The paradigm shift in corticosteroid therapy for idiopathic pericarditis: from high-dose regimens to precision therapy

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ABSTRACT

Idiopathic and recurrent pericarditis are increasingly recognised as autoinflammatory conditions driven by the NLRP3 inflammasome and interleukin-1 signalling. Despite this shift in understanding, the misuse of high-dose corticosteroids has often led to a 'massacre of the innocents', transforming acute events into chronic, steroid-dependent diseases.

This narrative review analyses the evolution of corticosteroid use in pericarditis, emphasising the transition from aggressive loading doses to the low-dose, 'rheumatological' strategies codified in the 2025 European Society of Cardiology (ESC) guidelines.

Evidence confirms that medium-dose prednisone (0.2–0.5 mg/kg/day), when integrated into triple therapy with non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine, reduces recurrence rates and iatrogenic toxicity compared to traditional high-dose regimens (1.0 mg/kg/day). A critical challenge remains the tapering process; rapid withdrawal triggers inflammatory rebounds, necessitating meticulous, long-lasting (months-years), C-reactive protein (CRP)-guided reductions as small as 5 to 1.25 mg every two to six weeks, similar to the tapering used in polymyalgia rheumatica. Chronic maintenance with low-dose prednisone (≤5 mg/day) may be considered in selected cases, with a good risk-benefit balance. The elderly usually require medium dosages (e.g. prednisone 12.5–25 mg), pregnant women no more than 10 mg, while children should avoid chronic corticosteroid therapy. CRP-negative cases may benefit from low doses (5 mg). Given the long-term duration of therapy, bone protection therapy is essential yet underutilised, while proton-pump inhibitors should be used only for concomitant NSAIDs therapy. Corticosteroids are generally ineffective in chronic idiopathic pericardial effusions with normal CRP.

In conclusion, modern management redefines corticosteroids as a precision tool rather than a blunt anti-inflammatory agent.

Introduction

Idiopathic pericarditis is increasingly recognised as a clinical manifestation of an underlying autoinflammatory process (1-6). In the majority of cases of pericarditis in Western countries (7), particularly those classified as idiopathic or recurrent and with an inflammatory phenotype, the pathogenesis is driven by the activation of the NLRP3 inflammasome (8-10). This intracellular complex triggers the massive release of pro-inflammatory cytokines, most notably interleukin-1beta (IL-1beta) and interleukin-1alpha (IL-1alpha) (4, 11). This IL-1-mediated pathway creates a self-perpetuating loop of inflammation that explains the clinical hallmarks of the disease: the rapid rise in C-reactive protein (CRP), the systemic febrile response, and the high propensity for recurrence (12).

This paradigm shift in understanding the autoinflammatory nature of the disease has revolutionised the therapeutic landscape. While traditional treatments relied heavily on broad-spectrum anti-inflammatory agents, modern management now includes targeted biological therapies (13-18). Interleukin-1 (IL-1) blockers, such as anakinra and rilonacept, have emerged as highly effective options for patients with multiple recurrences and steroid dependence, offering a steroid-sparing effect that was previously unattainable (19-24). These therapies, alongside the systematic use of colchicine and high-dose non-steroidal anti-inflammatory drugs (NSAIDs), form the current standard of care aimed at quenching the autoinflammatory fire. However, despite these advances, the

historical reliance on corticosteroids remains a deeply rooted clinical practice (25, 26). To understand why corticosteroids continue to represent both a vital resource and a significant clinical challenge, it is necessary to examine the historical context of their use.

The therapeutic landscape of pericarditis has undergone a profound transformation over the last two decades (11, 27). We now recognise it as a complex disease where the initial management strategy determines the long-term prognosis. Central to this evolution is the role of corticosteroids, a class of drugs that acts as a double-edged sword, capable of rapid clinical remission but also responsible for the most challenging cases of treatment-dependent recurrence.

This manuscript is a narrative review with an expert clinical perspective focused on the evolving role of corticosteroids in recurrent pericarditis. The literature reviewed was identified through a targeted, non-systematic search of PubMed/MEDLINE, prioritising guidelines, randomised clinical trials, pivotal observational studies and clinically relevant reviews. In addition, the discussion is informed by the authors' clinical experience in referral centres managing patients with recurrent pericarditis.

