Myocardial Infarction-triggered Thrombotic Microangiopathy

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Dear Editor,

Thrombotic thrombocytopenic purpura (TTP) is a microangiopathic hemolytic disorder that is induced by a marked reduction in the level of von Willebrand factor-cleaving protease, a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13 (ADAMTS-13).[1] Thrombotic microangiopathy (TMA) and TTP are characterized by microangiopathic hemolytic anemia, consumptive thrombocytopenia, renal and neurological involvement, pyrexia, and skin purpura..[2,3] Various triggers associated with the development of TTP have been identified, including bacterial and viral infections, tumors, and collagen vascular diseases. Although some reports available in literature describe myocardial infarction (MI) caused by TTP,[4] TTP caused by MI has not been reported. We report our 15-year experience (2001 to 2016) at our hospital regarding TTP and TMA cases and describe 2 patients in whom acute MI triggered TTP and were successfully treated using plasma exchange therapy.

Case 1 was a 48-year-old man admitted with a diagnosis of acute MI. On the second day of admission, he developed a high-grade fever (39°C) and thrombocytopenia (45×10^9/L). Within the next 2 days, his platelet count dropped to 20×10^9/L, hemoglobin dropped to 7.5 mg/dL. Lactate dehydrogenase level was 2500 IU/mL; leukocyte count, 20×10^9/L; indirect bilirubin, 2.4 mg/dL; direct bilirubin, 1 mg/dL; and serum creatinine, 2.3 mg/dL. A peripheral blood smear revealed schistocytes (at least 5 to 7 per powered field). Following a diagnosis of TTP, plasma exchange therapy was initiated using fresh frozen plasma replacement. His platelet count increased, and serum creatinine dropped to 1.7 mg/dL after 3 days of treatment. We assessed ADAMTS-13 activity 2 months after the MI and observed that it was less than 5%.

Case 2 was a 60-year-old man who developed TMA within a few days of acute MI. This patient also showed a low level of ADAMTS-13 activity (< 5%) and was treated with fresh frozen plasma replacement.

Systemic inflammation is known to cause an elevation in von Willebrand factor levels and a further reduction in ADAMTS-13 levels, eventually precipitating TTP, particularly in patients with low levels of ADAMTS-13. Patients with acute MI show elevated plasma von Willebrand factor levels with a parallel decrease in ADAMTS-13 and may trigger TTP or TMA.[4,5] In both cases, we hypothesized that acute MI was a source of inflammation in the setting of an underlying ADAMTS-13 deficiency, which could lead to endothelial damage and development of TMA or TTP. Clinicians should be aware of and consider this rare combination.

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