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Bentall Procedure as a Lifesaving Surgery: Aortic Dissection Stanford A Debakey Type II and Severe Aortic Regurgitation Suspected Marfan Syndrome

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Abstract: Aortic dissection is a life-threatening condition often associated with connective tissue disorders, particularly Marfan syndrome, which predisposes individuals to aortic dilation and rupture. The Bentall procedure, which involves the replacement of the ascending aorta and aortic valve, is an essential surgical intervention for patients with aortic dissection and valve disease. This study examines the role of the Bentall procedure in managing Stanford A DeBakey Type II aortic dissection, with a focus on patients with Marfan syndrome. The findings highlight the importance of early diagnosis, effective pharmacological management, and timely surgical intervention to improve patient outcomes. While recent studies show promising results, further research is needed to assess long-term outcomes and refine surgical techniques, including minimally invasive approaches, to reduce complications and enhance recovery.

Keywords: *Aortic Dissection; Bentall Procedure; Marfan Syndrome; Aortic Valve Replacement; Surgical Intervention.*

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I. INTRODUCTION

Aortic diseases encompass a variety of conditions that can affect this large artery, such as aortic aneurysm, aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer (PAU), traumatic aortic injury, pseudoaneurysm, aortic rupture, atherosclerosis, and inflammation (Papachristodoulou et al., 2025; Vignaraja et al., 2024). According to data from the Global Burden of Disease 2010, mortality rates from aortic aneurysms and dissections increased between 1990 and 2010, with higher rates among men. Ascending aortic dissection, which accounts for 58–62% of all aortic dissections, has a high mortality rate, reaching 73%. The annual incidence of aortic dissection is estimated to be approximately 2.5–6 cases per 100,000 population (Erbel et al., 2014). Marfan syndrome, an autosomal dominant connective tissue disorder, is often associated with aortic dissection due to mutations in the FBN1 gene, which encodes fibrillin-1, an important component of elastic microfibrils in connective tissue, particularly in the medial layer of the ascending aorta. Patients with Marfan syndrome are at high risk of developing aortic dissection, which can cause severe chest pain radiating to the back, often difficult to distinguish from symptoms of angina due to acute coronary syndrome (Grewal & Gittenberger-de Groot, 2018; Hiratzka et al., 2010).

Aortic dissection is a life-threatening medical condition that requires immediate intervention. One approach used in managing cases of aortic dissection with severe aortic regurgitation is the Bentall procedure. The Bentall procedure is particularly relevant for patients with DeBakey Type II aortic dissection, where the dissection is localized to the ascending aorta and is often associated with connective tissue disorders such as Marfan syndrome. Stanford type A aortic dissection, which includes DeBakey types I and II, is the most common form and requires immediate surgical intervention to prevent acute mortality (Lin et al., 2021; Nardi et al., 2021). Marfan syndrome, a genetic disorder leading to connective tissue abnormalities, can cause aortic dilation and dissection, which is one of the leading causes of death in individuals with the condition (Graf, 2022). Characteristic symptoms such as ectopia lentis, commonly found in Marfan syndrome, can play a crucial role in the diagnosis and management of aortic dissection. Given the high risk posed by aortic pathology, the Bentall procedure plays a vital role in reconstructing the aortic root and associated valves, aiming to restore hemodynamic stability and prevent further complications from aortic regurgitation and progressive dissection (Gupta et al., 2021).

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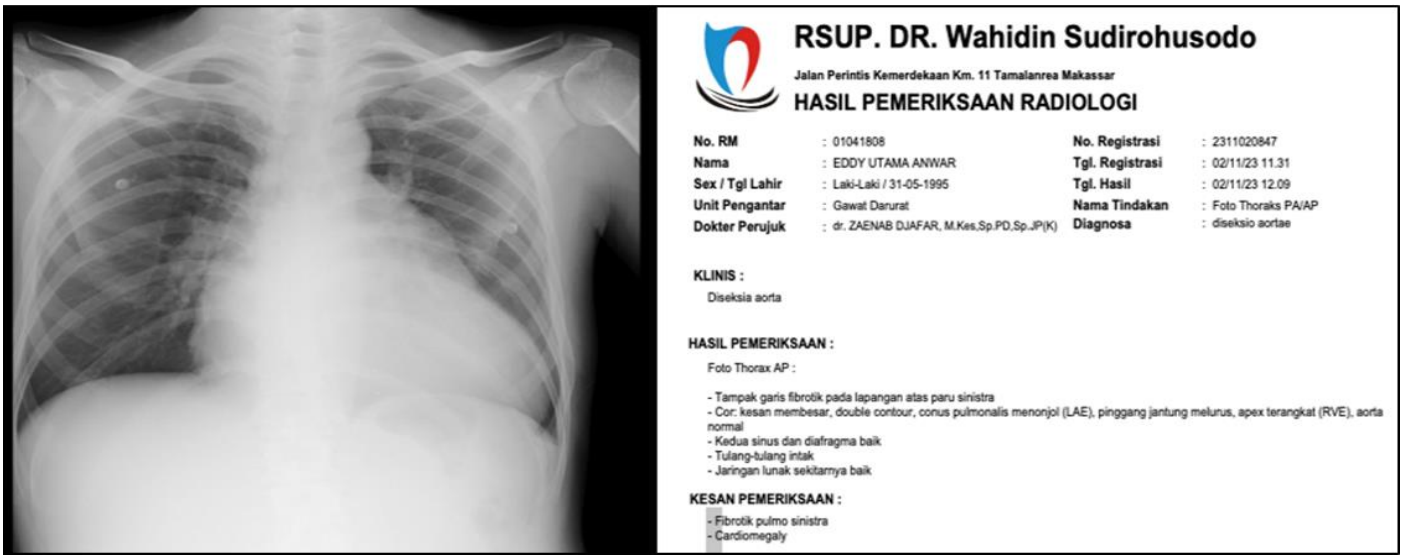
Recent studies have shown good outcomes after the Bentall procedure in patients with DeBakey Type II aortic dissection, demonstrating improved postoperative outcomes compared to patients with more extensive dissection (Biancari et al., 2024; Khachatryan et al., 2021; Le et al., 2024). Patients with Type II dissection tend to have better surgical outcomes, particularly in terms of intraoperative mortality and postoperative complications (Nardi et al., 2021). However, the success of surgical repair is often influenced by the extent of the dissection and the presence of additional factors, such as myocardial hypoperfusion, which can worsen the intervention and increase morbidity (Lin et al., 2021). Nevertheless, the Bentall procedure has evolved with several modifications, including minimally invasive techniques that demonstrate advantages in terms of faster recovery times and reduced complications related to postoperative ventilation (Shah et al., 2021). These modifications are particularly relevant for high-risk patients with aortic dissection, as they help reduce complications typically associated with more extensive surgical approaches. However, despite these advancements, further research is needed to explore these outcomes in greater depth, as well as the impact of various techniques and patient profiles, to provide more precise guidance in tailored management strategies.

