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IgG4-Related Coronary Arteritis With Acetylcholine-Induced Coronary Vasospasm

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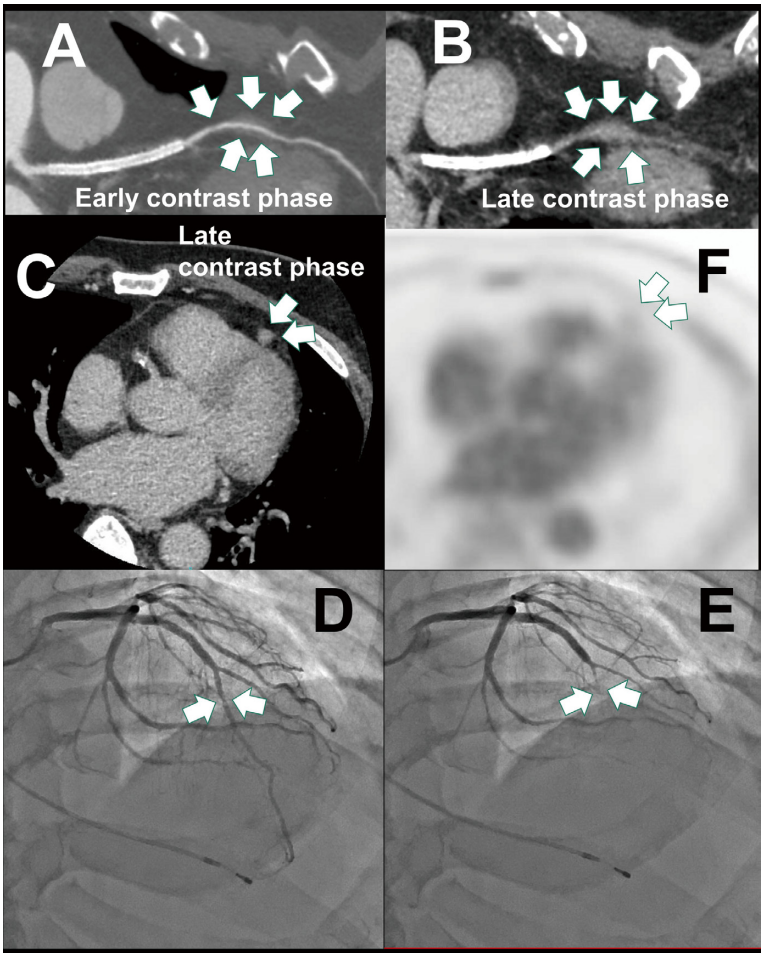

Figure. (**A**–**C**) ECG-gated computed tomography shows a thickened arterial wall of the mid-left anterior descending (LAD) coronary artery with moderate stenosis and late enhancement (white arrows). (**D**) Invasive coronary angiography demonstrates moderate stenosis at the same position (white arrows). (**E**) During an acetylcholine provocation test, the occlusion was induced at the segment with coronary artery wall thickening (white arrows). (**F**) ^{18}F-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography showed increased ^{18}F-FDG uptake in the thickened LAD (white arrows).

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A 72-year-old man with old inferior myocardial infarction and hepatocellular carcinoma presented with left-side and right-side shoulder pain on effort during the daytime and was referred to our department. His electrocardiogram (ECG) showed no ischemic ST-T changes. However, his serum troponin I was 466.2 pg/mL (normal range, ≤34.2 pg/mL). ECG-gated computed tomography showed a thickened arterial wall of the mid-left anterior descending (LAD) coronary artery with moderate stenosis and late enhancement (**Figure A**–**C**, white arrows). Invasive coronary angiography demonstrated moderate stenosis at the same position (**Figure D**, white arrows). The fractional flow reserve of the LAD stenosis was 0.73, and the coronary flow reserve and index of microcirculatory resistance were 3 and 7, respectively. An acetylcholine provocation test was performed because of the elevation of troponin I. The occlusion was induced at the segment with coronary artery wall thickening (**Figure E**, white arrows). ST elevation on precordial leads and chest pain were detected, but shoulder pain was not detected. ^{18}F-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography showed increased ^{18}F-FDG uptake in the thickened LAD (**Figure F**, white arrows), but no other abnormality suggesting immunoglobulin-4 (IgG-4)-related disease was detected. His IgG-4 level was 431 mg/dL (normal range, 11–121 mg/dL). Therefore, he was diagnosed with coronary arteritis-inducing effort angina due to IgG-4-related disease.[1] Several vasodilators had controlled his symptoms well and his hepatic cancer was endstage; therefore, immunosuppression therapy or coronary intervention was not performed. To the best of our knowledge, this is the first report of drug-induced vasospasm associated with IgG4-related coronary arteritis.

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