

surrogate marker CD244 was performed on muscle tissue. For clinical evaluation serum creatine kinase (s-CK) and functional index (FI) of myositis was used.

Results Patients significantly improved in Functional Index following treatment ($p=0.002$) but only one patient regained 100% muscle function. Serum CK-levels went back to normal in all patients after treatment ($p=0.004$). The CD28^{null} T cell frequencies were increased or unaffected for majority of the patients, 11 out of 14, which was statistically significant ($p<0.05$). The proportion of T_{regs} did not differ before and after treatment at group level, but for the majority of the patients the frequency was lower or unaffected (10 out of 14).

Conclusions Despite normalised CK-levels, patients only show partial functional improvement and many displayed persistent T cells in muscle tissue post-treatment. The relative number of regulatory T cells was unchanged or decreased, while the CD28^{null} T cell proportion was mainly increased post-treatment suggesting that high doses of glucocorticoid treatment might impair the regulation of autoreactive/pathogenic T cells including the CD28^{null} T cell populations in affected muscle.

5 PERSISTING CD28^{NULL} T CELLS, BUT NOT REGULATORY T CELLS, IN MUSCLE TISSUE OF MYOSITIS PATIENTS AFTER IMMUNOSUPPRESSIVE THERAPY

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Introduction Polymyositis (PM) and dermatomyositis (DM) are characterised by infiltration of macrophages and T cells in skeletal muscle tissue. Immunosuppressive treatment has limited effects on the number of infiltrating cells providing a basis for persistent immune reaction targeting muscle fibers. Regulatory T cells are key players in the maintenance of peripheral tolerance by controlling T cell reactivity to self-antigen. CD28^{null} T cells are a highly enriched subset of proinflammatory T cells in patients with autoimmune diseases and are suggested to be resistant to apoptosis. Our aim was to establish whether the persisting T cells in myositis tissue belong to the regulatory T cell subset or to the apoptosis resistant, proinflammatory CD28^{null} T cell subset.

Method Muscle tissue biopsies were obtained from 14 patients with PM/DM before and after 8 (4–16) month of treatment with glucocorticoids and additional immuno-suppressive drugs. Immunohistochemistry for CD3, FOXP3 and CD28^{null}