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Differential association of proinflammatory cytokines with left ventricular diastolic dysfunction in subjects with and without continuous ambulatory peritoneal dialysis

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Abstract *Background and aims:* The association between inflammation and left ventricular (LV) diastolic dysfunction in continuous ambulatory peritoneal dialysis (CAPD) and non-CAPD patients is not established. The objective of this study was to test the above association and whether inflammation interacts with CAPD to increase LV diastolic dysfunction risks.
Methods and results: 120 subjects with normal creatinine levels and 101 CAPD patients were recruited. Echocardiographic parameters were assessed in all patients. The participants were classified as having LV diastolic dysfunction by echocardiographic findings including mitral inflow E/A ratio < 1, deceleration time > 220 cm/s, or decreased peak annular early diastolic

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velocity in tissue Doppler imaging. Blood was sampled at the baseline for measurement of inflammation markers, including tissue necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). Subjects with LV diastolic dysfunction had higher proinflammation cytokines levels in both groups. Inflamed markers correlated significantly with echocardiography parameters for LV diastolic dysfunction in patients receiving CAPD. In a multivariate regression analysis adjusting for all the factors associated with LV diastolic dysfunction, inflammation is still significantly associated with left ventricular diastolic dysfunction (TNF-alpha, OR: 2.6, 95% CI: 2.0—3.35, $p < 0.001$; IL-6, OR: 1.26, 95% CI: 1.25—1.26, $p = 0.01$). In addition, the interaction of CAPD and inflammation significantly contributed to the development of LV diastolic dysfunction (CAPD* TNF-α: OR: 1.45, 95% CI: 1.13—1.79, $P = 0.004$).

Conclusion: We found inflammation plays a vital role for LV diastolic dysfunction especially in CAPD patients. A synergistic effect between CAPD and inflammation, especially TNF-α, would further aggravate LV diastolic dysfunction.

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Introduction

According to cross-sectional and population-based studies, approximately 50% of patients with heart failure (HF) have a normal or near normal ejection fraction and, therefore, are referred to as having diastolic heart failure or heart failure with normal ejection fraction (HFNEF) [1—3]. Also, other observational studies have shown that the prognosis of patients with HFNEF appears to be similar to that of patients with HF and reduced LV ejection fraction [4,5]. Until now there has been much controversy about the pathophysiology of HFNEF. There are several mechanisms associated with LV diastolic dysfunction proposed, including impaired LV relaxation, increased LV passive stiffness, pericardial and endocardial disease, impaired neurohormonal regulation and even genetic factors [6—8]. However, so far, the mechanism of cardiac diastolic dysfunction is not fully understood.

Immunoinflammatory activation has been demonstrated to play a pivotal role in the development and in the progression of heart failure by some studies [9—11]. Through influencing cardiac contractility, inducing hypertrophy, and promoting myocyte apoptosis and fibrosis, it has been suggested that inflamed cytokines contribute to left ventricular remodeling and to the progression of heart failure [10,12—14,39]. Besides, circulating levels of tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) have been found to be elevated in direct relation to deteriorating functional class of heart failure, predicting a worse outcome. Such inflamed cytokines could promote and maintain inflammation locally and stimulate fibroblasts to produce collagen and subsequently lead to increased myocardial stiffness and diastolic dysfunction [10]. The association between plasma levels of TNF-α and IL-6 and diastolic heart failure in some specific groups, such as patients with coronary artery disease or patients with newly diagnosed systolic heart failure has been suggested recently [15,16].

In patients with renal failure, it is now widely accepted that there is a high prevalence of inflammation and oxidative stress, both of which are associated with the high rate of cardiovascular events [17—19]. Similar findings are reported by several groups in both hemodialysis (HD) and continuous ambulatory peritoneal dialysis (CAPD) patients alike [20]. In addition to inflammation, patients with renal failure suffered more for fluid overload, a high prevalence of hypertension and LV hypertrophy (which is a physiological response to pressure and volume overload) which all, it is suggested, contribute to the higher prevalence of LV diastolic dysfunction [21,22]. In CAPD patients, an association between inflammation and fluid overload has been suggested [23,24]. However, there is no published study on the relationship between inflammation and LV diastolic dysfunction in CAPD patients and whether anti-inflammation medication (e.g. statin) has a specific role in this group of patients remains to be determined.

Tissue Doppler imaging (TDI) has been a simple, reproducible and widely available noninvasive tool for the assessment left ventricular diastolic function. In 2007, the European Working Group on HFNEF proposed a new diagnostic algorithm which implemented TDI techniques as the basic tool to help diagnose LV diastolic dysfunction and HFNEF [25].

Therefore, this study is designed to explore the possible association between LV diastolic dysfunction and inflammation reflected by serum IL-6 and tissue TNF-α levels in CAPD patients and subjects with normal creatinine. We would also investigate whether there is a synergistic effect for inflammation and CAPD for LV diastolic dysfunction.

Methods

Study subjects

We had two study groups. The first group (group 1) consisted 120 subjects with normal renal function (serum creatinine $<$1.5 mmol) recruited sequentially from the outpatient clinic or the cardiovascular ward of National Taiwan University Hospital and its affiliates from July 2007 to March 2009. The second group (group 2) enrolled 101 patients consecutively that had received CAPD for more than six months at the same hospital. These participants received conventional glucose-based lactate-buffered CAPD solutions (UltraBag; Baxter Healthcare SA, Singapore). Patients who had hepatic disease, history of myocardial infarction, coronary intervention, cardiac myopathy, pericardial disease, significant valvular heart disease ($\geq$moderate), chronic obstructive pulmonary disease, chronic atrial fibrillation,

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