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IMAGING VIGNETTE

Coronary Embolization Following Balloon Dilation of Lipid-Core Plaques

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BALLOON DILATION OF A CORONARY STENOSIS CONTAINING A LIPID CORE PLAQUE (LCP) may be complicated by distal embolization of plaque contents, leading to slow or no-reflow and peri-stenting myocardial infarction (MI) (1). Near-infrared spectroscopy (NIRS) (LipiScan Coronary Imaging System, InfraReDx, Inc., Burlington, Massachusetts) by a catheter-based system, can identify LCP in patients prior to balloon dilation (2). This report describes 4 cases (2 with clinical data and 2 with histological data) that elucidate the connections among: 1) balloon dilation of LCP; 2) slow or no-reflow; 3) peri-stenting MI; and 4) histological findings. By connecting the dots among these 4 cases, it is possible to envision the pathophysiology of a mechanism of peri-stenting infarction, and identify a possible means of prevention by the NIRS-guided use of a distal protection device.

Case 1. Pathologic Substrate Underlying a Circumferential LCP by NIRS: Ex Vivo Histopathological–Chemography Correlation

A post-mortem NIRS examination was performed in a blood perfused coronary artery of a 48-year-old man who suffered sudden coronary death. Figure 1C is a display of NIRS measurements made during pullback (x-axis, mm) and rotation (y-axis) within the artery. The NIRS examination indicated that a massive circumferential LCP, represented by circumferential yellow signal (Fig. 1C), described as a "napkin-ring" lesion, was present. Histological results confirm this measurement showing that a massive, circumferential LCP was present with numerous cholesterol crystals (Fig. 1A). An intracoronary thrombus was also seen at the distal edge of the LCP (Fig. 1B). The solid arrow between panels A and C, and the dashed arrow between panels B and C, colocalize the histologic specimens with their corresponding locations on the chemogram. This autopsy case documents the histological findings expected when the NIRS measurement yields a circumferential yellow signal.

