

Intracoronary ultrasound and angioscopic imaging facilitating the understanding and treatment of post-infarction angina

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We report on the use of intravascular ultrasound, coronary angioscopy and on-line quantitative angiography in an unstable patient soon after myocardial infarction. Combined intracoronary imaging made it possible to solve the therapeutic problem posed by an unusual angiographic appearance secondary to intracoronary thrombolysis during coronary recanalization. The pathological validation of the observations performed with angioscopy and intravascular ultrasound was made possible with the concomitant use of directional atherectomy.

Introduction

We present the case of a patient with post-infarction angina where the combined use of on-line quantitative analysis, intravascular ultrasound and angioscopic imaging greatly facilitated the therapeutic strategy and understanding of the pathological substrate of a patient with an unusual angiographic morphology after intracoronary thrombolysis.

Case report

A 49-year-old white Caucasian male with post-infarction angina pectoris was referred to our Institution for percutaneous coronary angioplasty. The patient had been admitted 9 days before to a different hospital with chest pain at rest lasting for more than 48 h. An ECG disclosed sinus rhythm with Q waves in leads II, III and aVF. Physical examination, chest X-ray, blood biochemistry (CPK 120 U.l^{-1}) and haematology were normal. Blood pressure was 135/70 mmHg. Coronary risk factors included smoking and a family history of coronary artery disease. After admission he had intermittent episodes of chest pain at rest. Intravenous nitroglycerine and heparin were started, and beta-blockers, calcium antagonists and aspirin were also given. Coronary angiography performed at the referring hospital revealed a severe eccentric stenosis with smooth borders in the mid right coronary artery which was identified as the culprit lesion. Left ventricular angiography disclosed an ejection fraction of 52% and an area of posterobasal hypokinesis.

On the following day the patient was transferred to our Institution for percutaneous coronary intervention. Repeat angiography of the right coronary disclosed a new overhanging, moving filling defect originating from

one of the borders of the stenosis (Fig. 1(a)). Some minor irregularities were noted in the proximal segment of the vessel, which were judged to correspond to a separate, non-significant stenosis. Quantitative angiography was performed on-line from the digital angiogram (ACA, Phillips DCI, Eindhoven, The Netherlands). Stenosis severity in the mid segment was 76%, obstruction diameter was 0.84 mm and the interpolated reference diameter was 3.55 mm. Interpolation of the reference dimensions showed inverted tapering of the vessel, which was thought to indicate the existence of a post-stenotic dilatation (Fig. 2(a)). The moving defect was identified as the tail of a thrombus and intracoronary thrombolysis was performed with rTPA (50 mg over 30 min). Repeat angiography 30 min later revealed the disappearance of this overhanging filling defect and an obstruction diameter of 0.82 mm, but was followed by the appearance of a new large contrast opacification protruding outside the luminal borders identified in previous views (Fig. 1(b)). The nature of this new opacification could not be ascertained, but the possibilities considered included major intraplaque bleeding, and extravasation of contrast medium or vessel dissection, all of them posing a serious problem to continuing thrombolytic treatment. In an attempt to obtain complementary information on the changes taking place within the arterial wall, two-dimensional ultrasound imaging of the proximal and stenotic segment was performed with a 4.3 Fr 30 MHz intracoronary probe (Cardiovascular Imaging Systems, Inc., CA). Proximal to the stenosis the vessel showed a well-defined ultrasonic three-layers appearance (Fig. 3).

The area comprised within the ultrasound-defined internal elastic lamina was 25.5 mm^2 , much larger than expected from the angiographically derived reference area (9.92 mm^2). Only 7.9 of the total 25.5 mm^2 (31%) corresponded to vessel lumen, the remaining corresponded to occluding material presenting low echodensity and speckling. Proximal to the stenosis, several luminal channels, that were best visualized during the

