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Special Article - Deep Vein Thrombosis

Platelet and Microvesicles Derived in Venous Thromboembolism: Call to Be Aware

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Short Communication

Venous Thromboembolism (VTE) affects a number of individuals it is frequently diagnosed in patients hospitalized for chronic diseases (i.e.cardiac, cerebral, inflammatory disease, trauma, surgical procedures) or having thrombophilia (i.e.factor V Leiden, mutation of pro thrombin G210A, deficiency of protein c and S, anti thrombin 3rd deficiency, mutation of MTHFR enzyme, hyperomocisteinemia) [1-7]. Hypercoagulative condition plays a crucial role for VTE consequently, factors of coagulative cascade are highly considered both as patogenethic tools, and in threatening the VTE. Diversely, there is a few interest on the role played by Platelets (P) in VTE. P is widely accepted as a pivotal mechanism involved in arterial thrombosis in fact anti platelet drugs are assigned both to prevent and to cure arterial thrombosis (Figure 1). However, there are such evidences from studies on pathophisyologic role of P and on possible efficacy of anti platelet drugs in preventing the VTE or in reducing its frequency [8-12]. Acetil salicilic acid was considered for VTE prophylaxis, achieving to the risk reduction of VTE in primary and in secondary prevention. Genetic and functional activation of P, spatial or volumetric modifications were found in specific setting of VTE patients (i.e. cancer, anti phospholipid syndrome, hematologic malignancy etc.) [13-15]. However, role of P in VTE is still debated or no cleared. About P in VTE, I believe as noteworthy to highlight on microvesicles P derived as a specific pathway of clot activation in VTE. Briefly, Microparticles (MPs) are phospholipid vesicles potentially derived from the bloodstream cells (leukocyte, platelet) and from endothelial cells. Tissue Factor (TF) and Phospholipids (Phsp) are two active players leading to procoagulant capability of MPs. In this light it is noteworthy to note that surface of P derived MPs is able to bind coagulative VIII, IX, V, and X activated factors of coagulative cascade [14].

We took attention on MPs in a group of patients affected by Deep Veins Thrombosis (DVT), and we found higher generation of MPs, of phospholipids in DVT patients than in matched group of healthy controls. On the contrary, clot time generation lowered as by demonstrated by prothrombinase induced clotting time. About MPs in VTE we summarize on mixed results. In acute VTE MPs were not found [15] whereas association between MPs with TF

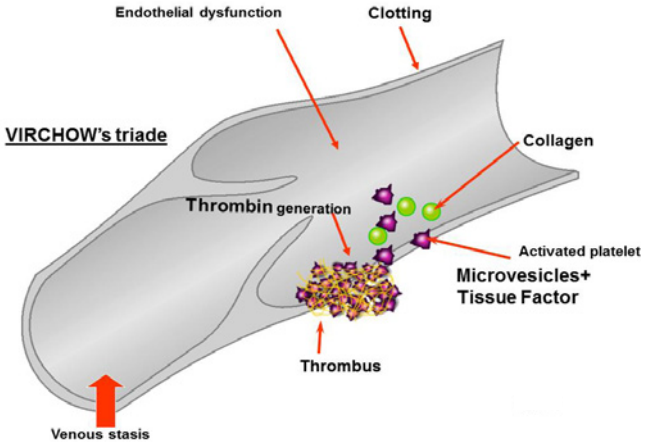

Figure 1: Mechanisms involved in provoking thrombosis. Figure focuses both on Virchow's triade components, on platelet and on platelet derived miscovesicles.

was demonstrated in unprovoked DVT [15,16-18]. Consistent data showed a putative role of Mps in patients with antiphospholipid syndrome, as a key mechanism, and to be able to lead to thrombotic consequences most frequently diagnosed in these patients [19,20]. We agree with opinion from authors that MPs cannot be used as a single lab coagulative marker to screen individuals for risk of VTE [21-23]. Rather, a network of lab markers including MPs, PiCT assay, and PLPs could be added to other known coagulative markers to achieve intriguing targets. Firstly, to elicit pro-thrombotic conditions, furthermore to focus on role played by P and its derived particle as a pathophysiologic mechanism in VTE [24-27]. Now we must to call be aware on role played by MPs and by the P in provoking VTE. It is particularly relevant in patients with cancer because MPs, P are closely related to the TF generation. This last pro thrombotic condition must be considered as crucial to explain high VTE frequency.

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