This study aims to further examine the Bentall procedure in the management of aortic dissection, particularly in patients with Marfan syndrome who also have severe aortic regurgitation. By reviewing clinical experience and outcomes of this procedure, this study is expected to provide further insights into the effectiveness and postoperative outcomes in patients with DeBakey type II aortic dissection. Additionally, this study aims to explore the potential modifications of the Bentall technique, including minimally invasive approaches, which may improve surgical outcomes in high-risk patients. The benefits of this study are not limited to the development of better management strategies for aortic dissection but may also contribute to the development of more tailored clinical guidelines for patients with connective tissue disorders such as Marfan syndrome, who are at high risk of aortic complications. By continuing to deepen our understanding of the Bentall procedure, it is hoped that more optimal approaches can be identified to improve patients' quality of life and reduce mortality rates from aortic dissection.

II. CASE REPORT

A 28-year-old male patient was admitted to the Emergency Department of the Wahidin Sudirohusodo Integrated Heart Center with complaints of shortness of breath that had been felt since the previous day and had occurred frequently over the past few months, accompanied by chest pain in the middle of the chest that felt like being cut, torn, and burning, radiating to the back without nausea or vomiting. The patient also experienced dyspnea on exertion (DoE), paroxysmal nocturnal dyspnea (PND), and orthopnea, but no fever or cough. The patient's medical history indicated no history of hypertension, diabetes mellitus, or family history of heart disease, although he had been smoking for 10 years (half a pack per day). The patient had previously been hospitalized with similar complaints and was diagnosed with acute decompensated heart failure, aortic dissection, and severe aortic regurgitation, and received routine therapy with furosemide, spironolactone, bisoprolol, digoxin, captopril, and lansoprazole. Physical examination revealed the patient was in severe condition with adequate nutritional status (height 184 cm, weight 70 kg, BMI 20.7 kg/m²), alert and oriented, blood pressure 145/55 mmHg (right arm), 128/65 mmHg (left arm), heart rate 105 beats per minute, respiratory rate 26 breaths per minute, and oxygen saturation 99% with nasal cannula. Further physical examination revealed minimal pitting edema on the bilateral dorsum of the feet and several characteristic signs of severe aortic regurgitation, such as Corrigan sign, Landolfi sign, Muller sign, Rosenbach sign, Traube sign, and Mayne sign. Cardiac examination revealed a grade 4/6 systolic murmur in the left lower anterior (LLSB) and a grade 3/4 diastolic murmur in the right upper anterior (RUSB), as well as hepatomegaly with the liver palpable 4 fingers below the costal arch.



RSUP. DR. Wahidin Sudirohusodo

Jalan Perintis Kemerdekaan Km. 11 Tamalanrea Makassar

HASIL PEMERIKSAAN RADIOLOGI

No. RM	: 01041808	**No. Registrasi**	: 2311020847
Nama	: EDDY UTAMA ANWAR	**Tgl. Registrasi**	: 02/11/23 11.31
Sex / Tgl Lahir	: Laki-Laki / 31-05-1995	**Tgl. Hasil**	: 02/11/23 12.09
Unit Pengantar	: Gawat Darurat	**Nama Tindakan**	: Foto Thoraks PA/AP
Dokter Perujuk	: dr. ZAENAB DJAFAR, M.Kes,Sp.PD,Sp.JP(K)	**Diagnosa**	: diseksio aortae

KLINIS :

Diseksia aorta

HASIL PEMERIKSAAN :

Foto Thorax AP :

- Tampak garis fibrotik pada lapangan atas paru sinistra
- Cor: kesan membesar, double contour, conus pulmonalis menonjol (LAE), pinggang jantung melurus, apex terangkat (RVE), aorta normal
- Kedua sinus dan diafragma baik
- Tulang-tulang intak
- Jaringan lunak sekitarnya baik

KESAN PEMERIKSAAN :

- Fibrotik pulmo sinistra
- Cardiomegaly

Fig 1 Chest X-ray at PJT (November 2, 2023)

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Interpretation: Enlarged heart with CTI 0.66, double contour, prominent pulmonary cone (LAE), straightened cardiac waist, elevated apex (RVE). Conclusion: Cardiomegaly

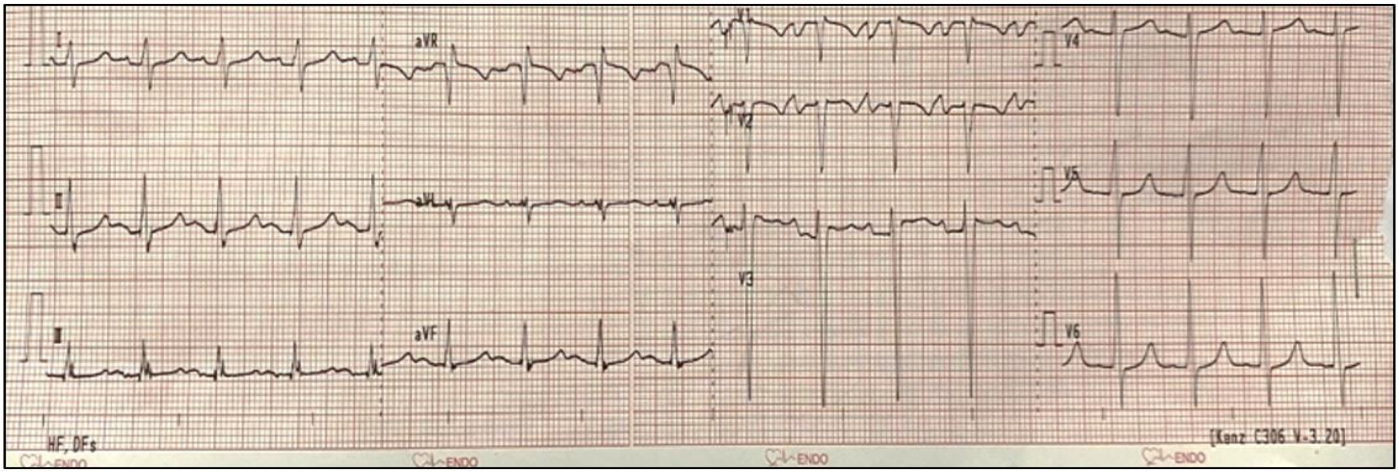

Fig 2 Electrocardiogram (ECG)

Sinus tachycardia, rate 105 beats per minute, regular, normal axis, P wave 0.12 seconds (the final negative portion of the P wave in V1 is >40 ms in width and >1 mm in depth), PR interval 0.20 seconds, QRS duration 0.10 seconds, inverted T waves in V1-V2, left ventricular hypertrophy (Cornell criteria), QTc 476 msec (Bazzet formula)

