Thrombotic Thrombocytopenia Purpura

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Hemolytic Anemia

Fever

signs, symptoms, and proper treatment. In TTP, clinicians must be informed on diagnosis and prompt treatment are crucial with these symptoms may be cared for in hospital departments and subsequently admitted to intensive care. Treatment for TTP is plasma exchange therapy and can be life-saving in some cases.

Platelets play a significant role in the formation of clots and the prevention of bleeding. The normal platelet count falls within the range of 150,000 to 400,000/mm³ (Korhonen, Länsio, & Lynch, 2011). When endovascular tissue is injured by an inflammatory process or traumatic damage, the endothelium loses its integrity. Platelets adhere to the intimal surface via binding with vWF, present in both endothelial and platelet membranes. The interaction of vWF with platelet glycoprotein Ib/IX results in the release of vWF multimers from the endothelial cell which aggregate with platelets.

The decreases in ADAMTS 13 deficiency can either be congenital, although one recent retrospective study concluded that patients who received platelet transfusions for treatment of TTP however, the ADAMTS 13 deficiency that causes TTP, can either be congenital, or acquired as a result of autoantibodies, drugs, or malignancies (Strecker, Trachtman, & Mark Andrew, 2011). TTP can occur spontaneously or trigged by an infection, pregnancy, or other stimuli of autoantibodies and as many as 30% of those affected report a family history (Rosse, 2014). Most commonly occurring in adults, primarily women, patients typically present with thrombocytopenia (with or without apheresis bleeding), hemolytic anemia, he~t, altered mental status or headache, neurological impairment or chest pain (Kappler, Ronan-Bentle, & Graham, 2014). TTP belongs to a group of disorders called thrombotic microangiopathies. Also included in this group are HUS and HELLP syndrome (Kappler et al., 2014). TTP is a systemic disease characterized by the formation of small intravascular clots within the microcirculation of the body and results in organ damage. TTP leads to organ damage and microvascular thrombosis (Kappler et al., 2014). Abdominal pain, chest pain, and neurological symptoms may also present as a result of organ damage and microvascular thrombosis.

Clinical presentation of TTP includes thrombocytopenia, hemolytic anemia, fever, altered mental status, headache, and neurological symptoms. Platelet transfusions for treatment of TTP however, the ADAMTS 13 deficiency that causes TTP, can either be congenital, or acquired as a result of autoantibodies, drugs, or malignancies (Strecker, Trachtman, & Mark Andrew, 2011). TTP can occur spontaneously or trigged by an infection, pregnancy, or other stimuli of autoantibodies and as many as 30% of those affected report a family history (Rosse, 2014). Most commonly occurring in adults, primarily women, patients typically present with thrombocytopenia (with or without apheresis bleeding), hemolytic anemia, he~t, altered mental status or headache, neurological impairment or chest pain (Kappler, Ronan-Bentle, & Graham, 2014). TTP belongs to a group of disorders called thrombotic microangiopathies. Also included in this group are HUS and HELLP syndrome (Kappler et al., 2014). TTP is a systemic disease characterized by the formation of small intravascular clots within the microcirculation of the body and results in organ damage. TTP leads to organ damage and microvascular thrombosis (Kappler et al., 2014). Abdominal pain, chest pain, and neurological symptoms may also present as a result of organ damage and microvascular thrombosis.

Plates are potent reactive oxygen species, which is important for the regulation of the inflammatory response.

In conclusion, TTP is a serious and potentially life-threatening disorder that requires prompt recognition and treatment. Delay in diagnosing and treating TTP can be fatal. Early diagnosis and prompt treatment of TTP are crucial in improving patient outcomes. Close monitoring of neurologic, cardiac, and renal complications as well as bleeding complications. TTP is a systemic disease characterized by the formation of small intravascular clots within the microcirculation of the body and results in organ damage. TTP leads to organ damage and microvascular thrombosis (Kappler et al., 2014). Abdominal pain, chest pain, and neurological symptoms may also present as a result of organ damage and microvascular thrombosis.

Significance of Pathophysiology

The acute state of widespread microvascular thrombosis and thrombocytopenia can lead to significant injury and precipitate poor outcomes for patients diagnosed with TTP. Patients are at risk for myocardial infarctions, cerebral vascular accidents, kidney failure, and other organ failure as a result of microvascular thrombosis and ischemia. The thrombi that accumulate in TTP are mostly platelets and do not include fibrin which differentiates TTP from other microangiopathies. In TTP, however, the ADAMTS 13 deficiency that causes TTP is not associated with fibrin deposition.

Nursing care must be directed at the prevention of bleeding, treatment of infection, and supportive care. Careful monitoring of neurologic, cardiac and renal function is important and even slight changes in assessment criteria require prompt evaluation.

Thrombotic Thrombocytopenia Purpura

Thrombotic Thrombocytopenia Purpura (TTP) is a rare but potentially fatal condition that occurs as an result of decreased levels of ADAMTS 13, a cleavage protein for von Willebrand factor (vWF), which causes platelet aggregation and thrombosis. The decreases in ADAMTS 13 deficiency can either be congenital, or acquired as a result of autoantibodies, drugs, or malignancies (Strecker, Trachtman, & Mark Andrew, 2011). TTP can occur spontaneously or trigged by an infection, pregnancy, or other stimuli of autoantibodies and as many as 30% of those affected report a family history (Rosse, 2014). Most commonly occurring in adults, primarily women, patients typically present with thrombocytopenia (with or without apheresis bleeding), hemolytic anemia, he~t, altered mental status or headache, neurological impairment or chest pain (Kappler, Ronan-Bentle, & Graham, 2014). TTP belongs to a group of disorders called thrombotic microangiopathies. Also included in this group are HUS and HELLP syndrome (Kappler et al., 2014). TTP is a systemic disease characterized by the formation of small intravascular clots within the microcirculation of the body and results in organ damage. TTP leads to organ damage and microvascular thrombosis (Kappler et al., 2014). Abdominal pain, chest pain, and neurological symptoms may also present as a result of organ damage and microvascular thrombosis.