Additional serologic T-cell activation markers (Fractalkine (CX3CL1), ENA-78 (CXCL5), Eotaxin-3 (CCL26), and TRAIL) were measured in 26 patients using multiplex-analyses. In general, no differences were found between sensitive and refractory GVHD patients at escalation of immunosuppressive therapy, whereas late values were significantly higher in sensitive as compared to refractory patients for ENA-78 and TRAIL with a similar trend in Eotaxin. Although patient numbers were low, the results support the findings with sFASL indicating that refractory patients did not have stronger T-cell activation than sensitive patients. The lower serum levels of most T-cell markers in the late (d20–90) period following start of immunosuppression in refractory GVHD suggest that salvage immunosuppressive regimens were efficiently suppressing the T-cell arm of the immune response.
Figure S1. NRM (A) and OS (B) measured from onset of GVHD
Steroid-refractory disease was associated with significantly higher NRM (p<0.0001, HR 27.8 95% CI 4.2-185.6), translating into a significantly worse overall survival of patients with refractory GVHD (p=0.0003 HR 13.9 95% CI 3.3 to 58.7)