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Chemotherapy-Induced Thrombocytopenia : Unravelling Mechanisms and Redefining Management Paradigms

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Abstract

Chemotherapy-induced thrombocytopenia (CIT) is a common hematologic toxicity encountered during systemic cancer therapy and represents an important cause of treatment interruptions. Practically, such interruptions mandate dose reductions in chemotherapy regimens which potentially compromise the efficacy and clinical response. Mechanisms underlying the pathophysiology include bone marrow suppression affecting megakaryocyte production, immune-mediated platelet destruction, thrombopoietin dysregulation and alterations in marrow microenvironment.

The incidence varies according to tumor type and chemotherapy regimen, with platinum-based and gemcitabine-containing regimens associated with highest risk. Current management approaches include chemotherapy dose modification, platelet transfusion and supportive care. Recently, thrombopoietin receptor agonists (TPO-RAs) have emerged as a promising therapeutic option to stimulate platelet production and maintain treatment continuity. This review summarizes the epidemiology, mechanisms, clinical consequences and evolving management strategies of chemotherapy induced thrombocytopenia.

Keywords: Chemotherapy Induced Thrombocytopenia; Drug Toxicity; Treatment Strategies; Low Platelet Count; Chemotherapeutic drugs

Abbreviations: CIT: Chemotherapy-Induced Thrombocytopenia; TPO-Ras: Thrombopoietin Receptor Agonists; PSE: Partial splenic embolization; CDK: Cyclin Dependant Kinase; ISTH: International Society on Thrombosis and Haemostasis; TPO: Thrombopoietin; CIT: Chemotherapy-Induced Thrombocytopenia; CTCAE: Common Terminology Criteria for Adverse Events; AABB: Association for the Advancement of Blood & Biotherapies;

Introduction

Chemotherapy-induced thrombocytopenia (CIT) is defined as peripheral platelet count below 100 x 109/L in patients receiving myelosuppressive chemotherapy [1]. It remains a frequent and clinically significant complication of cytotoxic chemotherapy, affecting almost 20–25% of individuals with solid tumors [2]. The incidence varies depending on tumor type, patient characteristics and treatment regimen. CIT is most observed in non-small cell lung cancers, ovarian cancers and colorectal cancers with a reported incidence of 25%, 24% and 18% respectively [3].

This site predilection reported in literature is attributed to the use of chemotherapeutic drugs-such as gemcitabine, platinum and taxane compounds- in these subsite cancers [4,5]. Though the list is exhaustive, common chemotherapy drugs implicated in thrombocytopenia have been summarised below in Table 1 [4-13]. The clinical manifestations of thrombocytopenia range from mild, asymptomatic cases with petechiae to those with life-threatening haemorrhagic events, such as intracranial or gastrointestinal bleeding [8]. CIT thus inevitably leads to delay in

subsequent chemotherapy cycles, dose reductions or permanent discontinuation of chemotherapy [14]. Despite advances in supportive care, the optimal management of CIT remains challenging. Traditional approaches focus on platelet transfusions and chemotherapy modification, while emerging therapies aim to stimulate platelet production through thrombopoietic pathways.

Pathophysiology of CIT

CIT pathogenesis involves myriads of pathways. Some notable and well understood mechanisms are described below

Table 1: Various chemotherapy agents and incidence of clinically significant thrombocytopenia associated with them [4-13].

Class	Agent	Incidence
Antimetabolites	Gemcitabine	30-65%
	Cytarabine	20-40%
	Capecitabine	25-30%
	5-Fluorouracil	10-15%
	Methotrexate	10-20%
Platinums	Carboplatin	25-80%
	Oxaliplatin	10-50%
	Cisplatin	5-10%
Alkylating agents	Busulfan	20-60%
	Carmustine	20-50%
	Cyclophosphamide/Ifosfamide	5-15%
Anthracyclines	Doxorubicin	15-20%
Taxanes & Vinca Alkaloids	Paclitaxel, Docetaxel Vincristine, Vinblastine	5-10%
Topoisomerase inhibitors	Etoposide	20-40%
	Irinotecan	5-10%
Hematological agents	Bendamustine	30-60%
	Fludarabine	20-40%
	Chlorambucil	10-30%

A: Bone marrow suppression

The primary mechanism underlying CIT is direct suppression of hematopoietic progenitor cells by cytotoxic chemotherapy. Drugs such as alkylating agents and platinum compounds lead to apoptosis and DNA damage in megakaryocyte precursors resulting in depletion of progenitors giving rise to platelets [15].