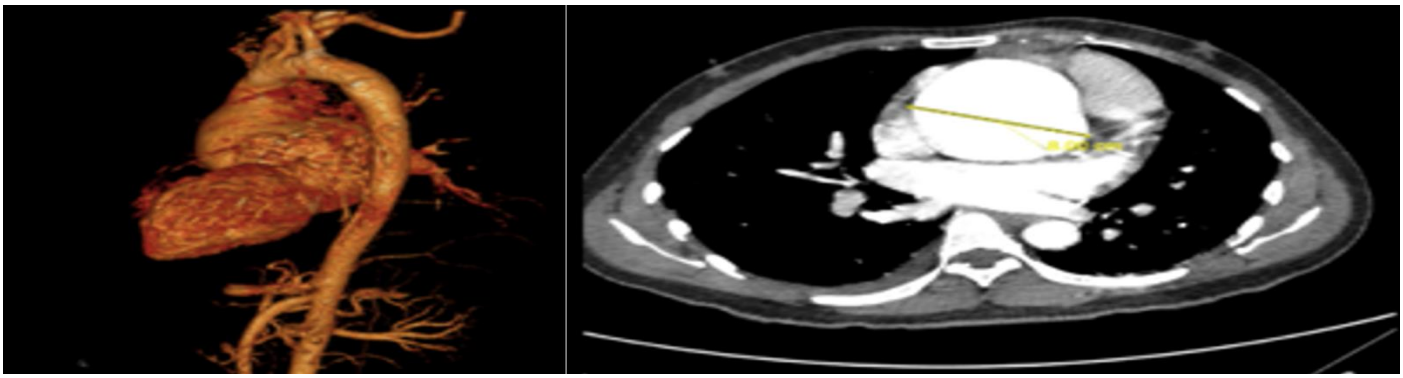

Fig 3 MSCT Thoraco-abdominal Angiography RSWS (August 31, 2023)

Conclusion: Ascending aorta dilation accompanied by tunica intima flap (double lumen) indicating Stanford A De Bakey Type II aortic dissection, cardiomegaly (LVH), bilateral pleural effusion.

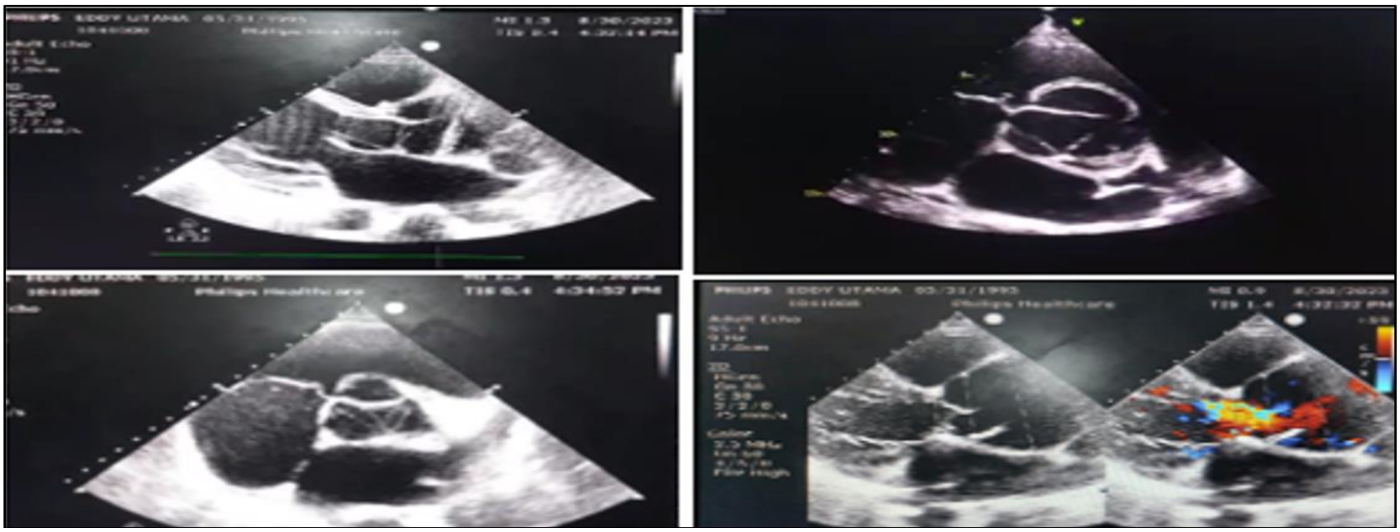

Fig 4 Echocardiography

(PLAX View: aortic root, PSAX Aortic view: Floating membrane with false lumen, true lumen, and dilated aortic root)

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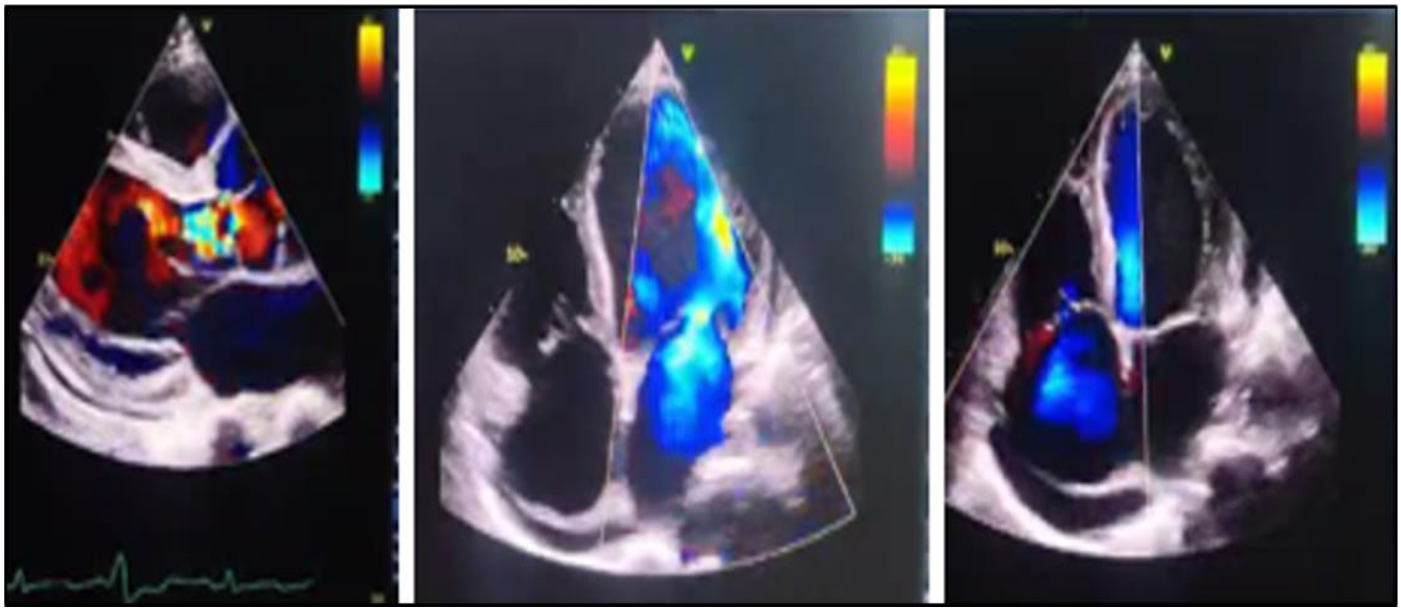

Fig 5 Echocardiography

(PLAX View, Apical Four Chambers: Severe aortic regurgitation, moderate mitral regurgitation, severe tricuspid regurgitation)

