Case Report

Right-Sided Heart Failure Presentation in Severe Valvular Aortic Stenosis: How to Deal with Diuretic Use?

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A R T I C L E   I N F O

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A B S T R A C T

Introduction: Patients with right-sided heart failure and severe aortic stenosis (AS) have many clinical challenges to overcome fluid retention. A typical diuretic therapeutic approach can be recommended for the guidance of removing fluid overload in severe AS with right ventricular (RV) failure. This case review aims to understand how to assess and manage RHF and severe AS.

Objective: This case report aims to describe diuretic in Severe Valvular Aortic Stenosis

Case Illustration: A 65-year-old female with worsening bilateral leg swelling, and she was aware of the shortness of breath for three months. Physical examination revealed a grade 3/6 systolic ejection murmur at the aortic area radiating to the neck, a grade 2/6 diastolic murmur at the pulmonic area, and a grade 3/6 systolic murmur at the apex radiating to the axilla, increased jugular venous pressure, prominent bilateral leg swelling, and minimal rhonchi at the base of the lungs. The chest X-ray showed worsening cardiomegaly in the last three months. Echocardiography revealed high gradient severe valvular AS, decreased systolic RV function, high probability of pulmonary hypertension (PH), and other valvular dysfunctions, including moderate mitral regurgitation, moderate pulmonary regurgitation, and mild tricuspid regurgitation. She was introduced to high-dose furosemide infusion. The average urine output production was 5 L/day, and the negative fluid balance was 3 L/day. Furosemide dose was adjusted daily according to urine output production, and she was discharged after six days of hospitalization.

Conclusion: This case report provides an example of RV failure in severe AS patients. Hemodynamic monitoring and the typical approach of diuretic therapies should be needed in the management of fluid overload in severe AS.

1. Introduction

Valvular aortic stenosis (AS) has some characteristics of clinical findings. Dyspnea, syncope, and chest pain are the usual complaints, and the electrocardiogram presents left ventricular hypertrophy. Right-sided heart failure is an unusual manifestation of aortic stenosis.1,2 Morbidity and mortality in cardiovascular disease are closely related to right ventricular (RV) dysfunction.

RV dysfunction is a well-known predictor of poor prognosis in patients with valvular heart disease. Tricuspid annular phase systolic excursion (TAPSE) is the most extensively studied among several echocardiographic measures of RV function. A cut-off TAPSE of 17 mm could identify RV dysfunction predicting a poor prognosis.3

The main clinical feature in patients with right heart failure is peripheral edema. Volume management is an important issue in acute right heart failure.4 Increased total blood volume (fluid overload) leads to systemic congestion (ascites and peripheral edema). Diuretics are the mainstay medical choice in patients with RV failure and fluid overload.5 Therefore, it is crucial to monitor hemodynamic parameters. Severe valvular AS can lead to largely hemodynamic changes.5 Stepped pharmacological and hemodynamic assessment, therefore, should be needed in our case.

This case demonstrates that unusual right-sided heart failure could be the main problem in severe valvular aortic stenosis. This case underscores the importance of appropriate diuretic management strategies in a patient with right-sided heart failure and severe valvular AS.
2. Case Illustration

A 65-years-old female, was referred from a private hospital to the emergency department (ED) due to worsening bilateral leg swelling following shock condition three days before admission. It was getting worse in the last two weeks. Three months previously, she had also recently become aware of exertional dyspnea. There were no complaints of chest pain, palpitation, or syncope. She has had hypertension since ten years ago. Her blood pressure was 105/76 mmHg, respiratory rate was about 22/minute, and heart rate was about 65/minute. Her jugular vein pressure (JVP) was R+5 cmH2O, and blood pressure in both arms was equal. She had rales heard at the base of the lungs. Auscultation revealed a grade 3/6 systolic ejection murmur at the aortic area radiating to the neck, a grade 2/6 diastolic murmur at the pulmonic area, and a grade 3/6 systolic murmur at the apex radiating to the axilla. The liver was palpable two fingerbreadths below the right costal margin. There was bilateral peripheral edema up to the thigh.

The ECG (figure 1A) was compatible with left ventricular hypertrophy, poor r wave progression, and low voltage at limb leads. Laboratory findings revealed haemoglobin 10.8 g/dL, ureum 67.9 mg/dL, creatinine 1.36 mg/dL, and N-terminal-pro brain natriuretic peptide (NT-pro BNP) 33473 pg/mL. Chest X-ray (CXR) (see figure 1B) showed marked cardiomegaly with a cardiothoracic ratio (CTR) was about 75%. CXR films now revealed marked cardiomegaly with cardiac waist disappearance compared to that of three months ago (see figure 1C).

