CASE REPORT

Intracoronary demonstration of adenosine-induced coronary collateral steal

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Abstract

A steal phenomenon was detected by intravascular Doppler guidewire in a patient with a well collateralised coronary vascular area supplied by a reopened left circumflex coronary artery. This phenomenon accounted for the fall in blood flow velocity reserve during hyperaemic conditions to 50% of the baseline value. The collaterals must have been the cause

of the steal phenomenon because complete revascularisation of the lesion barely reversed it.

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Figure 1 (A) Angiogram (left anterior oblique view) showing the occluded left circumflex coronary artery (LCX) with the guidewire positioned just proximal to the bifurcation of two posterolateral branches (large arrow). There was retrograde filling of the branches through collaterals from the left anterior descending artery (small arrow). (B) View of the recanalised LCX with the Doppler guidewire located distal to the previously occluded lesion (arrow).

Ten to 30% of patients with coronary artery disease undergoing dipyridamole stress testing and nuclear perfusion imaging show a fall in stress image activity below resting levels.¹ One postulated mechanism by which the pharmacological stress test produces myocardial ischaemia includes coronary steal via collaterals.¹⁻⁶ Coronary steal is commonly defined as an absolute or relative fall in coronary blood flow to a certain vascular region in favour of another supply area under conditions of coronary arteriolar vasodilatation.^{1 7 8} The mechanism for this redistribution of blood flow can be a fall in perfusion pressure at the origin of collateral vessels caused by proximal stenoses of coronary arteries supplying the collaterals or by proximal viscous friction developing at high flow states even in normal coronary arteries from which the collaterals arise.^{1 3 5}

In general, the term "steal" is a misnomer, because blood is not stolen from the collateral-dependent myocardial bed by an overall backward flow away from this area. There is a net decrease during hyperaemia in blood flow toward the area rather than a flow directed away from it. The decrease in blood delivery during hyperaemia translates into a flow rate below resting level in the collateral-dependent region which can be detected by non-invasive methods.⁹

In humans there have been no reports of invasive, quantitative assessment of adenosine-induced coronary collateral steal. With the advent of small intravascular Doppler guidewires (1/3 mm in diameter) it has become possible to verify directly the consistent but indirect and qualitative findings of non-invasive methods applied to pharmacologically induced steal phenomena.⁹

Case report

A 51 year old symptom free diabetic man with

Figure 2 Evidence of coronary artery steal. (A) Proximal coronary flow velocity trend plot before, during, and after intracoronary injection of 18 μ g adenosine. Coronary flow velocity reserve (CFVR) was 1.2. (B) CFVR measurement distal to the recanalised lesion showing a 50% decrease in coronary flow velocity after intracoronary injection of 18 μ g adenosine compared with baseline conditions (CFVR = 0.5). B, baseline; S, search mode for peak velocity (P) after adenosine injection.

Figure 3 Quantitative assessment of coronary collaterals to the LCX. (A) Velocity trend plot before (patency) and during balloon occlusion of the recanalised lesion showing that about half the blood flow—compared with conditions during patency—was provided by anterograde collateral flow (almost 50% was supplied by retrograde collateral flow). (B) Intracoronary Doppler flow velocity profile obtained during balloon occlusion of the recanalised LCX occlusion at a site far distal to that location (see fig 1B). There is an anterograde (positive) velocity profile throughout the entire cardiac cycle with an average peak flow velocity (BAPV) of 11 cm/s.

an electrocardiographically positive ergometric stress test and a reversible scintigraphic myocardial perfusion defect at the inferoposterior region underwent coronary angiography. The patient had no history of myocardial infarction. Cardiovascular risk factors included diabetes, smoking, and a family history of coronary artery disease.

Coronary angiography showed two-vessel coronary artery disease with an occlusion of the left circumflex coronary artery (LCX) just proximal to the bifurcation into two posterolateral branches (fig 1A). The right coronary artery showed a proximal 50% diameter stenosis. There was retrograde filling by collaterals of the epicardial segment distal to the LCX occlusion (fig 1A). The left ventricular (LV) angiogram was normal (ejection fraction = 80%). There were no regional systolic LV wall motion abnormalities. The LCX occlusion was recanalised with a laser wire.

Before percutaneous transluminal coronary angioplasty (PTCA), a Doppler guidewire (0.014 inch Flowire, Cardiometrics, Mountain View, CA, USA) was positioned distal to the recanalised LCX occlusion (fig 1B). Blood pressure and heart rate remained constant throughout the procedure.