Echocardiography revealed severe primary aortic regurgitation due to aortic dissection with measurements of the aortic root: aortic annulus 3.8 cm, sinus valsalva 4.2 cm, sinotubular junction 4.7 cm, and ascending aorta 5.9 cm, accompanied by an intimal flap and holodiastolic reversal flow. There is also severe tricuspid regurgitation, moderate mitral regurgitation, and preserved left ventricular systolic function with an EF of 53%. Right ventricular systolic dysfunction is noted with a TAPSE of 1.3 cm and S' Lat of 8 cm/s, along with mild to moderate pericardial effusion. Further laboratory tests at RSWS are still ongoing, and the following results have been obtained: Laboratory test results of the patient at the Integrated Heart Center of RSWS conducted on various dates between November 2 and November 28, 2023. Routine hematology tests showed fluctuations in white blood cell (WBC) counts, ranging from 10,200/µL and increasing to 19,200/µL on November 27, 2023, indicating a possible infection or inflammation. Additionally, the patient's hemoglobin (Hb) levels fluctuated between 4.8 g/dL and 15.2 g/dL, with significant decreases on November 24 and 26, indicating possible anemia. The platelet count (PLT) was also consistently low, ranging from 72,000 to 164,000/µL, indicating thrombocytopenia in some examinations. Coagulation factor tests showed that PT, INR, and aPTT values frequently exceeded the normal range, indicating coagulation disorders on several examination dates; Blood chemistry tests revealed electrolyte imbalances, particularly sodium (Na) levels, which continued to decline from 131 mmol/L on November 2 to 123 mmol/L on November 16, well below the normal range of 136–145 mmol/L, which may indicate hyponatremia. Potassium (K) levels also fluctuated, with the highest value on November 2 reaching 8.1 mmol/L, indicating hyperkalemia, before returning to normal in subsequent examinations. On November 28, SGOT and SGPT levels increased significantly, reaching 200 U/L and 507 U/L, respectively, indicating liver damage. A very high quantitative CRP level on November 28 (71.4) and extremely high procalcitonin (68.90) suggest significant infection or inflammation. Arterial blood gas analysis revealed metabolic acidosis on November 24, with a low pH (7.192) and extremely low bicarbonate (HCO3) levels, indicating an acidosis state. However, on November 25, pH and other arterial blood gas parameters returned to normal.

Based on the medical history, physical examination, laboratory tests, echocardiography, chest X-ray, and thoracoabdominal CT angiography, the patient was diagnosed with Stanford A DeBakey Type II aortic dissection, Severe Aortic Regurgitation, Severe Tricuspid Regurgitation, Moderate-to-Severe Functional Mitral Regurgitation, Congestive Heart Failure NYHA Class III, Marfan Syndrome, Congestive Liver Disease, Severe Hyperkalemia, and Mild Normoosmolar Hyponatremia. The therapy administered included a furosemide bolus of 80 mg IV, maintenance furosemide 80 mg IV, and other diuretics as indicated. Marfan Syndrome, Congestive Liver Failure, Severe Hyperkalemia, and Mild Normoosmolar Hyponatremia. Treatment included a 80 mg IV furosemide bolus, 40 mg IV furosemide every 8 hours, 5 mg IVabradine every 12 hours orally, 5 mg ramipril every 24 hours orally (delayed), perdipine 5 mcg/kg body weight/minute via an infusion pump, carvedilol 6.25 mg every 12 hours orally (delayed), spironolactone 25 mg every 24 hours orally (delayed), and correction of hyperkalemia with calcium gluconate 10% 10 ml in 100 ml of 0.9% NaCl. The patient was also given potassium chloride 5 grams every 8 hours orally for potassium correction. After 22 days of treatment, the patient was decided to undergo the Bentall procedure.

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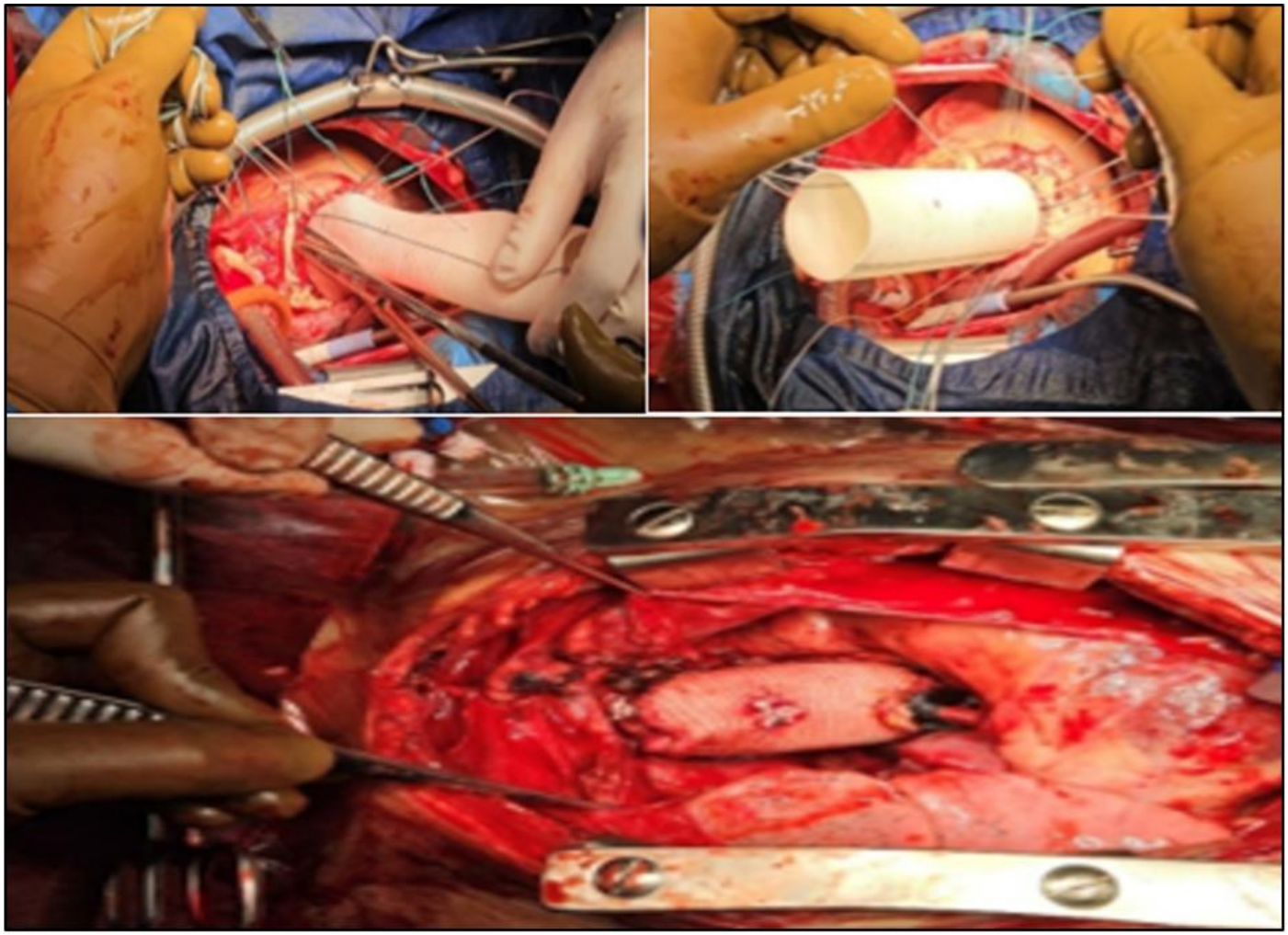

