Lymphomas and myeloma

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Hodgkin Lymphoma with Vertebral Involvement

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A 39 year old male presented with a 3 month history of progressive anterior chest pain radiating posteriorly. In addition he had interscapular discomfort exacerbated by alcohol. He had significant weight loss (5 kg in one month) and occasional drenching night sweats. There was no palpable lymphadenopathy or organomegaly. CT scan showed mediastinal lymphadenopathy and destruction of T2 and T3 vertebral bodies with an associated soft tissue paravertebral mass (image available). Mediastinal biopsy yielded a diagnosis of nodular sclerosing Hodgkin lymphoma (HL). Bone marrow trephine showed no evidence of infiltration. PET/CT confirmed bone involvement at T2/T3 making him a stage 4B. He had a Hasenclever score of 2. He was entered into the NCRI RATHL study for advanced HL. PET/CT scan after 2 cycles of ABVD showed no abnormal FDG uptake and he therefore went on to complete a total of 6 cycles of therapy. End of treatment CT scan showed resolution of adenopathy and the paraspinal mass but residual destruction of T2/3 vertebrae. Interscapular pain persisted on exertion but was not affected by alcohol ingestion. He received consolidation radiotherapy (30 Gy in 15 fractions) to T1-T4 vertebral bodies. The patient remains well without B symptoms after 4 months of follow up. Potential points for discussion: 1. Should we worry unduly when Hodgkin lymphoma involves bone? 2. Is interim PET scanning still reliable in assessing bony involvement? 3. Is consolidation radiotherapy to bone justified in the setting of a negative interim PET?

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Pleural effusion in a patient with Hodgkin's lymphoma

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A 28-year-old man was admitted at hospital in December 2009, with a 2 months story of left cervical and scapular disabling pain which was exacerbated with superior limb movements. He had Hodgkin's lymphoma (HL), sclerosis nodular type, stage IV-B, IPS-4, and he has been treated with multiple chemotherapy regimens and cervical radiotherapy (May-June/2009). At hospital admission, he was on the 5th cycle of 4th line chemotherapy with Gemcitabine, Vincristine and Liposomal Doxorubicin (GVD). We suspected of disease progression, but PET showed no abnormal uptake. However, TC images revealed a pleural effusion (PE) in the superior left and inter-cisural pleural space. A thoracocentesis was performed, with drainage of 650 ml yellow-citrine fluid. Laboratory tests revealed an exsudate without lipid excess, bacteriological and mycological tests were negative and no malignant cells were seen. Pleural biopsy showed no neoplasic cells. Central venous catheter (CVC) was removed and had no loss of integrity. As we have excluded infectious and malignant underlying conditions as well as CVC rupture, we thought that the PE could be secondary to radiation or to liposomal doxorubicin (LD) or both. We asked our patient again and realized that pain had been progressive, starting after the 2nd GVD cycle. Initially, the pain occurred only 2–3 days after chemotherapy, becoming persistent after the 4th cycle. The pain didn't overcome with corticosteroids and opioids, but it improved with anti-inflammatory drugs. The hypothesis of PE being secondary to synergism of LD and radiotherapy seemed probable, regarding the patient's complains. So, the 6th cycle (the last one) was made without doxorubicin and pain has decreased progressively. We presented this case because the diagnosis was difficult, as there are many possible etiologies for a PE in a patient with cancer doing chemotherapy. Management was also controversial, as not prescribing LD at 6th cycle could be questionable.

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Peripheral T cell Lymphoma – Lennert's subtype

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59 year old male presented in July 2006 with widespread lymphadenopathy. There were no B symptoms or organomegaly. Full blood count was normal but LDH raised. CT scan showed extensive lymphadenopathy and mild splenomegaly. Lymph node biopsy showed a lympho-epithelial T cell lymphoma of Lennert's subtype. Bone marrow was infiltrated making him a stage 4A. He initially declined treatment but commenced CHOP 3 months later in view of increasing peripheral lymphadenopathy with most nodes 3–4 cm. Following the third cycle he suffered a myocardial infarction. For the remaining chemotherapy etoposide replaced doxorubicin. Post treatment CT showed mild, low volume lymphadenopathy. Eight months later he developed a florid, infiltrative rash across his torso (image available). Biopsy confirmed peripheral T cell lymphoma with identical peak in T cell receptor gene rearrangement as that seen in initial biopsy. One month later he developed recurrent lymphadenopathy and stage 4 relapse was confirmed. He was treated with DHAP salvage chemotherapy with a view to autologous stem cell transplant. However despite an excellent response we deferred transplant as he was profoundly debilitated. Within 3 months the skin rash recurred followed by B symptoms and progressive lymphadenopathy and splenomegaly. LDH was raised but full blood count was normal. Twelve days later he developed severe thrombocytopenia and LDH increased dramatically. Severe pancytopenia ensued and bone marrow aspirate showed marked haemophagocytosis (image available). Despite treatment with high dose steroids, his condition continued to deteriorate. He died one week later. Potential points for discussion: Is Lennerts lymphoma a clinically distinct subtype of peripheral T cell lymphoma? What is the optimum first line treatment for Lennerts lymphoma? Is there a role for gemcitabine in the management of peripheral T cell lymphoma? Should patients with peripheral T cell lymphoma be transplanted in first remission?

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Peripheral T-cell lymphoma (PTCL), CMV or both?

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41-year-old male (past exposure to toxic paints, smoker) who began complaints of fever, sweating, cervical lymphadenopathy and hepatosplenomegaly (HSM), cutaneous rash, generalized pruritus and autoimmune hemolytic anemia (AIHA) in 06/2006. He had recurrent episodes with partial improvement on corticosteroids; was referred to our hospital in 05/2009: