CASE REPORT

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A Case of Isolated Septal Myocardial Infarction: Myocardial Perfusion-Metabolism Mismatch as a Tool for Diagnosis

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ABSTRACT

Isolated septal myocardial infarction is an uncommon condition with diagnostic difficulty due to small infarction size and anatomical variations. We report a case of isolated septal myocardial infarction, in which the diagnosis was confirmed not by electrocardiographic, echocardiographic, or angiographic findings, but by nuclear imaging. A 46-year-old man with chest discomfort exhibited ST-segment elevations in leads V1 and V2, and borderline abnormalities of the septal wall motion on echocardiography. Emergency coronary angiography demonstrated delayed flow in the second septal branch of the left anterior descending coronary artery. Intravascular ultrasound showed plaque in the proximal portion of the septal branch without evidence of plaque rupture. No balloon angioplasty or stent implantation was required because the flow delay in the septal branch disappeared after the intravascular ultrasound procedure. Myocardial perfusion-metabolism mismatch, as assessed by resting thallium-201 and iodine-123-beta-methyl-p-iodophenyl-pentadecanoic acid, was seen in the mid-septal region.

Septal myocardial infarction is commonly developed with anterior myocardial infarction because the feeding artery of the ventricular septum is a branch of the left anterior descending (LAD) coronary artery. Only a few cases of isolated septal myocardial infarction have been reported,[1-4] and available data on this condition are limited. We experienced a case of isolated septal myocardial infarction, in which myocardial perfusion-metabolism mismatch, as assessed by nuclear imaging, was useful in determining the site of the small myocardial infarction.

CASE REPORT

A 46-year-old man was admitted to Matsushita Memorial Hospital, Osaka, Japan because of an abnormal electrocardiogram. The patient had noticed exertional chest discomfort for a few months, but did not seek medical care because his symptoms improved with rest and had not changed in frequency or severity over time. One day before admission, he had a 30-minute episode of chest discomfort at rest in the morning. The next day, his chest symptoms reportedly recurred during rest in the morning and lasted for approximately one hour. The patient visited his physician in the afternoon and was found to have an abnormal electrocardiogram. He was transferred to the emergency room of the hospital, when his symptoms had already disappeared. The patient had a history of hypertension but had taken no drugs. He was a current smoker with a 25 pack-year history.

His physical examination was normal, other than a blood pressure of 189/130 mmHg. An electrocardiogram showed normal sinus rhythm, a heart rate of 65 beats per minute, ST-segment elevations in V1 and V2, and negative T waves in anterior and lateral leads [Figure 1]. His chest radiograph was unremarkable. His troponin T level was 1.74 ng/mL (reference, < 0.1 ng/mL). Echocardiography demonstrated borderline abnormalities of wall motion in the ventricular septum. A diagnosis of acute myocardial infarction was made, although the infarcted area remained undetermined.

Intravenous administration of isosorbide dinitrate, followed by heparin (5000 units), did not alter his electrocardiogram. Emergency coronary