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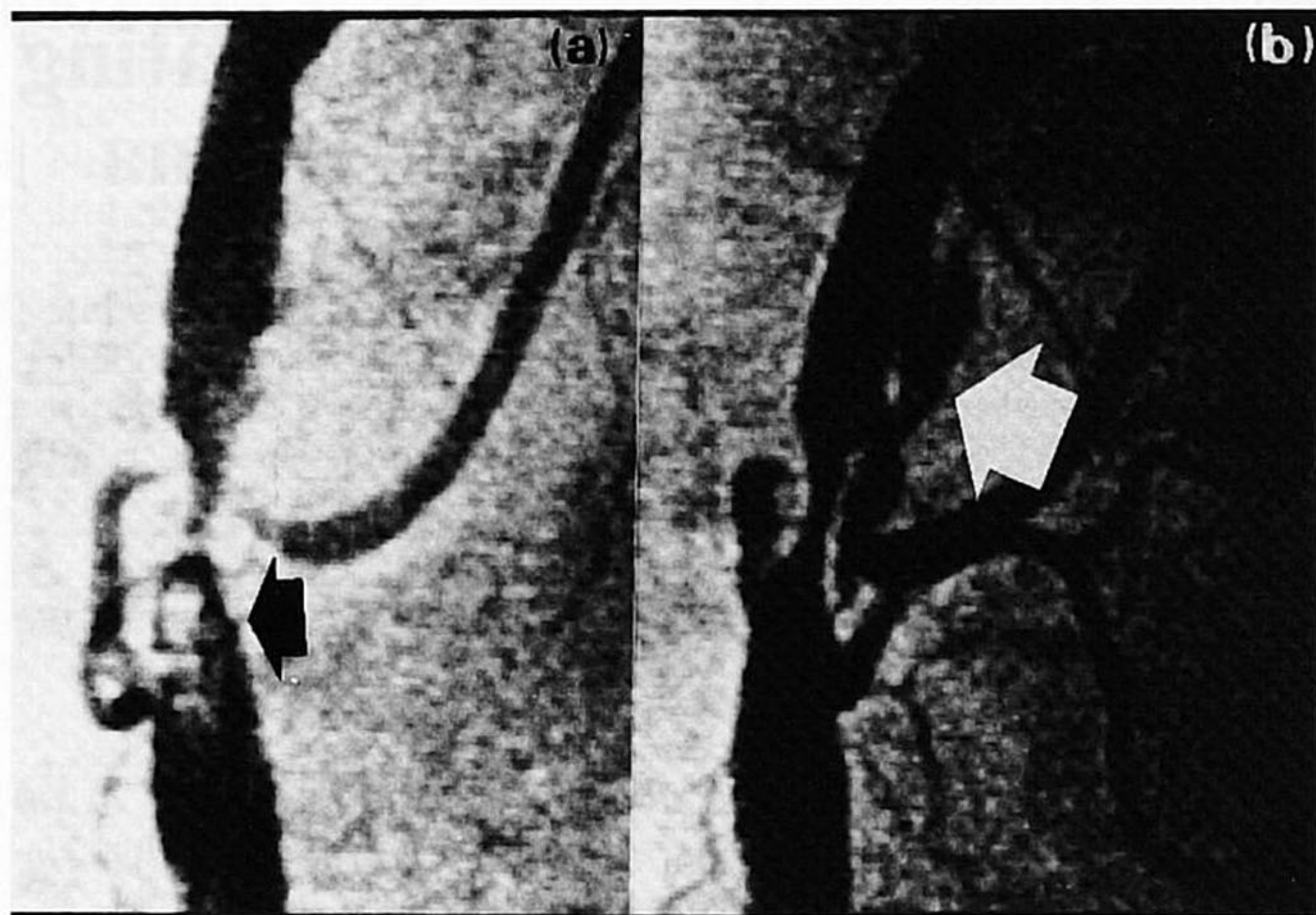


Figure 1 (a) Coronary angiography showing an overhanging, mobile filling defect (black arrow) distal to the mid right coronary artery stenosis, which was identified as the tail of a thrombus. Minor irregularities in the proximal segment were also noted, but thought to correspond to a separate non-significant stenosis. (b) Repeat angiography 30 min after intracoronary thrombolysis disclosed complete disappearance of the filling defect. However, a new large contrast opacification protruding out of the original luminal borders (white arrow) became evident.

