Case Report

Chronic Aortic Dissection Complicated by Necrotizing Pneumonia and Heart Failure: A Multisystem Challenge

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Abstract

Chronic aortic dissection is an infrequent but clinically significant condition with heterogeneous presentations. Its management becomes markedly more complex when complicated by superimposed infections such as necrotizing pneumonia and ensuing multi-organ dysfunction. We report a diagnostically challenging case that exemplifies the critical role of multidisciplinary collaboration in navigating such multifaceted clinical scenarios. A 44-year-old morbidly obese male with multiple comorbidities, including obstructive sleep apnea, asthma, hypertension, and dyslipidemia presented with chronic cough, dyspnea, orthopnea, and hemoptysis. Initially managed as community-acquired pneumonia, his condition progressively deteriorated despite empirical antibiotic therapy. Further investigations revealed bilateral pulmonary congestion, leukocytosis, and acute kidney injury. Blood cultures were positive for Staphylococcus lugdunensis. Cross-sectional imaging via CT pulmonary angiography and CT aortic angiography confirmed a Stanford Type A chronic aortic dissection, cardiomegaly, moderate aortic regurgitation, and necrotizing pneumonia with pleural effusion. Transthoracic echocardiography demonstrated global hypokinesia with a reduced ejection fraction of 42%. Management included intravenous antibiotics, diuretics, beta-blockers, and ultrasound-guided pigtail catheter drainage. Worsening renal function necessitated initiation of sustained low-efficiency dialysis (SLEDD), which yielded biochemical improvement. A coordinated multidisciplinary approach which encompassing cardiology, cardiothoracic surgery, nephrology, pulmonology, and infectious disease specialists, was essential to optimizing clinical outcomes. The patient was stabilized and discharged on home oxygen therapy with structured outpatient follow-up. This case highlights the diagnostic and therapeutic complexity of chronic aortic dissection in the context of concurrent severe infection and organ failure. Timely recognition, individualized management, and cohesive multidisciplinary care are paramount in mitigating morbidity and improving prognostic trajectories in such high-risk patients.

Keywords: *aortic dissection, necrotizing pneumonia, heart failure, multidisciplinary communication, renal dialysis*

INTRODUCTION

Chronic aortic dissection is a rare but serious condition that can lead to significant complications if not properly managed (Nusbacher et al. 1976). While the classic presentation involves severe chest or back pain, the clinical features of aortic dissection can be quite variable (Finkelmeier 1997). Aortic dissection is a life-threatening condition with high morbidity and mortality, particularly when it involves the ascending aorta (Stanford type A). Dissection of the ascending thoracic aorta is a surgical emergency, with mortality exceeding 1% per hour during the first 48 hours if left untreated. In contrast, dissections involving only the descending aorta (Type B) are generally managed with medical therapy unless there are signs of ongoing dissection, intractable hypertension, or end-organ ischemia (Finkelmeier 1997).

This case underscores the clinical complexity of managing chronic type A aortic dissection complicated by necrotising pneumonia and acute decompensated heart failure. The coexistence of these high-risk pathologies necessitated timely recognition, advanced diagnostics, and a coordinated multidisciplinary strategy. This highlights the importance of individualised, evidence-based care in addressing overlapping cardiovascular and infectious processes to optimise patient outcomes in complex clinical scenarios.

CASE REPORT

A 44-year-old nonsmoking male presented with a chronic cough that started in September 2024. Initially, the cough produced whitish sputum but progressed to haemoptysis over the past two weeks. He had multiple clinic visits during this period and was

treated for acute exacerbation of bronchial asthma (AEBA). In November 2024, he presented to the emergency department and was diagnosed with community-acquired pneumonia (CAP). He was prescribed a five-day course of Augmentin and a three-day course of azithromycin, after which he was discharged.

Over the subsequent two weeks, he developed worsening dyspnoea, reduced effort tolerance, orthopnoea, and symptoms consistent with the NYHA Class II functional status. He also reported pleuritic chest pain, which had persisted for the past two months. Upon presentation to the emergency department, the patient was alert. His vital signs were as follows: blood pressure of 150/68 mmHg, heart rate of 101 bpm, oxygen saturation of 98% on the nasal cannula at 3 L/min, and a documented fever of 38.7°C. Point-of-care ultrasound (POCUS) of the lungs revealed bilateral basal B-lines. Bedside echocardiography revealed moderate left ventricular contractility with an estimated ejection fraction (EF) of 40% (by eyeballing). The aortic root and descending aorta were normal in size, with no regional wall motion abnormalities observed.

His past medical history included morbid obesity with a BMI of 32.3 kg (weight 88 kg, height 165 cm) and severe obstructive sleep apnea (OSA) with a baseline AHI of 58 and lowest oxygen saturation of 87%. He is on CPAP therapy with a nasal mask, although compliance is suboptimal, with a recorded CPAP usage of >4 hours in only 21% of nights. He has had bronchial asthma since childhood and is managed under respiratory care with Symbicort 2 puffs BD as part of SMART therapy. He had a history of intensive care unit (ICU) admission without intubation and was last admitted for asthma exacerbation in February 2023. Additionally, he has hypertension and dyslipidemia, for which he is on perindopril 8 mg OD, indapamide 1.5 mg OD, amlodipine 5 mg OD, allopurinol 300 mg OD, and symbicort as needed. His family history was significant for ischaemic heart disease, with his father having three-vessel disease. There was no record of prior cardiac imaging, such as an echocardiogram, before this acute admission despite the presence of comorbidities.

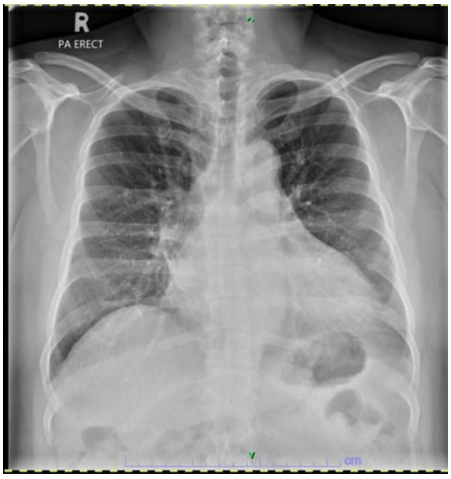

Figure 1: Initial CXR upon presentation to a casualty (November 2024)

Initial investigations revealed cardiomegaly with bilateral pulmonary congestion and basal infiltrates on chest radiography. The electrocardiogram revealed sinus rhythm with poor R-wave progression. Elevated troponin I (Troponin I) (41 ng/L) has raised concerns about myocardial strain. Laboratory parameters revealed leukocytosis (WBC 13 ×10^9/L), elevated C-reactive protein (CRP 4.2 mg/L), and acute kidney injury (creatinine 144.8 µmol/L, urea 6.9 mmol/L). Arterial blood gas analysis of room air revealed primary respiratory alkalosis (pH 7.45, pCO_2 27 mmHg, pO_2 103 mmHg, and HCO_3- 21 mmol/L), indicative of underlying respiratory compensation, likely due to increased breathing and underlying pulmonary pathology.

Following a cardiology assessment, the patient was admitted for comprehensive inpatient care with initial diagnoses of decompensated congestive cardiac failure (CCF), partially treated pneumonia, hepatic congestion, and acute kidney injury (AKI). Throughout hospitalisation, he remained intermittently febrile (maximum temperature 37.5°C), prompting the escalation of antimicrobial therapy due to concern for hospital-acquired infection (HAI). Initial empiric therapy with intravenous piperacillin-tazobactam was administered for three days, followed by a nine-day course of intravenous ceftriaxone. On Day 3, antimicrobial coverage was further increased to intravenous ceftazidime. Despite these measures, the inflammatory indices continued to trend upwards (WBC increased from 11.9 to 17.4 ×10^9/L; CRP increased to 7.48 mg/L).

Blood cultures taken on Day 4 revealed that *Staphylococcus lugdunensis,* a virulent coagulase-negative Staphylococcus, was increasingly recognised for its clinical significance. The subsequent cultures remained sterile. Sputum analysis was negative for acid-fast bacilli and significant bacterial growth,

whereas tuberculosis cultures were pending. Transthoracic echocardiography demonstrated a left ventricular ejection fraction (LVEF) of 42%, with global hypokinesia and moderate-to-severe aortic regurgitation (AR). Although the NT-proBNP levels were not available, the clinical signs were consistent with volume overload and systolic dysfunction. The patient continued to experience orthopnea, exertional dyspnea, and persistent tachycardia (>130 bpm).

