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AN ELDERLY CASE OF ISCHEMIC CARDIOMYOPATHY WITH SUCCESSFUL IMPROVEMENT IN LEFT VENTRICULAR FUNCTION AFTER PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

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Abstract: A 76-year-old woman was admitted to our hospital because of dyspnea and palpitation. A 12-lead EGG showed ventricular premature beats, but no evidence of old or new myocardial infarction. Chest X-ray showed pulmonary congestion and an increased cardio-thoracic ratio. Echocardiogram showed severe left ventricular chamber dilatation and diffuse hypokinesis of the left ventricular wall. The results of these noninvasive diagnostic procedures suggested dilated cardiomyopathy. On the twelfth hospital day, the patient developed chest pain with increased serum creatinine phosphokinase. Emergency cardiac catheterization was performed, and severe three-vessel disease was revealed. Staged PTCA was performed for all three vessels. One month after complete revascularization of the three vessels, the left ventriculography showed that left ventricular ejection fraction had improved to 49 % from 26 % at admission. In elderly patients with severe generalized poor left ventricular wall motion, in whom ischemic cardiomyopathy rather than dilated cardiomyopathy can be ruled out, PTCA should be considered as a treatment modality.

Index Terms

coronary angiography, ischemic cardiomyopathy, PTCA

Introduction

In 1970, Burch et al[1]. defined "ischemic cardiomyopathy", which has symptoms and a pathophysiology similar to those of dilated cardiomyopathy because of severe coronary artery disease. The prognosis of ischemic cardiomyopathy is very poor, and medical treatment is sometimes ineffective. We report a case of ischemic cardiomyopathy with severe three-vessel coronary artery disease in whicsh left ventricular function improved after staged percutaneous transluminal coronary angioplasty (PTCA) for complete revascularizatio of the diseased coronary arteries. We also review the differential diagnosis and treatment of ischemic cardiomyopathy.

Case report

A 76-year-old woman was admitted to our hospital because of dyspnea and a nine-month history of palpitation. There was no history of chest pain or oppression and no family history of cardiac disease. A 12-lead electrocardiogram (EGG) made at a private clinic showed ventricular extrasystole, but no evidence of myocardial infarction. Ten days before admission,

dyspnea began to occur regardless of rest or effort. One day before admission, a short run of ventricular extrasystole (PVC) was detected on EGG at the clinic. The following day, the patient was referred to our hospital. On admission, her height was 150 cm, body weight was 46.5 kg, supine blood pressure position was 150/70 mmHg, and pulse was irregular at 98 beats /min. On ausculation, holosystolic murmur (Levine 2/6) was heard at the apex, suggesting mitral valve regurgitation. Inspiratory crackles that were evidence of congestive heart failure were heard over both lung fields. An abdominal examination yielded normal results. There was peripheral edema, but no digital clubbing or cyanosis.

The results of laboratory examination were almost normal except for elevated serum lactic dehydeogenase (Table 1). Chest X-ray showed a cardiothoracic ratio of 60 % and bilateral pulmonary edema with bilateral pleural effusion (Fig. 1). An EGG demonstrated a short run of paroxysmal ventricular extrasystole and markedly depressed ST segment in leads V 5 and

Table 1. Laboratory data on admission

Urinalysis			γ-GTP	13	IU/l
Protein	(−)		ALP	149	IU/l
Glucose	(−)		AMY	57	IU/l
Occult blood	(−)		LDH	523	IU/l
			TP	6.5	g/dl
Hematology			Alb	3.9	g/dl
RBC	394×10^4	/μl	TC	191	mg/dl
WBC	5,600	/μl	TG	93	mg/dl
Ht	36.2	%	Scr	0.9	mg/dl
Hb	12.0	g/dl	BUN	14.8	mg/dl
PLT	16.5×10^4	/μl	UA	3.0	mg/dl
ESR	17	mm/h	K	4.0	mEq/l
			Cl	111	mEq/l
Biochemistry			Na	148	mEq/l
T-Bil	0.5	mg/dl			
GOT	40	IU/l	Serology		
GPT	35	IU/l	CRP	0.3	mg/dl