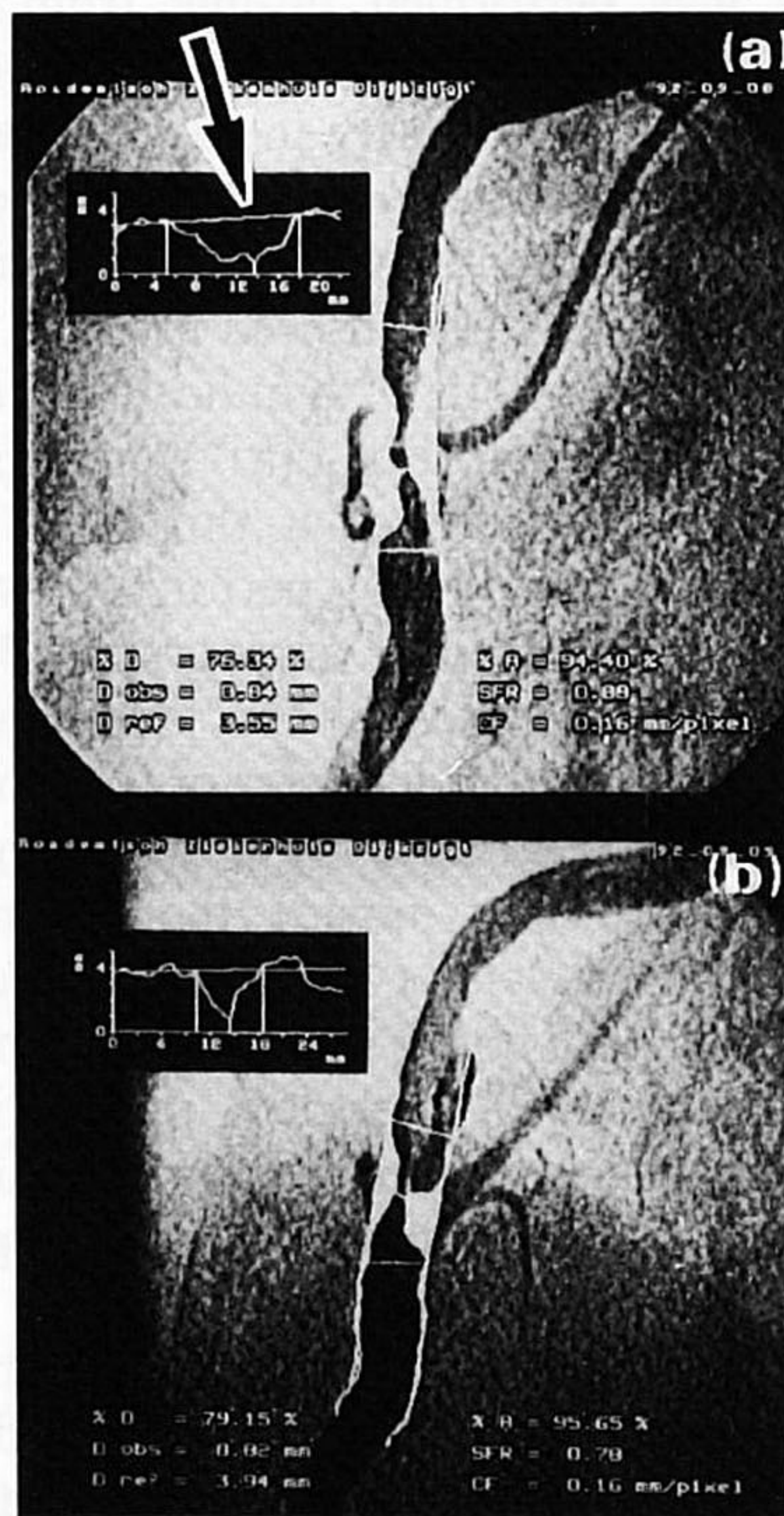


Figure 2 (a) On-line quantitative angiographic analysis performed prior to thrombolysis. An inverted tapering of the vessel was disclosed in the interpolated vessel diameter function (arrow). (b) Quantitative analysis after thrombolysis. Although there was no substantial change in obstruction diameter, significant remodelling of the plaque was evident (white plaque area), with normalization of the inverted vessel tapering found the day before.

injection of saline, were seen (Fig. 3). The penetration of contrast medium in these newly formed channels or lacunar cavities was identified as the cause for the protruding opacification. Based on this information, a