Fig 6 Bentall Procedure

III. DISCUSSION

➢ *Aortic Dissection*

Patients presenting to the hospital with the primary complaint of deep substernal chest pain that feels like being cut or torn, accompanied by pain radiating to the back, demonstrate characteristic clinical signs of aortic dissection. These symptoms have been present for several months, are recurrent, and are accompanied by cardiac congestion such as dyspnea on exertion (DoE), paroxysmal nocturnal dyspnea (PND), and orthopnea. Physical examination revealed a grade 4/6 systolic murmur in the left lower anterior (LLSB) and a grade 3/4 diastolic murmur in the right upper anterior (RUSB), along with hepatomegaly and minimal pitting edema on the bilateral dorsum of the feet. Based on these clinical findings, as well as the presence of characteristic signs of aortic regurgitation such as Corrigan sign, Landolfi sign, Muller sign, Rosenbach sign, Traube sign, and Mayne sign, it was concluded that the patient had aortic dissection with severe aortic regurgitation. Based on echocardiography findings, aortic root dilation and an intimal flap were identified, indicating aortic dissection in the ascending aorta, along with severe tricuspid regurgitation and moderate mitral regurgitation. This diagnosis was supported by the results of thoracoabdominal MSCT, which showed a dissected ascending aorta with true and false lumens, as well as the formation of an intimal flap, leading to a diagnosis of Stanford A DeBakey II aortic dissection (Isselbacher et al., 2022).

Aortic dissection is an acute medical condition characterized by a tear in the aortic intima, causing blood to enter between the intima and adventitia layers. This process results in the formation of two lumens, the true lumen and the false lumen, separated by an intimal flap. In this patient, the likely cause of aortic dissection is Marfan syndrome, which is characterized by connective tissue abnormalities and a predisposition to a more fragile and easily dissected aorta. Several pathognomonic characteristics found in the patient, such as plain pes planus, wrist sign, and thumb sign, support the suspicion of Marfan syndrome. The aortic dissection in this patient was classified as subacute-chronic phase, based on symptoms lasting several months and a history of rehospitalization with similar complaints. The aortic wall remodeling process that occurs in response to this dissection plays a role in worsening the condition of the aorta, increasing the risk of aortic rupture, and adding the risk of further complications, such as congestive heart failure and myocardial ischemia (Erbel et al., 2014; Isselbacher et al., 2022).

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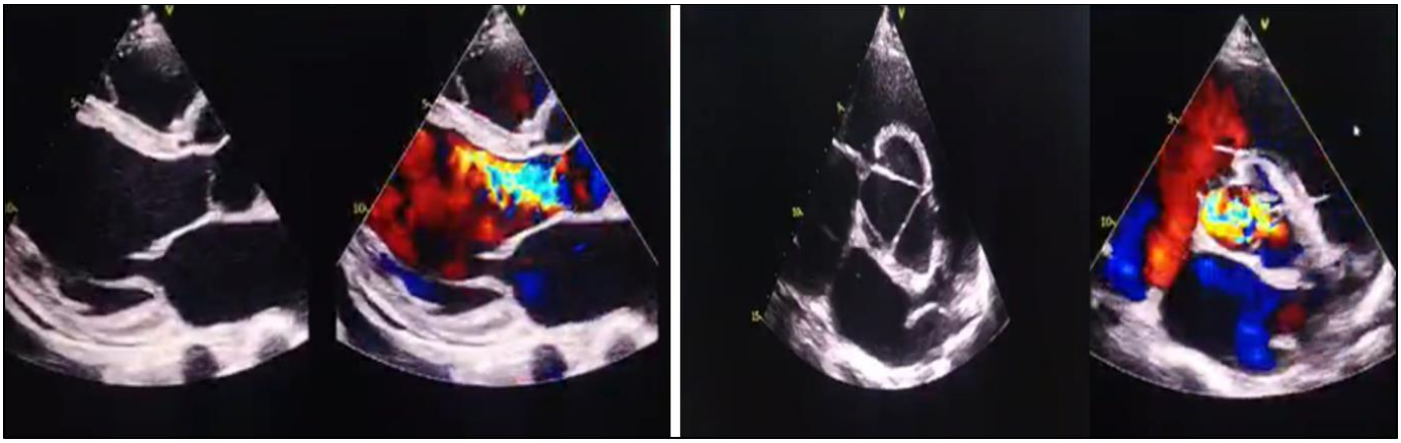

A **B**

Fig 7 View PLAX and PSAX

A. Dilatation of the entire heart chamber accompanied by regurgitant jet at the aortic valve (AR PHT 155 ms, AR VC 1.0 cm, regurgitant jet width > 65%); B. Three-cusped aorta accompanied by false lumen and regurgitant jet passing through the aortic valve

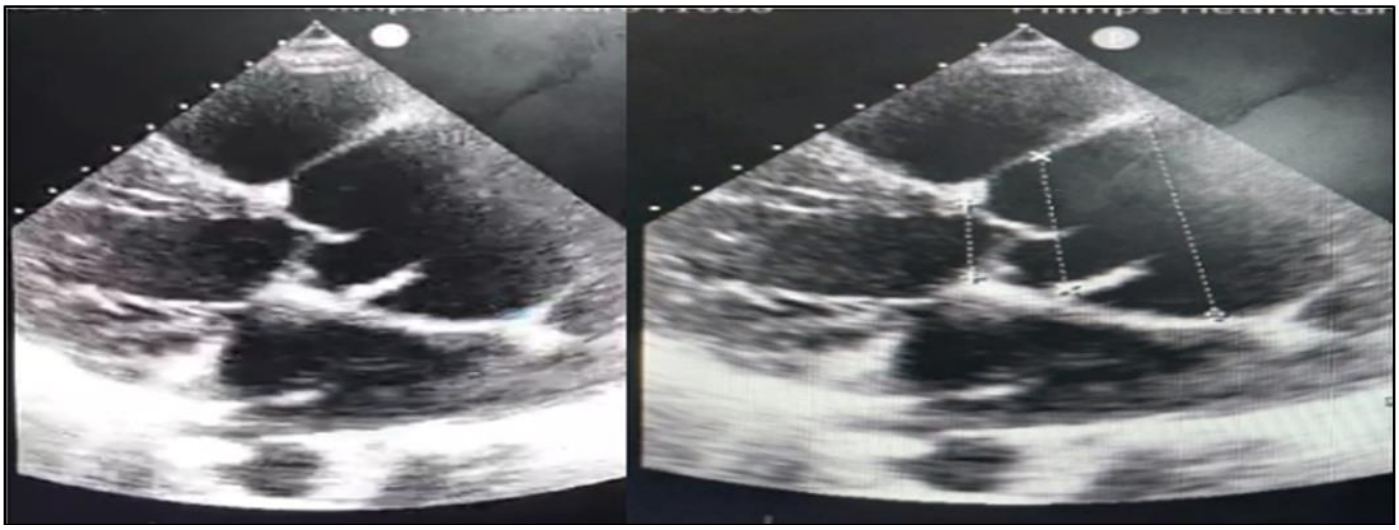

Fig 8 View of PSAX (Aortic Root) Dilated aortic root with (aortic annulus 3.8 cm, sinus valsalva 4.2 cm, Sinotubular Junction 4.7 cm, ascending aorta 5.9 cm) and positive intimal flap

