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Tc-99m Sestamibi Myocardial Perfusion Imaging After Coronary Artery Bypass Grafting for Ischemic Heart Failure

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ABSTRACT

Heart failure (HF), a clinical syndrome most commonly occurring due to ischemic heart disease, causes significant morbidity and mortality. The benefits of revascularization versus medical treatment for ischemic heart failure remain controversial. Thus, we assessed a patient diagnosed with ischemic heart failure before and 3 months after coronary artery bypass grafting by myocardial radionuclide imaging. Findings of Tc-99m sestamibi myocardial perfusion imaging revealed that the degree and area of ischemia significantly were reduced, and the systolic function of the left ventricle improved compared with the preoperative value. This suggests the benefit of revascularization in cases of ischemic heart failure.

INTRODUCTION

In most developed countries, ischemic heart disease is the main cause of heart failure [Pagliaro 2020], causing significant morbidity and mortality. Treatment options for ischemic heart failure include drug therapy and revascularization. However, the benefit of revascularization remains controversial for ischemic heart failure patients. Thus, myocardial nuclide imaging may be used to assess the benefit of revascularization by providing valuable data on ventricular function, myocardial perfusion, viability, and intraventricular synchrony. Herein, we report the results of myocardial radionuclide imaging in a patient with ischemic heart failure before and 3 months after coronary artery bypass grafting (CABG).

CASE REPORT

A 67-year-old male patient was admitted to our hospital on August 19, 2021, with "chest tightness and chest pain after repeated activities" of two months' duration. The pain was accompanied by shortness of breath, without sweating, nausea, vomiting, or syncope, with each episode lasting for approximately 10 minutes and relieved by rest. The patient's complaint had worsened in the week before admission, and the chest pain was not relieved after rest. Physical examination results on admission were as follows: body temperature, 36°C; blood pressure, 115/80 mmHg (1 mmHg=0.133 kPa); respiration, 20 beats/min; pulse, 84 beats/min; and regular rhythm. The patient was conscious, but had a poor general condition. Breathing was clear in both lungs, and examination revealed that they were solid to percussion. Examination of the abdomen and lower limbs was unremarkable. Laboratory test results were as follows: troponin I level, 0.053 μg/L; creatine kinase level, 61 U/L; creatine kinase isoenzyme MB (mass) level, 1.3 μg/L; lactate dehydrogenase level, 207 U/L; lactate dehydrogenase isoenzyme level, 36.0 U/L, hydroxybutyrate dehydrogenase level, 140 U/L; and N-terminal pro-B-type natriuretic peptide level, 1950 ng/L. Additionally, electrolyte, liver and kidney function, stool, and urine analyses showed normal findings.

The patient's electrocardiogram (ECG) revealed ST-T changes, and coronary angiography findings were as follows: left main artery: 80% stenosis of the ostium; left anterior descending artery (LAD): calcified parts of long lesions in the proximal and middle segments, 90% stenosis at the narrowest part, 90% stenosis in the middle and distal segments, thicker diagonal branches in the middle segment, and 90% stenosis in the proximal segment of the opening; left circumflex artery (LCX): 80% stenosis of the opening and occlusion of the first blunt marginal branch; and right circumflex artery (RCA): small vessel with 90% stenosis in the proximal segment. Echocardiography revealed the following: 1. The inner diameters of the left atrium and left ventricle were enlarged, and the inner diameter of the ascending aorta was widened; 2. left ventricular wall motion was impaired; 3. the mitral valve was slightly insufficient (+ degree), and the aortic and tricuspid valves were slightly stenosed; 4. Left ventricular systolic and diastolic functions were impaired (ejection fraction [EF]: 30%, left ventricle: 63 mm). Chest radiography revealed an enlarged heart, bilateral pleural effusion, and evidence of possible cardiac insufficiency. Chest computed tomography revealed the following: 1. Lung findings: multiple interlobular septal thickenings in both lungs, scattered nodular and patchy ground-glass exudates, pulmonary edema, and bilateral pleural thickening; 2. Cardiac findings: enlarged atrioventricular chambers, multiple calcifications in the walls of

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the aorta and left and right coronary arteries, and widened main pulmonary artery; and 3. Multiple enlarged mediastinal lymph nodes.

