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Thrombotic Thrombocytopenia Purpura

Ann Oliva
Otterbein University, ann.oliva@otterbein.edu

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Hemolytic Anemia

Fever

90% (Kappler et al., 2014).

Without prompt recognition and treatment, signs, symptoms, and proper treatment. Many areas of practice and because correct status or headaches, renal impairment, and hemolytic anemia, fever, altered mental

in adults, primarily women, patients

infection, pregnancy, or other stimulus of autoantibodies, drugs, or malignancies count (HELLP) syndrome (Kappler et al.,

Bentle, & Graham, 2014). TTP belongs to a

hemolytic anemia, and possible neurologic damage, along with thrombocytopenia, that occurs as result of decreased levels of TTP is a rare but potentially fatal condition

problems that TTP patients can present

in the inpatient or the critical care unit. The

Hemolytic Uremic Syndrome (HUS), and pregnancy-induced hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome (Kappler et al.,

The presenting signs and symptoms can vary in patients with TTP

basic metabolic panel but is typically more severe in HUS (Kappler et al.,

thrombocytopenia can occur as well as jaundice or dark urine related to organ failure as a result of microvascular thrombi and ischemia. The thrombi that

metalloproteinase with a thrombospondin type 1

llb/llla receptor and platelet aggregation (Konkle et al.,

normal platelet count falls within

TTP. The ADAMTS13 cleaving protease for von

The ADAMTS13 concentration is very low even due to congenital abnormalities or acquired autoantibodies, which leave an abnormal amount of ADAMTS13 in the blood. The lack of ADAMTS13 can result in widespread

the ADAMTS13-13 concentration is important.

Implications for Nursing Care

Nursing care must be directed at infection, pain control and symptom management, and close monitoring for renal function as well as pain patterns

thrombotic thrombocytopenia 

Significance of Pathophysiology

TTP is a challenging and dangerous disorder that requires prompt recognition and treatment. The recent discovery of the ADAMTS13 deficiency and increased availability of plasma exchange therapy has greatly increased an individual’s chance of surviving a TTP diagnosis. Treatment differences and drug interactions in the microangiopathy disorders can cause significant morbidity and mortality. Without prompt recognition and treatment for these patients, diagnosing and treating TTP would be a significant responsibility, especially because successful identification and treatment of a disorder like TTP can save a patient’s life.

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Signs and Symptoms

Introduction

Painful patients with Thrombotic Thrombocytopenic Purpura, or TTP, are often times seen in the emergency department and subsequently admitted to the inpatient or the critical care unit. The problems that TTP patients can present with vary greatly and atitude nursing assessment plus knowledge of the pathophysiology behind the diagnosis is vital to deliver excellent nursing care. The diagnosis of Thrombotic Thrombocytopenic Purpura or TTP is a rare but potentially fatal condition that occurs as result of decreased levels of ADAMTS13, a cleaving protease for von Willebrand factor (vWF), which causes platelet aggregation and microvascular thrombotic and subsequent and organ damage, along with thrombocytopenia, hemolytic anemia, and possible neurologic and renal impairment (Kappler, Ronan- Bentle, & Graham, 2014). TTP belongs to a group of disorders called thrombotic microangiopathies. Also included in this group are Hemolytic Uremic Syndrome (HUS), and pregnancy-induced hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome (Kappler et al., 2014). The decreased in ADAMTS-13 causing TTP, can either be congenital, idiopathic, or acquired as result of autoantibodies, drugs, or malignancies (Trachtman, 2013). TTP can occur spontaneously or be triggered by an infection, pregnancy, or of other stimuli of autoantibodies and as many as 30% of those affected relapse after a normalty (Rosove, 2014). Most commonly occurring in adults, primarily women, patients typically present with thrombocytopenia (with or without a of bleeding, hemolytic anemia, fever, altered mental status or headache, renal impairment, and abdominal or chest pain) (Kappler et al., 2014). Treatment for TTP is plasma exchange therapy, with the goal of replacing the missing ADAMTS-13 and removing the contributing antibody (Kappler et al., 2014). Patients presenting with these symptoms may be cared for in more severe cases of practice and because correct diagnosis and prompt treatment are crucial in TTP, staff must be informed in its signs, symptoms, and proper treatment. Without prompt recognition and treatment, mortality rates from TTP can be as high as 90% (Kappler et al., 2014).

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 thrombosis that accumulate in TTP are mostly platelets and do not include fibrin which differentiates TTP from Disseminated Intravascular Coagulation (DIC). Without prompt recognition and treatment, mortality rates from TTP can be as high as 90% (Kappler 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/mmc (Konkle/Hauser, Longo, Jameson, & Linsalata, 2015). When endothelial tissue is injured platelets adhere to the residual surface via von willebrand factor (vWF) and the platelet sticky site (GPIIb/IIIa receptor) is activated leading to aggregation of platelets, formation of a platelet plug and hemostasis (Korkele et al., 2015). Usually vWF is cleaved into smaller proteins by a metalloproteinase called a disintegrin and metalloprotease domain containing type 1 metalloproteinase (ADAMTS13). But in TTP, the ADAMTS13 concentration is very low due to congenital abnormalities or acquired autoantibodies, which leave an abnormal amount of ADAMTS13 in the blood. The lack of ADAMTS13 can result in widespread platelets leading to organ damage and can result in neurologic, cardiac, and renal complications as well as TTP. TTP is more common in pregnant women, patients with HIV, and cancer, can be induced by certain drugs. In patients who have undergone an organ or stem cell transplant (Konkle et al., 2015, Rosove, 2014)