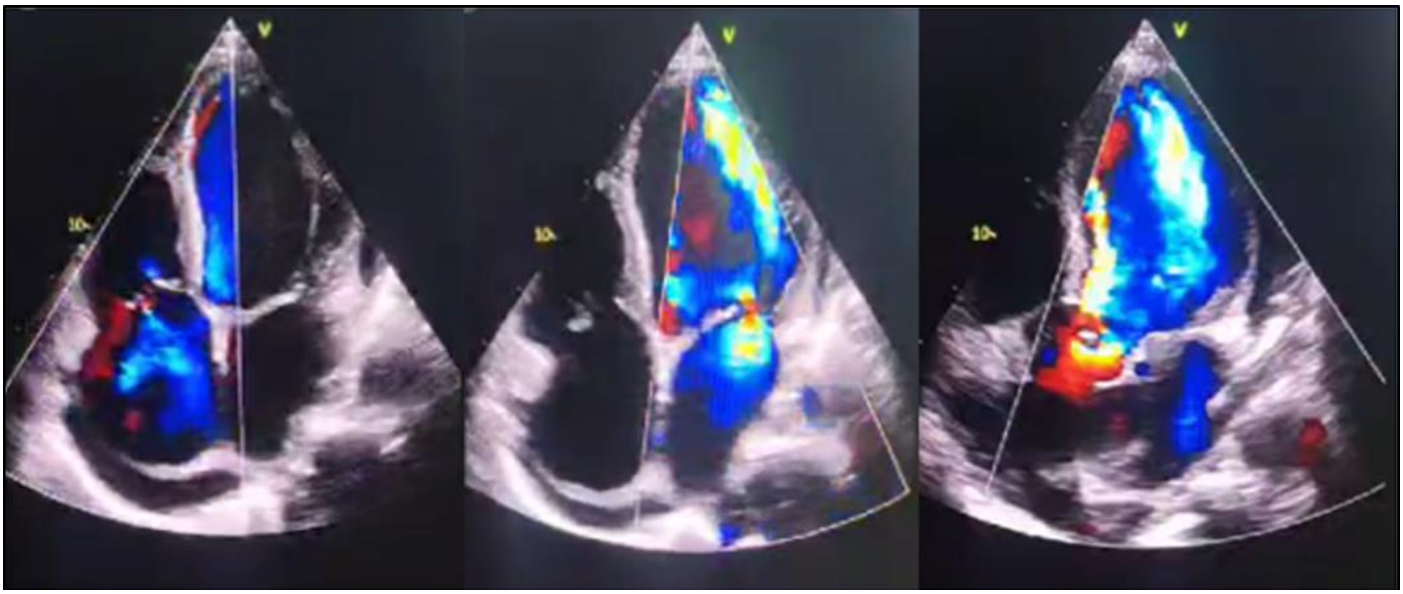

Fig 9 A4C and A5C views Severe tricuspid regurgitation, moderate mitral regurgitation, and severe aortic regurgitation

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To manage this patient, a diagnosis was established based on laboratory test results, echocardiography, and MSCT.

In this patient, further examinations were conducted, including echocardiography and thoracoabdominal aortic MSCT, to confirm the primary diagnosis. Echocardiography revealed the presence of a floating membrane accompanied by true-false lumens and aortic root dilation, suggesting aortic dissection in the ascending aorta, as well as severe aortic regurgitation, severe tricuspid regurgitation, and moderate mitral regurgitation. MSCT of the thoracoabdominal aorta revealed dilation of the ascending aorta, with a tunica intimal flap forming a double lumen (true lumen and false lumen), consistent with a Stanford A Debakey Type II aortic dissection.

These diagnostic tests provided a clear picture of the location of the dissection and the overall condition of the aorta. Using the Aortic Dissection Detection Risk Score system from the ACC/AHA and ESC, this patient was classified as high-risk due to meeting the criteria of chest pain and physical examination findings indicating a high likelihood of aortic dissection. Based on these findings, further medical interventions were initiated, including the administration of diuretic therapy with furosemide, ivabradine, and other medications to manage cardiac symptoms and prevent further complications. Additional therapies included correction of hyperkalemia with calcium gluconate and treatment to control blood pressure. With this approach, it is hoped that the patient's condition can be stabilized, the risk of aortic rupture reduced, and the patient prepared for the planned Bentall procedure as the next therapeutic step (Gawinecka et al., 2017; Isselbacher et al., 2022).

- *Marfan Syndrome*

Marfan syndrome is an autosomal dominant connective tissue disorder caused by mutations in the FBN1 gene, which encodes fibrillin-1, a glycoprotein that forms microfibrils in the extracellular matrix (ECM). These microfibrils are essential for maintaining tissue elasticity and structure, as well as regulating the bioavailability of growth factors such as transforming growth factor beta (TGFβ). Mutations in the FBN1 gene disrupt fibrillin-1 synthesis, leading to damage in the architecture of elastic microfibrils and loss of tissue homeostasis, which further compromises ECM integrity (Grewal & Gittenberger-de Groot, 2018). Additionally, studies indicate that FBN1 mutations increase TGFβ release, playing a role in the cardiovascular pathophysiology of Marfan Syndrome. TGFβ induces signaling through the Angiotensin II receptor (AT2), leading to increased apoptosis and loss of aortic wall integrity, making it stiffer and less distensible (Yu & Jeremy, 2018).

This pathogenic process significantly affects the aortic media, which in Marfan Syndrome undergoes degeneration due to microfibriLLSBr structural damage. Vascular smooth muscle cells (VSMC) in the media become less differentiated and exhibit immaturity. This leads to reduced expression of lamin A/C in smooth muscle cells, contributing to the thinning of elastic fibers and aortic wall weakness. Additionally, the immaturity of VSMC reduces normal levels of FBN1, exacerbating aortic degenerative conditions and increasing the risk of aortic root dilation or dissection, which are characteristic features of this syndrome. Patients with Marfan syndrome may exhibit skeletal manifestations including Plain Pes Planus, Wrist Sign, Thumb Sign, Arachnodactyly, and Dolichostenomelia, which are important indicators for diagnosing this syndrome. A definitive diagnosis of Marfan syndrome can be established if there is lens ectopia, evidence of FBN1 mutations, or the presence of at least seven skeletal findings and systemic manifestations, along with aortic root dilation or dissection (Grewal & Gittenberger-de Groot, 2018).

