

Submission No. : AAJS-0001

Session Title : APSN-ERA Joint Symposium

Session Topic : -

Date & Time, Place : June 16 (Sun) / 08:30-10:00 / Room 3 (GBR 104-105)

Genetic- and Kidney Disease-related Asymptomatic Hyperuricemic Mechanisms and Clinical Implications

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Uric acid (UA) is the final product of purine metabolism in humans and primarily excreted by the kidney via urate transporters. Hyperuricemia (HU) is defined as an elevated serum UA level, usually greater than 7.5 mg/dl in men and 6.5 mg/dl in women. Most people with HU are asymptomatic. Factors that contribute to HU include environmental and genetic factors. Previous GWAS have identified urate transporters as being involved in the process of UA excretion and in HU. Similar to dysfunctional variants in urate transporters, genetic risk variants in metabolism, as in Lesch-Nyhan syndrome, lead to rare pediatric syndromes of HU with early-onset of gouty arthritis and kidney stones. However, these genetic HU-related disorders account only for 10% of HU, while the majority develops HU as a result of impaired kidney UA clearance. So far, a causative role has only been documented for gouty arthritis, kidney stones and acute urate nephropathy. In contrast, asymptomatic HU has been implicated as potential risk factor for numerous disorders, such as metabolic syndrome, cardiovascular disease and CKD progression. So far, it has been controversially discussed as to whether asymptomatic HU is linked to these disorders by causation. Recent experimental and clinical evidence did not find urate-lowering therapy to delay CKD progression and hence disproved a causal link between asymptomatic HU and CKD progression but rather points toward an immunoregulatory role of soluble UA. In my lecture, I will summarize the latest findings on genetic- and kidney disease-related HU, and discuss a series of problematic assumptions on the differential effects of soluble versus crystalline UA, and provide evidence on the role of asymptomatic HU in CKD progression and the secondary immunodeficiency related to kidney disease. And finally, I will talk about potential therapeutic approaches on when and how to treat asymptomatic HU in patients with CKD.



APCN & KSN 2024

June 13(Thu) - 16(Sun), 2024 Coex, Seoul, Korea



*Promoting Sustainable Kidney Health:
The Asia-Pacific and Beyond*

Keywords: Kidney disease, Hyperuricemia, Inflammation