Improvement of Severe Heart Failure After Endovascular Stent Grafting for Thoracic Aortic Aneurysm

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Summary

Afterload is considered to be an important factor regulating heart failure. Aortic structure or pathology may affect afterload to various extents. However, the contribution of aortic diseases, such as aortic aneurysm or aortic dissection, to heart failure status has not been completely elucidated.

Here we describe a 78-year-old patient with severe heart failure who made a dramatic recovery from cardiac decompensation following endovascular thoracic aortic aneurysm surgery. He previously underwent graft replacement for impending rupture of the descending aorta and replacement of both the mitral valve and aortic valve to address valve regurgitation. Subsequently, his left ventricular (LV) function became severely depressed (13%) and serum brain natriuretic peptide (BNP) level remained high (approximately 880–3520 pg/mL). Conversely, his aortic arch was dilated to 70 mm and required surgical intervention. Despite his extremely high vascular surgery risk due to severely depressed cardiac function, stent grafting for thoracic aortic aneurysm was successfully performed. Furthermore, the severity of his depressed cardiac function and heart failure dramatically improved following stent grafting. The left ventricular ejection fraction improved from 13% presurgery to 55% postsurgery and the serum BNP level had significantly decreased to 70–240 pg/mL. These improvements helped to alleviate the patient’s heart failure symptoms, including shortness of breath.

This case suggests a possible beneficial effect of aortic aneurysm repair for improving cardiac function and heart failure; our study presents a new concept of another extrinsic factor that can affect cardiac function through modulation of afterload. (Int Heart J 2015; 56: 682-685)

Key words: Cardiac insufficiency, Aortic aneurysm repair, Afterload, Hemodynamic disturbance

Heart failure is a serious global problem, with over 30 million patients diagnosed worldwide. Survival with chronic heart failure has improved with the widespread use of pharmacotherapy, including β-adrenoreceptor blockers and agents that block the renin–angiotensin–aldosterone system, and with some cardiac devices including implantable cardiac defibrillators. These interventions modulate cardiac function itself, resulting in heart failure improvements. However, with some intractable cases, sufficient improvement cannot be achieved with these modalities, and mechanical support or transplantation is required. On the other hand, modulating extrinsic factors can also have a marked impact on the condition and symptoms of heart failure. When developing such interventions, it is of great importance to establish an association between cardiac function and the extrinsic factor.

Aortic disease progresses with aging through atherosclerotic mechanisms. This aortic disease is often complicated with cardiac problems such as ischemic heart disease or heart failure. The presence of heart failure significantly increases the risks associated with surgical intervention to address aortic diseases. For example, preoperative BNP levels can be used to independently predict the likelihood of cardiovascular events after vascular surgery. However, the exact association between heart failure and aortic disease has not been completely established. Decisions on whether patients with high BNP levels should avoid vascular surgery because of high operative risk are complicated.

Here we report the dramatic case of a 78-year-old man with advanced heart failure, who underwent successful endovascular surgery to treat a thoracic aortic aneurysm (TAA), resulting in remarkable improvement in both severely impaired left ventricular (LV) function and intractable symptoms of heart failure.

Case Report

A 78-year-old man was hospitalized for endovascular operation to address TAA. At the age of 76, he had undergone descending aortic reconstructive surgery with a synthetic graft because of impending rupture. After his previous surgery, he gradually developed pitting edema in the lower legs and dyspnea on exertion. Finally, he was diagnosed with heart failure 1
year after the operation. Echocardiography revealed diffuse LV wall hypokinesis [LV end-diastolic and end-systolic dimensions (LVDd/Ds): 55/46 mm, LV ejection fraction (LVEF): 34%] with severe mitral regurgitation, moderate aortic regurgitation, and moderate tricuspid regurgitation. Cardiac catheterization demonstrated no significant stenosis of the coronary arteries, an increased mean pulmonary capillary wedge pressure (29 mmHg), and a decreased cardiac index (2.12 L/minute/ m²). His serum BNP level was 1331 pg/mL on admission. His heart failure was considered to be the result of a valvular disorder and aortic and mitral valve replacement with tricuspid valveoplasty was performed. At that time, an aortic arch partial replacement was attached because of regional aortic arch expansion observed on a preoperative computed tomography (CT) scan. The aneurysm was not totally treated because of a surrounding massive perivascular thrombosis at the distal part of the aortic arch. Temporal, continuous hemodiafiltration was required after the operation due to the chronic kidney disease.

