Received: 2018.02.10
Accepted: 2018.04.07
Published: 2018.06.23

e-ISSN 1941-5923
© Am J Case Rep, 2018; 19: 739-743
DOI: 10.12659/AJCR.909448

Recurrent Unilateral Transudative Pleural Effusion Due to Low Flow, Low Gradient Severe Aortic Stenosis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Female, 86
Final Diagnosis: Severe aortic stenosis
Symptoms: Exertional dyspnea
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

Background: In symptomatic severe aortic stenosis (AS), the majority of patients have high gradient AS. However, some patients have an AS gradient less than 40 with a valve area under 1.0 cm². For patients with a low gradient, severe AS is difficult to detect and requires a high index of suspicion. Transcatheter aortic valve replacement (TAVR) is currently recommended for patients with moderate to high risk AS according to the Society of Thoracic Surgery (STS) risk score.

Case Report: Here we present the case of an 86-year-old female with recurrent pleural effusion over the course of 2-year; she had multiple thoracentesis procedures and was being considered for a pleurodesis. Later the patient was found to have severe AS; an echocardiogram showed an aortic valve (AV) area of 0.67 cm², AV mean gradient of 34 mmHg, and ejection fraction of 75%. The patient underwent a diagnostic cardiac catheterization and was treated with TAVR.

Conclusions: The diagnosis was made after exclusion of all other causes of unilateral pleural effusion and was confirmed by improvement of effusion following the TAVR procedure.

MeSH Keywords: Aortic Valve Stenosis • Dyspnea • Pleural Effusion

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/909448>

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Background

Symptomatic severe aortic stenosis (AS) is the third most common cause of heart disease after hypertension and coronary artery disease in developed countries. AS generally presents with exertional dyspnea, syncope, and exertional chest pain [1]. Severe AS is normally defined by a combination of an aortic valve (AV) area $\leq 1 \text{ cm}^2$ and a mean transvalvular pressure gradient $\geq 40 \text{ mmHg}$ [2]. However, some patients have a gradient less than 40 mmHg, left ventricle (LV) stroke volume index $\leq 35 \text{ mL/m}^2$ with a AV area under 1.0 cm^2 . This low-flow, low-gradient (LFLG) severe AS is difficult to detect and requires a high index of suspicion. It is further classified into classical LFLG-AS in which there is reduced LV ejection fraction (LVEF) and paradoxical LFLG-AS associated with preserved EF [3]. Echocardiogram is used for the diagnosis of LFLG-AS and it is confirmed with cardiac catheterization. Transcatheter aortic valve replacement (TAVR) is currently recommended for patients with true AS with moderate to high risk according to Society of Thoracic Surgery (STS) risk score.

Transudative pleural effusions according to Light's Criteria is defined as a ratio of pleural fluid protein to serum protein < 0.5 and a pleural fluid lactate dehydrogenase (LDH) to serum LDH ratio < 0.6 . Caused by either increased hydrostatic pressure, such as in heart failure, or decreased oncotic pressure, such as in liver cirrhosis or nephrotic syndrome [4].

Case Report

An 86-year-old non-smoking female with mild chronic obstructive pulmonary disease presented for recurrent exertional dyspnea secondary to pleural effusion to our cardiology clinic. She had a 2-year history of recurrent, right-sided pleural effusion with multiple thoracentesis procedures and was being considered for a pleurodesis.

On physical examination, she was found to be hypoxic, have diminished breath sounds, dullness to percussion on the right side with no crackles or wheezes detected, a grade of 4/6 ejection systolic murmur at the right upper sternal border with an absent A2 component and delayed carotid upstroke. The neck examination was negative for jugular venous distension.

Chest x-ray showed right-sided pleural effusion before the TAVR procedure (Figure 1). Thorax computed topography (CT) showed a moderate right pleural effusion before AV intervention (Figure 2). Laboratory results showed no leukocytosis, no coagulopathy, no hypoalbuminemia, normal platelet counts, normal thyroid function test, and the B natriuretic peptide (BNP) result was lower than 100 excluding cardiac etiology. QuantiFERON-TB Gold test for tuberculosis was negative (Table 1).

Figure 1. Chest x-ray showed right-sided pleural effusion before transcatheter aortic valve replacement procedure.

Figure 2. Computed topography scan thorax reveals moderate right pleural effusion before aortic valve intervention.

A thoracentesis was consistent with transudative pleural effusion with the pleural fluid protein to serum protein of 0.42, and the pleural fluid lactate dehydrogenase (LDH) to serum LDH ratio of 0.27. The results were negative for gram stain, fungal culture, and acid-fast bacilli stain and culture. Pathology reports showed no malignant cells were identified (Table 2).

The echocardiogram showed normal LV size, wall thickness, and systolic function; grade II diastolic dysfunction and EF 75%. It showed moderate dilated left atrium with left atrial volume index of 46 mL/m^2 and moderate calcific severe AS. The AV area was 0.67 cm^2 , AV mean gradient was 34 mmHg, with peak at 49 mmHg. The LV stroke volume index was below 35 mL/m^2 . The right atrial size was normal; the right atrial pressure was

Table 1. Laboratory values.

