Sleep-disordered breathing is associated with fibrosis and impaired diastolic function

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Funding Acknowledgements: Type of funding sources: Public hospital(s). Main funding source(s): University Hospital of Regensburg

Background: Emerging evidence is linking sleep-disordered breathing (SDB) to diastolic dysfunction. SDB-induced hypoxia may stimulate the development of cardiac fibrosis which then causes diastolic dysfunction. We investigated the association between SDB and myocardial collagen and PDGF mRNA levels and how collagen mRNA levels are linked to parameters of diastolic dysfunction.

Methods: RA biopsies were collected from 31 patients undergoing elective coronary artery bypass grafting in the prospective observational study CONSIDER-AF. Tissue mRNA levels of collagen type I (Col1A), PDGFα, and PDGFβ were quantified using real-time qPCR on ViIA 7 real-time PCR system. NT-pro-BNP levels were measured in the serum via ELISA blots. The apnea-hypopnea-index (AHI) was registered in portable SDB-monitoring. Echocardiography was performed to assess diastolic function.

Results: First, we investigated an association of SDB and cardiac fibrosis. AHI was correlated with tissue mRNA levels of Col3a (p = 0.0239, Fig A), PDGFα (p = 0.0414, Fig B), and PDGFβ (p = 0.0024, Fig C). Then, we investigated an association between cardiac fibrosis and diastolic dysfunction. Tissue mRNA levels of Col3A were inversely correlated with lateral (p = 0.0196, Fig D) and septal E’ (p = 0.0006, Fig E) as a marker for diastolic relaxation. Higher ratios of E/E’mean were correlated with increased tissue mRNA levels of Col3A (p = 0.0368, Fig F). NT-pro-BNP which is a diagnostic criterion for heart failure with preserved ejection fraction was correlated with Col3 levels (p = 0.0247, Fig G).

Discussion: Our findings indicate that SDB is linked to fibrosis which is associated with diastolic dysfunction.