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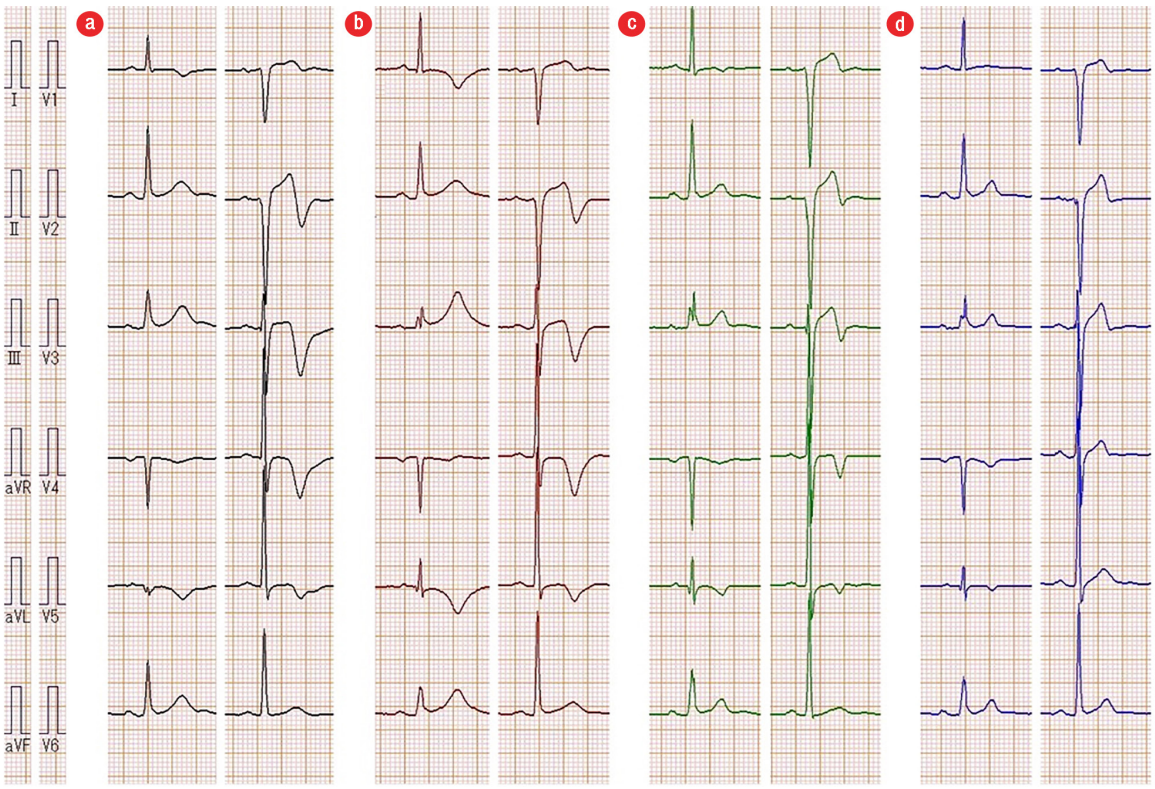

Figure 1: Serial electrocardiograms for a 46-year-old male. **(a)** On admission, ST-segment elevation in leads V1 to V2, and negative T waves in I, aVL, and V2 to V5 were seen. **(b)** These findings were still present the next day, **(c)** but improved five days after admission, and **(d)** almost disappeared one month later. Note: lack of septal q waves in V5 and V6 during the clinical course.

angiography, which was performed after initiation of aspirin (200 mg) and clopidogrel (300 mg), revealed no significant stenosis with a delayed flow in the second septal branch of the LAD [Figure 2a]. Intravascular ultrasound was done in the second septal branch for further examination because the delayed flow in the second septal branch of the LAD did not improve after administration of intracoronary isosorbide dinitrate, intravascular ultrasound of the LAD, and inserting a wire into the septal branch. Intravascular ultrasound showed a mild to moderate amount of plaque burden in the proximal portion of the septal branch without evidence of plaque rupture [Figure 2b], whereas no atherosclerosis was observed in the mid- to distal portions [Figure 2c]. No balloon angioplasty or stent implantation was required because the flow delay in the septal branch disappeared after the intravascular ultrasound procedure [Figure 2d].

Four days after admission, we performed myocardial perfusion and fatty acid metabolism assessment with thallium-201 and iodine-123-beta-methyl-p-iodophenyl-pentadecanoic acid (I-123-BMIPP). Thallium-201 (111 MBq) and I-123-BMIPP (111 MBq) were injected intravenously at rest, and 36 images over a 180-degree anterior arc were obtained five minutes or three hours after tracer injections with a digital gamma camera, equipped with a low-energy, high-resolution, and parallel-hole collimator. The acquisition lasted 50 beats per projection, was stored in a matrix of 64 × 64 pixels, and the images were reconstructed using a Hanning filter without attenuation or scatter correction. The bull's-eye map with thallium-201 demonstrated mildly reduced tracer uptake in the mid-septal region on the delayed images, whereas on I-123-BMIPP, the same reduction was observed in the early images with tracer washout in the delayed images [Figure 3].

