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Late presenters with ST-elevation myocardial infarction and thromboembolic complications: a treatment challenge: a case report

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Abstract

Background Late presenters with ST-elevation myocardial infarction pose a considerable challenge in the field of cardiovascular medicine. These individuals, who delay seeking medical attention after the onset of ST-elevation myocardial infarction symptoms, often face substantial difficulties in treatment. The coronavirus disease 2019 pandemic led to a significant increase in the number of late presenters. By increasing the frequency of complications such as thromboembolic events in the course of left ventricular thrombus, the pandemic necessitated the refinement of existing management strategies.

Case presentation In this paper, we present two White male patients of Central European (Polish) descent (50 and 64 years old) who, although reported to have had acute ST-elevation myocardial infarction, turned out to be ST-elevation myocardial infarction latecomers. In both cases, we were dealing with the presence of left ventricular thrombus and complications related to the central nervous system. On the basis of these two patients, we discuss the role of revascularization in latecomer ST-elevation myocardial infarction patients. We present the position of direct oral anticoagulants in the left ventricular thrombus treatment and show that, in limited cases of a huge thrombus, cardiac surgery is a treatment of choice.

Conclusions As left ventricular thrombus is still relatively common in ST-elevation myocardial infarction latecomers, we present the current state of knowledge on this topic, emphasizing the need for further research in this area.

Keywords Acute coronary syndrome, Late reperfusion, Left ventricle thrombus, Acute ischemic stroke

Introduction

During the coronavirus disease 2019 (COVID-19) pandemic, a significant reduction in primary percutaneous interventions (PPCI) was observed. The number of PPCIs in Europe between 2019 and 2020 decreased by 19%. Therefore, the pandemic has a severe impact on the diagnosis and treatment of ST-elevation myocardial infarction (STEMI) patients [1, 2]. The most important reasons were a limited number of hospital staff due to the illness, overload related to the hospitalization of COVID-19 patients, fear of contracting the virus, and misinterpretation of cardiac pain for respiratory causes.

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The prolonged time of the ambulance arrival to the patient and longer door-to-balloon time caused by the specific COVID-19 protocols for screening patients and preparing equipment and personnel in the catheterization laboratory was also not without significance. All the abovementioned reasons increased the pool of patients called STEMI latecomers, which defines patients with symptoms lasting more than 12 hours. This, in turn, has resulted in an increased number of MI complications, worse clinical outcomes, and higher mortality [3]. The treatment of STEMI late presenters is particularly difficult and involves an individual approach.

MI increases the risk of left ventricular thrombus (LVT), as it involves every aspect of Virchow's triad: blood stagnation, tissue injury, and blood hypercoagulability [4, 5]. The risk of LVT formation is particularly increased in the course of MI caused by left anterior descending artery (LAD) occlusion. Its occurrence is also correlated with the size of the necrotic area, so the risk is higher in the late-presenter STEMI patients [4–6]. The major consequence of LVT is systemic thromboembolism, including ischemic stroke. Therefore, it requires anticoagulation treatment, usually for about 3–6 months [4, 5, 7]. The standard treatment is to apply vitamin K antagonists (VKA), but the use of direct oral anticoagulants (DOACs) is gaining more attention. Although their use in the course of LVT is still off-label [7, 8], many studies show promising results [9]. However, the treatment in case of coexisting ischemic stroke is challenging, especially in terms of the risk of hemorrhagic conversion, and should be provided carefully.

In our article, we present two cases of STEMI late presenters with coexisting symptoms of peripheral embolism. We share our doubts and experiences, and finally, we discuss state-of-the-art knowledge on the topic.

Case description

Case 1 (November 2021)

A 50-year-old White male of Central European (Polish) descent with arterial hypertension, hyperlipidemia, and a history of ST-elevation myocardial infarction (STEMI) was admitted to the hospital for consecutive STEMI. The previous MI was 4 years ago, and it was STEMI of the inferior wall treated with PPCI of the right coronary artery (RCA). After discharge from the hospital, the patient did not follow any doctor's recommendations, quickly stopped taking all medications, and continued to smoke cigarettes.

On admission, the patient complained of chest pain lasting about an hour. It was accompanied by dizziness, nausea, and vomiting. There were no signs of pulmonary congestion, and the patient was hemodynamically stable. In the electrocardiography (ECG), there were Q waves in precordial leads with ST-segment elevation in V3–V6, I, and aVL (Fig. 1). Extensive anterior STEMI was diagnosed. The patient received a loading dose of antiplatelet drugs (300 mg of aspirin and 180 mg of ticagrelor) and was qualified for urgent coronary angiography, which revealed an occlusion of the LAD. PPCI was made, and a drug-eluting stent (DES) was implanted. There were no significant stenoses in the remaining vessels (Fig. 2).

