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A Perspective on Controlling Diabetes-Related Cardiovascular Risk

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Introduction

The U.S. Renal Data System (USRDS) reported a decrease of 1303 patients joining the renal replacement treatment field between 2010 (n 14 116,946) and 2011, which was a reason for celebration in the kidney community (n 14 115,643). This demonstrates that not many DKD patients began renal replacement therapy. Better care, a difficult metric for this population, may be indicated by this finding. Despite having severe CKD, fewer diabetic patients with high levels of comorbidity may have been given the "nondialysis" strategy since the risk of death may be greater than the risk of ESRD.

Description

In the Medicare population with heart failure, angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB) are only prescribed to 44.2% of ESRD patients receiving hemodialysis. Patients without CKD, on the other hand, had a prevalence utilisation rate of 57.3 percent, whereas those with CKD had a rate of 52.0 percent. The utilization rate for advanced CKD stages 4 through 5 was just 42.4%. Beta-blockers and other medications are prescribed to a vulnerable population whose rates of heart failure and acute myocardial infarction are rising instead of using anti-RAAS therapy. Patients with chronic kidney disease (CKD) and those without heart failure have nearly identical medication usage data. There probably aren't many reasons for this, and none of them are good. To begin, there may be a reluctance to use these medications until the very end for fear of accelerating the rate at which kidney decline progresses.

Despite the fact that an increase in ACEI medication has been associated with a decrease in glomerular filtration rate (GFR), these phenomena are described in great detail. If the GFR drops significantly, bilateral renal artery stenosis may occur, but this is uncommon. They observed a particularly sensitive subset of CKD patients who experienced acute kidney injury following the start of anti-RAAS therapy. CKD of any kind and severity can result in a sudden drop in GFR. However, this is a small percentage of patients who receive anti-RAAS treatment. This is also true for DKD sufferers. It is necessary to anticipate and accept a certain amount of elevated serum keratinize.

Benazepril's efficacy in treating patients with serum certainties between 3 and 5 mg/dl was demonstrated by them. In fact, if the serum keratinise level does not rise, the doctor should consider the following: 1) Determine whether the patient's lack of medication adherence is linked to drug resistance; 2) Increase the dose; and 3) Determine whether another factor increased the GFR while anti-RAAS therapy decreased it. After all, the additional medication a patient must take and the price of that antihypertensive agent make the difference between achieving a systolic blood pressure of 130 mmHg and 140 mmHg. However, this additional generic blood pressure-lowering medication might only add $50 to $100 (US) to annual costs. At the level of proteinuria and kidney survival, the benefit of lowering blood pressure between 130 and 140 mmHg is not immediately apparent. Even though intense blood pressure reduction is ineffective in diabetes (140 vs. 120 mmHg for major cardiovascular events), continuous anti-RAAS therapy may still be beneficial. A reanalysis of the Action to Control Cardiovascular Risk in Diabetes-Blood Pressure trial, in particular, raises the possibility that lower blood pressure may improve cardiovascular outcomes.

A stroke is a common complication of diabetes and is frequently detected as a silent sign of diabetic vasculopathy on computed tomography scans. As a consequence of this, the index stroke event is frequently "missed," and it is concerning that we frequently fail to inform patients of this significant finding due to the absence of a "clinical event." in the trial of tria for the secondary prevention of strokes of the small subcortical brain. Even abnormalities in the diffuse white matter were evidence of previous cerebrovascular events. Although a blood pressure of 140 mmHg is all that is required to prevent stroke, continuous anti-RAAS blood pressure reduction may be beneficial. This is due to the fact that ACEI inhibition is approved as a method for preventing secondary strokes. Proteinuria is a major risk factor for CKD, even more so than high blood pressure, and its presence is crucial to the disease: Publication of Improving Global Outcomes. CGA classification system (albuminuria, GFR category, and cause). By lowering albuminuria and, consequently, interstitial inflammation—the most significant indicator of a kidney's longevity—anti-RAAS therapy has the potential to be beneficial. Importantly, the recently published JNC 8 guidelines recommend using anti-RAAS therapy with caution in the elderly due to the risk of hyperkalemia and elevated serum creatinine. Those with proteinuria and those who can tolerate anti-RAAS therapy may be at risk if this guideline is strictly followed [1-5].

Conclusion

To summarize, the gap in anti-RAAS therapy cannot continue to widen. Given the volume and strength of data indicating that anti-RAAS therapies slow the progression of both DKD and non-DKD29, we cannot afford to stop using them. Hyperkalemia should be looked into for reasons other than taking anti-RAAS medication. Increases in serum potassium and keratinize concentrations must be tolerated at higher thresholds. As anti-RAAS treatment lasts longer, hyperkalemia must also be treated more carefully when it occurs.

Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

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