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Case Report

A successful case of percutaneous fistula closure in a patient with high output heart failure and extracardiac arteriovenous fistula

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ABSTRACT

Intra-cardiac shunt diseases may cause chronic ventricular volume overload, but extra-cardiac fistula could also cause high-output heart failure (HF).
A patient presented with high-output HF and significant extra-cardiac shunt flow. Although the size and shape of the patient's left ventricle suggested dilated cardiomyopathy, considerable origins were not identified except for a high-flow fistula between the right subclavian artery and right internal jugular vein. Right heart catheter examination revealed inappropriately high cardiac output. Left-to-right shunt ratio was calculated at 40.3% from an oximetry run, under the assumption that the left anonymous vein which was not contaminated with any shunt flow could be substituted for venous return from the upper body. We could determine the indication of fistula closure according to the estimated high left-to-right shunt ratio, reducing cardiac output by 42.7% which was similar to the pre-estimated left-to-right shunt ratio. Two months later, the patient's serum B-type natriuretic peptide level and left ventricular end-diastolic and end-systolic diameters were decreased.
The proposed method to estimate the left-to-right shunt ratio was useful in determining the indication for fistula closure in a patient with HF and a significant shunt fistula.
<**Learning objective:** A significant extracardiac left-to-right shunt more than 30–35% could cause heart failure with left ventricular dilatation similar to dilated cardiomyopathy. One can decide fistula closure with the finding of left-to-right shunt ratio calculated using a transcatheter oximetry run. It is mandatory that the venous oxygen saturation at the upper stream of fistula is determined precisely without the contamination of arterial blood. In this case, the oxygen saturation at the left anonymous vein substituted for it.>

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Introduction

The etiologies and pathophysiological origins of dilated cardiomyopathy are highly diverse [1]. While congenital intra-cardiac shunt diseases, such as patent ductus arteriosus, could cause chronic ventricular volume overload leading to left ventricular (LV) dilatation, extra-cardiac forearm arteriovenous shuts for hemodialysis could also be an origin of high-flow heart failure with LV dilatation [2,3]. Over 30–35% of the shunt flow volume/cardiac output (SFV/CO) ratio in patients on hemodialysis could cause high-output heart failure [3]. Flow volumes of forearm fistulas can be evaluated by Doppler ultrasound and finger pressure measurement [4,5], and blood flow suppression therapy is known to be effective in patients with high-output heart failure due to a high SFV/CO ratio [2,6]. However, a patient not on dialysis with clinically significant extra-cardiac left-to-right shunts is rarely observed; hence, a definitive method for evaluating the shunt ratio has not been reported nor has an indication for stunt closure been established.

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A patient with heart failure presented with significant left-to-right shunt between the right subclavian artery and right internal jugular vein. Here, we report the method used to calculate the left-to-right shunt ratio and how we treated this patient.

Case report

A woman in her seventies with exertional dyspnea that had worsened over the past few months was referred to Nagoya City University Hospital, after initial treatment for acute heart failure with pulmonary congestion at the previous hospital, with which we could expect left atrial pressure was elevated.

At the visit to our hospital, her symptoms and hemodynamics were well controlled to "compensated" with 30 mg of azosemide. Her blood pressure was 134/63 mmHg, heart rate was 95 bpm, SPO2 was 95% in room air, and respiratory rate was 19/min. During the physical examination, a systolic murmur was heard at the apex, and a continuous murmur was heard in the right subclavian area. Blood tests showed elevated B-type natriuretic peptide level of 300 pg/mL, and chest X-ray revealed an increased cardiothoracic ratio (58%). Transthoracic echocardiography showed apparent LV dilatation and moderately reduced LV systolic function as presented in Table 1, indicating a dilated cardiomyopathy (DCM)-like heart.

Although we searched for the cause of the LV dysfunction, secondary cardiomyopathy and coronary artery disease were dismissed by coronary angiography, ^{67}Ga scintigraphy, gadolinium-enhanced cardiac magnetic resonance imaging, and myocardial biopsy.

Then, we examined the cause of the continuous murmur in the right subclavian area by vascular ultrasound imaging. A shunt flow was found between the right subclavian artery and right internal jugular vein (Fig. 1A). Additional medical interviews brought forth information about the patient's past history and that she had tried to commit suicide by stabbing herself in the right chest with a knife 11 years previously. The shunt due to arteriovenous fistula made by the knife was not noticed at that time.

Table 1 Hemodynamics, echocardiographic, laboratory, and chest X-ray data.

