Posterior Reversible Encephalopathy Syndrome After Dinutuximab

Kayeleigh Higgerson DO
*Children's Mercy Hospital*

Chandni Dargan MD
*Children's Mercy Hospital*

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Research Abstract Title: Posterior Reversible Encephalopathy Syndrome After Dinutuximab

Submitting/Presenting Author (must be a trainee): Kayeleigh Higgerson, DO
Primary Email Address: khiggerson@cmh.edu

☐ Medical Student
☐ Resident/Psychology Intern (≤ 1 month of dedicated research time)
☐ Resident/Ph.D/post graduate (> 1 month of dedicated research time)
✓ Fellow

Primary Mentor (one name only): Alan Gamis, MD, MPH
Other authors/contributors involved in project: Chandni Dargan, MD; Joy Bartholomew, APRN

IRB Number: N/A

Describe role of Submitting/Presenting Trainee in this project (limit 150 words): performed chart review and literature search to formulate and write abstract/poster presentation regarding subject

Background, Objectives/Goal, Methods/Design, Results, Conclusions limited to 500 words

Background:
Posterior reversible encephalopathy syndrome (PRES) is a reversible disorder associated with edema of subcortical white matter and cortical gray matter that usually involves the occipital and parietal lobes. Endothelial injury secondary to abrupt blood pressure changes breaks down the blood brain barrier leading to brain edema.

Per the ANBL0032 Fall 2015 Study Progress Report, Dinutuximab was the probable cause of PRES in two patients. There have been published reports of PRES with other anti-GD2 antibody therapies but very few reports of Dinutuximab as the causative agent.

Objectives/Goal:
To report a case of PRES after Dinutuximab

Methods/Design:
Case Report

Results:
A 4-year-old male with stage IV neuroblastoma presented to the emergency department (ED) with new onset seizure activity described as generalized stiffening with his eyes rolled back five days after completion of his second course of Dinutuximab and IL-2. His blood pressure elevated in the
ED, as high as 156/129. He was agitated on exam with a nonfocal neurologic exam. He was admitted to the intensive care unit where he had recurrent seizure activity that progressed to status epilepticus. MRI of the brain was significant for an abnormal T2 and FLAIR signal hyperintensity involving the cortex and subcortical white matter of the occipital lobes, posterior parietal lobes, and posterior temporal lobes, most consistent with PRES. He remained inpatient until elevated blood pressures and seizure activity resolved. He tolerated further cycles or Dinutuximab without incident.

**Conclusions:**
Pediatric patients receiving anti-neoplastic therapy are at risk for developing PRES, mostly reported in patients with leukemia. The recognition of PRES is increasing likely secondary to increased awareness of this syndrome and improved radiographic technique. Hypertension is a common side effect of some antineoplastic agents and appears to be a key risk factor in developing PRES, as was likely the case with the patient described here. Our patient developed symptoms five days after completing antibody and IL-2 therapy. The half-life of Dinutuximab is ten days, versus one hour for IL-2. Hypertension is a known adverse effect of Dinutuximab but not IL-2. These factors support Dinutuximab as a contributing factor to his presentation rather than IL-2.

There have been other cases of PRES documented in patients receiving different forms of monoclonal antibody immunotherapy. There is not enough data to establish a causative association with monoclonal antibody therapy and PRES. However, the index of suspicion for PRES should be high, especially when patients present with acute neurological symptoms after receiving this therapy.