The shift in guidelines: from high-dose to medium-dose corticosteroids as a second-line strategy

The historical management of pericarditis was long shadowed by what can be defined as the 'Original sin': the early recommendations for high-dose regimens recommended in the first guidelines, based on expert opinion, that stated: "*The recommended regimen is: prednisone 1–1.5 mg/kg, for at least one month. Corticoids should be tapered over a three-month period. If symptoms still recur, return to the last dose that suppressed the manifestations, maintain that dose for 2–3 weeks and then recommence tapering. A common mistake is to use a dose too low to be effective"(28).*

Accordingly, for years, clinicians followed protocols using prednisone doses of 1.0–1.5 mg/kg/day for at least one month, followed by quick reductions every 2–3 weeks (28). This approach, however, triggered a 'Massacre of the innocents'. High doses provided an illusion of control but any attempt at tapering led to a rebound in the inflammatory process, effectively transforming an acute event into a chronic, steroid-dependent disease (29, 30).

The turning point in evidence-based management came from the realisation that high doses are not only unnecessary, but also harmful. A pivotal study provided a stark comparison: patients treated with high doses (1.0 mg/kg) faced a recurrence rate of 64.7% and a 23.5% incidence of severe side effects. In contrast, those treated with lower 'rheumatological' doses (0.2–0.5 mg/kg) saw recurrence rates drop to 32.6% and side effects to a mere 2.0%. These findings demonstrated that a lower initial dose, coupled with a very slow tapering protocol, is far more effective in achieving permanent remission (31). In the review process the Journal asked the authors to propose a therapeutic protocol suggesting how to taper corticosteroids, and in the Editorial accompanying the published paper, entitled "*Corticosteroids for recurrent pericarditis; on the road to evidence-based medicine*", Shabetai recommended to follow this new protocol in clinical practice (32)

Consequently, the 2015 European Society of Cardiology (ESC) Guidelines took this protocol, and the recent 2025 update codified this paradigm shift (33, 34).

In detail, the protocol suggests avoiding high doses, starting with 0.2 to 0.5 mg/kg/day and recommend a tapering of 2.5 mg/day every 2–4 weeks for doses of prednisone-equivalent between 25–15 mg, and then a tapering of 1.25–2.5 mg/day every 2–6 weeks for doses of prednisone-equivalent <15 mg. This translates into a duration of tapering of at least 7 months, similar to polymyalgia rheumatica (Table I). Patients who develop recurrence during corticosteroid tapering should continue colchicine and receive an NSAID on top of this treatment at the maximal tolerated doses, instead of increasing the dose of corticosteroids. During the tapering NSAIDs should be maintained, particularly for doses lower than 12.5 mg daily.

Figure 1 illustrates the historical evolution of corticosteroid use in pericarditis management: the traditional approach, based on prolonged high-dose corticosteroid therapy, has progressively been challenged by evidence showing that patients treated with high doses, compared with those receiving lower doses, experienced higher rates of adverse effects, recurrences and hospitalisations. These observations prompted a progressive revision of international recommendations. The subsequent 2015 ESC guidelines shifted the therapeutic approach toward combination therapy, the use of low-to-moderate corticosteroid doses, and a slow tapering strategy. This evolution ultimately led to the contemporary phenotype-based management strategy incorporated into the most recent ESC guidelines.

In the ESC Guidelines corticosteroids are recommended as a second-line therapy, to be reserved for patients with contraindications to NSAIDs or those who fail to respond to first-line therapy (NSAIDs, colchicine and lifestyle restriction). In contrast the United States statement recommends avoiding corticosteroids, with a rapid transition to anti-IL1 agents (35). In clinical practice we consider the use of anakinra when chronic therapy with prednisone at a dosage >10-15 mg daily is anticipated.

Despite these clear mandates, a significant reality gap persists. In daily practice, many patients are still subjected to rapid tapering schedules that do not account for CRP levels or clinical stability, leading to preventable relapses.

The challenge of tapering: clinical inertia and the risks of rapid withdrawal

A critical discrepancy persists between international recommendations and real-world clinical practice regarding the discontinuation of corticosteroid therapy. While the ESC guidelines provide a clear framework for a gradual reduction (36), many practitioners still implement rapid or step-ladder tapering schedules that are disconnected from the patient's biological response. This clinical inertia often stems from a misplaced urgency to withdraw corticosteroids due to fear of long-term toxicity, yet ironically, an accelerated withdrawal is the primary trigger for inflammatory rebounds and the subsequent development of chronic,

Table I. Example of corticosteroid tapering in recurrent pericarditis according to the 2025 European Society of Cardiology Guidelines (34).