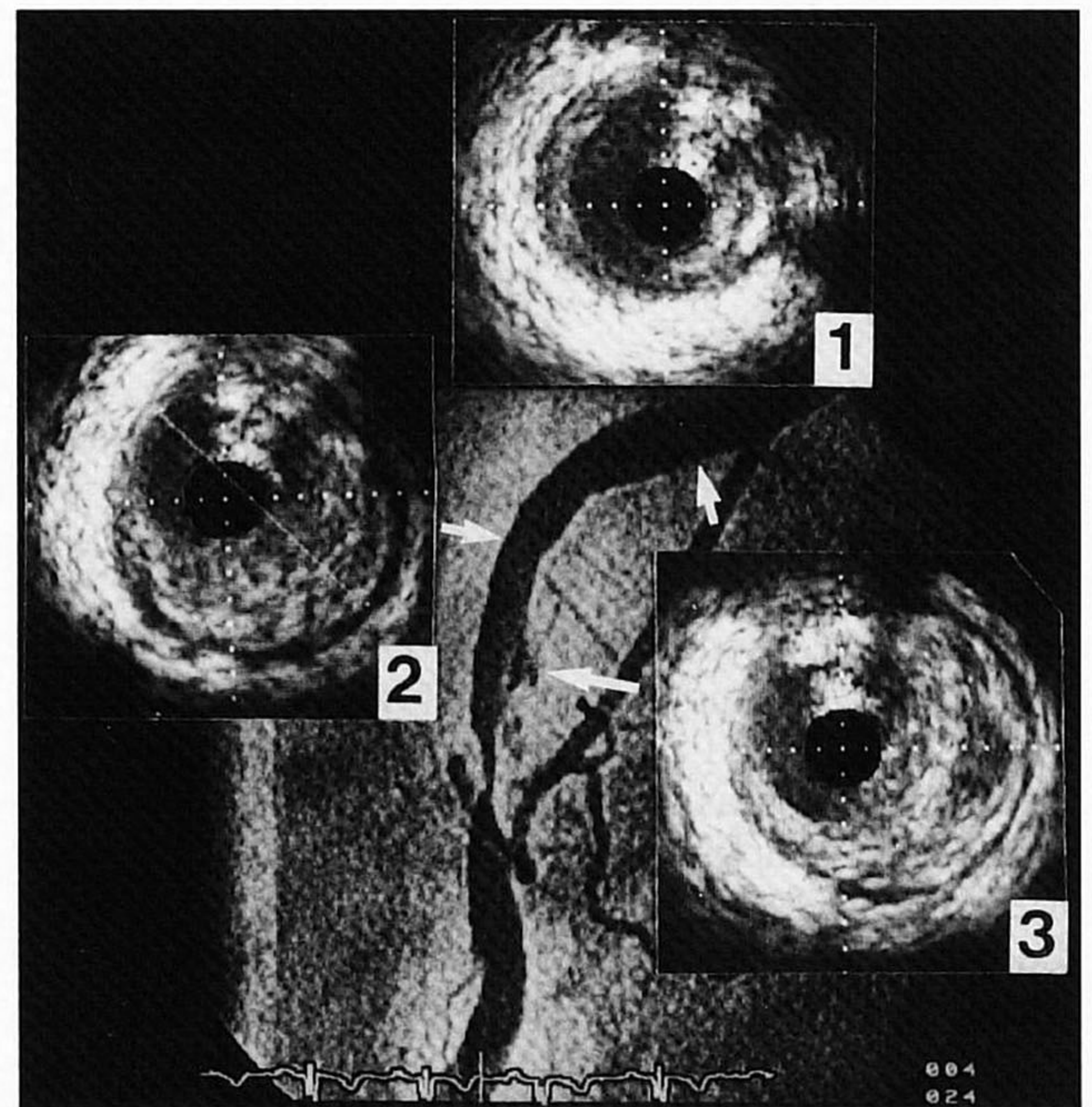


Figure 3 Intravascular ultrasound imaging immediately after intracoronary thrombolysis. In the proximal segment of the vessel, only minimal atheroma was disclosed following a concentric distribution (1). However, further advance of the probe revealed a larger vessel than expected from coronary angiography, occluded by homogeneous material of moderate echogenicity with a concentric lumen in the eccentric position (2). At the level of the protruding angiographic opacification (3), the occluding material showed areas of low echogenicity suggestive of newly formed vascular channels or lacunar spaces.

large mass of remaining thrombus was judged to be the underlying substrate of the images, in spite of the fact that intravascular ultrasound has a low sensitivity

