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A 62-year-old female with a past history of acute infero-posterior myocardial infarction 12 years ago and diabetes mellitus treated with insulin for 25 years, underwent follow-up coronary angiography. Although free from angina, diffuse 75% stenosis in proximal LAD and proximal LCX were detected. Suspecting silent myocardial ischemia due to long history of diabetes mellitus, we decided to perform PCI to LAD. Pre-PCI IVUS showed diffuse soft plaque with deep ultrasound attenuation (attenuated plaque) about 40mm in length. In concern of distal embolism and no-reflow phenomenon, predilatation was performed with a filter distal protection device (Filtertrap™). However, after the second balloon dilatation, coronary angiogram showed no-reflow phenomenon and blood pressure dropped to 60/ mmHg. IABP was applied and inotropic agent infusion started. Despite intracoronary nitroglycerin injection, coronary flow remained TIMI-I flow. IVUS revealed that the target lesion showed positive remodelling, moreover, the attenuated plaque broke away and disappeared. Coronary flow recovered to TIMI-II flow after stent implantation, nevertheless, precordial ST elevation was sustained and regional wall motion remained akinetic.

The no-reflow phenomenon is one of the catastrophic complications during PCI, and is known to be more complicated in ACS than in elective cases. Positive remodelling and/or attenuated plaque in IVUS findings are reported to be good predictors. We could not avoid this feared complication even with a filter distal protection device, perhaps because the plaque was too massive. Should we have used other protection devices? Or, avoid PCI initially?