Surgical transmitral thrombectomy to prevent recurrent stroke in acute myocardial infarction: Case report

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Research Article

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Abstract

Introduction: Left ventricular (LV)-thrombi occur in up to 14% of patients with acute myocardial infarction (AMI) in the era of primary percutaneous coronary intervention. For these patients, anticoagulant therapy (AC) is recommended by AMI-guidelines. When, despite AC, LV-thrombi lead to embolism, surgical thrombectomy is an option, which is not mentioned or not recommended in AMI-guidelines.

Case Presentation: We report a 46-year old female patient with AMI. An 80% stenosis of the proximal left anterior descending coronary artery was treated by a drug-eluting stent. Thrombi within the akinetic LV-apex became mobile despite AC and dual antiplatelet therapy, and a cerebellar stroke occurred. By a transmirtal surgical approach with endoscopic assistance the thrombi were completely removed. Postoperative course and 12-months follow-up were uneventful.

Conclusions: LV-thrombi should be observed carefully regarding changes in morphology. Surgical thrombectomy of LV-thrombi is a rare treatment option to prevent imminent embolism. Benefits versus risks of surgical removal of LV-thrombi need to be carefully weighted.

Introduction

Left ventricular (LV) thrombus formation is a complication of cardiac diseases with reduced systolic function like acute myocardial infarction (AMI), Takotsubo syndrome (TTS), dilated cardiomyopathy, myocarditis and other non-ischemic cardiomyopathies. LV thrombi may occur in up to 14% of patients with AMI in the era of primary percutaneous coronary intervention (PCI), in up to 5% with TTS, and are sporadically reported in patients with myocarditis, sarcoidosis or other cardiomyopathies [1][2][3][4][5].

Anticoagulant therapy (AC) with vitamin K antagonists for patients with LV thrombi to prevent stroke or peripheral embolism (S/E) is recommended by the European and American AMI-guidelines [6][7]. Thrombolytic therapy, on the contrary, may increase the risk for embolization of LV thrombi [8]. Despite therapeutic AC, however, mural LV thrombi may become mobile and lead to S/E [3][8][9][10]. Under these circumstances, surgical thrombectomy by various techniques has been reported as a therapeutic alternative since 1983.[11][12] However, benefit and risks of thrombectomy have only been described in case reports and case series, a randomized trial has not yet been performed [4][5][13][14][15][16]. Surgical thrombectomy of mobile LV thrombi is either not mentioned or not recommended in review articles and guidelines, since procedural risks are considered higher than the benefit [1][6][7][17]. Nevertheless, we referred a patient with AMI and intraventricular thrombi for surgical thrombectomy since she developed a mobile LV thrombus and a minor cerebellar stroke despite therapeutic AC and dual antiplatelet therapy.

Case Presentation

A 46-year old female was admitted in November 2020 because of increasing oppressive chest pain which started 12 hours before admission (Figure 1). Chest pain had resolved spontaneously on admission and did not recur. She had a history of migraine since 30 years, arterial hypertension since 8 years and was a
smoker with 24 pack years. Her menopause was just starting. Over the last three months the patient had experienced a high level of stress at work. Physical examination showed no abnormalities, her body-mass-index was 17 and blood pressure 130/100 mmHg.

Blood tests (Table 1) and the electrocardiogram (ECG) at hospital admission were indicative for an ST-elevation AMI, therefore, acetylsalicylic acid and prasugrel were started. During the following 12 hours, the ST-segment elevations disappeared and negative T-waves developed. Because troponin levels were decreasing and the patient was free of pain acute coronary angiography was not performed. Bedside echocardiography on day 1 showed apical ballooning and hyperkinesia of the basal parts of the LV. Bisoprolol was initiated because of sinus tachycardia and atorvastatin because of hypercholesterolemia. On day 3, coronary angiography showed an 80% stenosis of the proximal left anterior descending coronary artery (LAD) extending far beyond the LV apex, which was treated by PCI with a drug-eluting stent (Figures 2A and B). The remaining coronary arteries were without stenosis. Ventriculography in 30° RAO projection showed a large akinetic area of the entire LV apex, a LV ejection fraction (EF) of 37% and a questionable attenuation of contrast material in the apical region (Figure 3A). On day 5, echocardiography showed a LV EF of 41%, and one large sessile and two small mobile thrombi in the akinetic LV apex. Enoxaparin, followed by phenprocoumon, was added and the antiplatelet therapy was changed from prasugrel to clopidogrel.

In the night from day 9 to day 10, the patient complained about sudden headache, different in pain character from the known migraine. Cerebral magnetic resonance imaging (MRI) showed a small recent ischaemia in the right cerebellum. Except for headache, which resolved after paracetamol, she did not show any neurologic abnormalities. On day 10, the INR was 4.4 and enoxaparin was stopped. Echocardiography showed an improved left ventricular function. The large LV thrombus, however, was now highly mobile and protruding. Since AMI was complicated by a presumed embolic stroke and a further escalation of AC therapy was considered futile, the patient was referred for surgical thrombectomy. Preoperative cardiac computed tomography (Figure 3B) confirmed the presence of LV thrombi.

