Contrast-induced acute kidney injury/ contrast-induced nephropathy may be related to additional risk factors

To the Editor,

We read with interest the article entitled "Impact of continuation of metformin prior to elective coronary angiography on acute contrast nephropathy in patients with normal or mildly impaired renal functions" by Oktay et al. (1) published in the Anatolian Journal of Cardiology. The authors investigated the association of metformin treatment with contrast-induced acute kidney injury/contrast-induced nephropathy (CI-AKI/CIN). In this study, they concluded that periprocedural metformin treatment in patients with type 2 diabetes mellitus undergoing elective coronary angiography (CAG) with normal or mildly impaired renal functions (eGFR >60 mL/min/1.73 m²) was reliable with respect to the development of CI-AKI/CIN and lactic acidosis.

The exact pathophysiological mechanism underlying CI-AKI/ CIN is still a matter of debate. However, it probably involves the direct toxic effect of contrast exposure (probably caused by high contrast media volume) and decreased renal medullary blood flow (particularly caused by heart failure, hypovolemia, hemodynamic instability, or anemia) that may contribute to the subsequent development of medullary ischemia and oxidative stress (2). In addition, a number of additional potential risk factors other than metformin use may also be attributed to CI-AKI/CIN development, an issue not discussed in the paper. It is of clinical importance to determine the contributions of hyperuricemia, hypoalbuminemia, and microalbuminuria to the development of CI-AKI/CIN.

In recent years, studies on the relationship between hyperuricemia and CI-AKI/CIN gradually appeared. In a meta-analysis of 18 relevant studies involving a total of 13,084 patients, subjects with hyperuricemia had a significantly increased risk of CI-AKI/ CIN regardless of whether the effect size was adjusted or not. In this report, hyperuricemia in the subjects undergoing CAG and/ or percutaneous coronary intervention resulted in significantly greater in-hospital mortality and incidence of CI-AKI/CIN requiring renal replacement therapy (3). Kumar et al. (4) reported that prophylactic oral administration of allopurinol (300 mg/day) was better than N-acetylcysteine (600 mg bd) alone or with saline hydration in the prevention of CI-AKI/CIN.

Hypoalbuminemia and microalbuminuria were also proven to be independent predictors of CI-AKI/CIN in patients with T2DM. In a study by Yang et al. (2), the incidence of CI-AKI/CIN in the positive urine albumin group was reported to be significantly higher than that in the trace and negative groups (positive vs. trace vs. negative: 18.5% vs. 6.7% vs. 3.9%, p<0.001).

To better elucidate the effect of metformin treatment on CI-AKI/CIN development in patients undergoing coronary procedures with normal or mildly impaired renal functions, the abovementioned confounding factors should have also been considered as they may contribute to the risk.

Hilmi Umut Ünal, Yalçın Başaran*, Hadim Akoğlu Departments of Nephrology and *Endocrinology, Gülhane Training and Research Hospital; Ankara-*Turkey*

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Address for Correspondence: Dr. Hilmi Umut Ünal,

Sağlık Bilimleri Üniversitesi, Gülhane Eğitim ve Araştırma Hastanesi, Nefroloji Kliniği, Etlik, Ankara-*Türkiye* E-mail: hilmiumut@hotmail.com ©Copyright 2018 by Turkish Society of Cardiology - Available online at www.anatoljcardiol.com DOI:10.14744/AnatolJCardiol.2017.66915

Author`s Reply

To the Editor,

We would like to thank the readers for their valuable comments related to our article entitled "Impact of continuation of metformin prior to elective coronary angiography on acute contrast nephropathy in patients with normal or mildly impaired renal functions" published in Anatol J Cardiol 2017; 18: 334-9 (1). Contrast-induced nephropathy (CIN) is associated with longer hospital stay and increased morbidity and mortality (2). Although the exact pathophysiology of CIN is not well-defined, several mechanisms such as renal medullary hypoxia, direct toxicity of contrast media, cytokine-induced oxidative stress, and inflammation have been proposed (3). As the readers have mentioned, multiple patient-related risk factors such as hyperuricemia, hypoalbuminemia, and microalbuminuria may contribute to the development of acute CIN after coronary angiography (CAG) (4, 5).

