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CASE REPORT

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Management of the type-1 heparin induced thrombocytopenia (HIT) in the course of acute inferior myocardial infarction with high thrombus burden

Yüksek trombüs yükü olan akut inferior miyokart enfarktüsü seyrinde gelişen tip-1 heparin ilişkili trombositopeni vakasının yönetimi

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ABSTRACT

Heparin-induced thrombocytopenia (HIT) is a difficult clinical presentation to deal with and we know little about the management of these patients. Stopping of heparin is essential in treatment but some patients may need anticoagulation in the follow-up; however, we do not have enough data about the patients with a need of anticoagulant usage. We want to present a myocardial infarction patient who developed type I HIT after exposure to unfractionated heparin with a need of anticoagulation at the same time.

Keywords: myocardial infarction, heparin administration, heparin induced thrombocytopenia

ÖZ

Heparin ilişkili trombositopeni (HİT) başa çıkılması zor bir klinik tablodur ve bu hastaların yönetimiyle ilgili bilgimiz sınırlıdır. Heparin'in kesilmesi tedavinin esasıdır ancak bu hastaların bir kısmında antikoagülan kullanım ihtiyacı doğabilir ve takibinde antikoagülan ihtiyacı olan hastalarla ilgili net bir veri bulunmamaktadır. Bu yazımızda anfraksiyone heparin sonrasında tip I HİT gelişen ve aynı zamanda antikoagülan kullanım ihtiyacı bulunan miyokart enfarktüsü hastamızın sunumunu yapmayı amaçladık.

Anahtar sözcükler: miyokart enfarktüsü, heparin kullanımı, heparin ilişkili trombositopeni

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INTRODUCTION

Heparin-induced thrombocytopenia (HIT) is a challenging clinical disorder, but we have limited data about the management of these patients. Diagnosis is depending on the decreasing of platelet count under 150 000 per mm3 or lower than 50% of the basal level. There are two different types of HIT and they differ from each other regarding prevalence, mechanisms of development, times of onset and clinical presentations. Type II HIT has an immunological mechanism and presents with the development of HIT antibodies. Prevalence of type II HIT is as low as 0.2% in the literature [1]; however, type I HIT can be experienced after the procedures needing high active clotting time levels, such as coronary artery bypass grafting or mechanical valve surgeries. Prevalence of type II HIT is higher and reaches up to 30% after open-heart surgeries [2]. Type I HIT develops as a result of direct toxic effect of heparin on platelets. Type I HIT generally develops in the first few days after heparin exposure; on the other hand, type II HIT is known to have a later onset and starts after 5 days following heparin administration. Clinical presentations also differ between these two types of HIT: type I HIT presents with bleeding, whereas type II HIT presents with typical, reactive thrombosis complications. Stopping of heparin-derived anticoagulants is essential, but we have little evidence about the management of these patients, especially if they need oral anticoagulation in the follow-up. In this paper, we want to present a case of type I HIT patient with a need of oral anticoagulation.

CASE REPORT

A 66 years old male patient admitted to the emergency department with syncope. On his admission electrocardiography (ECG), acute inferior myocardial infarction (MI) with complete AV block was detected. A diagnostic coronary angiography was performed emergently with a temporary percutaneous pacing lead. The right coronary artery (RCA) revealed aneurysmatic anatomy with global thrombosis, and we performed iterative percutaneous balloon angioplasties with diameters of 3.0 and 4.0mm coronary balloons. We were unable to achieve a flow so we administered an intracoronary tirofiban via a microcatheter for perfusion of distal coronary bed. A TIMI-2 flow appeared following the tirofiban bolus, and sinus rhythm was accessed; we noticed ST-segment resolution on 12-lead ECG. We decided to continue the tirofiban infusion and planned a control coronary angiography. On his control coronary angiography, the RCA was still globally thrombosed, but near TIMI-3 flow existed in the distal coronary bed. We planned an oral anticoagulation for high thrombotic burden in the long term.

At the 36th hours of unfractionated heparin administration, the patient developed massive gastrointestinal bleeding, bleeding from the femoral access site, and subcutaneous bleeding from venous indwelling. His hemoglobin level was 6.7gr/dL, and thrombocyte count was 39 000/mm3. Following these results, type-1 heparin-induced thrombocytopenia (HIT) was diagnosed, and all antiplatelet and anticoagulant therapies were stopped, and 5 000IU protamine sulfate was given to him. We replaced four units of whole blood, and the patient became stable hemodynamically. Follow-up hemoglobin and thrombocyte counts were expressed in Figures1 and 2. We started clopidogrel 1x75mg on the day of the thrombocyte count at >50 000/mm3 and rivaroxaban 1x15mg with a thrombocyte count >100 000/mm3. The patient recovered excellently, and we stopped clopidogrel because of severe allergic reaction while continuing with rivaroxaban 1x20mg at the end of the 1st month.