Prednisone dose	Duration	Time from baseline
25 mg/day*	2 weeks	-
20 mg/day	2 weeks	1 month
15 mg/day	4 weeks	2 months
10 mg/day	4 weeks	3 months
7.5 mg/day	4 weeks	4 months
5 mg/day	4 weeks	5 months
2.5 mg/day	4 weeks	6 months
2.5 mg every other day	4 weeks	7 months

*For a starting dose of prednisone 25 mg the total duration of tapering will last minimum 7 months, similar to polymyalgia rheumatica. During the tapering non-steroidal anti-inflammatory drugs (NSAIDs) should be maintained, particularly for doses lower than 12.5 mg daily.

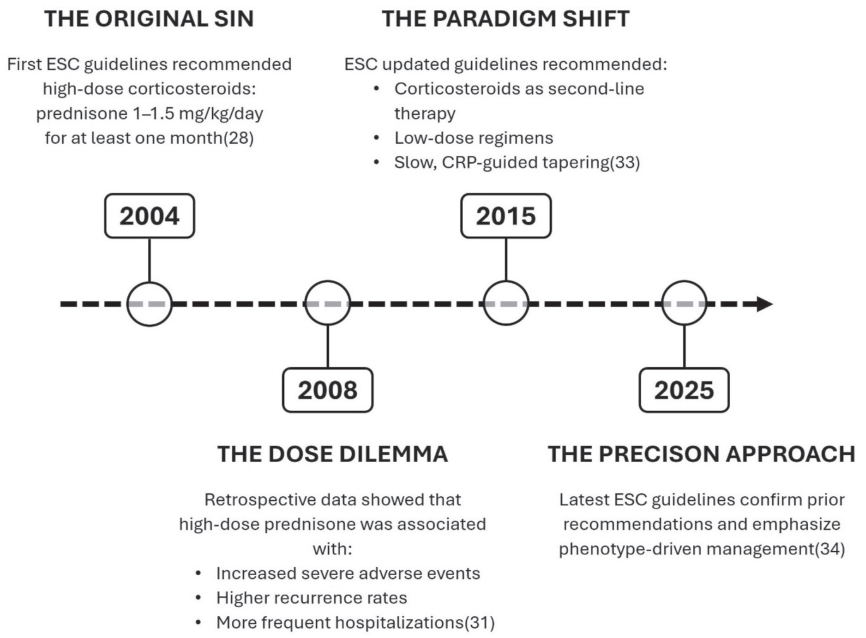

Fig. 1. The evolution of corticosteroid use in pericarditis management.
CRP: C-reactive protein; ESC: European Society of Cardiology.

recurrent pericarditis, with renewed use of corticosteroids.

The 2015 and 2025 guidelines emphasise that tapering must be a meticulous process, characterised by small dose decrements, often as low as 1.25 to 2.5 mg every 2 to 6 weeks, once the patient has achieved clinical stability. Crucially, this process should not be automatic. It must be strictly guided by the serial monitoring of CRP levels. A biologically guided tapering ensures that the anti-inflammatory coverage remains sufficient to suppress the autoinflammatory flare during the most vulnerable phases of treatment. To prevent the rebound phenomenon typical of autoinflammatory pericarditis, any symptom flare occurring during corticosteroid reduction should be addressed by increasing NSAID therapy, making every effort not to increase again the dose of prednisone. This strategy is crucial to maintain the steroid-sparing trajectory and break the cycle of recurrence (37).

In clinical reality, many relapses occur because the dose is reduced while CRP levels are still fluctuating or when the reduction exceeds the critical threshold (typically around 10–15 mg/day of prednisone). Failure to adhere to these slow, CRP-monitored protocols effectively transforms a manageable acute condition into a refractory disease state, necessitating higher cumulative doses of corticosteroids over time. Therefore, the success of the therapy depends less on the speed of withdrawal and more on the precision of the tail-end of the tapering schedule.

