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Review article

Thrombocytopenia associated with TAVI—The summary of possible causes

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ABSTRACT

Thrombocytopenia (TP) following transcatheter aortic valve implantation (TAVI) procedure is a common phenomenon but the underlying mechanisms are neither well known nor described. Postinterventional severe TP is related to worse early and late outcome. Moreover, the statement of enhanced platelet and coagulation activation might justify even stronger antiplatelet and anticoagulation therapy following TAVI procedure. Thus, the examination of the pathomechanisms responsible for TP post TAVI seems to be crucial.

Several hypotheses have been raised. TP can be caused by insufficient production or impaired platelet renewal. On the other hand, increased platelet activation, consumption and destruction might also be responsible for TP. These findings, mostly related to the procedure alone, need further investigation.

Here, we summarize the potential multifactorial causes of post TAVI thrombocytopenia.

Contents

Abbreviations: AS, aortic valve stenosis; BCMD, blood contacting medical devices; DAPT, dual antiplatelet therapy; DIC, disseminated intravascular coagulation; ES, Edwards Sapien; FS, Freedom Solo; F1+2, prothrombin fragments 1+2; GP, platelet glycoprotein; HIT, heparin-induced thrombocytopenia; HCA, homocysteic acid; HMWM-vWF, high molecular weight multimers of von Willebrand factor; IL-6, interleukin-6; MSTP, moderate-to-severe thrombocytopenia; NPSS, nonphysiological shear stress; PAP, plasmin-alfa$_2$-antiplasmin complex; PC, platelet count; PCI, percutaneous coronary interventions; PF-4, platelet factor-4; PMP, platelet-derived microparticles; PS, P-selectin; RANTES, regulated on activation, normal T-cell expressed and secreted; SIRS, systemic inflammatory response; STP, severe thrombocytopenia; TAT, thrombin-anti-thrombin-complex; TAVI, transcatheter aortic valve implantation; TP, thrombocytopenia; TRAP, thrombin receptor-activating protein; TTP, thrombotic thrombocytopenic purpura; UFH, unfractionated heparin; VEGF, vascular endothelial growth factor; vWF, von Willebrand factor; WBC, white blood cells.

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1. Introduction

Transcatheter aortic valve implantation (TAVI) is an alternative procedure for symptomatic patients with aortic valve stenosis (AS) who are not qualified for cardiac surgery [1]. It has been demonstrated that TAVI improves the quality of life and reduces symptoms related to valve disease [2]. Thrombocytopenia (TP) is one of the most common phenomenon observed after TAVI. However, the decrease of platelet count (PC) is not specific to TAVI procedure. It has been observed after coronary artery by-pass grafting, surgical aortic valve replacement (AVR), especially after Freedom Solo (FS) implantation, as well as after percutaneous coronary interventions (PCI) [3–6].

Thrombocytopenia defined as nadir PC $<150{,}000 \times 10^3/\mu L$ concerns almost 70% of patients after TAVI procedure. The exact mechanisms leading to platelet loss after TAVI procedure are less clear and the explanation of this phenomenon is crucial because major TP increases mortality. In addition, TP complicates the perioperative use of antiplatelet agents in these patients.

