ARTYKUŁ POGLĄDOWY / REVIEW PAPER
Otrzymano/Submitted: 08.06.2026 • Zaakceptowano/Accepted: 25.06.2026

Diagnostic errors and pitfalls in geriatric cardiology – a review illustrated by two difficult cases in older patients

Magdalena Wiciak[1], Robert Krion[2]

[1] Department of Cardiology and Cardiac Electrotherapy II, University Clinical Centre in Gdańsk

[2] Department of Emergency Medicine, Medical University of Gdańsk

Summary

Diagnostic error is among the most frequent and most serious threats to quality of patient care, and cardiovascular events are its leading cause. In older patients the risk increases because of atypical or oligosymptomatic presentation, multimorbidity, polypharmacy, frailty, cognitive impairment and communication barriers. Based on a structured PubMed search, this review discusses the mechanisms of error (cognitive and geriatric) and the principal pitfalls in the diagnosis of acute coronary syndromes (silent infarction, troponin interpretation, mechanical complications), heart failure with preserved ejection fraction, aortic stenosis, and rhythm and conduction disorders, including embolic stroke of undetermined source. The thesis is illustrated by two difficult cases: a silent apical infarction complicated by cardiac rupture in a 91-year-old, and a cardiomyopathy diagnosed during loop-recorder monitoring in a 71-year-old. Practical strategies to reduce error are presented. *Geriatria 2026;20:97-104. doi: 10.53139/G.20262030*

Keywords: diagnostic error, geriatric cardiology, silent myocardial infarction, troponin, atrial fibrillation, cardiac rupture

Introduction

Diagnostic error–understood as incorrect diagnosis of the disease or its absence- is one of the most common and most dangerous threats to patient safety. In the United States alone, diagnostic errors are estimated to account for nearly 800,000 deaths or permanent disabilities each year [1]. Unlike therapeutic errors, which are usually apparent, a diagnostic error often remains hidden until a complication occurs, which makes it harder to detect and correct.

Three disease groups: vascular events, infections and cancers, with vascular events – including myocardial infarction and stroke – constituting the single largest category [2]. Among these, myocardial infarction remains one of the most frequently missed diagnoses in emergency departments, and the symptoms that most often lead to error are chest pain and dyspnoea wrongly attributed to non-cardiac causes [3,4]. The scale and consequences of these mistakes make cardiology a field with particularly high diagnostic stakes.

Older patients are a particularly vulnerable group. Heart disease in this population more often follows an atypical or oligosymptomatic course, and the diagnosis is hindered by multimorbidity, polypharmacy, frailty, cognitive impairment and communication barriers such as hearing loss [5]. A further problem is the under--representation of older people – and especially frail, multimorbid patients – in clinical trials, which weakens the evidence base for decisions made in this population [5]. Because older people bear the greatest burden of cardiovascular disease, the consequences of a delayed or incorrect diagnosis are most severe in this group.

The aim of this paper is to discuss the mechanisms by which diagnostic errors arise in geriatric cardiology and the most important pitfalls in individual clinical domains – acute coronary syndromes, heart failure, valvular disease, and rhythm and conduction disorders – and to present practical strategies for reducing them. The argument is illustrated by two difficult cases from our own practice that bring the pitfalls discussed into focus.

Methods

A structured search of the PubMed/MEDLINE database covering publications up to May 2026 was performed. Groups of terms corresponding to the review topics were combined with Boolean operators, including: diagnostic error; misdiagnosis; myocardial infarction AND (silent OR atypical OR unrecognised); elderly OR older adults; high-sensitivity troponin; heart

ORCID: Magdalena Wiciak 0009-0007-9221-4063, Robert Krion 0009-0007-0857-2555

failure with preserved ejection fraction; aortic stenosis AND low-flow low-gradient; atrial fibrillation AND (screening OR monitoring); embolic stroke of undetermined source; hypertrophic cardiomyopathy; cardiac rupture; cognitive bias AND diagnostic error. The search was supplemented by current scientific-society guidelines and by screening the reference lists of the retrieved articles. Publications in English and Polish were considered, with preference given to guidelines, systematic reviews and meta-analyses, and large clinical studies; items were selected for relevance and methodological quality. The data were synthesised narratively, grouped around mechanisms of error and clinical domains. This is a narrative review with a structured search method rather than a full systematic review.

Mechanisms of diagnostic error in older people

Cognitive mechanisms

Most diagnostic errors have a cognitive basis. In the dual-process model, a distinction is drawn between fast, intuitive processes (System 1), based on pattern recognition, and slow, analytical processes (System 2). System 1 is efficient and indispensable in everyday practice, but in atypical situations it becomes a source of systematic distortion [6].

