lymphoma and the patient age he has been just followed up for the last 6 months. Blood cell count, serochemical findings including liver enzymes are normal and the liver mass is stable.

Discussion: Primary hepatic lymphoma is defined as lymphoma either confined to the liver or having major liver involvement. It represents less than 1% of all extranodal lymphomas. The exact cause of primary hepatic lymphoma is unknown, and there is no consensus on the best approach for management.

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Surgery and multiple successive chemotherapy helped to achieve a sustainable remission in patient with advanced MALT lymphoma

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Pt suffered from dramatic weight loss and acute pain, however, the CT did not revealed tumour and he was not referred to the oncologist. Only after suffering became unbearable he was admitted to the hospital with advanced lymphoma. Aggressive treatment improved conditions and remission was achieved. Pt E.A, 62 y.o. Diagnosis MALT lymphoma with the involvement of the small intestines and lymph nodes of mesentery with the secretion of the IgA lambda-type paraprotein, IVB stage Immunohistochemistry: proliferation of small lymphocytes with infiltration of intrafollicular structures. Phenotype: CD 20+, CD79+? CD5–, CD10–, CD23–, CD43+/–, Cd11+/–. In 1997 the pt noted the dramatic weight loss (14 kg), acute abdominal pain, vomiting. The level of the IgA lambda-type paraprotein was increased (15 g/l). CT did not detect tumour. In December 1999 due to intraperitoneal bleeding he underwent surgery with the resection of the small intestine, sanitisation and drainage of the abdominal cavity. Histological findings helped to set the diagnosis. February 2000 – January 2001 – 7 courses of chemotherapy (Leukeran, Vinblastin, Natulan, Prednisolonum). After that the pt was monitored. In December 2007 the increase of paraprotein (40 g/l) was observed. However the conditions remained stable, and interventions were not conducted. In February 2008 due to intoxication the chemotherapy (Vincristine, Carmustine, Alkeran, Cyclophosphan, Prednisolonum) was started. After 4 courses the pt had serious complications: granulocytopenia (4 grd) and the bilaterial mycotic pneumonia. New chemotherapy line (4 courses of Rituximab, Cyclophosphan, Fludara) started in December 2008 reduced intoxication symptoms. August – September 2009 – 2 administration of Rituximab to consolidate the response. From October 2009 – case monitoring. In December 2009 the paraprotein level was 0.9 g/l.

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A case report of myeloma in gastric MALT lymphoma with bone marrow(BM) involvement: Two distinct entities?

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Plasmocytic differentiation (PD) may been seen in a minority of B-cell non Hodgkin's lymphoma (NHL) but it's frequent in extranodal marginal zone B-cell lymphoma (MALT lymphoma). PD often manifests clinically with secretion on monoclonal immunoglobulin. In some cases, the distinction between a B-cell NHL with PD and a plasma cell neoplasm on the basis of immunohistochemical analysis on biopsy specimen may be challenging to diagnose. Recent data suggest that flow cytometric immunophenotyping (FCI) of plasma cell might help the differential diagnosis of these two entities. We report a case of MALT gastric lymphoma stage I that, after five years from diagnosis and treatment, showed BM involvement from nodal marginal zone B-cell NHL together gastric relapse, progressive increment of serum monoclonal component IgG-lambda and appearance of free light chain lambda on urine immunofixation. The patient received six cycles of R-CVP achieving a CR on gastric and BM biopsies specimens, but serum monoclonal component level was not affected and on the contrary it increased on. After 10 months BM biopsy detected an infiltration of 40% of small lymphoma cells stained with anti-CD20 monoclonal antibody associated with plasma cell differentiation. Rituximab $\times 4$ were performed, with further increase of serum monoclonal component IgG-lambda (4.9 gr/dl) and persistent BJ proteinuria. A new BM biopsy showed 90% of small lymphoid cell expressing CD20, CD79a, Bcl2 and lambda light chain. FCI of the BM was done and revealed an increased plasma cell population expressing CD38, CD138, CD20, CD56, CD117, but lacked CD 45 and cytoplasmatic light chain lambda. Further appropriate laboratory and radiographic studies defined the diagnosis of low-grade myeloma, small cell type, stage II (D–S) and stage III (ISS) and consistent treatment. FCI of plasma cell in correlation with the clinicopathologic data confirms its usefulness for the identification and characterization of myeloma cell.

