

# Abrupt Vessel Closure

A detailed look at the intraprocedural decision making required to handle this challenging presentation.

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**A** 52-year-old woman with hypertension, dyslipidemia, and known obstructive coronary artery disease presented with symptoms concerning for recurrent angina. Previous percutaneous coronary interventions (PCIs) were performed on the ostium of the second obtuse marginal artery (OM2) and also on the right coronary artery (RCA) and right posterior descending artery bifurcation using a two-stent Culotte technique. Drug-eluting stents were utilized in both cases. Outpatient nuclear perfusion imaging revealed new lateral wall ischemia, prompting left heart catheterization.

Cardiac catheterization demonstrated moderate left anterior descending (LAD) artery disease, moderate RCA in-stent restenosis, and severe left circumflex (LCX) disease involving the origin of the previous OM2 stent (Figure 1).

## DECISION POINT 1 What Are the Treatment Options?

Optimization of medical therapy, repeat PCI, and referral for coronary artery bypass grafting were each contemplated based on anatomy. Data from the COURAGE trial suggest that optimization of medical therapy is noninferior to a revascularization approach in terms of the hard endpoints of death and myocardial infarction.<sup>1</sup>

However, patients in this trial derived significant angina relief from revascularization. Given the presenting symp-

toms, a revascularization approach was therefore reasonable. In addition, nuclear subset data from the COURAGE trial revealed benefits in patients with underlying significant ischemic burden, as in our patient.<sup>2</sup> Given the focal nature of the culprit lesion, lack of diabetes, left ventricular dysfunction, and severe stenosis of the LAD, in addition to the patient's desire to avoid open heart surgery, PCI was advised. During the decision-making process, however, the patient reported acute anginal chest pain, with inferolateral ST depressions noted on the monitor.

## DECISION POINT 2 What Is Causing This Patient's Chest Pain? What Is Indicated Next?

The clinical syndrome and electrocardiogram findings suggested ischemia in an inferior or inferolateral distribution. Because the RCA was most recently engaged,



Figure 1. Right anterior oblique left coronary angiography revealed moderate LAD disease and critical culprit lesion in the LCX at the proximal edge of a previously placed drug-eluting stent (A). Left anterior oblique angiography of the RCA revealed moderate focal in-stent restenosis of the previously placed right posterior descending artery stent (B).



Figure 2. Emergent left coronary angiography using a 6-F, JL4 guide catheter revealed complete occlusion of the LCX.

immediate repeat angiography of this vessel was indicated, revealing normal perfusion and unchanged angiographic findings. No collaterals were apparent to the left coronary system. As such, a 6-F, JL4 guide catheter was placed in the left coronary artery because emergent PCI of a left coronary vessel seemed likely. Angiography revealed complete occlusion of the LCX at the level of the previously described lesion (Figure 2).

**DECISION POINT 3**

**What Can Explain the Angiographic Finding?**

Differential diagnosis for acute occlusion (abrupt ves-

sel closure) of the LCX includes coronary vasospasm, thrombosis (embolic or in situ formation), dissection, air embolus, or progression of acute coronary syndrome. Because the lesion was never wired, acute thrombosis seemed unlikely. Similarly, because chest pain and electrocardiogram changes started after RCA angiography, it seemed unlikely that air or thrombus emboli or catheter-induced dissection caused the vessel closure. Moreover, catheter-induced dissection should initiate in the left main or origin of the LCX, whereas angiography yielded normal findings in these segments. Thus, vasospasm and/or progression of the culprit lesion in acute coronary syndrome seemed most likely.

**DECISION POINT 4**

**What Is Indicated Next?**

Given the likely progression of acute coronary syndrome, with or without superimposed vasospasm, the patient received intracoronary nitroglycerin (200 µg), a heparin weight-based bolus, and glycoprotein IIb/IIIa receptor inhibitor therapy. Although bivalirudin is an option based on available data,<sup>3</sup> glycoprotein IIb/IIIa receptor inhibitors remain the gold standard in patients undergoing high-risk, complex lesion intervention, especially for complications such as abrupt vessel closure related to acute coronary syndrome or dissection.<sup>4,5</sup>

Repeat angiography revealed continued occlusion of the vessel. Therefore, once an activated clotting time (ACT) > 200 was confirmed, the lesion was wired with a short Cougar wire, and multiple balloon inflations from the proximal LCX into the OM2 were performed using a 2.5- X 12-mm Voyager compliant balloon (Abbott Vascular, Santa Clara, CA) (Figure 3A). Based on the focal and relatively proximal nature of the lesion, and

