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Elevated coagulation FIX and risk of thrombosis development

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Coagulation involves the regulated sequence of proteolytic activation of a series of zymogens to achieve an appropriate and timely hemostasis in an injured vessel in an environment that favors an anticoagulant state. Alteration of hemostatic balance between the prothrombotic and antithrombotic factors can result in insufficient inhibition of coagulation thrombosis or bleeding due to excessive antithrombotic treatment. Fibrin is the key component of thrombi and anticoagulant drugs that reduce thrombin formation which are effective in both prevention and treatment of thrombosis. Therefore, an increased circulating level of coagulation factor is a must for treatment mechanisms of both venous and arterial thrombosis. The existing anticoagulants may have only limited effects due to their modest therapeutic benefits, increased bleeding risks, narrow clinical applications and drug-induced thrombophilia. However, some new oral anticoagulants, when administered optimally, are associated with significant anti-ischemic benefits and lower bleeding risk when compared with heparin and vitamin K antagonists. Since factor IX (FIX) plays a key role in tissue factor-mediated thrombin production, it may represent a promising target for drug development. This review aims to summarize the current data for FIX and its role in the development of thrombosis (although thrombosis is a platelet-centric process and FIX may not have any direct and specific effect on platelets).

Biography

S M AlDallal has completed her PhD at University of Manchester, UK. She has published 14 articles in reputed journals and has experience in Haematology & Blood Bank Laboratory. She has also published several papers in national and multi-national journals. She is the senior of training courses of haematology technicians at general hospital laboratory in Kuwait.

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