	Before treatment	Just before procedure	After procedure
Hemodynamics			
Mean PCWP, mmHg	4	4	2
Mean PAP, mmHg	13	11	7
Mean RAP, mmHg	1	2	1
LVEDP, mmHg	10	8	5
AoP, mmHg		140/50	138/50
CO, L/min	6.03	6.25	3.58
CI, L/min/m^2	4.52	4.88	2.80
Echocardiographic data			
LVDd, mm	69		49
LVDs, mm	50		42
LVEF, %	39		42
LAD, mm	53		48
MR,	Moderate		Mild
Laboratory data			
BNP, pg/mL	300		78
Chest X-ray			
Cardiothoracic ratio, %58			49

PCWP, pulmonary capillary wedged pressure; PAP, pulmonary artery pressure; RAP, right atrial pressure; LVEDP, left ventricular end-diastolic pressure; AoP, aortic pressure; CO, cardiac output; CI, cardiac index; LVDd, left ventricular end-diastolic diameter; LVD, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; LAD, left atrial diameter; MR, mitral regurgitation; BNP, brain natriuretic peptide.

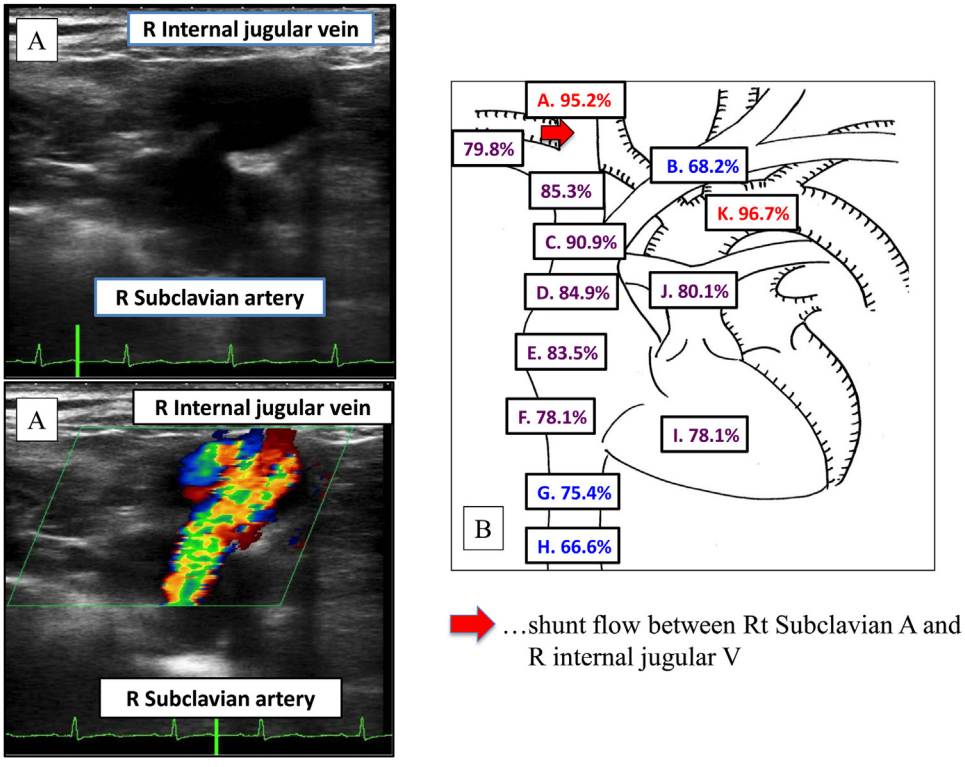

Fig. 1. (A) Vascular ultrasound imaging showing the amount of shunt flow from the right subclavian artery to the right internal jugular vein. (B) Map of the oximetry run during the catheter examination, presenting oxygen saturation in each vessel. (A) Right internal jugular vein; (B) left anonymous vein; (C) superior vena cava; (D) high right atrium; (E) middle right atrium; (F) low right atrium; (G) high inferior vena cava; (H) low inferior vena cava; (I) right ventricle; (J) main pulmonary artery; (K) ascending aorta. High oxygen saturation was observed in the right internal jugular vein.

