a much more frequent rate and is unrecognized because it usually does not proceed to a clinical event [5]. Recently performed intravascular ultrasound (IVUS) imaging in a patient provides evidence for this hypothesis.

CASE REPORT

A 48-year-old man presented with substernal chest pain over the previous 24 hr. The patient had elevated CK enzymes (482 IU/l) without Q-waves or ST segment elevation on ECG. During diagnostic catheterization, an occluded circumflex artery was observed. In addition, there was a 40% lesion in the left anterior descending (LAD) artery and a questionable lesion in the left main coronary artery observed in only one projection (Fig. 1). Prior to performing angioplasty on the circumflex artery, a decision was made to evaluate both the LAD and left main lesion by IVUS. The IVUS examination of the left main artery (Fig. 2) revealed a 9.4 mm² atherosclerotic plaque consisting of a central echogenic lucency and an echo-reflective fibrous capsule. The plaque encompassed only 39% of the vessel cross-sectional area (defined as the area of the external elastic membrane). There was a lucent line in the capsule, consistent with a tear. In addition, there was a mobile echo-reflective mass extending from the plaque into the lumen which was compatible with a thrombus (Fig. 3). Due to this observation of a spontaneous thrombus in the left main plaque, the patient was referred for CABG surgery rather than proceeding with any further percutaneous intervention.

DISCUSSION

Plaque rupture is now considered to be the major cause of acute coronary syndromes [5]. Evidence for this hypothesis comes from 1) angiography, which has demonstrated a high incidence of thrombosis during acute myocardial infarction [6]; 2) pathology, which has demonstrated intraluminal thrombus extending from a torn fibrous capsule, suggesting that the thrombogenic lipid core has mixed with blood, initiating thrombosis [3]; and 3) from angioscopy, which has demonstrated a high incidence of intraluminal thrombus in patients presenting with either unstable angina or myocardial infarction [7]. This theory is distinctly different from an earlier hypothesis that thrombosis found in pathologic specimens was secondary to stagnation from the occlusive disease and not the primary cause of the clinical syndrome [8]. Fuster [1] advanced the theory that plaques evolve by an

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intermittent process of spontaneous rupture, thrombosis, and resorption. An occlusive thrombus is thought to be a less frequent event, although it is this occurrence that brings the patient to clinical notice. We believe that this case report supports this latter theory. In our patient, intravascular ultrasound imaging demonstrated the presence of spontaneous rupture of a nonhemodynamically significant plaque in the left main coronary artery. This plaque was shown by ultrasound to be associated with an intraluminal thrombus that was not observed by angiography. These observations suggest that other cases of spontaneous thrombosis may also be clinically or angiographically silent.

Angiography is a form of projection imaging that may mask intraluminal pathology which may be revealed by techniques that image from within the artery. In addition,
intravascular ultrasound has the capacity to visualize the plaque in cross section and can discern tissue morphology as well as plaque components such as calcium, fibrous tissue, or lipid-rich areas [9]. Angioscopy has also demonstrated the presence of thrombosis, even in patients with stable angina [4]. Our case is unusual in that the lumen was not compromised and the size of the plaque was consistent with early compensatory enlargement and a normal-appearing angiogram. The occlusion in the circumflex was the cause of the patient’s chest pain and clinical presentation. It cannot be determined from these observations whether the circumflex occlusion was also due to a spontaneous rupture of plaque, or whether there was an embolus from the thrombus in the left main artery.

REFERENCES