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LETTER TO THE EDITOR

The Effect of Colchicine in Acute Myocardial Infarction

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Dear Editor,

Recently, an interesting study entitled "Colchicine Inhibits NETs and Alleviates Cardiac Remodeling after Acute Myocardial Infarction" was published in Cardiovascular Drugs and Therapy [1]. We congratulate the authors and would like to discuss some points about the effect of colchicine in acute myocardial infarction (AMI).

In this article, the authors found that colchicine inhibited cardiac inflammation and exerted its cardioprotective effects after AMI. They further showed that colchicine inhibited extracellular traps (NETs) formation could dramatically alleviate AMI-induced cardiac remodeling. It indicated that colchicine may also become a potential strategy to improve cardiac function after AMI [1]. Interestingly, in a rat model of AMI, short-term colchicine treatment after AMI attenuated the post-AMI inflammatory response and subsequent ventricular remodeling and dysfunction [2]. Moreover, in a randomized, double-blind trial involving patients recruited within 30 days after AMI, the results showed that patients who were randomly assigned to receive 0.5 mg of colchicine once daily led to a significantly lower risk of ischemic cardiovascular events than placebo [3]. It indicated that administration of colchicine elicits protective effects against AMI. However, time-to-treatment initiation and dose of colchicine after AMI need further explored.

In another randomized, double-blind, trial of low-dose colchicine (0.5 mg daily) or matching placebo in patients admitted with AMI, patients treatment with low-dose colchicine was safe and well tolerated, but was not associated with a significantly increased likelihood of achieving a C-reactive protein (CRP) level < 2 mg/L or lower absolute levels of CRP 30 days after an AMI [4]. It suggested that colchicine maybe not associated with lower risk of recurrent vascular events. In patients with a first episode of STEMI and occluded culprit coronary artery, high-dose colchicine (2-mg loading dose followed by 0.5 mg twice a day) given orally at the time of reperfusion for a short period did not reduce myocardial damage induced by ischemia-reperfusion and the resulting inflammation compared with placebo [5].

In conclusion, colchicine played a crucial role in AMI. Further studies exploring the timing, pharmacokinetics, and dose response of colchicine and other anti-inflammatory agents are needed to identify an effective therapy to reduce infarct size or limit remodeling. We hope that they will find our queries of interest for further experimental research on the field.

Declarations

Conflict of Interests The authors declare no competing interests.

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