During the next hours, the patient's symptoms of vertigo and nausea worsened. In the neurological

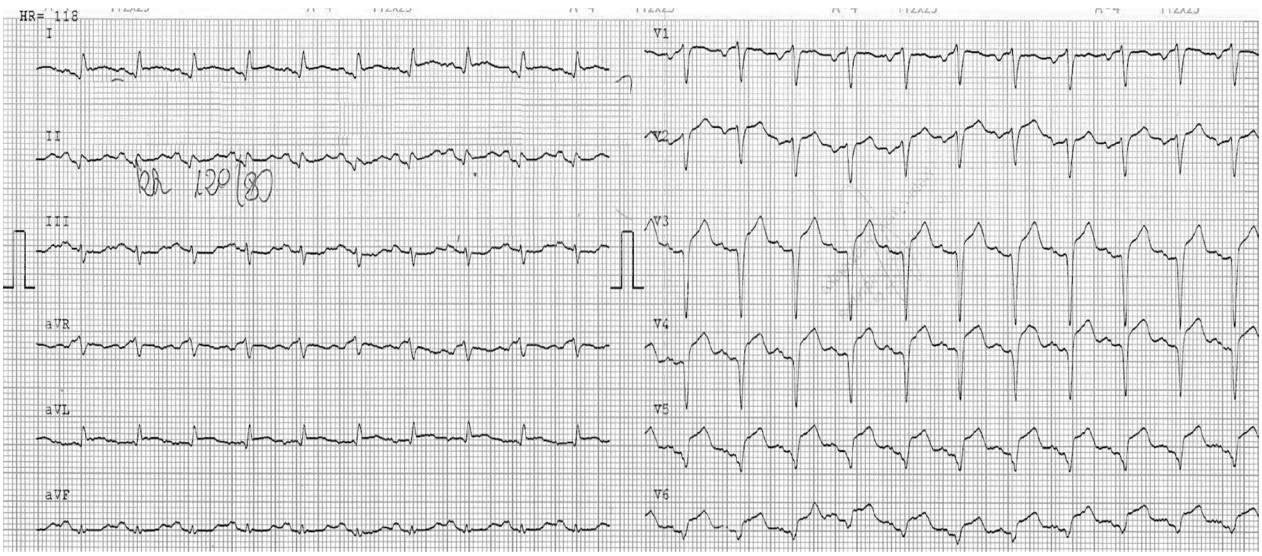

Fig. 1 The electrocardiograph of patient 1 on admission

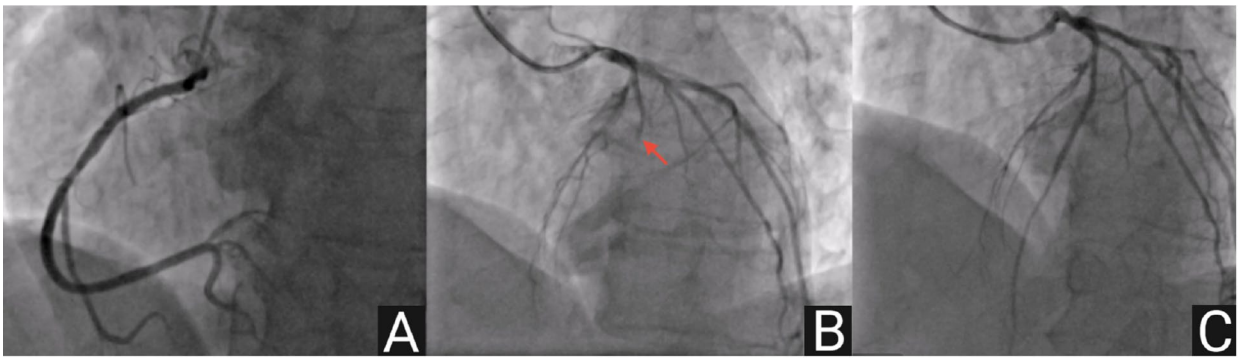

Fig. 2 Coronary angiography and revascularization of patient 1 on admission. (**A**) Left anterior oblique view, right coronary artery—no signs of restenosis in a previously implanted stent in the proximal right coronary artery and 40% of stenosis in the mid-right coronary artery. (**B**) Left anterior oblique cranial view, occlusion of the left anterior descending artery before revascularization; marked with an arrow. (**C**) Left anterior oblique cranial view, Left anterior descending artery after revascularization

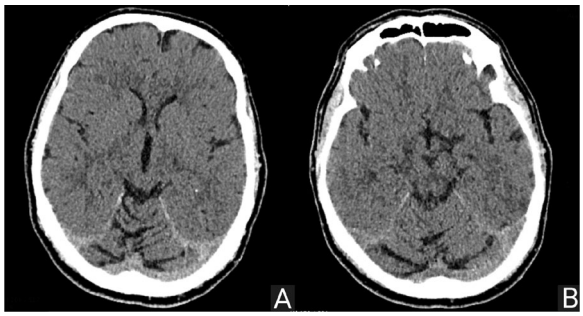

Fig. 3 Computed tomography scan of the head of patient 1 on admission (**A**) and control after several days (**B**). Brain and cerebellum without focal changes; no signs of intracranial bleeding