Acute kidney injury is considered multifactorial and is likely secondary to sepsis, hypoperfusion from decompensated heart failure, and underlying cardiorenal syndrome. Liver function tests revealed elevated transaminases and increased periportal echogenicity on abdominal ultrasonography, which is consistent with hepatic congestion. Viral hepatitis screening returned negative. On day 6 after admission, the patient developed mild haemoptysis (~5 mL), which prompted CT pulmonary angiography (CTPA). The scan excluded pulmonary embolism but revealed extensive pulmonary infection changes, cardiomegaly, dilation of the main pulmonary artery, and an enlarged ascending aorta measuring 4.6 cm.

Given the progression of respiratory distress, unilateral reduction in breath sounds, and new left-sided infiltrates on chest radiograph, a CT aortic angiography was subsequently performed. This confirmed the presence of a chronic Stanford type A aortic dissection.

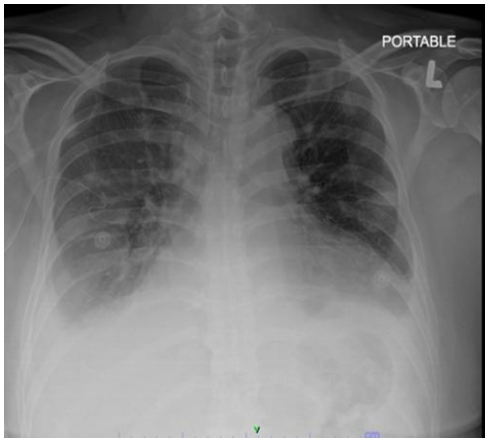

Figure 2: Repeated CXR upon deterioration in the ward.

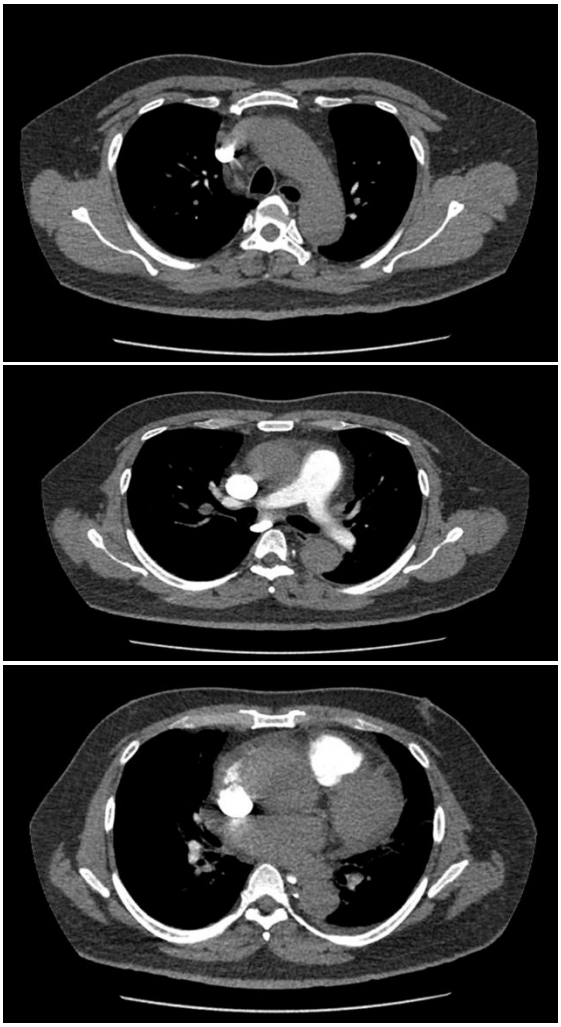

Figure 3: CTPA was performed, excluding pulmonary embolism, but revealed significant pulmonary infection changes, cardiomegaly, dilatation of the main pulmonary artery, and a dilated ascending aorta (measuring 4.6 cm).

