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Case Report

Extreme thrombocytosis in traumatic amputee and role of thromboembolism prophylaxis: a case report

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ABSTRACT

Platelets are the smallest blood component produced in the bone marrow that plays a fundamental role in the blood clotting process. A normal platelet count applicable to all adults is 150 to 400×10^9/l. Thrombocytosis develops when the platelet count exceeds 450×10^9/l. Thrombocytosis is classified into primary thrombocytosis and secondary (or extreme) thrombocytosis. Primary thrombocytosis is a chronic myeloproliferative disorder in which sustained megakaryocyte proliferation leads to an increase in the number of circulating platelets. Extreme thrombocytosis or reactive thrombocytosis, is defined as abnormally high platelet count in the absence of chronic myeloproliferative disease, secondary to an underlying events, disease, or the use of certain medications. Causes of reactive thrombocytosis include acute blood loss, acute infection, amputation, iron deficiency, asplenia, cancer, chronic inflammatory or infectious diseases. Secondary thrombocytosis resolves when the underlying event is managed. Extreme thrombocytosis may result in thromboembolic episode such as mesenteric vein thrombosis, pulmonary embolism and acute myocardial infarction. In patients who survive after trauma the platelet count displays a bimodal response with an initial decrease below baseline values, followed by an increase above the normal range after 1 week. We report a similar experience of a trauma patient with reactive thrombocytosis and discussion on importance of thromboprophylaxis.

Keywords: Extreme thrombocytosis, Trauma, Amputation, Thromboprophylaxis

INTRODUCTION

Extreme thrombocytosis defined as occurrence of platelets counts more than 1,000,000/µl is rare but dangerous phenomenon.[1] Its occurrence has been documented in orthopaedic surgeries and also in polytrauma patients, isolated orthopedic traumas and post-splenectomy apart from blood malignancies. Extreme thrombocytosis is often associated with thrombotic events such as mesenteric vein thrombosis, pulmonary embolism and acute myocardial infarction.[2] Though occurrence of thrombocytosis (platelet count above 450,000/µl) is common up to 20%, extreme thrombocytosis is rare approximately 1.4-5.7% of trauma patients.[1]

These patients are more susceptible to venous thromboembolism (VTE) (2%) and prolonged intensive care stay and increased mortality hence, these require careful consideration for thromboprophylaxis. Thromboprophylaxis is currently the standard in polytrauma patients once patients are optimized physiologically and required on further immediate surgical interventions with aspirin or low weight molecular heparin.[3]

We report a case of extreme thrombocytosis in traumatic amputee patient who underwent revision amputation and sequential debridement and wound management.

CASE REPORT

A 18 year old male was brought to the emergency department with history of train accident and traumatic amputation at right thigh level and degloving injury over left leg. Patient immediately received primary care at primary health care centre with intravenous fluids, analgesics, antibiotics, wound wash and ligation of bleeding vessels and pressure bandage of traumatic amputated stump and stay sutures and dressing of the degloved wound, and was referred to our level-II trauma centre with supplemental oxygen and intravenous crystalloid fluids.

On arrival, primary survey revealed no immediate life-threatening injuries with stable hemodynamics, full GCS, no obvious chest, abdomen-pelvic injuries. On secondary survey, there was traumatic amputation at the right mid-thigh level with no active bleeding and contamination, left leg showed extensive degloving injury from knee up to ankle with stay sutures, gross contamination, partly necrosed muscles, and partly exposed bone and neurovascular bundles in the lower end of left leg.

Chest X-ray, pelvic X-ray was normal, FAST- were negative. Patient was further resuscitated with crystalloids, broad spectrum antibiotics, tetanus immunoglobulin were administered.

After hemodynamically stable, and noting all extremity injuries, NCCT head and C-spine, and CECT torso was done considering dangerous mechanism of injury and ruled out head, spine, chest, abdomen, and pelvic injuries. All baseline laboratory investigations were sent (Table 1).

Table 1: Baseline serological investigations on the day of admission.

Parameters	Value	Units
Albumin	3.6	g/dl
Alkaline phosphatase	110	U/l
SGPT	38	U/l
SGOT	77	U/l
Bilirubin-conjugated	0.58	mg/dl
Bilirubin-total	1.06	mg/dl
Calcium ionised	1.02	mmol/l
Creatinine	0.43	mg/dl
Sodium	136	mmol/l
Potassium	4.8	mmol/l
Total calcium	8.6	mg/dl
Urea BUN	26.2/12.25	mg/dl

After further stabilizing the patient, carefully noting all the injuries, patient underwent right thigh revision amputation, thorough washing, debridement and dressing of left leg degloving injury. Patient was further resuscitated in intensive care unit with PRBC, FFP. Subsequently patient underwent further serial debridement for the degloved wound under spinal anaesthesia for 5 times and serial wound dressing was done under bedside sedation. Finally, skin graft was applied after wound was healthy. Early rehabilitation was started within 48 hours and continued after discharge. The patient is under follow up and presently active rehabilitation with artificial limb is provided.

Characteristic laboratory finding in this patient was the occurrence of extreme thrombocytosis (Figure 1). Platelet counts were 1,80,000/cumm on admission, 1,35,000/cumm on day 2, 1,90,00/cumm on day 5, followed by steep rise to 6,50,000/cumm, 7,00,000/cumm, 12,30,000/cumm respectively on day 10,11, and 14 days. Further on platelets dropped gradually to 10,60,000/cumm, 7,00,000/cumm on days 17 and 19, and reached normal values after day 26. The coagulation profile (prothrombin time, activated partial thromboplastin time), and other laboratory parameters were normal through-out the hospital course.

