www.aaem.pl

Reactive thrombocytosis resulting from frequent blood donations as an extremely rare cause of ST Segment Elevation Myocardial Infarction in the case of a 19-year-old male

Andrzej Jakub Sałacki^{1,2,A-D,F}, Andrzej Wysokiński^{2,E}

¹ Doctoral Studies, II Faculty of Medicine with English Language Divison, Medical University, Lublin, Poland ² Chair and Department of Cardiology, Medical University, Lublin, Poland

A – Research concept and design; B – Collection and/or assembly of data; C – Data analysis and interpretation; D – Writing the article; E – Critical revision of the article; F – Final approval of article

Sałacki AJ, Wysokiński A. Reactive thrombocytosis resulting from frequent blood donations as an extremely rare cause of ST Segment Elevation Myocardial Infarction in the case of a 19-year-old male. Ann Agric Environ Med. 2018; 25(4): 602-604. doi: 10.26444/aaem/74196

Abstract

Introduction. Thrombocytosis is a disease where the platelet count exceeds 600,000/µl. It is usually reactive in nature, less often clonal. Reactive thrombocytosis is usually a response to reactive inflammation or infection, splenectomy, iron deficiency, pregnancy, physical effort or cancer.

Case report. The case is described of a heart attack with ST-segment elevation in a 19-year-old male which was occurred due to intensive haematopoietic system renewal, caused by frequent blood donation which, in turn, caused reactive thrombocytosis. Reactive thrombocytosis is very rarely the cause of heart attack with ST-segment elevation, and is extremely rarely caused by blood donation. It is generally considered that reactive thrombocytosis is not a risk factor in the case of thromboembolic complications.

Conclusion. The presented case and the literature point to the risk of serious thromboembolic incidents, including heart attack, in the course of thrombocytosis.

Key words

ST segment elevation myocardial infarction, reactive thrombocytosis, blood donations

INTRODUCTION

Thrombocytosis is an elevated platelet count >600,000/ul [1]. This increase is most often reactive by nature (reactive thrombocytosis), or less frequently clonal (clonal thrombocytosis) in chronic myeloproliferative disorders: polycythemia vera, chronic myeloid leukemia, and particularly essential (primary) thrombocytosis [2, 3].

Reactive thrombocytosis (RT) is most often secondary to inflammatory disorders and infections, periods of haematopoietic system intensive restoration, splenectomy, iron deficiency, physical exertion, pregnancy, cancer (lung cancer, colon cancer) and myeloproliferative condition [4]. RT is an extremely rare cause of myocardial infarction in patients without atherosclerotic lesions of coronary vessels [5].

CASE REPORT

A 19-year-old male patient was transferred from ED to CICU due to suspected anterior myocardial infarction (STEMI). The patient reported very strong pains in the thorax. The patient had not undergone medical treatment up to that point and had not been taking any medicines. Upon admission, blood pressure was 150/80 mm Hg, BMI 21.3 kg/m². Physical

Address for correspondence: Andrzej J. Sałacki MD, Chair and Department of Cardiology, Medical Uniwersity of Lublin, 8 Jaczewskiego St, 20-954 Lublin, Poland e-mail: andrzej.salacki@umlub.pl

Received: 27.06.2016; accepted: 30.05.2017; first published 15.07.2017

examination did not show any deviation from the norm. Medical history showed a 3-day infection of the upper respiratory tract. The patient was a non-smoker and never used narcotics or anabolic steroids. He practiced swimming twice a week for 60 minutes. His medical history showed no instances of coronary disease in his family. The patient also reported that he has been giving blood very often for the last two years, every two months on average - 500ml five times in the last year. The last time he gave blood was 10 days ago. Laboratory tests showed: RBC 4.98 mln/ul (4,5-6,1), HGB 14.3 g/dL (14-18), HCT 43% (40-54), WBC- 19.32/ul (4-10.5), NEUT-16.62/ul (2.5-5.0), PLT 957 th/ul (130-400), MPV 8.5 fL (8-11), PCT 0.56% (0.14-0.36%), PDW 48.1 fL (40-60), potassium level 5.5 mmol/l, troponine I > 50,000 ng/ml (<0.04), CRP 0.6 mg/l (0-5), LDH 675 U/L(120-246), iron 27,0 ug/dl(65–175), lipidogram without any deviations from the norm. Additional tests (urine and blood) excluded the possibility of the patient taking narcotics (marijuana, cocaine, amphetamine or opiates). ECG showed regular sinus rhythm with the frequency of 55/min, normogram, ST-segment elevation in I, aVL and V2-V5, ST-segment depression in III and aVR (Fig. 1). Transthoracic echocardiogram (TTE) showed left ventricle apical akinesis with wall thinning in the apex area, and thinning of the apical segment of the interventricular septum, ejection fraction of 46%, mitral insufficiency and tricuspid insufficiency - both of the 1st degree, restrictive LV filling pattern, trace amount of fluid in the pericardial cavity and thickening of the pericardial layers to 0.4 cm. The patient was directed urgently for coronary angiography (Fig. 2). Prior to the procedure, the



