ELSEVIER

Blood 142 (2023) 6313

The 65th ASH Annual Meeting Abstracts

ONLINE PUBLICATION ONLY

627.AGGRESSIVE LYMPHOMAS: CLINICAL AND EPIDEMIOLOGICAL

Fat Embolism Syndrome from Bone Marrow Necrosis Following Peg-Filgrastim in a Patient with Primary Mediastinal B-Cell Lymphoma

Assile Koubeissy, DO[1], Natalie Rafaeli, MD[2], Pooja Gogia Bhasin, MD[1], Stephen Zachary Peeke, MD[1], Martin Bluth, MD PhD[3]

[1] Maimonides Medical Center, Brooklyn, NY
[2] MD Anderson Cancer Center, Houston, TX
[3] Maimonides Medical Center, Brooklyn, NJ

Background: The mechanism of atraumatic fat embolization syndrome (FES) is poorly understood. However, mechanical and biochemical processes are the two plausible theories that exist. FES from bone marrow necrosis caused by hemoglobinopathies, use of chemotherapy in hematopoietic malignancies and from use of G-CSF has been reported. In our case, a young male patient with primary mediastinal large B-cell lymphoma presented with microhemorrhages and punctiform ischemic brain lesions after receiving chemoimmunotherapy and pegylated G-CSF.
Case: A 29-year-old male recently diagnosed with primary mediastinal large B-cell lymphoma presented to our ED with severe bone pain after receiving his first cycle etoposide, prednisone, vincristine sulfate, cyclophosphamide, doxorubicin, and rituximab (DA R-EPOCH) with peg-filgrastim support. He was discharged with pain control and subsequently received his second cycle of DA-R-EPOCH (20% increase in etoposide, doxorubicin, and cyclophosphamide) with a retrial of peg-filgrastim. His WBC count rose to a peak of 41,100K/UL on the seventh day of his second cycle. Ten days after receipt of his 2 nd peg-filgrastim, he presented to the ED with complaints of generalized weakness, decreased oral intake and mild imbalance with his gait. His blood pressure was 103/71 mmHg, heart rate was 138 beats per minute, respiratory rate was 22 breaths per minute and oxygen saturation was 91% on room air; and he required 2 liters of supplemental oxygen. Laboratory studies showed Hb of 8.1 gm/dL, PLT of 47,000k/UL and WBC of 10,100k/UL. The WBC differential was consistent with myeloid predominance with a left shift and some enucleated RBC consistent with use of peg- filgrastim. LDH was 2874 U/L, albumin was 3.8 g/L, PTT 23.1 seconds, PT 13.8 seconds, INR 1.2. A CT with angiography of the chest showed no pulmonary embolism but an anterior mediastinal soft tissue mass, which decreased in size compared to his prior image, with erosive changes of sternum. Mediastinal and axillary lymphadenopathy also overall decreased in size. Due to developing confusion and mild left-sided facial weakness, MRI with and without intravenous contrast of the brain demonstrated numerous petechial microhemorrhages throughout the brain parenchyma in the supratentorial compartment and posterior cranial fossa with involvement of superficial and deep parenchymal regions. Many were seen clustered in the internal/external watershed regions. Transthoracic echocardiogram with bubble study demonstrated a patent foramen ovale (PFO). Prophylactic low molecular weight heparin was started and he was discharged after improvement of symptoms. He proceeded to complete 4 more cycles of standard dose R-EPOCH with prophylactic levofloxacin instead of peg-GCSF. His neurologic symptoms resolved and did not recur.
Conclusion: This case highlights a rare but severe complication of BMN causing fat embolization syndrome in a patient receiving G-CSF following R-EPOCH for high PMBCL with sternal bone involvement. We hypothesize that the rapid proliferation of myeloid cell lines induced by G-CSF led to vascular occlusion and relative hypoxemia. Furthermore, the prolonged incorporation of corticosteroids is known to cause adipose tissue disruption. His underlying disease involved invasion into his sternum which further would support the theory of bone marrow necrosis being able to dislodge into the vasculature. This case is important to alert physicians to be cognizant of this severe complication and to provide some evidence for continuing curative intent treatment with the omission of G-CSF support.

Disclosures No relevant conflicts of interest to declare.

https://doi.org/10.1182/blood-2023-189582