Serial computed tomography angiograms (CTAs) of the aorta were performed for surveillance and evaluation of disease progression. All the scans consistently revealed a Stanford type A (DeBakey type I) aortic dissection originating from the aortic root. The dissection extended superiorly into the brachiocephalic trunk, right common carotid artery, and proximal bilateral subclavian arteries and inferiorly into the descending thoracic aorta, terminating at the T9–T10 vertebral level. Stable fusiform dilatation of the ascending aorta was observed, ranging from 4.8 to 5.5 cm in anteroposterior diameter. There were no signs of active leakage, contrast extravasation, fat stranding, or retroperitoneal hematoma. A stable, short segment intramural thrombus was noted at the T11 level.

CTA imaging further revealed patent visceral branches, including the celiac trunk, superior mesenteric artery (SMA), renal arteries, and common iliac arteries. The patient had mild cardiomegaly with

a progressively decreasing right-sided pericardial effusion (from 1.7 cm to minimal residual thickness). A right thyroid nodule (2.0 × 1.9 cm) was noted and remained unchanged throughout the disease course.

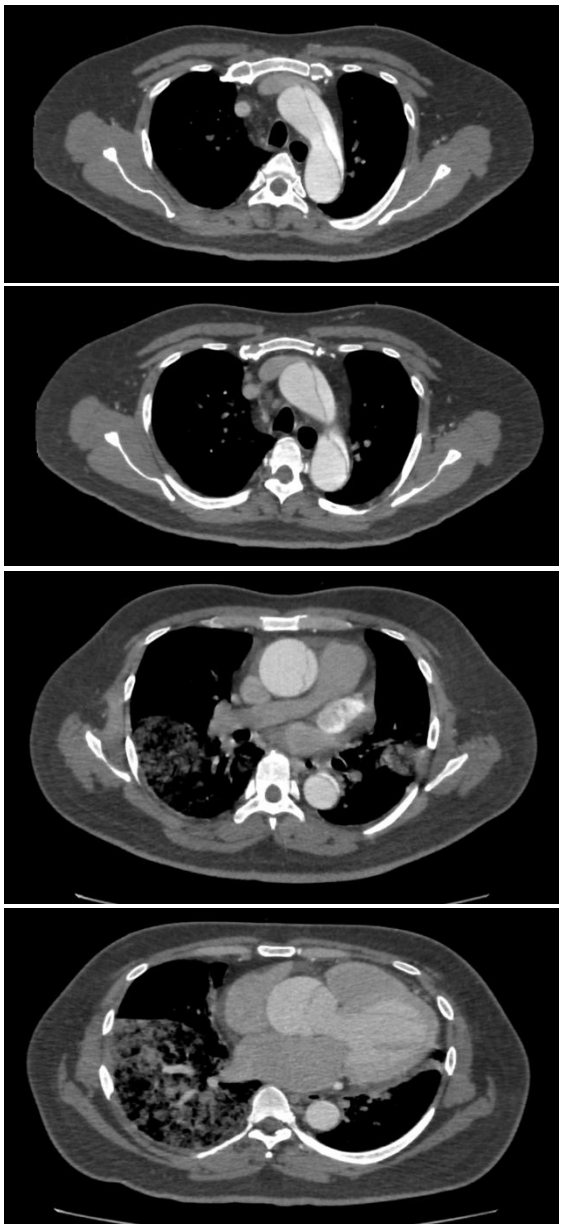

Figure 4 CTA dated 31/12/2024 showing the appearance of fusiform dilatation of the ascending aorta measuring 4.8 cm in maximal AP diameter. Unchanged aortic dissection with an intimal flap noted arising from the aortic root. Superiorly, it extends to the brachiocephalic trunk, the origin of the right common carotid artery, and the proximal right and left subclavian arteries. The true lumen gives rise to the brachiocephalic trunk and the right common carotid and right subclavian arteries. The intimal flaps also continue inferiorly involving the descending thoracic aorta up to the T9/T10 level, which is essentially unchanged. A small, short segment intramural thrombus noted at the T11 level is also similar in appearance. No periaortic fat streakiness or active contrast blushing suggested active leakage. There were no signs of retroperitoneal haematoma or draped aorta. The celiac trunk, SMA, both renal arteries and both common iliac arteries are opacified.

