CASE REPORT

Comprehensive Management and Preoperative Preparation in Aortic Stenosis: Strategies for Optimal Recovery in CHF Patients

Doharjo Manullang[1], Menang B Tarigan[2], Salomo GU Simanjuntak[3], Ivonne Situmeang[4], Evi R Simanjuntak[5], Reinhard DJ Hutahaeng[6]

1. Department of Internal Medicine, Faculty of Medicine,Universitas Methodist Indonesia.
2. Department of Internal Medicine, Faculty of Medicine, Universitas Methodist Indonesia.
3. Department of Internal Medicine, Faculty of Medicine,Universitas Methodist Indonesia.
4. Department of Public Health, Faculty of Medicine, Universitas Methodist Indonesia.
5. Department of Histology, Faculty of Medicine, Universitas Methodist Indonesia.
6. Department of Forensic and Medicolegal Sciences, Universitas Methodist Indonesia.

Correspondence:
doharjomanullang2025@gmail.com

ABSTRACT

Stenosis is the most common valvular heart disease in Europe and North America with fear of calcification in the elderly (2-7% of the population> 65 years) while congenital aortic stenosis is usually caused by rheumatic heart disease which is now rare and usually found in young age. Classification of Aortic Stenosis. Classification of Aortic Stenosis can be divided based on the severity divided into 3 parts of mild, moderate, severe and very severe. Echocardiography modality is one of the gold standard tools for determining Aortic Stenosis which in echocardiography can capture Peak aortic jet velocity, the mean pressure gradient aortic valve area. The following is a case report of a 36 year old man with CHF ec Aortic Stenosis ec Rheumatic Heart Disease. This case was only diagnosed about 1 month ago with complaints of shortness of breath and chest pain. And the patient immediately went to RSMH for further treatment. This case was appointed for the management and preparation for surgery as a definitive therapy so that it is expected to give the best results in this patient

Keywords: Aortic Stenosis, Classification, Diagnostic Test and Treatment.

INTRODUCTION

Aortic Stenosis (AS) is the most prevalent valvular heart disease in Europe and North America. Primary AS is characterized by calcification in the elderly (affecting 2–7% of the population over 65 years old), whereas congenital AS is typically associated with Rheumatic Heart Disease (RHD), which has become increasingly rare and is predominantly found in young adults. Key clinical indicators for managing AS include symptoms such as exertional dyspnea, angina, and syncope.

In the United States, approximately 4.2–5.6 million adults present with clinical valvular disease. In 2010, the population aged 65 and older reached approximately 40 million, with projections increasing to 55 million by 2020 and 72 million by 2030. Generally, the progression of AS is closely linked to the aging process. [1,2,3]

A study by Helsinki Aging utilizing echocardiography revealed that aortic valve calcification and degeneration correlate with advancing age. Valve calcification was observed in approximately 75% of patients aged 85–86 years. In cases of critical aortic stenosis, the prevalence was approximately 1–2% at ages 75–76 and increased to roughly 6% by ages 85–86. Furthermore, a study of 16,501 individuals by the National Heart, Lung, and Blood Institute—involving 11,911 clinical echocardiographic examinations in Minnesota—demonstrated a rising prevalence of AS in tandem with increasing age. [1,2,3]

ETIOLOGY

The etiology of Aortic Stenosis (AS) primarily involves senile calcification, degenerative processes, and Rheumatic Heart Disease (RHD). In developed countries, AS is predominantly caused by calcification and degenerative changes, which are frequently observed in elderly patients. Conversely, in developing nations, rheumatic disease remains the most common underlying cause of AS. [2,4]

CLINICAL MANIFESTATIONS

Aortic Stenosis (AS) is characterized by a classic clinical triad of symptoms: angina, syncope, and heart failure. Initially, patients often present with fatigue during daily activities; if left untreated, the full classic triad will manifest. According to the landmark report by Ross and Braunwald, the life expectancy for AS patients following the onset of angina is approximately five years, decreasing to three years for syncope, and approximately two years once heart failure develops.

Angina in AS results from a mismatch between oxygen supply and demand. Increased oxygen demand is driven by left ventricular hypertrophy (LVH), while supply is compromised by a reduction in mean arterial pressure (MAP) relative to coronary blood flow. Syncope occurs due to the heart's inability to increase cardiac output to meet systemic demands; for instance, orthostatic changes can lead to venous pooling, which reduces preload and subsequently further diminishes cardiac output. Heart failure represents an advanced clinical stage of AS and carries a poor prognosis. This condition arises as a consequence of progressive valvular obstruction, which initially triggers compensatory left ventricular hypertrophy.

Beyond clinical symptoms, AS can be detected through auscultation, characterized by a systolic murmur heard at the second right intercostal space (ICS). In addition to clinical manifestations and physical examination, echocardiography is essential for diagnosing AS. It provides a detailed anatomical assessment of the aortic valve, including the extent of calcification, left ventricular dimensions, and hemodynamic severity. Furthermore, echocardiography is critical for determining prognosis and the optimal timing for valvular intervention. [5,6,7]

Table 1 Stages of Aortic Stenosis include:

Stage	Group	Definition	Results	Handling
A	At risk	Aortic or Bicuspid Valve Sclerosis, V max <2 m/s	There is a >50% increased risk of myocardial infarction and cardiovascular death occurring after 5 years.	Cardiovascular risk factor assessment and primary prevention
B	Progresif	Mild to moderate calcification or rheumatic changes with reduced leaflet motion, Vmax 2-3.9 m/s, or mean transaortic pressure gradient 20-39 mmHg..	Hemodynamic progression is present.	Assess cardiovascular risk factors and primary prevention, conduct periodic clinical check-ups with echocardiographic monitoring, and educate the patient about the disease course and outcome.
C1	Asymptomatic aortic stenosis with normal left ventricular function	Severe calcification or rheumatic changes with reduced leaflet motion, Vmax >4 m/s or a mean transaortic pressure gradient ≥ m/s with EF ≥50%.	Symptoms can occur within 3 years in approximately 50-80% of attacks, with a low risk of sudden death. However, the severity of symptoms depends on the type of attack. If symptoms persist >50% and with very severe aortic stenosis (Vmax >5 m/s), the life expectancy is approximately 2 years.	Frequent clinical monitoring (≤6 months) and echocardiographic monitoring (≤12 months) during attacks and disease progression. Treatment or a Brain Natriuretic Peptide (BNP) test may be considered. AVR is an appropriate treatment for very severe aortic stenosis with asymptomatic symptoms.
C2	Severe asymptomatic aortic stenosis with an EF <50%	Severe calcification or rheumatic changes with decreased leaflet motion Vmax ≥ 4 m/s or a mean transaortic pressure gradient ≥ 40 mmHg with <50%	If other causes of left ventricular dysfunction are excluded, the EF will return to normal after AVR.	AVR may be recommended to preserve left ventricular function.
D1	Severe aortic	Severe calcification	Mortality can occur in	AVR is performed

	stenosis with symptoms and a high pressure gradient.	or rheumatic changes with decreased leaflet motion Vmax ≥ 4 m/s or a mean transaortic pressure gradient ≥ 40 mmHg.	approximately 50% of cases within 1 year, and in 70-80% of cases within 2 years if AVR is not performed.	immediately and is an effective therapy.
D2	Severe aortic stenosis with symptoms and a low pressure gradient and an EF <50%.	Severe calcification or rheumatic changes with decreased leaflet motion, AVA ≤ 1 cm2 with Vmax < 4 m/s with EF < 50% and Vmax ≥ 4 m/s with AVA ≤ 1 cm2, and slow flow as evidenced by a dobutamine stress test.	The 2-year mortality rate with therapy is approximately 20% compared to approximately 40% with AVR. Mortality is higher with surgery and the prognosis is lower in patients without any contractions.	AVR is performed immediately if severe aortic stenosis is found. EF improves after AVR even in patients without contractions.
D3	Severe symptomatic aortic stenosis with low flow and a low pressure gradient	Severe calcification or rheumatic changes with decreased leaflet motion, AVA ≤ 1 cm2 and V max < 4 m/s with EF > 50% or AVA ≤ 0.6 cm2/m2 with a Stroke Volume index < 35 mL/m2 with normal blood pressure	2-year mortality is approximately 50-70% if AVR is not performed.	AVR should be performed immediately in symptomatic patients if symptoms of severe aortic stenosis are found during the evaluation and no other cause is identified.

AVA : Aortic Valve Area, AVR : Aortic Valve Replacement, V_{max} = Aortic maximum Velocity

Table 2. Severity of Aortic Stenosis from AHA/ACC and European Association of Echocardiography/American Society of Echocardiography guidelines6:

	MILD	MODERATE	SEVERE	VERY SEVERE
Peak Aortic Jet Velocity (m/s)	2.0-2.9	3.0-3.9	≥ 4.0	≥ 5.0
Mean Preassure Gradient (mmHg)	< 20	20-39	≥ 40	≥ 60
Aortic Valve Area (AVA) (cm^2)	> 1.5	1.0-1.5	≤ 1.0	-
Indexed Aortic Valve Area (cm^2/m^2)	> 0.85	0.60-0.85	<0.60	-
Dimensionless index	>0.50	0.25-0.50	<0.25	-

ECHOCARDIOGRAPHY [13]

Berikut adalah terjemahan teks tersebut ke dalam bahasa Inggris medis yang formal dan sesuai dengan standar penulisan jurnal kardiologi:

ECHOCARDIOGRAPHY

Echocardiography is the cornerstone of diagnostic examination for aortic stenosis, as it allows for the assessment of valvular calcification, left ventricular function, and myocardial wall thickness, while also identifying other potential causes of valvular disease. Doppler echocardiography is the modality of choice for evaluating the severity of aortic stenosis. Ideal echocardiographic measurements for assessing the severity of the condition include the evaluation of flow, mean pressure gradients, ventricular function, cardiac dimensions, wall thickness, valvular calcification, blood pressure, and functional status.

There are four defining categories of aortic stenosis severity, as follows:

1. Cardiology (ESC).
   Classification of Aortic Stenosis Severity High-Gradient Aortic Stenosis (Aortic Valve Area [AVA] <1.0 cm2, Mean Gradient >40 mmHg). The severity of aortic stenosis can be assessed regardless of Left Ventricular Ejection Fraction (LVEF) and whether the flow is normal or reduced.
2. Low-Flow, Low-Gradient Aortic Stenosis with Reduced Ejection Fraction (AVA <1.0 cm2, Mean Gradient <40 mmHg, EF <50%). Low-dose dobutamine stress echocardiography is recommended to accurately differentiate true severe aortic stenosis from "pseudo-severe" aortic stenosis, where the aortic valve area increases to >1.0 cm2 with normalized flow. Furthermore, the presence of contractile reserve—defined as an increase in stroke volume by >20%—indicates a more favorable prognostic implication.
3. Low-Flow, Low-Gradient Aortic Stenosis with Preserved Ejection Fraction (AVA <1.0 cm2, Mean Gradient <40 mmHg, EF ≥50%, Stroke Volume Index [SVi] ≤35 mL/m2). This is typically found in elderly patients and is associated with small ventricular size, characterized by left ventricular hypertrophy and a history of hypertension.
4. Normal-Flow, Low-Gradient Aortic Stenosis with Preserved Ejection Fraction (AVA <1.0 cm2, Mean Gradient <40 mmHg, EF ≥50%, SVi >35 mL/m2). Patients in this category typically present with moderate aortic stenosis.

Indikasi Intervensi[8,9,10]

The indications for aortic valve intervention are summarized in the figure below:

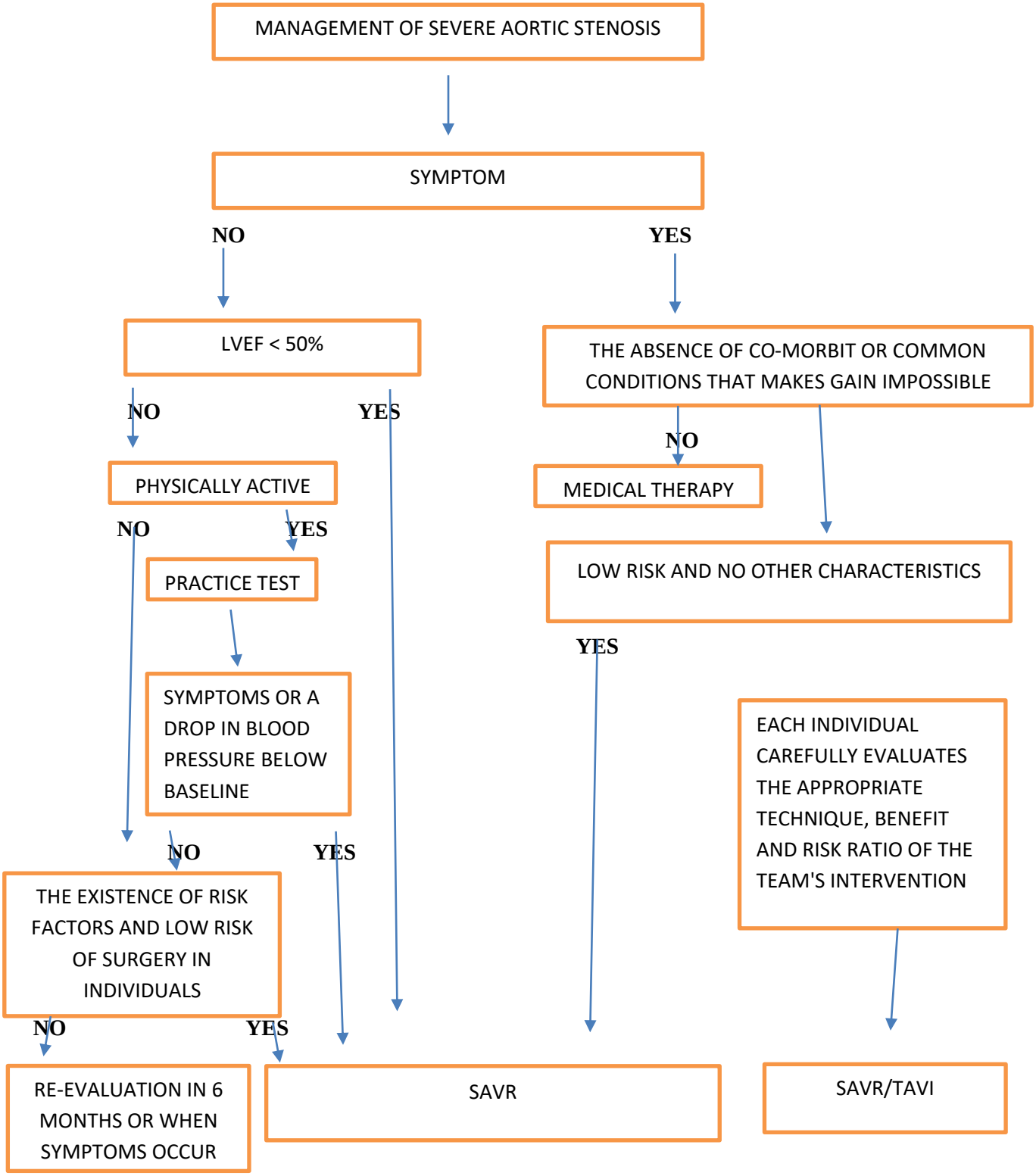


Figure 1: Management of Severe Aortic Stenosis[6]

INDICATIONS FOR INTERVENTION IN SYMPTOMATIC AORTIC STENOSIS.[11,12,13,14,15,16,17]

1. Patients with high mean gradient and reduced Ejection Fraction (EF).
2. Patients with low-flow, low-gradient aortic stenosis and reduced EF due to afterload mismatch; in these cases, left ventricular function typically improves following intervention.
3. Patients with low-flow, low-gradient aortic stenosis and preserved (normal) EF.
4. Patients with normal-flow, low-gradient aortic stenosis and preserved (normal) EF.

Several aspects must be considered by the Heart Team when determining the appropriate approach for Aortic Valve Replacement (AVR), specifically in choosing between Transcatheter Aortic Valve Implantation (TAVI) and Surgical Aortic Valve Replacement (SAVR), as detailed in Table 2 below.[8,9,10]

Table 3. Key Considerations for the Heart Team in Selecting Between SAVR and TAVI for Patients with Increased Surgical Risk[17]

	TAVI	SAVR
Karakteristik klinis		
STS/Euro SCORE II<4% (Logistic EuroSCORE I<10%)		+
STS/Euro SCORE II ≥ 4% (Logistic EuroSCORE I≥10%)	+	
Adanya Komorbiditas yang berat	+	
Umur < 75 tahun		+
Umur≥ 75 tahun	+	
Sebelumnya menjalani operasi bedah jantung	+	
Kelemahan	+	
Berkurangnya mobilitas atau atau sedang menjalani rehabilitas setelah menjalani suatu tindakan prosedur	+	
Dugaan Endocarditis		+
Aspek Teknik dan Anatomi		
Adanya akses yang baik untuk transfemoral TAVI	+	
Akses yang tidak baik untu TAVI		+
Gejala sisa nyeri dada akibat radiasi	+	
Porcelain Aorta	+	
Terlihat intact CABG saat dilakukan sternotomy	+	
Ketidaksesuaian prosthesis	+	

pada pasien		
Adanya jarak yang pendek antara ostia coronaria dengan annulus katup aorta		+
Ukuran dari annulus katup aorta yang diluar dengan TAVI		+
Morfologi katup (bicuspid, kalsifikasi), tidak baik untuk TAVI		+
Adanya trombus di aorta atau ventrikel kiri		+
Kondisis jantung selain stenosis aorta dan dilakukan intervensi secara bersamaan		
CAD berat dan wajib revaskularisasi dan CABG		+
Penyakit katup mitral yang berat dan diobati dengan tindakan bedah		+
Penyakit Katup Trikuspi yang berat		+
Aneurisma dari aorta asending		+
Hipertropi septal yang wajib dilakukan tindakn myectomy		+

CASE REPORT

Teks yang Anda berikan adalah bagian **Presentasi Kasus (Case Presentation)**. Dalam jurnal kedokteran, bagian ini harus ditulis menggunakan terminologi klinis yang objektif dan ringkas.
Berikut adalah terjemahannya dalam bahasa Inggris medis yang formal:

CASE PRESENTATION

A 36-year-old male (P) presented to the Emergency Department of RSMH on September 29, 2020, with a chief complaint of severe dyspnea. The patient reported experiencing shortness of breath for the past month, which was exacerbated by physical activity (exertional dyspnea) but unaffected by weather conditions. No wheezing was reported. To achieve comfort while sleeping, the patient required 2–3 pillows (orthopnea). Additionally, he experienced intermittent bilateral lower limb edema and recurrent chest pain.

In early February, the patient experienced a syncopal episode lasting approximately 15 minutes while playing badminton, preceded by dizziness and blurred vision. No further complaints occurred until mid-August, when he developed severe dyspnea. Following a blood test at the Palembang Health Laboratory (BBLK), he was informed of a potential cardiac condition. He subsequently sought consultation at RSMH with a Cardiovascular Specialist (SpPD-KKV) on September 1, 2020.

An initial echocardiography performed on September 1, 2020, revealed moderate Aortic Stenosis (AS) suspected to be secondary to Rheumatic Heart Disease (RHD), reduced left ventricular (LV) function, and moderate Pulmonary Hypertension (PH). He was discharged with a medical regimen consisting of Digoxin 0.25 mg once daily, Spironolactone 12.5 mg once daily, Furosemide 20 mg once daily, and Aspilet (Aspirin) 80 mg once daily. However, on September 29, 2020, the patient returned to RSMH due to worsening dyspnea

and was subsequently admitted for inpatient care.

Physical Examination
General condition: Weak, compos mentis consciousness, BP 110/88 mmHg, Pulse 97x/menit (regular isi dan tegangan cukup), RR : 26 x/ menit, T : 36^{o}C. BB : 65 kg TB : 173 cm, BBI = 21,72% (BBI Normal)

Berikut adalah terjemahan bagian **Pemeriksaan Fisik (Physical Examination)** ke dalam bahasa Inggris medis yang formal, biasanya ditulis dalam bentuk poin-poin atau narasi singkat di jurnal.

PHYSICAL EXAMINATION

- **Eyes:** Conjunctiva anemic (-/-), scleral icterus (-/-).
- **Mouth:** Cyanosis (-).
- **Neck:** Jugular Venous Pressure (JVP) 5+2 cmH_2O, thyroid enlargement (-).

Thorax

- **Lungs:**
  - **Inspection:** Symmetric chest expansion during static and dynamic phases.
  - **Palpation:** Normal tactile fremitus in both lung fields.
  - **Percussion:** Sonorous (resonant) in both lung fields.
  - **Auscultation:** Normal breath sounds; fine crackles (rales) detected in both lung bases.
- **Heart:**
  - **Inspection:** Non-visible apical impulse (ictus cordis).
  - **Palpation:** Palpable apical impulse, 2 fingers lateral to the left midclavicular line at the 6th intercostal space (ICS VI).
  - **Percussion:** Upper border at the left 2nd ICS; right border at the right parasternal line; left border 2 fingers lateral to the left midclavicular line.
  - **Auscultation:** Heart Rate (HR) 97 bpm, regular; Grade 3/6 systolic ejection murmur at the right 2nd ICS, radiating to the right carotid artery (neck).

Abdomen

- **Inspection:** Slightly distended (convex).
- **Palpation:** Soft, no hepatomegaly or splenomegaly (liver and spleen not palpable).
- **Percussion:** Tympanic; shifting dullness (-).
- **Auscultation:** Normal bowel sounds.