Technetium 99m sestamibi (Tc-99m MIBI) myocardial perfusion imaging revealed an enlarged left ventricular cavity and right ventricular structural abnormalities. A small part of the lateral wall of the left ventricle (LCX blood supply area) and inferior wall near the apex (RCA blood supply area) showed evidence of mild to moderate myocardial ischemia; the area of mild to moderate ischemia was 13% of the total left ventricular area. The left ventricular apical segment, anterior wall (LAD supply area), most of the lateral wall (LCX supply area), mid-inferior wall, and inferior wall basal segment (RCA supply area) revealed evidence of severe myocardial ischemia or infarction; the area of severe ischemia accounted for approximately 30% of the total left ventricular area. The drug stress ECG test results were negative, and the overall left ventricular systolic function was extremely poor.

The patient was diagnosed with very high risk acute coronary syndrome with coronary atherosclerotic heart disease, heart failure, grade IV impaired cardiac function, grade III hypertension, type 2 diabetes, and bilateral pleural effusion. The patient received diuretics, coronary dilators, antiplatelets, angiotensin-converting enzyme inhibitors, β-blockers, lipid-lowering drugs, and other treatments for secondary prevention of coronary heart disease, blood pressure, and blood sugar control. Additionally, simple closed bilateral thoracic drainage was performed, and the patient's symptoms resolved. After 12 days, CABG was performed.

Surgical exploration revealed that the left ventricle was significantly enlarged, and the main coronary artery and three vessels were diseased; coronary artery disease was severe and diffuse, and the vascular condition was poor. Bypass grafting was performed for the right coronary aorta (8-0 prolene line), obtuse marginal branch-diagonal branch-aorta (sequential perfusion) (8-0 prolene line), and left internal mammary artery-anterior descending branch (8-0 prolene line). The intraoperative cardiopulmonary bypass time was 161 min; the aortic occlusion time was 113 min. The operation lasted for 6 h 20 min. The coronary flow meter measured the blood flow of the bridge vessels as follows: the anterior descending artery, 93.6 mL/min; the right coronary artery, 40.4 mL/min; and the sequential bridge, 33.2 mL/min. Postoperatively, the patient received intravenous dopamine 8.2 μg/kg/min, epinephrine 0.03 μg/kg/min, and norepinephrine 0.08 μg/kg/min combined with inotropes and vasopressor therapy. The central venous pressure fluctuated at 6–8 mmHg, and the endotracheal tube was removed 2 days postoperatively. Finally, the patient was discharged 13 days postoperatively. Patient follow-up 3 months after discharge revealed that the symptoms significantly had improved; the patient's cardiac function recovered to grade II, and the 6-min walk test was grade 4 (500 m). Echocardiography revealed that the left ventricle and left atrium volume increased; the basal part of the ventricular septum was thickened; the left ventricular wall motion was weakened, and mitral valve insufficiency, aortic insufficiency, and decreased ventricular diastolic and systolic function were observed. The left ventricular EF was 40%, and the left ventricle diameter was 59 mm. Chest radiography revealed no obvious abnormalities.

Tc-99m MIBI myocardial perfusion imaging (Figures 1 and 2) revealed that the left ventricular cavity was enlarged (significantly smaller than that preoperatively). (Figure 1) (Figure 2) Moreover, the right ventricle was visualized, indicating that the right heart was overloaded. The left ventricular lateral wall (LCX blood supply area) and inferior wall (RCA blood supply area) showed evidence of mild to moderate ischemia, and the ischemic area accounted for 30% of the left ventricular area. The ischemic area was significantly smaller than that before surgery, and the degree of ischemia was significantly reduced. The drug stress ECG test results were negative. The overall systolic function of the left ventricle was poor, and the systolic synchronization of each ventricular wall was poor but improved compared with the preoperative value.