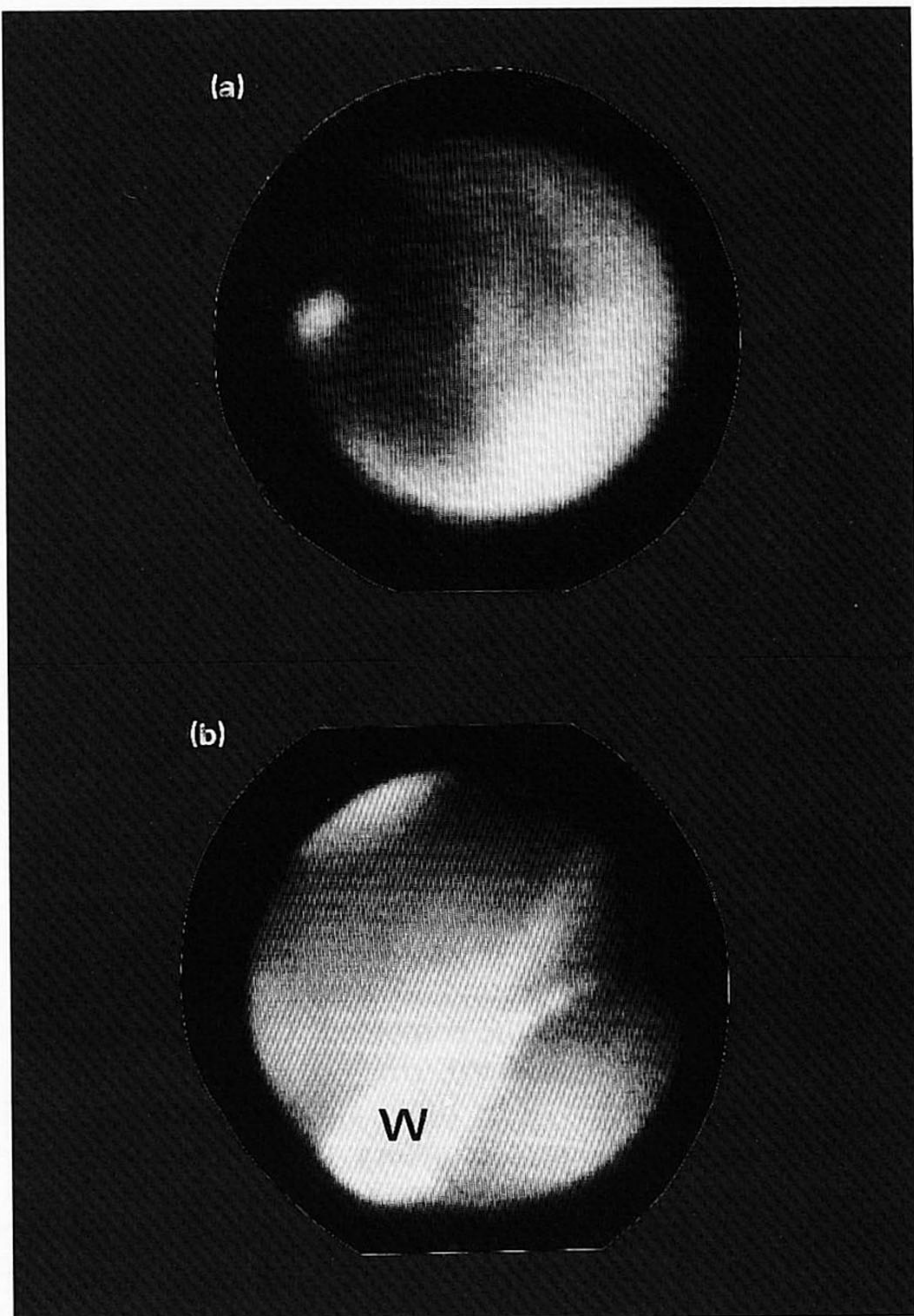


Figure 4 (a and b) Coronary angioscopy showing red material with large disruption of the vessel wall. The true vascular lumen could be identified by the position of the guidewire (W) used to cross the lesion.

in detecting thrombotic material^[1]. Since any contra-indication for thrombolysis was ruled out, an intravenous infusion of streptokinase (1 500 000 units) was started and continued overnight.