The role of combination therapy: maximising synergy to ensure remission

The success of modern pericarditis management relies not only on the careful titration of corticosteroids but, more importantly, on their integration into a comprehensive multidrug regimen. The contemporary therapeutic strategy is built upon the synergy between high-dose NSAIDs, colchicine and low-dose corticosteroids. This triple therapy is designed to attack the inflammatory process through multiple pathways simultaneously, thereby allowing for the use of the lowest possible corticosteroid dose and facilitating a smoother tapering process (4, 11, 38).

Colchicine plays a pivotal role in this setting, acting as a potent adjunct that significantly reduces recurrence rates and enhances the response to treatment. By inhibiting microtubule polymerisation and NLRP3 inflammasome activation, colchicine addresses the autoinflammatory core of the disease, providing a protective effect that is essential when attempting to reduce corticosteroid coverage (6, 39, 40). Furthermore, the systematic use of full-dose NSAIDs during the initial phase of treatment ensures adequate anti-inflammatory saturation. When these agents are used correctly in combination, corticosteroids can truly function as second-line support rather than the primary driver of the treatment, minimising the cumulative exposure and the associated iatrogenic risk (36).

In clinical practice, a therapy not according to the ESC guidelines of the first episodes is a clear risk factor for recurrences and chronicity (1, 41).

Duration of therapy

When corticosteroids are started in recurrent pericarditis, this therapy is anticipated to be a chronic therapy (Table I). In a large observational multicentric study of 370 patients the median duration of therapy with corticosteroids was 1.1 years (IQR 0.4–2.6); for comparison, the median duration of therapy with NSAIDs was 2.5 years (IQR 1.1–5.8), for colchicine 2.8 (1.3–5.0), and for anakinra 2.2 (0.9–5.0) (41).

Management of the chronic patient: re-evaluating gastroprotection and bone health

A significant challenge in the long-term management of pericarditis is the appropriate prevention of iatrogenic complications (42, 43). Current clinical practice often exhibits a paradoxical imbalance, in which patients are frequently overtreated with PPIs while remaining dangerously undertreated for steroid-induced bone loss.

The widespread use of PPIs in patients receiving corticosteroids is a common clinical habit that lacks scientific justification in the outpatient setting, condition in which corticosteroids are not indicated (44-47). While gastric protection is mandatory when corticosteroids are combined with high-dose non-steroidal anti-inflammatory drugs, they are not recommended when administered without NSAIDs in outpatients, particularly at the low doses recommended by modern guidelines. Excessive use of PPIs is not without risk, as long-term PPIs use has been associated with increased susceptibility to infections, potential interference with nutrient absorption and increased risk of osteoporotic fractures (44, 46). In particular, Abtahi *et al*. showed in a population-based cohort study that PPI use was associated with a higher incidence of fractures, with an additive effect when used together (48).

In contrast, the prevention of corticosteroid-induced osteoporosis is frequently overlooked despite its critical importance. As pericarditis often follows a recurrent or chronic course, what is initially intended as a short-term treatment usually evolves into a prolonged exposure to corticosteroids. In this context, the failure to initiate bone protection at the beginning of therapy represents a major clinical oversight (49). Bisphosphonates, such as alendronate, should be considered early in the treatment plan for patients expected to remain on corticosteroids for an extended period. This proactive strategy is essential to prevent the silent progression of bone mineral density loss and the subsequent risk of fractures (43, 50). Aligning the management of pericarditis with established rheumatological standards for bone health ensures that the benefits of inflammatory control are not negated by the long-term morbidity of skeletal complications. Notably, corticosteroid-induced osteoporosis predominantly occurs within six months after starting corticosteroids, and the process is detectable just after a few weeks (51); accordingly, bisphosphonates should be started soon, as recommended by rheumatological guidelines (52). Guidelines in the field of rheumatology suggest that any patient expected to receive a daily dose of prednisone equivalent to 2.5 mg or more for a duration exceeding three months should be evaluated for bone-sparing therapy (43).

In practice, the immediate initiation of bisphosphonates, such as alendronate, represents the most effective strategy to mitigate rapid bone loss, which is most pronounced during the first six months of corticosteroid use. This pharmacological intervention must be supported by adequate supplementation of calcium and vitamin D, tailored to the patient's nutritional status and baseline levels (52, 53).