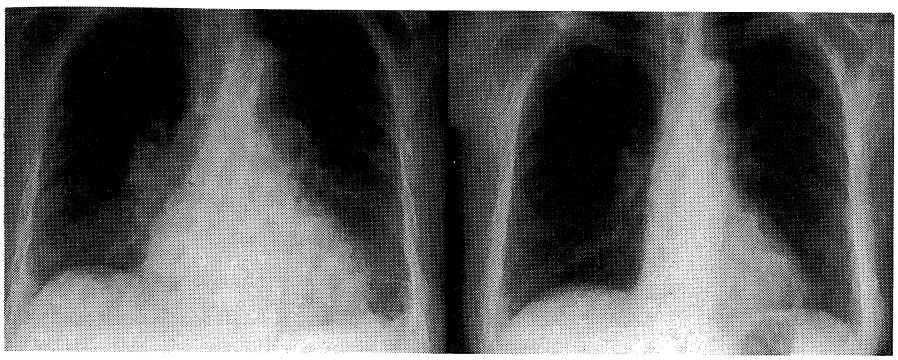

Fig. 1. Chest roentgenogram. A | B
Chest roentgenogram on admission showed cardiomegaly and bilateral pulmonary edema with bilateral pleural effusion (A). Chest roentgenogram after successful PTCA of three vessels showed decreased cardiothracic ratio and disappearing pulmonary edema (B).

V 6 (Fig. 2). A Holter EGG revealed ventricular extrasystoles (Lown grade IVb). An echocardiogram showed severe diffuse hypokinesis of the left ventricular wall, left ventricular wall thinning, dilatation of the left ventricular chamber, and moderate mitral regurgitation

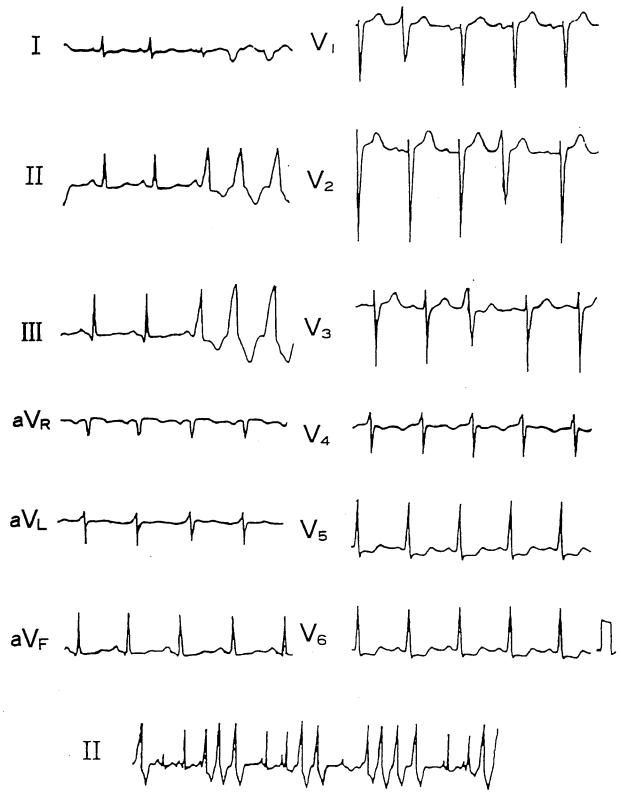

Fig. 2. Electrocardiogram on admission.
Electrocardiogram showed a short run of paroxysmal ventricular extrasystole and markedly depressed ST segment in leads V5 and V6.

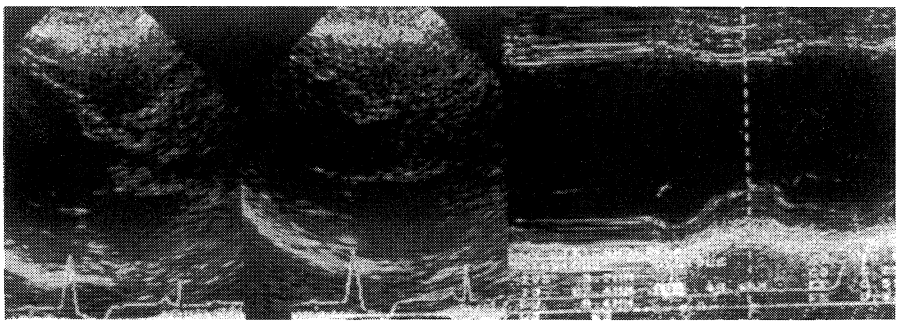

Fig. 3. Echocardiogram. A | B | C
Two-dimensional echocardiograms of end-diastole (A) and end-systole (B), and M-mode echocardiogram (C). These echocardiograms showed severe diffuse hypokinesis of the left ventricular wall, wall thinning, dilatation of the left ventricular chamber, and moderatc mitral regurgitation.

(Fig. 3).

