Myocardial fibrosis identified as new therapeutic target

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Myocardial fibrosis could be a future therapeutic target after researchers found it correlated with adverse cardiovascular outcomes in patients with obstructive sleep apnoea (OSA) referred for cardiac magnetic resonance (CMR). The study was presented today at EuroCMR 2016 by Dr Yaron Fridman, a cardiology fellow at the Heart and Vascular Institute, University of Pittsburgh Medical Center in Pittsburgh, Pennsylvania, US.

Dr Fridman said: "Fibrosis in the heart disrupts its normal architecture and function ultimately leading to cardiovascular morbidity and mortality. Patients with cardiovascular disease are at two to three times higher risk of having OSA. We hypothesised that quantifying myocardial fibrosis in patients with OSA could help identify those at increased risk of adverse cardiovascular outcomes."

The study included 1 094 patients who had been referred for CMR because of known or suspected heart disease. Of these, 324 had OSA and 770 did not have OSA. Sixteen healthy patients were included as a control group. Increased myocardial fibrosis was defined as an extracellular volume fraction (measured by CMR) of 30% or greater.

The investigators found that 30% of patients with OSA had a high degree of myocardial fibrosis. This was higher than healthy controls but similar to patients referred to CMR with known or suspected heart disease. After a median follow up of 2.3 years, there was no difference in the rates of heart failure hospitalisation or death between the OSA and

non-OSA groups. However, in patients with OSA, myocardial fibrosis was significantly associated with increased event rates, even after adjusting for age, kidney function, myocardial infarction size, and ejection fraction. In fact, each 5% increase in extracellular volume fraction was associated with a 1.6 times higher risk of heart failure hospitalisation or death.

Dr Fridman said: "Myocardial fibrosis was prevalent in patients with OSA. We found that we could quantify the amount of myocardial fibrosis and stratify patients' risk of poor outcomes. This is a powerful and robust way of identifying OSA patients who may be at higher risk for adverse cardiovascular events."

"The findings are particularly exciting because they identify a potential imaging biomarker for a pathological process occurring in the myocardium itself," continued Dr Fridman. "When there is myocardial fibrosis and the extracellular matrix is abnormal, the myocardium is less able to tolerate the vascular, ischaemic and haemodynamic insults seen in patients with OSA."

Dr Fridman said the findings point the way towards a personalised approach to treating cardiovascular disease based on the patient's amount of myocardial fibrosis. "We need to go beyond the traditional way of classifying and treating patients," he said. "For example, no two patients with OSA are alike, and yet we treat them the same. We assume that OSA always has the same effect on the heart, but that may not be true."

Dr Fridman continued: "Our research suggests that we should characterise patients with OSA, and other conditions, in more detail by assessing their amount of myocardial fibrosis. That may help us to be more precise in our treatment."

Dr Fridman concluded: "Large clinical trials are needed to determine if

using anti-fibrotic medications in patients with myocardial fibrosis identified by CMR improves cardiovascular outcomes. Ultimately, this is about better defining each patient's true risk and providing tailored treatment."

More information: Dr Fridman will present the abstract 'Myocardial Fibrosis is Prevalent in Obstructive Sleep Apnea and Associated with Hospitalization for Heart Failure or Death' during the session BEST Oral Abstracts which takes place on 13 May from 9:45 to 10:45 in the Main Auditorium.

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