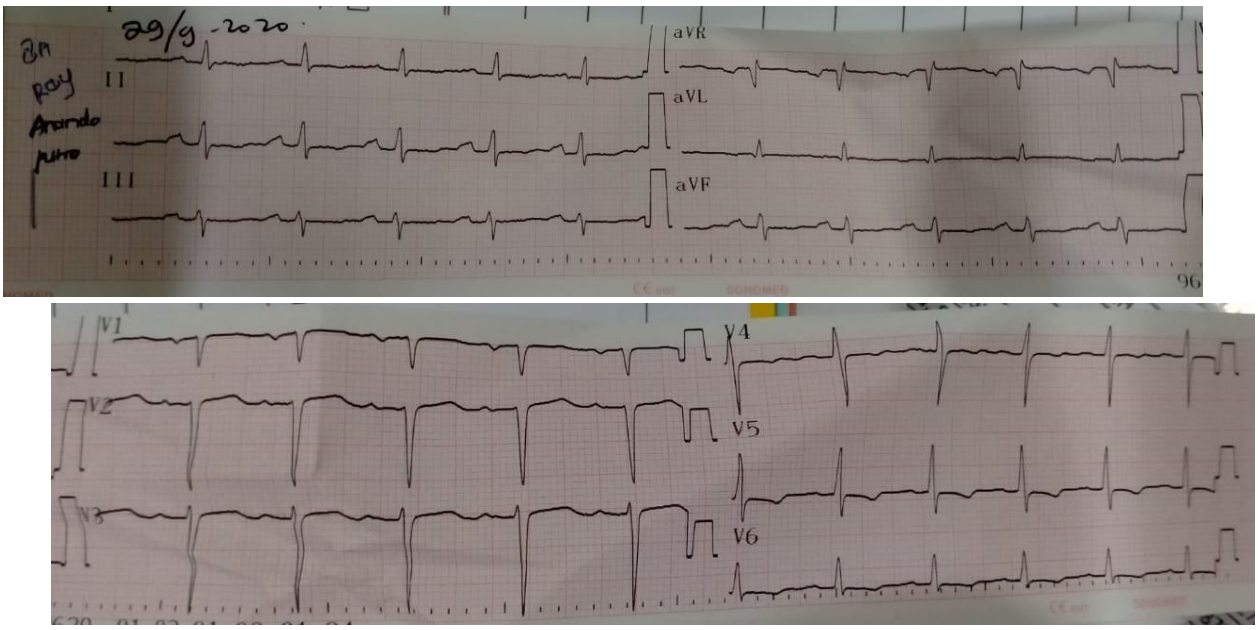
Genitalia & Extremities

- **External Genitalia:** Scrotal edema (-).
- **Extremities:** Pretibial edema (-), digital cyanosis (-), clubbing fingers (-).

Supporting investigation.
EKG (29-09-2020) :

Kepala :

97x/menit (regular isi dan tegangan cukup), RR : 26 x/ menit, T : 36^{o}C. BB : 65 kg TB : 173 cm, BBI = 21,72% (BBI Normal)
Kepala :



ELECTROCARDIOGRAM (ECG)

Rhythm: Sinus Rhythm (SR) Axis: Normal Heart Rate (HR): 100 bpm Findings:

- **P-wave:** P-mitrale in Lead II and inverted P-wave in V1.
- **Intervals:** PR interval 0.20 s; QRS duration 0.08 s.
- **Voltage Criteria:** SD+SV4>2.3 mV (Positive for Left Ventricular Hypertrophy [LVH] based on Peguero-Lo Presti criteria).

Conclusion: Normal Sinus Rhythm with Left Atrial Enlargement (LAE) and Left Ventricular Hypertrophy (LVH).

Foto Torak 29 September 2020

Berikut adalah terjemahan untuk hasil pemeriksaan **Radiologi (Chest X-Ray)** dalam terminologi medis internasional yang baku:

CHEST X-RAY (CXR)

Findings: The trachea is centrally located, and the superior mediastinum is not widened. There is an increase in bronchovascular markings with evidence of cephalization. No infiltrates or nodules are observed in either lung field. The cardiac silhouette is enlarged (cardiomegaly). Both the right and left diaphragms appear smooth, and the costophrenic angles remain sharp. The bony structures and soft tissues are within normal limits.

Impression:

- Cardiomegaly
- Pulmonary edema
- No radiographic evidence of pneumonia

LABORATORY RESULTS

Table 1. Laboratory Findings (September 20, 2020)

- Hematology: Hb: 15.9 g/dL, WBC: 13,090/mm³, Hct: 48%, Platelets: 271,000/mm³.
- Electrolytes & Chemistry: Calcium: 8.4 mg/dL, SGOT: 26 U/L, SGPT: 28 U/L, Albumin: 3.6 g/dL, Random Blood Glucose: 78 mg/dL.
- Renal Function: Urea: 36 mg/dL, Creatinine: 1.35 mg/dL (Elevated), Sodium: 137 mEq/L, Potassium: 4.1 mEq/L.
- Serology: SARS-CoV-2 IgG/IgM Antibody: Non-Reactive.

Laboratory Findings (September 01, 2020 – Cardiology Outpatient Clinic)

- Quantitative CRP: 13 mg/L (Elevated; Normal <5).
- ASO (Antistreptolysin O): Reactive.
- Rheumatoid Factor: Non-Reactive.

Laboratory Findings (August 25, 2020 – BBLK Palembang)

- WBC: 15,300/µL (Leukocytosis).
- Lipid Profile: Total Cholesterol: 216.50 mg/dL, Triglycerides: 68.20 mg/dL.
- Metabolic: Uric Acid: 9.09 mg/dL (Elevated), ALT/SGPT: 71.38 U/L (Elevated).
- Urinalysis: Appearance: Slightly turbid, Specific Gravity: >1.030, Protein: +1 (Proteinuria), Glucose/Blood/Ketone/Nitrite: Negative.

Laboratory Findings (August 28, 2020)

- Hepatitis Markers: HBsAg: Non-Reactive, Anti-HBs: Non-Reactive.
- Total Protein: 5.60 g/dL (Low).

