Letter to Editor

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2024.58.009099

Comment on: Association Between Urate-Lowering Therapy Initiation and All-Cause Mortality in Patients with Type 2 Diabetes and Asymptomatic Hyperuricemia

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ARTICLE INFO

Received: July 22, 2024

Published: August 07, 2024

Citation: Rimsha Bint-e-Hina and Anees Fatima. Comment on: Association Between Urate-Lowering Therapy Initiation and All-Cause Mortality in Patients with Type 2 Diabetes and Asymptomatic Hyperuricemia. Biomed J Sci & Tech Res 58(1)-2024. BJSTR. MS.ID.009099.

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A pervasive health concern, type 2 diabetes mellitus (T2DM) affects 9.3% of the global population as of 2019, with projections indicating that this figure will increase to 10.2% by 2030 and 10.9% by 2045. The substantial hazards to the quality of life and life expectancy of affected individuals are underscored by these figures, in addition to the well-established macro- and microvascular complications associated with T2DM [1]. The considerable association between hyperuricemia and an increased risk of developing T2DM is underscored by a 15-year cohort study conducted in the United States. Specifically, individuals aged 18–30 have a 1.87-fold higher risk, while US veterans with a history of gout have a 1.19-fold higher risk [2]. The prevalence of hyperuricemia among T2DM patients is highly variable, ranging from 10.7% to 45% [3].

The pathogenesis of T2DM and its complications is significantly influenced by oxidative stress. Various antioxidants, such as allopurinol, have potentially mitigated these effects. In particular, allopurinol has demonstrated efficacy in reducing inflammation and enhancing the expression of key regulators, indicating its potential to modulate oxidative stress and inflammation over two years [1]. According to an examination of prescriptions for urate-lowering drugs in Taiwan, benzbromarone and allopurinol account for approximately 92% of orders [2], with allopurinol being the most frequently prescribed agent. The utilization of allopurinol has been linked to a beneficial effect on long-term glycemic variability. This risk factor has recently been identified as independently associated with diabetes complications [1]. It is important to note that the risk of developing T2DM was reduced by 9% in individuals with asymptomatic hyperuricemia who took benzbromarone in comparison to those who took allopurinol [2]. Furthermore, allopurinol therapy has demonstrated a modest yet substantial impact on glycemic control, as exemplified by a small but significant increase in HbA1c levels in comparison to other treatment modalities [1].

The development of T2DM and pancreatic beta-cell dysfunction have been linked to elevated serum uric acid levels. Research has demonstrated that uric acid can potentially infiltrate pancreatic beta-cells, resulting in apoptosis, reduced insulin secretion, and inflammation. Urate- lowering pharmaceuticals may provide protection against these effects by reducing uric acid levels, potentially preventing the development of T2DM [2]. Additionally, mortality rates have been substantially reduced in comparison to those of non-initiators with the initiation of urate- lowering therapy, particularly among individuals without diabetes complications. This emphasizes the significance of the early implementation of urate-lowering treatment in managing diabetes [4].

Declaration of Interest Statement

Conflict of Interest

None.

Declaration

None.

Acknowledgments

None.

Funding

None.

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ISSN: 2574-1241

DOI: 10.26717/BJSTR.2024.58.009099

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