Component	Value	Ref range and units
WBC	10.4	4.8–10.8 K/uL
RBC	4.32	4.20–5.40 M/uL
Hemoglobin	13.3	12.0–16.0 g/dL
Hematocrit	39.2	37.0–47.0%
MCV	90.7	81.4–97.8 fL
RDW	42.5	35.9–50.0 fL
Platelet count	235	164–446 K/uL
Sodium	138	135–145 mmol/L
Potassium	3.8	3.6–5.5 mmol/L
Chloride	105	96–112 mmol/L
Co2	24	20–33 mmol/L
Anion gap	9.0	0.0–11.9
Glucose	117	65–99 mg/dL
Bun	22	8–22 mg/dL
Creatinine	0.85	0.50–1.40 mg/dL
Calcium	9.0	8.5–10.5 mg/dL
AST (SGOT)	17	12–45 U/L
ALT (SGPT)	6	2–50 U/L

Component	Value	Ref range and units
Alkaline phosphatase	88	30–99 U/L
Total bilirubin	0.4	0.1–1.5 mg/dL
Albumin	3.5	3.2–4.9 g/dL
Total protein	7.1	6.0–8.2 g/dL
Globulin	3.1	1.9–3.5 g/dL
LDH total	242	107–266 U/L
B natriuretic peptide	69	0–100 pg/mL
PT	13.4	12.0–14.6 sec
INR	0.99	0.87–1.13
TSH	0.800	0.350–5.500 uIU/mL
Carcinoembryonic antigen	1.4	0.0–3.0 ng/mL
Ca 19-9	19.7	0.0–35.0 U/mL
Ca 125	34.7	0.0–35.0 U/mL
Sed rate Westergren	28	0–30 mm/hour
Antinuclear antibody	None detected	None detected
Rheumatoid factor	<10	0–14 IU/mL

Table 2. Pathology reports.

Pleural fluid	Results
Color: body fluid	Yellow
Character: body fluid	Hazy
Total RBC count	<2000 cells/uL
Total WBC	231 cells/uL
Polys	1%
Lymphs	78%
Mononuclear cells	9%
Mesothelial cells – CSF	2%

Pleural fluid	Results
Fluid histiocyte	9%
Eosinophils – CSF	1%
PH	8
Total protein fluid	3.0
Body fluid LDH	66
Glucose, fluid	122
Body fluid amylase	27

estimated to be 3 mmHg. There was normal right ventricular size and systolic function. The right ventricular systolic pressure was estimated to be 45 mmHg (Figures 3–5).

The right heart catheterization showed right atrium was 8 mmHg, right ventricle was 45/10/13 mmHg, pulmonary artery was 45/25 (30) mmHg, and pulmonary wedge was 13 mmHg.

Cardiac output per thermal dilution was 4.77. Cardiac index per thermal dilution was 2.52.

Left side heart catheterization showed moderate to severe AS with peak to peak gradient 35 mmHg, mean gradient 35 mmHg, calculated AV area 0.61 cm², nonobstructive coronary artery disease, EF 65%, mildly dilated ascending aorta, moderate

Figure 3. Parasternal long axis view shows calcified aortic valve leaflets.

Figure 4. Continuous wave Doppler recording from apical 5-chamber view showing transvalvular peak jet velocity (3.49 m/s) and pressure gradients (mean gradient=33.89 mmHg) correspond to severe low gradient aortic valve stenosis.

Figure 5. (A, B) Diastolic function.

caliber distal aorta with 30% eccentric stenosis on right lateral wall, and normal appearing bilateral ilio-femoral arteries.

Discussion

AS is generally asymptomatic until stenosis is severe enough to cause symptoms. Classical symptoms are angina, syncope, heart failure, and sudden death [5]. During the evaluation of patients with severe AS, it is important to distinguish between true stenosis or pseudo stenosis as it affects the patient management plan. Dobutamine stress echocardiography, invasive valve study, or multidetector CT can be used to confirm severe stenosis when AV calcium score is >1200 AU in a female or >2000 AU in a male [6].

Patients with STS moderate- to high-risk AV stenosis have a high perioperative and postoperative mortality.

Pleural effusions can be seen in many conditions, including those with left-sided heart failure secondary to increased hydrostatic pressure, and it is generally present with bilateral pleural effusion with variation depending on which side is more prominent. However, unilateral pleural effusion secondary to severe AS in the absence of heart failure is unusual. Here, in a patient 86-year-old female with no significant lung disease, and severe low gradient flow AS.

The diagnosis was made after excluding of all other causes of pleural effusion mentioned in the table above and confirmed by lack of symptoms during follow-up visits on a regular basis with a series of chest x-rays and echocardiograms to ensure resolving effusion after the TAVR procedure (Figure 6).

Figure 6. Chest x-ray 1 month after transcatheter aortic valve replacement procedure reveal improvement in the right-sided effusion compared with before procedure.

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Conclusions

Pleural effusions can be seen in patients with severe AS complicated with hemodynamic significant heart failure and are generally present as bilateral pleural effusion. Unilateral pleural effusion is an unusual presentation, making this case an important example of how LFLG-AS can present with unilateral effusion even with the lack of significant manifestation of heart failure in a patient with mild diastolic dysfunction. The mechanism was likely due to diastolic dysfunction and increased hydrostatic pressure.

Conflict of interests

None.