Thrombopoietin-Receptor Agonism in Chemotherapy-Induced Thrombocytopenia

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Abstract

Chemotherapy-induced thrombocytopenia (CIT) is a common, dose-limiting toxicity of cytotoxic cancer therapy that frequently necessitates dose reduction, delay, or discontinuation, thereby compromising relative dose intensity and potentially diminishing therapeutic efficacy. Existing management strategies are largely supportive and do not address the underlying defect in thrombopoiesis. Romiplostim, a thrombopoietin-receptor agonist, directly stimulates megakaryocyte proliferation and platelet production. In prospective phase II studies and multicenter real-world cohorts, romiplostim has consistently demonstrated rapid and sustained platelet recovery in the majority of patients with CIT, enabling timely continuation of chemotherapy without recurrent thrombocytopenia and substantially reducing the need for platelet transfusions.

Available evidence indicates an acceptable safety profile, with no clear excess in thrombotic events or cumulative marrow toxicity beyond that expected in patients with active malignancy. However, these findings are derived predominantly from phase II and observational data, and important uncertainties remain regarding predictors of response and optimal dosing strategies. Romiplostim represents a rational, mechanism-based approach to CIT with the potential to preserve chemotherapy delivery and treatment intensity; adequately powered randomized trials are required to define its role in routine oncologic practice.

Keywords: Chemotherapy; Drug-Related Side Effects and Adverse Reactions; Romiplostim; Thrombocytopenia; Thrombopoietin Receptor Agonists

Letter to the Editor

Chemotherapy-induced thrombocytopenia (CIT) remains a frequent and clinically consequential complication of cytotoxic cancer therapy. Thrombocytopenia often necessitates chemotherapy dose reduction, treatment delay, or discontinuation, thereby compromising relative dose intensity and potentially diminishing therapeutic efficacy. Current management strategies largely rely on treatment modification or platelet transfusion, both of which address the consequence rather than the underlying pathophysiology of impaired thrombopoiesis[1].

Romiplostim,a thrombopoietin-receptor agonist that stimulates megakaryocyte proliferation and platelet production, has emerged as a potential therapeutic option for CIT. Early clinical investigations have demonstrated its capacity to restore platelet counts in patients whose chemotherapy had been interrupted or modified due to persistent thrombocytopenia. In a prospective phase II randomized study involving patients with solid tumors and CIT, platelet recovery occurred in the vast majority of patients receiving weekly romiplostim, whereas only a

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small proportion of those managed with observation alone achieved comparable recovery within the same timeframe[2].Importantly, correction of thrombocytopenia enabled resumption of chemotherapy without recurrent platelet decline in most treated patients Observational data from multicentre cohorts further support these findings. In a large real-world study of patients treated with romiplostim for CIT, the majority achieved a clinically meaningful platelet response, permitting continuation of systemic therapy without dose reduction or delay in a substantial proportion of cases [3.] Moreover, platelet transfusion requirements were markedly reduced, suggesting that pharmacologic stimulation of thrombopoiesis may offer a more durable supportive strategy.

The safety profile of romiplostim in this setting has also been reassuring. Long-term follow-up analyses have demonstrated sustained platelet responses without evidence of cumulative toxicity or clinically significant marrow fibrosis, while rates of thrombotic events appear comparable to those expected in patients with active malignancy receiving chemotherapy[4]. Such observations are particularly relevant given the hypercoagulable milieu associated with cancer.

Nevertheless, several questions remain unresolved. Predictors of response which includes marrow tumour infiltration, prior to pelvic irradiation, or specific cytotoxic regimens requiring further validation, and optimal dosing strategies have yet to be standardized 5. In addition, most available data derive from phase II trials or retrospective analyses, underscoring the need for adequately powered phase III studies.

Taken together, the existing evidence suggests that romiplostim may represent a rational and effective strategy to mitigate CIT while preserving the continuity and intensity of anticancer therapy. Definitive randomized trials are now warranted to clarify their role within routine oncologic supportive care.

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