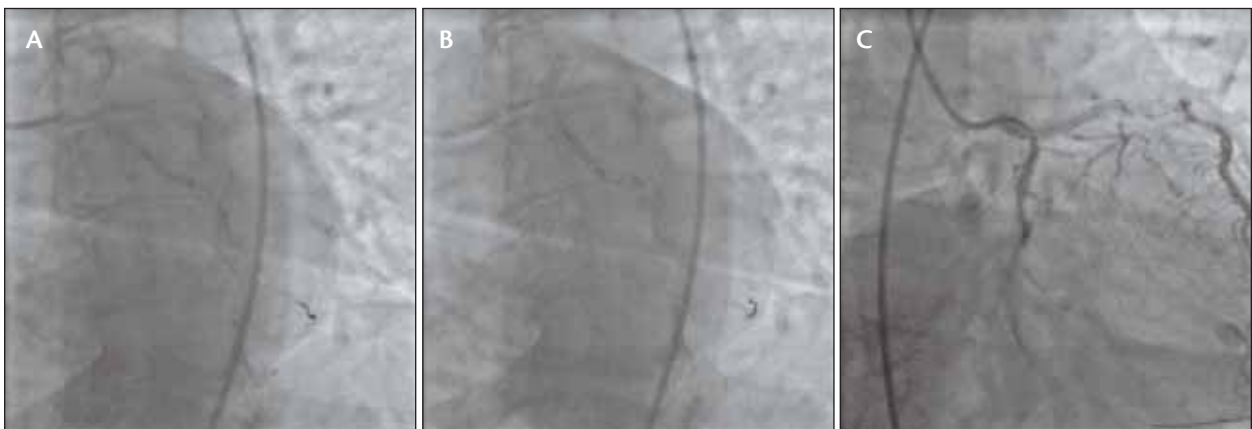


Figure 3. Multiple distal-to-proximal balloon inflations were performed using a 2.5- X 12-mm Voyager balloon over a short Cougar wire (Medtronic, Inc., Minneapolis, MN) (A). Dye hang-up was noted at the point of initial occlusion, indicating persistent poor antegrade flow and/or coronary dissection (B). Angiography revealed persistent poor antegrade flow despite multiple balloon inflations of the culprit lesion (C).

lack of obvious calcification, a short wire was used and was believed to be sufficient. In addition, although we chose to proceed with multiple balloon inflations, aspiration thrombectomy could have been attempted at this time as an initial strategy to improve antegrade flow.

After balloon inflations, angiography revealed dye hang-up at the proximal edge of the previously placed stent (at the culprit lesion, extending slightly into the OM2), consistent with coronary dissection (Figure 3B). Also, there was continued poor/no flow into the OM2 vessel, and the patient continued to note substernal chest pain (Figure 3C).

**DECISION POINT 5**

**What Explains the Angiographic Finding, and What Should You Do Next?**

Causes of poor flow in this situation include lack of sufficient anticoagulation and resultant refractory thrombus, coronary dissection, and inadvertent wire passage (and balloon dilation) behind the previously placed stent. The ACT was again checked and confirmed as therapeutic, and glycoprotein IIb/IIIa receptor inhibitors were already being utilized. Because balloon catheters were able to initially dilate past the previously placed stent, inadvertent wire passage behind the stent was thought unlikely. Therefore, the most likely etiology of poor flow appeared to be high-grade coronary dissection (type E or F with staining). The nomenclature for coronary dissection is shown in Table 1.<sup>6</sup> The decision was made to continue with balloon inflations using a larger balloon (3- X 12-mm Maverick [Boston Scientific Corporation, Natick, MA]), but this time the



**Figure 4.** Inability to advance a larger 3- X 12-mm balloon past the point of dye hang-up and poor guide support.

balloon was unable to be advanced past the dissection point (proximal edge of the previously placed stent). Figure 4 shows the balloon stuck at the lesion, with the guide catheter being pushed out of the coronary ostium as a result.

**DECISION POINT 6**

**What Explains the Angiographic Finding? What Can You Do Next?**

Inability to advance a larger balloon past the lesion can be due to propagation of dissection, impinging on

**TABLE 1. CORONARY DISSECTION CLASSIFICATION SYSTEM**

Grade	Description
A	Small radiolucent area within the lumen of the vessel
B	Linear, nonpersisting extravasation of contrast
C	Extraluminal, persisting extravasation of contrast
D	Spiral-shaped filling defect
E	Persistent lumen defect with delayed antegrade flow
F	Filling defect accompanied by total coronary occlusion
Length	Measure end-to-end for type B through F dissections
Staining	Persistence of contrast within dissection after washout of contrast from remaining portion of vessel

the central lumen and physically impeding balloon advancement. However, there remained some concern that the wire was behind the previous stent, allowing small balloons to pass the lesion but not larger balloons. The latter situation continued to seem unlikely because smaller balloons had passed freely and were dilated without difficulty. Compounding the problem, the 6-F, JL4 guide catheter did not appear to provide sufficient backup. To improve backup, and perhaps modify the dissected lesion to allow larger and/or noncompliant balloon advancement, a buddy wire was placed into the upper branch of the OM2 vessel (Figure 5A).

