Editorial

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Idiopathic Venous Thromboembolism (VTE) and its Association to Rare Molecular Clotting Abnormalities

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Editorial

Idiopathic (unprovoked) venous thromboembolism (VTE) may be defined as a VTE that occurs without triggering circumstances predisposing to VTE; yet, predisposing conditions that may induce a VTE are mainly prolonged immobilization, bone fractures, recent surgery, prolonged bed rest, active cancer [1]. So, idiopathic VTE, unassociated with surgery or trauma, is a chronic illness that warrants the implementation of strategies to prevent recurrence over a lifetime.

However according with these suggestions, from a clinical point of we may explain only nearly 25-40% of VTE [1,2]. So in order to explain the cause of the majority of cases of VTE, a pathophysiological association has been found also with other conditions as hormonal treatment, pregnancy, prolonged travel, chronic medical illness (as Chronic Obstructive Pulmonary disease, Inflammatory Bowel Disease and other immunopathological disease and so on) and molecular thrombophilia [3].

In last decades a lot of studies have been performed on inherited thrombophilia and recurrent VTE [4] but without a univocal therapeutic approach [5]. However, molecular thrombophilia is not only inherited thrombophilia but also another great number of molecular alterations that may lead to VTE.

In this group of molecular alterations antiphospholipid syndrome is the most studied and its diagnosis is well focused by ISTH criteria [6]. Other rare molecular alterations associated with VTE are the reduced availability of protein C, protein S and\or antithrombin as showed in their genetic deficiency [7].

Furthermore also anti-inflammatory functions of PC are known and include its ability to inhibit proinflammatory cytokine release in monocytes; this role should be thorough considered in clinical practice because it may change its activity during any pathological stress.

Furthermore also an impaired function of protein C system may lead to thrombosis and VTE by different mechanisms as impaired cleavage of activated clotting factor V and VIII or induced hypofibrinolysis [8]. This pathophysiological way is particularly relevant during infection or sepsis but it may be altered also in other conditions. Beside of inherited deficiency of clotting inhibitors as protein C, protein S and antithrombin, several illness have been associated to acquired reduction of these proteins as liver disease, cancer, nephrotic syndrome, disseminated intravascular coagulation, chronic use of such drugs as anti-vitamin K antagonist or oral contraceptives [9].

In these issues Scudiero et al. [10] showed an impaired function f protein C system during prolonged physical exercise [10]. In particular Authors describe a clear reduced action of protein C in last phases of strenuous physical activity and during recovery and rest. These data may so be implicated from several clinical points of view in particular for athletes and daily users of body gym to prevent vascular diseases as VTE.

So questions for daily clinical practice are the following, how intense should be the research of an apparent unprovoked VTE, in particular concerning clotting abnormalities? When he better moment to test levels of protein C and its activity? Should be considered primary and secondary pharmacological thromboprophylaxis only for main prothrombotic conditions or also for others? How long should be an antithrombotic treatment after a diagnosis of not congenital protein C system dysfunction?

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