Early postoperative echocardiography showed no signs of prosthetic valve dysfunction. The patient was discharged after a 1-month rehabilitation program; however, his heart failure symptoms remained and gradually worsened. The patient’s LV function was severely depressed (LVEF 13%) on echocardiography, and BNP level continued to increase up to 3521 pg/mL. Medical therapy, including bisoprolol (0.625 mg/day), perindopril (2 mg/day), furosemide (60 mg/day), spironolactone (25 mg/day), and pimobendan (2.5 mg/day), was not effective. On the other hand, his aortic arch aneurysm dilated up to 70 mm and required surgical intervention.

Because his operative risk was extremely high because of severely impaired LV function, endovascular intervention using a stent graft was performed (Figure 1). Unexpectedly, his postoperative clinical course was uneventful and he was discharged without marked complications. Furthermore, his severely depressed cardiac function showed a remarkable recovery when compared with the preoperative period. His LV function improved up to the normal range (LVEF 55%) (Table I), and serum BNP concentration markedly decreased from more than approximately 1000 pg/mL to under 200 pg/mL (Figure 2), resulting in a decrease in his diuretic requirements (furosemide decreased from 60 mg/day to 20 mg/day). His heart failure symptoms were also alleviated, and during the 11 months of follow-up he had no additional cardiovascular events.

**Discussion**

We have described the case of a man with advanced heart
failure who had undergone successful endovascular surgery for treatment of TAA, resulting in remarkable improvement of impaired LV function and refractory symptoms of heart failure.

The relationship between aortic structure abnormalities and heart failure has been described in a few published reports. Aortic stenotic lesions are expected to contribute to an increased afterload, leading to a negative impact on cardiac function. However, it has also been stated that nonstenotic involvement of the aorta could impact cardiac function. Lam, et al reported that an age-associated increase in aortic root diameter was associated with afterload changes resulting in cardiac remodeling and the development of heart failure. Aortic stiffness was also reported to enhance heart failure risk because the pulse wave travels faster, such that the reflected wave returns to the heart in late systole, increasing late afterload. Through these mechanisms, the magnitude of afterload was enhanced by various extrinsic factors other than blood pressure. This case suggests that other hemodynamic modulating factors derived from an aortic aneurysmal lesion can affect cardiac function and modify the conditions of heart failure.

The mechanism of hemodynamic parameter improvement after aortic aneurysm repair is unknown; however, Sugimoto, et al suggested a new perspective on blood flow dynamic improvement with aneurysm repair by introducing the concept of energy loss through altered blood flow with an aortic aneurysm. Flow turbulence reduces the efficiency of blood delivery around the dilated aortic lesion, causing local viscous energy loss and enhancing the systemic circulation afterload. The importance of the relationship between hemodynamics and energy loss on the aneurysmal aorta has been reported. Dilated aortic grafting ameliorates hemodynamic disturbances caused by flow disturbance and favorably impacts cardiac function. In the present case, previously depressed cardiac function secondary to valvular disease may render the patient more susceptible to the adverse effects of aneurysmal burden. These conditions may magnify the favorable effects of aortic aneurysm repair on cardiac function and may have a more minor impact in other cases.

Indeed, Cuypers, et al reported that hemodynamic parameters, including cardiac index and stroke volume, were improved after aortic aneurysm repair with both open and endovascular surgery. Our case and this previous report suggest the hypothesis that the presence of an aortic aneurysm has an adverse effect on LV function and that therapeutic intervention for aortic aneurysm ameliorates the LV functional deficit. To verify this hypothesis, we investigated changes in cardiac function for patients at our hospital who underwent aortic aneurysm repair by comparing cardiac function before and after the intervention (Table II). Ten patients were recruited for this analysis. We found that 8 of the 10 patients showed LVEF improvement on echocardiography after aortic aneurysm repair and the mean value of LVEF significantly increased from 64.9 ± 5.8% to 72.3 ± 5.6% perioperatively (P < 0.05) (Figure 3).

In conclusion, the presence of an aortic aneurysm has a substantial negative impact on cardiac function. Aortic aneurysm repair is a reasonable option for patients with aortic aneurysm exacerbating heart failure.

**Table II. Characteristics of 10 Patients With Aortic Aneurysm**

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OS indicates open surgery; EVAR, endovascular aortic repair; TAA, thoracic aortic aneurysm; AAA, abdominal aortic aneurysm; LVDd, left ventricular end-diastolic diameter; LVDs, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; and SV, stroke volume.

**DISCLOSURE**

**Conflicts of interest:** The authors declare no conflicts of interest.
REFERENCES