The most common cognitive traps in medical proceedings include anchoring (excessive attachment to the initial hypothesis), premature closure (stopping the search after the first plausible diagnosis), the availability heuristic (overweighting diagnoses easily recalled from memory), confirmation bias, base-rate neglect, and "diagnostic momentum" – the uncritical perpetuation of a diagnosis made earlier by another clinician [6]. In primary care, diseases with an atypical or non-specific presentation – including myocardial infarction – are misdiagnosed most often [7]. Anchoring is particularly important when a patient is admitted for an elective procedure for a defined indication: the team's attention focuses on that indication, and a coexisting, evolving disease process may go unrecognised.

Geriatric specifics

In older people, factors independent of clinical reasoning predispose to error. Acute illness often manifests not with typical organ-specific symptoms but with so-called non-specific presentations: a fall, delirium, an abrupt decline in functional status, immobility or anorexia. Such a picture is easily diverted onto a false diagnostic track. Multimorbidity masks symptoms, frailty and cognitive impairment hamper the gathering of a reliable history, and there is a widespread tendency to attribute complaints to "old age". The American Heart Association scientific statement on acute coronary syndromes in older adults explicitly identifies geriatric syndromes – frailty, multimorbidity and dementia – as factors that hinder diagnosis and treatment [5].

A separate, underappreciated source of error is polypharmacy. Drugs may both mask the symptoms of heart disease (e.g. beta-blockers blunting the tachycardic response to ischaemia or hypovolaemia) and mimic cardiac pathology or distort test results (diuretics, agents with anticholinergic activity, drugs affecting electrolyte balance). Interactions and adverse effects are easily mistaken for a new disease or for an exacerbation of an existing one.

A tool for structuring the assessment of an older patient is comprehensive geriatric assessment – a multidimensional, interdisciplinary diagnostic and therapeutic process that in the hospital setting reduces, among other things, the risk of delirium, falls and admission to long-term care. It is also worth remembering that in older, multimorbid patients an elevated troponin concentration more often reflects myocardial injury or a type 2 infarction (resulting from an oxygen supply–demand mismatch) than a classic type 1 infarction due to atherosclerotic plaque rupture; this distinction, defined in the Fourth Universal Definition of Myocardial Infarction, is often difficult and there is no simple discriminating tool [8,14].

Diagnostic pitfalls in selected areas of geriatric cardiology

Acute coronary syndromes

With age, the proportion of infarctions that are without pain symptoms or present with atypical symptoms – such as dyspnoea, weakness, altered consciousness, syncope or a fall – increases; in the very old, chest pain may be absent altogether. Silent myocardial infarction is not benign: but quite the opposite it is associated with a substantially increased risk of sudden cardiac death [9]. The symptom pattern also differs between the sexes, which is an additional pitfall [10]. Women experience silent heart attacks more often; they tend to have „masked heart attacks". In practice, this means that in an older patient the absence of typical anginal pain does not exclude acute ischaemia, and a low threshold of suspicion should also apply to non-cardiac presentations.

Interpreting troponin in older people is a separate challenge. The introduction of high-sensitivity assays has increased the detection of infarction but also the risk of overdiagnosis [11]. The Fourth Universal Definition of Myocardial Infarction distinguishes type 1 infarction (due to atherosclerotic plaque rupture or erosion) from type 2 infarction (secondary to an oxygen supply–demand mismatch, e.g. in tachyarrhythmia, anaemia, hypotension or hypoxaemia) and from myocardial injury, in which troponin is elevated without clinical evidence of ischaemia [8]. In older patients the non-infarction causes of a troponin rise are numerous – renal failure, heart failure, pulmonary embolism, sepsis or inflammatory states [12] – and in patients with chronic kidney disease the concentration rises as glomerular filtration declines [13]. The 99th-percentile value (the upper reference limit) is further influenced by age, sex and the choice of reference population [14]. The trend on serial measurement (a significant rise or fall) is therefore decisive, rather than a single value; the rapid 0/1 h and 0/2 h algorithms retain high sensitivity but lose specificity in older patients and in renal disease, which must be taken into account [8,12,15]. Once an acute coronary syndrome has been diagnosed in older patients, an invasive strategy is generally beneficial, reducing mortality at the cost of a higher bleeding risk [16].