The 3- X 12-mm Maverick was now able to be advanced with use of the buddy wire, and sequential distal-to-proximal inflations were performed. Although every attempt was made to maintain placement of both wires, the buddy wire was inadvertently pulled back by receding balloon inflations (Figure 5B). Despite successful balloon dilations throughout the course of the vessel, both distal to and proximal to the previously placed stent, there remained no antegrade flow. Further, placement of both drug-eluting and bare-metal 2.5- X 12-mm stents was attempted at the site of dissection, but we remained unable to reach the proximal edge of the prior stent. With each attempt, the guide was pushed out of the coronary ostium, indicating a lack of support and resistance to forward stent advancement.

**DECISION POINT 7**

**What Is Causing Failure at This Juncture? What Are the Available Options?**

The differential diagnosis of the potential etiologies for failure at this juncture includes (1) poor backup, (2) continued severe dissection with impingement of the central vessel lumen, (3) wire passage behind the previous stent allowing balloon passage but not stent passage, and (4) the wire not being in the true lumen. Although changing guide backup to a larger-diameter guide or alternate curve is a possibility, the technique

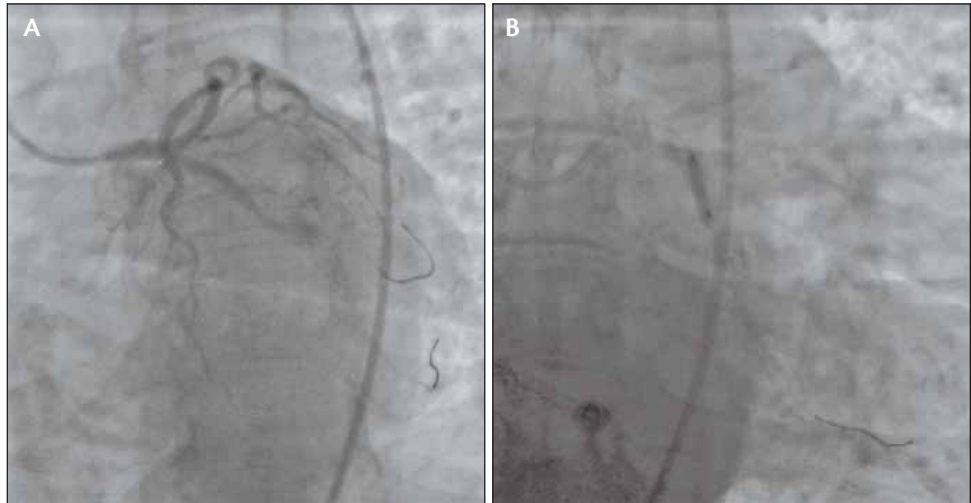


Figure 5. A buddy wire has been placed in the upper branch of the OM2 to improve support for balloon and/or stent advancement (A). After the buddy wire technique allowed distal advancement of the larger 3- X 12-mm Maverick balloon, multiple distal-to-proximal balloon inflations were performed (B).