The previous hospital's initial treatment was continuous dobutamine infusion up to 5 mcg/kg/minute. Echocardiography was performed in cardiac intensive care. The aortic valve seems to be heavily calcified with the tricuspid valve. Maximum aortic systolic velocity (Vmax) was 4.3 m/s, mean aortic valve pressure gradient (mean PG) was 51 mmHg, and aortic valve area (AVA) by continuity equation was 0.75 cm2.

Figure 1. ECG showing sinus rhythm with left ventricular hypertrophy and poor r wave progression (A). Chest x-ray film showing marked cardiomegaly with displacement of right heart border and disappearance of cardiac waist (on admission day) (B). Chest x-ray film showing cardiomegaly (on 3 months before admission) (C).
Other valvular dysfunctions were moderate aortic regurgitation, moderate mitral regurgitation, mild pulmonic regurgitation with early diastolic velocity (TR Vmax) 3.1 m/s (high probability of PH), RV systolic function was decreased, showing a TAPSE of 13 mm. RV systolic pressure was 46 mmHg. Mean pulmonic artery (PA) pressure was 38 mmHg. Other hemodynamic assessments revealed ejection fraction (EF) of 20%, stroke volume index (SVI) was 87 ml/m2, estimation of pulmonary capillary wedge pressure (ePCWP) was 26 mmHg, and segmental myocardial analysis revealed global hypokinesis.

As initial treatment, his breathing was supported with a nasal canule with 3 L of O2. The patient had been given intravenous furosemide (40 mg). Urine production had been observed up to 2900 cc for 6 hours. This patient ultimately was decided to give furosemide continuous infusion up to 20 mg/hour. Preload optimization was closely evaluated by central venous pressure (CVP). Targetted urine output production was 3-5 L/day. During observation in the intensive care unit, the average urine output was 8 L/day, and negative fluid balance was 5 L/day. CVP monitoring showed about 10-12 mmHg. The last furosemide dose was down-titrated up to 10 mg/hour and switched to intravenous 20 mg for twice daily due to improving clinical leg swelling. Inward, the average urine output production was 2.5 L/day, and negative fluid balance was 1 L/day. After six days of hospitalization, intravenous furosemide was stopped and converted to oral furosemide 40 mg daily; then, she was discharged home.

3. Discussion

The triad of syncope, angina, or dyspnea is the classic clinical presentation of severe AS. Most patients remain asymptomatic due to the left ventricular's adaptive mechanism (LV) until advanced narrowing of the aortic valve orifice occurs. The natural history of AS has poor outcomes unless the patient undergoes aortic valve replacement.6 The symptoms usually occur in the sixth decade, and the average survival is about four years after severe symptoms. In untreated patients, the survival is usually to be 2, 3, or 5 years after the onset of heart failure, syncope, or chest pain, respectively.1,6

RV dysfunction is rarely found due to the patient's early death.1 Only a few studies have been published about the negative prognostic impact of RV failure in AS. Aortic valve replacement could temporarily reduce systolic PA pressure (sPAP); however, sPAP levels were increased 12 months after the procedure. Galli et al. observed that RV dysfunction is found in one-quarter of patients with AS.7

The progressive LV remodeling leads to increase LV end-diastolic pressure transmitted to the left atrial, inducing an increased pulmonary post-capillary pressure. The increased sPAP will directly induce RV dysfunction in the final stage.2 The RV and LV share common fibers encircling both ventricles, which affect the interdependence observed between RV and LV.7 Prolonged elevation in LV afterload induces LV hypertrophy and fibrosis.8 LV hypertrophy produces angiotensin I and catecholamines, which cause RV remodeling and
progressive deterioration in AS.7,9 Galli et al. stated that cardiovascular (CV) mortality was related to RV dysfunction alone, whereas RV and LV dysfunction have been a significant predictor of CV mortality.2

Based on echocardiography, this patient had high gradient severe valvular AS due to Vmax > 4 m/s, mean PG > 40 mmHg, and AVA by continuity equation was < 1 cm² with normal flow (SVi > 35 mL/m²). It was also complicated due to the presence of RV dysfunction and a high probability of PH. Age-related calcific degeneration is the most common cause of AS.10 The patient with severe AS is relative "afterload fixed and preload dependent," which means cardiac output does not improve with afterload reduction. Thus, all afterload reducing agents are contraindicated. Hemodynamic monitoring with a central venous can be informative in such a case.4

