Review Article

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Aortic dissection: An Updated Review of Pathogenesis, Diagnosis and Management

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ABSTRACT

BACKGROUND & OBJECTIVE: Aortic dissection is a cardiovascular emergency requiring urgent attention and is associated with severe complications and an increased risk of death. Early diagnosis and timely management are critical for improving patient outcomes. This review aims to summarise the current understanding of the pathogenesis, classification, diagnosis, and management of aortic dissection.

Methodology: Relevant English-language studies available up to 2025 were identified through searches of PubMed, Google Scholar, and ScienceDirect. Keywords included "aortic dissection," "pathophysiology," "classification," "diagnosis," and "management." Peer-reviewed articles, clinical studies, and guideline papers published in English were included. Data were extracted and synthesised qualitatively.

Results: Aortic dissection occurs when a tear in the intimal layer allows blood to form a false lumen. Important risk factors include hypertension and connective tissue disorders. The Stanford classification remains the standard for guiding management choices. Diagnosis is primarily based on imaging, especially computed tomography angiography, with biomarkers such as D-dimer providing supportive information. Stanford Type A dissections require immediate surgical intervention, while Stanford Type B dissections are usually managed medically or with endovascular techniques.

Conclusion: Aortic dissection is a complex condition and demands swift identification and collaborative management. While advancements in imaging modalities, biomarker discovery, and endovascular interventions have enhanced diagnostic precision and patient prognoses, further investigation is essential to refine therapeutic approaches.

KEY WORDS: Aortic Dissection; Stanford Classification; D-Dimer; Diagnosis; Computed Tomography Angiography

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INTRODUCTION

Aortic dissection (AD) occurs when blood enters the aortic media through an intimal defect, leading to formation of a false lumen.[1] This process can impair organ perfusion or lead to aortic rupture and sudden death. AD is classified by its anatomical extent rather than just the location of the initial tear, a distinction that is fundamental to its management.[2] Although relatively rare, with an incidence of around 3 cases in 100,000 people per year, yet it carries a high risk of complications and early death.[3]

Two main systems are the DeBakey classification, based on the site of origin, and the Stanford classification, based on ascending aortic involvement.[4] Stanford Type A dissections represent almost two-thirds of cases and are associated with poorer results.[5] This disease mainly affects the elderly (average age 60–65 years). It is more common in men, with a ratio of approximately 2:1 between men and women.[4-6] Women tend to present later and with atypical features, which contribute to late diagnosis and poor prognosis.[7] Early detection by means of a high index of suspicion and appropriate imaging is essential to improve the outcome. This review summarises current knowledge on the pathogenesis of AD, risk factors, diagnosis and treatment, and the progress made to date.[1-8]

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Pathogenesis:

The aortic wall consists of three layers: the intima, media, and adventitia.[7] AD usually starts with an intimal tear, which allows blood to enter the media and form a false lumen, which can travel along the length of the vessel.[1-3] Medial degeneration is a key factor in this mechanical failure, characterised by elastin breakdown, loss of smooth muscle cells, and disruption of collagen architecture.[7] This progressive weakening of the aortic wall reduces its ability to withstand normal physical stress. Moreover, proteins like fibrillin are essential for maintaining wall integrity, and genetic mutations in pathways like TGF-β signalling can worsen the instability of the extracellular matrix.[9] The progression of the disease is influenced by both mechanical stress and biological processes. Inflammatory activity, particularly macrophage infiltration and the release of proinflammatory cytokines, further compromises the integrity of the aortic media.[8-10] Hypertension intensifies these mechanisms through increased mechanical stress, consequently accelerating disease progression.[3-6-10]

Stanford Type A vs Type B aortic dissection:

Clinical management of AD is mainly guided by the Stanford classification system, which is preferred over the DeBakey method for its usefullness in immediate decision making.[1,11] The basic distinction lies in whether the ascending aorta is affected: Type A involves the ascending segment (proximal to the brachiocephalic artery), while Type B is restricted to the descending aorta (distal to the left subclavian artery's origin). If a tear starts distal to the subclavian artery but extends back into the ascending aorta, it is classified as Type A.[8] This distinction is important for triage, since Type A dissections often require urgent surgical intervention, In contrast, patients with Type B dissections who have no complications are usually treated medically.[11] The DeBakey classification provides a more precise anatomical breakdown and categorizes aortic dissection according to its origin and extent: in Type I, the intimal tear originates in the ascending aorta and propagates distally, Type II remains limited to the ascending segment, whereas Type III involves only the descending portion of the aorta.[1] DeBakey Types I and II fall under Stanford Type A, while Type III falls under Stanford Type B.[11] Type B dissections are further subdivided into complicated and uncomplicated categories. Complicated cases, characterised by malperfusion, persistent pain, uncontrolled hypertension, or rupture, often require urgent endovascular or surgical treatment, whereas uncomplicated cases are managed conservatively.[12]