EVIDENCE FOR CORONARY STEAL

The coronary flow velocity reserve (CFVR) obtained proximal to the recanalised LCX lesion when 18 μ g intracoronary adenosine was given was 1.2 (fig 2A). The adenosine-induced CFVR measured distal to the previous occlusion was 0.5, indicating a flow rate during hyperaemia that was half that at rest—that is, a steal phenomenon in the vascular region where the Doppler wire was positioned (fig 2B and 1B). During a short period of several cardiac cycles, retrograde flow velocity profiles could be detected that corresponded to “steal” in the true sense of the word (fig 2B).

EVIDENCE FOR EXTENSIVE COLLATERALS TO THE LCX

Figure 3 shows the flow velocity profiles in the LCX posterolateral branch during occlusion of this branch proximal to the position of the Doppler wire (fig 1B). This is evidence for anterograde and retrograde collateral flow to this coronary vascular area with an average anterograde peak flow velocity of 11 cm/s (anterograde flow). The collateral flow lasted for the entire cardiac cycle. Relative to the flow velocity in the non-occluded vessel during baseline conditions, the anterograde average peak flow velocity during balloon occlusion amounted to about half the baseline flow velocity (fig 3A). Additionally, retrograde occlusion flow velocity was also about 50% of baseline flow velocity (12 cm/s). Baseline and occlusion flow velocities were measured after completion of the revascularisation procedure.

DISTAL CFVR AFTER PTCA AND STENT IMPLANTATION

CFVR after PTCA and before stent implantation measured at the distal location of the Doppler wire that corresponded to all CFVR

A

Distal to the
LCX lesion:
pre-PTCA

B

Distal to the
LCX lesion:
post-PTCA
/pre-stent

C

Distal to the
LCX lesion:
post-stent

Figure 4 Decrease of coronary steal after interventional therapy. (A) Velocity trend plot of CFVR measurement ($= 0.5$) obtained immediately after recanalisation of the LCX occlusion (same plot as figure 2B). (B) CFVR increased to $0.8-1.1$ after PTCA of the lesion to a residual stenosis of 30% in diameter which still indicates some steal. (C) After stent implantation by high pressure balloon expansion, CFVR increased only to 1.2 despite no angiographically visible, residual stenosis. APV, average peak velocity; B, baseline; D, diastole; P, peak velocity after intracoronary adenosine injection; S, search mode for P and systole, respectively.

measurements was $0.8-1.1$ (fig 4B). This indicates that a small amount of the blood flow during resting conditions was directed away from the vascular area distal to the recanalised LCX lesion, even after PTCA that resulted in a residual stenosis of only about 30–40% in diameter.

CFVR after PTCA and stent implantation at the previously occluded location was no longer influenced by reversed flow, but finally reached a value of only 1.2 (fig 4C). Angiographically, there was no stenosis detectable after stent implantation.

Discussion

Coronary steal has been studied experimentally, modelled theoretically, and demonstrated indirectly by dipyridamole perfusion scans and positron emission tomography imaging in humans.^{14,58} There are different categories of steal including one relating to the presence of an intact collateral circulation, subendocardial steal, and coronary branch steal at vascular bifurcations. Subendocardial steal and coronary branch steal do occur in the absence of collaterals.^{7,8} So far, there has been only one case report demonstrating direct intracoronary evidence of dipyridamole-induced coronary steal.⁶ However, this study did not provide evidence for collaterals, thus leaving room for speculation whether coronary branch steal or collateral steal was the cause of flow reversal during arteriolar dilatation. The steal phenomenon in one posterolateral branch of the LCX coronary artery demonstrated in the present study was probably caused by the extensive collaterals to this region and not by the recanalised LCX lesion (that is, branch steal).

A coronary artery branch steal would be caused by the hyperaemia-related decrease of microvascular resistance in the LCX branch adjacent to the posterolateral branch originating from the same bifurcation (fig 1B). The fall in resistance there would be more pronounced than in the vessel containing the Doppler wire. This could be the case when an occlusion is distal but not proximal to the bifurcation, as in the present case. Thus complete revascularisation of an occlusion *distal* to the bifurcation would have restored hyperaemic flow more completely than in the present case, where the steal barely disappeared and hyperaemic flow was only 20% instead of $\geq 100\%$ higher than at rest. The fact that the restoration of the hyperaemic flow was much less than expected points to the collaterals between the left anterior descending and the LCX posterolateral region which continued to be present after revascularisation and served as a conduit to the lower resistance contralateral vascular region during hyperaemia.

The findings of the present study imply that an inadequate functional result after complete revascularisation may be related to the presence of a well developed collateral circulation that can vastly influence the distribution of regional vascular resistances and cause coronary steal. The question arises whether revascularisation in such a case is necessary or even counterproductive. On the other hand, coronary steal caused by uneven epicardial resistances (that is, branch steal) may be successfully treated by PTCA because regional resistances are thus equalised.

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