The patient was reinvestigated on the following day. Repeat quantitative angiography revealed significant remodelling of the stenosis, with marked disappearance of the plaque bulk and considerable expansion of the extraluminal opacification. The obstruction diameter remained virtually unchanged (0.82 mm). The reference diameter was 3.94 mm (0.49 mm increase from the previous day). Marked attenuation of the inverted vessel tapering, demonstrated during quantitative analysis the day before, was evident (Fig. 2(b)). Coronary angioscopy (Baxter Edwards, Irvine, CA) was performed to discriminate between the components of the stenosis on the grounds of their chromatic characteristics. This angioscopy system includes a proximal compliant cuff to abolish antegrade blood flow in order to facilitate visualization of the lumen, while Ringer's lactate is flushed. A mobile optical bundle with 5 cm capacity is manipulated to obtain images over the coronary segment of interest. The luminal surface showed a very

irregular morphology, with wall disruption, intraluminal flaps and large areas of red material extending from the proximal right coronary segment to the site of the stenosis (Fig. 4), confirming that the occluding material identified by intravascular ultrasound was thrombus. Vessel disruption and areas of yellowish material were also seen. Directional atherectomy was performed using a 7 Fr atherocatheter (DVI, San Diego, CA). Three passages were done at the stenosis site. The retrieved material showed areas of dense fibrous tissue and thrombus, the latter containing cholesterol crystal clefts and necrotic debris (Fig. 5).

The immediate result of atherectomy was good as judged from the images obtained with the 11 Fr atherectomy catheter, and the atherectomy system, including the guidewire, was removed. However, repeat angiography performed with a conventional 7 Fr diagnostic coronary catheter revealed a major ostial dissection of the right coronary artery which rapidly led to acute vessel occlusion. The dissection could not be recrossed with the guiding wire and the procedure was abandoned. No emergency surgery was performed. The patient developed transient 1 mm ST elevation in leads II, III

