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Case Report

Post-coronary Artery Bypass Grafting Heparin-induced Thrombocytopenia in a Patient with Kaposi's Sarcoma: A Complex Hematological Challenge

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Abstract

Coronary artery disease is increasingly prevalent in today's world. Coronary artery bypass grafting (CABG) plays a significant role in the treatment of this disease. Systemic comorbidities are often found in coronary artery patients, especially requiring careful management post-CABG. Among these comorbidities, one important group that affects treatment management is oncological diseases. In addition to known systemic comorbidities, different clinical conditions that can arise later, such as heparin-induced thrombocytopenia (HIT), can also be observed during the surgical hospitalization process. Keeping in mind the rarely encountered and potentially fatal course of HIT in cardiovascular surgery is crucial for early diagnosis and treatment management. In this case, we aim to present our diagnosis and treatment approach to HIT following CABG in a 69-year-old female patient followed with a diagnosis of Kaposi's sarcoma.

Keywords: Coronary artery bypass grafting; coronary artery disease; heparin-induced thrombocytopenia; Kaposi's sarcoma.

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Kaposi Sarkomlu Bir Hastada CABG Sonrası Heparine Bağlı Trombositopeni: Karmaşık Bir Hematolojik Sorun

Özet

Koroner arter hastalığı (KAH) günümüzde giderek artan sıklıkla görülmektedir. Koroner arter bypass greftleme operasyonları (CABG), bu hastalıkların tedavisinde önemli bir yer teşkil etmektedir. Koroner arter hastalarında, sistemik ek hastalıklar da sıklıkla bulunmaktadır. Bu husus, özellikle CABG sonrasında hasta yönetimi açısından büyük dikkat gerektirmektedir. Bu ek hastalıklar arasında tedavi yönetimini etkileyen önemli gruplardan birisi de onkolojik hastalıklardır. Bilinen sistemik ek hastalıklar yanında, heparin ilişkili trombositopeni (HİT) gibi sonradan ortaya çıkabilen farklı klinik durumlar da cerrahi yatış sürecinde görülebilmektedir. Nadir karşılaşılan ve mortal seyredebilen HİT tablosunun kalp damar cerrahisinde her daim akılda bulundurulması, erken tanı ve tedavi yönetiminde hayati rol oynamaktadır. Bu olguda, Kaposi sarkomu tanısıyla takip edilen 69 yaşındaki kadın hastada, CABG sonrası gelişen HİT tablosundaki tanı ve tedavi yaklaşımımızı sunmayı amaçladık.

Anahtar sözcükler: Koroner arter baypas ameliyatı; koroner arter hastalığı; heparin ilişkili trombositopeni; Kaposi sarkomu.

Introduction

Kaposi's sarcoma (KS) is a malignant vascular tumor first described by M. Kaposi in 1872.[1] There are types of this rare neoplasm that are associated with acquired immunodeficiency syndrome (AIDS). Classic KS (CKS), not associated with HIV infection, tends to have a better prognosis.[2] It

is often observed in Eastern Europe and Mediterranean countries, with rare mucosal involvement.[3]

Heparin-induced thrombocytopenia (HIT) is also rare but can have a fatal course. After the development of an immune reaction against platelet factor 4, it can lead to a significant decrease in platelet count in the systemic circulation. Simultaneously, free thrombi complexes can be observed.[4] There are two clinical subtypes, with type 1 having a better prognosis and Type 2 having an approximately 30% mortality rate.[5]

Considering that the relationship between coronary artery patients and heparin treatment can begin before cardiopulmonary bypass (CPB), the fact that HIT can occur in cardiac surgery patients before, during, and after surgery should be kept in mind. In our case, we aim to present our successful treatment approach to HIT following coronary artery bypass grafting (CABG) in a 69-year-old female patient followed with a diagnosis of KS.

Case Report

A 69-year-old female patient presented to our clinic with complaints of chest pain. Her functional capacity was determined as New York Heart Association (NYHA) class 3. Arterial blood pressure was 145/90 mmHg, heart rate was 76 beats/min, respiratory rate was 15 breaths/min, and she was in sinus rhythm. The patient had a known history of hypertension, insulin-dependent diabetes mellitus, and a diagnosis of KS. There were noticeable red-purple nodular lesions on her hands and lower extremities (Fig. 1). Chest X-ray showed a normal cardiothoracic ratio and clear pleural sinuses. Transthoracic echocardiography revealed a mild reduction in left ventricular contractility, segmental motion defect, ejection fraction of 50%, and pulmonary artery pressure of 20 mmHg.