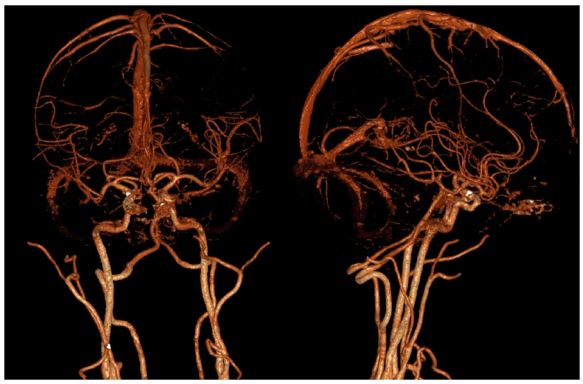

Fig. 4 Head and neck computed tomography angiography of patient 1—no lesions in the main vessels

examination, the patient exhibited convergent strabismus on the right, coarse horizontal nystagmus to the right, and tongue deviation to the left. In the upper limbs, positive deliberate movement signs were noted on the left, with reflexes being weaker on the left compared with the right. In the lower limbs, the reflexes were also weaker on the left. The patient walked on a wider base and had a positive Romberg test. The computer tomography (CT) of the head showed no significant changes (Fig. 3A). Additionally, head and neck CT angiography (angio-CT) did not reveal any hemodynamically significant lesions in large arteries (Fig. 4). Finally, on the basis of the symptoms, neurological examination, and imaging results, transient ischemia of the cerebellum and brainstem was diagnosed. Transthoracic echocardiography (TTE) revealed akinetic–dyskinetic tip and apical segments of the left ventricle (LV) with significantly reduced left ventricular ejection fraction (LVEF; 35%). In addition, a round thrombus was found in the left ventricular tip (Fig. 5). Hemodynamically significant valvular defects were not present. Repeated blood tests revealed normal

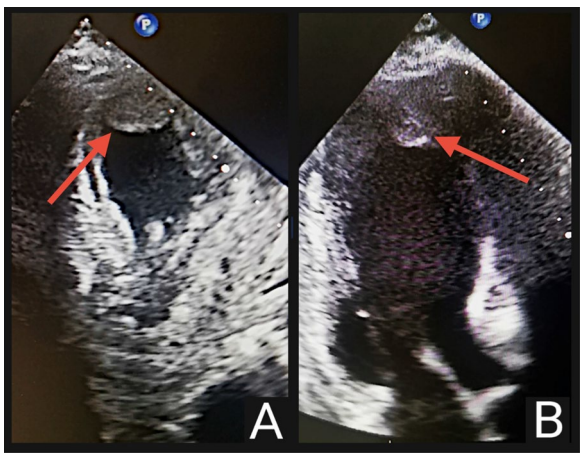

Fig. 5 The transthoracic echocardiography image of patient 1; the thrombus is marked with arrows, modified four-chamber cardiac view (**A**) and three-chamber cardiac view (**B**) projections

troponin T and creatine kinase myocardial band (CK-MB) levels (Table 1). The biochemical results, together with the echocardiography examination, indicated that we were dealing with STEMI latecomer. When questioned carefully, the patient described the chest pain he presented with on admission as atypical. Moreover, he reported feeling worse for about 2 weeks.

Low-molecular-weight heparin (LMWH) at a therapeutic dose was included in the treatment (1 mg/kg orally). The dual antiplatelet treatment was continued. However, ticagrelor was changed for clopidogrel (loading dose of 300 mg, then continued with 75 mg daily). In the following days, a gradual improvement in the neurological condition was observed. Control CT of the brain still showed no pathological changes (Fig. 3B).

On the tenth day of hospitalization, LMWH was changed to dabigatran 2×150 mg, and the patient, in good general condition, with no neurological deficits, was discharged home. In addition to anticoagulants, he was ordered to take one antiplatelet drug (clopidogrel 75 mg), statin (rosuvastatin 20 mg), ezetimibe (10 mg), and medications typical for heart failure with reduced LVEF (HFrEF) including a beta-blocker (BB), angiotensin-converting enzyme inhibitor (ACEI), and eplerenone (with no SGLT2 inhibitors, as they were not in treatment guidelines at that time). The patient was cautioned to take prescribed drugs systematically. In control TTE after 3 months, the thrombus was not present. However, left ventricular function did not improve, as LVEF was still 35%. Dabigatran was discontinued, and aspirin (75 mg daily) was started, with a recommendation to take double antiplatelet therapy for 12 months from the index MI. Due to the risk of LVT recurrence, a follow-up TTE was scheduled in the following 3 months [4, 10].

Table 1 Blood test results of patient 1 (case 1) and patient 2 (case 2)

Marker	Case 1	Case 2	Reference
Leucocytes (cells × 10^3/μl)	12.5	11.7	4.00–10.00
HbA1c (%)	5.4	5.8	4.5–5.7
Natriuretic peptide B (pg/ml)	694.3	4923	2.0–125.0
Troponin T (ng/ml)—baseline	0.004	0.027	0.000–0.014
Troponin T (ng/ml)—peak value	0.027	0.027	0.000–0.014
CK-MB (ng/dl)—baseline	1.87	1.8	0.10–4.94
CK-MB (ng/dl)—peak value	3.8	1.8	0.10–4.94
K^+ (mmol/dl)	4.31	3.95	3.7–5.4
LDL (mg/dl)	176.2	177.8	—
HDL (mg/dl)	75	62	>40
Non-HDL (mg/dl)	195.5	203.6	—
Total cholesterol (mg/dl)	270	266	140–190
CRP (mg/l)	23.48	10.1	0.0–5.0
Creatinine (mg/dl)	1.1	1.08	0.7–1.2