ECHOCARDIOGRAPHY (September 01, 2020)

Findings:

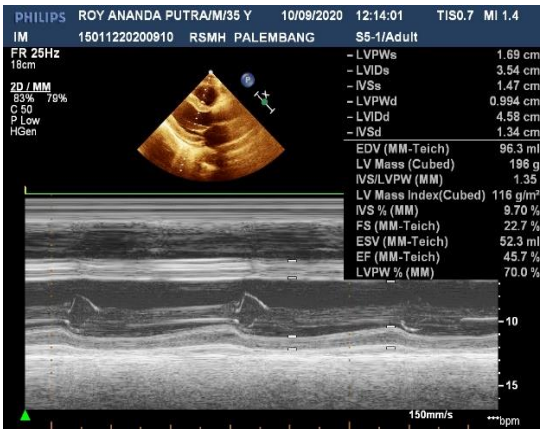
- Chamber Dimensions: Left Ventricular (LV) dilatation.
- Wall Motion: Hypokinetic global wall motion.
- Valvular Assessment:
  - Aortic Valve: Severe Aortic Stenosis (AS) with evidence of vegetations.
  - Mitral Valve: Mild Mitral Regurgitation (MR).
  - Aortic Valve: Mild Aortic Regurgitation (AR).
  - Tricuspid Valve: Moderate Tricuspid Regurgitation (TR).
- Functional Assessment:
  - Left Ventricular Function: Ejection Fraction (EF): 45.7% (Reduced).

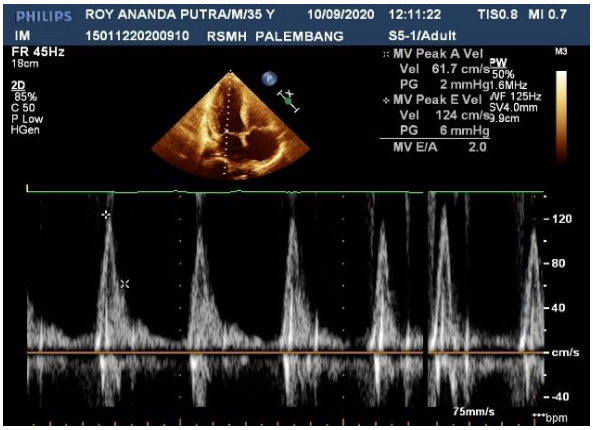
    - Diastolic Function: Grade III diastolic dysfunction.

Impression:

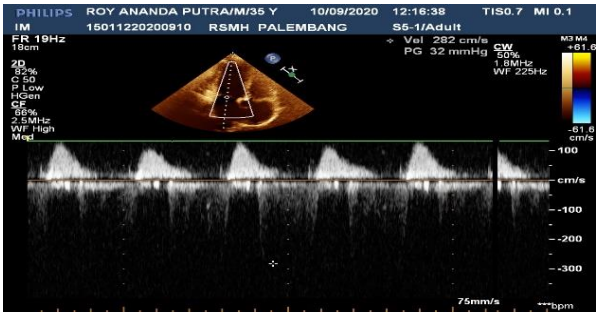
- Severe Aortic Stenosis secondary to Rheumatic Heart Disease (RHD).
- Moderate Pulmonary Hypertension (PH).
- Reduced Left Ventricular Function.

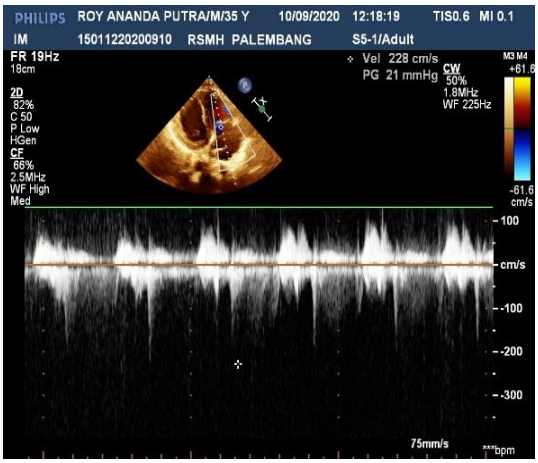
.

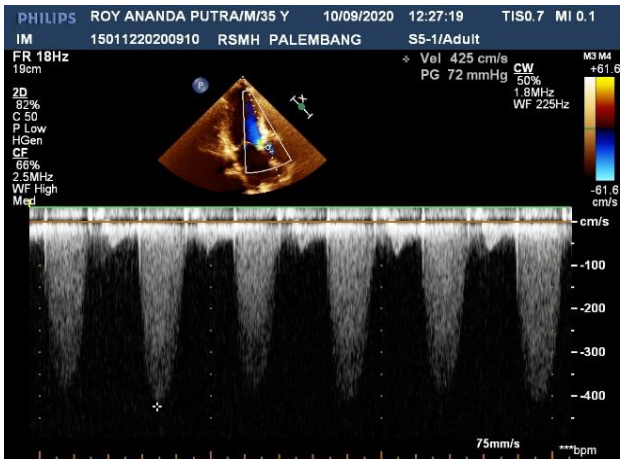

PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:14:01 TIS0.7 MI 1.4
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 25Hz
18cm
2D / MM
LVPWs 1.69 cm
LVIDs 3.54 cm
IVSs 1.47 cm
LVPWd 0.994 cm
LVIDd 4.58 cm
IVSd 1.34 cm
EDV (MM-Teich) 96.3 ml
LV Mass (Cubed) 198 g
IVS/LVPW (MM) 1.35
LV Mass Index(Cubed) 116 g/m²
IVS % (MM) 9.70 %
FS (MM-Teich) 22.7 %
ESV (MM-Teich) 52.3 ml
EF (MM-Teich) 45.7 %
LVPW % (MM) 70.0 %
150mm/s
***bpm

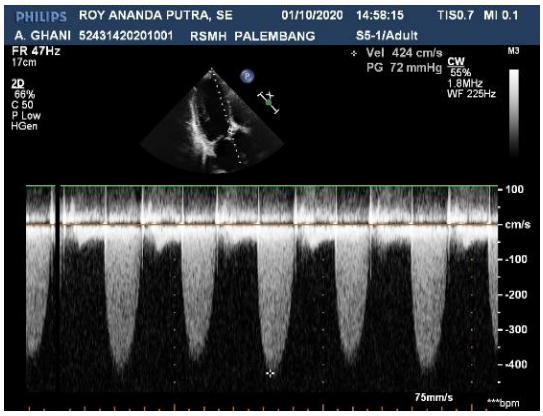

PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:11:22 TIS0.8 MI 0.7
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 45Hz
18cm
MV Peak A Vel
Vel 61.7 cm/s
PG 2 mmHg
MV Peak E Vel
Vel 124 cm/s
PG 6 mmHg
MV E/A 2.0
120
80
40
cm/s
-40
75mm/s
***bpm


PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:09:54 TIS0.7 MI 1.4
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 25Hz
18cm
LA Dimen 3.9 cm
AoR Diam 2.9 cm
LA/Ao (MM) 1.34
150mm/s
***bpm


PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:16:38 TIS0.7 MI 0.1
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 19Hz
19cm
Vel 282 cm/s
PG 32 mmHg
100
cm/s
-100
-200
-300
75mm/s
***bpm


PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:18:19 TIS0.6 MI 0.1
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 19Hz
18cm
Vel 228 cm/s
PG 21 mmHg
CW
50%
1.8MHz
WF 225Hz
100
cm/s
-100
-200
-300
75mm/s
***bpm


PHILIPS ROY ANANDA PUTRA/M/35 Y 10/09/2020 12:27:19 TIS0.7 MI 0.1
IM 15011220200910 RSMH PALEMBANG S5-1/Adult
FR 18Hz
19cm
Vel 425 cm/s
PG 72 mmHg
CW
50%
1.8MHz
WF 225Hz
cm/s
-100
-200
-300
-400
75mm/s
***bpm



DIAGNOSIS

Congestive Heart Failure (CHF) secondary to Severe Aortic Stenosis (AS) of Rheumatic origin (RHD), complicated by Acute Pulmonary Edema, Secondary Pulmonary Hypertension (PH), and Acute Kidney Injury (AKI).

THERAPEUTIC MANAGEMENT

The patient received the following medical regimen:

- **Intravenous (IV) Access:** Normal Saline (NaCl 0.9%) at a rate of 10 micro-drops per minute.
- **Diuretics:** IV Furosemide 20 mg (1 ampule) every 12 hours and oral Spironolactone 12.5 mg once daily.
- **Inotropic Support:** Oral Digoxin 0.25 mg once daily.
- **Antiplatelet Therapy:** Aspirin (Aspilet) 80 mg once daily.
- **Gastroprotective Agent:** IV Ranitidine 50 mg (1 ampule) every 12 hours.
- **Electrolyte Supplement:** Potassium Chloride (KSR) once daily.
- **Bowel Management:** Bisacodyl (Dulcolax) suppository as needed (*pro re nata*) and Liquid Paraffin/Glycerin (Laxadine syrup) 15 mL (1 tablespoon) twice daily.

SPECIALIST CONSULTATIONS

Thoracic and Cardiovascular Surgery (October 02, 2020)

- **Assessment:** Currently, no surgical intervention is indicated from a Thoracic and Cardiovascular Surgery standpoint.
- **Recommendation:** Continued observation and monitoring of the patient's clinical status are required before further surgical planning. *(Consultant: Dr. Gama, Sp.BTKV)*

Dentistry and Oral Maxillofacial (October 01, 2020)

- **Diagnosis:** Retained Roots (Gangrene Radix) and Gingivitis.
- **Recommendation:** To eliminate the source of infection, tooth extraction and scaling are recommended. The procedure may be performed once written clearance/consent from the primary physician (Cardiology) is obtained. The patient is advised to improve oral hygiene.

Allergy and Clinical Immunology Division (October 02, 2020)

- **Assessment:** Congestive Heart Failure (CHF) secondary to Aortic Stenosis (AS), suspected Rheumatic Heart Disease (RHD) + Moderate Pulmonary Hypertension (PH) + History of Hepatitis B infection.
- **Recommendation:** Evaluate Erythrocyte Sedimentation Rate (ESR/LED) and proceed with the

Dentistry consultation for focal infection screening.

CLINICAL FOLLOW-UP (September 30 – October 03, 2020)
Subjective (S): The patient reported a gradual improvement in dyspnea (shortness of breath).

Objective (O):

- **Blood Pressure (BP):** 80–100 / 60–70 mmHg
- **Heart Rate (HR):** 90–100 bpm
- **Respiratory Rate (RR):** 20–22 breaths/min
- **Temperature (T):** 36.5°C – 37.0°C

Assessment (A): Congestive Heart Failure (CHF) secondary to Aortic Stenosis (AS) of Rheumatic origin (RHD), complicated by secondary Pulmonary Hypertension (PH), Acute Kidney Injury (AKI), and resolved Pulmonary Edema.

Plan (P):

- **Inotropic Support:** IV Dobutamine (1 ampule in 100 cc Normal Saline) at a rate of 10 micro-drops per minute.
- **Diuretics:** Discontinued IV Furosemide; transitioned to oral Furosemide 20 mg (1/2 tablet) twice daily; Spironolactone 12.5 mg once daily.
- **Beta-Blocker:** Low-dose Bisoprolol 0.625 mg once daily.
- **Antiplatelet:** Aspirin (Aspilet) 80 mg once daily.
- **Other Medications:** Digoxin 0.25 mg once daily, Potassium Chloride (KSR) once daily, Coenzyme Q10 once daily.
- **Bowel Management:** Bisacodyl (Dulcolax) suppository as needed (*pro re nata*).

Berikut adalah terjemahan bagian **Diskusi (Discussion)** dalam format jurnal medis yang mengalir secara naratif dan menggunakan bahasa akademik yang kuat:

DISCUSSION

Aortic stenosis (AS) is classically characterized by a clinical triad of heart failure, angina, and syncope. In this case, the patient's history revealed dyspnea, dizziness, intermittent chest pain, and a history of syncope. The dyspnea was indicative of heart failure, as evidenced by its exertional nature and the presence of orthopnea, requiring the patient to use 2–3 pillows to sleep. This clinical assessment was further supported by radiographic findings of cardiomegaly.

