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Classification of bleeding disorders pdf

Skip to Main Content Skip Nav Destination Inherited Bleeding Disorders| March 26, 2015 Skip to Main Content Skip Nav Destination Condition in which the blood's ability to coagulate (form clots) is impaired Medical conditionCoagulopathyOther namesBleeding disorderPlateletsSpecialtyHematology Coagulopathy (also called a bleeding disorder) is a condition in which the blood's ability to coagulate (form clots) is impaired.[1] This condition can cause a tendency toward prolonged or excessive bleeding (bleeding diathesis), which may occur spontaneously or following an injury or medical and dental procedures.[citation needed] Of note, coagulopathies are sometimes erroneously referred to as "clotting disorders"; a clotting disorder is a predisposition to clot formation (thrombus), also known as a hypercoagulable state or thrombophilia. Signs and symptoms Coagulopathy may cause uncontrolled internal or external bleeding. Left untreated, uncontrolled bleeding may cause damage to joints, muscles, or internal organs and may be life-threatening. People should seek immediate medical care for serious symptoms, including heavy external bleeding, blood in the urine or stool, double vision, severe head or neck pain, repeated vomiting, difficulty walking, convulsions, or seizures. They should seek prompt medical care if they experience mild but unstoppable external bleeding or joint swelling and stiffness.[citation needed] Mechanism The normal clotting process depends on the interplay of various proteins in the blood. Coagulopathy may be caused by reduced levels or absence of blood-clotting proteins, known as clotting factors or coagulation factors. Genetic disorders, such as hemophilia and Von Willebrand disease, can cause a reduction in clotting factors.[2] Anticoagulants such as warfarin will also prevent clots from forming properly.[2] Coagulopathy may also occur as a result of dysfunction or reduced levels of platelets (small disk-shaped bodies in the bloodstream that aid in the clotting process).[citation needed] Acute traumatic coagulopathy In 2003, Karim Brohi, Professor of Trauma Sciences at Queen Mary University of London, first introduced the term Acute Traumatic Coagulopathy (ATC),[3] establishing that coagulopathy induced by trauma results in: more severe bleeding multi-organ failure high mortality Treatment If someone has coagulopathy, their health care provider may help them manage their symptoms with medications or replacement therapy. In replacement therapy, the reduced or absent clotting factors are replaced with proteins derived from human blood or created in the laboratory. This therapy may be given either to treat bleeding that has already begun or to prevent bleeding from occurring.[citation needed] Critical care One area of treatment is managing people with major bleeding in a critical setting, like an emergency department.[1] In these situations, the common treatment is transfusing a combination of red cells with one of the following options:[citation needed] Blood plasma Prothrombin complex concentrate, factor XIII, and fibrinogen Fibrinogen with tranexamic acid The use of tranexamic acid is the only option that is currently supported by a large, randomized, controlled clinical trial, and is given to people with major bleeding after trauma.[4] There are several possible risks to treating coagulopathies, such as transfusion-related acute lung injury, acute respiratory distress syndrome, multiple organ dysfunction syndrome, major hemorrhage, and venous thromboembolism. Laboratory findings in various platelet and coagulation disorders[citation needed] Condition Prothrombin time Partial thromboplastin time Bleeding time Platelet count Vitamin K deficiency or warfarin Prolonged Normal or mildly prolonged Unaffected Unaffected Disseminated intravascular coagulation Prolonged Prolonged Prolonged Decreased Von Willebrand disease Unaffected Prolonged or unaffected Prolonged Unaffected Hemophilia Unaffected Prolonged Unaffected Unaffected Aspirin Unaffected Unaffected Prolonged Unaffected Thrombocytopenia Unaffected Unaffected Prolonged Decreased Liver failure, early Prolonged Unaffected Unaffected Unaffected Liver failure, end-stage Prolonged Prolonged Prolonged Decreased Uremia Unaffected Unaffected Prolonged Unaffected Congenital afibrinogenemia Prolonged Prolonged Prolonged Unaffected Factor V deficiency Prolonged Prolonged Unaffected Unaffected Factor X deficiency as seen in amyloid purpura Prolonged Prolonged Unaffected Unaffected Glanzmann's thrombasthenia Unaffected Unaffected Prolonged Unaffected Bernard–Soulier syndrome Unaffected Unaffected Prolonged Decreased or unaffected Factor XII deficiency Unaffected Prolonged Unaffected Unaffected C1INH deficiency Unaffected Shortened Unaffected Unaffected See also Trauma triad of death Hypocoagulability Hypercoagulability References ^ a b Hunt, Beverley J. 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