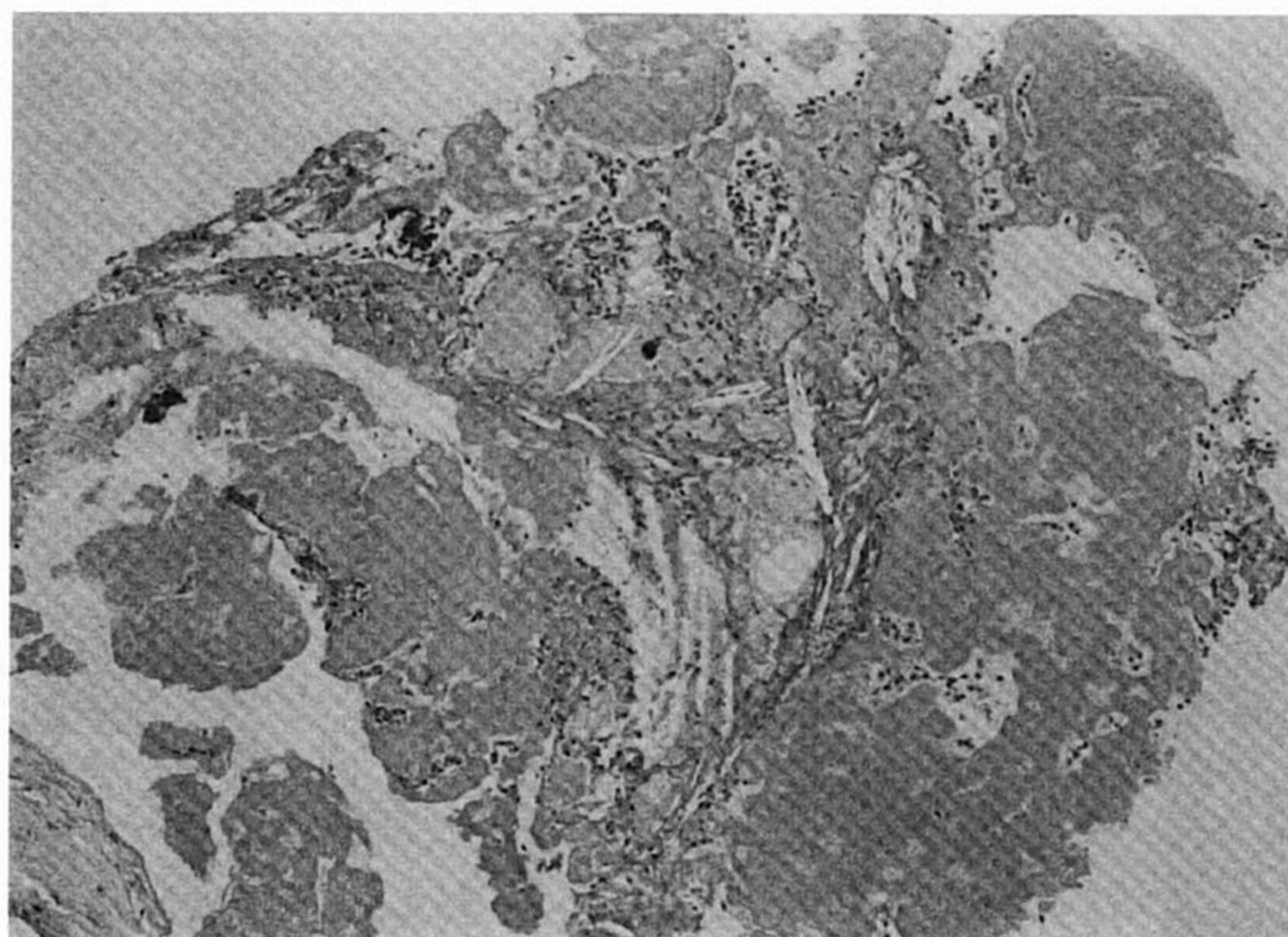


Figure 5 Atherectomy specimen obtained from the lesion showing fresh thrombotic material in association with areas of cholesterol crystal clefts and inflammatory cells (centre of the picture).

and aVF and ST depression in the anteroseptal leads without concomitant haemodynamic embarrassment. The CPK rose to $492 \text{ U} \cdot \text{l}^{-1}$. After this episode of reinfarction the patient followed an uncomplicated evolution and was discharged 8 days later.

Discussion

It is well known that the complication rate of coronary angioplasty in unstable patients is higher than in stable patients, particularly in the presence of coronary thrombus. In those cases, intracoronary thrombolysis prior to coronary intervention was used in an attempt to decrease the procedural risk^[2]. However, the induction of a lytic state may facilitate the extension of plaque fissures to deep layers of the vessel wall, leading to the formation of an intramural or extravascular haematoma and causing subsequent vessel dissection or lumen compression^[3]. This was suspected from the angiographic image shown in Fig. 1(b). The additional information obtained with intravascular ultrasound imaging excluded such potential complications and suggested continued thrombolytic treatment for the dissolution of an otherwise unsuspected large thrombotic mass. The criteria used for ultrasonic characterization of the occluding material were later reinforced by angioscopic examination.

Other observations made in this case illustrate several interesting aspects of the pathophysiology of myocardial infarction and post-infarction angina. Plaque rupture and exposure of plaque material to the bloodstream has been suggested as a common initial event of acute coronary syndromes^[4]. During plaque rupture, embolization of atheromatous material has been shown to occur^[4]. This is consistent in the present case with the histopathological findings in the atherectomy-retrieved tissue which showed cholesterol crystal clefts isolated from plaque material and embedded in the retrieved

thrombus. The fresher tail of the thrombus, that had developed at the narrowest stenotic point in the interval from the diagnostic angiogram and was first lysed during the administration of intracoronary thrombolytics, was probably the combined result of preexisting thrombus and high shear stress at that point of the vessel^[5]. As observed by other authors, remodelling of stenosis morphology secondary to the lysis of older thrombus was observed during angiography after sustained thrombolysis and anticoagulation^[6]. However, the present case illustrates how visual angiographic assessment may underscore the degree of remnant coronary thrombosis. The concomitant use of on-line computerized angiographic analysis can improve the sensitivity of coronary angiography and offer additional data that otherwise might be missed by the observer, such as changes in tapering characteristics of the vessel or modifications in luminal dimensions secondary to thrombolysis (Fig. 2).

Angioscopic and intravascular ultrasound findings have been previously validated *in vitro*^[1], suggesting that the information obtained with both methods can be complementary in the study of coronary artery disease. The present case shows how different imaging techniques can contribute to the solution of a specific problem found during coronary intervention, and how tissue retrieved by directional atherectomy can facilitate the validation *in-vivo* of such observations. Although we feel that the unfortunate ostial dissection that complicated this case was related to the withdrawal of the atherectomy catheter, the possibility that this could be related to the use of concomitant intracoronary imaging cannot be ruled out. Should further studies demonstrate the safety of intracoronary ultrasound and angioscopic imaging, the harmonious integration of data obtained by these two techniques may contribute to the understanding of the anatomopathological substrate of coronary syndromes, and subsequently influence the therapeutic management.

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