In an acute right heart failure setting complicated by hypotension, excessive volume resuscitation cause ischemia due to excess RV preload. The CVP optimum is about 8-12 mmHg to restore or maintain total preload for optimal condition.4 Diuretic therapies have clear benefits for the patient with fluid overload. There are three main classes of diuretics: (i) loop diuretics, which act in sodium channel in the loops of Henle; (ii) thiazide diuretics, which act in the distal nephron convoluted tubules; and (iii) potassium-sparing aldosterone antagonists, which inhibit aldosterone action on mineralocorticoid receptors. Intravenous furosemide is considered in specific case.5 Figure 2 shows the management of acute right heart failure focuses on managing volume/preload and perfusion.

Patients with acute decompensation may be hard to resolve in the diuretic resistance condition. They may not respond to intravenous loop diuretics due to several conditions, including low cardiac output, hypotension, elevated CVP, venous congestion, and acute kidney injury. Early and aggressive high-dose diuretic strategy in patients failing to respond to initial diuretic is safe; however, the efficacy is still unclear.4 In our case, the high dose furosemide continuous infusion had a good response because the urine output production was > 5 L/day. The next daily dose was adjusted to reach the goal of urine output production of 3-5 L/day. Table 1 explains stepped pharmacological care for diuretic use responding to urine output production. A hypotensive setting is not contraindicated in diuretic treatment. Hypotension is a marker of a critically ill patient in this setting, requiring vasoconstrictors and consideration for mechanical circulatory support.11

| UO goals to be assessed daily from randomization to 96 h |
|--------------------------------------------------------|
| UO >5 L/d = reduce current diuretic regimen if desired |
| UO 3-5 L/d = continue current diuretic regimen        |
| UO <3 L/d = see diuretic grid                          |

24-h assessment

UO recommendations as above

Advance to next step on grid if UO <3 L/d

48-h assessment

UO recommendations as above

Advance to next step on grid if UO <3 L/d

Consider dopamine or dobutamine at 2 ug/kg/h if SBP <110 mmHg and EF <40% or RV systolic dysfunction

Consider nitroglycerin or nesiritide if SBP >120 mmHg (any EF) and severe symptoms

Table 1. Stepped pharmacological care: treatment algorithm from the CARRESS-HF trial.4

Note; CARRESS-HF indicates Cardiorenal Rescue Study in Acute Decompensated Heart Failure; EF, ejection fraction; IV, intravenous; Loop, loop diuretic dose in furosemide equivalents; LVAD, left ventricular assist device; RV, right ventricular; SBP, systolic blood pressure; and UO, urine output.

| Current Dose | Daily Loop Dose | Suggested Dose | Thiazide |
|--------------|-----------------|----------------|---------|
| A <80 mg     | 40 mg IV bolus 5 mg/h | 5 mg metolazone once daily | None |
| B 81-160 mg  | 80 mg IV bolus +10 mg/h | 5 mg metolazone twice daily |
| C 161-240 mg | 80 mg IV bolus+20 mg/h | 5 mg metolazone twice daily |
| D >240 mg    | 80 mg IV bolus+30 mg/h |                         |
In cases of hypotension, inotropic and vasopressor may be needed to maintain adequate perfusion. Inotropes increase myocardial contractility and failing RV stroke volume while reducing RV end-diastolic volume and pressure. Milrinone and dobutamine could affect inotropic and vasodilator properties. Therefore, they could be used cautiously in hypotensive patients; however, it rarely occurs if LV preload is adequate improving cardiac output (see figure 2).⁴

In AS and RV failure, diuretic strategies help prevent edema and maintain fluid levels, yet they are associated with renal dysfunction. It is crucial to educate patients on what they should do to improve daily activity because adherence to therapies is a cornerstone to achieve optimal outcomes.⁵

4. Conclusion

This case report provides an example of RV failure in severe AS patients. Hemodynamic monitoring and stepped care of diuretic therapies should be needed in the management of fluid retention in severe AS. Patients should know how to manage, reduce, and monitor the side effects associated with diuretic use, if diuretic therapies are necessary.

5. Declarations

5.1. Ethics Approval and Consent to participate
Patient has provided informed consent prior to involve in the study.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: FR. Design: FR. Control/supervision: CT, AF, HM. Data collection/processing: FR. Extraction/Analysis/interpretation: FR. Literature review: CT, AF, HM. Writing the article: OH. Critical review: CT, AF, HM. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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