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## Platelets, Thrombosis and Life Threating Activation, why Good Changes in Bad and Ugly

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## Editorial

(Xeno-) antigens after transfusion (and transplantation), Game changers and disturbance in Hemostasis Thrombosis & Life-threating activation of death receptors.

Platelets are lifesaving a-nucleated blood cells, which are playing pivotal rule in rescue or loss of patients with bleedings disorders affected due to different primary and/or secondary diseases. Platelet disorders lead to defects in primary haemostasis, and have marks and indications dissimilar from coagulation factor deficiencies (disorders of secondary haemostasis). There are raising evidences that (transfused) platelets might being involved in the bleeding disorders, cancer metastasis [1,2], and thrombosis, post transfusion. The later processes are strictly regulated by multifactorial dissimilar processes [3,4]. Hypothetically it would be great if we could monitor and affect platelets pathophysiology, timely and externally in any patient with bleedings disorders.

Aime T Franco et al Blood 2015 [5] postulated that the basic scientists who are studying platelets' functions are thinking more beyond the traditional hemostasis and thrombosis models, while the practicing hematologists requisite appreciate platelet relevance in a wide range of disease processes. The body's reaction to vessel wall injury is rapid activation and adhesion of platelets to the injured subendothelium [6]. The initial haemostatic plug, composed primarily of platelets, is stabilized further by a fibrinogen conversion to fibrin network generated in secondary haemostasis. Bleeding stops solely as a result of the primary haemostatic plug formation initiated by good functioning platelets. When platelets do not function properly, primary haemostatic disorders occur that are characterized by prolonged bleeding time, and the characteristic local indications are petechiae and purpura [6,7]. Hemarthrosis and muscle hematomas are not present in primary haemostatic disorders. Cowan and Robson 2015 [8] postulated that the most dangerous indicators of platelets function disorders are the systemic 3 development of a life-threatening consumptive coagulopathy and thrombosis (Figure 1), characterized by posttransfusion thrombocytopenia and bleeding, [8-12] which is balanced at the opposite extreme by indigenous complications of graft loss due to thrombotic microangiopathy [5,8,9,12].

The contributing mechanisms to thrombosis and/or bleeding disorders might include pro-anti-inflammatory processes, vascular injury, heightened innate, humoral and cellular immune responses, and molecular incompatibilities that are affecting the regulation of coagulation. [4-8] There also appear to be organ-specific factors that have been linked to vascular heterogeneity between subjects. Overcoming coagulation dysfunction will necessitate a permutation of genetic and pharmacological strategies.

Qioa et al. [10] suggested another immunological mechanism and proteins which might play important rule in hemostasis, vessel wall injury, platelet-based autoimmune disorders, and thrombosis namely the human platelets express  $Fc\gamma RIIa$ , the low-affinity receptor for the constant fragment (Fc) of immunoglobulin (Ig) G. This receptor is also found on neutrophils, monocytes, and macrophages. Engagement of



this receptor on platelets by immune complexes triggers intracellular signaling events that lead to platelet activation [9] and pathologic thrombosis.

Camera et al. [11] in their review suggested that TF is main cause, which is well established occurred via the contribution of vessel wall-derived tissue factor (TF) to atherothrombosis. Although the pathophysiological relevance of the blood-borne TF is still a matter of debate, and controversies on the presence of platelet-associated TF still exist. These findings are in line with the evidence that platelets are heterogeneous in their functions and only a subset of them is involved in the hemostatic process [6,11].

Taken together, it would be great if externally, patients' platelets with defect and-or any kind of deficiencies could be removed with healthy ones, autologously. Regrettably up to now no Medical Researcher and /or study group were succeeded to exchange bad- with good platelets due to uncountable problems, in the blood banks. I hope in the near

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future we can remove all defects from all kind of blood cells, and from any kind of patients successfully. Then with simple manipulations Medici could repair all damages timely and return improved blood cells to the same patients, autologous.

## References

- 1. Leblanc R, Lee SC, David M, Bordet JC, Norman DD, et al. (2014) Interaction of platelet-derived autotaxin with tumor integrin  $\alpha V\beta 3$  controls metastasis of breast cancer cells to bone. Blood. 124: 3141-3450.
- Stegner D, Dütting S, Nieswandt B (2014) Mechanistic explanation for platelet contribution to cancer metastasis. Thromb Res 133: S149-157.
- Furie B, Flaumenhaft R (2014) Thiol isomerases in thrombus formation. Circ Res 114: 1162-1173.
- Poredos P, Jezovnik MK (2007) The role of inflammation in venous thromboembolism and the link between arterial and venous thrombosis. Int Angiol 26: 306-311.
- 5. Franco AT, Corken A, Ware J (2015) Platelets at the interface of thrombosis, inflammation, and cancer. Blood 126: 582-528.
- Ivanciu L, Stalker TJ (2015) Spatiotemporal regulation of coagulation and platelet activation during the hemostatic response in vivo. J Thromb Haemost

13: 1949-1959.

- Lusher JM (1996) Screening and diagnosis of coagulation disorders. Am J Obstet Gynecol. 175: 778-783.
- Cowan PJ, Robson SC (2015) Progress towards overcoming coagulopathy and hemostatic dysfunction associated with xenotransplantation. Int J Surg S1743-9191.
- Williamson LM, Stainsby D, Jones H, Love E, Chapman CE, et al. (2007) The impact of universal leukodepletion of the blood supply on hemovigilance reports of posttransfusion purpura and transfusion-associated graft-versushost disease. Transfusion 47: 1455-1467.
- Qiao JB, Li J, Zhang XF (2015) Analysis and Treatment of Multiple Severe Venous Vascular Malformation Syndrome Combined with Coagulopathy. Chin Med J (Engl) 128: 2546-2548.
- Camera M, Toschi V, Brambilla M, Lettino M, Rossetti L, et al. (2015) The Role of Tissue Factor in Atherothrombosis and Coronary Artery Disease: Insights into Platelet Tissue Factor. Semin Thromb Hemost 41: 737-746.
- 12. Petz LD, Calhoun L, Yam P, Cecka M, Schiller G, et al. (1993) Transfusionassociated graft-versus-host disease in immunocompetent patients: report of a fatal case associated with transfusion of blood from a second-degree relative, and a survey of predisposing factors. Transfusion 33: 742-750.

Page 2 of 2