In our study, our main purpose was to evaluate the association between metformin continuation during CAG and CIN in patients with normal or mildly impaired renal functions. There-



fore, we excluded all patients with eGFR < 60 mL/min/1.73 m². In addition, the risk of CIN was assessed using the Mehran risk score, which was moderate. Unfortunately, the baseline patient characteristics in our study were relatively preserved in terms of renal functions, and as the number of patients with hyperuricemia was relatively limited (only six patients), we did not perform subgroup analysis for patients with hyperuricemia in terms of CIN. Moreover, in our study population, there were no patients with hypoalbuminemia. Hence, the impact of these risk factors on CIN mentioned by the readers need to be confirmed in further clinical trials aiming for this purpose.

Veysel Oktay, İlknur Çalpar Çıralı, Ümit Yaşar Sinan, Ahmet Yıldız, Murat Kazım Ersanlı Department of Cardiology, University of İstanbul, Institute of

Cardiology; İstanbul-*Turkey*

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Address for Correspondence: Dr. Veysel Oktay İstanbul Üniversitesi Kardiyoloji Enstitüsü Kardiyoloji Anabilim Dalı, Haseki, İstanbul-*Türkiye* Phone: 0212 459 20 00 E-mail: drvoktay@gmail.com ©Copyright 2018 by Turkish Society of Cardiology - Available online at www.anatoljcardiol.com

An unusual complication during reimplantation of implantable cardioverter defibrillator (ICD) after ICD leads extraction: Distal migration of anchoring sleeve

To the Editor,

In the last decade, the use of pacemakers and implantable cardioverter defibrillators (ICDs) has increased. Consequently, the number of device- or procedure-related events requiring system removal, such as lead failure or infection, has also increased. In the literature, procedure-related complications involving loss, unsuccessful, or incomplete removal of intravascular objects have been described (1-3). However, loss of the anchoring sleeve during pacemaker implantation is extremely rare. In this paper, we report our experience of distal migration of the anchoring sleeve during ICD implantations after lead extraction procedure.

A 72-year-old man with ischemic cardiomyopathy and a left ventricular ejection fraction as low as 25% was followed up for many years. He received a dual-chamber ICD for primary prophylaxis 6 years ago. He presented with elective replacement interval and lead failure due to retraction of the atrial and ventricular lead of his ICD. The passive fixation atrial and ventricular leads were planned to be removed and single-chamber ICD implantation was planned. The lead extraction was performed in the supine position under local anesthesia and light sedation with fluoroscopy guidance via the left subclavian vein. The next generation in mechanical lead extraction TightRail[™] Spectranetics system with firm steady traction the leads could be mobilized from the right atrium/right ventricular (RV) apex and removed. After the leads were removed, subclavian venous access was protected and bleeding was controlled. Then single-chamber ICD leads were inserted through the vascular sheath. Following placement of the RV lead, we realized that the anchoring sleeve in the RV lead had slid to the tip of the distal coil. Because of the risk of embolism, a new anchoring sleeve was positioned close to the lead connector and sutures were made at the site of introduction into the vein. The pulse generator was then connected to the electrode and secured in the pocket. Implantation was completed after the incisions were closed in layers. During the follow-up 1 year after the procedure, the sleeve was in the same position and the patient's clinical course was uneventful. To the best of our knowledge, this is the first case in which a distal migration of the anchoring sleeve occurred and had a permanent stable position without any complications.

Anchoring sleeves, which are composed of silicone rubber, secure the lead from moving and protect the lead insulation and conductors from damage caused by tight sutures at the site of introduction into the vein. Embolism and migration of lead fragments are well-known complications of lead extraction procedures, occurring in 0.1%–0.2% of these procedures (1, 4). However, the loss of the anchoring sleeve during pacemaker implantation is extremely rare. Mutual interference manipulations and maneuvers of leads may cause the distal migration the anchoring sleeve into the subclavian vein. Moreover, in our case, the especially large subclavian vein entrance because of lead extraction may have caused the problem. During pacemaker implantation, the operator should ensure that the anchoring sleeve is positioned close to the lead connector pin to prevent the inadvertent passage of the sleeve into the vein. However there is no data on the migration of the sleeve of the endocardial leads. Anchoring sleeves and outer insulation coating of endocardial leads are similar because both are composed of silicone rubber. Therefore, if the sleeve is stable and the risk of embolism is low, no problem may occur.