



Introduction

Coronary artery dissection is a rare but significant nonatherosclerotic cause of acute coronary syndrome, with the potential to progress to fatal vessel occlusion. Dissections can occur spontaneously or iatrogenically, typically following a percutaneous coronary intervention (PCI), where disruption of the coronary intima creates a false lumen between the vessel wall layers. Due to its rarity, there is ongoing debate regarding the optimal treatment strategies.

Case Presentation

A 66-year-old female with a history of coronary artery disease, status post drug-eluting stents placed in the left anterior descending (LAD) and left circumflex (LCX) arteries, presented one month post-procedure with acuteonset dyspnea and angina. Laboratory evaluation revealed elevated troponin (1.966 ng/mL) and brain natriuretic peptide (1,400 pg/mL); however, the ECG showed no acute ischemic changes. The patient was started on dual antiplatelet therapy (DAPT), metoprolol succinate, and losartan, and was promptly taken for coronary angiography.

Cardiac catheterization revealed a significant 15mm edge dissection at the site of the previously placed ostial circumflex stent, extending into the media of the proximal LCX. An everolimus-eluting stent was successfully placed to cover the edge dissection.

Utilizing a Drug-Eluting Stent for an latrogenic Coronary Artery Dissection: Case Report

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Discussion

Remote coronary artery dissections post-stenting, although rare, have been documented in the medical literature. Case reports, such as one by Dogan et al., illustrate that in-stent dissections can occur even years after stent implantation and may be successfully managed with coronary re-stenting. The American Heart Association has recognized the risk of dissection extension following PCI for spontaneous coronary artery dissection, noting the potential for propagation both upstream and downstream within the stented vessel. As a result, conservative therapy is typically preferred, with revascularization reserved for high-risk cases characterized by ongoing ischemia, significant vessel occlusion, or hemodynamic instability.

However, Madhavan et al. highlighted that very-late stentrelated major adverse cardiovascular events (MACE) can occur between one and five years post-PCI, underscoring the need for long-term vigilance in patients with prior stent placement. In this reported case, the dissection was likely iatrogenic, secondary to the previously placed stent. Given the patient's acute symptoms and significant troponin elevation despite medical management, PCI was pursued due to concerns for further dissection propagation and acute vessel closure.

Conclusions

This case contributes to the growing body of evidence on the delayed complications of coronary stenting, such as coronary artery dissection, as seen in our case. The timing of these complications can vary, with dissections occurring anywhere from months to years after stent placement, reinforcing the need for ongoing clinical surveillance and timely intervention when late adverse events arise.

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