Images in Hospital Medicine: Posterior Reversible Encephalopathy Syndrome After High Dose Cytarabine in Acute Myelogenous Leukemia

October 11, 2016

Posterior Reversible Encephalopathy Syndrome (PRES) After High Dose Cytarabine (HiDAC) in Acute Myelogenous Leukemia

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Case Presentation:

A 68-year-old female with newly diagnosed acute myelogenous leukemia (AML) status post high-dose cytarabine (HiDAC) presented with ataxia, dysarthria, and thrombocytopenia with a platelet count of 10 k/uL. Computed tomography (CT) head showed scattered cortical subarachnoid hemorrhage (Figure A, solid arrow). Brain magnetic resonance imaging (MRI) showed bilateral, posterior subcortical white matter hyperintensity (Figure B, dashed arrow) consistent with vasogenic edema (i.e., posterior reversible encephalopathy syndrome (PRES)).¹⁻³ She had no associated blood pressure lability, kidney failure, or immunomodulatory therapy to suggest an alternative etiology of the PRES. She was supported in the intensive care unit (ICU) and eventually made good neurological recovery.

Discussion:

PRES is a syndrome that typically presents with either encephalopathy, loss of vision, seizure, headache or focal neurological deficit.¹⁻³ It is a well-described phenomenon in the setting of malignant hypertension, eclampsia, immunosuppression, renal failure and sepsis.¹⁻³ Many patients who develop PRES have complete reversibility and recovery significantly.¹ It is less described in induction chemotherapy with HiDAC for AML and should be recognized as a potential complication.² On brain imaging, PRES is commonly seen as bilateral well-defined subcortical vasogenic edema involving the white matter of parieto occipital region.¹⁻³ Other frequently affected areas are frontal region, brain stem, cerebellum, and basal ganglia.¹⁻³ The most accepted mechanism of action behind the vasogenic edema is thought to be an acute rise in the blood pressure which overcomes autoregulation of cerebral blood flow.¹⁻³ A second pathophysiological theory is it may be due to a direct injury to the endothelial tissue compromising the blood brain barrier.¹⁻²
Intracerebral bleeding or subarachnoid hemorrhage are uncommon features of PRES. The pathophysiology behind the hemorrhage is similar to the edema. These patients usually have associated bleeding or coagulation disorder. Presence of hemorrhage is associated with worse prognosis. Other indicators of poor prognosis are status epilepticus and presence of large ischemic infarction on MRI. Treatment consists of initiation of supportive care, control of hypertension and immediate discontinuation of the drug thought to be responsible for PRES if applicable.

References:

