



Figure. Pulmonary arteriole intimal and medial thickening seen in a patient with bronchiolitis obliterans

vessel wall proliferation. Routine screening for PH in patients with BO may assist in diagnosing PH. The treatment for PH differs from BO and there is little understanding of their interplay post-HSCT; further investigation is needed.

291

CT Scan Frequently Misses the Diagnosis of Posterior Reversible Encephalopathy Syndrome (PRES) after Stem Cell Transplant

Christopher E. Dandoy¹, **Luke Linscott**², **Stella M. Davies**¹, **Kasiani C. Myers**¹, **Javier El-Bietar**¹, **Ranjit Chima**³, **Abigail Pate**⁴, **Hector Wong**⁵, **Sonata Jodele**¹. ¹Bone Marrow Transplantation and Immune Deficiency, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; ²Pediatric Neuroradiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; ³Division of Critical Care Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; ⁴Cincinnati Children's Hospital Medical Center, Bone Marrow Transplantation and Immune Deficiency, Cincinnati, OH; ⁵Pediatric Critical Care, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Background: Posterior reversible encephalopathy syndrome (PRES) is a clinical syndrome characterized by vision changes, altered mental status, and seizures that are typically caused by acute rise in blood pressure and resolve over time. PRES has been reported after stem cell transplant (SCT) in association with hypertension from calcineurin inhibitors and steroids. The radiologic evaluation of PRES after SCT has not been well described.

Design/Methods: A retrospective review of all cases of PRES in SCT recipients from January of 2004 to December of 2013 was performed. PRES was diagnosed if the patient developed encephalopathy, headache, seizures or visual disturbances with a CT and/or MRI showing imaging findings compatible with PRES.

Results: Twenty-two of 838 (2.6%) transplanted patients were diagnosed with PRES, all of them allogeneic SCT recipients. The majority (64%) were male with a median age of

9.4 years (IQR 4.9–12.1) at time of SCT. Nine patients had a marrow failure syndrome (41%), 7 of which had Fanconi Anemia; 7 patients had an immune deficiency (32%); 5 had an underlying malignancy (23%); and 1 patient had a metabolic syndrome (4%). Twelve patients (55%) had a myeloablative preparative regimen. All patients received cyclosporine for graft versus host disease prophylaxis after SCT and 11 (50%) were treated with additional immunosuppressants including steroids for GVHD at the time of event. PRES was diagnosed at a median of 49 days (IQR 31–88) after SCT. Nineteen patients (86%) presented with seizures, the other 3 (14%) had altered mental status. All patients underwent a brain CT and/or MRI, 21 of 22 patients (95%) received a CT scan when they became symptomatic, which was diagnostic of PRES in 8 of the 21 studies (38%). Eighteen patients (82%) received MRI, 17 of the 18 (96%) were consistent with PRES. The one MRI not consistent with PRES was done 20 days after initial diagnosis, subsequent to resolution of the abnormal CT findings. Notably in 13 patients initial CT scans did not demonstrate findings of PRES, which were subsequently found on MRI. The median time elapsed between CT and an MRI examination was 20 hours (range: 3.6 hours to 9 days). The overall survival in patients that developed PRES was 35% (8 of 23 survived).

Conclusion: CT scan serves as a good diagnostic test in emergency situations to rule out CNS bleed or infection in SCT patients with acute mental status changes or seizures, but it is not an adequate radiologic study for diagnosing of PRES after SCT. Patients with clinical symptoms suggestive of PRES, but negative CT, should undergo MRI of the brain after the acute event is controlled to assist in diagnosis. MRI results diagnostic of PRES would prompt physicians to provide good hypertension control and aid decision-making regarding invasive diagnostic procedures (like lumbar puncture) or intensification of empiric antimicrobial therapy.

292

A Pilot Study of Donor Enteral Human Milk to Modulate the Gut Microbiome in Children Receiving Stem Cell Transplant

Stella M. Davies¹, **Cynthia Taggart**², **Kelly E. Lake**¹, **Doyle V. Ward**³, **Ardythe L. Morrow**⁴. ¹Bone Marrow Transplantation and Immune Deficiency, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; ²Division of Bone Marrow Transplantation and Immune Deficiency, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; ³Broad Institute, Cambridge, MA; ⁴Perinatal Institute, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Background: The gut microbiome is an important immune modulator, and previous work has shown important changes in the gut microbiome associated with the occurrence of GVHD. Studies in premature babies at risk for necrotizing enterocolitis have shown that enteral human milk reduces gut inflammation and bacterial translocation (Morrow et al, 2012). We hypothesized that enteral human milk given in the peri-transplant period would be well tolerated and would modify the microbiome and reduce inflammation.

Methods: In a pilot study, we treated 10 children <2 yrs with donor enteral milk, and collected stool samples for microbiome analysis and blood samples for analysis of inflammatory markers. Milk was obtained from the Mother's Milk Bank of the Northeast. Milk donors underwent standard donor screening and milk was pasteurized before use. As prebiotic and anti-inflammatory benefits of are thought to derive from human milk oligosaccharides produced by *fucosyltransferase2* (FUT2) and related gene biosynthesis