Left main stem coronary disease: the case for percutaneous coronary intervention in a high risk patient with complex disease

K Kosuga, H Tamai

Although coronary balloon angioplasty is an established treatment for patients with coronary artery disease, the results of balloon angioplasty of unprotected left main coronary artery (LMCA) stenoses have been disappointing because of the high mortality rate in both in-hospital (9.1%) and follow up periods. Current in-hospital mortality after initial coronary artery bypass graft surgery (CABG) in unprotected LMCA without myocardial infarction (MI) is 2.3% in highly experienced centres. Therefore, the procedure has been controversial, and CABG has been considered the only effective treatment for this lesion. One reason for high in-hospital mortality in balloon angioplasty for unprotected LMCA was haemodynamic deterioration during balloon inflation that would lead to cardiogenic shock and/or fatal arrhythmia. Another reason was acute occlusion caused by recoil or dissection of the LMCA or left coronary arteries that would lead to fatal MI. The reason for high mortality at long term follow up derived from restenosis occurred mainly within six months after the procedure. In order to improve the results of percutaneous coronary intervention (PCI) in unprotected LMCA, other devices and procedures were mandatory instead of balloon angioplasty.

Recently, directional coronary atherectomy (DCA) and coronary stenting have gradually been applied to this lesion. They can protect against acute occlusion in LMCA both during and after PCI, and attain larger post-minimal lumen diameter that can improve the late restenosis rate. Some studies have shown the feasibility, safety, and efficacy of PCI with these new devices in this lesion. Especially in CABG low risk patients, four institutions showed 0% of in-hospital mortality, results that were comparable with those in highly experienced centres. Therefore, the procedure has been controversial, and CABG has been considered the only effective treatment for this lesion. One reason for high in-hospital mortality in balloon angioplasty for unprotected LMCA was haemodynamic deterioration during balloon inflation that would lead to cardiogenic shock and/or fatal arrhythmia. Another reason was acute occlusion caused by recoil or dissection of the LMCA or left coronary arteries that would lead to fatal MI. The reason for high mortality at long term follow up derived from restenosis occurred mainly within six months after the procedure. In order to improve the results of percutaneous coronary intervention (PCI) in unprotected LMCA, other devices and procedures were mandatory instead of balloon angioplasty.

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Drug eluting stents (DES), such as the sirolimus eluting stent (Cypher, Jonson & Jonson-Cordis, Miami, Florida, USA), have recently been reported to be effective in reducing restenosis rates in selected patients. Moreover, this stent has been increasingly used in PCI for unprotected LMCA with promising results, especially the low restenosis rate. However, before we apply this type of stent to all LMCA stenoses in the “real world” intervention, we must reconsider risks involved in undertaking PCI for this lesion.

CASE REPORT
A 72 year old woman was admitted as an emergency to our hospital in February 2003 with congestive heart failure. Although the patient had no coronary risk factors, she had suffered an MI in December 1996. At that time, she underwent emergency balloon angioplasty for the mid portion of left anterior descending coronary artery (LAD) with successful recanalisation of the occluded lesion. She also underwent balloon angioplasty in both the left circumflex coronary artery (LCx) and the right coronary artery (RCA) a month later. As contrast angiography showed a pseudoaneurysm of left ventricle at the cardiac apex, she underwent surgical resection and reconstruction with a patch. She had been asymptomatic for six years following this treatment until her admission.

In the cardiac care unit, a Swan-Gantz catheter was inserted via the internal carotid vein. The haemodynamic data showed Forrester subset 4. Although oxygenation and medication could improve the symptoms, her haemodynamic state could not be fully restored. Moreover, cardiac enzyme, electrocardiography, and echocardiography showed the possibility of recent MI in the lateral wall of the left ventricle. Left ventriculography showed asynergy in the anteroseptal and anterolateral walls with decreased ejection fraction (20%) on day 4 (fig 1). Coronary angiography (CAG) showed diffuse narrowing of the LMCA with 90% stenosis in the distal bifurcation (fig 2). Chronic total occlusion was found in the proximal LAD just below the ostium of relatively large sized first diagonal branch with 99% stenosis in its middle portion. Collateral formation from other vessels to the LAD was poor. Moreover, there was 90% stenosis in the ostium of the LCx. The RCA had 50% stenosis in the proximal site and 90% stenosis in the posterior descending branch. The culprit lesion for the recent MI was considered to be the diagonal branch, and some invasive treatment was needed to improve the congestive heart failure.

The patient was considered to be at high risk of CABG because of the history of open heart surgery, decreased left ventricular function, and the presence of active congestive heart failure. Therefore, after informed consent was obtained, we planned to undertake PCI for the left main, LCx, and diagonal branch. There were difficulties in performing PCI for her left main disease. The lesion was diffuse, moderately calcified, and located in the distal bifurcated LMCA involving relatively large three vessels; LCx, high lateral branch, and LAD diagonal branch.