B: Alterations in bone marrow milieu:

Chemotherapy disrupts stromal cells, cytokine signaling and marrow niche that supports haematopoiesis. Reduced secretion of essential growth factors like Thrombopoietin (TPO) and interleukins from stromal cells, increased oxidative stress and inflammation in endothelial cells and cytokine production shift towards molecules like transforming growth factor beta (TGF-β) results in impaired megakaryocyte differentiation and inefficient platelet regeneration post-chemotherapy [15,16].

C: Immune Mediated Platelet Injury

In certain scenarios, chemotherapy and immunotherapy drugs can mount an immune response against platelets de novo resulting in their accelerated destruction in spleen and clearance from circulation. T-cell mediated platelet destruction, immune checkpoint inhibitor associated thrombocytopenia and drug induced anti-platelet reactive antibodies are some plausible mechanisms underlying immune mediated platelet injury [17].

D: Megakaryocyte Dysfunction and Thrombopoietin Dysregulation

Thrombopoietin is normally required for megakaryopoesis. Chemotherapy interferes with this physiological pathway by reducing megakaryocyte sensitivity to TPO, blunting TPO elevation in response to thrombocytopenia, particularly by impairing its synthesis from liver [18,19]. Taken together, these factors contribute to delayed to sluggish platelet recovery post chemotherapy.

Risk Factors

The development of chemotherapy-induced thrombocytopenia (CIT) is influenced by a combination of patient-related, disease-related and treatment-related factors. Patient related factors such as advanced age, poor performance status, baseline cytopenias and hepatic or renal dysfunction have been associated with

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an increased risk of CIT due to reduced bone marrow reserve and altered drug metabolism [6]. Disease-related factors like bone marrow infiltration by malignancy and prior exposure to radiotherapy, further compromise hematopoietic capacity and predispose patients to thrombocytopenia [4].

Among treatment-related variables, the type of drug, dose and schedule of chemotherapy play a critical role, with agents such as platinum compounds, gemcitabine and alkylating agents being particularly implicated. Combination chemotherapy regimens and higher cumulative doses are associated with greater myelosuppression [4]. Additionally, prior lines of chemotherapy received may amplify the risk. Identification of these risk factors is essential for early recognition, risk stratification and optimization of therapeutic strategies to minimize treatment interruptions and maintain dose intensity.

Clinical Presentation

The clinical presentation of chemotherapy-induced thrombocytopenia (CIT) varies widely, ranging from asymptomatic laboratory abnormalities to clinically significant bleeding. The Common Terminology Criteria for Adverse Events (CTCAE) categorizes thrombocytopenia into four grades based on platelet counts as shown in Table 2 [7]. The risk of bleeding correlates with both the severity of thrombocytopenia and the presence of additional risk factors such as infection, mucosal damage from chemotherapy, or concurrent use of anticoagulants.

Table 2: CTCAE grading for thrombocytopenia [7].

Grade of thrombocytopenia	Platelet count
1	< Lower limit of normal to 75,000 / microlitre
2	75000-50000 / microlitre
3	50000-25000 / microlitre
4	< 25000 / microlitre

Most patients with mild to moderate thrombocytopenia remain asymptomatic and are identified incidentally on routine blood counts. As platelet levels decline, patients may develop mucocutaneous manifestations such as petechiae, purpura, ecchymoses and epistaxis. Gingival bleeding and menorrhagia are also commonly reported. In cases of severe thrombocytopenia, more serious bleeding complications may occur, including gastrointestinal haemorrhage, haematuria and rarely, life-threatening intracranial hemorrhage [8].

Management Strategies

The primary goals in the management of CIT are to prevent clinically significant bleeding, maintain planned chemotherapy dose intensity and minimize treatment interruptions. Unlike other chemotherapy-induced toxicities such as neutropenia, where primary prophylaxis with granulocyte colony-stimulating factors is well established, there are currently no universally recommended preventive strategies for thrombocytopenia. Thus, current management strategies are predominantly reactive, i.e. implemented after the development of thrombocytopenia, rather than preventive [20].

Further, before initiating specific interventions, it is imperative to evaluate for secondary causes of thrombocytopenia, such as infection, coagulopathy or concurrent medications that may exacerbate platelet suppression [10]. The management strategies encompass thrombopoietin receptor agonists, platelet transfusions, chemotherapy dose modifications and other miscellaneous modalities.

