Perspective
Volume 7:3, 2022

Clinical Gastroenterology Journal

ISSN: 2952-8518 Open Access

Action to Control Cardiovascular Risk in Diabetes

Richard Guyer*

Department of Digestive Diseases, University of South Florida, USA

Introduction

A cause for celebration in the kidney community, the U.S. Renal Data System (USRDS) revealed a drop of 1303 patients entering the renal replacement treatment arena between 2010 (n 14 116,946) and 2011 (n 14 115,643). This shows that few DKD patients started renal replacement treatment. This observation might indicate better care, a challenging metric for this population. However, fewer diabetic patients with high degrees of comorbidity may have been offered the "nondialysis" approach, despite advanced CKD, given that the risk of death may outcompete the risk of ESRD.

Description

Angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB) are only prescribed to 44.2% of ESRD patients receiving hemodialysis in the Medicare population with heart failure, according to the USRDS. In contrast, patients without CKD had a prevalent utilisation rate of 57.3%, while those with CKD had a rate of 52.0%. Stages 4 to 5 of advanced CKD had a utilisation rate of only 42.4%. Rather than using anti-RAAS therapy, beta-blockers and other drugs are prescribed in a vulnerable population in which acute myocardial infarction and heart failure rates are worsening. The medication usage data in CKD patients with or without heart failure are nearly identical. The reasons for this are likely few, and none of them are good. First, there may be reticence to use these drugs until the bitter end for fear of accelerating the progression rate of kidney decline.

Despite the fact that a reduction in glomerular filtration rate (GFR) has been noted since the introduction of ACEI medication, these phenomena in great detail. Bilateral renal artery stenosis may exist if there is a sharp drop in GFR, but it is typically absent. They observed a particularly sensitive subset of CKD patients who had acute kidney injury after initiating anti-RAAS therapy, CKD of any origin and of sufficient degree may trigger a sudden fall of GFR. However, this is the minority of patients treated by anti-RAAS therapy. This is also true of the DKD population. Some degree of serum keratinize elevation must be anticipated and tolerated.

They demonstrated the successful use of benazepril in patients with serum certainties of 3 to 5 mg/dl. In reality, the doctor should take these into account if the serum keratinise does not rise: (1) assess whether drug resistance is related to no adherence to medication by the patient, (2) increase the dose, and (3) determine if another factor increased the GFR as the anti-RAAS therapy decreased it. After all, the difference of achieving a systolic blood pressure of 130 mmHg vs 140 mmHg is an additional medication that a patient must take and the cost of that antihypertensive agent. However, the cost of this additional, generic blood pressure-lowering agent might only represent an additional $50 to $100 (U.S.) annually. The advantage of reducing blood pressure between 130 and 140 mmHg is not readily apparent at the glomerular level in relation to proteinuria or at the level of kidney survival. The continuous use of anti-RAAS therapy may still be beneficial even though intense blood pressure-lowering is ineffective in diabetes (140 vs. 120 mmHg for major cardiovascular events). In particular, a reanalysis of the Action to Control Cardiovascular Risk in Diabetes-Blood Pressure trial raises the possibility that better cardiovascular outcomes may result from lower blood pressure.

Stroke is a frequent complication of diabetes and is frequently seen on computed tomography scans as a silent sign of diabetic vasculopathy. As a result, the index stroke event is frequently "missed," and alarmingly, we frequently fail to advise patients of this important finding because no "clinical event" had place. In the tria for the secondary prevention of small subcortical strokes trial. even diffuse white matter abnormalities represented prior cerebrovascular events. Continuous anti-RAAS blood pressure lowering may be protective against stroke, however the blood pressure only needs to be lowered to 140 mmHg. This is because ACEI inhibition is approved as a measure for secondary stroke prevention. Even more than blood pressure, proteinuria is a major risk factor for the development of CKD, and its existence is essential to the Kidney Disease: Improving Global Outcomes publication. CGA classification scheme (cause, GFR category, albuminuria). Anti-RAAS therapy is potentially salutary by reducing albuminuria, and thereby interstitial inflammation, the most important predictor of a kidney's longevity. Importantly, the recently released JNC 8 guidelines advise caution in the use of anti-RAAS therapy in the elderly due to the risk of increasing the serum creatinine and hyperkalemia. Stringent adherence to this guideline may leave those with proteinuria and those who could tolerate anti-RAAS therapy at risk [1-5].

Conclusion

The gap in anti-RAAS therapy can no longer grow any larger, to sum up. We cannot afford to stop using anti-RAAS therapies given the strength and volume of data indicating it slows the progression of both DKD and non-DKD,29. In the event of hyperkalemia, reasons other than anti-RAAS medication should be investigated. It is necessary to raise the thresholds for tolerating increases in serum potassium and keratinize concentrations. As anti-RAAS therapy is prolonged, hyperkalemia must also be treated when it arises more carefully.

Acknowledgement

We thank the anonymous reviewers for their constructive criticisms of the manuscript. The support from ROMA (Research Optimization and recovery in the Manufacturing industry), of the Research Council of Norway is highly appreciated by the authors.

Conflict of Interest

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

References

1. Palmisano, Brian T., Lin Zhu, and John M. Stafford. "Role of estrogens in the regulation of liver lipid metabolism." *Clin Gastroenterol J* (2017): 227–256.
2. Barazzoni, Rocco, Gianluca Gortan Cappellari, Maurizio Ragni, and Enzo Nisoli. "Insulin resistance in obesity: an overview of fundamental alterations." *Clin Gastroenterol J* 23 (2021): 149-157.

***Address for Correspondence:** Richard Guyer. Department of Digestive Diseases, University of South Florida, USA, E-mail: richguyer21@tmh.org*

Date of Submission: 07 June, 2022, Manuscript No. cgj-22-74689; **Editor assigned:** 08 June, 2022, Pre QC No. P-74689; **Reviewed:** 13 June, 2022; QC No. Q-74689; **Revised:** 18 June, 2022, Manuscript No. R-74689; **Published:** 24 June 2022, DOI: 10.37421/2952-8518.2022.7.167

3. Bertoluci, Marcello Casaccia, and Viviane Zorzanelli Rocha. "Cardiovascular risk assessment in patients with diabetes." *Clin Gastroenterol J* 9 (2017) 1-13.

4. Drucker, Daniel J. "Diabetes, obesity, metabolism, and SARS-CoV-2 infection: the end of the beginning " *Clin Gastroenterol J* 3 (2021): 479-498.

5. Kulbay, Merve, Nathan Bernier-Parker, and Jacques Bernier. "The role of the DFF40/CAD endonuclease in genomic stability." *Clin Gastroenterol J* 26 (2021): 9–23.

How to cite this article: Guyer, Richard. "Action to Control Cardiovascular Risk in Diabetes." Clin Gastroenterol J 7 (2022): 167.