CRP, C-reactive protein; HBA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein

Case 2 (January 2022)

A 64-year-old White male of Central European (Polish) descent, not undergoing treatment for any reason, with no history of smoking, was admitted for PPCI in the course of STEMI of the anterior wall. He was referred by the ambulance team as having chest pain lasting 2 hours with an elevation of the ST segment over the anterior wall in ECG. In the ambulance, the patient received a loading dose of antiplatelet drugs (300 mg of aspirin and 180 mg of ticagrelor) and 5000 units of heparin intravenously, and the pain subsided. On admission, the patient seemed sluggish, his speech was impaired, and there was less muscle strength in the limbs on the right side. When asked, the patient admitted that he had weakened muscle strength in the right limbs and had had difficulty speaking for about 12 hour. For this reason, a CT of the head was performed, which revealed large bilateral hypodense areas in the occipital and parietal lobe (Fig. 6A). The consulting neurologist diagnosed ischemic stroke beyond the time window for intravenous thrombolysis or mechanical thrombectomy. After diagnosing the stroke, the question arose whether the patient needed urgent PPCI. The chest pain turned out to be atypical and was no longer present. There were no signs of pulmonary congestion, and the patient was hemodynamically stable. In the ECG (Fig. 7), not only ST elevations but also QS waves in precordial leads were present. TTE showed an akinetic aneurysm of the apex, significantly reduced LVEF (25%), and a large balloting clot in the LV. On the basis of the echocardiographic image (Fig. 8) and the presence of QS waves in ECG on admission, the patient was classified as a STEMI late presenter. Blood tests (Table 1) confirmed the diagnosis, and PPCI was not done. The pharmacological treatment of MI with one antiplatelet drug (aspirin at a dose of 75 mg) and low-molecular-weight heparin at a therapeutic dose (1 mg/kg orally) was applied, and the patient was transferred to the neurology department. During the next days, the neurological condition temporarily deteriorated. In the CT scans, numerous diffuse hemorrhagic foci were visualized in the previously ischemic areas (Fig. 6B). Only after 2 weeks from the admission, when a significant reduction of hypodense areas and resorption of hemorrhagic lesions were observed, the patient underwent coronary angiography. The study showed a three-vessel form of coronary artery disease, including obstructed LAD (Fig. 9). The large thrombus observed in the LV on admission was still present in TTE. After the heart team discussion, the patient was qualified for cardiac surgery. Finally,

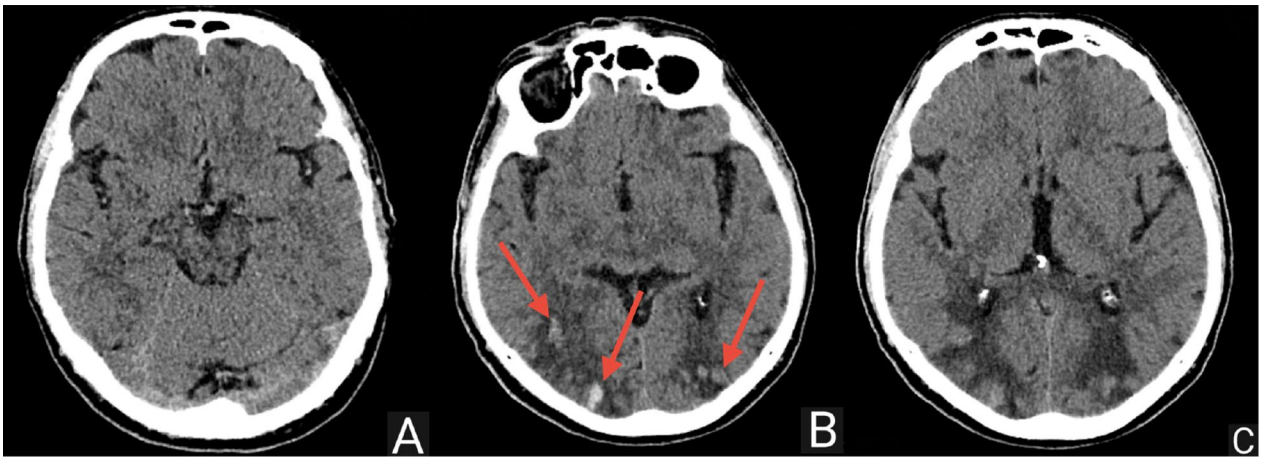

Fig. 6 Head computed tomography scans of patient 2 on admission—large bilateral hypodense areas in the occipital and parietal lobes (**A**). Third day of hospitalization; hemorrhagic foci marked with arrows (**B**). Tenth day after admission; partial resorption of hemorrhagic changes (**C**)

