Anti-inflammatory Therapies for Cardiovascular Disease in 2026

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Chronic low-grade inflammation is a recognised pathophysiological driver in the progression of atherosclerosis and its thrombotic complications, contributing significantly to residual cardiovascular risk despite optimal lipid-lowering and antiplatelet therapies. The clinical dilemma for practitioners involves identifying patients who may benefit most from targeted anti-inflammatory interventions and integrating these into existing treatment paradigms. The immediate takeaway is that while several agents show promise, their precise role and patient selection criteria continue to evolve.

KEY TAKEAWAYS

- The Pivot: Targeted anti-inflammatory therapies are moving beyond broad immunosuppression to specific cytokine inhibition.
- The Data: Interleukin-1β inhibition has demonstrated a reduction in major adverse cardiovascular events (MACE).
- The Action: Clinicians should consider the inflammatory burden in high-risk patients, particularly those with elevated high-sensitivity C-reactive protein (hs-CRP).

Inflammation plays a central role in the initiation, progression, and destabilisation of atherosclerotic plaques. Endothelial dysfunction, leukocyte recruitment, and the release of pro-inflammatory cytokines contribute to plaque growth and vulnerability. Traditional risk factor modification, including statin therapy, addresses some inflammatory components indirectly, but a significant residual inflammatory risk persists in many patients with established cardiovascular disease (CVD). This residual risk is often quantified by persistently elevated levels of high-sensitivity C-reactive protein (hs-CRP), a biomarker of systemic inflammation.1

Targeting Inflammatory Pathways

The concept of directly targeting inflammatory pathways to reduce cardiovascular events gained substantial traction following the Canakinumab Anti-inflammatory Thrombosis Outcome Study (CANTOS). This trial investigated canakinumab, a monoclonal antibody that selectively neutralises interleukin-1β (IL-1β). The trial demonstrated that canakinumab, at a dose of 150 mg subcutaneously every three months, led to a statistically significant reduction in the primary composite endpoint of nonfatal myocardial infarction, nonfatal stroke, or cardiovascular death. The hazard ratio (HR) for the primary endpoint was 0.85 (95% CI 0.79-0.92; P), compared with placebo. This effect was observed in patients with a history of myocardial infarction and an hs-CRP level of ≥ mg/L.2 The reduction in cardiovascular events was independent of lipid-lowering effects, as canakinumab did not significantly alter lipid profiles. However, an increase

in fatal infections was observed in the canakinumab group (incidence rate 0.31 vs 0.18 events per 100 patient-years), necessitating careful patient selection.2

Beyond IL-1β inhibition, other inflammatory targets are under investigation. Colchicine, an ancient anti-inflammatory agent, has shown promise in reducing cardiovascular events. The Colchicine Cardiovascular Outcomes Trial (COLCOT) and the Low-Dose Colchicine for Secondary Prevention of Cardiovascular Disease (LoDoCo2) trial both demonstrated a reduction in cardiovascular events in patients with recent myocardial infarction or chronic coronary disease, respectively. In COLCOT, colchicine 0.5 mg daily reduced the primary composite endpoint of cardiovascular death, resuscitated cardiac arrest, myocardial infarction, stroke, or urgent hospitalisation for angina leading to revascularisation, with an HR of 0.77 (95% CI 0.61-0.96; P = .02).3 LoDoCo2 similarly reported an HR of 0.69 (95% CI 0.57-0.83; P) for the primary composite endpoint of cardiovascular death, nonfatal myocardial infarction, nonfatal ischaemic stroke, or ischaemic-driven coronary revascularisation.4 The mechanism of action for colchicine involves inhibition of microtubule polymerisation, leading to reduced neutrophil activation and inflammasome inhibition.3,4 Gastrointestinal side effects are the most common adverse events with colchicine.3,4

Methotrexate, a folate antagonist with anti-inflammatory properties, was investigated in the Cardiovascular Inflammation Reduction Trial (CIRT). This trial enrolled patients with a history of myocardial infarction or multivessel coronary artery disease and type 2 diabetes or metabolic syndrome. CIRT did not demonstrate a reduction in cardiovascular events with low-dose methotrexate (15-20 mg weekly) compared to placebo. The HR for the primary composite endpoint of nonfatal myocardial infarction, nonfatal stroke, or cardiovascular death was 1.01 (95% CI 0.89-1.16; P = .84).5 This outcome suggests that a broad anti-inflammatory approach without specific targeting of the IL-1β pathway may not be effective in this population, or that the anti-inflammatory effects of methotrexate at these doses are insufficient to impact cardiovascular outcomes.5

Ongoing research continues to explore novel targets, including inhibitors of the NLRP3 inflammasome, which is a key component of the innate immune system involved in IL-1β activation. Other areas of interest include therapies modulating T-cell responses and B-cell activity, though these are largely in earlier phases of development for cardiovascular indications. The challenge remains to identify specific inflammatory pathways that are both amenable to therapeutic intervention and critical for driving cardiovascular events, while also ensuring a favourable safety profile.

Looking ahead to 2026, the landscape of anti-inflammatory therapies for CVD is expected to evolve with a greater emphasis on precision medicine. Biomarkers beyond hs-CRP, such as specific cytokines or genetic markers, may help identify patients most likely to benefit from targeted anti-inflammatory interventions and those at higher risk of adverse events. Further refinement of dosing strategies and combination therapies could also optimise efficacy while mitigating side effects.

The integration of these therapies into routine clinical practice will require robust risk-benefit assessments and clear guidelines for patient selection. While the promise of directly addressing inflammation in CVD is significant, ongoing research must continue to balance therapeutic efficacy with safety, particularly concerning immunosuppression and infection risk. The coming years will likely see a more nuanced approach to anti-inflammatory strategies, moving beyond broad-spectrum agents to highly targeted interventions based on individual patient inflammatory profiles.

CLINICAL IMPLICATIONS

The data from trials like CANTOS and LoDoCo2 underscore a critical shift in our understanding of cardiovascular disease management: inflammation is not merely a bystander but an active participant in atherothrombosis. For clinicians, this means moving beyond the traditional lipid-centric view and considering the inflammatory burden, particularly in patients with elevated hs-CRP despite optimal statin therapy. The availability of agents like colchicine, with its established safety profile and low cost, presents an immediate opportunity to address residual inflammatory risk in a broader patient population. However, the increased risk of fatal infections with canakinumab necessitates careful patient selection and a thorough risk-benefit assessment, likely reserving it for specific high-risk individuals.

The pharmaceutical industry faces the challenge of developing more targeted anti-inflammatory agents with improved safety profiles. The failure of methotrexate in CIRT highlights that not all anti-inflammatory mechanisms are equally effective in cardiovascular disease. Future drug development must focus on specific inflammatory pathways that are mechanistically linked to atherothrombosis, rather than broad immunosuppression. This precision medicine approach will be crucial for identifying therapies that offer significant cardiovascular benefits without undue systemic side effects. Companies developing NLRP3 inflammasome inhibitors, for instance, will need to demonstrate clear efficacy and safety advantages over existing options.

For patients, these developments offer hope for reducing recurrent cardiovascular events, particularly for those who remain at high risk despite current standard of care. However, it also introduces complexity. Patients will need to understand the rationale for these new therapies, the potential side effects, and the importance of adherence. The cost-effectiveness of these treatments, especially for biologics, will also be a significant consideration for healthcare systems and patient access. Integrating these anti-inflammatory strategies into clinical guidelines will require robust evidence on long-term safety and cost-effectiveness, ensuring that the benefits outweigh the risks and financial implications for a wide patient demographic.

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