After standard preparation and local anaesthesia, an 8 French sheath was positioned into the femoral artery. Intravenous heparin was given to achieve an activated clotting time > 300 seconds. Anatomical lesion characteristics

Abbreviations: CABG, coronary artery bypass graft surgery; CAG, coronary angiography; DCA, directional coronary atherectomy; DES, drug eluting stents; IVUS, intravascular ultrasound; LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; LMCA, left main coronary artery; MI, myocardial infarction; MLD, minimal lumen diameter; PCI, percutaneous coronary intervention; QCA, quantitative coronary angiography; RCA, right coronary artery

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were carefully evaluated in a view appropriate to visualise the distal bifurcation and the extension of the atheromatous lesion over the ostium of the LCx and/or LAD diagonal branch. The lesion length was 11.2 mm by quantitative coronary angiography (QCA). Reference vessel size was small, 2.33 mm, and minimal lumen diameter (MLD) was 0.61 mm. Intra-aortic balloon pumping and temporary pacing were employed before PCI to maintain blood pressure and heart rate during the procedure.

Baseline intravascular ultrasound (IVUS) was performed using a commercially available system (Boston Scientific Corporation/Cardiovascular Imaging System Inc, San Jose, California, USA). As the stenosis in the LMCA was too tight to pass the IVUS device through, balloon pre-dilatation was performed using a 2.0 mm balloon at 8 atm after dilatation of the mid portion of diagonal branch. The imaging catheter was advanced to the diagonal branch and standard motorised pullback was performed at a rate of 0.5 mm/s. The resulting image is shown in fig 3. IVUS imaging showed that the vessel size of the LMCA was about 3.5 mm, larger than that revealed by QCA. The MLD by IVUS imaging was 1.5 mm and the plaque area was 76.7% after the pre-dilatation.

DCA was performed from the LAD diagonal branch to the LMCA with a Flexi-cut M device (Devices for Vascular Intervention, Redwood City, California, USA) (fig 4). After a total of 24 cuts for debulking with balloon inflation (maximum 7 atm), the ostium of the high lateral branch and LCx was occluded by plaque shift (fig 4C). We could pass the intermediate wire (Asahi Intecc, Nagoya, Japan) through the ostium of both branches, and DCA was also performed from both vessels to the LMCA with IVUS guidance (fig 5). After a total of seven cuts for debulking with balloon inflation (maximum 8 atm), we attempted coronary stenting
from the proximal LAD to the LMCA (fig 5C). After stent implantation at 16 atm (S670 stent with 3.5 mm diameter and 15 mm length; Medtronic, Minneapolis, Minnesota, USA), kissing balloon inflation was performed with a 3.5 mm balloon in the LAD diagonal branch and a 2.5 mm balloon in the high lateral branch (fig 6A). These procedures were completed without complication (fig 6B).

After PCI the patient’s congestive heart failure promptly improved. She was discharged without in-hospital complications. An eight month follow up CAG showed no restenosis in the LMCA, LCx, high lateral branch, and proximal LAD diagonal branch, and the patient has remained alive and asymptomatic to date.

**DISCUSSION**

In this case, the reference vessel size estimated by QCA was smaller than that estimated by IVUS. In many cases of LMCA stenosis, especially in a distal bifurcated lesion, atherosclerosis distributes from the LMCA to the proximal LAD or LCx. Therefore, this kind of discrepancy occurs sometimes, and IVUS is useful for determining the correct size of the LMCA. The IVUS image can also show the distribution and

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**Figure 4** Directional coronary atherectomy (DCA) procedures. Panel C shows the occluded high lateral branch and left circumflex coronary artery after DCA. HL, high lateral branch; LCX, left circumflex coronary artery, RAO, right anterior oblique.

**Figure 5** Wiring and recanalisation for high lateral branch. Panel C shows the positioning of the coronary stenting. HL, high lateral branch.

**Figure 6** Kissing balloon inflation and final angiogram. Successful dilatation was attained in the distal bifurcated LMCA, HL, high lateral branch; LCX, left circumflex artery.
characteristics of lesion plaque. These facts show the importance of IVUS as a decision making tool when considering PCI for the treatment of unprotected LMCA.

Stent implantation in unprotected LMCA can improve procedural and late clinical outcomes. However, concerns about stenting related complications such as stent jail, stent thrombosis, and in-stent restenosis remain. Although DES can dramatically decrease the restenosis rate, these devices are not effective for improving the initial results of PCI in unprotected LMCA. Before we apply DES for all LMCA stenoses, we must reconsider these points. For complex lesions in unprotected LMCA such as diffuse lesions, those in small vessels, bifurcated lesions, and heavily calcified lesions, there may be still some limitations for coronary stenting, even in the era of DES.

In this patient, stenting had the high possibility of stent jail by plaque shift because the lesion was diffuse, moderately calcified, and located in a distal bifurcation with the narrowing ostium in the LCx. As debulking seemed to improve the result of PCI in these lesions, we performed IVUS guided DCA before stenting. With IVUS guidance, debulking was performed more precisely and aggressively, and produced a large lumen area and low residual plaque burden in this lesion. Although side branch occlusion occurred after the debulking, we could pass the wire through the occluded lesions in this case. If the stent had been placed there first, we could not have passed the wire through the lumens because of the obstacles presented by the stent struts. Moreover, the stent was easily delivered and dilated after the debulking, even if the wire could not have been passed through the struts. This also shows the importance of IVUS as a decision making tool when considering PCI for the treatment of unprotected LMCA.

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