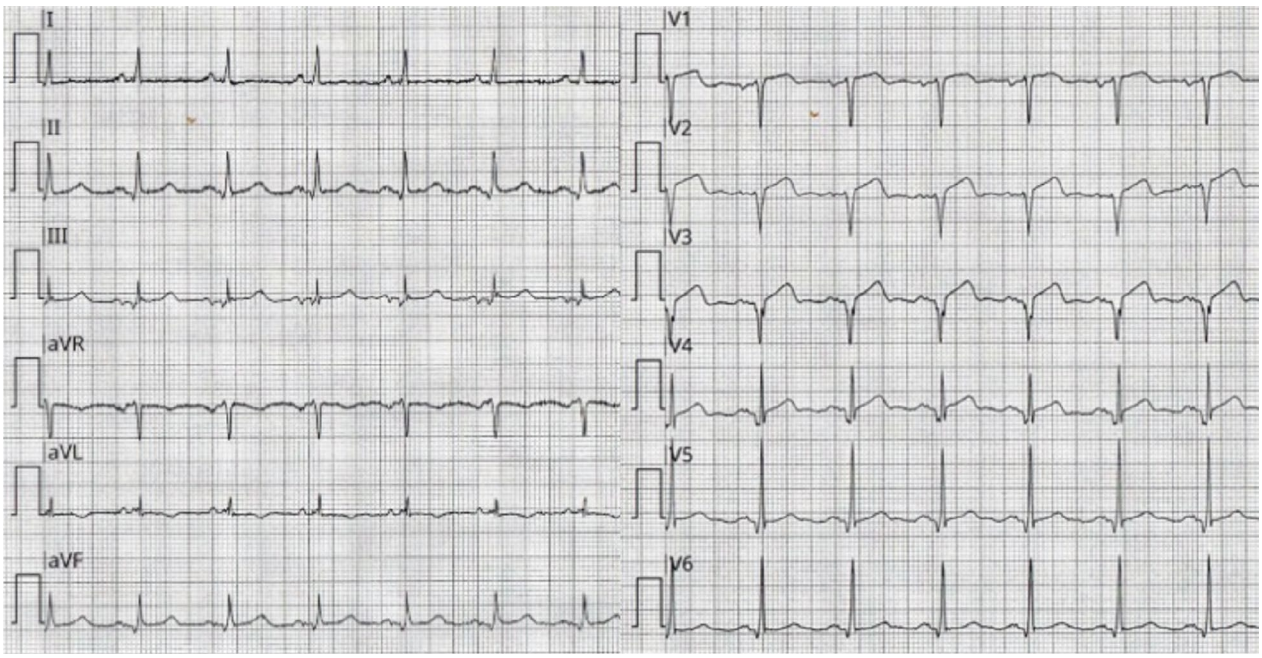

Fig. 7 The electrocardiogram of patient 2 on admission

after 5 weeks spent in our hospital, he was transferred to the cardiac surgery clinic. The left ventricular aneurysm resection with left ventricular reconstructive surgery and coronary artery bypass grafting (CABG) was performed (Fig. 10). When it comes to CABG, sequential coronary artery bypass was implanted in the marginal branch I and marginal branch II. The postoperative period was complicated by fluid accumulation in the left pleural cavity, which required pleural drainage. The patient spent 4 weeks in the cardiac surgery department, but then left for home in good condition, with an LVEF of 35%; on typical treatment, including a high dose of statin (atorvastatin 80 mg), BB, ACEI, SGLT2 inhibitor, and one antiplatelet drug (aspirin 75 mg); and without anticoagulation.

Discussion

General discussion

The COVID-19 pandemic had a massive impact on the healthcare system, leading, among other things, to an increase in the number of patients with acute coronary

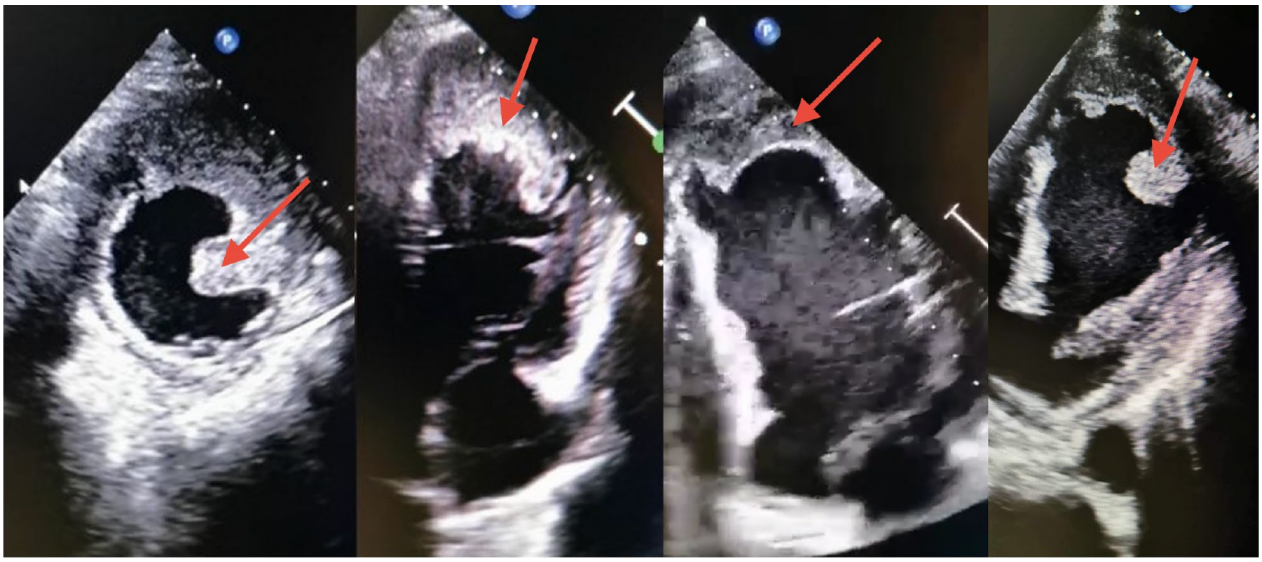

Fig. 8 Transthoracic echocardiography images of patient 2 on admission; the thrombus is marked with arrows

