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CASE REPORT

Outcome of Aortocoronary Bypass in a Patient with a History of Hemophilia

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Abstract

Hemophilia is a congenital disorder of the blood coagulation system linked to the X chromosome and manifested by a deficiency of blood coagulation factor VIII (hemophilia type A) or factor IX (hemophilia type B). We present our clinical case involving the combination of myocardial infarction (MI) and hemophilia type A. A 42-year-old man with a known history of hemophilia A was admitted to the emergency department of an external center with complaints of pressing chest pain radiating to the left arm. He was treated with 300 mg of ecopyrine and 300 mg of Plavix, and a significant increase in troponin level was detected. The patient was then transferred to the emergency department of our hospital.

In the emergency department, he was evaluated by cardiology. The control troponin level was 72, and the patient was admitted to the coronary intensive care unit with a preliminary diagnosis of non-ST elevation myocardial infarction (NSTEMI). Coronary angiography was planned. Internal medicine and hematology clinics were consulted to adjust anticoagulant therapy for the procedure, and dual antiplatelet therapy (ecopyrine + Plavix) was administered.

After coronary angiography, the patient was evaluated by a joint council of cardiovascular surgery and cardiology, and coronary bypass surgery was recommended. He was transferred to the cardiovascular surgery unit for preoperative preparation. During this period, he was also evaluated by the hematology clinic due to his hemophilia A history. Based on the clinic's recommendation, specific factor VIII (FVIII) therapy was administered pre- and postoperatively, along with daily aPTT monitoring.

No complications occurred during surgery. Cardiopulmonary bypass was initiated once the activated clotting time (ACT) exceeded 450. No bleeding complications were observed in the postoperative period. The patient was successfully discharged.

Keywords: Bypass; coronary angiography; FVIII, hemophilia A.

Hemophilia is a congenital disease of the blood coagulation system, linked to the X chromosome, and develops with a deficiency of blood clotting factor VIII (hemophilia type A) or factor IX (hemophilia type B). The number of people with hemophilia in the world is about 400,000 people [1]. In Russia, there are about 7,500 people with hemophilia [2]. It was previously believed that the presence of hemophilia protects against the occurrence of coronary heart disease [3]. However, subsequently published studies have shown that with improved treatment of hemophilia, the life expectancy of these patients increases, and the number of risk factors and concomitant diseases, including coronary artery disease, also increases [4]. Indeed, in a study conducted in Canada on 294 hemophilia patients, arterial hypertension was found in 30.3%, diabetes in 10.5%, and dyslipidaemia in 22.4%.

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There were 14 cases of ischaemic heart disease; 3 patients underwent coronary artery bypass surgery, and 9 patients underwent percutaneous coronary intervention. The combination of myocardial infarction (MI) and hemophilia type A is very rare. As of 2006, only 36 cases of MI due to hemophilia were described in the world literature [5]. We present our own clinical case involving the combination of myocardial infarction and hemophilia type A.

Case Report

A 42-year-old male patient, diagnosed with hemophilia A about 3 years ago and who had not received any treatment for hemophilia A, was admitted to the emergency department of an external centre with complaints of pressing chest pain radiating to the left arm. A significant increase in troponin value was observed, and the patient was treated with 300 mg ecopirin and 300 mg plavix. The patient was then transferred to the emergency department of our hospital.

In the emergency department, he was evaluated by cardiology. Echocardiography showed an ejection fraction of 65%, and rheumatic mitral valve disease was suspected. The control troponin level was 72, and the patient was admitted to the coronary intensive care unit with a prediagnosis of non-ST myocardial infarction. Coronary angiography was planned (Figs. 1–3). The patient was consulted to the internal medicine and haematology clinics for adjustment of anticoagulant treatment for angiography, and dual antiplatelet therapy (ecopirin + plavix) was administered due to acute coronary syndrome.

