Contrast nephropathy: Risk factors and the role of beta blockers

Novel treatments and an understanding of CN are important to reduce the acute rates of hospitalizations and the increased risk of death associated with renal failure (1-4). I read with great interest the manuscript-published presented by Akgüllü et al. (3) entitled "A clinical study about contrast nephropathy: risk factors and the role of beta blockers." regarding contrast nephropathy as this outcome is associated with deleterious effects both economic and clinical, including prolonged hospital stays, increased readmission rates, increased morbidity and mortality, and acute care dialysis. The topic is timely and needs further study, which Akgüllü et al. (3) have provided. Some of the methodological approaches to the study were helpful in understanding the role of beta-blockers and CN. Approaches such as patients being well-hydrated, low osmolality solution, collection of baseline creatinine levels, and use of changes in these levels as a maker of CN, were helpful for pursuing answers to the study questions. The challenges of the study can be found in the dose of each beta-blocker, pretest differences between groups, and the statistical approach. Is there a dose-response relationship with 5 mg/day nebivolol, 25 mg/day carvedilol, and 50 mg/day metoprolol groups? The differing doses may be a significant confounding variable as stronger effects may have been exerted by larger doses, although no differences were found in CN between the different beta-blockers. Probably the greatest challenges to interpreting the study findings are the pretest differences in gender, hypertension, hyperlipidemia, family history, ejection fraction, contrast dosage, and some medications. These pretest differences can be risk factors for CN (gender, hypertension, hyperlipidemia) and renal failure, and may confound the understanding of the results (5, 6). Additionally, a different statistical approach, such as ANCOVA, for controlling serum creatinine, previous beta-blocker usage and diabetes status could have helped to ascertain the individual effects of each beta-blocker on CN. It should be noted that both family history and HDL were identified as risk factors for CN, but the odds ratio for HDL (1.005) suggests that the effect is relatively small. The family history variable (OR=3.159) shows more promise, but the pretest differences make the interpretation speculative. Additionally, it would have been helpful to measure C-reactive protein and uric acid levels to understand whether the pretest or post-test levels of inflammation have influenced the results of the study. It has been widely understood that nutritional status, hyperlipidemia, and inflammation, and possibly hyperuricemia interact significantly in various patient populations, and they may be a factor in CN at least as it is related to creatinine levels and kidney function (7). Finally, a stronger case for the need to compare the beta-blockers would have strengthened the study. Overall, the study was well-designed, and the conclusions reached by the authors of the study suggest novel findings that will contribute to the literature an understanding about the effects of various beta-blockers and risk factors for CN.

Rodney G. Bowden
Executive Associate Dean Brown Foundation Endowed Chair, Professor of Health Education, College of Health and Human Sciences, Baylor University; Texas-*USA*

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Address for Correspondence: Prof. Dr. Rodney Bowden, One Bear Place #97304
Waco, TX 76798-7304, Texas-*USA*
Phone: 254-710-3111 E-mail: Rodney_Bowden@baylor.edu
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