Discussion: The complete color transformation of the entire skin due to hemosiderin accumulation is to the best of our knowledge the first reported observation in CD30+ lymphoproliferation/ALCL patient. We speculate that hemosiderin loaded macrophages resulted from the paraneoplastic process by some still unknown mechanism.

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Primary mediastinal B cell lymphoma

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A 51 year old male presented with a one month history of progressive facial and chest swelling, hoarseness and dry cough. He developed acute onset anterior chest pain and presyncope. He had no weight loss or sweats and no haemoptysis. He was a non-smoker with no significant past medical history. On examination he had facial swelling and distended neck and chest wall veins. Breath sounds were reduced at the right apex. There was no peripheral lymphadenopathy or hepatosplenomegaly. Clinically the impression was of superior vena caval obstruction syndrome. Full blood count, biochemistry and LDH were normal. CT scan showed a 10.6 by 6.4 cm anterior mediastinal mass (image available). There was no other lymphadenopathy. Biopsy revealed a diffuse, moderately large lymphoid cell infiltrate with diffuse sclerosis (image available). Immunohistochemistry was positive for CD20, CD79a, BCL2, BCL6 and MUM1. CD5, CD30, cyclin D1, CD21 and CD23 were negative. Proliferation fraction was 70%. A diagnosis of primary mediastinal B cell lymphoma was made, stage 1AX. He was commenced on dexamethasone prior to starting definitive chemotherapy in the form of R-CHOP. CT scan after 3 courses showed >50% reduction in the mass which now measured 6.6 by 2.5 cm. Following a further 3 courses of R-CHOP the mass reduced to 5.2 by 1.5 cm. A PET/CT scan on completion of chemotherapy showed a complete metabolic response (image available). In view of this result, radiotherapy was omitted. The patient remains well in clinical remission 9 months post presentation. Potential points for discussion: 1. Is primary mediastinal B cell lymphoma a distinct histological entity? 2. Optimal first line chemotherapy for primary mediastinal B cell lymphoma 3. The role of rituximab 4. The utility of PET/CT imaging 5. The role of consolidation radiotherapy

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Relapsing multiple myeloma with difficult peripheral blood stem cells mobilization and uncommon adverse effects of novel agents

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A 53-year-old man presented in November 2008 with left sided chest wall swelling and was diagnosed as multiple myeloma, Durie-Salmon Stage III and serum beta2-microglobulin 3.2 mg/L). Investigations confirmed left fourth rib plasmacytoma with 20% abnormal plasma cells in bone marrow and monoclonal protein in serum of IgA lambda subtype. Following 5 cycles of VAD regime from November 2008 till March 2009, he attained complete remission and planned for autologous PBSCT and thalidomide. However, he failed twice peripheral blood stem cells (PBSC) mobilizations using high dose cyclophosphamide and refused bone marrow harvesting. In October 2009, he had successful PBSC mobilization with filgastrim and plerixafor yielded 3.7×106 CD34+ cells/kg in 3 leukapheresis sessions. Two days after the mobilization, he developed worsening renal function, detection of urine Bence Jones, abnormal plasma cells in peripheral blood and raised serum free light chain. There were also severe thrombocytopaenia, multiple lytic lesions, myelomatous deposits in the pelvic muscles and pleura and cardiogenic shock with diastolic dysfunction secondary to suspected early amyloid cardiomyopathy. He was treated with bortezomib, dexamethasone and thalidomide dose was continued but was later withheld due to intolerable adverse effects. It was complicated by severe peripheral neuropathy NCI-CTAE Grade III and deterioration in cognitive function with MMSE of 13. This occurred about 2 weeks after second cycle of VD which was thought to be associated with bortezomib. Other possible causes of deterioration in cognitive functions were excluded. A week later, he developed severe pneumonia and massive haemorrhagic pleural effusion after which he succumbed. In this case, we would like to highlight the uncommon adverse effects of novel agents, namely subcutaneous plerixafor in mobilizing patients with multiple myeloma and bortezomib. Further management of these issues are to be discussed.

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Six cases of ABO discrepancies after intensive chemotherapy for B cell lymphoma

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In ABO blood typing, the discrepancies between forward (cell) grouping and reverse (serum) grouping are observed in the cases of disease-related immunosuppresion. However, it is obscure that these phenomena are related to some types of treatment, such as intensive chemotherapy for malignant diseases. We retrospectively analyzed 2156 specimens for ABO blood typing in our hospital between January 2005 and December 2005, and compared forward and reverse typing tests in each case. ABO typing was performed by gel column centrifugation method at room temperature. Anti-A or anti-B antibodies were negative (zero), trace (w+) and weakly positive (1+) in 30 specimens of 24 cases (19 cases of group A, 4 cases of group B and one case of group O). In these cases, 12 cases were examined for the first time and the other 12 cases (11 in group A and one in group O) had been examined before. In latter 12 cases, decrease of anti-B antibodies was observed in eight cases (seven in group A and one in group O), including six cases of B-cell lymphoma (B-NHL) in group A. All these cases of B-NHL received intensive chemotherapy with rituximab. Decrease of serum immunoglobulin (IgG, IgA and IgM) was observed in all the cases after B-NHL treatment. The ABO discrepancies in these cases may be related to decrease of anti-B antibodies caused by treatment-related severe immunosuppression (decrease of serum immunoglobulin). Anti-B agglutination reaction might be influenced more easily than anti-A, as the amount of group B antigen on red blood cells is less than that of group A antigen. Further studies are needed to clarify between this phenomenon and other complications (such as severe viral infections).

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Anaplastic large T/null cell lymphoma (ALCL) while treating Langerhans cell histiocytosis (LCH)

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We report a case of a 27 year-old woman, previously healthy, with the diagnosis of a LCH and an ALCL. In Jun/2007 the patient (pt) noted a left parasternal lump, with progressive