

Mini-report on the association between serum uric acid and incident metabolic syndrome

Eiji Oda*

Medical Check-up Center, Tachikawa General Hospital, Asahioka 1-24, Nagaoka, Niigata, 940-8621 Japan

Metabolic syndrome (MetS) is a constellation of interrelated metabolic risk factors that appear to directly promote the development of cardiovascular disease (CVD) and may be a systemic manifestation of adipose tissue dysfunction characterized by an increased aggregation of activated macrophages into adipose tissue induced by chronic energy overload, which is related to many other complex pathophysiological mechanisms including insulin resistance [1]. Many epidemiological studies have been reported regarding the association between serum uric acid (SUA) and CVD including MetS [2-8]. Some studies that have adjusted for multiple risk factors suggest that SUA is an independent risk factor of CVD [9-12] and increasing evidence suggests that SUA may contribute to the development of MetS. The increased SUA levels observed in MetS has been attributed to hyperinsulinemia because insulin reduces the renal excretion of SUA [13]. However, hyperuricemia often precedes the development of hyperinsulinemia [8,14]. Despite many epidemiological studies have demonstrated a cross-sectional association between SUA and MetS [15-18], longitudinal studies regarding baseline SUA as a predictor of incident MetS are limited [19-21]. The author reported that baseline SUA is an independent predictor of incident MetS in a Japanese health screening population [22].

The multivariable adjusted hazard ratios (HRs) of incident MetS through three years were calculated for each 1 SD increase in baseline SUA, for the higher quartiles of baseline SUA compared with the lowest quartile, and for baseline hyperuricemia defined as ≥ 7.0 mg/dL for men and ≥ 6.0 mg/dL for women in apparently healthy 1,606 men aged 51.7 ± 9.4 years and 953 women aged 51.6 ± 9.4 years who visited a medical check-up center in Japan.

The HRs (95% confidence interval; p value) were 1.282 (1.097-1.499; 0.002) in men and 1.354 (1.041-1.762; 0.024) in women for 1 SD increase in baseline SUA, 2.206 (1.344-3.620; 0.002) in men and 3.110 (1.121-8.627; 0.029) in women for the highest quartile of baseline SUA compared with the lowest quartile, and 1.900 (1.376-2.622; <0.001) in men and 2.088 (1.040-4.190; 0.038) in women for baseline hyperuricemia adjusting for the pre-existing components of MetS, age, smoking, drinking, physical activity, use of antihypertensive, antihyperlipidemic, and antidiabetic medications and histories of coronary heart disease and stroke. However, no significant association was found between longitudinal changes in SUA and incident MetS.

The above results suggested that baseline SUA is an independent predictor of incident MetS. Animal studies show that decreasing SUA levels can prevent or reverse features of MetS [23-25]. Management of hyperuricemia may be beneficial to prevent individuals from these CVD-related morbid conditions. However, there is not an enough evidence to support SUA lowering therapy for non-gout individuals with hyperuricemia [26].

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*Correspondence to: Eiji Oda, Medical Check-up Center, Tachikawa General Hospital, Asahioka 1-24, Nagaoka, Niigata, 940-8621 Japan, E-mail: ijie@venus.sannet.ne.jp

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