The patient was diagnosed with isolated septal myocardial infarction. His clinical course was uneventful, and his peak level of creatine kinase was 823 U/L (measured on admission). Electrocardiographic abnormalities gradually improved, although lack of septal q waves in V5 and V6 persisted [Figure 1]. Follow-up angiography, performed six months later, showed good blood flow

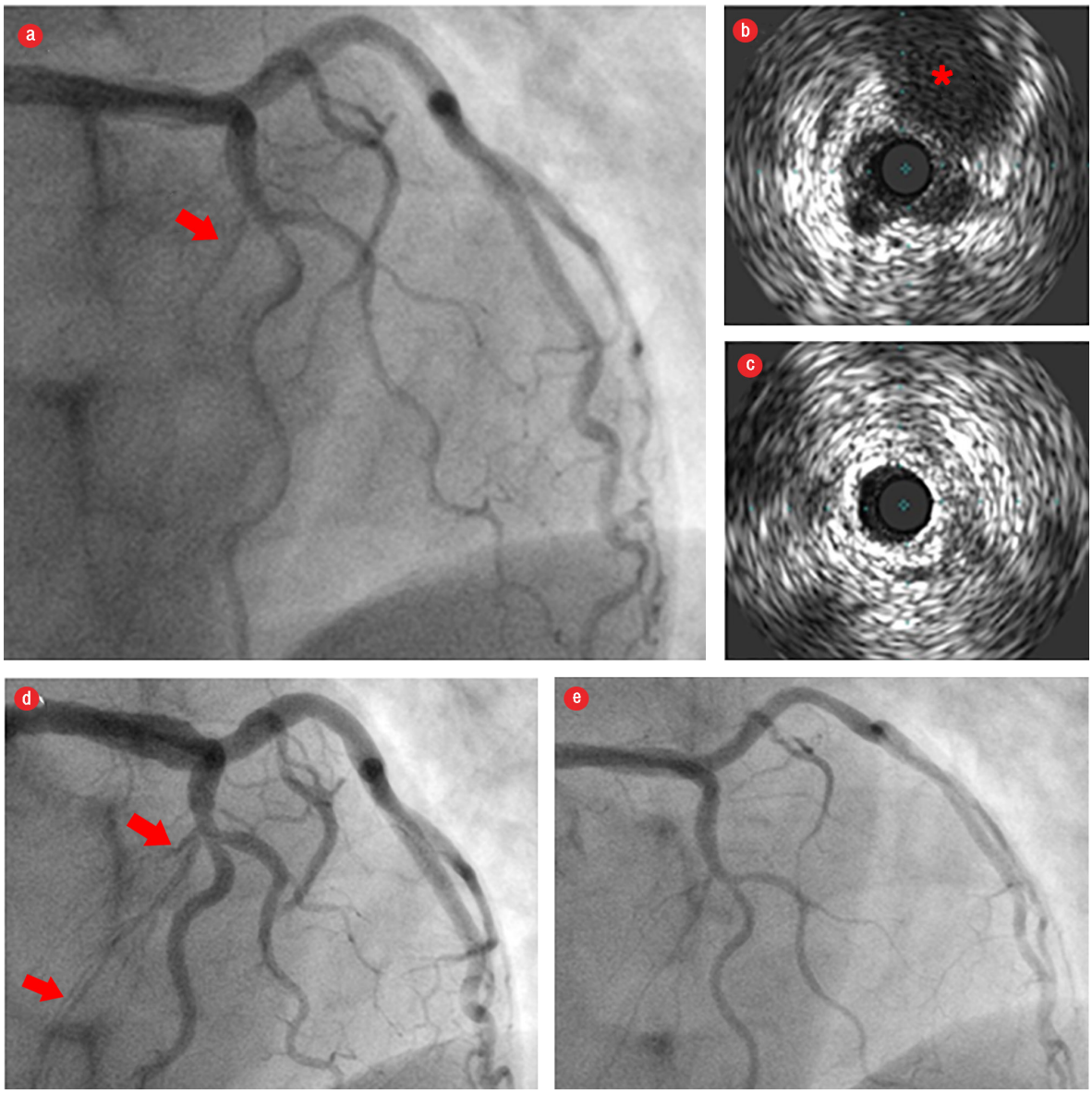

Figure 2: Coronary angiography and intravascular ultrasound imaging. **(a)** We observed no significant stenosis in the main artery, but flow delay in the second septal branch of the left anterior descending was present (red arrow). **(b)** Intravascular ultrasound images revealed a mild to moderate amount of plaque burden in the proximal portion of the septal branch, in the absence of atherosclerotic changes in the left ascending coronary artery (asterisk), whereas **(c)** no atherosclerosis was observed in the mid- to distal portions of the septal branch. **(d)** Following the procedure, angiography revealed improved flow in the septal branch with no flow delay. **(e)** Follow-up angiography performed six months later, demonstrated good blood flow in the septal branch.

in the second septal branch of the LAD [Figure 2e]. The patient was doing well with daily aspirin 100 mg and statins for more than two years after discharge.

