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COMMENTARY

A new character on the scene of cardiorenal syndrome

Francesco Natale, Emanuela Lo Priore, Luigi Aronne, Alessandro Siciliano, Maria Credendino, Chiara Cirillo, Chiara Granato, Paolo Calabrò, Maria Giovanna Russo and Raffaele Calabrò

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Renal dysfunction is related to increased arterial stiffness and left ventricular (LV) hypertrophy, which are two important risk factors for cardiovascular events. In addition, arterial stiffness and LV mass have been linked to each other and to early signs of renal damage, such as microalbuminuria.[1]

Arterial stiffness has a strong influence on the kidney because of the distinctive structure of the renal microcirculation. Certain tissues, such as the brain, heart, skin and skeletal muscle tissues, contain precapillary arterioles and metarterioles that disperse the majority of the mean and pulsatile energy content of the advancing pressure and flow waveform before it reaches the capillary. In contrast, glomerular capillaries are located between afferent and efferent arterioles. Because efferent arteriolar resistance is physiologically greater than afferent resistance, the mean and pulsatile pressures in the glomerulus are relatively elevated. This high hydrostatic pressure ensures the maintenance of an elevated glomerular filtration fraction, which is normally $\sim$20% of renal plasma flow, but exposes the glomerular capillary to potentially harmful pulsatile pressures if arterial stiffness and pulse pressure are high. Moreover, the myogenic tone of the afferent arteriole is influenced by pressure pulsatility.[2] Therefore, if the pulse pressure becomes greater than the mean pressure, renal vascular resistance will rise, and renal blood flow will fall.

The pathophysiology that links aortic stiffness to cardiovascular morbidity and mortality in hypertensive subjects may be associated with the reduced compliance of the large arteries that modifies the timing of wave reflections and thus determines ventricular load. The net effect of these hemodynamic changes may be ischemia, particularly in the subendocardium, which, if chronic, is associated with interstitial fibrosis and the development of heart failure.[3]

Chen *et al.*[4] have found that a left ventricular ejection fraction lower than 40% and an increased brachial-ankle pulse wave velocity were independently associated with declines in renal function and progression to a renal end point. In our opinion, these findings are relevant because they support the hypothesis that increased arterial stiffness is one of the mechanisms involved in the cross talk between the kidneys and the heart. Thus, as suggested by Chen *et al.*, cardiorenal syndrome may be renamed as cardiovascular renal syndrome.

According to the pathophysiology mentioned above, the treatment of arterial stiffness could be useful for improving the prognosis in patients with cardiorenal syndrome.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Dr F Natale, E Lo Priore, L Aronne, A Siciliano, M Credendino, C Cirillo, C Granato, P Calabrò, GR Maria and R Calabrò are at the Department of Cardiology, Second University of Study of Naples, Naples, Italy.
E-mail: natalefrancesco@hotmail.com