The electrocardiogram, too, can be a trap. Left bundle branch block and a paced rhythm mask typical ST-segment elevation, where the Sgarbossa criteria are helpful; infarction of the infero-basal wall requires posterior leads, and early ischaemia may manifest only as tall, "hyperacute" T waves [8]. In a patient with permanent atrial fibrillation, assessment of the ST segment is additionally hampered.

A particularly dangerous, though rare, pitfall lies in the **mechanical complications of infarction.** Free-wall rupture, ventricular septal rupture and papillary muscle rupture are infrequent – below one per cent in the reperfusion era – but carry a high and almost unchanging mortality [17,18]. Free-wall rupture may be abrupt (a "blow-out"), leading to electromechanical dissociation and immediate death, or subacute ("oozing"), with progressive tamponade; a contained form is the pseudoaneurysm. These complications usually occur within the first days of infarction, more often in older patients, in a first infarction recognised late, and in single-vessel disease with poor collateral circulation. They often remain unrecognised until catastrophic tamponade, and the principal differentiating investigation is echocardiography [17,19]; surgical management is addressed in current consensus documents [20,21].

Illustrative case 1. A 91-year-old man with persistent atrial fibrillation (figure 1) was scheduled for pacemaker implantation due to tachycardia-bradycardia syndrome. On admission, he denied chest pain and was considered in good condition. He had no history

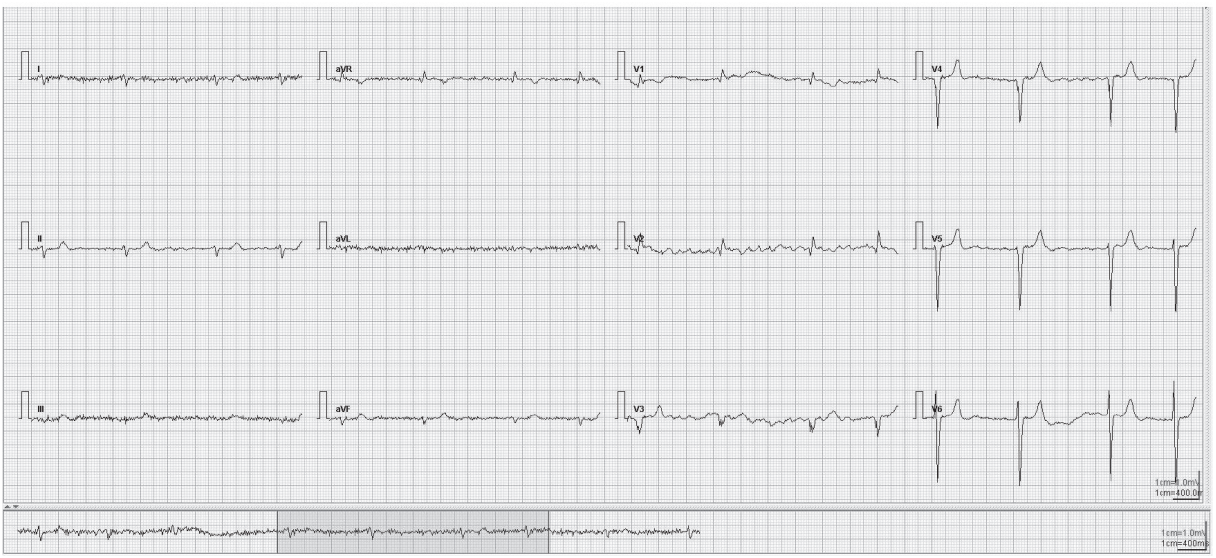

Figure 1. Admission ECG (case 1) – atrial fibrillation with slow ventricular response (~50 bpm); personal data removed

of previous myocardial infarction. Laboratory tests revealed no significant abnormalities (figure 2). An echocardiogram performed 4 months earlier revealed an LVEF of 60% and no regional wall motion abnormalities. After uncomplicated VVI pacemaker implantation, the patient died suddenly on the second day. Post-mortem examination revealed cardiac tamponade (750 ml of blood) due to apical rupture in the course of a recent apical infarction, accompanied by significant coronary artery stenosis. The case combines several pitfalls: silent infarction in a very elderly person in the period between the performance of ECHO and admission to hospital, anchoring attention to the indication for pacing, and the need to differentiate post-infarction rupture from iatrogenic perforation by the lead.