DISCUSSION

The site of myocardial infarction was not easy to determine in our case. Electrocardiographic, echocardiographic, and angiographic findings were consistent with septal myocardial infarction, but not definitive enough to make a diagnosis of isolated septal myocardial infarction. This may be explained by anatomical variations of the septal branch; in our case, the culprit lesion (i.e., the second sepal branch of the LAD) was not large. Electrocardiographic changes are reported to vary in patients with septal myocardial infarction.[1,2,5] By contrast, nuclear imaging with thallium-201 and I-123-BMIPP (to determine

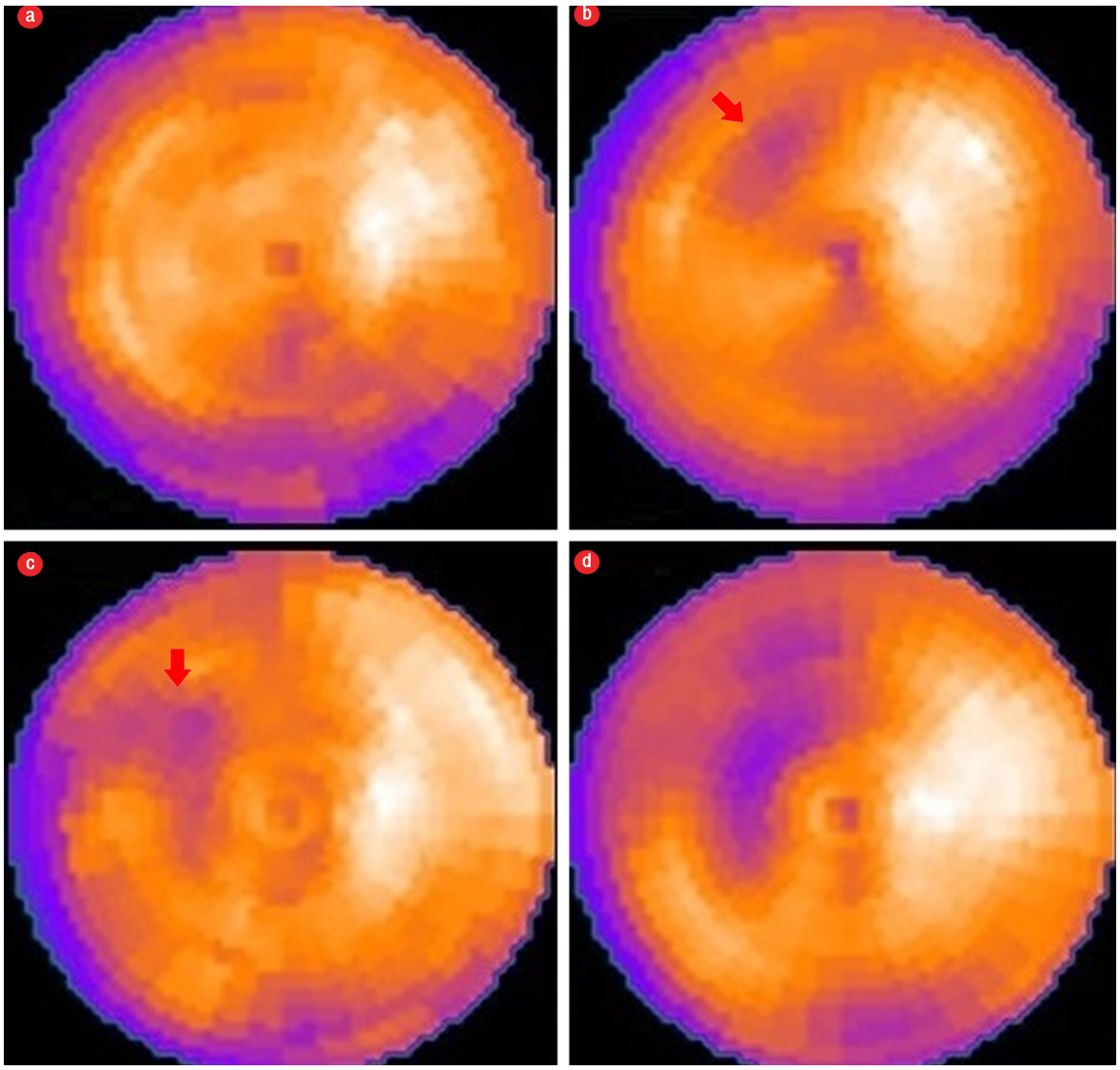

Figure 3: Resting nuclear imaging. **(a)** Thallium-201 bulls-eye map showed no perfusion defect five minutes after injection, **(b)** but mildly reduced tracer uptake or reverse redistribution in the ventricular septum three hours later (red arrow). **(c)** Mildly reduced I-123-beta-methyl-p-iodophenyl-pentadecanoic uptake on the initial bulls-eye map (red arrow) with **(d)** high washout on the delayed image.

perfusion-metabolism mismatch) was informative despite the relatively small amount of infarction and the uncommon site limited to the mid-septum.

Myocardial perfusion imaging at rest was useful in assessing myocardial viability. The good uptake of thallium-201 on the initial images in our case suggests that severe damage was unlikely in the ventricular septum. Such small damage can also be undetectable on cardiac magnetic resonance, although the radiation-free technique is reported to be useful in the diagnosis of isolated septal myocardial infarction.[3,4] Nevertheless, we observed mildly reduced tracer uptake on the delayed images of thallium-201 in our case. This finding, so-called reverse redistribution, has been associated with less severe damage, such as nontransmural myocardial infarction, patent infarct-related coronary artery, and myocardial salvage.[6,7] Given that the determinant of thallium-201 redistribution is a balance between its influx and efflux,[8,9] the latter seemed predominant in our case. This phenomenon is due, in part, to a rapid recovery of thallium-201 washout rate after the improvement of coronary perfusion pressure,[10] although the kinetic behavior of thallium-201 in biological tissue is not fully understood.

Myocardial imaging with I-123-BMIPP indicated impaired fatty acid metabolism in the mid-septum, findings consistent with myocardial damage