Physical examination remains a vital diagnostic tool; auscultation in AS typically reveals a systolic murmur at the second right intercostal space (ICS). Consistent with this, a grade 3/6 systolic murmur was identified at the same location in this patient.

Echocardiography is the cornerstone for evaluating AS, providing essential data on valvular anatomy, the extent of calcification, left ventricular dimensions, and hemodynamic severity. It is also pivotal in determining prognosis and the optimal timing for intervention. Doppler echocardiography allows for the calculation of the peak pressure gradient using the modified Bernoulli equation:

$$\Delta P = 4 \times (VAV)2$$

where VAV represents the transvalvular peak velocity. In this patient, the echocardiogram demonstrated a peak

pressure gradient of 72 mmHg with a VAV of 425 cm/s (4.25 m/s) and an Ejection Fraction (EF) of 45.7%. These findings classify the patient as having **Symptomatic Severe Aortic Stenosis with High Gradient.**

The etiology of AS varies; while senile calcification is common in the elderly, inflammatory processes such as Rheumatic Heart Disease (RHD) are more prevalent in younger patients. In this case, RHD is the most likely etiology, supported by the patient's young age and positive inflammatory markers, including elevated CRP and reactive ASO titers.

The clinical indications for intervention in symptomatic aortic stenosis include:

1. Patients with a high mean gradient and reduced EF.
2. Patients with low-flow, low-gradient AS and reduced EF due to afterload mismatch, where LV function is expected to improve post-intervention.
3. Patients with low-flow, low-gradient AS and preserved EF.
4. Patients with normal-flow, low-gradient AS and preserved EF.

Based on these criteria, the patient in this case falls into the first category: **severe aortic stenosis with a high gradient and reduced ejection fraction**, making him a clear candidate for valvular intervention.

CONCLUSION

We have reported a case of Symptomatic Severe Aortic Stenosis secondary to Rheumatic Heart Disease (RHD) with a high pressure gradient. This condition was diagnosed only one month prior to admission following the onset of clinical symptoms, including dyspnea and chest pain. The current pharmacological management includes intravenous Dobutamine infusion at 10 micro-drops per minute, Spironolactone 12.5 mg once daily, Aspirin (Aspilet) 80 mg once daily, Digoxin 0.25 mg once daily, Potassium Chloride (KSR) once daily, Furosemide 20 mg once daily, Bisoprolol 0.625 mg once daily, and Coenzyme Q10 once daily, with symptomatic treatment using Bisacodyl (Dulcolax) and Liquid Paraffin (Laxadine). Given the severity and symptomatic nature of the disease, Aortic Valve Replacement (AVR) is highly recommended as the definitive intervention.

REFERENCES

1. M. Panggabean Marulam et al. Stenosis Aorta. Setiaiti S, Alwi I, Sudoyo A, Simadibrata SB, Fahrial A, Editor : Buku Ilmu Penyakit Dalam PAPDI. Edisi VI.2014;1188-91.
2. Thoenes Martin. Patient screening for early detection of aortic stenosis (AS)—review of current practice and future perspectives.
3. Eveborn Gry Wisthus et al. The evolving epidemiology of valvular aortic stenosis. The Tromsø Study. Eveborn GW, et al. Heart 2013;99:396–400.
4. Watkins David A et al. Rheumatic Heart Disease Worldwide. JACC.72.No.12.2018 September 18: 1397-416
5. H. Brian. Aortic Stenosis: Diagnosis and Treatment. . (Am Fam Physician. 2016 ;93(5):371-378.
6. Kanwar et al. Management of Patient With Aortic Valve Stenosis. Mayo Clin Proc.2018;93(4):488-508.
7. J. Matthew et al. Diagnosis and Management of Valvular Aortic Stenosis. Clinical Medicine Insight : Cardiology 2014:8(s1)15-24.

8. Juan Siordia. Transcatheter Aortic Valve Replacement. A Review Article. J Cardiothorac Med.2016;4(2):423-436.
9. Everett RJ et al. Timing of intervention in aortic stenosis : a review of current and future strategies. Heart 2018;104:2067-2076.
10. Pibarot P. Moderate Aortic Stenosis and Heart Failure with Reduced Ejection Fraction. JACC : Cardiovascular Imaging vol 12.No 1,2019:172-84.
11. Taramasso M. Review Article Emerging Approaches of Transcatheter Valve Repair. Cardiology Research and Practice Vol 2010.
12. Taniguchi Tomohiko et al. Contemporary issues in severe aortic stenosis: review of current and future strategies from the Contemporary Outcomes after Surgery and Medical Treatment in Patients with Severe Aortic Stenosis registry. Taniguchi T, et al. Heart 2020;106:802–809.
13. Baumgartner Helmut et all. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. European Journal of Echocardiography (2009) 10, 1–25.
14. Andrei D Mărgulescu. Assessment of aortic valve disease - a clinician oriented review. World J Cardiol 2017 June 26; 9(6): 481-495.
15. Wagner Gernot et al. Comparison of transcatheter aortic valve implantation with other approaches to treat aortic valve stenosis: a systematic review and meta-analysis. Wagner et al. Systematic Reviews (2019) 8:44.
16. Salah AM Said. Currently Applied Non-Surgical Techniques for Treatment of Severe Aortic Stenosis in Inoperable High-Risk Adult Patients. J Clinic Experiment Cardiol 2011, S:3.
17. Baumgartner et al. 2017/ESC/EACT Guidelines for the management of Valvular Heart Disease. European Heart Journal (2017) 38,2739-2791.