Interval progression was noted across imaging studies. On 13 December 2024, aortic root dissection was first detected, with the ascending aorta dilated to 5.5 cm. Associated findings included bilateral lung collapse/consolidation, which was most pronounced in the right lower lobe and suggestive of infection, and a pericardial effusion measuring up to 1.7 cm. By 20 December, ECG-gated imaging suggested a smaller ascending aortic diameter (4.8 cm), possibly due to improved resolution. However, pulmonary consolidations had worsened, with low attenuation areas in the right lower lobe and lingular segment of the left upper lobe, raising suspicion for necrotising pneumonia. Minimal perihepatic, perisplenic, and paracolic free fluid was also noted. By 31 December, imaging demonstrated stable dissection and a stable aortic size, but the right pleural effusion had worsened, causing near-complete collapse of the right middle and lower lobes. Low-attenuation consolidation persisted in the right lower lobe, which was consistent with ongoing necrotising pneumonia.

The patient was referred to the cardiothoracic surgery (CTC) team for evaluation of potential surgical intervention. Given the chronic nature of the dissection, the absence of active leakage, and the patient's current comorbid burden, conservative management with close radiologic surveillance was advised. Moreover, decompensated heart failure was managed with bisoprolol, intravenous diuretics, and careful fluid balance. The introduction of an SGLT2 inhibitor was deferred until infection resolution.

The case and CTA reports were updated to the CTC Team. The patient's condition and clinical progress were noted. The patient is currently stable despite an ongoing infection. Given this stability, the plan is to allow progression to a subacute Stanford type A presentation. The CTC team will be updated again once the infection has resolved.

The immediate plan is to focus on treating ongoing infection while closely monitoring trends in total white cell count (TWC) and C-reactive protein (CRP) levels. Pending blood culture and sensitivity results were traced. Blood pressure control is a priority, with a target systolic blood pressure (SBP) of 100–110 mmHg and a heart rate of 60–80 beats per minute. Oral antihypertensive medications will be optimised accordingly. If the patient's blood pressure remains persistently elevated, intravenous labetalol will be initiated. The CTC team will be reinformed once the infection has resolved.

Necrotising pneumonia required an extended 12-week course of intravenous antibiotics. Serial blood cultures confirmed the clearance of bacteremia. A pigtail catheter was inserted to drain the worsening right pleural effusion; analysis confirmed exudative fluid without malignant cells. The patient's AKI necessitated two sessions of sustained low-efficiency dialysis (SLEDD), which reduced creatinine from 390 µmol/L to 77 µmol/L. Following clinical stabilisation, the patient was discharged with home oxygen therapy and scheduled for multidisciplinary follow-up in cardiology, respiratory medicine, nephrology, and infectious disease.

This case underscores the complexity of managing chronic aortic dissection in a patient with overlapping cardiopulmonary pathology and multiorgan dysfunction. This highlights the importance of early cross-sectional imaging in unexplained cardiopulmonary decline, the potential for infectious complications in chronic dissection, and the necessity of coordinated multidisciplinary care in optimising outcomes for high-risk patients.

DISCUSSION

This case highlights the intricate challenges in managing patients with multiorgan dysfunction, including decompensated congestive cardiac failure (CCF), necrotising pneumonia, hospital-acquired infection (HAI), and Stanford type A chronic aortic dissection. The convergence of systemic inflammation, infectious burden, and cardiovascular compromise necessitated a sophisticated, multidisciplinary approach, underscoring the importance of precise clinical judgment and collaboration across specialities.

The patient developed necrotising pneumonia, a rapidly progressive and destructive pulmonary infection characterised by tissue necrosis, cavitation, and systemic inflammation, resulting in increased morbidity and mortality (Grabczak et al. 2016). Despite initial broad-spectrum antibiotic therapy, persistent fever and escalating inflammatory markers have raised concerns of an unresolved infectious process. Subsequent blood cultures revealed Staphylococcus lugdunensis, a coagulase-negative staphylococcus with increasing recognition for its pathogenic potential, particularly in severe infections such as prosthetic valve endocarditis (Becker et al. 2014). Parapneumonic effusion further complicates the respiratory status, necessitating pigtail catheter drainage, which yields exudative, nonmalignant fluid.

In light of persistent leukocytosis and ongoing febrile episodes, antibiotic therapy was escalated according to sepsis management protocols (Evans et al. 2021), resulting in an extended 12-week intravenous antibiotic course due to the protracted nature of pneumonia and its associated complications. Serial blood cultures confirmed the clearance of S. lugdunensis, indicating progress in the resolution of the bacteremia.

