

Fatal thrombocytopenia associated with intracardiac mass

Platelets are anuclear blood cells whose function is to form blood clots when we injure ourselves, to prevent excessive blood loss. Thrombocytopenia is a relative low number of platelets in the blood. Thrombocytopenia has several possible causes, including the trapping of platelets in spleen, reduced production of platelets and increased breakdown of platelets. Some examples of an increase in platelet breakdown include pregnancy, idiopathic thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome, infection, autoimmune diseases and medications (e.g., heparin). Thrombocytopenia may often be asymptomatic and can only be revealed by full blood cell count. Alternatively, it may cause excessive bruises and/or bleeding. In some cases, platelet count is so low that dangerous internal bleeding occurs.

In this case, the author reports a 22-year-old male presented with progressive breathlessness on exertion, increasing jaundice, easy bruising and petechial rash over a period of 15 days. The patient had no history or family history of common disorders that may cause these symptoms. Importantly, the patient had severe thrombocytopenia, revealed by peripheral blood smear evaluation, along with an elevation in direct bilirubin level, total leukocyte count and neutrophil count. Accordingly, several possible causes of thrombocytopenia were examined. For instance, bone marrow study failed to detect any possible abnormality in hematopoiesis, excluding the possibility that there was a decrease in platelet production. Also, abdominal ultrasounds showed normal-sized spleen, therefore, ruling out that platelets were trapped in the spleen. Further, color Doppler excluded the possibility of deep vein thrombosis in abdomen or lower limbs. Also, the patient had no recent exposure to heparin. Anti-phospholipid antibody, anti-system lupus erythematosus (SLE) antibodies and anti-PF4 antibodies were negative, so thrombocytopenia of this patient was not likely to be caused by antibody-induced

autoimmune responses. Both ITP and TTP were ruled out in this case as there was no evidence of end organ damage and micro-angiopathic hemolytic anemia, and the patient showed no response to steroids. Since there were normal lactate dehydrogenase and haptoglobin levels, hemolysis was also excluded. Anti-infection and anti-inflammatory medications as well as platelet transfusion failed to improve the symptoms of this patient. He died of cardiac arrest just 1 day after an episode of melena.

As most common causes of thrombocytopenia were excluded, what was the possible explanation of this fatal thrombocytopenia? The patient had facial puffiness and soft tender hepatomegaly, which is a common sign of right ventricular inflow obstruction. Indeed, cardiac auscultation found long mid-diastolic murmur in tricuspid area and harsh phasic ejection systolic murmur, grade III/VI, without ejection click at pulmonary area. Further evidence came from trans-thoracic echocardiography, which showed a homogenous mobile mass obstructing right ventricular inlet and outlet, similar to large right atrial myxoma. The finding was confirmed by contrast-enhanced computerized tomography of chest and abdomen. Therefore, the fatal thrombocytopenia appears to be associated with intracardiac mass in the right ventricle, which may resemble large right atrial myxoma.

Thrombocytopenia has been reported to be associated with cardiac tumors.^[1-4] Large cardiac tumors often result in intracardiac blood flow obstruction, eventually leading to right heart failure. Cardiac tumor-associated thrombocytopenia is often associated with other hematologic disorders such as anemia or erythrocytosis as well. The mechanism by which intracardiac tumor leads to thrombocytopenia remains unclear, although it has been postulated that abnormal mechanical shear stress, caused by tumor-induced flow obstruction, may be responsible for the increase in the breakdown of platelets.

In conclusion, the current study presented a rare case with fatal thrombocytopenia associated with intracardiac mass. It provides useful insights into the clinical complications of thrombocytopenia associated with intra-cardiac tumors as well as how to diagnose such conditions. This report will have important implications for helping other patients with similar thrombocytopenia associated with intra-cardiac tumors.

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