Coronary angiography identified an 80% stenosis in the left main coronary artery and widespread coronary artery disease, with no stenosis in the carotid arteries. Pre-operative hemogram revealed a hematocrit of 38% and a platelet count of 214,000. The patient, prepared for CABG, was referred to the oncology clinic for consultation regarding KS. With a history of radiotherapy to the extremities, the patient was deemed suitable for the operation. Informed consent was obtained.

Median sternotomy was used for the operation. The left internal mammalian artery and saphenous vein were prepared as grafts. After appropriate heparinization (300 U/kg), standard aortic arterial and bicaval two-stage venous cannulations were performed. The activated clotting time (ACT) reached 480, and CPB was initiated. Antegrade and retrograde blood cardioplegia were administered after aortic cross-clamping to achieve cardiac arrest. Normothermia was maintained, and cardiac protection was ensured by repeating cardioplegia every 20 min. Bypass grafting operations on the right coronary artery, circumflex artery, diagonal artery, and left anterior descending coronary artery were successfully performed. The patient was weaned off CPB in spontaneous sinus rhythm. Additional heparin (5000 units) was administered based on ACT monitoring during CPB. The aortic cross-clamp time was 79 min, and the total perfusion time was 118 min. The patient was extubated in the 5th h of intensive care admission. Drains were removed on the 2nd day postoperatively, and the patient was transferred to the ward. On the 6th day, the patient was discharged with stable hemodynamics and normal hematologic values.

After discharge, during the 1st-week follow-up, high blood sugar and arrhythmia were detected in the patient. She was placed under intensive care monitoring, and blood sugar regulation was achieved. In the follow-up transthoracic echocardiogram, no intracardiac thrombus was observed, but atrial fibrillation was identified. The patient was started on amiodarone and low molecular weight heparin treatment. On the 2nd day of intensive care monitoring, sinus rhythm was achieved. However, coldness and discoloration were detected in the distal part of the left upper extremity, and the radial pulse could not be palpated during the examination. Arterial Doppler ultrasound revealed monophasic flow in the left radial artery and triphasic flow in the left ulnar artery. Low molecular weight heparin was discontinued, and intravenous heparin infusion was initiated. On the 3rd day of intensive care monitoring, the patient reported shortness of breath. A repeat transthoracic echocardiogram revealed a pulmonary artery pressure of 50 mmHg. The control lung X-ray and tomography, which were evaluated as normal, showed a more than 50% decrease in platelet count in the control hemogram compared to the previous day (from 144,000 to 52,000). Six hours later, when the platelet count was rechecked and found to be 38,000, a diagnosis of HIT was considered due to the clinical presentation of arterial thrombosis and pulmonary embolism. Platelet replacement was not performed. Heparin was discontinued, and consultation with the hematology clinic was sought. Fondaparinux, a factor Xa inhibitor, was initiated for the patient with a positive thrombocyte aggregation test. On the 2nd day of 2.5 mg fondaparinux treatment, the platelet count was assessed as 23,000, and the treatment dose was increased to 5 mg. On the 3rd day, the control platelet count was 25,000. Discoloration and coldness

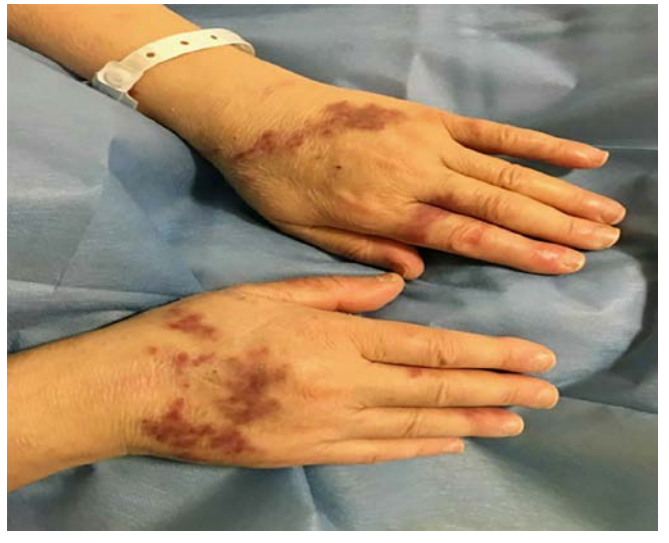

Figure 1. Red-purple-colored nodular lesions in Kaposi sarcoma.