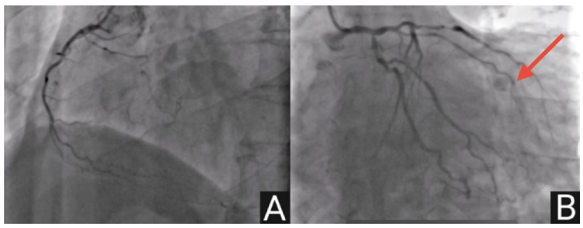

Fig. 9 Multiple lesions in right coronary artery, left anterior oblique 30° view (**A**), and left coronary artery with left anterior descending artery occlusion (marked with an arrow), right anterior oblique caudal view (**B**) of patient 2

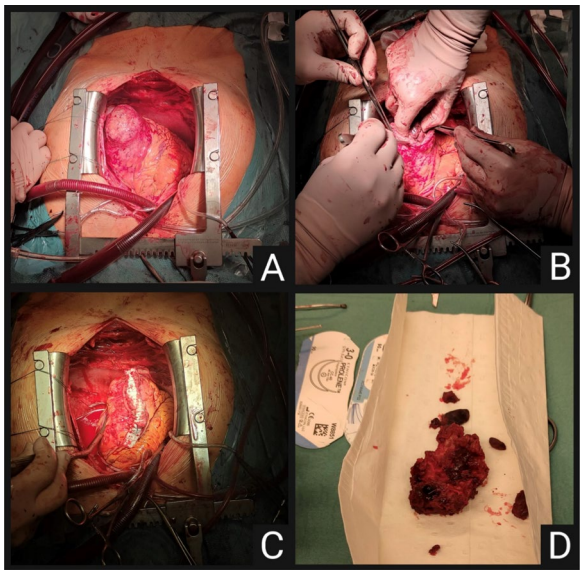

Fig. 10 Cardiac surgery of patient 2: the aneurysm of the apex (**A**); thrombus removal (**B**); GORE-TEX patch (**C**); and removed thrombus (**D**)

syndrome (ACS) who were admitted to the hospital late, beyond the time window for PPCI recommended by the guidelines [1–3]. This meant that we again often encountered complications of MI that occurred sporadically before the pandemic.

Late opening of the coronary artery in the course of STEMI occurs when the interval between symptom onset and reperfusion is more than 12 hours [11]. For years, studies have been conducted to answer the question of whether it is worthwhile and safe to open the infarct-related artery (IRA) outside the reperfusion time window [11]. There is a concept that the late opening of an IRA may improve the healing process. The proposed underlying positive effects include an influx of inflammatory cells into the infarct area, inhibition of the apoptosis among salvaged cardiomyocytes, an awakening of hibernating myocardium, acceleration of collagen synthesis, and preservation of non-myocyte cell components. Thanks to these processes, the scar is smaller and richer in collagen [12, 13], which reduces the risk of unfavorable remodeling and improves the geometry of the LV. Finally, it could have a positive effect on the LVEF and, thus, on the long-term prognosis by reducing the risk of dangerous arrhythmias. There are certainly many variables that affect the success of late reperfusion. These are primarily partial IRA occlusion and the presence of coronary collateral circulation [14].

The results of experimental studies are encouraging [15]. Also, the clinical studies performed in the second

and third decades of the current century show promising results in the invasively treated cohort. In a meta-analysis made by Bouisset *et al.*, coronary revascularization of latecomer STEMI patients was associated with better short- and long-term clinical outcomes [16]. In a recent, large, nationwide, prospective Korean registry, inverse steep differences in the use of PPCI and mortality rates were found between both early (< 12 hours) and late (12–48 hours) presenters [17]. In the DECOPI trial (2–15 days after MI), an improved LVEF was observed. However, there were no differences in clinical outcomes at 2 years [18]. In the study of Dong *et al.*, the late percutaneous interventions (PCI) strategy (defined as the time of PCI-mediated reperfusion > 24 hours) was associated with a reduced risk of major adverse cardiovascular events (MACE) in MI patients with preserved LVEF compared with conservative strategies [19].

Data presenting the advantage of the conservative strategy were mainly derived from two fundamental studies: TOAT and OAT. In the TOAT study, PPCI with stenting was performed 3 days–6 weeks after anterior STEMI. PPCI failed to improve LV performance, though increased exercise tolerance and improved quality of life were observed. In the OAT trial, PPCI was not shown to be of any benefit in patients who presented 3–28 days after acute MI with a persistent total occlusion of the infarct-related artery [20, 21]. However, the data supporting the conservative treatment of STEMI latecomers are based on evidence from the beginning of the century, and whether the progress in interventional techniques, device improvement, and currently used medications do not shift the paradigm can be discussed. Summarizing the research results, it seems that PPCI should be offered to all patients with STEMI presenting 12–48 hours from symptom onset. The European Society of Cardiology (ESC) advises that patients presenting at > 12 hours with signs of ongoing ischemia, heart failure, or electrical instability should undergo a PPCI strategy regardless of the time of presentation (class I recommendation). In patients presenting at 12–48 hours, routine PPCI should be considered in all patients (class IIa). For those presenting beyond 48 hours, the ESC advises that either angiography or a non-invasive test for the presence of residual myocardial ischemia or viability should be performed. Still, routine PPCI of an occluded IRA is not recommended (class III) [11, 13, 27].

