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Detection of a coronary arterial thrombus by indium-111-oxine-labeled platelet scintigraphy

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Coronary arteriography revealed significant left anterior descending coronary artery stenosis in a 72-year-old man with a history of myocardial infarction. Stenting of the stenotic vessel was performed. Twelve hours after stenting the patient complained of chest pain but emergent coronary arteriography did not show sign of any coronary arterial stenosis. Under suspicion of coronary thrombus formation, indium-111-oxine-labeled platelet scintigraphy was performed 5 days after stenting, and revealed accumulation of indium-111-oxine in the area corresponding to the stent implantation site.

Key words: platelet scintigraphy, stent, coronary heart disease

INTRODUCTION

Post-implantation thrombus is a grave complication of coronary arterial stent implantation. The incidence of subacute thrombosis during the first 2 weeks after stenting is relatively low, but serious early thrombus-related complications have been reported. When the vascular endothelium is damaged during stent implantation, a thrombus may form subacutely, and hyper-activation of plateletmediated vascular repair may lead to restenosis.² Indium-111-oxine-labeled platelet scintigraphy is useful for localizing and quantifying thrombotic activity.³ Although identification and localization of coronary arterial thrombi in swine has been reported previously, 4 no studies in humans have been published to date. In the present report, we describe the local accumulation of indium-111-oxine in the coronary artery after coronary arterial stent implantation.

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CASE REPORT

A 72-year-old male patient with a history of myocardial infarction was admitted to our hospital for evaluation for coronary insufficiency. Electrocardiography showed abnormal O waves in leads V₁₋₄. Echocardiography demonstrated reduced motion of the anterior wall of the left ventricle. Coronary arteriography revealed significant stenosis in the proximal left anterior descending artery, and a Wiktor stent was placed successfully on the target lesion (Fig. 1). Antiplatelet therapy had been started prior to the arteriography. About 12 hours after stent placement, the patient complained of chest pain, and electrocardiography demonstrated pseudonormalization of T waves in leads V1 through V4. Under suspicion of acute closure, emergent coronary arteriography was performed but neither restenosis nor thrombus was observed. Five days after stent placement, indium-111-oxine-labeled platelet scintigraphy was conducted by labeling the patient's platelets with indium-111-oxine as previously described,3 and indium-111-oxine accumulation was observed in the area corresponding to the site of stent placement (Figs. 2, 3).

DISCUSSION

Radioscintigraphy has been used to visualize thrombi in

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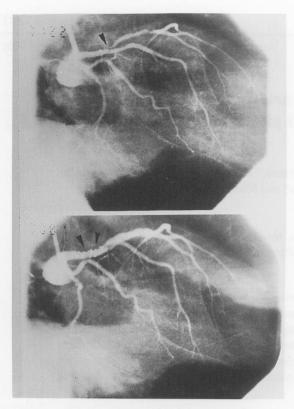


Fig. 1 Coronary angiography. The top panel shows the coronary stenosis. The bottom panel shows the coronary artery after intracoronary stent implantation.



Fig. 2 Whole-body scintigraphy with indium-111-oxine-labeled platelets. The left panel shows the anterior view, which reveals accumulation at the stent site. The right panel shows the posterior view.

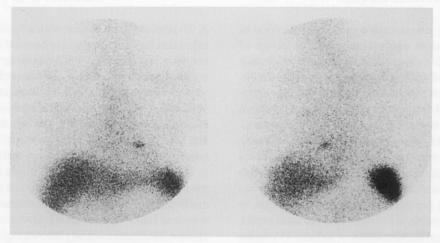


Fig. 3 Scintigraphic images in the anterior (left) and left oblique views (right), following the injection of indium-111-oxine-labeled platelets. The area of accumulation corresponds to the stent site.

the large pelvic arteries and in aortic aneurysms⁵ but no successful visualization of a coronary thrombus in humans has been reported. Our patient accumulated indium-111-oxine in an area that corresponded to the coronary artery that had been stented, although neither acute restenosis nor thrombus formation had been observed by angiography.

The stent used in this case was a Wiktor stent, and its location could be verified by plain chest roentgenography. The site in four different projections correlated with the accumulation site indicated in three different projection on scintigrams. This correlation was the basis for our conclusion that the indium accumulation occurred at the site of the stent, even though coronary arteriography did

not identify a thrombus unequivocally. Sakatani et al. observed a thin film-like thrombus along the inner edge of the stent by vascular endoscopy, which was not demonstrated by coronary arteriography. In addition, Goto et al. found that a thrombus may develop on the outer edge of the stent in an experimental model. These date support the likelihood that a thrombus can develop which is not visualized readily by angiography. We also performed indium scintigraphy 5 days after the occurrence of chest pain. Active platelet aggregation if the thrombus was located on the outside of the stent.

Restenosis most commonly occurs a few months after coronary stenting and is due to the proliferation of smooth muscle cells rather than thrombosis. Thus antiplatelet therapy is ineffective prophylaxis against restenosis.8 Platelet accumulation after coronary stenting is caused by vascular endothelial disruption.⁴ Injury to the vascular endothelium sufficient to induce thrombus formation increases the risk of acute restenosis.² A relationship between thrombus formation at the site of stenting and coronary arterial restenosis has been reported based on gross morphology⁹ and histologic analysis.¹⁰ The present study used noninvasive means to document thrombus formation. The significance of indium-111-oxine-labeled platelet accumulation in terms of developing restenosis requires further study but we believe that platelet accumulation has predictive value for identifying patients at risk of restenosis because platelet activation indicates that significant endothelial damage occurred during stent implantion. In fact, our patient did develop restenosis in the chronic stage. A large number of cases must be studied before indium accumulation can be definitely linked with restenosis.

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Vol. 15, No. 1, 2001 Case Report 51