Table 1. The treatment and PLT counts in HIT

Day (n)	1.	2.	3.	4.	5.	6.	7.	8.	9.
Treatment (mg)	Fx 2.5	Fx 5	Fx 5	Fx 5	Fx 5	Fx 5+W 2.5	Fx 5+W 2.5	Fx 5+W 5	W 5
PLT (*1000)	48	23	25	39	48	71	73	91	108

PLT: Platelets; HIT: Heparin-induced thrombocytopenia; Fx: Fondaparinux; W: Warfarin.

in the left upper extremity decreased, but the radial pulse still could not be palpated. On the 4th day of fondaparinux treatment, the platelet count increased to 39,000. A control transthoracic echocardiogram revealed a decrease in mean pulmonary artery pressure to 30 mmHg, and the patient, whose shortness of breath had resolved, was placed under ward follow-up. On the 5th day of treatment, the platelet count was 48,000, and the radial artery pulse could be palpated. On the 6th day, the platelet count was 71,000, and oral warfarin was added to the treatment. On the 9th day, the platelet count was 108,000, and the INR value was 2.1. With stable hemodynamics, the patient was discharged with the discontinuation of fondaparinux and the initiation of oral warfarin treatment. During the 1-week follow-up, the patient's functional capacity was determined to be NYHA Class 1. The INR value was 2.3, and the platelet count was 129,000. Consultation with the hematology clinic resulted in the planning of 1 year of oral warfarin treatment, with monthly follow-up appointments. Daily platelet values after fondaparinux treatment are shown in Table 1.

Discussion

The classic type of KS is not associated with AIDS. This well-known subtype, characterized by a generally benign course, typically presents itself with nodular lesions of a purple color on the extremities. Commonly observed in Eastern Europe and Mediterranean countries, CKS is mostly confined to the skin, with rare instances of visceral involvement.[6] Our patient, who tested negative for HIV, had typical reddish-purple nodular lesions on the hands and feet. A histopathological examination confirmed the diagnosis of CKS, and the patient had undergone radiation therapy for the lesions on the hands and feet. In addition, there was no evidence of visceral involvement in outpatient.

Heart surgery treatment regimens often involve frequent use of heparin; however, HIT is a rare occurrence. It can be observed postoperatively at a rate of 1.9%. There are two different clinical types: Type 1, which is non-immune and often leads to a normalization of platelet counts after discontinuation of heparin, and Type 2, which is immune-related and more dangerous. In Type 2, serious complications such as bleeding in 53%, thromboembolism in 44%, and death in 33% of cases can occur.[7] Our patient experienced Type 2 HIT after surgery.

In addition to low platelet levels, bleeding, and thromboembolic symptoms, play a crucial role in the diagnosis of HIT. Clinical suspicion is essential, and three specific tests can be used under laboratory conditions. These include serotonin release assay, ELISA, and platelet aggregation tests. Platelet aggregation testing is more accessible in Turkey, with a reported specificity of over 80% and sensitivity of over 90%.[8] HIT 4T's score is a useful guide to predict heparin-induced thrombocytopenia through symptoms and clinical features. It estimates HIT possibility. Our patient's 4T score was 4 (total possible score is 8.) and HIT probability was 8–29% according to 4T score. We acted rapidly and stopped heparin treatment before necrotizing lesions occur thus the 4T score is low.

In treatment, discontinuation of heparin is recommended as a priority. In addition, platelet replacement is not recommended as it may exacerbate thromboembolic events.[9] Treatment options include non-thrombin inhibitors such as danaparoid, thrombin inhibitors like hirudin and argatroban, and factor Xa inhibitors like fondaparinux. In our case, heparin was discontinued as soon as HIT was suspected, and platelet replacement was not performed to avoid exacerbating thromboembolic events, as recommended in the literature. The diagnosis of HIT was confirmed by thrombocytopenia, thromboembolic manifestations, and a positive platelet aggregation test. Among the treatment options, fondaparinux, which is rapidly available, was preferred, providing effective treatment.

In CKS, cases involving lymphedema, chronic venous insufficiency, and vascular thrombosis affecting the extremities have been reported.[10] However, occurrences of post-CABG thromboembolic events and HIT in CKS, as in our case, have not been documented in the literature.

Conclusion

Systemic comorbidities commonly encountered in cardiac surgery patients are usually known in the pre-operative period, and measures are taken for post-operative management accordingly. However, for rare but potentially fatal conditions that can develop later, such as HIT, clinical suspicion and awareness should always be at the forefront. Hence, the coexistence of CKS and HIT after CABG has been emphasized.

Disclosures

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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