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Letter to the Editor

Colchicine inhibits endothelial inflammation via NLRP3/CRP pathway

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Dear Editor,

Kajikawa et al. [1] reported that short-term administration of low-dose colchicine significantly alleviated endothelial inflammation with reduction of serum hs-CRP concentration in CAD patients. This finding may recognize a novel role of colchicine in anti-inflammatory atheroprotection, whereas the underlying mechanisms need further exploration.

Colchicine is implicated in various diseases through NLRP3 suppression. Martinez et al. [2] found that short-term colchicine therapy dramatically reduced the expression levels of IL-1β, IL-6 and IL-18 by blockade of NLRP3 inflammasome activation in ACS patients. Otani et al. [3] observed that colchicine mitigated NSAID-induced small bowel injury via repression of NLRP3 inflammasome activation and IL-β maturation. These observations suggest that colchicine can exert anti-inflammatory effects via NLRP3 inflammasome inactivation.

NLRP3 activity is positively associated with the levels of CRP expression and inflammatory response. Yan et al. [4] delineated that the stimulation of NLRP3 inflammasome increased CRP and MCP-1 levels, while NLRP3 knockdown reduced CRP and MCP-1 levels in HUVECs. Paul et al. [5] reported that NLRP3 activation elevated the production of IL-1β and IL-6 with up-regulation of CRP expression, which exacerbated atherogenesis. These findings indicate that colchicine may ameliorate endothelial inflammation through the NLRP3/CRP pathway. However, more experiments are necessitated to verify this view in the future.

Conflicts of interest

The authors report no relationships that could be construed as a conflict of interest.

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References

[1] M. Kajikawa, Y. Higashi, H. Tomiyama, T. Maruhashi, S. Kurisu, Y. Kihara, et al., Effect of short-term colchicine treatment on endothelial function in patients with coronary artery disease, Int. J. Cardiol. 281 (2019) 35–39.
[2] G.J. Martinez, S. Robertson, J. Barraclough, Q. Xia, Z. Mallat, C. Bursill, et al., Colchicine acutely suppresses local cardiac production of inflammatory cytokines in patients with an acute coronary syndrome, J. Am. Heart Assoc. 4 (2015), e002128.
[3] K. Otani, T. Watanabe, S. Shimada, S. Takeda, S. Itani, A. Higashimori, et al., Colchicine prevents NSAID-induced small intestinal injury by inhibiting activation of the NLRP3 inflammasome, Sci. Rep. 6 (2016), 32587.
[4] Y. Li, P. Wang, X. Yang, W. Wang, J. Zhang, Y. He, et al., SIRT1 inhibits inflammatory response partly through regulation of NLRP3 inflammasome in vascular endothelial cells, Mol. Immunol. 77 (2016) 148–156.
[5] P.M. Ridker, From C-reactive protein to interleukin-6 to interleukin-1: moving upstream to identify novel targets for atheroprotection, Circ. Res. 118 (2016) 145–156.

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