TnI [ng/ml]	0,01
CK-MB [ng/ml]	4,2
BNP [pg/ml]	211
NT-pro-BNP [pg/ml]	1870
CRP [mg/l]	8,08
Kreat. [mg/dl]	1,5
Chol. [mg/dL]	198

Figure 2. Laboratory test results

In a patient after recent pacemaker implantation, tamponade must be differentiated from lead perforation of the cardiac wall; imaging of the lead position and device interrogation are helpful [22,23,24]. A post-infarction aetiology is supported by the presence of freshly infarcted, softened myocardium around the rupture site and by a normal device interrogation. This distinction matters not only clinically but also medico-legally.

Heart failure with preserved ejection fraction

Heart failure with preserved ejection fraction (HFpEF) predominates in older people, especially women with hypertension, obesity and atrial fibrillation, and its diagnosis is frequently delayed or missed. The symptoms – exertional dyspnoea, fatigue, oedema – are non-specific and readily attributed to multimorbidity, obesity, lung disease or ageing [25,26]. **A normal left ventricular ejection fraction paradoxically lulls vigilance, even though it does not exclude significant heart failure.**

The HFA-PEFF algorithm aids diagnosis: after an initial pre-test assessment it integrates functional and morphological echocardiographic criteria with natriuretic peptide concentrations and, in equivocal cases, recommends diastolic stress echocardiography or invasive haemodynamic assessment [26,27]. An alternative is the H2FPEF score. It should be noted that **natriuretic peptide concentrations** may be falsely low in obese patients and falsely high in atrial fibrillation, renal failure and old age, which further complicates interpretation and can cause both missed and overdiagnoses [26,27].

Aortic stenosis

Calcific aortic stenosis is the most common acquired valvular disease in older people, and its frequency rises with age. It is sometimes underdiagnosed and undertreated, despite the availability of effective therapies including transcatheter aortic valve implantation (TAVI) [28]. An important pitfall is its paucity of symptoms: older patients often unconsciously limit their activity and attribute dyspnoea to "old age", so that symptoms go unreported and the lesion – despite being severe – is regarded as asymptomatic.

The most diagnostically difficult forms are the low-gradient variants: classic low-flow, low-gradient stenosis with reduced ejection fraction, and paradoxical low-flow, low-gradient stenosis with preserved ejection fraction, typical of older patients with a small, hypertrophied left ventricle. In these situations the classic gradient criteria fail to reveal the severity of the lesion; dobutamine stress echocardiography – which distinguishes truly severe from pseudo-severe stenosis and assesses flow reserve – and, in equivocal cases, assessment of the degree of valve calcification on computed tomography are helpful [28,29]. Underestimating the severity of the lesion delays interventional treatment and worsens prognosis.

Rhythm and conduction disorders and embolic stroke of undetermined source

Detecting paroxysmal atrial fibrillation in older people can be difficult, because the episodes are often asymptomatic. The 2024 ESC guidelines recommend opportunistic screening for atrial fibrillation in people aged 65 years and older by pulse-taking or an ECG recording [30], and active, prolonged monitoring incre-

ases detection severalfold [31,32]. However, the clinical benefit of detecting subclinical atrial fibrillation is not unequivocal – in a trial using an implantable loop recorder, increased detection did not translate into a significant reduction in strokes [33] – which raises the question of the arrhythmia-burden threshold that justifies anticoagulation.

Growing importance is attached to the concept of atrial cardiopathy as a substrate for embolism independent of documented atrial fibrillation; its markers include left atrial enlargement, an abnormal P wave and elevated NT-proBNP [34]. In embolic stroke of undetermined source (ESUS), in-depth cardiac work-up is sometimes required; randomised trials have not shown superiority of direct oral anticoagulants over acetylsalicylic acid in an unselected ESUS population, which underscores the need to identify the specific embolic mechanism [34]. In some patients a patent foramen ovale remains relevant, and its causal relationship to the stroke is assessed with scores such as RoPE [35]. It should be remembered that prolonged monitoring may reveal not only atrial fibrillation but also clinically significant conduction disorders, pauses or a cardiomyopathic substrate – and it was precisely this situation that became the crux of the second case.

Illustrative case 2. A 71-year-old man with recurrent embolic stroke of undetermined source (three events within eight months) was referred for implantation of a loop recorder to search for atrial fibrillation. Monitoring did not reveal atrial fibrillation but instead disclosed advanced conduction-system disease, and further work-up showed hypertrophic cardiomyopathy with extensive late gadolinium enhancement on magnetic resonance imaging (figure 3). On the first echocardiogram, left ventricular hypertrophy had been missed (limited acoustic window, lower-resolution equipment). The case illustrates anchoring on atrial fibrillation as the sole source of embolism and the limitations of echocardiography. It exemplifies pitfalls common to the whole spectrum of older adults.

