**Reviewer’s report**

**Title:** The Predictive Value of the Prognostic Nutritional Index for Postoperative Acute Kidney Injury in Patients Undergoing On-Pump Coronary Bypass Surgery

**Version:** 0  **Date:** 27 Jan 2019

**Reviewer:** GL Hicks

**Reviewer’s report:**

The paper for review is a single institutional retrospective observational study comprised of 336 consecutive patients with normal serum creatinine levels elective undergoing on-pump CABG. The purpose of the study was to identify postoperatively patients who developed AKI by Acute Kidney Injury Network (AKIN) criteria based on the occurrence of creatinine changes within the first 48 h after CABG surgery. The authors used common pre and postoperative variables as well as prenutritional index scores calculated using the following formula: PNI = serum albumin levels (g/dl) 

\[ \text{PNI} = \text{serum albumin levels (g/dl)} - 10 + \text{total lymphocyte count (per mm}^3) - 0.005. \]

The authors preoperative bias was that decreased immunonutritional status as indicated by PNI might influence postoperative outcome which in this study was the incidence of AKI.

AKI developed in 82 (24.4%) of all patients. In univariant analysis, the PNI was independently predictive of AKI (OR: 0.829, 95% CI: 0.783-0.877, p < 0.001). Moreover, C-reactive protein (CRP), a history of diabetes mellitus, and positive inotropric usage were independent risk factors for AKI. Using multivariant analysis adding PNI to the process improved greatly the discrimination of the predictive occurrence of AKI.

The authors concluded that decreased PNI could be associated with AKI.

The authors are to be congratulated on recognizing (as others have reported) a preoperative factor which can be a surrogate for poorer postoperative outcomes. Unfortunately the study has a binary conclusion, that is either a patient does or doesn't have AKI. Further, although PNI can be considered a marker of nutritional status the relative arbitrary limits of what is considered normal and abnormal leaves some area of gray. My questions for the authors are as follows:

1. There is a marked difference in mortality between AKI and non AKI patients, as well as double the rate of inotropic usage, and use of IABCP. This would indicate the AKI group had worse hemodynamics intra and postoperatively setting them up for AKI. How do the authors differentiate these factors from those related to PNI

2. In patients with low PNI as a separate analysis what were the results of subset analysis?
3. Unfortunately the authors did not include frailty indices as another indicator of preoperative status. Do they believe that low PNI and frailty are similar in their effects on outcome?

4. Believe there is an error in Table 1 as the incidence of IABCP usage in the nonAKI group was 5/254 or 1.9% not 6%

5. Given the authors conclusion about predictive value of PNI what chances have they made in their preoperative nutritional or physical rehab for patients to improve outcomes?

I would ask the statistical editor for the predictive power of the 84 patients in the AKI group as well as the selection of variables which have been well described in the past. I would reserve my acceptance of the paper for both this review and answers to the above questions

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