Right heart catheterization showed a compensated cardiac pressure condition as presented in Table 1, while the patient's cardiac output was 6.03 L/min (cardiac index, 4.52 L/min/m^2), which seemed inappropriately high with regard to her cardiac performance. Oximetry run revealed a significantly elevated oxygen saturation level of 95.2% at the right internal jugular vein where shunt flow was provided from the right subclavian artery (Fig. 1B). We assumed that the left anonymous vein, which was not contaminated with any shunt flow, was substituted for venous return from the upper body, while the mean of saturation levels of upper and lower inferior vena cava was used for the saturation of venous return from the lower body as usual. If the venous return volume ratio between the upper and lower body was assumed to be 3:1, the estimated mixed venous oxygen saturation was calculated as 68.9% using the following formula: S(mixed venous)O_2 = [3 × S (upper body)O_2 + S(lower body)O_2]/4 [7]. Furthermore, using this estimated mixed vein oxygen saturation, we calculated the left-to-right shunt ratio at 40.3% using the following formula: [left-to-right shunt ratio = ($SPAO_2$ − S(mixed venous)O_2)/($SLAO_2$(= $SAoO_2$) − S (mixed venous)O_2) × 100] [8].where SXO_2 = oxygen saturation at the place of X; PA = pulmonary artery; LA = left atrium, Ao = aorta.

Actually using this case's data of oximetry run, S(mixed venous) O_2 = [3 × 68.2 + (75.4 + 66.6)/2)]/4 = 68.9%, and left-to-right shunt ratio = [(80.1 − 68.9)/(96.7 − 68.9)] × 100 = 40.3%.

From this result, the left-to-right shunt volume was considered to cause heart failure, and we determined that this patient was a candidate for fistula closure.

The fistula closure procedure was performed by a team consisting of cardiologists, radiologists, and cardiovascular surgeons. In a hybrid operating room, an 8-Fr sheath was inserted into the right brachial artery under direct visualization using a cut-down method, and a 4-Fr sheath was inserted percutaneously into the right femoral artery with vascular ultrasound imaging, and a 12-Fr sheath was inserted into the left femoral artery using a cut-down method. A pull-through system was established between the right brachial artery and the left femoral artery. With angiographic guidance, two 13-mm-diameter, 50-mm-long GORE-Viabahn stent-grafts (WL Gore & Associates, Inc., Flagstaff, AZ, USA) were deployed at the right subclavian artery. To avoid residual shunt flow, after checking no acute ischemic symptom was observed, the right internal mammary artery and right vertebral artery were embolized with coils. Particularly, ischemia of vertebral artery area was dangerous so that we confirmed the good collateral circulatory network of arteries including the circle of Willis to be by magnetic resonance angiography before the procedure. Finally, we confirmed that there was no residual shunt flow (Fig. 2).

Just before and after the procedure, the patient's hemodynamic conditions were re-evaluated by catheter examination. Cardiac output was significantly reduced from 6.25 L/min to 3.58 L/min, representing a reduction ratio of 42.7%, which was similar to the left-to-right shunt ratio before the procedure (Table 1).

Two months later, the LV end-diastolic and end-systolic diameters decreased to 55 mm and 44 mm, respectively, and the patient's serum B-type natriuretic peptide level decreased from 300 pg/mL to 78 pg/mL (Table 1).

Discussion

There are two types of categories in high output heart failure [9]. One is caused by an unusual systemic condition which needs higher cardiac output, the other is exacerbated by excessive preload for the left ventricle which is forced by mechanical process.

