International Journal of Preventive Medicine

Letter to Editor Open Access

Comment on: Prevention of Renal Damage by Treating Hyperuricemia

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How to cite this article: Nasri H, Ardalan M, Rafieian-Kopaei M. Comment on: Prevention of renal damage by treating hyperuricemia. Int J Prev Med 2015;6:10.

DEAR EDITOR,

A recently published article by Nickavar, entitled "prevention of renal damage by treating hyperuricemia in the esteemed "International Journal of Preventive Medicine" was read with great interest. She presented the child with proteinuria, hypertension, and glomerular sclerosis secondary to hypouricosuric hyperuricemia, who was treated by uric acid lowering management. They observed the decrement of proteinuria significantly by uric acid lowering agents.[1] At this paper, I would like to remember a few points. Recently, much attention had been paid toward the impact of hyperuricemia on kidney injury.[2,3] In addition to various well-known risk factors such as hypertension and diabetes, several "non-traditional" risk factors may contribute to the higher risk of renal injury compared to the general population.[4-8] In a study on 60 patients with type II diabetes without a history of gout, we detected a significant positive association between body mass index and serum uric acid levels ($r = 0.428$, $P = 0.001$). After adjustment for weight, a significant positive association of serum uric acid with level of proteinuria was seen ($r = 0.47$, $P < 0.001$) too,[9] moreover, the association of serum uric acid with level of blood pressure was significantly positive.[10] Accordingly, Jalalzadeh *et al.*, conducted a single-blind, randomized cross-over clinical investigation consisting 55 hemodialysis patients with serum uric acid level >6.5 (men) and >5.5 mg/dL (women).[11] They detected the a reduction of blood pressure by allopurinol treatment in hemodialysis patients.[11] Thus, likewise to the beneficial effect of Allopurinal in study of Nickavar, this study also revealed that allopurinol therapy may act as a new potential therapeutic approach to control of blood pressure in patients on hemodialysis.[12-14] Interestingly it was shown that treatment of hyperuricemia increases 1,25(OH) $_2$D levels, proposing that hyperuricemia may have a suppressive effect on 1-α hydroxylase activity.[15] It is possible that high plasma uric acid levels reduce 1,25(OH) $_2$D levels by suppressing 1-α hydroxylase and, while low 1,25(OH) $_2$D levels can stimulate parathyroid hormone (PTH) that high uric acid levels should be associated with elevated PTH levels too.[2,15-21] In accord with this hypothesis, Chen *et al.*, in *in vivo* study on Sprague Dawley rats, found uric acid suppresses 1-α hydroxylase *in vitro* and *in vivo*.[15] In fact, a reduction in 1,25(OH) $_2$D secondary to reduced 1-α hydroxylase enzyme activity contributes to the development of secondary hyperparathyroidism in patients with chronic kidney disease.[2,15-21] Indeed, the direct association of hyperuricemia and Vitamin D metabolism has two consequences. First, it is well understand that hyperuricemia by itself is an independent risk factor for kidney damage in various nephropathies like IgA nephropathy or diabetic kidney disease.[2,16-24] Many studies, support the hypothesis that elevated uric acid levels might have an injurious effect, resulting to inflammation, endothelial dysfunction, and vascular disease.[2,9,11,15-17] Second, except the deleterious effect of hyperuricemia on hypertension and aggravation of some type of nephropathies, its harmful effect of Vitamin D production is of significant importance.[15-23] Many studies had shown, support Vitamin D as a negative regulator of the circulating and local tissue renin-angiotensin system (RAS), since RAS has a critical implication in the physiology of sodium and volume homeostasis, hence, excess activity of RAS is associated with high blood pressure, kidney disease,

and diabetes.[15-31] Thus, the cast presented by Nickavar, further supports the implication of hyperuricemia in the aggravation of kidney damage.

Received: 08 Mar 14 **Accepted:** 07 Aug 14
Published: 20 Feb 15

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DOI:
10.4103/2008-7802.151815