CLINICAL VIGNETTE

Cytomegalovirus-induced Coagulopathy

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Case Report

A 52-year-old African American male with history of renal transplantation for end stage renal disease secondary to focal segmental glomerulosclerosis presented to the hospital with three days of fevers to 39.5 degrees Celsius. In addition he reported chills, diarrhea, hematochezia and productive cough. His past medical history was significant for renal transplantation 18 months prior, in which the donor was CMV positive. His transplantation was complicated by acute rejection, treated with immunosuppressants and plasmapheresis, with ultimately preserved graft function. In addition, he had a history of hemophilia B and hepatitis C. The patient was admitted for further evaluation of his fevers. His initial labs were remarkable for creatinine of 2.5 (baseline 1.7) as well as CMV viremia with viral load 3 million copies/ml. Ganciclovir was initiated for presumed CMV viremia, nephropathy, and colitis. Additionally, intravenous immunoglobulin (IVIG) was started as adjunctive therapy for CMV nephropathy.

Seven days after admission, his INR unexpectedly increased from 1.3 to 5.5, and his PTT increased from 38.0 to 55.5 seconds with associated bleeding from line sites. Because of the patient’s history of hemophilia B, factor IX levels had been followed and were stable (38-43%) with factor IX concentrate replacement. The fibrinogen level was found to be decreased at <25 mg/dl and D-dimer >10,000 ug/L. Lactate dehydrogenase (LDH) was elevated at 444 U/L and haptoglobin depressed at <8 mg/dl. His hemoglobin was stable at 11.9-12.4 and his platelet count, which was initially 106,000-139,000 decreased to approximately 60,000 over the next week. A peripheral smear showed moderate schistocytosis. His acute kidney injury progressed and his creatinine worsened to 6.4. Of note, the CMV viral load increased from 166,400 to 481,800 copies/ml two days prior to the onset of coagulopathy, having previously declined from admission with ganciclovir treatment (See Figures 1-4).
Conclusion

Treatment of CMV-associated coagulopathy should consist of supportive transfusions and treatment of underlying CMV infection. However, one case report demonstrated possible benefit from plasma exchange in patients without thrombotic thrombocytopenic purpura, possibly due to clearance of pro-inflammatory cytokines, activated coagulation factors, and removal of viral particles from the bloodstream5.

Physicians should be aware of the association between DIC and severe CMV viremia in immunocompromised patients. Every attempt should be made to treat the underlying CMV infection. However, plasma exchange or plasmapheresis should be considered if the patient’s condition is worsening.

REFERENCES


Background

DIC is a rare but important complication of CMV viremia. There have been three case reports of disseminated CMV infection precipitating DIC in immunosuppressed patients1,2. Rat models of CMV infection reported abnormal coagulation studies similar to DIC involving endothelial infection, thrombosis, and organ hemorrhage3. In vitro experiments show that endothelial cells infected with CMV induce a procoagulant phenotype with local generation of thrombin4.