Published Online: 2026 January 4

**Letter**

# Colchicine and Chemotherapy Induced Cardiotoxicity: Emerging Evidence of Cardioprotection

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**Received:** 3 January, 2026; **Accepted:** 3 January, 2026

*Keywords:* Colchicine, Cardioprotective, Chemotherapy Induced Cardiotoxicity

*Dear Editor,*

We read with great interest the recent publication by Peng et al., which evaluated the cardioprotective effects of low-dose colchicine against doxorubicin-induced cardiotoxicity in experimental (both in-vivo and in-vitro) settings (1). Indeed, doxorubicin-related cardiotoxicity represents a multifactorial clinical process, primarily manifested by a decline in left ventricular ejection fraction and the development of cardiomyopathy (2). The major finding of Peng et al. was that low-dose colchicine enhanced autolysosome degradation; as a result, attenuation of doxorubicin-induced mitochondrial damage and a reduction in reactive oxygen species accumulation were observed. In addition, low-dose colchicine (0.1 mg/kg) was associated with improved myocardial structural organization and reduced pathological cardiac remodeling. Beyond these effects, colchicine has been reported to modulate calcium handling through its microtubule-disrupting properties, a mechanism that has been implicated in its anti–atrial fibrillation effects (1).

We were also interested in the recent research conducted by Safarpour et al., which examined the cardioprotective properties of colchicine against 5-fluorouracil (5-FU) induced cardiotoxicity. Five-fluorouracil caused significant cardiac injury, indicated by elevated levels of cardiac enzymes, cyclooxygenase-2 (COX-2), malondialdehyde, and tumor necrosis factor-α (TNF-α) expression, along with histopathological alteration. Furthermore, 5-FU disrupted electrocardiographic parameters, including ST-segment elevation and prolonged QRS duration. Data reported from this animal study indicate that treatment with colchicine attenuated these effects by reducing cardiac enzyme levels, oxidative stress, histopathological degeneration, and COX-2 expression in cardiac tissue. Moreover, colchicine ameliorated electrocardiographic abnormalities, increased circulating blood cell counts and total antioxidant capacity, and prevented treatment-associated body weight loss (3).

Furthermore, Hutchins et al. reported that colchicine has a cardioprotective role on anthracycline-induced cardiomyopathy (4). Extending the evidence regarding the cardioprotective role of colchicine, Kim et al. demonstrated that colchicine may also improve outcomes following pericardiocentesis in patients with malignant pericardial effusion. In their cohort, 91% of patients had a confirmed pericardial effusion secondary to malignancy (most commonly due to lung cancer (63%), with cardiac tamponade occurring in approximately 86% of cases). Significantly, most patients presented with advanced metastatic disease, underscoring colchicine's potential role in managing severe cancer-related cardiovascular complications (5).

While the mechanisms of colchicine remain partially understood, its well-established anti-inflammatory properties in cardiovascular disease, including pericarditis, post-operative atrial fibrillation, and



**How to Cite:** Alizadehasl A, Nemati A, Azarfarin A, Parsi-Moud A, Parsimood E. Colchicine and Chemotherapy Induced Cardiotoxicity: Emerging Evidence of Cardioprotection. Multidiscip Cardio Annal. 2025; 16 (1): e169330. https://doi.org/10.69107/mca-169330.

atherosclerosis, are of great significance (4). Recent studies have shown that colchicine's ability to downregulate inflammatory factors such as TNF-α, interleukin-1β (IL-1β), and IL-6 suggests a multifaceted role in cardiac protection (3-5). Colchicine permanently attaches to tubulin, preventing the formation of microtubules. This interferes with various cellular functions, such as neutrophil adhesion, the production of TNF-α, and the activation of the NLRP3 inflammasome (3). Given the growing interest in colchicine as a cardioprotective agent, we encourage the authors of these studies to present detailed data regarding the physiological pathway expressions in their experiments.

All in all, we congratulate the authors of these articles for elucidating the role of colchicine in the cardiotoxicity of chemotherapy drugs. We hope that future studies will examine the effect of colchicine on the cardiotoxicity of novel chemotherapy drugs and its effects according to patients' body weight, renal function, and the administration methods.

## Footnotes

**AI Use Disclosure:** The authors declare that no generative AI tools were used in the creation of this article.

**Authors' Contribution:** Az. A. and E. P. M. contributed to the conception of the letter and critical appraisal of the topic. E. P. M., Ar. A., and A. N. drafted the manuscript, and A. P. M. critically reviewed and revised it. All authors reviewed and approved the final version and agree to be accountable for all aspects of the work.

**Conflict of Interests Statement:** The authors declare no conflict of interest.

**Funding/Support:** The present study received no funding/support.

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