Hypertrophic cardiomyopathy in older people is sometimes unrecognised or mistaken for hypertensive hypertrophy, and its picture differs from the sarcomeric form in younger patients. A rigid wall-thickness cut-off

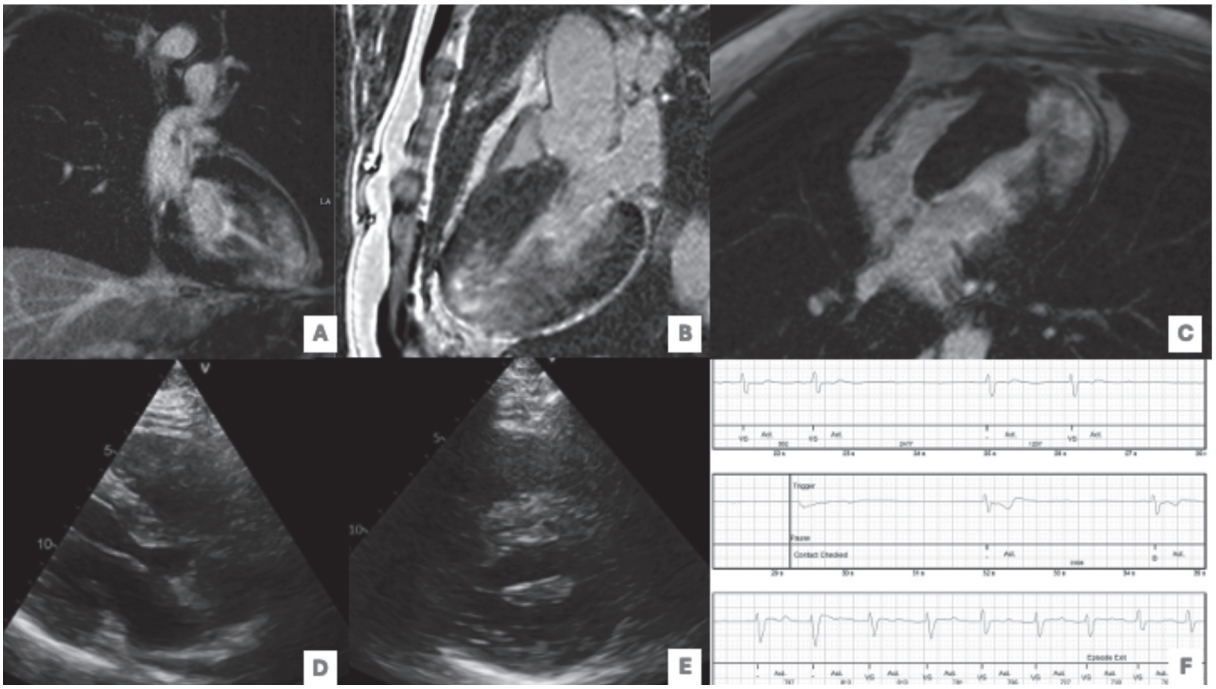

Figure 3. A-C Cardiac MRI imaging in 2-chamber (A), 3-chamber (B) and 4-chamber (C) view with extensive LGE. D-E Transthoracic echocardiography showing evident LV hypertrophy.F Report from the AVB episode registered by the loop recorder

of ≥15 mm distorts the diagnosis, so thresholds adjusted for age, sex and body surface area have been proposed [36]. Cardiac magnetic resonance with assessment of late gadolinium enhancement has key diagnostic and prognostic value – the amount of late enhancement correlates with the risk of sudden death [37] – and tissue characterisation allows differentiation of cardiomyopathy phenotypes and subgroups, including patients with fibrosis and without obstruction [38].

Imaging and biomarker pitfalls

Transthoracic echocardiography, although widely available, has limitations dependent on the quality of the acoustic window, the operator's experience and the equipment – which in the second case led to the hypertrophy being missed. Image quality can be improved with a contrast agent (delineation of the endocardium), but when the result is equivocal or discordant the decisive method is cardiac magnetic resonance, which allows precise assessment of wall thickness and mass and tissue characterisation. Chest-wall deformities such as pectus excavatum may compress the heart and distort assessment of the right ventricle (paradoxical septal motion, apparent inflow abnormalities), which is sometimes misinterpreted or masks true pathology [39].

The interpretation of biomarkers requires equal caution. Troponin should be read dynamically and in the context of age, renal function and the choice of reference values [8,13], and natriuretic peptides with account taken of obesity, atrial fibrillation and renal failure [25,26]. In the diagnosis of left ventricular hypertrophy the thresholds should be adjusted for demographic characteristics [36]. The overarching practical conclusion is simple: a discrepancy between the clinical picture and the result of a single test in an older patient should prompt verification with a higher-resolution method or a multimodal approach, rather than premature closure of the diagnosis.

How to reduce errors – practical conclusions

- Maintain vigilance for ischaemia even in patients admitted for elective procedures; consider serial ECGs and troponin measurement at any deterioration, and interpret the result dynamically [3,4,8,12].
- Differentiate type 1 and type 2 infarction from myocardial injury, taking account of age, renal function and comorbidities [8,12-15].
- Treat non-specific symptoms (dyspnoea, weakness, syncope, a fall, delirium) as a possible mask of heart disease in older people [5,9].
- Account for the effect of polypharmacy – drugs may mask or mimic cardiac symptoms and distort test results.
- Use comprehensive geriatric assessment as a framework for the systematic evaluation of an older patient [40].
- When the clinical picture and imaging disagree, verify the diagnosis with a higher-resolution method (cardiac magnetic resonance) or a multimodal approach [36-39].
- Apply structured strategies to reduce cognitive bias (deliberate consideration of alternatives, a diagnostic time-out, checklists), especially when anchored on a procedural indication [6].
- Differentiate mechanical complications of infarction from iatrogenic complications (lead perforation) using echocardiography, imaging and device interrogation [17,22-24].
- Make key decisions as a team (heart team) and use post-mortem examination as a tool for verifying the diagnosis and providing feedback to the team.

Limitations

This is a narrative review based on a structured but single-source (PubMed) search, without dual, independent study selection, which entails a risk of omitting some reports and of selection bias. The cases presented are illustrative and serve to highlight the pitfalls discussed rather than to support inferences about frequency or causation.

Conclusions

Diagnostic errors in geriatric cardiology arise from the overlap of cognitive mechanisms and the specifics of the older patient – atypical presentation, multimorbidity, polypharmacy and frailty. The most dangerous pitfalls concern silent myocardial infarction and its mechanical complications, the interpretation of troponin and the electrocardiogram, the underdiagnosis of heart failure with preserved ejection fraction and aortic stenosis, and rhythm and conduction disorders, including embolic stroke of undetermined source. Awareness of these pitfalls, dynamic interpretation of investigations, a low threshold for imaging verification, and structured reduction of cognitive bias – supported by comprehensive geriatric assessment and teamwork – can genuinely

improve the safety of older cardiac patients. Both cases presented show that it is not a lack of knowledge but premature closure of the diagnostic process that most often leads to a tragically consequential oversight.

Conflict of interest
None

Correspondence address
Magdalena Wiciak
Department of Cardiology and Cardiac Electrotherapy II, University Clinical Centre in Gdańsk
ul. Mariana Smoluchowskiego 17, 80-214 Gdańsk
☎ (+48 58) 584 47 60
mwiciak@uck.gda.pl