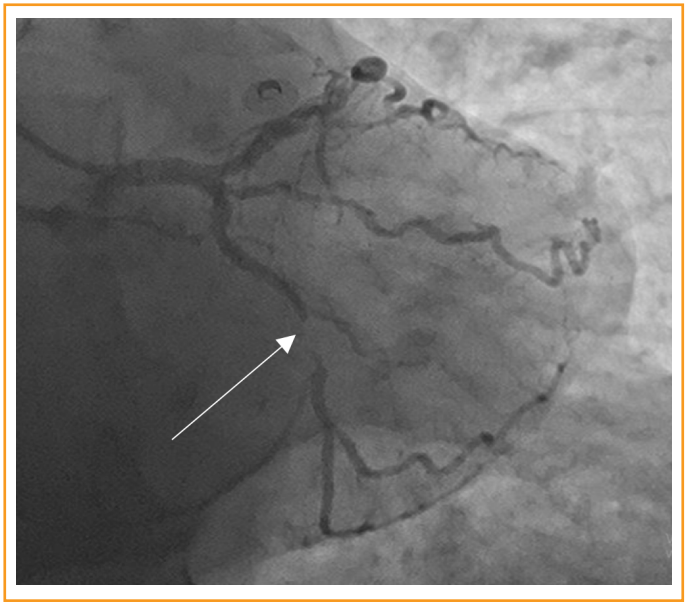

Figure 1. Coronary angiography image. CX (circumflex artery) middle segment, total occluded.

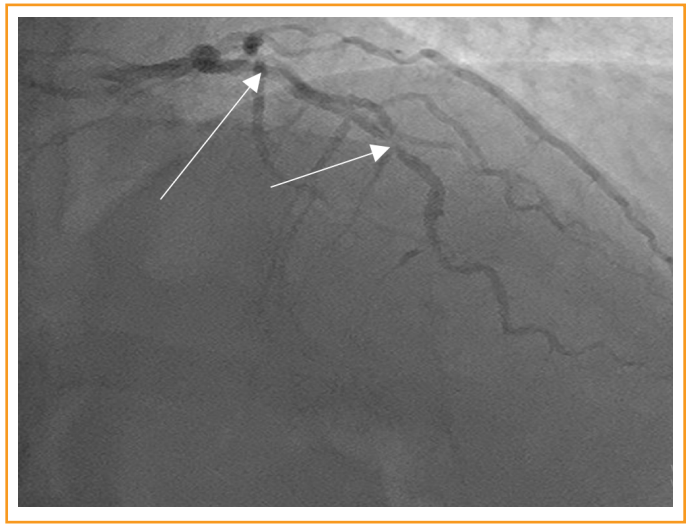

Figure 2. Coronary angiography image. LAD (left descending artery) proximal and middle segments, severe stenosis is present.

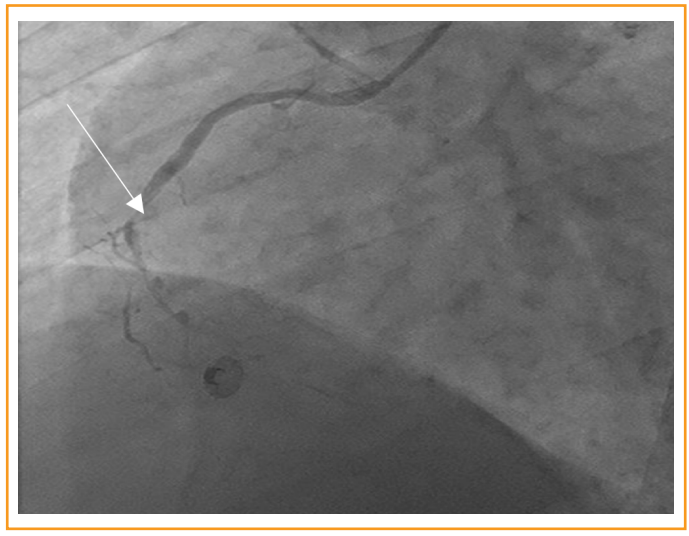

Figure 3. Coronary angiography image. RCA (right coronary artery) middle segment, subtotally occluded.

The patient underwent coronary angiography performed by cardiology. Coronary main vessels were visualised, and a coronary stent procedure was attempted. However, since the stent procedure could not be performed, the patient was evaluated by the cardiology and cardiovascular surgery (CVS) council for aortocoronary bypass surgery. Coronary bypass surgery was recommended, and the patient was transferred to the cardiovascular surgery ward for preoperative preparation.

The patient was evaluated by the haematology clinic due to his history of hemophilia A and the surgical indication. HEMOFIL-M treatment was initiated with

the recommendation of the hemophilia clinic and administered as 4,500 units on the day of surgery and 2,000 units daily for the first 3 postoperative days. The patient underwent elective surgery 6 days after coronary angiography, following the completion of preoperative preparations. During the operation, heparinisation was applied, and cardiopulmonary bypass was performed while maintaining the ACT value >450. Saphenous vein grafts were routinely used for the coronary arteries. The procedure was completed without complications. At the final stage, heparin was neutralised with protamine.

After the operation, the patient was transferred to the intensive care unit and extubated after 4 hours. In the postoperative period, no significant complaints from the drains were noted. The patient's blood tests and vital signs showed no serious abnormalities, and there was no decrease in hemogram values. Daily activated partial thromboplastin time (aPTT) and factor VIII monitoring were performed upon the recommendation of the haematology clinic. The patient was transferred to the ward after 3 days of intensive care monitoring and was successfully discharged a few days later.

Discussion

There are very few published studies on the combination of hemophilia and MI. Girolami et al. [5] analyzed 36 cases of MI in patients with hemophilia. The mean age of patients with hemophilia was 44 years. Twenty-two cases of MI occurred during or immediately after infusion of factor VIII concentrate. The authors emphasized that the high incidence of MI occurring after infusion of factor VIII or prothrombin complex concentrate suggests that a thorough clinical evaluation of each patient is necessary to develop an adequate therapeutic approach. Replacement therapy with blood clotting factors was performed in all cases.

Mannucci et al. [6] presented recommendations for the management of hemophilia patients with acute coronary syndrome. Radial access is recommended because it is safer in terms of bleeding. It is advised to administer 40 U/kg factor VIII bolus during diagnostic coronary angiography and stenting, 20 U/kg bolus 12 hours later, and to maintain the blood level at 80 U/dL during heparin administration. When dual antiplatelet therapy is used, it is recommended to administer factor VIII at a dose of 50 U/kg every other day. If treatment with two antiplatelet agents lasts up to 1 month, it is recommended to maintain the blood concentration of factor VIII at 30 U/dL. The use of bare metal stents is preferred.

Regarding the presented clinical observation, this is the first case of a combination of MI and hemophilia among more than 7,000 cases of MI in our clinic over 10 years. The lack of practical experience in the treatment of MI with such a rare and dangerous pathology as hemophilia did not allow us to decide to perform coronary angiography. In addition, invasive intervention, as mentioned above, should be accompanied by the administration of factor VIII, which is not available in routine emergency cardiology practice. An additional obstacle in the treatment of MI in a patient with hemophilia is the need for continuous monitoring of the level of factor VIII in the blood when using anticoagulants and antiplatelet agents, which is also not a routine practice.

The mild course of myocardial infarction, without ST segment elevation on ECG, without hemodynamic disturbances, and without any complications, led us to opt for conservative treatment during the patient's first MI. However, recurrent MI developed more aggressively due to occlusion of the trunk of the left coronary artery (the electrocardiographic sign of this localization of thrombosis is ST segment elevation in aVR and ST segment depression in leads V3–V6, which was confirmed by autopsy), pulmonary edema, and cardiogenic shock, leading to death.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Conflict of Interest: The authors declare that there is no conflict of interest.

Informed Consent: The patient agrees to the use of information about himself/herself in a journal article or mature presentation, or for use in a thesis presentation.

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