DISCUSSION

The main cause of heart failure in more than 50% of patients in North America and Europe is ischemic cardiomyopathy [Pagliaro 2020], and ischemic heart failure has become a major threat for mankind. Despite the recommendation of drug treatments and revascularization therapy in the 2021 European Society of Cardiology guidelines [McDonagh 2021], there are limited data on the benefit of surgical treatment for ischemic heart failure versus medical treatment. For ischemic heart failure (EF ≤35%), the primary outcome of mortality was not significantly different between the CABG and MT groups at a median follow-up of 56 patients. However, in a randomized trial of 1,212 patients with ischemic

Table 1. Comparison of preoperative and 3 months after surgery of the area and degree of myocardial ischemia by Tc-99m MIBI myocardial perfusion imaging

	Area of mild-moderate ischemia		Area of severe ischemia
Preoperative	A small part of the lateral wall and inferior wall near the apex of the left ventricle	Left ventricular apical segment, anterior wall, most lateral walls, mid-inferior wall, and basal segment of inferior wall	Mild-moderate ischemic area 13%, severe ischemia 30%
3 months after surgery	Left ventricular lateral wall, inferior wall	-	The ischemic area of mild to moderate ischemia was approximately 30%

heart failure (EF ≤35%) by Velazquez et al. [Velazquez 2016], the median survival time of patients in the CABG group was 7.73 years, and the median survival time of patients in the MT group was 6.29 years. Thus, the median survival was 1.44 years longer in the CABG group. Moreover, Wolff et al. [Wolff 2017] systematically reviewed studies comparing revascularization and drug therapy in patients with EF ≤40%. Compared with drug treatment, there was a significant mortality reduction with CABG (hazard ratio, 0.66; 95% confidence interval, 0.61-0.72; $P < 0.001$).

The prognosis of CABG in patients with ischemic heart failure is significantly associated with the degree of myocardial viability [Neumann 2018], and currently, the benefits of CABG in patients with ischemic heart failure are controversial. We compared the myocardial viability of a patient before and 3 months after CABG to evaluate myocardial survival in patients with ischemic heart failure. (Table 1) Preoperative myocardial nuclide imaging in this case indicated that a small part of the lateral wall of the left ventricle and inferior wall near the apex of the myocardium had mild-to-moderate ischemia, and the area of mild-to-moderate ischemia accounted for approximately 13% of the total left ventricular area. The left ventricular apical segment, anterior wall, most of the lateral wall, middle segment of the inferior wall, and basal segment of the inferior wall showed severe myocardial ischemia, and the area of severe ischemia accounted for ~30% of the total area of the left ventricle. The myocardial radionuclide re-examination 3 months postoperatively indicated that the

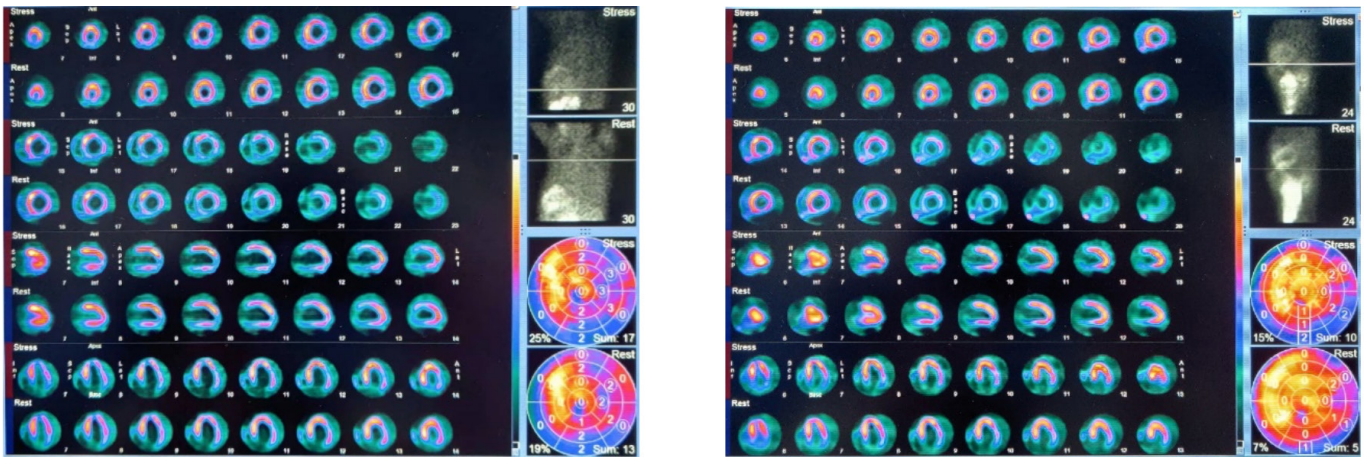

Figure 1. Myocardial imaging analysis of Tc-99m MIBI myocardial perfusion imaging before operation (A) and 3 months after operation (B).