Leukaemias

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Bone marrow necrosis: A challenging condition

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Bone marrow necrosis (BMN) is a rare entity, which is generally associated with malignancy. Hematologic malignancies are the underlying cause in almost 60% of the cases, with acute leukemia being the most frequent etiology accounting for 40%. Prognosis is generally poor but in some cases repopulation of the BM cavity with normal hematopoietic cells is seen. Treatment includes supportive measures and treatment of the underlying cause. A 54-year-old man with idiopathic myelofibrosis since 2004, treated with splenectomy and hidroxiurea, was asymptomatic until November/2009, when he presented with asthenia, anorexia and weight loss. Full blood count showed increasing basophiles but bone marrow aspiration didn't reveal leukemic transformation (LT). Two months later, he developed an intense and disabling pain on the lower back leading to hospitalization. Laboratory findings included Hg – 8.7 g/dl, white cell count – 8.07×10^9/L, platelets – 307×10^9/L, LDH – 1000 U/L (normal range: 67–190 U/L). During hospitalization, he developed pancytopenia and LDH raised up to 7354 U/L. Bone marrow (BM) aspiration and biopsy showed chronic myelofibrosis with extensive necrosis and no blasts. BM scanning revealed 99m Tc uptake only in proximal third of the left humerus, right humerus's head and both femur's heads. There was also an intense

uptake in liver related with extramedullary hematopoiesis. Arterial blood hypoxemia raised the suspicion of fat and/or necrotic BM embolization to pulmonary arteries confirmed by ventilation-perfusion scintigraphy. Supportive measures were initiated (platelets and red blood cells transfusion, analgesia, oxigenotherapy) with clinical improvement. At present, he has recovered from pancytopenia and pain, but still needs oxigenotherapy. Despite the efforts to find a cause, we didn't know why this patient developed BMN. Nevertheless, as BMN develops before leukemic diagnosis in some cases, he will be keeping in close surveillance.

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Relapsing t-acute lymphoblastic leukemia post allogeneic peripheral blood stem cell transplantation

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An 18-year-old Malay man presented with progressively enlarging multiple swellings at the neck region in January 2008 and was diagnosed as T-cell acute lymphoblastic leukemia (T-ALL)-intermediate risk (normal cytogenetics). He completed 3 cycles of BFM-90 Block A–B regimes in June 2008 and successfully attained complete remission (CR) after the first cycle. While on maintenance with oral mercaptopurine and methotrexate, he relapsed in September 2008. He attained CR2 following HyperCVAD chemotherapy. He underwent HLA-matched sibling donor (younger sister) allogeneic peripheral blood stem cell transplantation (PBSCT) in November 2008 with a total of $4.2 \times 10\text{^}6$ CD34+ cells/kg. Conditioning regimen was cyclophosphamide 60 mg/kg and total body irradiation 12 Gy. Graft versus host disease (GvHD) prophylaxis was methotrexate, cyclosporine and prednisolone. Neutrophil and platelet engraftment was at days +17 and +15, respectively. Two months post PBSCT, he developed cyclosporine nephrotoxicity but was successfully reintroduced. Eight months later, he relapsed with 80% blast in peripheral blood and was advised for chemotherapy but he declined further treatment. Short tandem repeat (STR) study showed complete donor chimaerism. He was treated symptomatically with blood and blood products transfusion. He succumbed to his illness in January 2010. We plan to follow up the donor. The points that we would like to discuss are the treatment options of relapse T-ALL post allogeneic PBSCT and role of novel agents. Interestingly, the possibility of donor cell leukemia.