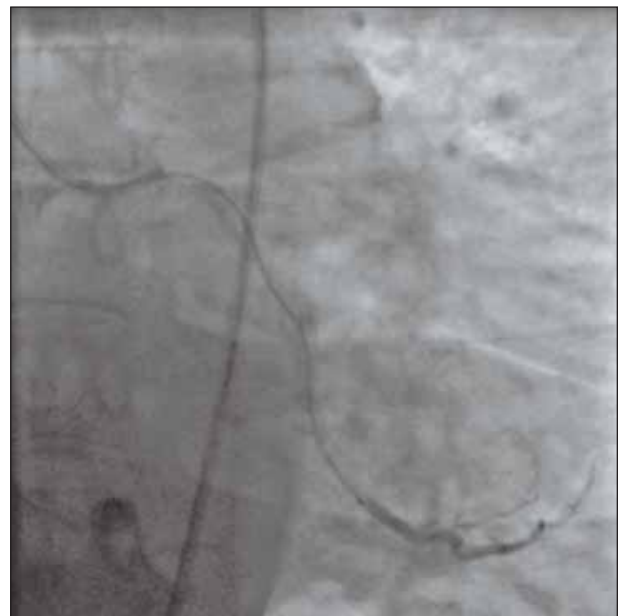
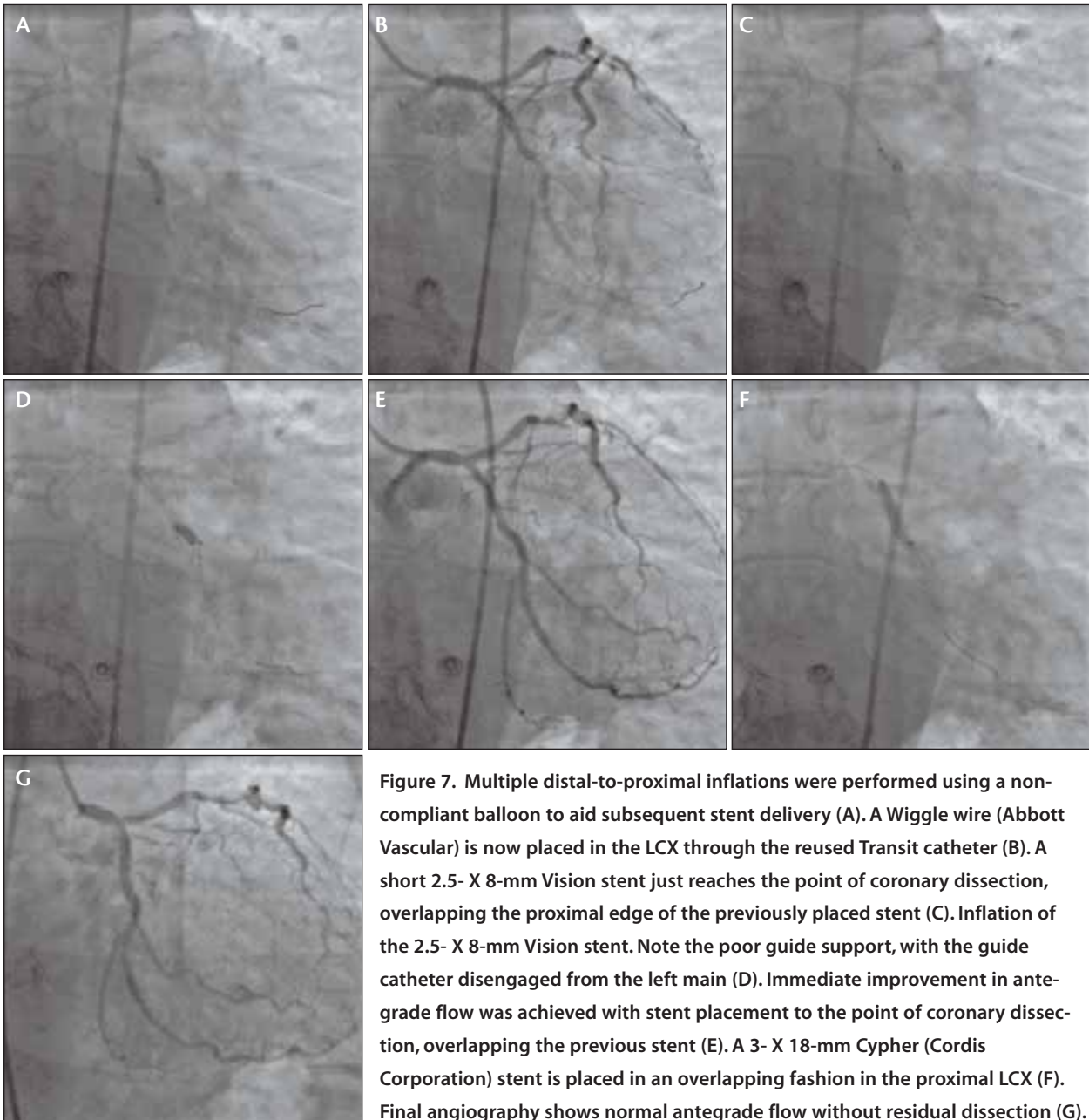


Figure 6. A Transit catheter (Cordis Corporation, Bridgewater, NJ) is placed across the lesion into the distal vessel. Contrast injection through the central lumen of the catheter revealed normal distal vessel flow without dissection, confirming wire placement in the true lumen and making wire passage behind the previous stent unlikely.

has a failure rate of at least 20%, with the possibility of losing wire position in the setting of known coronary dissection. Therefore, backup exchange was not initially considered to be a realistic option. It is imperative at