A: Thrombopoietin Receptor Agonists (TPO-RAs): The Wonder Molecules

TPO is the main cytokine which regulates the development and maturation of megakaryocytes. Clinical trials by Moskowitz et al. [24] and Vadhan-Raj et al. [25] evaluated the utility of recombinant TPO for managing thrombocytopenia. The trials were closed early in their course since the recombinant TPO gave rise to anti-TPO autoantibodies which cross reacted with endogenous TPO and further resulted in greater thrombocytopenia and delayed platelet recovery. This formulated the basis for generation of TPO-RAs, a class of drugs that bind to and activate the TPO receptor to stimulate megakaryocyte production and differentiation, without the added problem of auto-antibody production.

Although their action is like endogenous TPO, they lack its peptide sequence [10]. There are currently four available TPO-RAs: romiplostim, eltrombopag, avatrombopag and lusutrombopag. To date, only studies involving romiplostim have shown the maximum benefit in CIT [26]. Rompilostim is a subcutaneously administered TPO analogue given by a weekly regimen of 1 microgram/kg till platelet count improves to >50,000 per microlitre [26]. Its efficacy has been validated in several trials. In a retrospective study by Parameswaran et al., administration of romiplostim to patients with CIT increased platelet counts to >1,00,000 / microlitre in 95% patients with severe thrombocytopenia.

Further, this increment had a sustained effect such that 60% of patients of the study group received atleast 2 more cycles of same chemotherapy without dose reduction [27]. In a

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large observational cohort of 173 patients with solid tumors, lymphoma or myeloma, romiplostim administration doubled the median platelet count in the intervention cohort (112 × 109/L vs 54 × 109/L). 79% of patients with solid tumors were able to continue treatment without dose reductions or any delays due to thrombocytopenia.

89% of patients could complete treatment without platelet transfusions [28]. However, patients with prior pelvic irradiation, bone marrow invasion and prior temozolomide were refractory to romiplostim therapy in their study. Al-Samkari H et al. [28] in their recent phase III randomized trial on patients with CIT demonstrated that a significantly higher proportion of patients receiving romiplostim were able to maintain their planned chemotherapy dose intensity without requiring dose reductions compared to those receiving placebo (84% vs 36% patients) [29].

Various studies have also evaluated the efficacy of oral thrombopoetin receptor agonists in managing CIT. Kellum et al. [30] in their study found that patients treated with eltrombopag 50 mg daily had higher platelet counts at the start of subsequent treatment cycles. However, the primary end point difference in platelet count from day 1 to the lowest platelet point in cycle 2 was not reached. In another phase II trial, eltrombopag administration in CIT patients led to higher platelet counts, lower incidence of grade 3 or 4 CIT, faster recovery of cytopenia and less dosage reduction/treatment delays [31]. Avatrombopag is the most recent oral TPO-RA being popularised for greater efficacy as compared to eltrombopag.

In a phase III randomised trial by Al-Samkari et al. [28] Avatrombopag almost doubled the nadir platelet count (51,000 per microlitre vs 29,000 per microlitre) in the entire intervention cohort with an absolute benefit of 76% [32]. Chen et. al in their study on paediatric acute lymphoblastic leukaemia patients demonstrated that those who received avatrombopag 20 mg daily had a shorter duration of grade 3 thrombocytopenia (a mean of 3.3 vs 4.7 days) and a shorter time to platelet recovery (a mean of 7.9 vs 9.7 days). Further, platelet transfusions could altogether be avoided in almost 23% of such patients [33]. No trials have been done till date to compare the efficacy of romiplostim as compared to oral TPO-RA agents.

B: Platelet Transfusions: Where we stand?

There are certain pitfalls with the approach of platelet transfusions for tackling CIT. The transfusions provide only a short duration of improvement of mere 3-5 days and the platelet increment is unpredictable.10 Transfusion-related acute lung injury is still an obstacle to effective blood transfusion and patients may even become refractory to platelet transfusions [9,10]. Further, receiving multiple transfusions can lead to immune sensitization in the recipient, thus rendering this therapeutic approach ineffective. Although single-donor platelets theoretically limit donor exposure and are expected to decrease the risk of alloimmunization, clinical studies have shown comparable rates of HLA alloimmunization and platelet refractoriness between single donor platelets and platelets from multiple donors [9].