DISCUSSION

Coronary aneurysms can be congenital or can develop secondarily to various conditions such as atherosclerosis, trauma, previous coronary interventions, Kawasaki's disease, mycotic emboli, or systemic lupus erythematosus [3]. Aneurysmatic anatomy disrupts the laminar pattern of coronary blood flow, and this may result in the formation of coronary thrombus. In the setting of acute coronary syndromes following the correction of the blood flow of the distal coronary bed, in most cases with coronary aneurysm, we are not be able to perform stenting because of inappropriate vessel size [4]. Some authors offer the administration of thrombolytic therapy in cases where distal blood

Figure-1: Timeline of hemoglobin follow-up

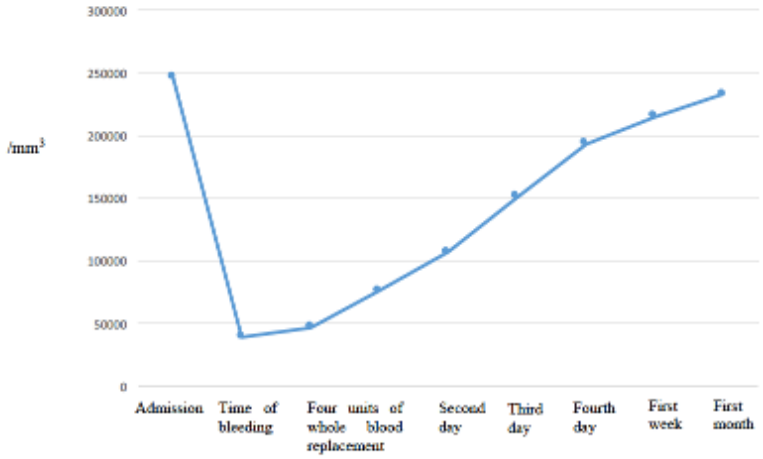

Figure-2: Timeline of thrombocyte count follow-up

flow fails to restore with traditional percutaneous balloon angioplasty techniques [5]. In our patient, the coronary anatomy revealed diffuse aneurysmal dilatation starting from the proximal portion of the RCA and extending distal part of it. We measured the diameter of the RCA as 5.7mm, and we decided that the stenting of this highly thrombosed, long segment aneurysm, might not be the optimal treatment choice. On the other hand, we evaluated the restoration of normal sinus rhythm, resolution of ST-segment elevation, and cessation of chest pain as the signs of successful reperfusion of distal coronary bed, so long-term oral anticoagulation was decided as the follow-up treatment.

Heparin-induced thrombocytopenia (HIT) resembles two distinct clinical presentations in clinical practice as type I and type II. The main difference between them is the time of development of thrombocytopenia [2]. Type I HIT generally develops in the first 5 days after exposure to heparin. In our case, thrombocytopenia developed at 36th hour after heparin administration. These two types of HIT also have different clinical features. Type I HIT is a non-immunological pathology and generally presents with bleeding. Type II HIT is a rare, immune-mediated disorder and shows a paradoxical thrombogenic status despite profound thrombocytopenia [6]. Massive gastrointestinal bleeding was the clinical presentation in our case. We were unable to search HIT antibodies by the time of the onset of thrombocytopenia and clinical presentation, as bleeding directed us to type I HIT as a definitive diagnosis. Oral anticoagulation is highly recommended for type II HIT because of the prevention of future thrombosis. But there is not enough data about the management of type I HIT patients, especially if they have the need

for an oral anticoagulation, as in our case. The main concern rises from the risk of bleeding which is already highly prevalent in type I HIT patients. Previous studies offer the bridging with a non-heparin parenteral anticoagulant within the period of the recovery of thrombocyte count and after thrombocyte count exceeds 150 000/mm3, starting with a vitamin-K antagonist oral anticoagulant in type II HIT patients; but no data exists about type I HIT patients [7].

We have limited choices for non-heparin parenteral anticoagulants, the likes of bivalirudin and fondaparinux, and these treatments require laboratory monitoring and have high costs in practice, as well as the fact that access to these medications may not be easy. Recently, some studies have been published about the role of direct oral anticoagulants (DOAC) in the HIT treatment. There is strong evidence for the use of DOACs safely in the HIT patients, and rivaroxaban has the most significant proof among them [8, 9]. The authors claim that there is no need for bridging with another non-heparin parenteral anticoagulant in the case of rivaroxaban usage and recommend the start of rivaroxaban at the time of diagnosis of HIT. They report that the possibility of thrombosis declines as low as 2.2% with the rivaroxaban treatment [10]. Of course, these recommendations are proven for type II HIT patients. There is no universal consensus for the management of the type I HIT. We encounter type I HIT, especially in the first few days of open-heart surgeries, and in most cases, cessation of heparin treatment can be enough for the recovery. But we do not have any previous data about the management of the type I HIT patients who need long term oral anticoagulation, such as in our case.

CONCLUSION

Differential diagnosis of two different types of HIT is essential in the management of HIT patients. Oral anticoagulation is essential in the treatment of type II HIT disorders, but the need of bridging therapy with a non-heparin parenteral anticoagulant can be problematic for physicians. We do not have any clinical data about the efficacy and safety of oral anticoagulation in type I HIT patients who already present with high bleeding risk. Novel oral anticoagulants are promising choices regarding the efficacy and safety in the treatment of HIT patients, but we need further clinical studies in this area.

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