The Updated Strategies in the Management of Hyperuricemia and Gout in Taiwan

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There is a significant advancement in the treatment of hyperuricemia and gouty in the current Taiwan, but gout was only a rare disease before the World War II. The first case was diagnosed in 1915, and the second was a tophaceous gout described by a Japanese Dr Tsuda in 1920. However, the case number increased dramatically after the War II, and it was majorly due to changing life style. A large-scaled community-based cohort study in a remote Kinmen island found the transformation with younger onset, increased female patients, and more with metabolic syndrome but less severe tophaceous gout. The most recent studies disclosed that the prevalence of hyperuricemia is around 20%, and gout 1-2%. The prevalence rate is much higher among aborigines, which genetically belong to Malayo-Polynesian, around 50% for hyperuricemia and 10% for gout. The studies found Atayal familial gout locus linked to ADMCKD on 1q21 region, HGPRT (Tsou) gene, and reduced fractional UA clearance, which connect to gout and hyperuricemia in aborigines. A prospective cohort study from the nationwide MJ Health Screening Centers reported hyperuricemia was an independent risk factor of mortality from all causes, total cardiovascular disease and ischemic stroke. Another study from databank of CGMH found that hyperuricemia is strongly associated with chronic kidney disease (CKD). Furthermore, from our national Longitudinal Health Insurance Database (LHID), gout linked to cancer risk and increased mortality from cardiovascular, endocrine and renal causes.

National guidelines for gout and hyperuricemia was developed. Asymptomatic hyperuricemia was suggested to trace the underlying predisposing factors, like diuretics, anti-TB drugs or changing life styles, and treatment of metabolic syndrome. As a gold standard, acute gout was treated by NSAIDs and/or colchicine, or corticosteroid local injection. Chronic gout was treated by uricosuric agents or xanthine oxidase inhibitors to keep the SUA less than 6mg/dL. Allopurinol hypersensitivity was not uncommon in the daily practice, some developed Stevens-Johnson syndrome or toxic epidermal necrolysis. The pharmacogenetic study found that HLA-B5801 highly related to these side effects in Chinese populations. Febuxostat, a nonpurine xanthine oxidase inhibitor, safer for renal and liver dysfunction, is under investigation. The newly developed recombinant urate oxidase, like Rasburicase and PEGylated uric oxidase are now used for tumor lysis syndrome or refractory gout.

The advancements of biomolecular engineering have created many biological agents. IL-1β is the most important pro-inflammatory cytokine in MSU crystal-induced acute inflammation. The clinical

trials of two IL-1 inhibitors, IL-1β fully humanized monoclonal antibody (canakinumab), and a fusion protein IL-1 Trap (rilonacept) provided promising data in prophylaxis or treatment of acute flare-up in chronic gout. Targeting the inflammasome activation may be the direction for the future gout therapy and prevention.