In this heavily pretreated patient population, survival rates for patients responding to bendamustine salvage therapy were encouraging. We believe that well-selected double refractory patients might benefit from bendamustine as salvage treatment, but further prospective clinical trials are needed in this situation.

P2.19 Light chain cast nephropathy in a patient with Waldenström's macroglobulinemia

X. Galloo[1], C. Caron[2], L. Pipeleers[3], K. Wissing[3], K. Fostier[2], F. Trullemans[2], C. Tielemans[3], R. Schots[2], A. De Becker[2]

[1]Vrije Universiteit Brussel, Brussel, Belgium, [2]Hematology, UZ Brussel, Brussel, Belgium, [3]Nephrology, UZ Brussel, Brussel, Belgium

A 65-year-old man, diagnosed with Waldenström's macroglobulinemia (WM), who had repeatedly declined systemic treatment presented with anuric renal failure and hyperviscosity syndrome (HVS). Viscosity was increased to 3.2 and immunoglobulin M-levels (IgM) to 54.80g/L. Plasma exchange was initiated to relieve HVS-symptoms. At presentation plasma creatinin had increased from 1.32 mg/dl to 7.16 mg/dl and eGFR had decreased from 54ml/min/1.73m2 to 8 ml/min/1.73m2. Urgent haemodialysis was started, dexamethason was also administered, however renal function did not improve. Kidney biopsy revealed protein sediments in the tubules, kappa positive on immunofluorescence, compatible with light chain cast-nephropathy. Serum free light chain kappa was elevated (1120 mg/L), a repeat bone marrow aspirate confirmed the diagnosis of WM. Since there was a light chain excess and a biopsy proven light chain cast nephropathy haemodiafiltration with endogenous reinfusion (HFR) was initiated and continued for the first time in a patient with WM. During diagnostic work-up dexamethasone was started, however renal function did not improve. Subsequently systemic treatment was initiated with R-COP (rituximab, cyclophosphamide, vincristine and prednisolone), in accordance to a previous report. There was a sufficient improvement in renal function to stop haemodialysis, unfortunately the patient progressed after 3 treatment cycles and deceased of splenic rupture.
WM is a rare low-grade lymphoproliferative disorder, histopathologically defined as a subset of lymphoplasmacytic lymphoma (LPL). WM is characterized by bone marrow infiltration and clonal proliferation of B-lymphocytes responsible for IgM monoclonal gammopathy of any concentration. Most patients diagnosed with WM have symptoms attributable to tumor infiltration and/or monoclonal protein. Renal disease in WM has been reported, but it is rare and cast nephropathy associated with WM in particular is very rare. To our knowledge this is the sixth case report of WM-associated cast-nephropathy.

P2.20 Galectin expression in the multiple myeloma microenvironment

J. Muller[1], J. Caers[1], M. Binsfeld[1], S. Dubois[1], G. Carmeliet[2], Y. Beguin[1], R. Heusschen[1]

[1]University of Liège, Liège, Belgium, [2]KU Leuven, Leuven, Belgium

Galectins are a protein family characterized by a conserved carbohydrate recognition domain, which enables them to bind to and cross-link glycoproteins. In addition, they exert intracellular functions independent of their sugar-binding capacity. Galectins have been implicated in diverse physiological and pathological processes, including cancer. In fact, galectins have been shown to contribute to multiple aspects of tumorigenesis, including neoplastic transformation, tumor cell survival, immune escape, angiogenesis and metastasis. This makes these proteins interesting targets for therapy. At this point, the role of galectins in multiple myeloma is not well understood. Interestingly, initial reports suggest a similar multi-faceted role for galectins in this disease.
Multiple myeloma is a hematological malignancy of monoclonal plasma cells characterized by bone marrow infiltration of these cells and osteolytic bone lesions. Galectins have been shown to modulate the proliferation and survival of multiple myeloma cells and the differentiation, proliferation and adhesion of osteoblasts and osteoclasts. In the current project, we aim to elucidate the role of galectins in the 5TGM.1 model with a focus on their role in osteolytic bone disease. We profiled the mRNA expression of all murine galectin family members in 5TGM.1 myeloma cells before and after propagation *in vivo*. In addition, we established primary osteoclast cultures and we confirmed successful generation of polykaryotic osteoclasts by TRAP staining and reporter gene expression validation. Also, we determined galectin mRNA expression during osteoclast differentiation from monocytic precursors. Our results indicate that the expression of several galectins is significantly altered during these processes.
These initial results provide us with target galectins for gain- and loss-of function experiments to further dissect their contribution to multiple myeloma biology and the development of osteolytic bone disease.

P2.21 Post-Transplant Lymphoproliferative Disorder following solid organ transplantation

J. Dallemagne[1], M.C. Ngirabacu[1], V. De Wilde[2], B. Bailly[2], F. Benghiat[2], M. Maerevoet[2], M.F. Dehou[2], N. Meuleman[1], D. Bron[1]

[1]Institut Jules Bordet, ULB, Brussels, Belgium, [2]Erasme, ULB, Brussels, Belgium

Background
Post-Transplant Lymphoproliferative Disorder (PTLD) is one of the most frequent cancer following solid organ transplantation. Its incidence has been noted to vary according to EBV history and time from transplant.

Population and methods
We reviewed all cases of patients which had developed PTLD after solid organ transplantation (heart, lung, kidney and liver) from 1987 to 2010, in Erasme hospital, Brussels. PTLD was defined in conformity with WHO criteria. We analyzed baseline information, EBV history, PTLD characteristics, treatment and outcome.

Results
We identified 28 cases of PTLD. Two cases had no information concerning EBV status, one lacked data and was not considered. Average time from transplant at diagnosis was 6,22 years with 7 cases that had occured during the first year post-transplant (25,9%). Prior to development of PTLD, in those with information concerning EBV sero-status (25 cases), 6 had presented a sero-conversion (24%) and 13 an EBV reactivation (52%). According to WHO criteria classification, 23 were monomorphic PTLD (85,2%), 3 were polymorphic PTLD (11,1%,) and 1 was an early lesions PTLD (3,7%). At diagnostis, the majority were a stage IV (63%); 22,2% a stage IE; 7,4% a stage I; 3,7% a stage II; 3,7% a stage III. Concerning treatment, 77,8% benefited from a reduction of immunosuppression; 77,8% were given rituximab (alone (37%) or combined with chemotherapy (40,8%)). After treatment, 14 achieved complete remission (CR), 3 had a partial response (PR), 3 had a progressive disease, 5 died soon after diagnosis, 2 were lost to follow-up. Regarding patients with CR, 5 relapsed (2 attained CR with second line treatment, 3 died). As to patients with PR, 1 died, 2 obtained CR after second line treatment.

Conclusion
Our series confirms a trend towards a late onset of PTLD (< 26 %