A 64-year-old man was referred to our coronary care unit because of a 2-week history of frequent, severe chest oppression at rest and after a 10-m walk in the morning. Following a diagnosis of unstable angina, the patient underwent emergency cardiac catheterization. The coronary arteriogram demonstrated an anomaly of a right dominant single coronary artery arising from the right sinus of Valsalva. The left main coronary artery (LMCA) was branched at the proximal site of the right coronary artery (RCA). No significant fixed stenosis was detected during intravenous drip infusion of nitroglycerine, except for a slight narrowing of the middle of both the RCA and LMCA. Left ventriculography showed slight hypokinesis in the anterior wall with an ejection fraction of 70%. The LMCA showed no obvious compression during a complete cardiac cycle on biplane coronary arteriography. The LMCA branching from the RCA was seen to pass between the ascending aorta and the pulmonary arterial trunk in an ECG gated multislice CT scan (Aquilion; Toshiba Co, Tokyo, Japan) (Fig 1).

After a period of 2 days to allow for the washing out of diltiazem and isosorbide mononitrate, the anterior wall ischemia was documented in exercise stress thallium scintigraphy (Fig 2), accompanied by chest oppression and significant ST-segment depression in leads V3–6. Ten days after undergoing the emergency cardiac catheterization examination, the patient underwent an acetylcholine provocation test using a 2.3Fr Transit Catheter™ (Cordis Co, Miami, FL, USA) within a 6Fr guiding catheter, which was inserted into the LMCA. The spontaneously spastic lesion (70% reduction in diameter) in the LMCA (Fig 3A, B) became a total obstruction as early as 50 s after intra-LMCA infusion of acetylcholine.

**Key Words:** Acetylcholine; Single coronary artery; Spasm
infusion of 20μg acetylcholine (Fig 3C,D), followed by persistent severe chest oppression and ST-segment elevation in leads I, aVL and V3–6 (Fig 4). Subsequent angiography showed no significant lesion after intracoronary infusion of isosorbide dinitrate (E,F).

The patient has been free from angina for 6 months on a regimen of 200 mg of diltiazem, 40 mg of isosorbide mononitrate and 15 mg of nicorandil per day.

Discussion

Isolated single coronary artery, which occurs in 0.024% of the population, is recognized as one of the potentially serious coronary anomalies, together with ectopic coronary origin from the pulmonary artery and large coronary fistulae. Some investigators have suggested that patients with a single coronary artery may be predisposed to sudden death or acute myocardial infarction and spasm of the aberrant coronary artery is thought to be one of several mechanisms that explain these critical complications. Yamamoto et al first reported ergonovine-provoked spastic stenosis in the RCA branching from the left coronary artery in a patient with a single coronary artery and to our knowledge, the present report is the first to describe the induction of totally occlusive spasm of the LMCA between the great arteries in a patient with a RCA type single coronary artery. By performing the acetylcholine provocation test, spasm of the aberrant coronary artery was shown to be a potential mechanism for sudden death in such patients. At the same time, it was proven that the attack could be prevented by the pharmacotherapy. Coronary arterial bypass grafting or percutaneous transluminal coronary angioplasty is recommended if the spastic angina persists despite intensive medical intervention.

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References