Following current European guidelines, AD is staged chronologically: the acute phase covers the first 14 days, the subacute phase spans from 15 to 90 days, and the condition is considered chronic once it persists beyond the 90-day mark.[2,10]

Risk factors and predispositions:

While hypertension remains the most prevalent risk factor for AD, the condition is also frequently observed in aging populations and males. Additional contributors to aortic fragility include various connective tissue diseases, traumatic events, and the use of sympathomimetic stimulants.[1,4,5] The most critical modifiable factor is hypertension, which increases mechanical stress and accelerates medial degeneration.[1,7] Heritable connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome, are strongly correlated with AD.[1,9,13]

Marfan syndrome (FBN1 mutation) results in cystic medial degeneration, whereas Loeys-Dietz syndrome (TGFBR mutations) is characterised by an aggressive disease course even at smaller aortic diameters.[9] Vascular Ehlers-Danlos syndrome (COL3A1 mutation) leads to profound vascular instability and increased rupture risk.[13] Congenital conditions, including bicuspid aortic valve disease and Turner syndrome, further increase susceptibility.[13] Inflammatory disorders, such as giant cell arteritis, contribute to wall failure through immune-mediated damage.[7] Finally, a family history of thoracic aneurysm represents a non-syndromic genetic predisposition affecting smooth muscle function.[14]

Clinical presentation:

The presentation of AD is notoriously varied, necessitating a high index of clinical suspicion (Table I). Most patients experience abrupt, severe thoracic or dorsal pain, although presentations vary widely across individuals.[7,11] Clinicians must differentiate AD from aortic mimics, such as acute myocardial infarction, pulmonary embolism, and tension pneumothorax, as the misadministration of thrombolytic therapy for a misdiagnosed coronary event can be fatal.[7,20] Physical findings, such as pulse deficits or blood pressure differentials between the arms, support suspicion but are not consistently present.[4,11] While chest X-rays may reveal indirect findings like a widened mediastinum, they lack sufficient diagnostic precision to rule out the condition.[11] Initial management prioritises rapid blood pressure and heart rate control while awaiting definitive confirmation via imaging.[11,12,15–17]

AD biomarkers:

Biomarkers are an adjunct for early risk stratification, though imaging remains the gold standard.[9,15] The most utilised is D-dimer; it is highly effective in ruling out AD in low-risk patients but has limited specificity.[15,18] Cardiac troponins, interleukin-6, and C-reactive protein may be elevated but lack specificity.[2,15] Emerging biomarkers, such as matrix metalloproteinases and smooth muscle myosin heavy chain, show potential but are not yet established in routine practice.[2,9,19]

Diagnosis guidelines:

The AHA/ACC and ESC provide structured protocols for diagnosis, including the Aortic Dissection Detection Risk Score (ADD-RS) (Figure 1).[20] This tool stratifies patients based on high-risk predispositions and pain characteristics; a score >1 indicates high risk requiring immediate definitive imaging, whereas in low-risk patients (ADD-RS ≤1), a negative D-dimer may be used for exclusion.[2,18,20]

Computed tomography angiography (CTA) is considered the first-line imaging modality in emergency settings due to its rapid acquisition and 98–100% accuracy.[2,21] ECG-gated CTA further improves precision by reducing motion artefacts in the ascending aorta.[21] For unstable patients, transoesophageal echocardiography (TEE) serves as a rapid bedside tool to visualise entry tears and pericardial effusion.[15,22] Magnetic resonance imaging (MRI) offers excellent anatomical and functional assessment but is typically reserved for stable patients due to logistical challenges in acute monitoring.[2,20] Transthoracic echocardiography (TTE), though less sensitive for the descending aorta, remains a valuable screening tool for proximal involvement and valve assessment.[20]