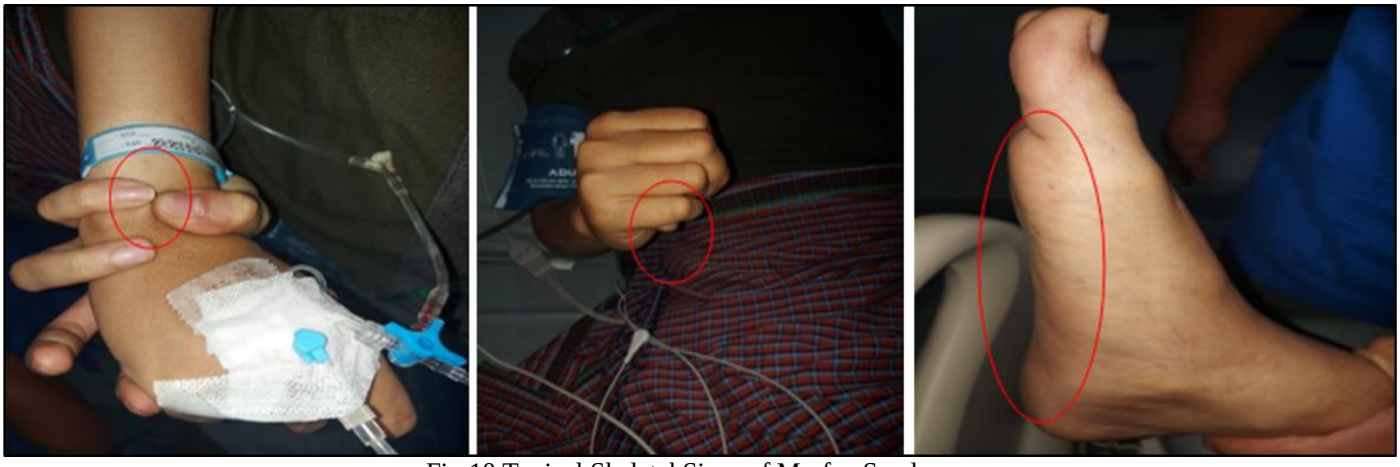

Fig 10 Typical Skeletal Signs of Marfan Syndrome
(Wrist Sign, Thumb Sign, Plain Pes Planus)

In this patient, suspicion of Marfan syndrome was based on physical examination findings of skeletal manifestations (5 points) including flat feet, wrist sign, thumb sign, arachnodactyly, and dolichostenomelia. The definitive diagnosis of Marfan Syndrome is the presence of ectopia lentis, evidence of a mutation in the FBN-1 gene, or the presence of at least 7 points of systemic skeletal

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manifestations accompanied by aortic root dilation or dissection.

In the 2010 nosological revision, diagnostic criteria for Marfan syndrome were divided into several different scenarios. If there is no family history of Marfan syndrome, the diagnosis can be established with four scenarios. First, if there is aortic root dilation or dissection and ectopia lentis, the Marfan diagnosis can be confirmed without the need for other systemic signs. Second, if there is aortic root dilation or dissection and an FBN1 mutation is identified, a diagnosis of Marfan syndrome can be made even in the absence of ectopia lentis. Third, if there is aortic root dilation or dissection without ectopia lentis and the FBN1 status is unknown or negative, the diagnosis can be confirmed with the presence of other systemic findings consistent with the latest scoring system (≥7 points). Finally, if ectopia lentis is present but without aortic root dilation or dissection, FBN1 mutations must be identified to confirm the diagnosis of Marfan syndrome. If FBN1 mutations are not associated with cardiovascular disease, the patient is categorized as ectopia lentis syndrome (Loeys et al., 2010).

- *Management of Aortic Dissection*

Initial pharmacological therapy in patients with aortic dissection aims to control blood pressure and reduce left ventricular pressure, which is important for reducing stress on the aortic wall (Nordon et al., 2011). Maintaining systolic blood pressure below 110 mmHg is key to preventing further damage to the aortic wall, especially in patients who do not undergo open-heart surgery (Gudbjartsson et al., 2020). The first-line therapy for controlling blood pressure in patients with aortic dissection is the use of beta-blockers (BB), which not only lower blood pressure but also heart rate, with a target of 60–80 beats per minute. If patients are intolerant to BBs or do not respond optimally, alternative therapies include non-dihydropyridine calcium channel blockers (CCBs) such as verapamil and diltiazem, which act through negative inotropic and chronotropic effects. However, the long-term use of CCBs for aortic dissection still lacks sufficient evidence (Gudbjartsson et al., 2020). Other antihypertensive agents such as ACE inhibitors, ARBs, and alpha-blockers may be considered if heart rate is controlled with BB or CCB, although there are no strong recommendations for their use in patients with aortic dissection, despite studies suggesting potential benefits of ACE inhibitors in preventing aortic aneurysm progression (Nordon et al., 2011).

This patient was diagnosed with Stanford A DeBakey Type II aortic dissection, accompanied by valvular heart disease involving aortic, tricuspid, and mitral regurgitation. The patient exhibited typical symptoms of aortic dissection and congestive heart failure, with a high risk indicating that immediate surgical intervention was necessary. As medical therapy, the patient was administered furosemide 40 mg/8 hours intravenously, ivabradine 5 mg/12 hours orally, ramipril 5 mg/24 hours orally, perdipine 5 mcg/kg body weight/minute via a syringe pump, carvedilol 6.25 mg/12 hours orally, and spironolactone 25 mg/24 hours orally. Based on the evaluation of the therapy, the patient's systolic blood pressure was within the range of 100-130 mmHg, with a controlled heart rate between 60-80 beats per minute. This therapy was effective in controlling the patient's hemodynamic condition; however, the final decision was to proceed with surgical intervention to address the aortic dissection involving the ascending aorta and damaged aortic valve (Isselbacher et al., 2022).

