Colonization of CF patients' upper airways with *S. aureus* contributes more decisively to upper airway inflammation than *P. aeruginosa.*

Janhsen WK, Arnold C, Hentschel J, Lehmann T, Pfister W, Baier M, Böer K, Hünniger K, Kurzai O, Hipler UC, Mainz JG (2016) Colonization of CF patients' upper airways with *S. aureus* contributes more decisively to upper airway inflammation than *P. aeruginosa. Med Microbiol Immunol* 205(5), 485-500.

Abstract

In cystic fibrosis (CF) patients' airways, inflammatory processes decisively contribute to remodeling and pulmonary destruction. The aims of this study were to compare upper airway (UAW) inflammation in the context of Staphylococcus aureus and Pseudomonas aeruginosa colonization in a longitudinal setting, and to examine further factors influencing UAW inflammation. Therefore, we analyzed soluble inflammatory mediators in noninvasively obtained nasal lavage (NL) of CF patients together with microbiology, medication, and relevant clinical parameters. NL, applying 10 mL of isotonic saline per nostril, was serially performed in 74 CF patients (326 samples). Concentrations of the inflammatory mediators' interleukin (IL)-1β, IL-6, IL-8, matrix metalloproteinase (MMP)-9, and its anti-protease TIMP-1 were quantified by bead-based multiplexed assay, neutrophil elastase (NE) via ELISA. Culture-based microbiology of the upper and lower airways (LAW), as well as serological and clinical findings, were compiled. Our results

indicate that UAW colonization with S. aureus significantly impacts the concentration of all measured inflammatory mediators in NL fluid except TIMP-1, whereas these effects were not significant for P. aeruginosa. Patients with S. aureus colonization of both the UAW and LAW showed significantly increased concentrations of IL-1β, IL-6, IL-8, MMP-9, and slightly elevated concentrations of NE in NL fluid compared to non-colonized patients. This work elaborates a survey on S. aureus' virulence factors that may contribute to this underestimated pathology. Serial assessment of epithelial lining fluid by NL reveals that colonization of the UAW with S. aureus contributes more to CF airway inflammatory processes than hitherto expected.

Involved units

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