At surgery, cardiopulmonary bypass was established from the superior and inferior vena cava to the ascending aorta. The aorta was cross-clamped and the left atrium was opened via the interatrial groove. The thrombus could be viewed through the mitral valve. Endoscopy facilitated visualization of the intracardiac structures, and one large (measuring 2 cm) and three smaller thrombi were removed from the LV apex (Figure 4A and B). The times for aortic cross-clamping, cardiopulmonary bypass and the whole surgical procedure were 36, 72, and 210 minutes, respectively. Histological examination confirmed the diagnosis of organizing thrombi. Postoperative echocardiography showed an improved systolic function and apical hypokinesia without thrombi. Since no residual thrombus was visible and systolic function had improved, no AC therapy was prescribed. Dual antiplatelet therapy with acetylsalicylic acid and clopidogrel was continued postoperatively. Due to the recent stroke and low body weight of 53 kg prasugrel or ticagrelor were considered contraindicated [18]. She was discharged on the 8th postoperative day with acetylsalicylic acid, clopidogrel for 1 year, ramipril, bisoprolol, atorvastatin and pantoprazole.
Twelve months later, she was in a stable clinical condition, the ECG had normalized and echocardiography showed only inferoapical hypokinesia. Cardiac MRI showed a LV EF of 56%, inferoapically a slight hypokinesia and a diffuse fibrosis in the corresponding area but no subendocardial or transmural scar. Test results for thrombophilia, comprising activated protein C resistance, free protein S and homocysteine; activity of antithrombin III, protein C and S, disclosed no abnormalities.

Conclusions

We report a case of a young lady with acute anterior wall MI and a recanalized LAD, either spontaneously or due to antiplatelet therapy, with Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 and a high-grade stenosis. The reversible wall motion abnormality can be explained by stunned myocardium in the supply area of the large LAD, extending to the inferior wall. This assumption is supported by cardiac MRI during follow-up which showed a mild hypokinesia and a diffuse fibrosis in the inferoapical area without scar formation. TTS might be a differential diagnosis because of emotional stress preceding the event, akinesia of the entire LV apex and an only slight elevation of cardiac markers compared to the extent of wall motion abnormality. Emotional stress, however, may also induce acute coronary plaque instability and lead to AMI [19][20].

LV thrombi in patients with rapidly recovering LV function differ from thrombi in transmural myocardial infarction where the wall motion abnormalities usually persist. The rapid improvement of contractility may promote the transformation of initially mural and adherent thrombi to mobile and protruding thrombi which are prone to embolize despite therapeutic AC [8][9][13]. Surprisingly, short-term echocardiographic follow-up investigations of LV thrombi during AC therapy to detect changes in mobility and morphology are not recommended in current guidelines or review articles [1][6][7][17].

Headache in ischaemic stroke is reported by 15-27% of acute stroke patients [21][22]. Headache at stroke onset is associated with a history of migraine, cardioembolic aetiology, vertebrobasilar stroke, especially with cerebellar stroke [21][22]. All these factors were present in our patient.

Left ventriculotomy is the most frequently used approach for surgical thrombectomy [11][12][13]. The usual concern with surgical removal of thrombi is the risk of ventriculotomy in a friable myocardium. Ventriculotomy in patients with AMI, TTS or myocarditis may increase the risk for cardiogenic shock, arrhythmia, myocardial necrosis and haemorrhage. Therefore, other approaches for LV thrombectomy with endoscopic assistance were developed like transmitial or trans-aortic removal [4][13][14][16]. These approaches allow clear visualization of the thrombus and the LV cavity and eliminate the need to incise the LV myocardium.

This case shows that patients with LV thrombi have to be carefully monitored regarding a change in thrombus morphology. When there is a high risk of imminent or recurrent systemic embolism from a highly mobile LV thrombus, surgical removal by transmitial thrombectomy with endoscopic assistance may be a safe and successful treatment option.
Declarations

I. Funding – no funding

II. Conflicts of interest – no conflicts of interest (all authors)

III. Ethics approval – not applicable, retrospective, case report

IV. Consent to participate – not applicable, retrospective, case report

V. Consent for publication – was given by the patient

VI. Availability of data and material – are available in the records of Klinik Landstrasse and Allgemeines Krankenhaus Wien

VII. Code availability – not applicable

VIII. Authors contributions

Matthias Hasun 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published; coronary angiography, magnetic resonance imaging.

Wilfried Wisser 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published; cardiac surgery.

Maria Heger 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published; echocardiography.

Lisa Sow 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published; electrocardiogram, clinical care of the patient.

Nina Schönbrunn 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published; electrocardiogram, clinical care of the patient.

Josef Finsterer 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; 3) final approval of the version to be published, neurological investigation.

Claudia Stöllberger 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content;
Franz Weidinger 1) substantial contributions to conception and design, acquisition of data, analysis and interpretation of data; 2) drafting the article and revising it critically for important intellectual content; and 3) final approval of the version to be published.

References


Table

Table 1 Results of blood tests of the patient with acute coronary syndrome-associated left ventricular thrombus

<table>
<thead>
<tr>
<th>Parameter (Normal range)</th>
<th>Admission</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 10</th>
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<td>Leucocytes (3.9-10.2 G./L)</td>
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<td>NM</td>
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</tbody>
</table>

NM= not measured

Figures
Figure 1
Timeline of the course of the disease.

Figure 2
Coronary angiography three days after onset of symptoms shows an 80 % stenosis of the left anterior descending artery (A) which was treated by a drug-eluting stent (Xience Sierra 2.75/15 mm) (B). The left anterior descending artery wraps around the left ventricular apex to the inferior wall of the left ventricle (LCA= left coronary artery). The right coronary artery (RCA) shows no stenosis.
Figure 3

Ventriculography in 30° right anterior oblique projection, 3 days after onset of symptoms, shows a large akinetic area of the left ventricular apex and a questionable attenuation (arrow) of contrast material in the apical region (A). Preoperative computed tomography, 10 days after onset of symptoms, confirms the thrombi in the left ventricle (B).

Figure 4
Intraoperative image of the ventricular thrombus (A) entrapped in the apex (APM = anterior papillary muscle, PPM = posterior papillary muscle). One large and three smaller thrombi were removed (B).

Supplementary Files

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- CareChecklist.jpg