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A Narrative Assess of Autoimmune Diseases Affecting Hemostasis

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Introduction

Hemostasis is a homeostatic mechanism that seeks to balance pro- and anti-coagulant forces in order to maintain blood flow within the circulation. Simply put, an excess of procoagulant forces can cause thrombosis, while an excess of anticoagulant forces can cause bleeding. A wide range of congenital disorders are associated with bleeding or thrombosis. Furthermore, there is a wide range of autoimmune diseases that can result in either bleeding or thrombosis. Autoantibodies against clotting factors, for example, can cause bleeding, the most common of which is acquired haemophilia A. Antiphospholipid syndrome, a condition caused by autoimmune-mediated antibodies against phospholipids, is another example of a prothrombotic milieu. Furthermore, there are a variety of autoimmunity-promoting environments that can result in a variety of antibodies.

Hemostasis is a homeostatic physiological mechanism that aims to balance procoagulant and anticoagulant forces in order to keep blood flowing through the circulation. When procoagulant forces are in excess, thrombosis can occur; conversely, anticoagulant forces in excess can result in pathological bleeding. Furthermore, natural procoagulants and anticoagulants interact to maintain normal physiological hemostasis. Congenital disorders associated with bleeding or thrombosis include haemophilia and von Willebrand disease, as well as thrombophilic conditions caused by a lack of natural anticoagulants or the presence of clotting factor variants associated with thrombosis. Furthermore, there are numerous autoimmune diseases that can cause hemostasis dysfunction and thus either a bleeding or thrombosis phenotype [1].

Description

Adhesive plasma protein that otherwise promotes platelet plug formation by facilitating attachment and immobilisation of blood platelets to sites of vascular injury. Alternatively, auto-immune-mediated antibody generation against various hemostasis components can promote a prothrombotic environment. Antiphospholipid syndrome, for example, is caused by antibodies produced against phospholipids. Additional prothrombotic conditions, such as heparin-induced thrombotic thrombocytopenia and vaccine-induced thrombotic thrombocytopenia, can result from the generation of antibodies against platelet factor 4. This narrative review discusses the interaction between various autoimmune diseases and hemostasis, which can result in acquired hemostasis disorders and either bleeding or thrombosis [2].

Physiological hemostasis maintains blood flow in the circulation, preventing bleeding or thrombosis. Hemostasis is normally characterised by a balance of procoagulant and anticoagulant forces, indicating an equilibrium of procoagulant and anticoagulant blood components. Another popular way to look at disrupted hemostasis leading to a procoagulant state and possible thrombosis is through the Virchow's Triad, in which intravascular vessel wall damage, blood flow stasis, and/or the presence of hypercoagulability are key components. Hemostasis actually reflects the interaction of multiple processes, which can conventionally and conveniently be separated into 'primary hemostasis', 'secondary hemostasis' and 'fibrinolysis'. However, while this division is useful for laboratory-aided investigations of hemostasis dysfunction, it does not accurately represent the in vivo hemostatic system, in which all of these processes interact at multiple points.

Autoimmune disorders that cause bleeding are characterised by the production of autoantibodies directed against procoagulant components of hemostasis. This is not typical of the development of alloantibodies in patients who are deficient in certain coagulation factors and receive replacement therapy. Alloantibodies will develop in a large proportion of haemophilia A and B patients at some point during their treatment because the coagulation proteins they are unable to produce naturally may be perceived as 'foreign' by the immune system when provided as replacement products. Autoantibodies can also occur spontaneously in patients who do not have natural deficiencies and instead acquire a deficiency as a result of a disease process or even immune system dysregulation.In these settings, the development of autoantibodies against naturally occurring proteins is triggered by certain event (usually encompassing a strong stimulation of the immune system by infections, inflammation, pathologies like cancer or certain therapies) at a certain point of the patient's life [3].

Anti-FVIII autoantibodies are produced in a number of autoimmune conditions, including systemic lupus erythematosus, rheumatoid arthritis, multiple sclerosis, Sjogren syndrome, and temporal arteritis. Inflammatory bowel disease, infections, diabetes, hepatitis, respiratory or dermatological diseases, blood (haematological) malignancies, or certain solid cancers are all conditions that can lead to the development of these inhibitors. An association has also been reported with the use of certain drugs, such as penicillin or interferon, and with pregnancy, primarily in the post-partum period. Affected people develop complications as a result of abnormal, uncontrolled bleeding into the muscles, skin, and soft tissue, which can occur spontaneously, during surgery, or as a result of trauma. Nosebleeds, bruising throughout the body, solid swellings of congealed blood, blood in the urine, and gastrointestinal or urogenital symptoms are all possible.

As a result, a complex interplay is at work. For example, everyone is at risk of COVID-19, and the majority of people have been immunised against it. This general population includes both healthy and sick people, as well as cancer, APS, and other pathology patients. As a result, a cancer patient may be exposed to COVID-19, which may increase their risk of thrombosis or the development of an autoimmune condition. Here, an unfortunate synergy may occur, resulting in a pathology that is greater than the sum of its parts.

Although acquired haemophilia A is the most common acquired factor defect, antibodies can develop to all other clotting factors, including the most well-known. Most historical causes of acquired deficiency were caused by the use of bovine thrombin and fibrinogen in some surgeries to help seal wounds. Unfortunately, the bovine thrombin used was frequently 'contaminated' with trace amounts of bovine, resulting in the development of anti-bovine FV antibodies that cross-reacted with human. In Japan, where a national

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Collaborative Research Group operates, up to 200 patients with autoimmune FV deficiency have been identified. These researchers discovered a relatively mild type of bleeding diathesis that was associated with a lower mortality rate than previously reported in the literature for both autoimmune conditions [4,5].

Conclusion

Immune, complement, and hemostasis pathways are all intricately linked. Dysfunction in one of the pathways may have an effect on another. The current narrative review has concentrated on the relationship between autoimmunity and hemostasis dysfunction. Depending on the type of antibodies produced, hemostasis dysfunction can manifest as either bleeding or thrombosis. Antibodies can be produced against procoagulant clotting proteins, either clearing them from circulation or inhibiting their function, resulting in bleeding diathesis, as seen in acquired haemophilia A. Alternatively, antibodies may be generated against hemostasis pathway components that act to moderate procoagulant activity. Antibodies against this protein, for example, can cause its clearance or reduce its activity.

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Conflict of Interest

There are no conflicts of interest by author.

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