Beyond precision therapy: the need for precision clinical decision-making in recurrent pericarditis

Dear Editors,

We read with great interest the timely narrative review by Bizzi *et al*. (1), "The paradigm shift in corticosteroid therapy for idiopathic pericarditis: from high-dose regimens to precision therapy". The authors provide a timely and comprehensive overview of the evolution of corticosteroid therapy, thoughtfully outlining the transition from historical high-dose regimens to the contemporary low-dose, slow-tapering approach endorsed by the 2025 European Society of Cardiology (ESC) Guidelines (2). Their central message – that corticosteroids should be regarded as a precision tool rather than a broadly acting anti-inflammatory therapy – represents an important conceptual advance and offers a valuable framework for contemporary pericarditis management.

Despite these important strengths, one aspect deserves further consideration. Although the concept of precision therapy is consistently emphasised throughout the manuscript, its translation into everyday clinical decision-making could be further developed. Corticosteroids are repeatedly described as a precision intervention (1), and the authors discuss several clinically relevant determinants of treatment selection, including phenotype-based management and the consideration of anakinra when prolonged prednisone therapy at doses >10–15 mg/day is anticipated. However, these elements are not integrated into a structured decision framework that could guide clinicians in choosing between continued corticosteroid-based management, gradual tapering, and earlier escalation to interleukin-1 (IL-1) inhibition. Consequently, an opportunity remains to translate the concept of precision therapy into a more operational approach to individualised treatment selection.

This gap is clinically important because recurrent pericarditis is increasingly recognised as a heterogeneous autoinflammatory syndrome rather than a single disease entity (3). Therapeutic decisions are influenced by substantially more than corticosteroid dose or C-reactive protein normalisation. The inflammatory phenotype, recurrence burden, previous corticosteroid exposure, response to colchicine, contraindications to conventional therapy, anticipated duration of glucocorticoid treatment, patient age, pregnancy, access to biologic agents, and the risk of cumulative glucocorticoid toxicity may all contribute to selecting the most appropriate therapeutic strategy (2, 4). Although several of these variables are individually addressed throughout the review (1), their integration into a practical phenotype-based decision algorithm could further assist clinicians in translating the concept of precision therapy into individualised treatment decisions.

The discussion of IL-1 inhibitors further illustrates this issue. The review provides an excellent overview of both the efficacy and the current limitations of IL-1 inhibitors, including cost, reimbursement policies, accessibility and unresolved questions regarding treatment duration (1). Importantly, the authors acknowledge the differing positions of European and US recommendations and note that, in their clinical practice, anakinra is considered when prolonged prednisone therapy at doses >10–15 mg/day is anticipated. However, the broader question of which patients might benefit from earlier biologic intervention to reduce cumulative corticosteroid exposure remains less clearly defined. This question is becoming increasingly relevant as growing evidence suggests that prolonged corticosteroid dependence itself contributes to recurrent disease and treatment-related morbidity (5-7).

The next step in the evolution of precision medicine in recurrent pericarditis may therefore lie not only in further refining corticosteroid dosing and tapering, but also in translating existing phenotype-based principles into practical treatment algorithms that facilitate individualised therapeutic decision-making. Ultimately, precision medicine is achieved not simply by prescribing lower corticosteroid doses or tapering them more gradually, but by selecting the most appropriate therapeutic strategy for the individual patient. Building on the framework provided by Bizzi *et al*. (1), a more explicit integration of clinical phenotype, corticosteroid dependence and the appropriate timing of IL-1 inhibition could further strengthen the clinical impact of this excellent review and help bridge the gap between evidence and everyday clinical practice.

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