trunk, arms, hands, face and oropharynx; there was no evidence of liver dysfunction. No mutations suggestive of hereditary hemorrhagic telangiectasias were identified. An IgG-kappa monoclonal gammopathy was detected at a concentration of approximately 0.7 g/dl without lytic bone lesions and the bone marrow biopsy revealed 7% plasma cells, consistent with monoclonal gammopathy of undetermined significance (MGUS). The bilateral perinephric fluid collections developed slow and progressive over 5 years from completely normal appearing kidneys. A liver biopsy was normal without any evidence of elevated pressures through the hepatic or portal systems. Therapeutic phlebotomy was initiated but ultimately discontinued due to development of shortness of breath following phlebotomy. Microscopic intrapulmonary shunting was identified and slowly progressed with worsening hypoxia and shortness of breath, finally requiring continual supplemental oxygen. There was no evidence of pulmonary hypertension on echocardiogram or right heart catheterization. The pathofysiology of the TEMPI syndrome is unclear.

Abstracts posters stem cell biology and transplantation P.05 – P.17

P.05. Imatinib and nilotinib do not prevent adhesion and migration of human CD34+ cells in vitro and in immunodeficient NSG mice

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Background
The BCR-ABL tyrosine kinase inhibitor (TKI) imatinib has previously been shown to also inhibit the tyrosine kinase c-kit, the stem cell factor receptor. Nilotinib is 30 times more potent than imatinib to inhibit BCR-ABL in vitro. But very few information is available on its inhibitory effects on c-kit and thus on $CD34^+$ cell adhesion and migration since this receptor is implicated in these biological processes.

Aims
To compare, in vitro and in vivo, the inhibitory effects of imatinib and nilotinib on adhesion, migration and engraftment capacity of human cord blood $CD34^+$ cells.

Results
Analysis of VLA-4, VLA-5 and CXCR-4 cell surface expression by flow cytometry after 48 hours of culture have shown that both VLA-4 and VLA-5 expression (n=3) were significantly decreased in presence of imatinib or nilotinib at physiological concentrations (1 and 5 µM) while CXCR-4 expression was not affected (n=3). However, nor imatinib nor nilotinib decreased the adhesion of $CD34^+$ cells to retronectin (n=4). Further, migration through a SDF-1 gradient was not affected by a 48-hour cell culture in presence of TKIs (n=3). Finally, we compared the impact of imatinib and nilotinib on engraftment in a xenotransplantation model. Twenty-five NSG mice sublethally irradiated and inoculated intravenously with 6.10^5 human $CD34^+$ cells, were treated orally with a placebo, imatinib 150 mg/kg/day or nilotinib 75 mg/kg/day for 42 days. Bone marrow chimerism was analyzed by flow cytometry. No significant differences were seen between mice treated with imatinib (47.7 % ± 5.3; n=8; p=0.4130) or placebo (52.5 % ± 2.7; n=9), while engraftment of human CD34+ cells was slightly decreased (40.6 % ± 4.4; n=8; p=0.0314) in mice treated with nilotinib.

Conclusion
TKIs do not prevent adhesion and migration of human cord blood $CD34^+$ cells both in vitro and in NSG mice even if chimerism was slightly lower in mice given nilotinib.

P.06. Evidence for expansion of host-derived CMV-specific CD8+ T cells after allogeneic transplantation with nonmyeloablative conditioning

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It has been suggested that significant numbers of host-derived CMV-specific T cells could persist in patients given grafts following nonmyeloablative conditioning. In the current study, we challenged this hypothesis by assessing chimerism levels among CMV-specific CD8+ T cells (labelled by specific pMHC multimers) around day 40, 100 and 180 after allo-HCT in a cohort of 24 patients given allogeneic grafts after nonmyeloablative conditioning. Four of 17 CMV-seropositive recipients given grafts from CMV-seronegative donors had higher (>25%) proportion of cells of recipient origin among CMV-specific CD8+ T cells (ranging from 32.4 to 100%) than among other CD8+ T cells. Interestingly, the 2 patients with CMV-specific CD8+ T cells of 100% recipient origin on day 100 had relatively high counts of CMV-specific CD8+ T cells on that day (13.1 and 14.7 cells/µL), demonstrating that high number of CMV-specific CD8+ T cells of recipient origin could persist after allo-HCT in a proportion of nonmyeloablative recipients.

P.07. Adaptation of a murine chronic GVH model to study graft versus myeloma effect after allogeneic transplantation

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To elucidate the mechanisms behind graft versus tumor effect (GVT) and graft versus host disease (GVH), our laboratory adapted a murine model of allogeneic bone marrow (BM) transplantation using B10.D2 donor mice and Balb/cJ recipient mice that were inoculated with MOPC-315.BM myeloma cells. Balb/cJ recipient mice were intravenously (IV) injected with $2,5x10^5$ luciferase transfected MOPC-315.BM cells. At day 30 after inoculation, 6 mice received an autologous transplantation (Balb/cJ cells) and 8 mice received an allogeneic transplantation (B10.D2 cells) by IV injection of $10x10^6$ BM cells and $70x10^6$ spleen cells. Prior to transplantation, both groups of mice were irradiated with 6 Gy. Tumor development, before and after transplantation was followed by measuring their bio-luminescence using VIVOVISION IVIS 200 (Xenogen). Immune responses were followed by taking blood samples before transplantation (day -2), and at days 7 and 19 after transplantation, analysing lymphocyte counts and NK, NKT and T-cell subpopulations. When mice showed signs of paraplegia or signs of GVH disease, they were sacrified and analysed for immune activation and regulation in different organs (blood, spleen, lymph nodes, thymus and bone marrow). In vivo imaging showed disappearance of the luciferase signal in 7 of the 8 allografted mice, whereas all mice that received an autologous transplantation developed myeloma disease. The recovery of myeloma diseased mice by this allogeneic transplantation could be attributed to an immune graft versus myeloma effect. Further analysis of the cellular kinetics showed a decrease in regulatory T cells and activation of both CD4 and CD8 T lymphocytes in the allografted mice.This model will be used for studying the mechanisms behind graft versus tumour effect (antigen mismatches, activation of T cell subpopulations) and the effects of immune suppressors (e.g. rapamycin) on the graft versus tumour effect.

P.08. Bone marrow-derived mesenchymal stromal cells failed to prevent experimental xenogeneic graft-versus-host disease

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Background
Graft-versus-host disease (GVHD) is a life-threatening complication