EBV-reactivation and Post-transplant Lymphoproliferative Disorder Treated with Rituximab

We read the recent paper on profile of EBV-associated infectious mononucleosis (IM) by Balasubramanian, *et al*. [1] with interest. They concluded that EBV associated IM is more common in preschool children. Here we highlight another dreaded complication due to reactivation of EBV post-allogeneic stem cell transplant (SCT) leading to post-transplant lymphoproliferative disorder (PTLD). Mortality rates due to PTLD are reported to be as high as 50–90% [2]. Various therapeutic approaches are suggested for EBV-associated PTLD including anti-B-cell treatments such as rituximab (Anti-CD20 monoclonal antibody) [3]. Rituximab alone or combined with low-dose chemotherapy is an effective therapy for EBV-associated PTLD [4,5]. We describe here successful treatment of EBV induced PTLD with rituximab in an infant post-allogeneic SCT.

An 11-month-old girl was referred to our centre for matched sibling allogeneic SCT. She was diagnosed as a case of familial hemophagocytic lymphohistiocytosis (HLH) at the age of three months and treated as per HLH-2004 protocol. Her elder brother was complete 6/6 match with her on HLA typing. She received reduced intensity conditioning regimen and her doner stem cells (CD34 positive cells) were infused. GVHD prophylaxis consisted of cyclosporine and methotrexate. She had neutrophil and platelets engraftment on Day +22 and +26, respectively. FISH studies performed on day +30 showed 97% XY (donor) cells and 3% XX (recipient) cells.

On day +45, she developed high grade fever and neck swelling with difficulty in swallowing. Bilateral tonsils were enlarged. Hepatosplenomegaly was also noted. A possibility of PTLD due to EBV was considered. Her EBV DNA copy numbers were raised to 72700. She was started on injection rituximab 375 mg/m^2 IV weekly × 4 doses. Her fever disappeared 48 hours later and tonsils gradually became normal size by day +52. Her repeat EBV DNA copy numbers were 1900 after 3 weeks. She was discharged on day +81 post-transplant and is doing well till date (18 months post-transplant). Early detection of EBV induced PTLD and aggressive treatment with Rituximab is a key to survival in patients who have undergone allogeneic SCT.

VIKAS DUA AND SATYA PRAKASH YADAV
Pediatric Hematology Oncology and BMT Unit,
Sir Ganga Ram Hospital, Delhi, India.
satya_1026@hotmail.com

REFERENCES

1. Balasubramanian S, Ganesh R, Kumar JR. Profile of EBV-associated infectious mononucleosis. Indian Pediatr. 2012;49:837-8.
2. Ocheni S, Kroeger N, Zabelina T, Sobottka I, Ayuk F, Wolschke C, *et al*. EBV reactivation and post transplant lymphoproliferative disorders following allogeneic SCT. Bone Marrow Transplant. 2008;42:181-6.
3. Yadav SP, Chinnabhandar V. Rituximab usage in children: a double edged sword. Indian Pediatr. 2012;49:335-6.
4. Serinet MO, Jacquemin E, Habes D, Debray D, Fabre M, Bernard O. Anti-CD20 monoclonal antibody (Rituximab) treatment for Epstein-Barr virus-associated, B-cell lymphoproliferative disease in pediatric liver transplant recipients. J Pediatr Gastroenterol Nutr. 2002;34:389-93.
5. Gross TG, Orjuela MA, Perkins SL, Park JR, Lynch JC, Cairo MS, *et al*. Low Dose Chemotherapy and Rituximab for Post transplant Lymphoproliferative Disease (PTLD): A Children's Oncology Group Report. Am J Transplant. 2012; 12:3069-75.

Psoas Abscess: Primary or Secondary?

It is important to explain the classification of primary and secondary iliopsoas abscess, with regard to a recent article by Mondal, *et al*. [1].

Iliopsoas abscess may be classified into primary or secondary according to the pathogenesis [2,3]. In 1992, Gruenwald, *et al*. [4] proposed a new classification of iliopsoas abscess according the organism, therefore those due to *Staphylococcus aureus* should be classified as primary and those that were not caused by this bacterium as secondary. This hypothesis was refuted because many iliopsoas abscesses secondary to spondylodiscitis were caused by *Staphylococcus aureus*. From then until today, the classification of iliopsoas abscess is based on the form of spread of infection. Therefore, primary abscesses are due to a bacteremia distant to iliopsoas muscle and secondary iliopsoas abscess occurs as a result of direct spread of infection to the iliopsoas muscle by contiguity from an adjacent structure [2,3], such as the case presented by Mondal, *et al*. [1]

Recent studies place special emphasis on understanding the characteristics of primary and secondary abscesses, and it is particularly important that