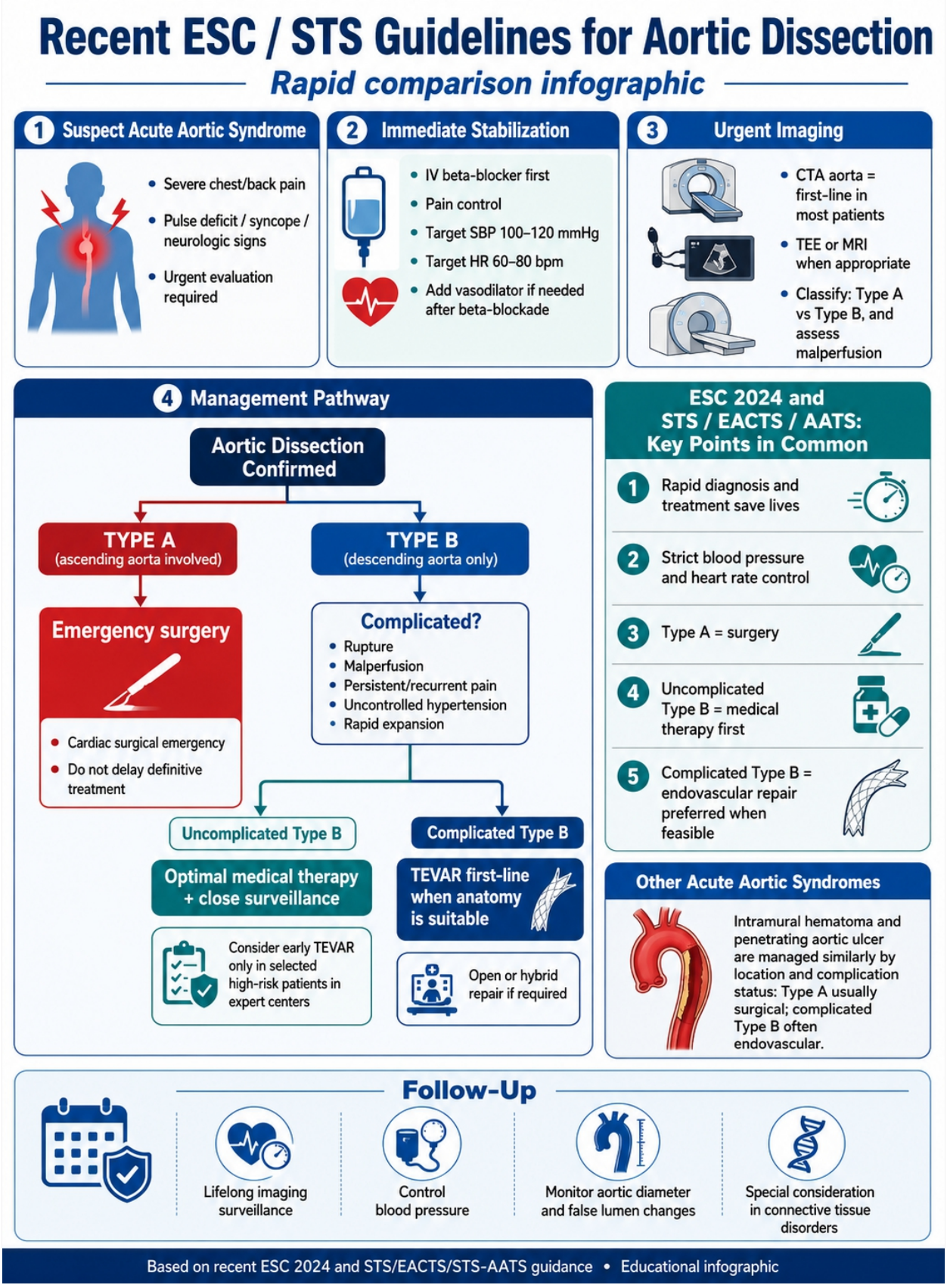

Figure 1: The STS/AATS clinical practice guidelines on the management of type B aortic dissection.[27]

Table I: Clinical features and diagnostic significance in aortic dissection.

