Effects of allopurinol, a xanthine oxidase inhibitor, on TNF-α-induced endothelial cells

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Tumor necrosis factor (TNF) is a kind of cytokine involved in infection protection and antitumor action by expressing cell adhesion molecules such as vascular cell adhesion protein-1 (VCAM-1), and by inducing apoptosis and inflammatory mediators. VCAM-1 is known to exacerbate cardiovascular disease and is a risk factor for cardiovascular disease events. Gout is not just a disease of the joints, it causes inflammation throughout the body and affects various organs. Since TNF-α is induced under inflammatory conditions such as hyperuricemia, we investigated an effect of allopurinol, a treatment agent for hyperuricemia, on VCAM-1. Human Umbilical Vein Endothelial Cells (HUVEC) were cultured confluently. Allopurinol (0.1-100 μM) was treated 20 minutes before TNF-α (10 ng/mL) exposure. The amount of VCAM-1 induced by TNF-α was evaluated using Western blotting. VCAM-1 protein levels in cultured HUVEC increased 24 hours after TNF-a exposure, which allopurinol suppressed the significantly ($p < 0.05$, $n = 4$). Allopurinol is thought to inhibit the induction of VCAM-1 by TNF-α and may decrease cardiovascular disease events.