Defining the toxicity threshold and the safety of low-dose regimens

The clinical management of long-term corticosteroid therapy requires a precise understanding of the dose-response relationship regarding systemic toxicity. Evidence suggests a clear bimodal distribution of risk: while dosages exceeding 10 mg per day of prednisone are consistently associated with a significant increase in iatrogenic morbidity, doses below 5 mg per day demonstrate a remarkably high safety profile (54-56). In the context of recurrent pericarditis, where complete withdrawal often triggers a relapse, achieving a stable maintenance dose within this low range can be considered a successful therapeutic outcome.

The safety of these ultra-low doses is supported by data from other inflammatory conditions, most notably the GLORIA trial (56), which evaluated the addition of 5 mg of prednisolone to standard care in older patients with rheumatoid arthritis. The study concluded that such a low dose does not significantly increase the overall burden of adverse events over a two-year period, while maintaining its anti-inflammatory efficacy. In pericarditis management, this implies that the goal of therapy does not necessarily have to be the absolute discontinuation of corticosteroids. For many patients, a maintenance dose of 2.5 to 5 mg per day provides sufficient suppression of the autoinflammatory pathway without incurring the metabolic, cardiovascular or skeletal risks typical of higher dosages.

Furthermore, current clinical experience indicates that patients who remain stable on 5 mg or less per day can lead a normal life with minimal monitoring, provided that the initial high-dose phase was avoided (54). This approach shifts the focus from an obsessive pursuit of complete suspension to a strategy of clinical stabilisation. By accepting a safe minimal dose, clinicians can prevent the dangerous cycle of withdrawal and relapse that characterises the most difficult to treat cases of steroid-dependent pericarditis. This nuanced perspective on dosing ensures that the treatment remains sustainable for the patient over the long term. Hydroxychloroquine may be considered in these cases to further minimise corticosteroids exposure (57).

Figure 2 illustrates the key principles for the safe use of corticosteroids in patients with pericarditis. In particular, it emphasises that corticosteroids should be considered a second-line therapy, to be used in combination with NSAIDs and colchicine in cases of incomplete response to first-line treatment and administered at low-to-moderate doses. The figure also highlights the importance of early prevention of corticosteroid-related complications, particularly osteoporosis. Regarding long-term management, it illustrates that maintenance therapy with very low corticosteroid doses may be sustained over time in selected patients, whereas treatment discontinuation requires a slow tapering strategy guided by clinical stability and CRP levels.

Phenotype-based therapy and management of specific clinical subsets

The clinical heterogeneity of pericarditis necessitates a tailored pharmacological approach, as the risk-benefit ratio of corticosteroids varies significantly across different patient populations.

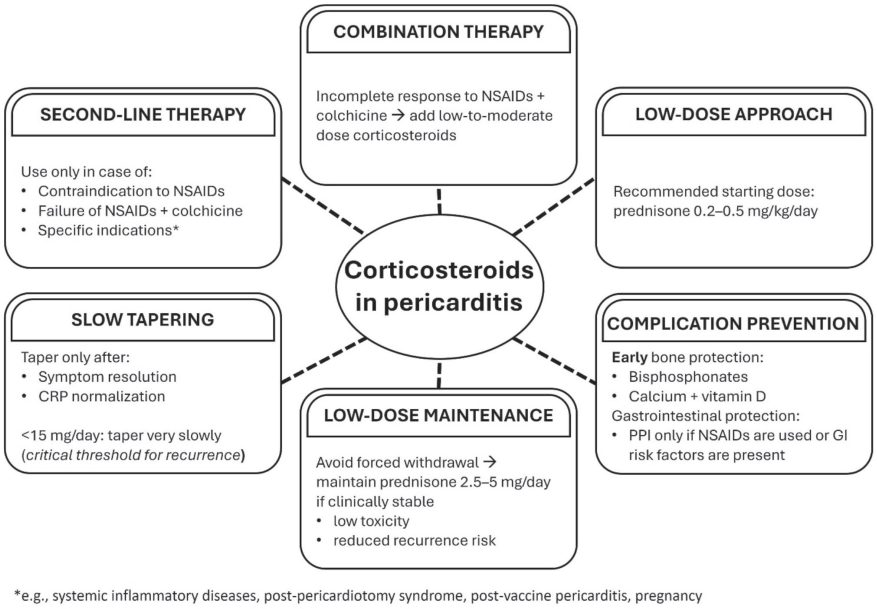

Fig. 2. Key principles for the safe use of corticosteroids in pericarditis.
CRP: C-reactive protein; GI: gastrointestinal; NSAIDs: non-steroidal anti-inflammatory drugs; PPI: proton pump inhibitor.