due to reduced flow in the septal branch of the LAD. It is worth noting that myocardial uptake of I-123-BMIPP, as compared with thallium-201 uptake, was more severely impaired in our case. These findings are known as a perfusion-metabolism mismatch,[11,12] and are explained by the ability of the myocardium to use not only free fatty acids but also other substrates as an energy source — such as glucose, lactate, or ketones — to protect the heart from direct energy deprivation.[13,14] Among these alternative sources, glucose is a major energy substrate in the presence of severe coronary disease.[15,16] Thus, it is reasonable to consider that a switch from free fatty acid metabolism to glucose metabolism had developed in our case due to blood flow reduction in the septal branch of the LAD. On the delayed images of I-123-BMIPP, increased washout was observed in the mid-septal region. Although the clinical significance of delayed images of I-123-BMIPP remains to be elucidated, the relation between washout rates of I-123-BMIPP and improvement of ventricular function was observed in patients with successfully reperfused ST-segment elevation myocardial infarction.[17]

It is intuitive to consider that the mechanism of pure septal infarction in our case was due to the semi-occluded lesion of the proximal portion in the second septal branch of the LAD. Intravascular ultrasound exhibited progressed atherosclerosis at the culprit site, but no plaque rupture was detected in our case. It remains controversial whether rupture or erosion of the plaque surface was attributable to the onset. Given that his symptoms were associated with conditions at rest in the morning, coronary artery spasm should also be considered as a possible trigger for the myocardial infarction; however, we did not perform a spasm provocation test. Spontaneous coronary artery dissection can cause an isolated septal myocardial infarction,[4] but the possibility is less likely in our case because of the absence of dissection on angiographic and intravascular ultrasound images.

CONCLUSION

We experienced a case of isolated septal myocardial infarction, in which the diagnosis was confirmed by perfusion-metabolism mismatch on nuclear imaging. Given the rarity of this condition, clinical data should be accumulated for the diagnosis and treatment.

Disclosure

The authors declared no conflicts of interest. No funding was received for this study.

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