Clinical Feature	Clinical Significance	Description
Chest pain	Most common symptom	Sudden, severe, tearing chest pain
Back pain	Common in Stanford Type B	Pain radiating to interscapular region
Pulse deficit	Suggests branch vessel involvement	Reduced/absent pulses
Blood pressure difference	Suggests vascular compromise	Inter-arm BP discrepancy
Syncope	Indicates severe disease	May suggest tamponade or rupture
Neurological deficits	Indicates malperfusion	Stroke or spinal cord ischemia
Hypotension/shock	Poor prognostic sign	Suggests rupture or tamponade
Imaging findings	Confirms diagnosis and guides intervention	Presence of a visible intimal flap separating the true and false lumens; may show entry/re-entry tears or branch vessel involvement.

Treatment guidelines:

The management of acute AD requires immediate haemodynamic stabilisation followed by definitive surgical or endovascular intervention. Initial therapy is directed at the rapid reduction of systolic blood pressure (target 100–120 mmHg) and heart rate control to minimise shear stress on the aortic wall. Intravenous labetalol or esmolol are the preferred first-line agents due to their anti-impulse effects, reducing the dP/dt (the rate of rise in ventricular pressure), which is the mechanical force driving the dissection, to prevent further propagation of the intimal tear.[23,10]

Acute Stanford Type A aortic dissection is a surgical emergency necessitating the immediate replacement of the ascending aorta.[24–25] In modern practice, advanced approaches such as the Frozen Elephant Trunk (FET) technique have gained prominence for complex cases involving the aortic arch, allowing for a single-stage repair of the arch and proximal descending aorta.[10,26] In specialised centres, survival rates for Stanford Type A repair have reached 85–90%.[25] In contrast, uncomplicated Stanford Type B AD is primarily managed with intensive medical therapy.[9,10] Complicated Stanford Type B cases require thoracic endovascular aortic repair (TEVAR).[10,26] Recent guidelines emphasise the role of multidisciplinary aortic care teams and an increasing reliance on endovascular techniques.[10] Lifelong imaging surveillance is mandatory for all survivors due to the persistent risk of late complications.[10,26]

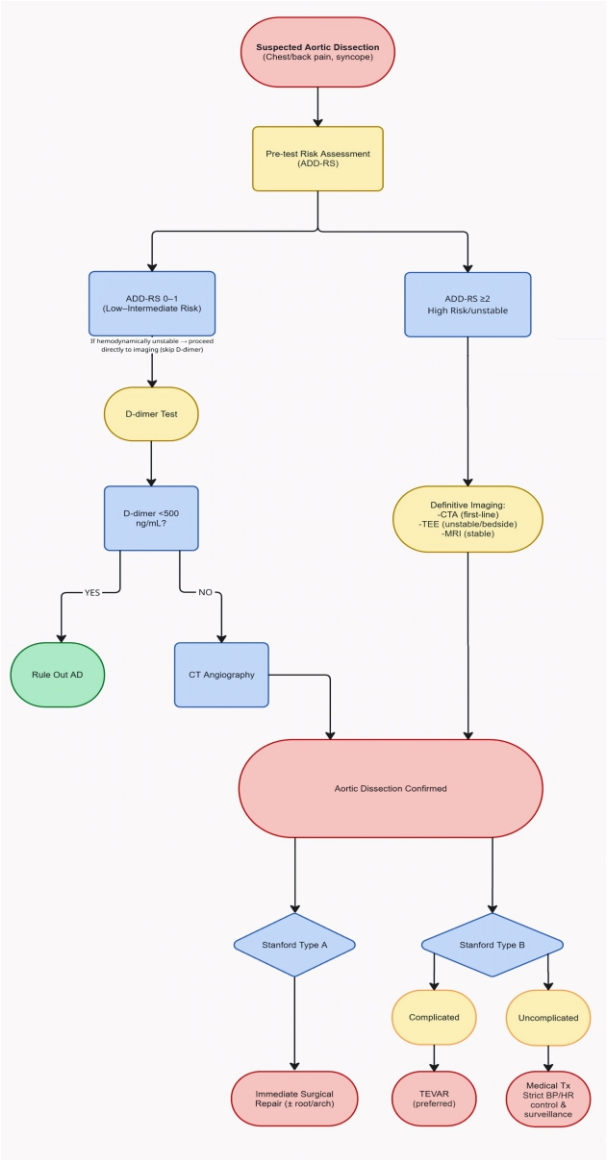

Figure 2: Diagnostic and management algorithm for suspected aortic dissection. Patients presenting with symptoms suggestive of aortic dissection undergo pre-test risk stratification using the Aortic Dissection