In older adults, for instance, the management strategy should prioritise the avoidance of systemic toxicity, following the principle that less is often more. In this subset, initial prednisone doses should ideally not exceed 12.5 to 25 mg/day, mirroring the successful protocols used in polymyalgia rheumatica (58, 59); bone protection is mandatory. This cautious approach is supported by the observation that older patients are more susceptible to the metabolic and neuropsychiatric side effects of corticosteroids, making the transition to low-dose maintenance even more critical (59).

Specific considerations must also be applied to the paediatric population and pregnant patients (1, 22, 60-64). In *children*, chronic use of corticosteroids is strictly discouraged due to the high risk of growth impairment and permanent endocrine disruption. For these patients, early consideration of IL-1 inhibitors is often preferable to prolonged corticosteroid exposure. Conversely, in *pregnancy*, prednisone is generally considered safe at low doses, but it should ideally be maintained below 10 mg per day to minimise the risk of gestational diabetes and pregnancy-induced hypertension. Recent literature, including the 2025 updates on obstetric outcomes in inflammatory diseases, reinforces the importance of using the lowest effective dose to ensure both maternal and fetal safety (30, 65, 66).

A challenging scenario involves patients with recurrent symptoms despite a negative CRP (67). In these cases, the inflammatory driver is often less intense, and the use of high-dose corticosteroids is clinically unjustified. Some evidence suggests that a very low dose of prednisone, typically around 5 mg per day, is sufficient to manage symptoms such as asthenia and pain without escalating to more toxic regimens (67).

This phenotype-driven approach ensures that corticosteroid therapy is not applied as a one-size-fits-all solution but is instead calibrated to the specific biological and clinical needs of the patient, thereby optimising outcomes while minimising long-term iatrogenic harm.

Limitations of IL-1 inhibitors

Interleukin-1 inhibitors, particularly anakinra (68), rilonacept (23), and goflikicept (69) that inhibit either IL-1 alpha or IL-1 beta, have significantly changed the therapeutic management of recurrent pericarditis, especially in patients with a marked inflammatory phenotype and corticosteroid-dependent or refractory disease. The United States statement recommends avoiding corticosteroids, with a rapid transition to anti-IL1 agents soon after NSAIDs and colchicine failure (35). On the other hand, the ESC Guidelines consider anti-IL1 agents as a third-line therapy, after low-medium doses of corticosteroids, with the possible exception of pediatric cases, in which chronic corticosteroid use is certainly contraindicated. In Europe only anakinra is available. In clinical practice we consider the use of anakinra when chronic therapy with prednisone at a dosage >10–15 mg daily is anticipated. However, despite the remarkable efficacy of anakinra and rilonacept, several limitations still restrict their widespread use in routine clinical practice.

First, the economic cost of these therapies and the heterogeneous reimbursement policies across different healthcare systems significantly limit accessibility in many countries. In this context a cost-effectiveness analysis should take into account also the cost of the specific drug and the marked reduction in hospital admissions associated with the use of these drugs (70). In several settings, treatment with anakinra or rilonacept remains restricted to specialised referral centres and/or requires dedicated approval pathways. In addition, although currently available studies support a generally favourable safety profile, long-term real-world data in recurrent pericarditis remain more limited than for conventional anti-inflammatory therapies (18-20, 70), even if the safety profile of these old drugs is well established in other conditions such as rheumatoid arthritis and autoinflammatory diseases (71-75). Furthermore, relapses following treatment discontinuation are not uncommon, and the optimal duration of therapy and the most appropriate tapering strategy after remission have not yet been definitively established (19, 27, 41). Moreover, the daily subcutaneous administration of anakinra and the possible injection-site reactions, although usually manageable, may adversely affect treatment adherence and quality of life in some patients (19, 70).

Therefore, despite the major therapeutic advances provided by IL-1 inhibitors, corticosteroids still maintain a clinically relevant role in many patients when used according to modern low-dose and slow-tapering protocols, particularly in

regions in which anakinra, rilonacept or goflikicept are not available.

