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The Use of Fondaparinux in The Setting of Antithrombin Deficiency: A Case Report

Manar Omar[1] and Maher Al-Hajjaj[2*]

[1]*Laboratory Medicine, Almalakiya Clinics, Doha, Qatar*

[2]*Department of Surgery, University of Aleppo, Aleppo, Syria.*

[*]**Corresponding author:** Maher Al-Hajjaj, Department of Surgery, University of Aleppo, Aleppo, Syria.

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Abstract

Heparin is an anticoagulant (blood thinner) that prevents blood clots. Heparin-induced thrombocytopenia (HIT) is a potentially life-threatening complication of taking the drug heparin. HIT occurs when heparin binds to a protein in platelets called PF4. We present a case of 59 years old patient with severe liver cirrhosis who developed HIT after using heparin. We administrated fondaparinux-specific anti-Xa drug with daily monitoring of levels of antithrombin. After several times of monitoring, we reached an acceptable range of blood levels of fondaparinux. Our patient did not have any thrombotic or hemorrhagic accidents. Follow-up for one month showed no complains.

Introduction

Heparin-induced thrombocytopenia (HIT) occurs in up to 5% of patients who are exposed to unfractionated heparin for 5 or more days. Due to early recognition and discontinuation of heparin therapy, the mortality rate in response to HIT has decreased from 20% to 2% [1].

This complication occurs as a result of antibodies binding to the highly immunogenic complex of heparin and platelet factor 4 (PF4). These antibodies subsequently activate platelets through their Fc receptors, causing the release of prothrombotic platelet-derived microparticles, which in turn promote thrombin generation and contribute to a hypercoagulable state [2].

The use of alternative anticoagulants in patients with suspected HIT remains an unanswered question. Fondaparinux, a factor Xa inhibitor, has been used for the treatment of patients with HIT but has not gained Food and Drug Administration (FDA) approval for this indication [3].

Various case reports and case studies concluded that fondaparinux may be an effective alternative anticoagulation agent for the treatment of HIT [4,5].

Here, we reported a rare case of the use of fondaparinux as an alternative of heparin because of heparin-induced thrombocytopenia. We succeeded to prevent thrombus and bleeding at one time.

Case Presentation

A 59-year- old male patient with advanced liver cirrhosis was admitted to prepare for trans jugular intrahepatic portosystemic shunt (TIPS) procedure. The past medical history was remarkable for chronic hepatitis B virus ten years ago. Because of incompliant with his anti-virus drugs, he reached the stage of severe cirrhosis. In the last two months, he had a newly developed portal hypertension and hepatic hydrothorax. During hospital admission, he developed thrombocytopenia and a pulmonary embolus after the initiation of heparin treatment. He was diagnosed with heparin-induced thrombocytopenia (HIT). Because of severe liver impairment, we decided to start with fondaparinux (7.5 mg subcutaneously daily) for anticoagulation treatment instead of heparin.

To stay in the therapeutic reference, we aimed to test antithrombin levels.

After 36 hours of administration of fondaparinux, we found that the patient had very low antithrombin activity, 20% (reference range 75%-125%). Because of the risk of developing new thrombosis and bleeding, we decided to monitor fondaparinux-specific anti-Xa levels. After 24 hours of administration of therapeutic doses of anticoagulation, we found low levels of fondaparinux-specific anti-Xa at 0.28 mcg per mL. We were expecting a range of 0.5 to 1.5 mcg per mL. After achieving normal levels of antithrombin, we had normal levels of fondaparinux specific anti-Xa.

We had a strategy of daily monitoring of levels of anti-Xa with sometimes administration of antithrombin concentrate when we had low levels of fondaparinux. This led to achieving a good balance between bleeding and thrombus. Our patient did not have any accidents over 25 days of follow-up. He was discharged after the TIPS procedure was successful. Follow-up for another one month showed no complaints.

Discussion

Routinely monitoring of levels of fondaparinux-specific anti-Xa is not necessary. Nevertheless, we had a case that we monitored levels of fondaparinux in the setting of antithrombin deficiency.

There is limited data evaluating the use of fondaparinux for the treatment of suspected or confirmed HIT [3].

However, several cases reported that we can use fondaparinux in the setting of HIT.

There are no guidelines instruct physician how to use fondaparinux in HIT. Several monitoring of levels of fondaparinux and addition of appropriate supplementation of antithrombin can be necessary to ensure that the fondaparinux level is within the therapeutic range.

We need further and large studies regarding monitoring antithrombin levels and supplementation in patients who underwent the administration of antithrombin agents in the setting of congenital or acquired antithrombin deficiency.

However, patients with decompensated cirrhosis cannot undergo surgical treatment due to either severe liver dysfunction or lack of matching liver donors. During the early stages of thrombosis, thrombolytic therapy can achieve good outcomes; but increased dosing may be required and the probability of intestinal bleeding may increase.

Furthermore, indications for thrombolytic therapy should be strictly acknowledged. Patients with decompensated cirrhosis may not meet the indications for thrombolytic therapy due to grade C liver function according to Child–Pugh classification, history of gastrointestinal bleeding, thrombocytopenia, and coagulation disorders [6].

In this case, we had a patient with advanced liver function impairment because of chronic hepatitis B. He is going to underwent TIPS. In the setting of the use of heparin, he developed HIT. We stopped heparin and we started with fondaparinux. With close and repeated monitoring of antithrombin levels, we achieved a balance between bleeding and thrombus.

Conclusion

In patients with antithrombin deficiency receiving fixed fondaparinux dosages, regular monitoring of antithrombin and fondaparinux-specific anti-Xa levels may be necessary. Further studies are needed to find international guidelines for such cases.

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