DISAPPEARANCE OF MITRAL VALVE REGURGITATION AFTER SUCCESSFUL PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

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Percutaneous transluminal coronary angioplasty has been reported to improve several clinical parameters. Functional papillary muscle dysfunction, which is also known to induce mitral valve regurgitation, is reversible after revascularization. We described a patient, with a 95% stenosis of proximal right coronary artery, whose mitral valve regurgitation disappeared after successful percutaneous transluminal coronary angioplasty.

PERCUTANEOUS transluminal coronary angioplasty was introduced by Gruentzig in 1977! has been reported to improve several clinical parameters2-4 and is now widely used for revascularization in cases with coronary arteriosclerotic stenotic lesions. The posterior papillary muscle has a predominantly single blood supply from the posterior descending coronary artery and its terminal branches. Rupture of this muscle is reported 6 to 12 times more frequently than that of the anterolateral papillary muscle, which has a dual blood supply from the left anterior descending and circumflex vessels. Disturbance in coronary blood flow may result in dysfunction or even necrosis of the papillary muscle, resulting in mitral valve regurgitation.

CASE REPORT

A 57-year-old man was awakened from sleep due to chest pain, and was admitted to a local hospital. The electrocardiogram showed sinus rhythm, normal axis deviation, complete right bundle branch block, no sign of left ventricular hypertrophy and no abnormal ST-T changes. His symptoms were relieved after taking 2 tablets of nitroglycerin sublingually. He was referred to our hospital for through evaluation of the angina pectoris. On admission, the heart rate was regular, about 80 beats per min, blood pressure was 120/60 mmHg. The chest was clear to auscultation. A 3/6 degree systolic ejection murmur was detected in the left parasternal intercostal IV. The electrocardiogram showed sinus rhythm, normal axis deviation, no sign of left ventricular hypertrophy and no abnormal ST-T changes. There was no enzymatic evidence of significant myocardial necrosis. Exercise electrocardiography was performed on a treadmill according to the modification of the standard Bruce protocol. The test was terminated at 18 min 30 sec when the patient was becoming fatigued. ST-T changes were unclear due to complete right bundle branch block. A color doppler echocardiogram revealed second degree

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Fig. 1. (A) The color doppler echocardiogram in parasternal long axis view before angioplasty showed a second degree mitral regurgitation. (B) The coronary angiogram in LAO view showed a 95% stenosis of the proximal right coronary artery with delayed filling. (C) The left ventriculogram before angioplasty in RAO view during ventricular systole and diastole (Upper and lower panel). The left ventriculogram showed the entire left atrium was opacified by contrast material during ventricular systole (arrows).
mitral valve regurgitation (Fig. 1A). Coronary angiography and left ventriculography were performed in the cardiac catheterization laboratory. The coronary angiogram showed a 95% stenosis of the proximal right coronary artery (Fig. 1B). There was also

Fig. 2. (A) No regurgitation can be observed in the color doppler echocardiogram after angioplasty. (B) The proximal right coronary artery showed a 50% stenosis (arrows) and (C) no regurgitation can be observed in the left ventriculogram after angioplasty in the RAO view during ventricular systole (upper panel).
50% stenosis of the mid left anterior descending coronary artery and obtuse marginal branch of the left circumflex coronary artery, with collateral blood vessels from left circumflex coronary artery to the right coronary artery observable. The left ventriculogram on ectopic free injections showed third degree mitral valve regurgitation with wall motion reduced at the apical, diaphragmatic and posterobasal segments (Fig. 1C). Thallium-201 single photon emission computed tomography showed reduced activity after exercise in the inferoposterior region with redistribution at the delayed image. Radionuclide left ventriculography showed reduced wall motion in the posterior wall with an ejection fraction of 41.6%. Which increased to 50.2% during exercise testing. Approximately 1 month after admission, PTCA was performed successfully, according to the procedure initially described by Gruentzig et al, using a Hartzler angioplasty catheter (Advance Catheter Systems), with the right coronary artery stenosis reduced to 50%. We routinely evaluate the results of PTCA with a thallium-201 single photon emission computed tomogram, radionuclide left ventriculogram, Double Master exercise test, ergometer bicycle exercise test and treadmill test, 1 week after PTCA and 3–6 months later when the patient is re-admitted for a follow-up coronary angiogram.

To detect possible early restenosis, the patient was readmitted to the hospital 3 months later for complete reevaluation including cardiac catheterization, No evidence of electrocardiographic change and no systolic ejection murmur were detected. The thallium-201 scintigram, radionuclide left ventriculogram and treadmill test also demonstrated considerable improvement. No mitral valve regurgitation was detected by color doppler echocardiography (Fig. 2A). The left ventriculogram on ectopic free injections showed improvement of the wall motion at the apical, diaphragmatic and posterobasal segments. No progression in the coronary arteries nor mitral valve regurgitation were observed (Fig. 2B, 2C).

DISCUSSION

We described a patient with ischemic heart disease which led to functional mitral regurgitation due to papillary muscle dysfunction. The patient was suffering from angina pectoris due to 95% stenosis of the proximal right coronary artery, with a reduction in the motion of the posterobasal region of the left ventricle clearly observable. This abnormality, which was compatible with other non-invasive methods, was considered to be due to ischemic myocardium. In this patient, there was no evidence of myocardial infarction, cardiomegaly or change in the cardiothoracic ration. The disappearance of the mitral regurgitation after successful revascularization was suggested to be due to improved papillary muscle function and improved motion of the diaphragmatic and posterobasal segments of the left ventricular wall. We documented some studies which reported an improvement in papillary muscle function leading to a reduction in the degree of mitral regurgitation after revascularization. Therefore, in cases of ischemic heart disease exhibiting a combination of critical ventricular dysfunction and mitral regurgitation, we suggest that by improving the coronary blood flow by conventional and/or interventional therapy, mitral regurgitation and ventricular dysfunction may also be ameliorated.

REFERENCES

