

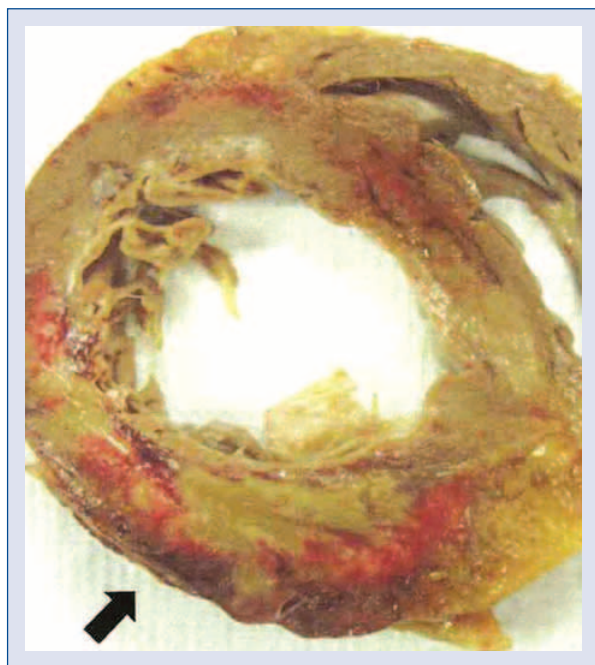
## ‘No-reflow’ phenomenon

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A 57-year-old man presented with new anginal symptoms nine years after three-vessel coronary artery bypass grafting. Cardiac catheterization revealed severely and diffusely diseased saphenous vein graft to the obtuse marginal coronary artery. Percutaneous coronary intervention was complicated by the ‘no-reflow’ phenomenon. The patient suffered a periprocedural myocardial infarction (peak troponin 26.3 ng/mL) and died from low-output cardiogenic shock four days after the procedure.

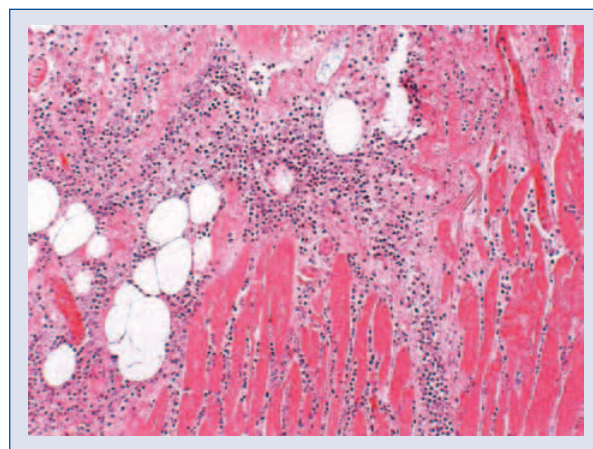
Gross autopsy findings demonstrated myonecrosis of the posterolateral wall (Fig. 1, black arrow).



**Figure 1.** Gross autopsy findings of posterolateral wall of the myocardium — arrow demonstrates extensive myonecrosis of the posterolateral wall of the myocardium on autopsy in the distribution of the obtuse marginal artery.

The infarction is in the distribution of the obtuse marginal coronary artery and extends further than the distribution of the obtuse marginal coronary artery secondary to low perfusion resulting from cardiogenic shock. Histopathologic examination revealed massive myonecrosis and abundant polymorphonuclear leukocytes consistent with recent myocardial infarction (Fig. 2). It is highly unusual and interesting that the hematoxylin and eosin stain demonstrated merely the inflammatory cells and necrosis, rather than debris resulting from procedure-related embolization.

The ‘no-reflow’ phenomenon is defined as inadequate myocardial perfusion through a given segment of the coronary circulation without evidence of vessel obstruction. After placement of an embolic filter protection device, the graft was successfully recanalized with a bare metal stent. Immediately after the stent was placed, the ‘no-reflow’ phe-



**Figure 2.** Hematoxylin and eosin stain demonstrating the damaged posterolateral wall of the myocardium — massive myonecrosis and abundant polymorphonuclear leukocytes are consistent with recent myocardial infarction.

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nomenon was observed despite initial filter protection and subsequent administration of intracoronary vasodilators. The underlying pathophysiology is not completely understood, but it may occur as a result of vessel microembolization or microcirculatory spasm and edema. The 'no-reflow' phenomenon is associated with increased hospital mortality and periprocedural myocardial infarction. Pathologic changes in the myocardium are similar to those seen in atherothrombotic acute myocardial infarction.

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