Advancements in management of different types of thrombocytopenia.

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Introduction

Numerous inherited and acquired conditions can lead to thrombocytopenia. Reduced production of bone marrow, increased consumption, increased destruction, splenic sequestration, or a combination of these factors may all contribute to thrombocytopenia. We have concentrated on a few of the severe acquired causes of thrombocytopenia in this review. Over the past five years, tremendous progress has been made in our knowledge of the aetiology, diagnostic procedures, and therapeutic approaches for immune thrombocytopenia, heparin-induced thrombocytopenia, thrombotic thrombocytopenic purpura, and atypical hemolytic uremic syndrome.

Thrombocytopenia, defined as a platelet count below 150 10 9/L, can have a variety of reasons, which can be broadly categorised as congenital and acquired. Thrombocytopenia after acquisition may be immune or non-immune. Increased sequestration and/or destruction in the periphery, as well as a combination of decreased production and sequestration, may all contribute to thrombocytopenia. Reviewing the peripheral blood smear is one of the first procedures in the examination of thrombocytopenia in order to rule out pseudo-thrombocytopenia caused by platelet clumping When coupled with a complete blood count, a thorough patient history, and a physical examination, the peripheral blood smear may also point to further reasons of thrombocytopenia [1].

One further characteristic on the peripheral blood smear that aids in determining the cause of thrombocytopenia is platelet size. Other characteristics include the presence of schistocytes, polychromasia, or spherocytes. A significant underlying medical disease, such as thrombotic thrombocytopenic purpura (TTP) or heparin-induced thrombocytopenia, could be presaged by thrombocytopenia (HIT). Therefore, it is crucial that the treating physician assess thrombocytopenia as soon as possible to avoid delaying the therapy for some dangerous illnesses. The pathogenesis and treatment of some of the major acquired causes of thrombocytopenia that are encountered in the hospital context will be the primary emphasis of the current review's recent revisions.

Immune thrombocytopenia

Most immune thrombocytopenia (ITP) patients either have minimal muco-cutaneous bleeding or are asymptomatic when they first show. Since platelet autoantibodies cannot be reliably detected by a diagnostic test, ITP continues to be a diagnosis of exclusion. However, in addition to being therapeutic, a quick response to steroids or intravenous immunoglobulin (IVIG) may also help with ITP diagnosis. In the pathophysiology of ITP, platelet autoantibodies, T cell-mediated autoimmunity, or both, play a role in platelet destruction and decreased platelet production.

Despite the fact that platelet autoantibodies are not employed in the diagnosis of ITP, current research indicates that the absence of platelet-bound antibodies may be a predictor of rituximab therapy resistance in ITP patients. In individuals with comparable platelet counts, the bleeding phenomenology varies widely, and it may be influenced by things like concurrent drugs, patient age, and platelet reactivity. The risk of bleeding may be reduced in ITP patients with lower platelet counts and higher platelet reactivity, according to some research. The most terrifying ITP complication is intracranial haemorrhage, which is likely to occur if there has been extensive bleeding from other places in the past [2].

Rituximab, a monoclonal anti-CD20 antibody, has a 50–60% initial response rate, but only 20% of patients still have the response after five years. There was no difference in the percentage of patients in complete remission between the two groups at 18 months, according to a new randomised control trial contrasting rituximab with a placebo. In a first-line scenario, combining rituximab with one to three cycles of high-dose dexamethasone may lead to higher response rates than dexamethasone alone.

Heparin-induced thrombocytopenia

Unfractionated heparin (UFH), despite the widespread use of direct oral anticoagulants (DOACs) and other parenteral anticoagulants, is still crucial to the care of hospitalised patients. Therefore, for the treating physician, knowing the pathogenesis and management of HIT is still highly important. HIT is a disorder with several paradoxes. In this condition, thrombocytopenia is linked to thrombosis rather than bleeding and an anticoagulant causes thrombosis. Contrary to other thrombocytopenias, HIT increases the risk of clotting while receiving platelet transfusions. Contrary to other drug-induced thrombocytopenias, acute HIT cannot be treated with vitamin K antagonist anticoagulants, and simply ceasing the offending medication (heparin) is not sufficient [3].

Platelet factor 4 (PF4)-heparin or endogenous glycosaminoglycan complexes produce HIT antibodies. These IgG antibodies, in particular, have a subgroup that can cross-

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link the Fc receptor IIA (FcR IIA), activating platelets and monocytes and producing consumptive thrombocytopenia and venous and/or arterial thrombosis.

In patients undergoing orthopaedic surgeries, spontaneous HIT commonly develops without any heparin exposure.

Thrombotic thrombocytopenic purpura

Multiple disease states share microangiopathic hemolytic anaemia (MAHA) and thrombocytopenia. Since TTP is caused by a severe (10%) ADAMTS13 (a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13 or von Willebrand cleaving protease) deficiency, it is the easiest of these to diagnose [4].

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