Myocardial Infarction in a Splenectomized Patient with Immune Trombocytopenia

Ilknur NIZAM1,*, Mehmet Ali Erkurt2, Ilhami Berber2, Emin Kaya2, Irfan Kuku2, Mustafa Koroglu3

1Department of Internal Medicine, Faculty of Medicine, Inonu University, Malatya, TURKEY
2Turgut Ozal Medical Center
3Karabuk Education and Research Hospital
*Corresponding author: ilknizam@yahoo.com

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Abstract Immune trombocytopenia is an autoimmune disease characterised by abnormal platelet destruction. Corticosteroids and intravenous immune globulin are main first line treatment options. Splenectomy is a preferred second line treatment option for unresponsive patients at many centers. Secondary thrombocytosis is a possible complication awaiting this group of patients after splenectomy. Here, we present a case of myocardial infarction at a patient who had undergone splenectomy for ITP, which is a rare event seen with secondary thrombocytosis due to splenectomy.

Keywords: immune thrombocytopenia, splenectomy, myocardial infarction


1. Introduction

Immune trombocytopenia (ITP) is an autoimmune disease in which the immune system targets platelets and megakaryocytes, resulting in decreased platelet counts and increased risk of bleeding [1]. The pathophysiology of ITP includes both antibody-mediated and T cell–mediated platelet and/or megakaryocyte destruction [2]. Thrombocytopenia can cause bleeding; the aim of treatment is to stop the bleeding and also decrease the risk of possible clinically-significant bleeding in the future [3]. Nearly 70-80% of newly-diagnosed, treatment-naive patients respond to prednisone based treatment schemes, but most of the patients have recurrent thrombocytopenia when prednisone dose is tapered which leads to further treatment [4,5]. Besides corticosteroids, intravenous immune globulin (IVIG), and splenectomy are main options of treatment where novel therapies including rituximab and the thrombopoietin receptor agonist, eltrombopag, is a safe and efficient novel agent in resistant and relapsed ITP [11,12]. Elevated risk of thrombosis has been reported with eltrombopag, especially with known thrombophilia and chronic liver disease [13]. Besides, patients with elevated von Willebrand factor (vWF) levels had been found to have a higher tendency for thrombosis [14].

Here, we present a case of myocardial infarction at a patient who had undergone splenectomy for ITP.

2. Case Presentation

A 45-year-old woman had been diagnosed as ITP 11 years ago and she had been followed up until she had severe bleedings that didn’t respond to corticosteroids and IVIG. Splenectomy was performed 1 year ago due to steroid refractoriness. Platelet count increased rapidly after the operation where platelet count was over 700,000/mm3 at the first month of operation. Following this first-month visit, she was admitted to the emergency service of another health center with severe chest pain where she was diagnosed with myocardial infarction by electrocardiography and elevation at cardiac markers. The platelet count at the time of MI diagnosis was 571,000/mm3. Immediate coronary angiography was performed and left anterior descending (LAD) artery was observed to be occluded. The cardiologists avoided any further treatment because of her primary diagnosis of ITP and treated the patient with a beta blocker, acetylsalicylic
acid and atorvastatin. Initial ECG was not available as it was performed in another center but the patient came to the cardiology outpatient unit for the 1st month followup and T wave abnormality could be observed at her ECG (Figure 1). At transthoracic ecocardiography performed at this visit, the ejection fraction was 55% and she had no wall motion abnormalities. She had no risk factors for coronary artery disease. She was not obese, she had no history of hyperlipidemia, diabetes mellitus (DM) or hypertension (HT). She had no family history of coronary artery disease. Protein C, protein S, and homocysteine workup was within normal ranges, and factor V Leiden mutation was not detected. vWF level was normal.

Figure 1. ECG, one month after MI (Negative T waves)

3. Conclusion

There is no consensus on the optimal treatment for patients with ITP who suffer acute MI. Antiplatelet therapy is contraindicated as ITP increases the risk of bleeding. Percutaneous coronary intervention has been performed and reported as successful by some authors for patients with chronic ITP, where concomitant therapy with acetylsalicylic acid, clopidogrel, and unfractionated heparin caused no major bleeding [15,16,17]. Our patient underwent coronary angiography and administered acetylsalicylic acid and no bleeding occurred during the intervention or the followup therapy.

The platelet count at primary thrombocytosis is higher compared to secondary thrombocytosis and primary thrombocytosis is more likely to cause thromboembolic complications compared to secondary thrombocytosis. Therefore, it may be concluded that higher platelet counts may affect clinical outcomes [18]. Besides higher platelet counts, primary thrombocytosis is significantly associated with an increased incidence of both arterial and venous thromboembolic complications. In secondary thrombocytosis, thromboembolic events are restricted to the venous system and occur only in the presence of other risk factors [19]. Our patient had no risk as she wasn’t obese or had any risk factor for coronary artery disease such as hyperlipidemia, DM or HT. Laboratory tests regarding thrombophilia were normal. vWF level was normal.

An arterial thrombus concomitant with secondary thrombosis is a rare occasion meriting clinical consideration. Physicians should be aware that even a patient with secondary thrombosis may suffer from clinical conditions arising from arterial thrombi. Besides, each patient with ITP should be followed up carefully for possible thrombotic predispositions.

References


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