INTRODUCTION

Rotational atherectomy is a widely used modality in percutaneous coronary revascularization, especially for heavily calcified coronary arteries [1–5]. Due to the unique mechanism of rotational atherectomy, there have been reports of unusual complications, including fracture of coronary wire, guidewire bias, coronary spasm leading to myocardial infarction, and fracture of the drive shaft causing acute ischemia [6–9]. We provide the first case reported of entrapment of an atherectomy burr in the left anterior descending artery (LAD) and a technique for its removal.

CASE REPORT

An 83-year-old male with a history of atrial flutter and congestive heart failure underwent an adenosine Cardiolite scan, which revealed a medium to large area of mild reversible ischemia in the septal/anteroseptal and apical territories. He subsequently underwent coronary angiography, which revealed a heavily calcified, completely occluded proximal LAD (Fig. 1), a 60%–70% stenosis of the proximal left circumflex, and significant disease in the distal dominant right coronary artery (RCA). The mid- to distal LAD filled via collateral flow from the distal RCA. We were asked to attempt percutaneous revascularization of his LAD.

A 7 Fr guiding catheter was chosen and placed into the ostium of the left main artery. Several guidewires were used in an attempt to cross the occlusion; eventually it was crossed with a stiff Cross-It 300 (Guidant, Temecula, CA) guidewire (Fig. 2). Numerous balloon catheters would not cross the lesion. We then decided to perform rotational atherectomy. A rotational atherectomy guidewire was advanced alongside our existing guidewire into the distal LAD. We then removed the original Cross-It wire. A 1.25 mm diameter rotational atherectomy burr was then advanced to the lesion and atherectomy was performed at 150,000 rpm, with passage through the calcified area into the mid-LAD (Fig. 3). We then attempted to retract the burr from the LAD; however, we were unable to retrieve the device. It would freely advance distally, but would not come back through the original calcified lesion (Fig. 4). Intracoronary nitroglycerin did not resolve the problem.

The guiding catheter was then withdrawn approximately 1 cm from the ostium of the left main, leaving the rotoblator burr and wire across the lesion. A second guiding catheter was then placed into the left main artery via the left femoral approach. We then used a 2 mm diameter Ace Balloon (Scimed, Maple Grove, MN: a premounted balloon on a 0.014” guidewire) and passed this into the LAD. We advanced the guidewire distally into a septal branch just beyond the original calcified lesion but proximal to the entrapped rotoblator burr. Balloon dilatation was then performed at the site of the calcified area, with near-full expansion occurring at 8 atm of pressure (Fig. 5). After the dilatation, the burr was easily removed. Angiography revealed flow into the distal LAD without evidence of coronary dissection or perforation (Fig. 6). A very tight, calcified lesion distal to the area of entrapment remained, which had not been treated. Therefore, it was determined that the patient would ultimately be better served with surgical revascularization and he underwent bypass with three conduits (LIMA to the LAD, saphenous vein graft to the posterior descending artery, saphenous vein graft to the circum-

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Received 18 February 2002; Revision accepted 6 March 2002
DOI 10.1002/ccd.10263
Published online in Wiley InterScience (www.interscience.wiley.com).
flex). The patient had an uneventful postoperative course and was discharged on postoperative day 4.

**DISCUSSION**

Rotational atherectomy continues to be a widely used modality in interventional cardiology, especially in calcified coronary arteries [1–5]. As has been discussed, this technique has associated risks, some unique to the device [6–14]. The objective of this case report is to present a unique complication of rotational atherectomy entrapment and outline a strategy for burr removal.

We surmised that the small burr tip was able to pass through the calcified area without removal of a significant amount of calcified tissue. The burr could not be
retrieved back through a presumed ledge of calcium that had opened wide enough to allow forward passage of the burr but incomplete ablation prevented burr withdrawal. It did not appear that there was a device failure as the burr passed easily through calcified lesion and it easily advanced distally. Also, inspection of the device upon removal did not reveal any malfunction. The entrapment also did not appear to be due to spasm as nitroglycerin did not alleviate the problem.

Once we were able to create enough of a channel in the calcified area, the device was easily removed. Perhaps if a larger-diameter burr was utilized, it may have resulted in more tissue being removed during the initial pass.

REFERENCES