

## The role of nebivolol in the prevention of contrast-induced nephropathy

### *Kontrast nefropatisinin önlenmesinde nebivololün rolü*

I read with great interest the manuscript presented by Avcı et al. (1), regarding contrast-induced nephropathy. Renal insufficiency, hypertension along with preexisting diabetes are associated with contrast-induced nephropathy (CIN) and are of paramount importance in understanding how to prevent and mediate the effects of CIN (2). CIN is considered the third most common cause of acute renal failure (hospital acquired) after surgery and hypotension (3). Discovering new treatments such as nebivolol is necessary and warranted to avoid the seven-fold increase in mortality associated with CIN (4). The authors have chosen a timely and essential topic worthy of study. The challenges in this study concern the methodology associated with patient recruitment, timeline of data collection and the statistical analysis. The non-random assignment and unequal sample sizes along with relatively small sample size makes the inferences taken from this study somewhat speculative. Non-random assignment that was based on physician discretion only, may have introduced bias in the selection of patients for the study. If the physicians were not versed in the understanding of research and patient recruitment, they may have recruited in a way that introduced biases in the patient selection. A randomized or permuted randomized block procedure could have addressed the potential for bias and helped create equal sample sizes. Additionally some patients were previously prescribed and used a beta-blocker prior to study while others began using the beta-blocker only a few days prior to the study intervention. Since the study is about the use of a novel beta-blocker, it would have been helpful to control for this through a cluster randomization to assure an equal number of patients who previously used a beta-blocker was present in each beta-blocker group. Additionally, serum creatinine measures were taken 48 hours post intervention with some evidence suggesting the creatinine levels may peak at 72 hours and continue to decline for 10 days (5). The study may have missed the peak levels for creatinine. Also, the authors used an exclusion criterion of  $\geq 1.2$  mg/dl but there is some evidence that  $\geq 1.5$  mg/dl might be a better cut-point to demonstrate underlying chronic kidney disease (6). It is unknown how many

of the patients who had creatinine levels between 1.2-1.5 mg/dl were included in each group. Finally, an ANCOVA that took into consideration the level of serum creatinine, smoking status, previous beta-blocker use, diabetes, hyperuricemia, and metabolic syndrome could have helped to ascertain the individual effects of each beta-blocker and provided a good observation of how nebivolol might reduce CIN. Therefore, I think the findings should be taken in context based on the challenges with patient recruitment, methodology and statistical analysis. The authors chose a topic of great interest and need and I am appreciative of their work.

**Rodney G. Bowden**

**Associate Dean for Graduate Studies and Research  
Professor of Health Education, Baylor University  
Waco, Texas-USA**

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**Address for Correspondence/Yazışma Adresi:** Rodney G. Bowden, MD, PhD, Associate Dean for Graduate Studies and Research Professor of Health Education, Baylor University, Waco, Texas-USA Phone: +1 254 710 40 20 E-mail: rodney\_bowden@baylor.edu

**Accepted Date/Kabul Tarihi:** 05.09.2011 **Available Online Date/Çevrimiçi Yayın Tarihi:** 29.09.2011

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doi:10.5152/akd.2011.165