As treatment for congestive heart failure and paroxysmal ventricular extrasystole, salt and water intake were restricted, and diuretics and nitroglycerin were administered. However, little improvement occurred. On the 12 th hospital day, the paitent complained of chest pain. An EGG showed unremarkable change, but serum creatinine phosphokinase (1,294 U/L) and CPK-MB (114 U/L) were elevated. Emergency catheterization was performed because of suspected acute myocardial infarction. Selective coronary arteriography showed that the right coronary artery was completely obstructed close to its origin and that there were considerable stenotic lesions with delayed filling in the left anterior descending artery (LAD) and the circumflex branches (LCX) of the left coronary artery. There was jeopardized collateral from the left coronary artery, which supplied the terminal branches of the right cronary artery (Fig. 4). A left ventricular angiogram showed a dilated chamber with generalized poor wall motion (Fig. 5). A Swan-Ganz thermodilution catheter was inserted to measure pulmonary capillary wedge pressure and cardiac output. Because of her age and very severe left ventricular dysfunction, we decided to perform coronary angioplasty rather than hypass surgery on the patient. Cardiac surgeons were alerted to the possibility of emergency surgery. Percutaneous

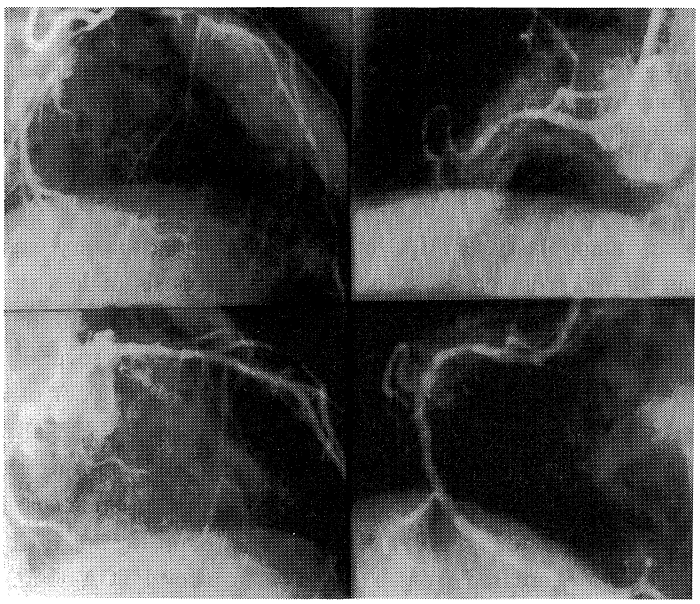

Fig. 4. Coronary angiograms. A | B / C | D

The right coronary artery before PTCA in LAO 60 view (A), the left coronary artery before PTCA in RAO 30 view (B), the right coronary artery after PTCA (C), and the left coronary artery after PTCA (D). Before PTCA, the right coronary artery was completely occluded close to its origin, and jeopardized collateral vessels were seen from the left coronary artery. There were also considerable stenoic lesions with flow-delay in the left anterior descending and circumflex branches of the left coronary artery. Complete coronary revascularization was obtained with PTCA.

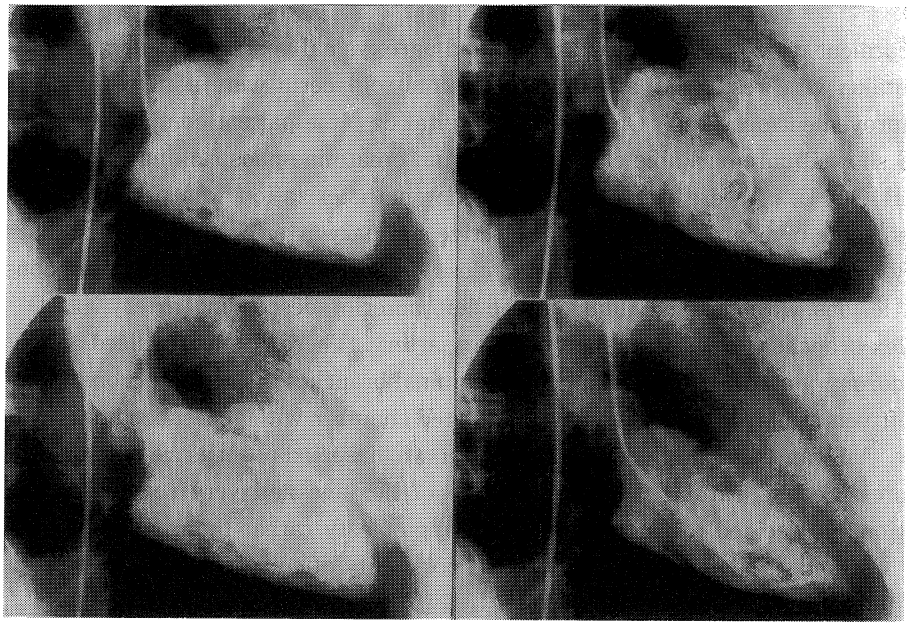

Fig. 5. Left ventriculogram in RAO view. A | B / C | D

Left ventriculogram of end-diastole before and after PTCA (A, B), and of end-systole before and after PTCA (C, D). Ejection fraction was 26%. One month after successful PTCA of all three vessels, LV wall motion improved with an ejection fraction of 49%, although severe hypokinesis of the left ventricular apex and posterior wall persisted.

