Coma And Thrombocytopenia In Kenya: Case Report

M Gelchsheimer, O Seiler, O Wenker

Citation

Abstract

ANSWERS TO THE QUESTIONS AND DISCUSSION

1. WHAT COULD BE THE FINAL DIAGNOSIS?
Thrombotic thrombocytopenic purpura (TTP) and immune thrombocytopenic purpura (ITP) are uncommon multisystem disorders with increased platelet destruction. In ITP, an autoimmune antibody (usually IgG) arises and interacts with the patient's own platelets. TTP is sometimes associated with predisposing conditions such as pregnancy, cancer, exposure to certain drugs, bone marrow transplantation and HIV-1 infection (1). It is a life-threatening multisystem disease characterized by thrombocytopenia, microangiopathic hemolytic anemia, fluctuating neurological signs, progressive renal failure, and fever (2, 3). Depressed consciousness can rapidly progress to coma and generalized seizures. In both, TTP and ITP, a main symptom is bleeding, which can include bruising ("ecchymosis") and tiny red dots on the skin or mucous membranes ("petechiae"). In some instances bleeding from the nose, gums, digestive or urinary tracts may also occur. Rarely, bleeding within the brain occurs.

2. WHAT IS THE CAUSE OF SUCH A DISEASE?
Although the cause of thrombotic thrombocytopenic purpura is unknown, many drugs, including penicillin, antineoplastic chemotherapy agents, and oral contraceptives, have been associated with the syndrome (4). Quinine and quinine-containing beverages such as tonic water are other classic examples of drug-induced purpura. An abnormal interaction between the vascular endothelium and platelets which occurs in certain organs leads to thrombosis, endothelial proliferation, minimal inflammation and microangiopathic hemolysis (5). Recent studies suggest that endothelial cell perturbation and apoptosis caused by an as yet unknown plasma factor(s) may lead to the release of abnormal von Willebrand factor which facilitates the deposition of platelet microthrombi. (1). Histologically, there are widespread micro-thrombi and reactive endothelial proliferation. Thrombi consist of masses of platelets and entrapping erythrocytes and fibrin.

Its frequency is estimated to be only 3.7 cases per year per 1 million persons, with mortality rates ranging from 10% to 20% (4). Thrombocytopenic purpura is considered chronic when it has lasted more than 6 months. The onset of illness may be at any age. Adults more often have the chronic disorder and females are affected two to three times more than males. The onset of illness may be at any age. In most cases remissions can be attained, and cures are now common—although approximately one-half of the patients will relapse. While relapses are usually milder, they still carry a significant mortality and preventive therapies are not always effective (6). Idiopathic autoimmune thrombocytopenic purpura during childhood is usually self-limited.

3. WHAT ARE THE TREATMENT OPTIONS FOR THIS DISEASE?
Exchange transfusions of plasma or plasma-cryosupernatant remain the cornerstone of the treatment of TTP/ITP along with corticosteroids, platelet inhibitor drugs, vincristine and splenectomy.

If the doctor thinks a drug is the cause of the thrombocytopenia, standard treatment involves discontinuing the drug’s use. Infection, if present, is treated vigorously since control of the infection may result in a return of the platelet count to normal. The treatment of thrombocytopenic purpura is determined by the severity of the symptoms (6). In some cases, no therapy is needed. In most cases, drugs that alter the immune system’s attack on the platelet are prescribed. These include corticosteroids (i.e., prednisone) and/or intravenous infusions of immune
Thrombotic thrombocytopenic purpura (TTP) is a rare disorder characterized by microangiopathic hemolytic anemia and thrombocytopenia. The diagnosis of TTP is based on evidence of microvascular occlusion with schistocytes on peripheral blood smear and low platelet count. A complete blood count should be done for diagnosis. A low platelet count will establish thrombocytopenia as the cause of purpura. The presence of schistocytes or fragmentocytes may confirm the diagnosis of thrombocytopenic purpura.

4. WHAT IS THE DIFFERENTIAL DIAGNOSIS?
A complete blood count should be done for diagnosis. A low platelet count will establish thrombocytopenia as the cause of purpura. The presence of schistocytes or fragmentocytes may confirm the diagnosis of thrombocytopenic purpura.

References
5. Charles L, Bennett, MD, PhD; Peter D. Weinberg, BS; Karine Rozenberg-Ben-Dror, PharmD; Paul R. Yarnold, PhD; Hau C. Kwaan, MD, PhD; and David Green, MD, PhD; Thrombotic Thrombocytopenic Purpura Associated with Ticlopidine: A Review of 60 Cases. Annals of Internal Medicine, 1 April 1998. 128:541-544. Available online at: http://www.acponline.org/journals/annals/01apr98/ticloid.html
Coma And Thrombocytopenia In Kenya: Case Report

Author Information

Michael Gelchsheimer, M.D.
Swiss Air-Ambulance REGA

Olivier Seiler, M.D.
Section Chief, Medical Department Fixed Wing, Swiss Air-Ambulance REGA

Olivier Wenker, M.D.
Associate Professor of Anesthesiology and Critical Care, MD Anderson Cancer Center, The University of Texas