References

1. Newman-Toker DE, Nassery N, Schaffer AC, et al. Burden of serious harms from diagnostic error in the USA. BMJ Qual Saf. 2024;33(2):109-120. doi:10.1136/bmjqs-2021-014130. PMID: 37460118.
2. Newman-Toker DE, Schaffer AC, Yu-Moe CW, et al. Serious misdiagnosis-related harms in malpractice claims: the „Big Three" – vascular events, infections, and cancers. Diagnosis (Berl). 2019;6(3):227-240. doi:10.1515/dx-2019-0019. PMID: 31535832.
3. Sharp AL, Baecker A, Nassery N, et al. Missed acute myocardial infarction in the emergency department – standardizing measurement of misdiagnosis-related harms using the SPADE method. Diagnosis (Berl). 2020;8(2):177-186. doi:10.1515/dx-2020-0049. PMID: 32701479.
4. Kwok CS, Bennett S, Azam Z, et al. Misdiagnosis of acute myocardial infarction: a systematic review of the literature. Crit Pathw Cardiol. 2021;20(3):155-162. doi:10.1097/HPC.0000000000000256. PMID: 33606411.
5. Damluji AA, Forman DE, Wang TY, et al. Management of acute coronary syndrome in the older adult population: a scientific statement from the American Heart Association. Circulation. 2023;147(3):e32-e62. doi:10.1161/CIR.0000000000001112. PMID: 36503287.
6. O'Sullivan ED, Schofield SJ. Cognitive bias in clinical medicine. J R Coll Physicians Edinb. 2018;48(3):225-232. doi:10.4997/JRCPE.2018.306. PMID: 30191910.
7. Kostopoulou O, Delaney BC, Munro CW. Diagnostic difficulty and error in primary care – a systematic review. Fam Pract. 2008;25(6):400-413. doi:10.1093/fampra/cmn071. PMID: 18842618.
8.. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). J Am Coll Cardiol. 2018;72(18):2231-2264. doi:10.1016/j.jacc.2018.08.1038. PMID: 30153967.
9. Cheng YJ, Jia YH, Yao FJ, et al. Association between silent myocardial infarction and long-term risk of sudden cardiac death. J Am Heart Assoc. 2021;10(1):e017044. doi:10.1161/JAHA.120.017044. PMID: 33372536.
10. Ferry AV, Anand A, Strachan FE, et al. Presenting symptoms in men and women diagnosed with myocardial infarction using sex-specific criteria. J Am Heart Assoc. 2019;8(17):e012307. doi:10.1161/JAHA.119.012307. PMID: 31431112.
11. McCarthy CP, Wasfy JH, Januzzi JL. Is myocardial infarction overdiagnosed? JAMA. 2024;331(19):1623-1624. doi:10.1001/jama.2024.5235. PMID: 38656331.
12. Chauin A. The main causes and mechanisms of increase in cardiac troponin concentrations other than acute myocardial infarction (Part 1). Vasc Health Risk Manag. 2021;17:601-617. doi:10.2147/VHRM.S327661. PMID: 34584417.
13. Chesnaye NC, Szummer K, Bárány P, et al. Association between renal function and troponin T over time in stable chronic kidney disease patients. J Am Heart Assoc. 2019;8(21):e013091. doi:10.1161/JAHA.119.013091. PMID: 31662068.
14. Krintus M, Kozinski M, Boudry P, et al. Defining normality in a European multinational cohort: critical factors influencing the 99th percentile upper reference limit for high sensitivity cardiac troponin I. Int J Cardiol. 2015;187:256-263. doi:10.1016/j.ijcard.2015.03.282. PMID: 25838227.
15. Verardi R, Iannopollo G, Casolari G, et al. Management of acute coronary syndrome in elderly patients: a narrative review through decisional crossroads. J Clin Med. 2024;13(20):6034. doi:10.3390/jcm13206034. PMID: 39457985.
16. Improta R, Di Pietro G, Piccialuti A, et al. Invasive and conservative management of elderly patients presenting with acute coronary syndrome: a meta-analysis. Int J Cardiol. 2024;417:132523. doi:10.1016/j.ijcard.2024.132523. PMID: 39255918.
17. Gong FF, Vaitenas I, Malaisrie SC, Maganti K. Mechanical complications of acute myocardial infarction: a review. JAMA Cardiol. 2021;6(3):341-349. doi:10.1001/jamacardio.2020.3690. PMID: 33295949.
18. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. JACC Cardiovasc Interv. 2019;12(18):1825-1836. doi:10.1016/j.jcin.2019.04.039. PMID: 31537282.
19. Lorusso R, Cubeddu RJ, Matteucci M, et al. Ventricular pseudoaneurysm and free wall rupture after acute myocardial infarction: JACC Focus Seminar 4/5. J Am Coll Cardiol. 2024;83(19):1902-1916. doi:10.1016/j.jacc.2023.10.054. PMID: 38719370.
20. Kaczorowski DJ, Takeda K, Atluri P, et al. 2025 AATS expert consensus document: surgical management of acute myocardial infarction and associated complications. J Thorac Cardiovasc Surg. 2025;170(5):1327-1344. doi:10.1016/j.jtcvs.2025.04.013. PMID: 40320004.
21. Cubeddu RJ, Lorusso R, Ronco D, et al. Ventricular septal rupture after myocardial infarction: JACC Focus Seminar 3/5. J Am Coll Cardiol. 2024;83(19):1886-1901. doi:10.1016/j.jacc.2024.01.041. PMID: 38719369.
22. Vatterott PJ, Syed IS, Khan AH. Lead extraction imaging. Card Electrophysiol Clin. 2018;10(4):625-636. doi:10.1016/j.ccep.2018.07.007. PMID: 30396577.

