

Pulmonary Hypertension in Renal Disease: Epidemiology, Potential Mechanisms and Implications

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Commentary

By Professor Richard Glasscock

In a thought-provoking in-depth review Kawar and colleagues have provided a scholarly and thorough analysis of the confusing data and perplexing pathophysiology of pulmonary hypertension in subjects with end-stage renal disease treated with hemodialysis or peritoneal dialysis. Pulmonary hypertension (PH), defined as pulmonary mean arterial pressure of >25mmHg, at rest, as assessed non-invasively by echocardiography is relatively common in such patients, but the prevalence varies widely (from 30 to 60%). A variety of mechanisms have been proposed such as cardiac dysfunction and fluid overload, bone and mineral disorders, arterio-venous fistula effects, uremic 'vasculopathy' and adverse effects of bio-incompatible membranes, to name a few. However, PH, so defined, can occur prior to dialysis therapy and the prevalence may be somewhat lower in peritoneal than in hemodialysis-treated patients. Echocardiography alone may not be sufficient for accurate diagnosis, especially in fluid overloaded patients. Right heart catheterization (RHC), where pulmonary capillary resistances can also be determined, might be the 'gold standard' for diagnosis. Few studies have included this measurement, and when RHC has been performed the pulmonary capillary wedge pressure (PCWP) is elevated in the majority of cases, but some (around 10%) show a pre-capillary form of PH, not accompanied by elevated PCWP. Subjects with PH have poorer survival. There is no consensus on the best approach to diagnosis and no interventional studies have been reported. A major question is whether PH contributes undependably

to excess mortality or whether it is simply a 'marker' of the severity of underlying cardiac dysfunction. The heterogeneity of the pathophysiology of PH is an impediment to better understanding of its role in morbid and mortal events seen in dialysis patients.

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