Delay in invasive treatment of MI significantly increases the risk of complications, including mechanical and thromboembolic ones. Studies show that LVT forms in 5–15% of patients with STEMI [4, 5]. The highest risk of its formation is between the fourth and seventh day from the onset of symptoms. Factors predicting LVT formation are anterior MI involving the apical segments and delayed revascularization [4–6, 23–25]. The ventricular thrombus poses a risk of peripheral embolization. Embolic events occur frequently in the first 3–4 months after MI. In the pre-PPCI era, the frequency of thrombotic events was 22.3% of patients with LVT, and in the PPCI era, it was reduced to 5.5% [4].

Oral anticoagulation with VKA is advocated in the treatment of LVT, with the administration of LMVH (1 mg/kg orally) as a bridge therapy till the effective therapeutic range of VKA is achieved within 2 days [7]. Various societal guidelines recommend 3–6 months of anticoagulation with VKA (or longer—up to thrombus resolution) [4, 5, 7–9, 26, 27]. During this time, TTE should be used to confirm the thrombus resolution. For patients in the intensive care unit with large and mobile LVT and for whom surgical therapy is being evaluated, unfractionated heparin (UFH) with target activated partial thromboplastin time (aPTT) ~60 seconds (50–70 seconds) may be a reasonable alternative [7]. A follow-up TTE should be performed in 3 months to assess the potential recurrence of thrombus, especially if there has been no improvement in LVEF [4, 10].

Recently, the use of DOACs is gaining more attention. Although their use in the course of LVT is still off-label [7], most single-center observational studies and case series show promising results [9, 28–34]. In one of the studies, although not designed and powered for a direct comparison between DOACs versus VKA, a similar rate of total LVT regression, MACE, and bleeding complications with both anticoagulant strategies was observed [35]. In an observational study of 2328 consecutive patients undergoing coronary angiography ± percutaneous coronary intervention for acute MI, there was greater and earlier LV thrombus resolution in the DOAC group compared with patients treated with warfarin. Moreover, major bleeding events during the follow-up period were lower in the DOAC group, with no difference in rates of systemic thromboembolism [36]. Only one randomized trial performed so far showed that apixaban is noninferior to warfarin for the treatment of patients with LV thrombus after acute MI [37]. However, a multicenter retrospective cohort study evidenced that DOAC treatment was associated with a higher risk of stroke and systemic embolism than VKA therapy [8].

Because of the lack of supportive data, surgical removal of LVT should be limited to rare circumstances, such as the inability to tolerate anticoagulation therapy and cases when the patient needs cardiac surgery for other reasons [7].

The most serious complication of LVT is acute ischemic stroke (AIS). Diagnosis of stroke requires a CT scan to distinguish between hemorrhagic and ischemic subtypes to choose an appropriate therapy. The therapeutic time

window for thrombolysis or mechanical thrombectomy is limited to 4, 5, or 6 hours, respectively. Recommendations for the conservative treatment of cardioembolic stroke in the course of LVT are the same as for uncomplicated LVT. The patients should be treated with VKA anticoagulation therapy for at least 3 months (class I). If intolerant to VKA therapy because of non-hemorrhagic adverse events, anticoagulation with an LMWH, dabigatran, rivaroxaban, or apixaban is an alternative to VKA therapy (class IIb) [26].

The use of triple therapy comes at the cost of increased bleeding complications, which is further associated with an increase in mortality. However, it is recommended in patients with ACS. Hence, balancing the ischemic benefits against bleeding events is a common clinical dilemma. In patients with an indication for VKA in combination with antiplatelet therapy, the dose of VKA should be regulated so that the international normalized ratio (INR) is in the lower part of the recommended target range with a time in the therapeutic range > 70% [38, 39].

Randomized trials have demonstrated that DOACs are more effective than VKAs in preventing strokes in atrial fibrillation (AF) and carry a lower risk of intracranial hemorrhage [40]. There are differences in the mechanism of thrombus formation in the LV and the left atrium (LA). However, the results from AF trials, which focus on thrombus in the LA, may be applicable to thrombus in the LV. However, large, randomized trials are required to confirm this hypothesis.

There is still the question of when to start oral anticoagulant treatment in the case of MI, LVT, and AIS. Generally, in the case of an increased risk of intracranial bleeding, that is, large infarcts and dual antiplatelet therapy, it seems reasonable to introduce bridging treatment with LMWH before starting VKA.