23. Hamada Y, Sakaki M, Watanabe Y, et al. Delayed right ventricular pacemaker lead perforation 9 years after implantation. Am J Case Rep. 2023;24:e940291. doi:10.12659/AJCR.940291. PMID: 37401051.
24. Feldt K, Dalén M, Meduri CU, et al. Reducing cardiac tamponade caused by temporary pacemaker perforation in transcatheter aortic valve replacement. Int J Cardiol. 2023;377:26-32. doi:10.1016/j.ijcard.2023.01.015. PMID: 36640966.
25. Gharagozloo K, Mehdizadeh M, Heckman G, et al. Heart failure with preserved ejection fraction in the elderly population: basic mechanisms and clinical considerations. Can J Cardiol. 2024;40(8):1424-1444. doi:10.1016/j.cjca.2024.04.006. PMID: 38604339.
26. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42(36):3599-3726. doi:10.1093/eurheartj/ehab368. PMID: 34447992.
27. Pieske B, Tschöpe C, de Boer RA, et al. How to diagnose heart failure with preserved ejection fraction: the HFA-PEFF diagnostic algorithm. Eur Heart J. 2019;40(40):3297-3317. doi:10.1093/eurheartj/ehz641. PMID: 31504452.
28. Vahanian A, Beyersdorf F, Praz F, et al. 2021 ESC/EACTS guidelines for the management of valvular heart disease. Eur Heart J. 2022;43(7):561-632. doi:10.1093/eurheartj/ehab395. PMID: 34453165.
29. Mogensen NSB, Ali M, Carter-Storch R, et al. Dobutamine stress echocardiography in low-gradient aortic stenosis. J Am Soc Echocardiogr. 2024;37(11):1023-1033. doi:10.1016/j.echo.2024.06.017. PMID: 38972614.
30. Van Gelder IC, Rienstra M, Bunting KV, et al. 2024 ESC guidelines for the management of atrial fibrillation developed in collaboration with the EACTS. Eur Heart J. 2024;45(36):3314-3414. doi:10.1093/eurheartj/ehae176. PMID: 39210723.
31. Nies RJ, Nettersheim FS, Braumann S, et al. Right ventricular dysfunction and impaired right ventricular-pulmonary arterial coupling in paradoxical low-flow, low-gradient aortic stenosis. Eur J Heart Fail. 2024;26(11):2340-2352. doi:10.1002/ejhf.3329. PMID: 38887164.
32. Gladstone DJ, Wachter R, Schmalstieg-Bahr K, et al. Screening for atrial fibrillation in the older population: a randomized clinical trial. JAMA Cardiol. 2021;6(5):558-567. doi:10.1001/jamacardio.2021.0038. PMID: 33625468.
33. Svendsen JH, Diederichsen SZ, Højberg S, et al. Implantable loop recorder detection of atrial fibrillation to prevent stroke (The LOOP Study): a randomised controlled trial. Lancet. 2021;398(10310):1507-1516. doi:10.1016/S0140-6736(21)01698-6. PMID: 34469766.
34. Migdady I, Russman A, Buletko AB. Atrial fibrillation and ischemic stroke: a clinical review. Semin Neurol. 2021;41(4):348-364. doi:10.1055/s-0041-1726332. PMID: 33851396.
35. Kent DM, Wang AY. Patent foramen ovale and stroke: a review. JAMA. 2025;334(16):1463-1473. doi:10.1001/jama.2025.10946. PMID: 40720119.
36. Shiwani H, Davies RH, Topriceanu CC, et al. Demographic-based personalized left ventricular hypertrophy thresholds for hypertrophic cardiomyopathy diagnosis. J Am Coll Cardiol. 2025;85(7):685-695. doi:10.1016/j.jacc.2024.10.082. PMID: 39772357.
37. Kiaos A, Daskalopoulos GN, Kamperidis V, et al. Quantitative late gadolinium enhancement cardiac magnetic resonance and sudden death in hypertrophic cardiomyopathy: a meta-analysis. JACC Cardiovasc Imaging. 2023;17(5):489-497. doi:10.1016/j.jcmg.2023.07.005. PMID: 37632503.
38. Neubauer S, Kolm P, Ho CY, et al. Distinct subgroups in hypertrophic cardiomyopathy in the NHLBI HCM Registry. J Am Coll Cardiol. 2019;74(19):2333-2345. doi:10.1016/j.jacc.2019.08.1057. PMID: 31699273.
39. Raggio IM, Toselli L, Valle M, et al. Cardiac decompression and right ventricular function improvement after bar removal in patients with pectus excavatum. Int J Cardiovasc Imaging. 2024;40(11):2383-2390. doi:10.1007/s10554-024-03243-3. PMID: 39312109.
40. Veronese N, Custodero C, Demurtas J, et al. Comprehensive geriatric assessment in older people: an umbrella review of health outcomes. Age Ageing. 2022;51(5):afac104. doi:10.1093/ageing/afac104. PMID: 35524746.