Clinical Utility of Echocardiographic Hemodynamic Monitoring during Manual Compression of Arteriovenous Shunt in a Patient with High-Output Heart Failure

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INTRODUCTION

An arteriovenous fistula for hemodialysis increases cardiac output by decreasing systemic vascular resistance and occasionally causes high-output heart failure in patients receiving chronic hemodialysis.[1] Although some studies have tested the hemodynamic influence of shunt flow by using noninvasive methods,[2,3] a robust strategy for the assessment remains to be established. We present the case of a woman who underwent kidney transplantation with arteriovenous fistula in whom echocardiographic assessment played an important role in the management of refractory heart failure.

CASE PRESENTATION

A 67-year-old woman with a history of kidney transplantation for polycystic kidney disease for 9 years was admitted to the cardiology section for worsening heart failure. She had been diagnosed with chronic heart failure due to hypertensive heart disease and hospitalized for worsening of symptoms three times within 1 year. Coronary artery disease had been ruled out by coronary angiography 1 year before admission. On admission, the patient's blood pressure was 134/46 mm Hg, pulse rate was 62 beats/min, and body temperature was 37.1°C. A clinical examination revealed jugular vein distension, third heart sound, and late-inspiratory fine crackles at the lower lung field. She had a brachiobasilic arteriovenous fistula in her left lower arm, which had been created for hemodialysis 13 years prior. The patient denied any signs of limb ischemia due to a steal phenomenon caused by the fistula. Chest radiography showed lung congestion with apparent cardiomegaly (Figure 1). Her estimated glomerular filtration rate was 42.6 mL/min, and her plasma brain-type natriuretic peptide level was 1,138.8 pg/mL. Transthoracic echocardiography (Figure 2, Video 1) showed a mildly enlarged but hyperkinetic left ventricle with hypertrophy, a severely enlarged left atrium, mild mitral regurgitation, restrictive left ventricular (LV) filling pattern, and severe pulmonary hypertension, suggesting elevated LV filling pressure due to a hypertrophied left ventricle. Intravenous nitroglycerin and furosemide were started in addition to oral administration of furosemide (100 mg/d), tolvaptan (15 mg/d), amlodipine (10 mg/d), olmesartan (40 mg/d), and doxazosin (2 mg/d), which had already been administered before the worsening. After admission, her symptoms were gradually relieved, and chest radiography confirmed the improvement of the lung congestion, resulting in termination of intravenous furosemide and nitroglycerin on the ninth day. However, soon after their discontinuation, lung congestion recurred, and intravenous medication was resumed and continued thereafter. To determine the contribution of the shunt flow via the arteriovenous fistula to heart failure, echocardiography was performed during its manual compression, which revealed decreases in LV end-diastolic dimension from 62 to 57 mm, in cardiac output from 5.2 to 4.5 L/min, in pulmonary artery systolic pressure from 76 to 53 mm Hg, and in the ratio of transmitral early diastolic to late diastolic flow velocity from 4.30 to 2.76 after 10 min of occlusion, without significant change in mitral regurgitation, suggesting reduced LV preload and decrease in the filling pressure (Figure 3, Videos 2 and 3). To further confirm these echocardiographic findings, invasive hemodynamic monitoring was repeated during the same maneuvers. Similarly to the echocardiographic study, pulmonary artery wedge pressure with large v waves and pulmonary artery pressures were dramatically decreased (Figure 4). Cardiac output estimated using Fick's principle decreased from 7.1 to 5.5 L/min, indicating 1.6 L/min of the shunt flow. On the basis of this dramatic improvement in hemodynamics by manual compression, surgical closure of arteriovenous fistula was performed. Immediately afterward, her symptoms diminished, and intravenous medication was discontinued without any sign of recurrence. The patient was discharged on the 40th day. Echocardiography 6 months after discharge showed a reduction of LV size, which revealed prominent apical hypertrophy, and a slight decrease in tricuspid regurgitation within the grade of mild, along with decrease in LV filling pressure suggested by transmitral flow pattern and tricuspid regurgitation velocity (Figure 5, Video 4).

DISCUSSION

Increased cardiac output caused by arteriovenous fistula occasionally results in symptomatic heart failure in hemodialysis patients,[1] but the exact incidence is still unknown.[2] Although the fistula is thought to be unnecessary in patients after kidney transplantation with stable graft function, there is still controversy as to whether to close the well-functioning fistula in these patients,[3] partly because of the difficulties in assessing how much it affects hemodynamics.

In this particular patient, echocardiographic evaluation played an important role in the diagnosis and decision of therapeutic strategy. First, the initial echocardiographic findings showed an enlarged and

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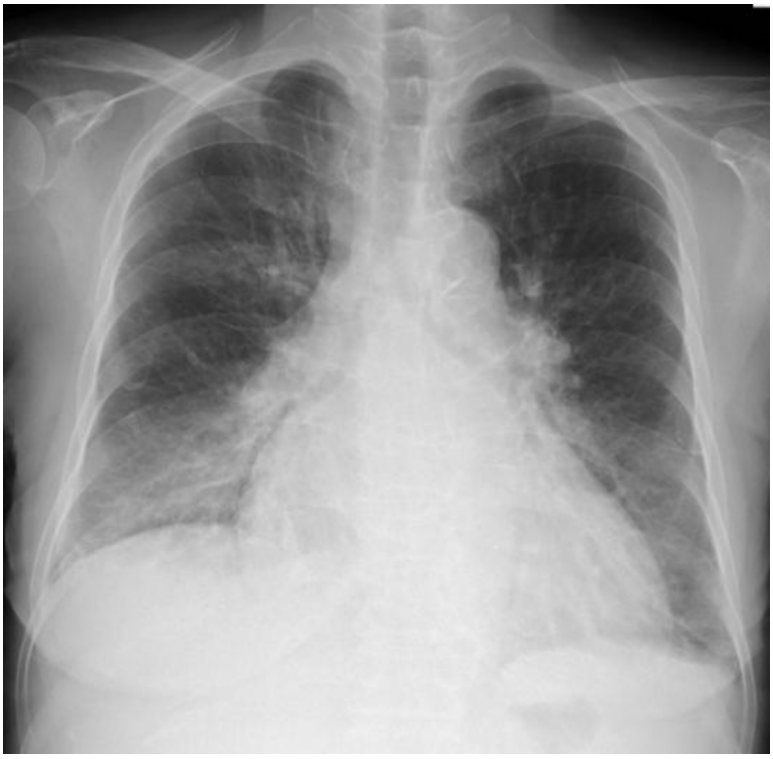

Figure 1 Chest radiography at admission, showing lung congestion with apparent cardiomegaly.

hyperkinetic left ventricle, which was different from the usual manifestation of patients with heart failure with preserved ejection fraction, smaller LV volume, and cardiac output.[4] This motivated the physicians to consider whether the arteriovenous shunt could cause the high-output heart failure. Second and more important, the noninvasive observation of hemodynamics by echocardiography during manual occlusion of the fistula suggested a decrease in LV filling pressure as well as preload and cardiac output. This finding led to testing for further invasive confirmation. Echocardiographic observation of cardiac output during fistula occlusion has been reported to be one of the noninvasive methods used to assess the hemodynamic effects of arteriovenous fistula.[2,3] Our case is the first in which changes in hemodynamics have been observed comprehensively, including not only cardiac output and preload changes but also changes in LV filling pressure. Because heart failure generally occurs in a situation in which a load over the cardiac capacity exists for a certain duration, hemodynamics in patients with arteriovenous fistulas depend on both of the amount of shunt flow and the degree of LV diastolic dysfunction. On the basis of the absence of signs of limb ischemia as well as the estimated shunt flow, the amount of shunt flow was not considerably large. Nevertheless, the patient demonstrated refractory heart failure. This suggested that "moderate" flow volume through the fistula was large enough to influence the patient's hemodynamics in whom LV diastolic function was severely impaired. On the basis of the prominent apical hypertrophy after the shrinkage of the left ventricle, underlying hypertrophic cardiomyopathy might have been the cause of this LV diastolic dysfunction, which could not be ruled out from the obtained medical record. Little has been reported regarding hemodynamic changes after closure of arteriovenous fistulas in patients after kidney transplantation. This case reports useful and unique findings suggesting that comprehensive hemodynamic monitoring by echocardiography during temporary occlusion of arteriovenous fistulas could be a useful strategy to evaluate the influence of the shunt flow on LV filling pressures, especially in patients with LV diastolic dysfunction.

CONCLUSIONS

We report a case of refractory heart failure in which echocardiographic evaluation played an important role for the assessment of hemodynamic influence of the arteriovenous fistula. Echocardiography can provide useful information on the hemodynamic changes during manual occlusion of arteriovenous fistulas, leading to successful management of heart failure. Future study is needed to test hemodynamic changes before and after the creation or ligation of arteriovenous fistula in dialysis patients.

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SUPPLEMENTARY DATA

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