Management of the patients

The two presented patients were referred to the hospital with the diagnosis of acute STEMI, while they both turned out to be STEMI latecomers. As the risk of pericardial inflammation after MI increases if STEMI is not treated invasively, the pericardial origin of the chest pain in the two patients was very likely. However, the noncardiac origin of the chest pain was also possible [41]. Since admission to the hospital, the first patient presented nausea, vomiting, and dizziness. These symptoms were not severe and were initially considered as symptoms related to MI. The presence of chest pain reduced our vigilance, and despite the presence of Q waves in precordial leads in ECG and accompanying neurological symptoms, the patient was treated as having acute STEMI with the need for PPCI. The diagnosis of transient ischemic attack was made only after PPCI when the neurological symptoms worsened. In accordance with previously applicable guidelines for stroke treatment, it was too late for intravenous alteplase or mechanical thrombectomy. The result of TTE and low repeated levels of troponins allowed for the diagnosis of the past MI with LVT and thromboembolic complications. Because the patient was saturated with antiplatelet drugs, we decided to treat him with LMWH (1 mg/kg orally).

Only after a control CT, where no ischemic changes nor transformation to hemorrhage were detected, did we start oral anticoagulant treatment. Due to the numerous data indicating the high effectiveness of DOACs in the course of LVT, the expected poor cooperation with the patient, and his reluctance to frequent blood draws for INR testing despite acting off-label, our decision was to implement DOAC. The LVT was no longer present in the 3-month follow-up. However, LVEF remained unchanged, which proves that coronary intervention on the occluded LAD performed on the distant day of the MI did not improve the left ventricular function. In summary, the first patient was not treated optimally. He was not initially diagnosed with TIA. Moreover, due to the presence of a post-infarction scar and an LVT, the PPCI was performed too hastily and possibly unnecessarily.

In the second case, due to the more pronounced neurological symptoms and our increased vigilance after the previous case, TTE was taken on admission. The visualization of the apical akinetic aneurysm with a huge LVT changed the management of the patient. The primary invasive strategy was not used, and pharmacological treatment of MI was applied. Coronary angiography was performed only after 2 weeks—the patient was then qualified for CABG, left ventricular constructive surgery, and LVT removal. As in the first case, also the second patient was outside the therapeutic window for stroke treatment. LMWH was started (1 mg/kg orally) and continued until the surgery. Due to the large area of cerebral infarction, we decided to use only one antiplatelet drug. It appeared to be the right decision because the second antiplatelet drug could further increase the severity of intracranial bleeding.

Based on these two cases, it seems reasonable that, if ST-segment elevation in the precordial leads is accompanied by Q waves, TTE on admission is an essential tool for better patient evaluation. Active thrombus search seems to be necessary in such patients, especially when the chest pain is atypical and neurological symptoms are present. The early diagnosis of LVT and AIS may change the treatment strategy and prevent neurological complications. Active thrombus search becomes very important due to the higher use

of mechanical circulator devices, which could be contraindicated in the case of LVT and can be catastrophic if used.

Conclusions

Patients who present late with an acute MI are a heterogeneous population, and the clinical decision regarding PPCI should not be the same for all. In the case of STEMI latecomers with LVT and AIS, especially when the area of cerebral injury is large, initial conservative treatment of MI with a postponed invasion strategy seems more reasonable. Moreover, a good solution seems to be a bridging therapy with LMWH in the first few days, when the risk of hemorrhagic transformation is highest. Cardiac surgery is sometimes necessary in the LVT treatment, especially when the LVT is huge and there is a need for CABG. Finally, randomized research is needed on DOACs as an alternative to VKA in the course of LVT.

Abbreviations

ACS	Acute coronary syndrome
AF	Atrial fibrillation
AIS	Acute ischemic stroke
Angio-CT	Computer tomography angiography
CABG	Coronary artery bypass grafting
CRP	C-reactive protein
CT	Computer tomography
DES	Drug-eluting stent
DOACs	Direct oral anticoagulants
IRA	Infarct-related artery
LAD	Left anterior descending artery
LMWH	Low-molecular-weight heparin
LVEF	Left ventricular ejection fraction
LVT	Left ventricular thrombus
MACE	Major adverse cardiovascular events
MI	Myocardial infarction
PPCI	Primary percutaneous interventions
RCA	Right coronary artery
STEMI	ST-elevation myocardial infarction
TTE	Transthoracic echocardiography
UFH	Unfractionated heparin
VKA	Vitamin K antagonist

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Author contributions

JT: writing—review and editing, methodology, and conceptualization. MW: writing—review and editing, visualization, and supervision. BW: writing—review and editing, methodology, and formal analysis. DGL: writing—review and editing, formal analysis, and validation. KW: resources, conceptualization, and investigation. DZ: resources, conceptualization, and investigation. PC: resources, conceptualization, and investigation. JZ: resources, conceptualization, and investigation. KM: writing—review and editing, visualization, and formal analysis. MZ: resources, supervision, and funding acquisition.

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None.

Availability of data and materials

The data will be shared upon request to the corresponding author.

Declarations

Ethics approval and consent to participate

Due to the retrospective nature of the research, the Institutional Review Board statement has been excluded. Informed consent was obtained from all subjects involved in the study.

Consent for publication

Written informed consents were obtained from the patients for publication of this case report and any accompanying images. Copies of the written consents are available for review by the Editor-in-Chief of this journal.

Competing interests

The authors have no competing interests to declare.

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