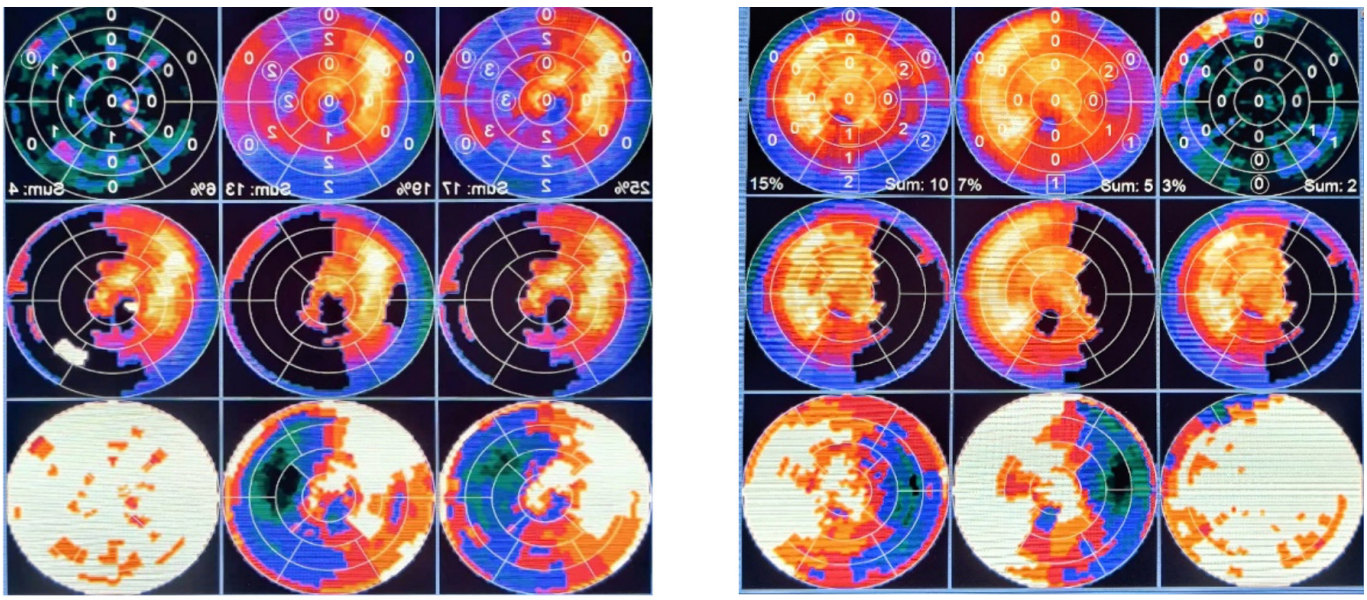

Figure 2. The degree and area of myocardial ischemia with Tc-99m sestamibi myocardial perfusion imaging before operation (A1) and 3 months after operation (B1).

left ventricular lateral wall and inferior wall showed "mild to moderate myocardial ischemia." The area and degree of myocardial ischemia after CABG improved significantly.

CONCLUSIONS

The degree of myocardial ischemia significantly improved after CABG, and the comparison of EF before surgery (30%) and 3 months after surgery (40%) by color Doppler ultrasound also confirmed that the patient's cardiac function was significantly improved compared with that before surgery. In terms of myocardial survival, we found that CABG was beneficial in improving myocardial ischemia in patients with ischemic heart failure. In the future, the benefit of CABG in patients with ischemic heart failure needs to be further confirmed by the evaluation of a large number of Tc-99m MIBI myocardial perfusion imaging ischemic areas and viable myocardium.

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