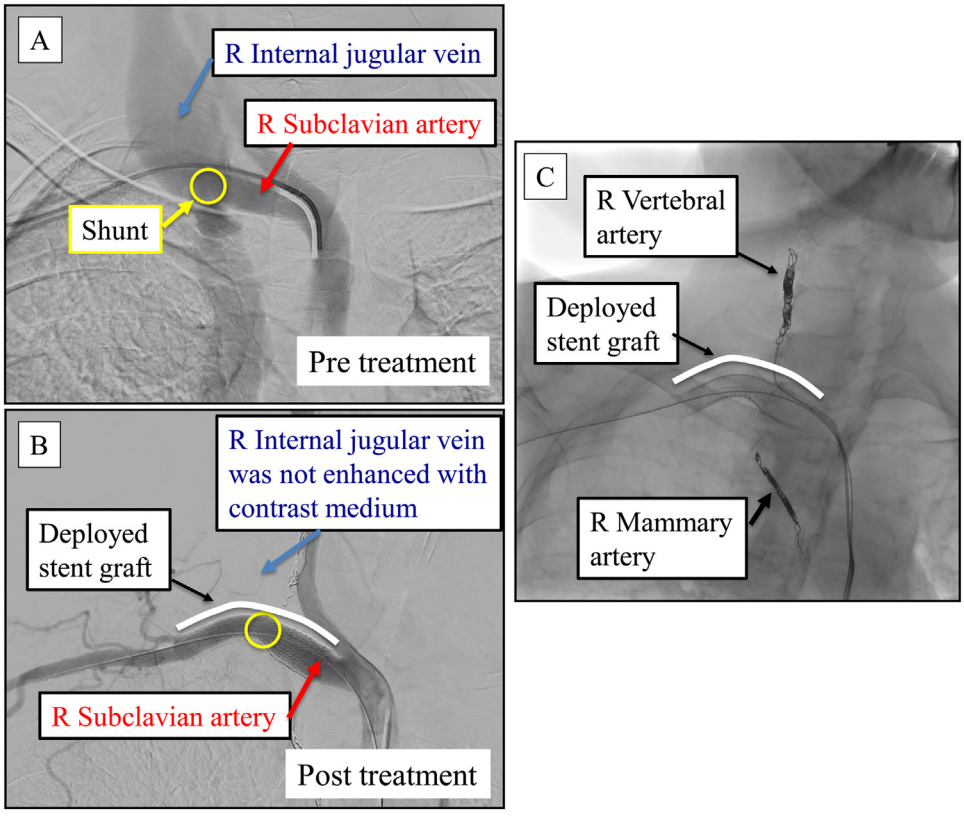

Fig. 2. Right subclavian angiographies of (A) pre- and (B) post-percutaneous fistula closure. The contrasted right internal jugular vein was observed in the pre-treatment angiography, and the right internal jugular vein was not contrasted after treatment. (C) Fluoroscopy of the treatment area after all procedures of percutaneous fistula closure had been done.

The former is derived from anemia, fever, exercise, inappropriate vasodilation (allergy, beriberi, sepsis, etc.), metabolic disorders (hyperthyroidism, pheochromocytoma, etc.) and so on. In these conditions, LV ejection fraction and heart rate must be higher, and the net volume of peripheral circulation is equal to the volume ejected from the left ventricle. On the other hand, the latter is originated from regurgitative valve disease (mitral valve regurgitation or aortic valve regurgitation), intracardiac shunt disease (ventricular septal defect), extracardiac shunt (patent ductus arteriosus, forearm hemodialysis shunt, etc.) and so on. These conditions have an important characteristic in common, that is, a partial volume of ejected blood from the left ventricle returns to the left ventricle again before perfusing peripheral tissue. This means that the net volume of blood which actually circulates peripheral tissue is smaller than the total blood volume which is ejected from the left ventricle. Then, according to the Frank–Starling's law, the left ventricle needs more end-diastolic volume to produce adequate blood output for satisfying the systemic demand. For the production of the increased stroke volume, the left ventricle should generate more work and is gradually damaged. Furthermore, the increased LV end-diastolic volume should provoke increased end-systolic volume and pressure due to the arterio-LV coupling mechanism, leading to deterioration of energy efficiency based on the mechano-energetics theory reported by Sunagawa et al. [10] Thus, through these mechanisms, the left ventricular performance should have deteriorated in a high output condition, brought about by the dilated and hypokinetic left ventricle during 11 years.

Sometimes, the forearm shunt for hemodialysis can contribute to high-flow heart failure, presenting with a dilated left ventricle that resembles DCM [3]. However, the fistula does not always work adversely; it is crucial to diagnose whether the left-to-right shunt is a cause of heart failure. A high volume extracardiac shunt with a left-to-right shunt ratio >30–35% or a shunt flow volume >2 L/min, which are considered adverse for cardiac function in dialysis patients [3], could cause heart failure. One could determine the indication for shunt closure if the left-to-right shunt ratio in patients with extracardiac fistula can be calculated.

The method we used to estimate the left-to-right shunt ratio seems applicable to shunts at any location in the blood circulation. An important point for using this method is identifying the substitutional vessel, which does not receive any shunt flow. Balloon occlusion test is also considered for evaluation of shunt ratio, however in this case, pseudoaneurysm was diagnosed beside the fistula, thus we avoided examination.

Patients with heart failure who have a DCM-like heart with excessive shunt flow can be improved by shunt closure. In this regard, we consider it is extremely important to estimate the left-to-right shunt ratio precisely in patients with high output heart failure and an extracardiac shunt disease.

Conflict of interest

The authors declare that they have no conflict of interest.

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