Owing to the above issues as well as the promising results of TPO-RAs, platelet transfusions are not routinely advocated for mild to moderate thrombocytopenia. When platelets are ≥50,000 per microlitre (CTCAE grade 1–2) with low risk of active bleed (less than WHO grade 2), Association for the Advancement of Blood & Biotherapies (AABB) and National Institute for Health & Care Excellence (NICE) guidelines do not recommend any transfusion [21,22]. Even when platelet count falls to a value between 25000 per microlitre to 50,000 per microlitre (CTCAE grade 3), pharmacological intervention is advocated instead of platelet transfusions [4]. Its role remains largely confined to management of clinically significant and severe thrombocytopenia. The cut-off for transfusing platelets, however, is varied.

Estcourt et al. [23] in their review compared platelet transfusion thresholds of a standard trigger level of 10,000 per microlitre vs 20000 per microlitre or 30,000 per microlitre. They transfused patients at risk of severe bleed based on various factors such as previous chemotherapy, performance status, rather than solely on the basis of platelet counts. It was observed that platelet transfusion threshold of < 10,000 per microlitre did not increase the risk of bleeding and resulted in fewer unnecessary transfusions. International Society on Thrombosis and Haemostasis (ISTH) has endorsed an empirical platelet transfusion strategy for a platelet count of < 10,000 per microlitre20 while AABB and NICE recommend transfusion for patients with serious bleeding risk (World Health Organization grade 2 or higher) even in context of less severe thrombocytopenia (i.e. platelet count more than 25,000 per microltre) [21,22].

C: Miscellaneous Strategies

Apart from TPO-RAs and platelet transfusions, reducing the chemotherapy frequency or dosage to decrease the incidence and severity of CIT is routinely advocated as an adjunct measure, particularly when the therapy is not standard or not of curative intent [2]. Oprelvikin, a recombinant human Interlekin-11, has been approved by FDA for treatment of grade 3-4 thrombocytopenia. Its use is, however, limited owing to significant cardio-pulmonary toxicity concerns [34]. Trilaciclib, an intravenous cyclin dependant kinase (CDK) 4/6 inhibitor, has been approved for the treatment of bone marrow suppression caused by chemotherapy in lung cancer. Grade 3 or 4 hematologic adverse events were reduced by half in trilaciclib-treated patients when compared to placebo, with grade 3 or 4 thrombocytopenia occurring in 18% of trilaciclib-treated patients (vs 33% in placebo-treated arm); though cost and availability are certain concerns [9].

In patients with liver cirrhosis, splenic sequestration often contributes to persistent thrombocytopenia, which can complicate the administration of systemic chemotherapy. Partial splenic

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embolization (PSE) offers a minimally invasive strategy to mitigate hypersplenism by reducing splenic blood flow, thereby increasing circulating platelet counts. This approach has shown effectiveness in improving hematologic parameters, enabling safer delivery of chemotherapy in selected cirrhotic patients [4]. Antifibrinolytic agents like ε-aminocaproic acid and tranexamic acid have been considered for managing bleeding in thrombocytopenic cancer patients when platelet transfusions are ineffective. However, their clinical benefit remains unproven, and their use may increase thrombotic risk, particularly in cancer patients [4].

Future Directions

The development of thrombopoietic agents, particularly thrombopoietin receptor agonists (TPO-RAs), represents a promising therapeutic approach for managing CITs. Large, well-designed randomized controlled trials are required to better define their efficacy, optimal timing, dosing strategies and long-term safety in patients undergoing chemotherapy. Identification of predictive biomarkers and risk stratification models to enable early identification of patients at high risk for CIT is also an area of active research. Improved understanding of the bone marrow milieu, standardization of platelet transfusion practices and incorporation of real-world data into clinical decision-making frameworks will further help optimize management.

Conclusion

Chemotherapy-induced thrombocytopenia (CIT) is a common and clinically significant complication of systemic anticancer therapy that can adversely affect both patient safety and treatment delivery. It arises from multifactorial mechanisms, including bone marrow suppression, impaired megakaryopoiesis and immune-mediated platelet destruction. Clinically, CIT not only increases the risk of bleeding but also frequently necessitates chemotherapy dose reductions or delays, thereby compromising relative dose intensity and potentially impacting oncologic outcomes. Current management strategies include chemotherapy modification, platelet transfusions and thrombopoietin agonists. A deeper understanding of biology underlying CIT, along with implementation of high-quality clinical trials is essential to optimize management and improve patient outcomes.

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