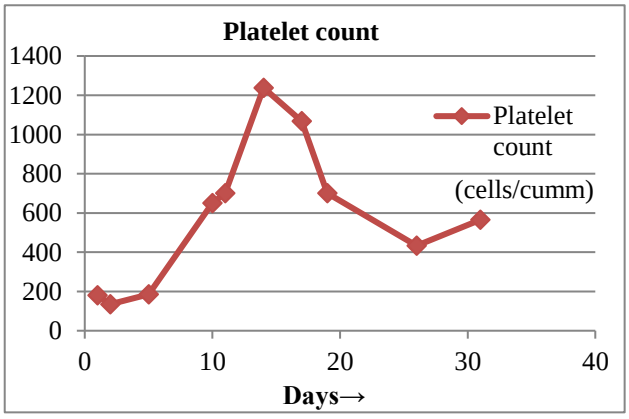

Figure 1: Platelet trend during hospitalization.

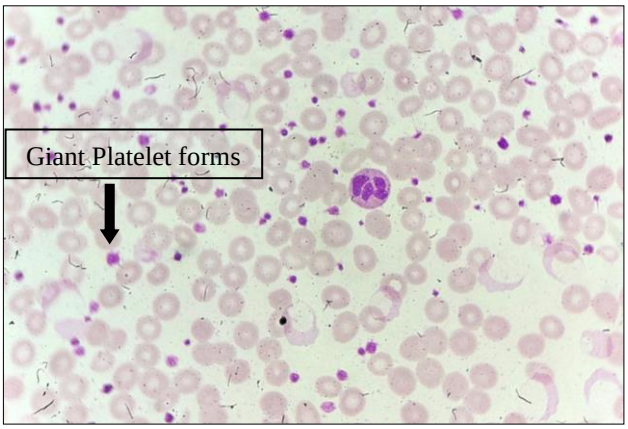

Figure 2: Photomicrograph of Leishmann stain of peripheral blood smear showing reactive thrombocytosis along with anisocytosis.

DISCUSSION

Secondary thrombocytosis develops due to the stimulation of thrombopoiesis by IL-6 during the acute phase of injury followed by lack of concomitant platelet consumption.

Another reason for this event is probably an increment in circulating thrombopoietin. IL-6 along with thrombopoietin, stimulates thrombopoiesis although IL-6 is able to support mega-karyo-poiesis. Patients with grievous injuries have the highest plasma levels of this long-lived cytokine.[4]

The laboratory workup of secondary thrombocytosis includes complete blood count, peripheral blood smear, erythrocyte sedimentation rate (ESR), iron studies, rheumatoid factor (RF), antinuclear antibody (ANA) and C-reactive protein (CRP).[5]

Reactive thrombocytosis is associated with an increased risk of venous thromboembolism (VTE) in patients after severe trauma during the recovery phase and is associated with an increased risk of mortality after ICU and hospital discharge. Although secondary thrombocytosis is benign in nature, the underlying aetiology of thrombocytosis can be associated with an increased risk of adverse outcomes.[5,6]

Venous thromboembolism (VTE) is a potential outcome of injury, surgery, and critical illness. Traumatic patients in the ICU are at risk for this condition, prompting routine use of mechanical or pharmacologic prophylactic measures. Furthermore, trauma and surgical patients often have real or perceived contraindications to prophylaxis that affect the timing of preventive measures and the consistency with which they can be applied.[7] Routine surveillance with venous duplex is neither indicated nor feasible for all trauma patients as it does not decrease the risk of VTE or fatal pulmonary embolism (PE). In addition, false positive results lead to unnecessary therapeutic anticoagulation.[8]

Treatment with anti-platelet drugs like aspirin is usually not suggested as the risk of thrombosis is very low in secondary thrombocytosis. Nevertheless, it can be considered for patients with very high platelet count and complications of thrombocytosis are present, or at risk of developing complications.[5]

Direct oral anticoagulants (DOACs) like dabigatran, rivaroxaban, apixaban and edoxaban can also be recommended as latest anticoagulants to prevent complications.

Antithrombotic prophylaxis can be given in oral (warfarin, coumarin) or injectable (unfractionated and fractionated heparin) forms. However, the limitation of oral drugs is that in case patient is taken for emergency exploration, effects of oral drug continues and can have bleeding complications. This limitation can be overcome by unfractionated heparin. Venous thromboembolism (VTE) prophylaxis with either low-dose unfractionated heparin (UFH) or low molecular weight heparin (LMWH) is recommended for hospitalized trauma patients, with LMWH being preferred for most patients.[9]

Low molecular weight fractionated heparin offers better scope as dose is titrated as per body weight and the effect is not very long lasting. Thus, the medication can be stopped and patient can be taken for emergency exploration without the risk of bleeding and providing adequate antithrombotic prophylaxis.

According to Western Trauma Association (WTA) Algorithms Committee guidelines, for most trauma patients, pharmacologic prophylaxis should continue uninterrupted throughout the hospital stay and at times after discharge. Avoiding preventable and non–evidence-based delays to the initiation and missed doses of pharmacologic prophylaxis should be a best-practice focus of all trauma centers, and it has clearly been associated with decreased rates of VTE events.[10]

In adverse situations, thromboembolic phenomenon is the most common and is associated with fatal outcome. Plateletpheresis is not permanent, but a very useful option in reducing the platelet count.[11]

CONCLUSION

Although secondary thrombocytosis is benign, the underlying cause of thrombocytosis can sometimes be associated with an increased risk of adverse and severe outcomes. In our patient though no thromboembolic event developed secondary to thrombocytosis but we should always make an effort for early diagnosis and appropriate management in such cases to avoid complications and mortality.

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