Thrombotic Thrombocytopenia Purpura

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Hemolytic Anemia

Mortality rates from TTP can be as high as 50% (Kappler et al., 2014). TTP can be triggered by an infection, pregnancy or medications, and can be induced by specific triggers such as streptococcal infections or chemotherapy. Patients with TTP typically present with thrombocytopenia (Rosove, 2014). Most commonly occurring in pregnant women, patients with HIV infection, and can be induced by specific triggers such as streptococcal infections or chemotherapy.

Thrombocytopenia Purpura

Patients presenting with TTP can exhibit many different symptoms due to the pathological pressures of TTP. Symptoms include microangiopathic hemolytic anemia, renal impairment, neurological impairment, and fever are seen, although not necessarily all together (Strecker-McGraw & Mark Andrew, 2011). Blood transfusions may be required for these patients, and severe anemia may result in death (Kappler et al., 2014). The reticulocyte count may be elevated and schistocytes can be seen on peripheral blood film. Hemolytic anemia will cause a decreased haptoglobin, elevated reticulocyte count, elevated indirect bilirubin from intravascular hemolysis. Coagulation studies will be normal, differentiating TTP from other causes of hemolysis. Coagulation studies will be normal, differentiating TTP from other causes of hemolysis.

The acute state of widespread microvascular thrombi and thrombocytopenia can lead to significant injury and precipitate poor outcomes for patients diagnosed with TTP. Patients with TTP typically present with thrombocytopenia (with or without a spleen) and microangiopathic hemolytic anemia without other explanations (Strecker et al., 2014).

Plateslets 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 (Konkle lauer, Langos, Jameson, & Lucas, 2013). When endothelial injury is present, platelet adherence and activation (resulting in aggregation) can block blood vessels, causing clot formation. In TTP, platelets adhere to the intimal surface via binding with vWF, present in both endothelial tissue and plasma, and intercellular signals leading to activation of platelet glycoprotein IIb/IIIa receptor and platelet aggregation (Korkele et al., 2013). Usually vWF is involved in smaller proteins by a plasma metalloprotease called a disintegrin and metalloprotease domain containing type 1 metalloproteinase with a thrombospondin type 1 domain (ADAMTS 13). In TTP, however, the ADAMTS 13 concentration is very low either due to congenital abnormalities or acquired autoantibodies, which leaves large unfragmented vWF that can block blood vessels and can result in organ damage and can result in neurological, cardiac, and renal complications as well as other complications. TTP is more common in pregnant women, patients with HIV infection, and can be induced by certain medications that patients who have undergone an organ or stem cell transplant (Konkle et al., 2015, Rosove, 2014).

Significance of Pathophysiology

The acute state of widespread microvascular thromboemboli 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 patients with ADAMTS 13 deficiency. In TTP, platelets are required for clotting and can contribute to the development of microangiopathic hemolytic anemia without red cell destruction (Strecker-McGraw & Mark Andrew, 2011). Therapy with plasma exchange is seen as possible as recommended to replace the ADAMTS 13 and reduce the antibodies associated with its destruction (Kappler et al., 2014). Platelet transfusions in the setting of thrombocytopenia in patients with TTP is generally contraindicated as and has been historically associated with an increased mortality, although one recent retrospective study concluded that patients who received platelet transfusions did not have an increased risk of death as a result of the transfusions (Trachtman, Liu, & Grossman, 2015).

Implications for Nursing Care

Nursing care must be directed at frequent assessment of patient symptoms, prevention of bleeding, infection, and supportive care. Careful monitoring of neurologic, cardiac and renal function is important as well as even slight changes in assessment criteria are significant. Pharmacologic therapy may include corticosteroids, immune suppressive medications, and blood pressure regulators. Refractory patients may receive treatment with dual antiplatelet therapy. Blood transfusions may be indicated for refractory patients. Platelet products for bleeding may be used. Central pain management is vital for pain management and associated with microvascular thromboemboli is important.

Signs and Symptoms

Anemia

Thrombocytopenia

Purpura

Examples of Purpura

Signs and Symptoms

Underlying Pathophysiology

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