Detection Risk Score (ADD-RS). Low-to-intermediate-risk patients (ADD-RS 0–1) undergo D-dimer testing, while high-risk or unstable patients (ADD-RS ≥2) proceed directly to definitive imaging. Confirmed dissections are subsequently classified according to the Stanford system, guiding management toward urgent surgical repair for Type A dissections and either thoracic endovascular aortic repair (TEVAR) or optimal medical therapy for Type B dissections depending on the presence of complications.

CONCLUSION

AD remains a life-threatening cardiovascular emergency requiring rapid diagnosis and coordinated multidisciplinary management. Advances in imaging techniques, particularly computed tomography angiography, alongside the integration of clinical risk scores and biomarkers, have significantly improved the early detection and management of the disease. Current guidelines emphasise early risk stratification, aggressive haemodynamic control, and timely intervention based on the anatomical type and clinical complexity.

While open surgical repair remains the cornerstone for Stanford Type A dissections, there is a clear shift toward less invasive endovascular approaches for Stanford Type B cases. Patient outcomes have been further enhanced by the increasing role of specialised aortic centres and multidisciplinary aortic care teams. Despite these advances, challenges remain in optimising early diagnosis, improving risk prediction, and establishing the routine clinical utility of biomarkers for personalised treatment. Future research should focus on genetic profiling, targeted molecular therapies, and long-term longitudinal data to further refine treatment strategies. Continued emphasis on early diagnosis, stringent risk factor control, and adherence to evidence-based guidelines will be crucial in reducing the high morbidity and mortality associated with this disease.

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Conflict of Interest:

Authors declare no conflict of interest.

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REFERENCES

1. Nienaber CA, Clough RE, Sakalihasan N, Suzuki T, Braverman AC, Zheng J, et al. Aortic dissection. Nat Rev Dis Primers. 2016;2:16053. doi: 10.1038/nrdp.2016.53.
2. Sayed A, Munir M, Bahbah EI. Aortic dissection: a review of the pathophysiology, management and prospective advances. Curr Cardiol Rev. 2021; 17(1):e230421186875. doi: 10.2174/1573403X16666201014142930.
3. Clouse WD, Hallett JW, Schaff HV, Spittell PC, Rowland CM, Ilstrup DM, et al. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clin Proc. 2004; 79(2):176-80. doi: 10.4065/79.2.176.
4. Evangelista A, Isselbacher EM, Bossone E, Gleason TG, Eusanio MD, Sechtem U, et al. Insights from the international registry of acute aortic dissection: a 20-year experience. Circulation. 2018; 137(17):1846-60. doi: 10.1161/-Circulationaha.117.031264.
5. Howard DPJ, Sideso E, Handa A, Rothwell PM. Incidence, risk factors, outcome and projected future burden of acute aortic dissection. Ann Cardiothorac Surg. 2014;3(3):278-84. doi: 10.3978/j.issn.2225-319X.2014.05.14.
6. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J. 2014; 35(41):2873-926. doi: 10.1093/eurheartj/ehu281.
7. Gawinecka J, Schnürath F, von Eckardstein A. Acute aortic dissection: pathogenesis, risk factors and diagnosis. Swiss Med Wkly. 2017 147:w14489. doi: 10.4414/smw.2017.14489.
8. Bossone E, LaBounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management. Eur Heart J. 2018; 39(9):739-49. doi: 10.1093/eurheartj/ehx696.
9. Alfson DB, Ham SW. Type B aortic dissections. Cardiol Clin. 2017; 35(3):387-410. doi: 10.1016/j.ccl.2017.03.007.
10. Bedi VS, Swain P, Yadav A. Medical therapy versus TEVAR for uncomplicated type B aortic dissection. Indian J Thorac Cardiovasc Surg. 2019; 35(Suppl 2):174-8. doi: 10.1007/s12055-019-00813-w.
11. Harky A, Hussain SMA, MacCarthy-Ofosu B, Ahmad MU. Role of thoracic endovascular aortic repair in connective tissue disorders. Braz J Cardiovasc Surg. 2020; 35(4). doi: 10.21470/1678-9741-2019-0158.
12. Albornoz G, Coady MA, Roberts M, Davies RR, Tranquilli M, Rizzo JA, et al. Familial thoracic aortic aneurysms and dissections. Ann Thorac Surg. 2006; 82(4):1400-5. doi: 10.1016/j.athoracsur.2006.05.023.
13. Isselbacher EM. Thoracic and abdominal aortic aneurysms. Circulation. 2005; 111(6):816-28. doi: 10.1161/-01.CIR.0000154569.08857.7A.
14. Loeys BL, Schwarze U, Holm T, Callewaert BL, Thomas GH, Pannu H, et al. Aneurysm syndromes caused by mutations in the TGF-β receptor. N Engl J Med. 2006; 355(8):788-98. doi: 10.1056/NEJMoa055695.
15. Bossone E, Czerny M, Lerakis S, Russo CV, Evangelista A, Eagle KA, et al. Imaging and biomarkers in acute aortic syndromes. Curr Probl Cardiol. 2021;46(3):100654. doi: 10.1016/j.cpcardiol.2020.100654.
16. Spanos K, Kölbel T. Role of endoluminal techniques in chronic type B aortic dissection. Cardiovasc Intervent Radiol. 2020;43(12):1808-20. doi: 10.1007/s00270-020-02570-x.
17. Fukui T. Management of acute aortic dissection and thoracic aortic rupture. J Intensive Care. 2018;6:15. doi: 10.1186/s40560-018-0287-2.
18. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, et al. 2010 ACCF/AHA guidelines for thoracic aortic disease. Circulation. 2010; 121(13):e266-369. doi: 10.1161/CIR.0b013e3181d4739e.

