

Uric acid in diabetic nephropathy

Wen-Ling Lee^{a,b,c}, Peng-Hui Wang^{b,d,e,f,*}

^aDepartment of Medicine, Cheng-Hsin General Hospital, Taipei, Taiwan, ROC; ^bInstitute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan, ROC; ^cDepartment of Nursing, Oriental Institute of Technology, New Taipei City, Taiwan, ROC; ^dDepartment of Obstetrics and Gynecology, Taipei Veterans General Hospital, Taipei, Taiwan, ROC; ^eDepartment of Medical Research, China Medical University Hospital, Taichung, Taiwan, ROC; ^fFemale Cancer Foundation, Taipei, Taiwan, ROC

DEAR EDITOR,

We read Dr. Xia and colleagues' article published in the April issue of the *Journal of the Chinese Medical Association* with interest.¹ The author conducted a cross-sectional study to evaluate the correlation between serum uric acid level and two diabetes mellitus (DM)-related morbidities (diabetic nephropathy and diabetic retinopathy).¹ The authors found that serum uric acid level is independently associated with diabetes nephropathy as the authors found that DM patients who had a higher serum uric acid level (≥420 µmol/L for men and ≥ 360 µmol/L for women) also had a significantly higher prevalence of diabetes nephropathy, and a more severe proteinuria (high urinary albumin excretion) and worse renal function (lower estimated glomerular filtration rate).¹ We congratulate the success of the authors' publication, but we have some confusions about the current publication and hope to see the authors' kind response.

At first, this is a cross-sectional study, it is unknown whether a higher serum uric acid level is a cause or an end product in DM patients with diabetes nephropathy.¹ It may need a longitudinal study to give an answer. Of course, it is not easy to conduct a longitudinal study, but the authors could try to use the Cox proportional hazard methods or "receiver operating characteristic" (ROC) strategies to establish the correlation between serum uric acid level and proteinuria (qualitative data) or other targets, such as diabetes nephropathy and diabetes retinopathy. These methods are widely used to evaluate the correlation between studied items and targets.²-⁴ As shown by authors, decreased estimated glomerular filtration rate might also impair the urine excretion of uric acid. Therefore, it is hard to claim the higher serum uric acid is independently associated with diabetes nephropathy.

According to the different prevalence of diabetes nephropathy and diabetes retinopathy, the authors supposed that the

pathogenesis can differ. Could the authors kindly explain what does the different pathogenesis of diabetes nephropathy and diabetes retinopathy mean? We totally agree with the complex and complicated pathogenesis of DM-associated morbidities, but the threshold of end-organ damage is also different. For example, microvascular system damage occurs much early than macrovascular system does.

The above-mentioned questions do not criticize the scientific value of the authors' contribution, and we are looking forward to learning the authors' kind response.

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*Address correspondence. Dr. Peng-Hui Wang, Department of Obstetrics and Gynecology, Taipei Veterans General Hospital, 201, Section 2, Shi-Pai Road, Taipei 112, Taiwan, ROC. E-mail address: phwang@vghtpe.gov.tw; pongpongwang@gmail.com (P.-H. Wang).

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