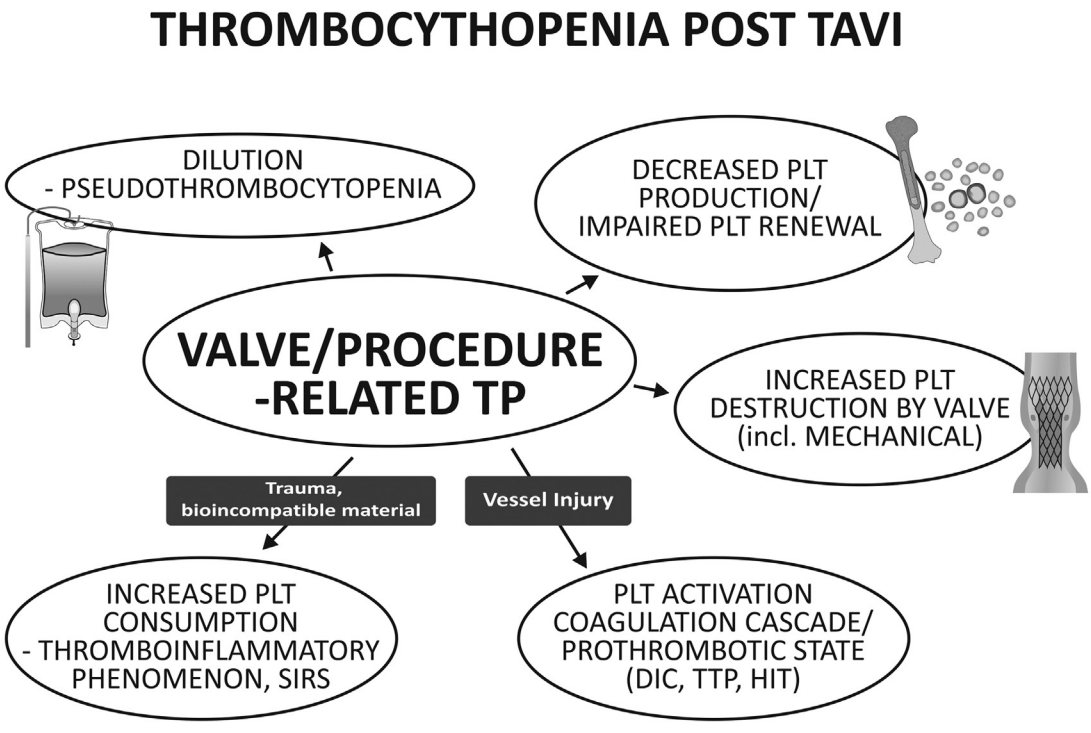
According to some previous studies, this kind of TP, occurring early after TAVI, might be a direct consequence of the procedure alone and related to either procedural or post-procedural adverse events such as vascular complications], bleeding, and multiple blood transfusions [7]. The potential causes of TP might be different. Despite being a less invasive option as compared to AVR, TAVI procedure still requires the use of large catheters, mini-invasive surgical access, high doses of unfractionated heparin (UFH) and, at least in some centers, dual antiplatelet therapy (DAPT). Thus, there are few factors which can lead to TP during TAVI procedure such as inflammatory response as a result of the surgical access, shear stress related to the device implantation, intraoperative administration of iodinated contrast, or heparin and antiplatelet drugs in the postprocedural period. In this respect, it is worth mentioning the differences between “platelet consumption” and “platelet destruction”. Platelet consumption is a broader concept which describes the decrease in PC due to immune or non-immune mechanisms at the valve level or the remote one. The second term relates to the mechanical destruction of platelets at the prosthesis level. On the other hand, TP after TAVI could be caused by insufficient platelet production, platelet dilution or a combination of these factors [8].

We provide the summary of the existing data regarding the possible causes of TP following TAVI procedures (Fig. 1).

2. Review

2.1. The consequences of platelet decrease and the potential strategy to reduce this phenomenon

Thrombocytopenia following TAVI procedures is related to the increased adverse events rate such as death, stroke, acute kidney injury, bleeding, and vascular complications [9–11]. Since TP might be early or late, so may be its consequences. Interestingly, mild TP does not increase the rate of adverse outcomes, and it is mostly resolved at patients’ discharge without association with adverse sequelae [2,3]. A significant decrease of platelet count, so called



DIC – disseminated intravascular coagulation; HIT – heparin-induced thrombocytopenia; PLT – platelets; TAVI – transcatheter aortic valve implantation; TP – thrombocytopenia; TTP – thrombotic thrombocytopenic purpura; SIRS – systemic inflammatory response.

Fig. 1. The causes of thrombocytopenia following TAVI procedures.