Fascial fibroblasts were obtained from fascia biopsy of a patient with EF and were stimulated with pre- and post-treatment serum of a patient with EF and conditioned medium of fascial fibroblasts by treatment for 4 and 10 weeks, compared to before treatment. Finally, fascial fibroblast proliferation was significantly increased by stimulation with IL-4. Furthermore, infiltration of CCR3-positive T cells was specific to the fascial tissue of EF. Therefore, we focused on fascial fibroblasts and aimed to determine the role of interleukin-4 (IL-4) in eosinophil and helper T cell infiltration and fibroblast proliferation.

Methods: Fascial fibroblasts were obtained from fascia biopsy of a patient with EF, and were stimulated with pre- and post-treatment serum of a patient with EF and healthy control, followed by microarray to analyze gene expression.

Results: By microarray analysis, CCL7 and CCL11 expression of fascial fibroblasts was increased. In addition, TGF-β and periostin in IL-4 stimulated facial fibroblast conditioned medium were also increased. In treatment, TGF-β and periostin in EF serum were gradually decreased by treatment for 4 and 10 weeks, compared to before treatment. Finally, fascial fibroblast proliferation was significantly increased by stimulation with IL-4. Furthermore, infiltration of CCR3-positive T cells was specific to the fascial tissue of EF.

Conclusions: In IL-4, enhancement of the proliferation of CCR3 ligands, TGF-β, and periostin from fascial fibroblasts. As a result, it promotes the migration of eosinophils and CCR3-positive T helper cells to the fascia and fibrosis. These results suggest that inhibition of IL-4 pathway could be a novel strategy for eosinophilic fascitis.

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Figure 1. The relative expression of autophagy-related genes