Figure 1. ECG upon admission (regular sinus rhythm with frequency of 55/min, normogram, ST-segment elevation in I, aVL and V2-V5, ST-segment depression in III and aVR)

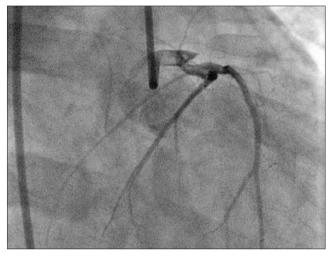


Figure 2. Left anterior descending artery: anterior interventricular branch closed in the proximal segment

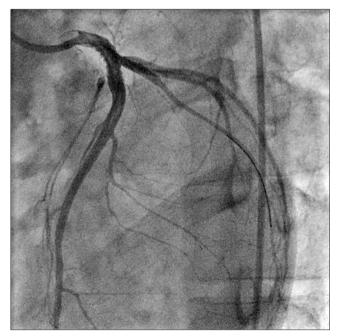


Figure 3. Bifurcation of the left anterior descending artery with the diagonal branch – after percutaneous angioplasty

patient was administered 180 mg of Ticagrelor and 300 mg of acetylsalicylic acid.

After the procedure, it was discovered that the patient had the anterior interventricular branch of the left coronary artery (LAD) closed in the proximal segment. Aspiration thrombectomy was conducted and effective percutaneous angioplasty of the bifurcation of the left anterior descending artery with the diagonal branch followed immediately. Thrombi was drawn and two anti-proliferative stents (DES) were implanted (Fig. 3). Abciximab infusion was also performed. Control TTE showed improved LV contractility, EF= 52%. During the 3rd and the 4th day of hospitalization, the maximum observed body temperature was 37.8 °C.

During the patient's stay at hospital, the levels of C and S proteins, IgA, IgM and IgG anticardiolipin antibodies, homocysteine fibrinogen as well as the total protein concentrations were tested and lupus anticoagulant test, antithrombin activity test, protein electrophoresis and lipoprotein electrophoresis were performed. The results were within the referential values. The factor V Leiden was absent. The final morphology (after 8 days of hospitalization) showed RBC 3.53 mln/ul, HGB 11.3 g/dL, HCT 31.1%, WBC 7.65/ul, PLT 590 th/ul. The patient was directed to the Department of Early Cardiac Rehabilitation of a Resort Hospital and advised treatment (including iron supplementation), further cardiological and haematological control.

After three months, the patients was in a good condition, control tests showed WBC 6.92/ul, RBC 5.25 mln/ul, HGB 14.6 g/dL, HCT 44%, PLT 470 th/ul, iron level 39 ug/dl, TIBC 378 ug/dl (250–450), transferrin 313 mg/dl (215–365), ferritin 44 ng/ml (22–322). More intensive iron supplementation was prescribed.

DISCUSSION

Thrombocytosis is the second most common cause of death in patients with polycythaemia vera (PV) or essential thrombocytosis (ET) [6]. The available data show that the frequency of cardiovascular complications caused by ET varies from 4%–21%. Thrombosis is more frequent in the coronary vascular bed area (75%) than in the venous area (25%) [7]. However, one should bear in mind that only slightly over 10% of patients with elevated platelet count suffer from myeloproliferative cancer. The remaining cases constitute thrombocytosis with other medical conditions [8]. However, reactive thrombocytosis is not a risk factor in the case of thromboembolic complications [9].

A large-scale retrospective study comprising 732 patients with thrombocytosis showed that 87.7% suffered from RT. Thromboembolic complications were present in 12.4% of patients with primary thrombocytosis, compared to 1.6% patients with RT. All patients with RT who also developed thrombotic complications had additional risk factors, such as a previous operations or cancer changes [10]. Another retrospective study comprising 318 patients without myeloproliferative conditions and after splenectomy, showed that thrombocytosis developed in 75% of patients, without a significant rise in thromboembolic incidents [11]. Yet, in another retrospective study comprising 129 patients with the platelet count over 1 mln/uL, Buss et al. [12] showed a constant frequency of incidents of thrombosis among patients with myeloproliferative conditions and patients with RT. The available data indicates RT as the reason for ST Segment Elevation Myocardial Infarction (STEMI) in a variety of cases [1, 13–17]. The phenomenon of reactive thrombocytosis after splenectomy is the one best known so far. The peak of the platelet count occurs 2–4 weeks after the procedure, and after that they begin to slowly decrease. In some cases, normalization takes place after a few years and remains that way for a long time [18]. As a result, thromboembolic complications are so rare that there is no need to administer anticoagulants continuously [19].