transluminal coronary angioplasty (PTCA) of the right coronary artery was performed first, and the lumen was dilated from complete obstructio to stenosis of less than 25 %. Staged PTCA was successfully performed for the LAD and LCX on 9 and 37 days after the first procedure, respectively (Fig. 4). Before PTCA, LV wall motion was severely hypokinetic with an ejection fraction (EF) of 26 %. One month after successful PTCA of all three vessels, except for severe hypokinesis from the apex to the posterior wall, LV wall motion improved with an EF of 49 % (Fig. 5). The hemodynamics also improved from Forrester type III with a mean pulmonary capillary wedge pressure (PCWP) of 8 mmHg and a cardiac output index (CI) of 1.46 l/min/m to Forrester type I with a PCWP of 8 mmHg and a CI of 2.39 l/min/m. The cardio-thoracic ratio decreased from 60 % to 48 % after PTCA (Fig. 1). In addition, ventricular premature beats on Holter EGG improved from Lown IVb to II. A myocardial scintigram with I^{123}-BMIPP performed three months after PTCA showed a defect from the apex to the inferior wall. The patient was discharged without any symptoms, and remained asymptomatic during six months of follow-up.

Discussion

1. The differential diagnosis of this case

Coronary angiography revealed that our patient had severe left ventricular dysfunction due to three-vessel coronary artery diasease. Severe left-sided heart failure and arrhythimia were

also present in this case. In 1970, Burch et al[1,2]. reported a case of ischemic heart disease with severe left ventricular dysfunction mimicking dilated cardiomyopathy. It is important in selecting therapy to distinguish ischemic cardiomyopathy from dilated cardiomyopathy, becase reperfusion in ischemic cardiomyopathy sometimes improves left ventricular dysfunction. However, noninvasive diagnostic techniques are still insufficient for a differential diagnosis of these disease, so a coronary arteriogram is needed for the final diagnosis. Our tentative diagnosis of our patient at admission was actually dilated cardiomyopathy because of severe generalized hypokinesis of the left ventricular wall, left ventricular dilatation, and thinning of the left ventricular wall on echocardiography. Nakamura et al[3]. reported about the differential diagnosis of ischemic cardimyopathy and dilated cardiomyopathy, and listed the characteristic echocardiographic features of ischemic cardiomyopathy such as local wall thinness, unequal local wall motion, and increased local echo-intensity. They suggested the possibility of using echocardiography to distinguish between these two diseases. Iskandrian et al[4]. showed the importance of preserved right ventricular function to distinguish ishemic cardiomyopathy from dilated cardiomyopathy. Recently, it was noted that ischemic cardiomyopathy is related to the hibernating myocardium[5]. Positron emission tomography (PET) is considered to be a possible tool for identifying the ischemic cardiomyopathy[6,7]. However, PET is not available at every hospital, and it is expensive. From the point of view of clinical availability, recent reports indicate that low-dose dobutamine stress echocardiography is useful for detecting the hibemating myocardium[8,9]. Thus, there is still controversy about which noninvasive tecnnique best disinguishes ishchemic cardiomyopathy from dilated cardiomyopathy. In our patient, we made a diagnosis of ischemic cardiomyopathy based on coronary angiography before other noninvasive examinations, such as a dobutamine stress echocardiogram, were performed.

2. PTCA for severe three-vessel disease

Severe three-vessel coronary artery diasease is not always an indication for PTCA, coronary artery bypass grafting (CABG) may be required. However, in a patient with poor ventricular wall motion, such as ejection fraction of less than 25 %, and age more than 75 years old, CABG is a high risk procedure, and PTCA might be more appropriate. In our patient, PTCA improved left ventricular dysfunction because of the hibernating myocardium, which had severe stenoses of the left coronary arteries and jeopardized collateral circulation to the right coronary artery from the left coronary arteries. Recent reports on PTCA for chronic total occluded coronary artery disease showed the relative usefulnes of this procedure for the improvement of left ventricular function, compared to therapy without PTCA[10,11,12]. In our patient, not only the occluded right coronary artery but also the other two stenotic lesions were revascularized after staged PTCA. In the end, the three-vessel were completely revasculized on three months after admission, and left ventricular function was observed to improve. With respect to improvement of left ventricular function after PTCA, increased amplitude of R wave on ECG, no delay in coronary angiography, and the existence of post-infarction angina pectoris appear to be more predictive than TI-201 cardiac scintigram[13]. In our patient, these three predictive clinical factors were not found before PTCA.

The high prevalance of restenosis after PTCA for three-vessel disease raises questions about the ideal therapy for this syndrome. Further study will be necessary to overcome the diffi-

culties in diagnosing and treating ischemic cardiomyopathy.

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