Key message: adhering to the 2025 ESC guidelines to correct historical errors

The management of pericarditis has reached a critical juncture where the integration of clinical evidence into routine practice is no longer optional but essential for patient safety. The primary conclusion of this review is the necessity of following the ESC 2025 guidelines (76), which reinforce the shift away from the high-dose corticosteroid regimens of the past. Therapy of the first attacks not according to the ESC guidelines is an important risk factor for recurrences and a longer duration of disease (41). By strictly adhering to these protocols, clinicians can avoid the cycle of dependency and recurrence that has historically characterised the treatment of idiopathic and recurrent pericarditis.

Furthermore, the transition to the 2025 standards requires a fundamental change in how the success of therapy is measured (58). It is no longer sufficient to achieve rapid symptomatic relief at the cost of long-term stability. The current guidelines prioritise a sustainable clinical course, where the use of corticosteroids is carefully calibrated according to the patient's specific inflammatory phenotype. This adherence to evidence-based medicine ensures that the mistakes of previous decades, specifically the use of aggressive loading doses and rapid tapering schedules, are replaced by a more disciplined and biologically guided approach.

Corticosteroids as a precision tool: efficacy through rheumatological dosing and gradual withdrawal

The final and perhaps most significant message for the clinician is the redefinition of corticosteroids as a precision instrument rather than a blunt anti-inflammatory tool. When managed according to modern rheumatological principles (54-56), these drugs remain an invaluable resource in the treatment of refractory pericarditis. Their efficacy, however, is strictly dependent on the use of medium-low starting doses on top of colchicine and NSAIDs and an exceptionally slow, non-automatic tapering process. By treating corticosteroids as a precision tool, the clinician can suppress the autoinflammatory IL-1 mediated loop without inducing the systemic instability that leads to disease chronicity.

Chronic idiopathic pericardial effusion

Acute pericarditis can cause pericardial effusion in 50–60% of cases (1). On the other hand, the majority of pericardial effusions are not associated with acute pericarditis. Chronic severe idiopathic pericardial effusion in the absence of evidence of pericarditis, usually with normal CRP, is a poorly understood nosological entity. No study has ever shown any efficacy of corticosteroids in this condition.

Conclusions and future perspectives

Corticosteroids are a double-edged sword in recurrent pericarditis. According to the 2025 ESC guidelines, corticosteroids may be added at low to moderate doses as triple therapy in cases of incomplete response to aspirin/NSAIDs and colchicine. In cases of recurrence, every effort should be made not to increase their dose or to reinstate corticosteroids (34).

The elderly usually require medium dosages (*e.g.* prednisone 25 mg), pregnant women no more than 10 mg, while it should be avoided giving chronic corticosteroid therapy to children. CRP negative cases may benefit from low doses (5 mg).

Chronic maintenance with really low-dose prednisone (5 mg/day or less) may be considered in selected cases, with a good risk-benefit balance.

In clinical practice we consider use of anakinra when chronic therapy with prednisone at doses greater than 10–15 mg daily is anticipated.

The final goal of therapy is not merely the suppression of symptoms, but the achievement of a stable, long-term remission with minimal iatrogenic impact. This requires a meticulous adherence to slow tapering protocols and a proactive stance on bone health, ensuring that the treatment of the pericardium does not come at the expense of the patient's overall skeletal and metabolic integrity.

Looking toward the future, the therapeutic landscape is expected to be further refined by the increasing availability of targeted biological therapies (77). The success of IL-1 blockers, such as anakinra and rilonacept, has already provided a viable exit strategy for the most refractory, steroid-dependent patients. Future research will likely focus on identifying specific biomarkers beyond CRP that can predict the clinical course and response to treatment, allowing for an even more personalised approach. Additionally, the role of genetics in understanding the predisposition to the autoinflammatory phenotype may open new avenues for early intervention before the disease enters a chronic phase (78, 79).

In summary, the future of pericarditis management lies in the synergy between the disciplined use of traditional drugs and the strategic integration of modern biologics. By considering corticosteroids as a precision tool rather than a first-line solution and by prioritising the prevention of long-term side effects, clinicians can transform the prognosis of this challenging condition. The ultimate objective remains a holistic model of care where the precision of the anti-inflammatory response is matched by a rigorous commitment to the patient's long-term safety and quality of life.

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