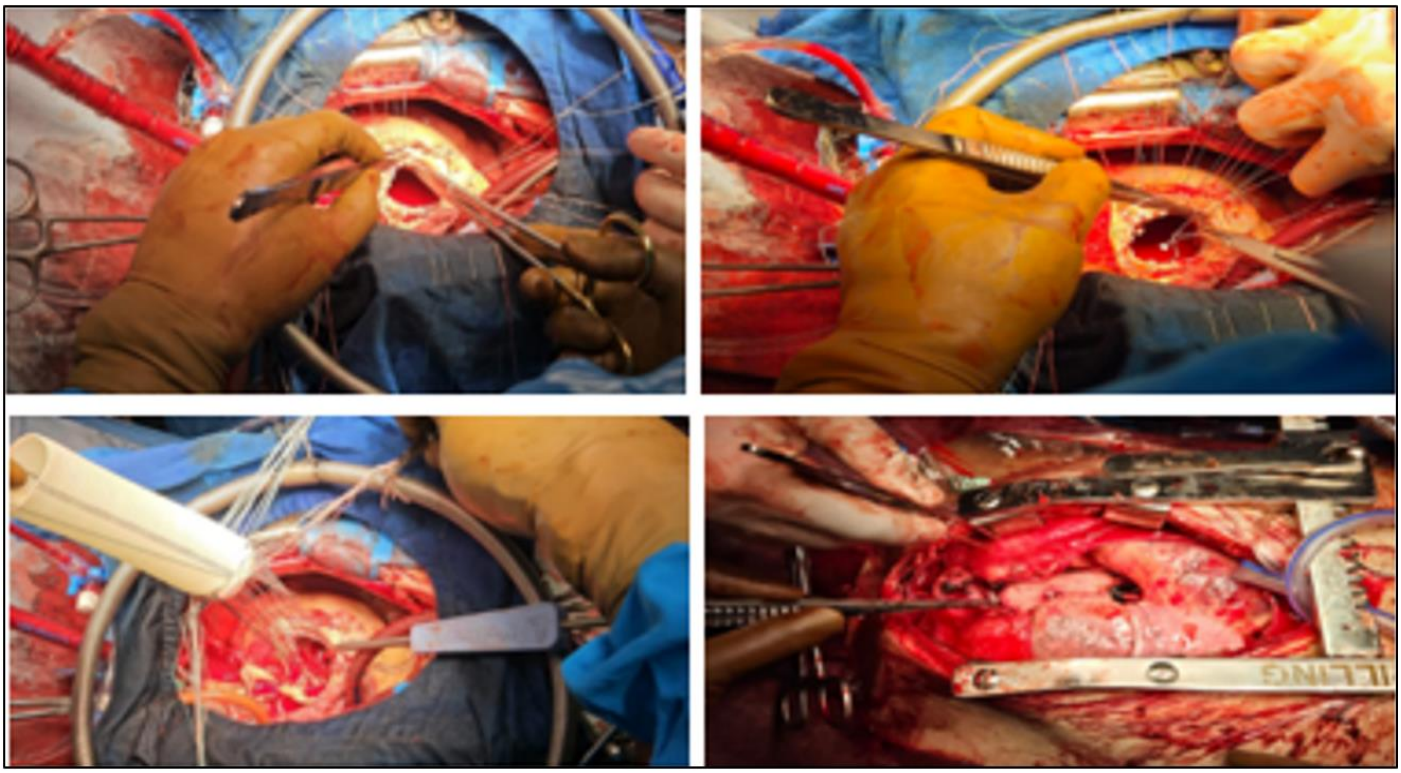

Fig 11 Bentall Procedure

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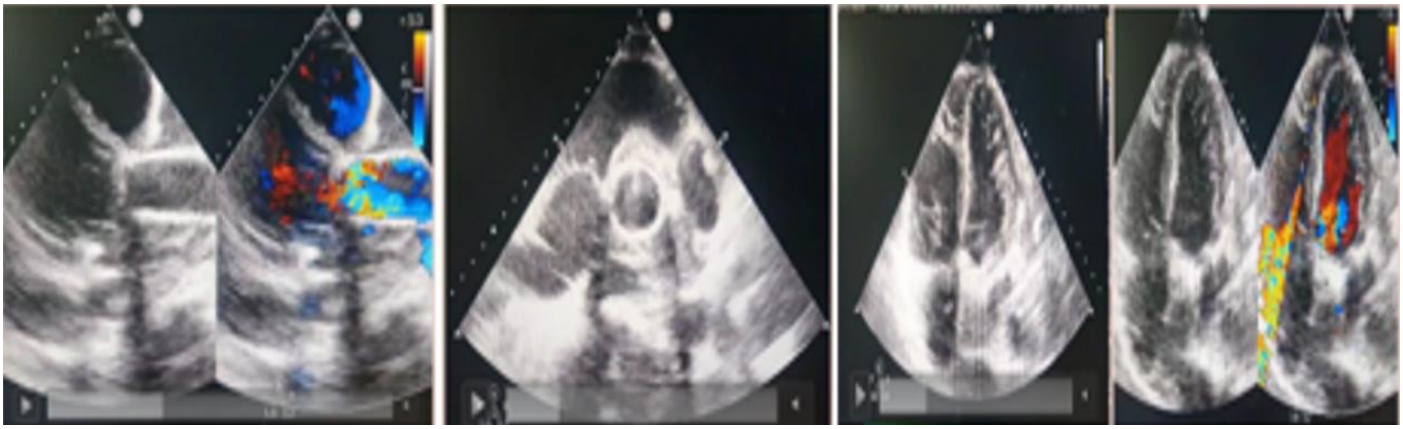

Fig 12 Echocardiography evaluation after Bentall Procedure

The recommended surgical procedure for this patient is the Bentall Procedure, a procedure used to address ascending aortic dissection involving aortic valve disease. Based on echocardiography and thoraco-abdominal MSCTA results, the patient showed dilation of the aortic root (aortic annulus 3.8 cm, sinus valsava 4.2 cm, sinotubular junction 4.7 cm, and ascending aorta 5.9 cm), which is also confirmed by the presence of an intimal flap, indicating a double lumen configuration consistent with Stanford A DeBakey Type II aortic dissection. The Bentall procedure is the primary surgical option for patients with type A aortic dissection, especially if the aortic root dilation exceeds 4.5 cm and is accompanied by intimal tear or connective tissue disease. This procedure involves replacing the ascending aorta and aortic valve using a Composite Valve Graft (CVG), which can be a mechanical or prosthetic valve. Although effective, this procedure requires lifelong anticoagulation and carries risks of complications such as stroke, bleeding, infective endocarditis, and hemolysis (Noori et al., 2022). Given these considerations, surgical therapy remains the primary option for patients, given the high mortality rate if aortic dissection is not promptly treated.

One of the primary limitations of this research is the lack of long-term data on the outcomes of the Bentall procedure, particularly in high-risk patients with connective tissue disorders such as Marfan syndrome. While recent studies show favorable short-term results, the long-term benefits and risks, including complications such as anticoagulation-related issues, stroke, and endocarditis, require further exploration. Additionally, the study relies on retrospective data, which may have inherent biases and limitations in terms of patient selection and outcome assessment. Further prospective studies with larger sample sizes are necessary to provide a more comprehensive understanding of the effectiveness and complications associated with the Bentall procedure in aortic dissection management.

This research has significant implications for the management of aortic dissection, particularly in patients with Marfan syndrome. By exploring the effectiveness of the Bentall procedure and its modifications, such as minimally invasive techniques, the study contributes to refining surgical approaches for high-risk patients. The findings could lead to more tailored treatment protocols, improving surgical outcomes and quality of life for patients with aortic dissection. Furthermore, the research underscores the importance of early detection and personalized treatment strategies, highlighting the need for continuous evaluation of new surgical techniques and pharmacological interventions to enhance patient prognosis and reduce long-term complications.

IV. CONCLUSION

In conclusion, the Bentall procedure is a vital surgical intervention for managing aortic dissection, particularly in patients with Marfan syndrome and concomitant aortic valve disease. Despite the advancements in surgical techniques and therapeutic interventions, the mortality and morbidity associated with aortic dissection remain high, especially in the acute phase. Future research should focus on further improving surgical techniques, exploring alternative pharmacological treatments, and evaluating long-term patient outcomes to provide more effective and personalized treatment strategies. Additionally, there is a need for more detailed studies on the role of genetic testing and early intervention in patients with connective tissue disorders to prevent the progression of aortic complications.

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