Adenosine-Induced Vasospasticity in a Myocardial Bridge... Endothelial Dysfunction?

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We report the case of a fit 90-year-old woman presenting with increasing episodes of angina. Diagnostic angiography identified a mid-left anterior descending artery myocardial bridge (MB) (Figure 1, Online Video 1) as well as a moderate diagonal disease. We performed fractional flow reserve measurement of the diagonal branch, which was negative. Interestingly, after adenosine infusion, the myocardial bridge showed severe and fixed spastic narrowing (throughout the cardiac cycle, not just during systole) (Figure 1, Online Video 2).

Adenosine produces vasodilation by binding to its receptors (A2a) on the surface of vascular smooth muscle cells, activating adenylate cyclase and leading to an increase in cyclic adenosine monophosphate (cAMP) concentration and cAMP-dependent protein kinase activation. The latter results in potassium channel opening, resulting in hyperpolarization of vascular smooth muscle cells, inhibits the entry of Ca\(^{2+}\) and also activates inducible nitric oxide synthase (1).

Endothelial function is impaired in patients with MB (2). Angelini et al. (3) described transient extreme spasticity in response to acetylcholine at the site of myocardial bridging (evidence of endothelial dysfunction because the normal endothelium has a vasodilatory response to acetylcholine testing). It seems that the same mechanism is responsible for the spastic response to adenosine observed in our patient, supporting the presence of endothelial dysfunction in the MB segment.

**FIGURE 1** Snapshot Showing Bridging in the Mid-Left Anterior Descending Artery Before Adenosine Infusion

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APPENDIX For accompanying videos, please see the online version of this article.