19. Nazerian P, Mueller C, Soeiro AM, Leidel BA, Salvadeo SAT, Giachino F, et al. Diagnostic accuracy of ADD-RS plus D-dimer. Circulation. 2018; 137(3):250-8. doi: 10.1161/Circulationaha.117.029457.
20. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The international registry of acute aortic dissection (IRAD). JAMA. 2000; 283(7):897-903. doi: 10.1001/jama.283.7.897.
21. Nienaber CA, von Kodolitsch Y, Nicolas V, Siglow V, Piepho A, Brockhoff C, et al. Diagnosis of thoracic aortic dissection by noninvasive imaging. N Engl J Med. 1993; 28(1):1-9. doi: 10.1056/NEJM199301073280101.
22. Budeanu RG, Broemmer C, Budeanu AR, Pop M. ECG-gated vs non-gated CT angiography in ascending aortic dissection. Tomography. 2022;8(5):2426-34. doi: 10.3390/-tomography8050202.
23. Ikram S, Jabeen K, Inam S, Inam A, Hassan S, Mushtaq S, Qureshi MA. The continuing menace of extended spectrum beta lactamase (ESBLs): a centre based study. Pak Postgrad Med J. 2023;34(3):154-157. doi:10.51642/ppmj.v34i03.578.
24. Wang J, Li Y, Li Y, Mao T, He X, Yu F, et al. Endovascular stent-graft placement in Stanford type B aortic dissection. Ann Vasc Surg. 2016; 36:298-309. doi: 10.1016/j.avsg.2016.03.016.
25. Isselbacher EM, Preventza O, Black JH 3rd, Augoustides JG, Beck AW, Bolen MA, et al. 2022 ACC/AHA guideline for the diagnosis and management of aortic disease. Circulation. 2022; 146(15):e334-482. doi: 10.1161/CIR.0000000000001106.
26. Franzen D, Kaleschke G, Tzikas S, et al. 2024 ESC guidelines for the management of peripheral arterial and aortic diseases. Eur Heart J. 2024. doi: 10.1093/eurheartj/ehae458.
27. MacGillivray TE, Gleason TG, Patel HJ, Aldea GS, Bavaria JE, Beaver TM, et al. The STS/AATS clinical practice guidelines on the management of type B aortic dissection. Ann Thorac Surg. 2022;113(4):1073-1092. doi:10.1016/-j.athoracsur.2021.11.002.

Authors' Contributions:

ZN & SK: Conceptualization & study design.

ZN & SK: Data Collection and manuscript drafting, data Analysis and critical review.

ZN & SK: Manuscript drafting & proof reading.

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