ST Segment Elevation Myocardial Infarction in a 19-yearold man is an extremely rare case. The available data does not provide a case where it occurred as a result of reactive thrombocytosis caused by giving blood regularly, especially in such a young patient. Thus, this case requires a detailed diagnosis with respect to diseases which might cause hypercoagulability, but have not yet been diagnosed. It should also be stressed that the patient had no additional risk factors like smoking, obesity or dyslipidaemia. However, the fact that one vessel (of the artery that caused the myocardial infarction) was occluded is very characteristic in the case of a myocardial infarction in a young patient, since it is usually connected with LAD, as in the reported case.

Upper respiratory tract infection was a favourable situation for RT, but the short duration and mild severity(CRP 0.6mg/l on the day of hospital admission) were not predisposing to a high thrombocytosis as presented. This level of thrombocytosis might be observed in chronic or prolonged inflammations. Long-lasting iron deficiency (continued also in a control test after 3 months), caused by often occurring phlebotomies, was not a dominant cause of thrombocytosis, because despite a persistently low iron level, the number of platelets normalized.

The reported case presents the possibility of a significant vascular incident in the case of RT, and reflects on the nature of the described problem, as well as encouraging the carrying out of further tests and developing methods to prevent similar situations.

REFERENCES

- Aundhakar SC, Mahajan SK, Mane MB, Lakhotiya AN. Reactive thrombocytosis leading to acute myocardial infarction. J Assoc Physicians India. 2013; 61: 745–747.
- Tefferi A, Hanson CA, Inwards DJ. How to interpret and pursue an abnormal complete blood cell count in adults. Mayo Clin Proc. 2005; 80: 923–936.
- 3. Schafer AI. Thrombocytosis. N Engl J Med. 2004; 350: 1211-1219.
- Sułek K. 1000 practical questions in hematology. Publishers 7 i pół. 1996; 8: 151–155.
- 5. Kelly SV, Burke RF, Lee KS, Torloni AS, Lee RW, Northfelt D, et al. Acute myocardial infarction: An unusual presentation of essential thrombocytosis in a 17- year-old man. Clin Adv Hematol Oncol. 2008; 6: 133–136.
- Vannucchi AM, Barbui T. Thrombocytosis and thrombosis. Hematology Am Soc Hematol Educ Program. 2007; 1: 363–370.
- Scheffer MG, Michiels JJ, Simoons ML, Roelandt JRTC. Thrombocythemia and coronary artery disease. Am Heart J. 1991; 122: 573-576.
- Dmoszyńska A, Robak T, Hus I. Basic hematology. 3rd edition. Czelej 2015.
- Williams B, Morton C. Cerebral vascular accident in a patient with reactive thrombocytosis: A rare cause of stroke. Am J Med Sci. 2008; 336: 279–281.
- Griesshammer M, Bangerter M, Sauer T, Wennauer R, Bergmann L, Heimpel H. Aetiology and clinical significance of thrombocytosis: Analysis of 732 patients with an elevated platelet count. J Intern Med. 1999; 245: 295–300.
- Boxer MA, Braun J, Ellmann L. Thromboembolic risk of postsplenectomy thrombocytosis. Arch Surg. 1978; 113:808–809.
- Buss DH, Stuart JJ, Lipscomb GE. The incidence of thrombotic and hemorrhagic disorders in association with extreme thrombocytosis: An analysis of 129 cases. Am J Hematol. 1985; 20: 365–372.
- Tai YT, Yu YL, Lau CP, Fong PC. Myocardial infarction complicating post splenectomy thrombocytosis, with early left ventricular mural thrombus formation and cerebral embolism: A case report. Angiology. 1993; 44: 73–77.
- Nielsen H. Fatal acute myocardial infarction associated with post splenectomy thrombocytosis. Ugesker Laeger. 1988; 150: 2125–2126.
- Aundhakar SC, Mahajan SK, Mane MB, Lakhotiya AN. Reactive thrombocytosis leading to acute myocardial infarction. J Assoc Physicians India. 2013; 61: 745–747.
- Ghaffari S, Pourafkari L. Acute myocardial infarction in a patient with post-splenectomy thrombocytosis: A case report and review of literature. Cardiol J. 2010; 17: 79–82.
- 17. Dumrongmongcolgul N, Mankongpaisarnrung C, Sutamtewagul G, Hosiriluck N, Chen T, Trujillo, et al. Reactive Thrombocytosis Associated with Acute Myocardial Infarction following STEMI with Percutaneous Coronary Intervention. Case Rep Cardiol. 2013.
- Lewis SM. The spleen. In: Hoffbrand AV, Catovsky D, Tuddenham EG, editors. Postgraduate hematology. 5th Ed. Blackwell Publishing, Oxford 2005: 364.
- Williams B, Morton C. Cerebral vascular accident in a patient with reactive thrombocytosis: A rare cause of stroke. Am J Med Sci. 2008; 336: 279–281.