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Acute lymphoblastic leukemia: A case-report of difficult management

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Introduction: The conventional treatment for acute lymphoblastic leukemia (ALL) is directed at the destruction of all leukemia cells in the bone marrow (BM), lymphoid system and those in sanctuary sites.
Case-Report: A 29-year old man had presented in 1993 with anorexia and weight loss with one month of evolution. The patient had a KPS of 90% and presented soft palate petechiae and leg equimosis. Hepatosplenomegaly was absent, but had cervical, axilar and inguinal lymph node enlargement. The WBC was $51 \times 103/\mu L$, with 81% of circulating blasts on blood smear. He had a Hb level of 7.4 g/dL and a platelet count of $13 \times 103/\mu L$. The BM was hypercellular with massive invasion of L2 lymphoblasts (90%). Immunophenotyping showed expression of CD10, CD19, CD20, CD34 and HLADr. Metaphases were absent on BM chromosomal analysis. CSF was free of blasts. Pre-B-cell ALL was diagnosed and treatment with LINKER protocol was initiated. He was free of blasts in the BM by D+14. The patient has also undergone prophylactic intrathecal methotrexate (IM), cranial radiotherapy and finished the maintenance chemotherapy (CT) in 1996. Five years after the diagnosis, he had an isolated CNS relapse and was submitted to HCVAD protocol, with IM and cranial-spinal irradiation and subsequent allogeneic stem cell transplantation (AlloSCT) on 2nd remission. Nine years after AlloSCT, he had a 2nd relapse with CNS (CNS 2, 0.16% blasts) and BM involvement. The karyotype was complex and the immunophenotyping was positive for CD9, CD10, CD20, CD34 and TdT. The patient initiated a 3rd line CT with ALL-BFM90 protocol and achieved a complete remission after the 1st cycle. He has been proposed for a 2nd AlloSCT.
Comment: This is a rare case of ALL with long-term remission after CNS relapse and prior irradiation. The CNS was the probable sanctuary for leukemic cells, despite the previous treatment with high-dose CT and AlloSCT. The available therapeutic options for CNS treatment are still limited and of difficult management.

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Fludarabine induced hemolytic anemia

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Introduction: AMF 32 years old lady presented to us in Jan. 2010 suffering from generalized weakness, jaundice & dark urine as after her 2nd scheduled cycle of chemotherapy. In Nov. 2009 she first presented in with generalized fatigue & blood picture of pancytopenia HB6.3 TLC14 PLT65 On examination axillary, cervical lymphadenopathy & hepatosplenomegally were only found. Bone marrow aspirate was done revealing low normocellular bone marrow with 4% blasts 8% immature lymphocytes 72% lymphocytes IPT showed Mature B lineage positive for CD19CD20CD22CD79b&IgM. Diagnosis of Lymphocytic lymphoma was confirmed &the patient received two cycles of Fludara cyclophosphamide combination chemotheraby. In Jan. 2010 before the third cycle she came suffering from deterioration of her general condition, hematuria & jaundice.
Diagnostic considerations: She is not known to be hypertensive or diabetic Physical examination revealed jaundice with hepatosplenomegally & axillary lymphadenopathy and her vital signs were pulse 120/min. Blood pressure120/80, temperature 37°C. Complete blood picture showed HB5.1 TLC2.2 PLT36. Liver functions showed total bil.4.2 direct bil.2. Coomb test was positive for direct test & negative for indirect one Reticulocytes 2.7%. Bone marrow aspirate repeated & showed low normocellular bone marrow with 2% blasts 29% lymphocytes. Abdominal sonography showed marked hepatosplenomegally with no dilated intrahepatic biliary radicals.
Treatment: Immediate blood components transfusions were given in addition to liver support. Steroids 1 mg/kg was started with addition of colony stimulating factors.
Results: Mild improvement in the patient's general condition with Total bil. Continued to rise up to 8.1, pancytopenia still persistent. Question 1: What causes haemolytic anaemia with fludarabine? Question 2: Is there any predictor for fludara induced hemolysis? Question 3: What is manag. of refractory cases?