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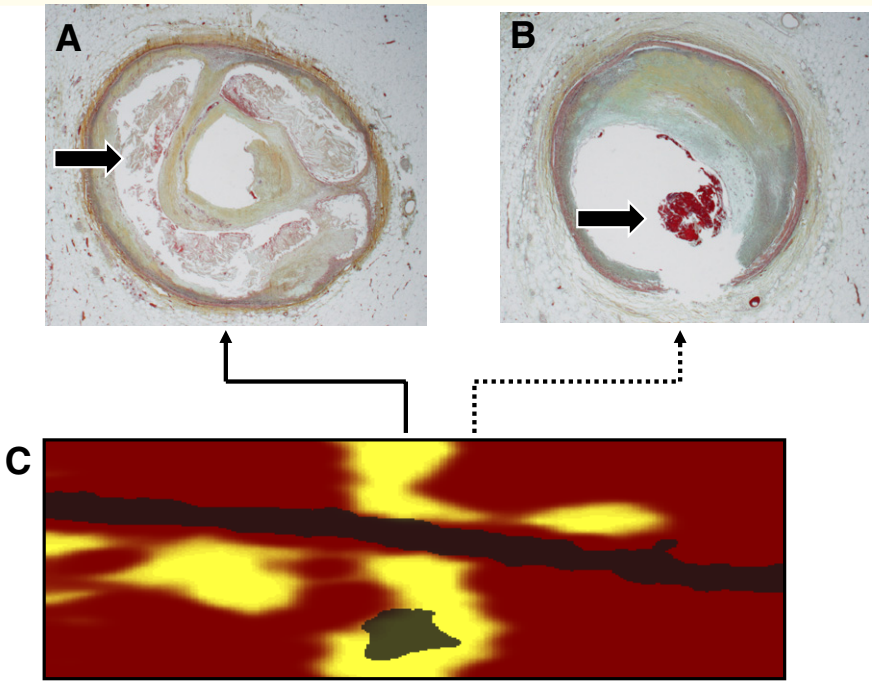
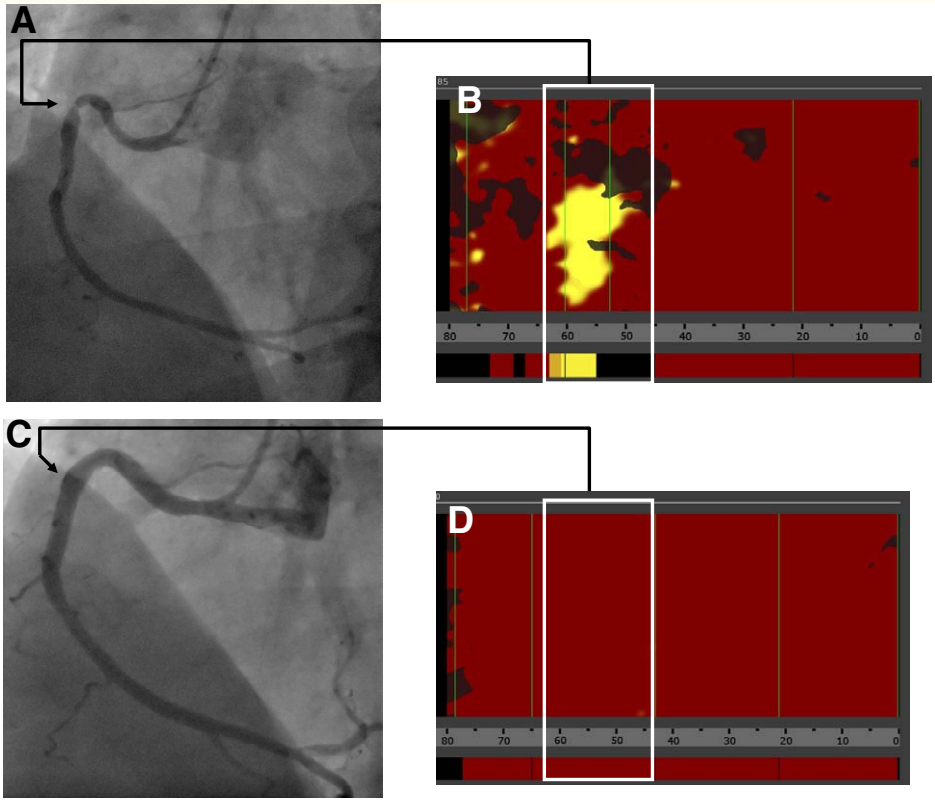
Figure 1. Circumferential LCP: Histopathological–Chemographic Correlates



Case 2. Post-Stenting No-Reflow Following Balloon Dilation of Stenosis and Disappearance of a Circumferential LCP NIRS Signal

A 71-year-old male with unstable angina had a hazy tight culprit lesion in the proximal right coronary artery with Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 (Fig. 2A, arrow). Pre-intervention NIRS revealed a prominent circular yellow lesion occupying >50% of the vessel circumference (Fig. 2B). Stent deployment followed by inflation of a noncompliant balloon led to an excellent result at the lesion site (Fig. 2C, arrow), but was complicated by no-reflow with deterioration to TIMI flow grade 1 associated with deep ST-segment depression. Intracoronary adenosine and nitrates were administered with eventual return of brisk flow and resolution of ST-segment changes. Repeat NIRS interrogation revealed a complete disappearance of the LCP (Fig. 2D). These findings document that intervention on an extensive circumferential LCP resulted in no-reflow associated with the disappearance of the LCP, suggesting that cholesterol-laden plaque embolized downstream and caused the no-reflow.

Figure 2. No-Reflow Following Stenting of Circumferential Yellow LCP



Case 3. Post-Stenting No-Reflow and Infarction Following Balloon Dilation of Circumferential Yellow NIRS Signal

A 62-year-old man with stable angina underwent coronary angiography, which demonstrated a complex hazy ulcerated culprit lesion in the mid-right coronary artery (Fig. 3A, solid arrow). Neither the angiogram nor an intravascular ultrasound image indicated the presence of thrombus. NIRS demonstrated a large yellow signal spanning the circumference of the culprit site (Fig. 3B, white rectangle), indicating the presence of a napkin-ring LCP; a smaller LCP was evident distally (Fig. 3, open arrow). Balloon angioplasty was performed (Fig. 4A, arrow), which led to prompt no-reflow (Fig. 4B, arrow) associated with severe bradyarrhythmia and profound hypotension (Fig. 4C). After brief cardiopulmonary resuscitation and pharmacological support with atropine and dopamine, physiologic rhythm and blood pressure were restored and stenting resulted in excellent angiographic outcome. However, the patient developed a peri-stenting nontransmural infarction (peak creatine kinase of 512 ng/ml) and required an additional day of hospital care in an intensive care unit. The circumferential LCP observed was similar in appearance to the post-mortem NIRS examination in Case 1, establishing a link between NIRS measurements, histological findings, and catastrophic events during stenting.

Figure 3. **"Napkin-Ring" Circumferential LCP**

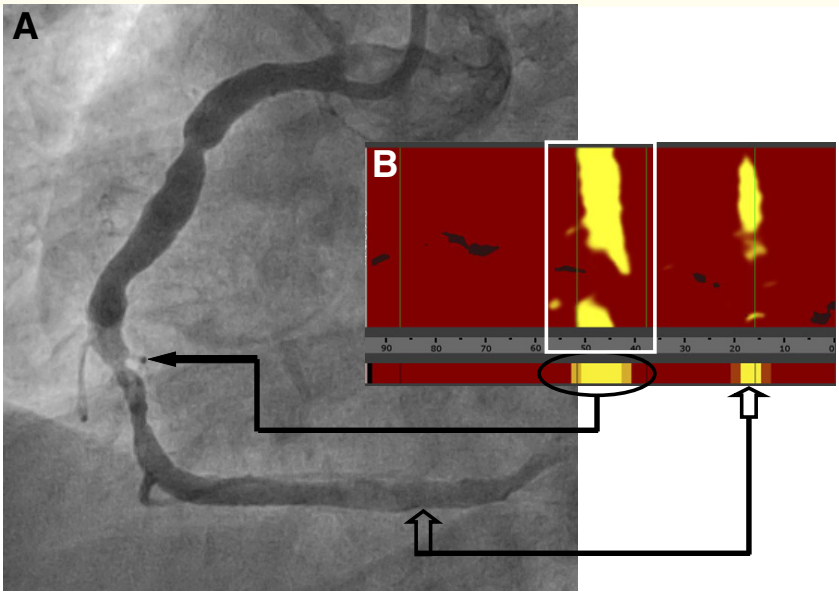
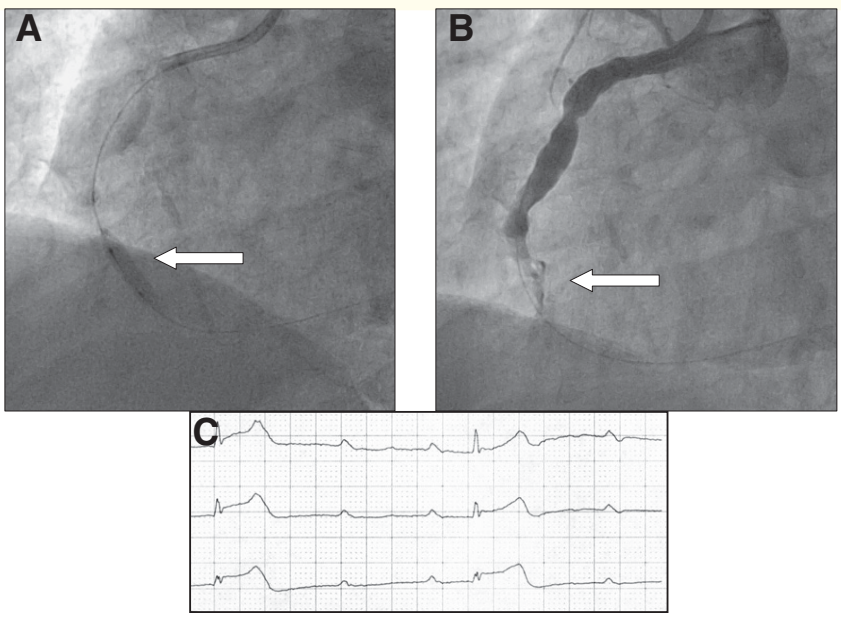


Figure 4. **Profound No-Reflow After Balloon Dilation of LCP**



Case 4. Refractory and Fatal No-Reflow Following Angioplasty: Angiographical-Histopathological Correlates

A 63-year-old woman presented with a nontransmural MI. Coronary angiography revealed a complex hazy ulcerated culprit lesion in the mid-right coronary artery (Fig. 5A, arrows). The case was performed 5 years before the availability of NIRS. Balloon angioplasty was complicated by severe coronary no-reflow. A stent was placed with an acceptable angiographic result at the lesion site (Fig. 5B, black arrows), but with further exacerbation of severe no-reflow distally (Fig. 5B, white arrows). Arteriolar vasodilators were administered which improved epicardial coronary flow. However, there was a myocardial blush grade score of 0 to 1 with profound and persistent hypotension refractory to all measures including aggressive intracoronary pharmacologic treatment, intra-aortic balloon pump support, and parenteral pressor support. The patient died from refractory hypotension and shock 36 h after the coronary intervention. At autopsy, histopathologic

analysis revealed extensive cholesterol emboli plugging the distal right coronary artery circulation (Fig. 6, arrows). This case links the angiographic and pathologic findings associated with balloon dilation, no-reflow, and cholesterol emboli: The angiographic pattern of a hazy ulcerated plaque in this case is similar to that in Case 3, in which balloon dilation of a napkin-ring LCP resulted in profound no-reflow, and demonstrates embolization of cholesterol crystals similar to those seen in Case 1.

Figure 5. Refractory No-Reflow After Balloon Dilation of Ulcerated Plaque

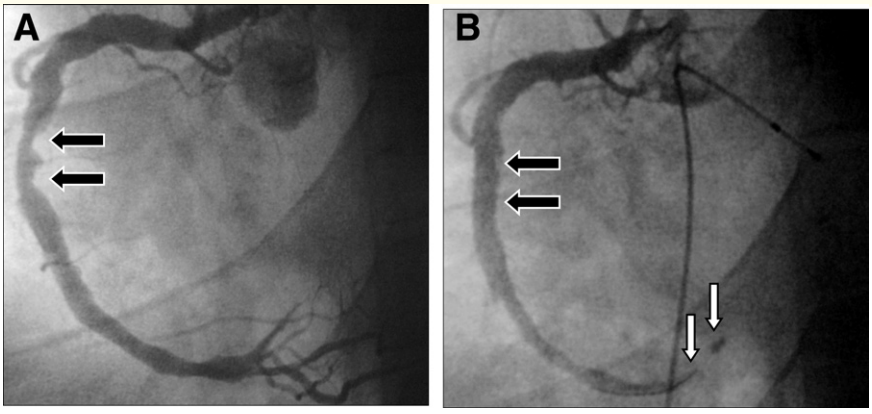
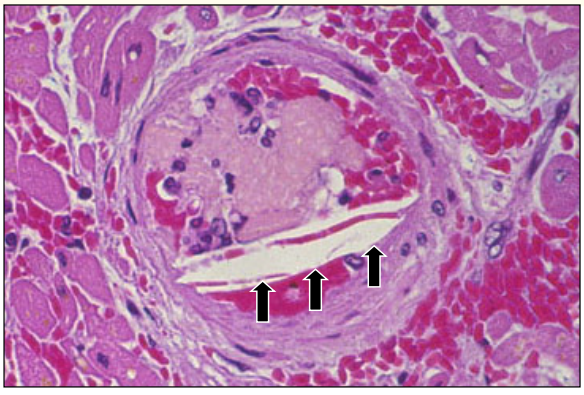


Figure 6. Angiographic-Pathological Correlates of Fatal No-Reflow



DISCUSSION

These findings provide insight into the pathophysiology of distal embolization following balloon dilation of coronary stenoses, an important and potentially fatal complication of stenting, and suggest a means of prevention. It appears likely that dilation/stenting of a large LCP results in embolization of cholesterol crystals and other debris into the distal vessels causing no-reflow that may in turn lead to a catastrophic coronary event. Additional studies are needed to quantitate the ability of NIRS to predict the occurrence of peri-stenting infarction and to test, in a randomized trial, the strategy of NIRS-guided use of a distal protection device.

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