The patient's decompensated heart failure, precipitated by the severe inflammatory burden of pneumonia, manifested as orthopnoea, reduced exercise tolerance, and echocardiographic evidence of global hypokinesia with an ejection fraction of 42%. In the context of moderate-to-severe aortic regurgitation, pharmacologic management focuses on optimising fluid balance and minimising myocardial stress. Diuretics were administered to alleviate pulmonary congestion, whereas beta-blockade with bisoprolol was introduced to attenuate myocardial oxygen demand, limit further aortic dilation, and reduce the risk of disease progression (Mcdonagh et al. 2021). Initiation of SGLT2 inhibitors was deferred until resolution of the infectious process, in alignment with current guidelines (Mcdonagh et al. 2021).

The incidental identification of a Stanford type A chronic aortic dissection, with an ascending aortic diameter of 4.6 cm, added complexity to management. Given the absence of rupture, malperfusion, or hemodynamic instability, conservative management was pursued with serial imaging and blood pressure control, as per established guidelines (Erbel et al. 2014; Isselbacher et al. 2022). The development of acute kidney injury (AKI), attributed to both septic shock and cardiorenal syndrome (CRS), necessitates the initiation of sustained low-efficiency dialysis (SLEDD), resulting in improved renal function and volume status (Bellomo et al. 2012). Furthermore, the management of concurrent gout was initially achieved with colchicine, followed by long-term allopurinol therapy.

This case highlights the necessity of a tailored, multidisciplinary treatment approach that incorporates expertise from cardiology, cardiothoracic surgery, nephrology, pulmonology, and infectious diseases. Ongoing monitoring, heart failure optimisation, and the completion of antibiotics emphasise the importance of individualised, dynamic care in complex, high-risk patients.

CONCLUSION

This case highlights the intricate relationships among infection, cardiovascular disease, and renal dysfunction, emphasising the need for early recognition, multidisciplinary care, and long-term follow-up. The patient's complex hospital course was

marked by necrotising pneumonia, decompensated heart failure, and chronic aortic dissection requiring a tailored and dynamic approach to treatment. Managing heart failure in the context of aortic dissection poses unique challenges, as aggressive volume reduction risked compromised perfusion.

Ultimately, this case reinforces the importance of individualised treatment plans, timely intervention, and ongoing surveillance in managing high-risk patients with multiple comorbidities. Future efforts should focus on refining management strategies to improve clinical outcomes in similar complex cases.

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REFERENCES

1. Becker, K., Heilmann, C. & Peters, G. 2014. Coagulase-negative staphylococci. Clin Microbiol Rev 27(4): 870-926.
2. Bellomo, R., Kellum, J. A. & Ronco, C. 2012. Acute kidney injury. Lancet 380(9843): 756-766.doi: 10.1016/j.ekir.2020.08.007. PMID: 33163730; PMCID: PMC7609891.
3. Erbel, R., Aboyans, V., Boileau, C., Bossone, E., Bartolomeo, R. D., et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). European Heart Journal 35(41): 2873-2926.
4. Echefu G, Stowe I, Lukan A, Sharma G, Basu-Ray I, et al. Central vein stenosis in hemodialysis vascular access: clinical manifestations and contemporary management strategies. Front Nephrol. 2023;3:1280666. doi: 10.3389/fneph.2023.1280666. PMID: 38022724; PMCID: PMC10664753.
5. Evans, L., Rhodes, A., Alhazzani, W., Antonelli, M., Coopersmith, C. et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med 47(11): 1181-1247.
6. Grabczak, E. M., Krenke, R., Przybylski, M., Kolkowska-Lesniak, A., Chazan, R. & Dzieciatkowski, T. Prevalence of Pulmonary Infections Caused by Atypical Pathogens in Non-HIV Immunocompromised Patients. Adv Exp Med Biol 2016; 935: 1-11.
7. Isselbacher, E. M., Preventza, O., Hamilton Black, J., Augoustides, J. G., Beck, et al. 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation 146(24): e334-e482.
8. Mcdonagh, T. A., Metra, M., Adamo, M., Gardner, R. S., Baumbach, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. European Heart Journal 42(36): 3599-3726.