**Figure 7.** Multiple distal-to-proximal inflations were performed using a non-compliant balloon to aid subsequent stent delivery (A). A Wiggle wire (Abbott Vascular) is now placed in the LCX through the reused Transit catheter (B). A short 2.5- X 8-mm Vision stent just reaches the point of coronary dissection, overlapping the proximal edge of the previously placed stent (C). Inflation of the 2.5- X 8-mm Vision stent. Note the poor guide support, with the guide catheter disengaged from the left main (D). Immediate improvement in antegrade flow was achieved with stent placement to the point of coronary dissection, overlapping the previous stent (E). A 3- X 18-mm Cypher (Cordis Corporation) stent is placed in an overlapping fashion in the proximal LCX (F). Final angiography shows normal antegrade flow without residual dissection (G).

this point, however, to confirm that the last two possibilities do not exist. Therefore, a Transit catheter was advanced to the distal vessel, with contrast injection through the catheter lumen showing good distal flow past the lesion, effectively ruling out wire passage in a false lumen and making wire passage behind the previous stent less likely (Figure 6). Moreover, the Transit catheter allowed for an exchange to a stiffer All-Star wire (Abbott Vascular) for added backup, while maintaining use of the same guide catheter. The Transit catheter was removed and stent placement was

attempted again with a 2.5- X 12-mm Vision stent (Abbott Vascular). However, despite use of the stiffer All-Star wire, the stent still would not reach the lesion.

#### DECISION POINT 8

##### What Are the Available Options?

The options at this point include repeat balloon inflations over the stiffer wire, changing to a different wire, repeating the buddy wire technique, or changing guide catheter backup, which might also include upsizing to a 7-F system. Repeat balloon inflations with a noncompli-

ant 2.5- X 12-mm balloon were performed, because it was the safest of available options (Figure 7A). However, stent placement was still not possible, and there remained continued TIMI 1 antegrade flow. Although changing guide backup was still a consideration, the decision was made to replace the Transit catheter and switch to a Wiggle wire for optimal support (Figure 7B). This wire has a zig-zag configuration along its intracoronary length that increases support through wire contact and friction with the vessel wall. In addition, the wire may alter the tortuosity or trajectory of the artery in such a way as to improve balloon and/or stent delivery. This allowed for successful placement of a short 2.5- X 8-mm Vision stent at the proximal edge of the previous stent, the site of dissection initiation, with resultant TIMI 3 antegrade flow (Figure 7C through E). Due to their improved deliverability, a bare-metal stent was chosen. A 3- X 18-mm Cypher was placed in an overlapping fashion in the proximal LCX to achieve the final successful result (Figure 7F and G). The patient's chest pain dissipated, and she was transferred to the recovery room in a hemodynamically stable condition. Glycoprotein IIb/IIIa receptor inhibitors were maintained for an additional 18 hours, clopidogrel 600 mg was loaded, and the patient was discharged at 36 hours without further complication.

**CONCLUSION**

PCI is fraught with potential intraprocedural complications that require rapid pattern recognition, complex decision making, and a multitude of available tools to get the operator and patient out of harm. Too often we focus on the indications and contraindications of procedures and fail to spend adequate time addressing the art of the procedure itself. The current case illustrates the development of abrupt vessel closure in a semielective patient and the thought process and technical challenges that this situation represents for the interventionalist.

Several take-home points are illustrated. First, elective procedures may turn into emergent interventions due to catheter-induced dissection, coronary vasospasm, air or thrombus emboli, or progression of an unstable lesion, as in the present case. Second, significant coronary dissections, both spontaneous and iatrogenic, can impair antegrade flow, make it difficult to discern true from false lumen, and impede the advancement of balloons and stents. Such dissections typically occur in calcified and tortuous vessels, oftentimes with previous stents in place, compounding the problem. Techniques to allow increased support and the advancement of balloons and stents include the use of two wires (buddy

wire) and the Transit catheter, the latter to allow changing to more supportive wires and visualization of the distal vasculature. Multiple balloon inflations with compliant, and then noncompliant, balloons may also be useful to change vessel architecture and tack up residual dissections. Third, changing guide backup over both the long coronary wire and a 0.035-inch exchange wire looped in the ascending aorta is possible and should be considered if all else fails; however, this is not recommended as an initial strategy in the setting of coronary dissection due to the possibility of losing distal wire position in an already compromised vessel. Finally, tacking up the dissection is vital, even with a short bare-metal stent, and will result in resolution of symptoms and reconstitution of antegrade flow. Therefore, efforts must be directed toward achieving safe yet rapid stent placement to the site of dissection initiation. ■

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