Intramural Coronary Amyloidosis Mimicking Effort Angina Pectoris Preceding Fatal Heart Failure

Miwako Ishida, MD; Hisahito Shinagawa, MD, PhD; Yoko Yamada, MD, PhD; Tomohiro Mizutani, MD, PhD; Atsuko Hara, MD, PhD; Junya Ako, MD, PhD; Takayuki Inomata, MD, PhD

A 72-year-old woman was referred for examination of exertional angina. Exercise stress test indicated broad redistribution of thallium-201 uptake (Figure B) associated with ST-segment depression on electrocardiogram (Figure A), but coronary angiography showed no stenotic lesions (Figure C). The patient continued to have angina despite maximum pharmacotherapy under a possible diagnosis of coronary artery spasm or microvascular angina. The patient died because of progressive heart failure at 14 months after the first visit.

At autopsy (Figure D), dilation of both cardiac ventricles suggesting cardiac failure was observed macroscopically. Histopathology was characterized by AL and lambda-type amyloid fibril deposits predominantly in the intramural coronary arteries (Figure G) and partly in the myocardial interstitium (Figure F). Figure E shows the unequal distribution of amyloid deposition in the vascular walls: scant deposits in the subepicardial coronary arteries and deposit-filled cavities in the occluded subendocardial coronary arteries.1

Although heart failure due to hypertrophied and restrictive myocardium is the most common cardiac manifestation, angina symptoms may precede the onset of heart failure in some patients with primary amyloidosis.2 Clinicians must consider amyloidosis as a differential diagnosis when evaluating patients with angina pectoris with no angiographically apparent lesions.

Disclosures

The authors declare no conflict of interest.

References
