中文題目：高尿酸血症增加低風險 ST 段上升心肌梗塞病人之一年死亡率

英文題目：**Hyperuricemia increased one-year mortality in patients with acute STEMI and low cardiovascular risk**

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Background

Hyperuriacemia as an independent predictor of short- and long-term prognosis in patients with myocardial infarction remained controversial. Hence, the study aimed to assess association between hyperuricemia and mortality in patients with ST-segment elevation myocardial infarction(STEMI) undergoing primary percutaneous coronary intervention(pPCI).

Materials and methods

In our five-year retrospective cohort, we analyzed 944 STEMI patients undergoing pPCI; with 88% male, the median of age 57 years and TIMI risk 4 in score. We divided these patient into hyperuricemia (uric acid >7mg/dl) and normal uric acid (uric acid ≤ 7mg/dl) groups. The study end point was six-month and one-year mortality.

Results

Baseline characteristics were similar in two groups except creatinine. Patients with hyperuricemia (n=293) had higher six-month (8.2% vs. 4.2%, P=0.019) and one-year mortality (9.6% vs. 5.0%, P=0.014). In univariate analysis, hyperuricemia was associated with higher one-year mortality (Hazard ratio: 1.967, 95% CI: 1.18-3.27). However, after adjustment for age, gender, body mass index, and creatinine, hyperuricemia was no longer a significant risk factor. In the subgroup analysis divided by Killip's classification, hyperuricemia remained an independent predictor of one-year mortality in patients at Killip I, even after adjustment for potential confounders. However, the association is not significant in patients at Killip II to IV. There was a significant interaction between hyperuricemia and Killip's classification associated with one-year mortality (P for interaction=0.037). The same association was found between hyperuricemia and six-month mortality.

Conclusions

We suggest that the association between hyperuricemia and the outcome of STEMI patients is dependent upon disease-severity and has the greatest impact on low risk groups (i.e., patients classified as Killip I).