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Clinical Utility of D-Dimer Monitoring in Predicting Treatment Response in Complex Breast Cancer Cases

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Abstract

Breast cancer remains a heterogeneous disease, with complex and aggressive cases presenting challenges in predicting and monitoring treatment response. D-Dimer, a fibrin degradation product, has emerged as a potential biomarker reflecting tumor-associated hypercoagulability and systemic disease activity. Accumulating evidence suggests that elevated baseline D-Dimer levels correlate with advanced disease and poor prognosis, while serial monitoring may provide dynamic insights into therapeutic efficacy. Reductions in D-Dimer during treatment often indicate favorable response, whereas persistent or rising levels may signal treatment resistance or early relapse. This narrative review explores the physiological and pathophysiological basis of D-Dimer elevation in breast cancer, summarizes clinical evidence supporting its utility in monitoring treatment response, and highlights future directions for integrating D-Dimer assessment into personalized patient management strategies. Incorporating this biomarker into clinical practice could enhance the precision of therapy monitoring and improve outcomes in complex breast cancer cases.

Keywords: D-Dimer, Breast Cancer, Treatment Response, Biomarkers, Prognosis

Abbreviations

CA 15-3 – Cancer Antigen 15-3, **CEA** – Carcinoembryonic Antigen, **DVT** – Deep Vein Thrombosis, **EMT** – Epithelial–Mesenchymal Transition, **IL** – Interleukin, **ROS** – Reactive Oxygen Species, **TNF-α** – Tumor Necrosis Factor-alpha, **VTE** – Venous Thromboembolism

Introduction

Breast cancer is the most commonly diagnosed malignancy among women worldwide and remains a leading cause of cancer-related mortality [1-2]. Its biological heterogeneity ranges from indolent hormone receptor-positive tumors to highly aggressive triple-negative or HER2-positive subtypes [3]. This heterogeneity poses substantial challenges in predicting therapeutic outcomes and tailoring treatment strategies, particularly in complex cases involving advanced disease, multi-drug resistance, or metastatic spread. Accurate and timely monitoring of treatment response is critical for optimizing patient outcomes, guiding therapy modifications, and minimizing unnecessary toxicity [4-6].Current monitoring strategies largely rely on imaging modalities, including mammography, ultrasound, MRI, and PET/CT scans, alongside pathological assessment of tumor biopsies. While these approaches provide valuable anatomical and histological information, they have limitations. Imaging can be costly, logistically challenging, and may not capture subclinical disease progression in real time. Similarly, repeated biopsies are invasive and often impractical, particularly for metastatic or deep-seated tumors. As such, there is an unmet need for minimally invasive, cost-effective biomarkers that can dynamically reflect tumor activity and predict response to therapy [7-9].

The coagulation and fibrinolytic systems have emerged as critical modulators of tumor biology. Malignant tumors can induce a hypercoagulable state by releasing procoagulant factors, activating platelets, and promoting fibrin deposition. This prothrombotic environment not only supports tumor growth and metastasis but also generates measurable circulating biomarkers. D-Dimer, a fibrin degradation product formed during plasmin-mediated breakdown of cross-linked fibrin, reflects systemic fibrin turnover and hypercoagulability. Elevated D-Dimer levels have been associated with higher tumor burden, metastatic potential, and poor prognosis in various malignancies, including breast cancer [10-11].Recent clinical investigations suggest that D-Dimer may serve not only as a prognostic marker but also as a dynamic indicator of treatment response. Serial measurements during chemotherapy, targeted therapy, or immunotherapy can provide real-time insights into tumor activity, offering potential advantages over conventional imaging or laboratory assessments. For instance, reductions in D-Dimer levels may indicate effective tumor cytoreduction, whereas persistent elevation or a rising trend may signify suboptimal response or early recurrence [12-14].

Aim

The aim of this review is to evaluate the clinical utility of D-Dimer monitoring in complex breast cancer cases, with a focus on its potential to predict treatment response, assess disease progression, and guide personalized therapeutic strategies. The review seeks to synthesize current evidence on the physiological and pathophysiological basis of D-Dimer elevation, its prognostic significance, mechanistic underpinnings, and translational implications, highlighting opportunities for integrating this biomarker into routine clinical practice for improved patient outcomes.

Methods

This narrative review was conducted to summarize and critically appraise the current evidence on the clinical utility of D-Dimer monitoring in predicting treatment response in complex breast cancer cases. A comprehensive literature search was performed using electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar, covering publications up to December 2025. Search terms included combinations of: “D-Dimer,” “breast cancer,” “treatment response,” “prognosis,” “biomarkers,” and “coagulation.”

Studies were considered for inclusion if they:

1. Investigated D-Dimer levels in breast cancer patients.
2. Evaluated D-Dimer as a prognostic or predictive biomarker in treatment monitoring.

3. Included clinical, mechanistic, or translational data relevant to therapeutic response.

Both observational and interventional studies, as well as meta-analyses, systematic reviews, and relevant preclinical studies, were considered to provide a comprehensive perspective. Exclusion criteria included studies not available in English, studies with insufficient methodological detail, or reports focusing exclusively on non-breast malignancies without translational relevance.

Data extraction focused on:

- Baseline and serial D-Dimer measurements.
- Correlation with treatment response, disease progression, or recurrence.
- Mechanistic insights linking coagulation and tumor biology.
- Clinical implications and potential for integration into practice.

The narrative synthesis emphasized trends, mechanistic interpretations, and translational relevance rather than statistical meta-analysis, reflecting the heterogeneity of study designs and endpoints. The review aimed to provide an integrative perspective on D-Dimer as a dynamic biomarker in complex breast cancer management.

Physiological and Pathophysiological Basis

D-Dimer is a fibrin degradation product generated during the breakdown of cross-linked fibrin by the fibrinolytic system. Under normal physiological conditions, coagulation and fibrinolysis exist in a tightly regulated balance: coagulation prevents excessive bleeding by forming fibrin clots, while fibrinolysis ensures that clots are degraded once vascular repair is complete. D-Dimer serves as a sensitive marker of ongoing fibrin turnover, reflecting the activity of both coagulation and fibrinolysis [15-16].In the context of malignancy, this balance is disrupted. Tumors, including breast cancers, actively manipulate the coagulation cascade to create a prothrombotic microenvironment that supports growth, invasion, and metastasis. Tumor cells can express procoagulant molecules such as tissue factor, release microparticles that trigger clotting pathways, and stimulate platelets to aggregate. This hypercoagulable state promotes fibrin deposition around tumor cells, facilitating their adhesion, evasion from immune surveillance, and dissemination through the vasculature. The resulting increase in fibrin formation and turnover leads to elevated circulating D-Dimer levels, even in the absence of clinically apparent thrombosis [17-19].

Therapeutic interventions further modulate this coagulation-tumor interplay. Cytotoxic chemotherapy induces tumor cell apoptosis and necrosis, releasing procoagulant cellular contents into the circulation. This can transiently increase D-Dimer levels, reflecting enhanced fibrin turnover rather than solely disease progression. Conversely, effective treatment that reduces tumor burden typically results in a gradual decline in D-Dimer, mirroring decreased prothrombotic activity. In complex breast cancer cases, where tumor heterogeneity and aggressive biology are pronounced, these dynamic shifts in coagulation biomarkers can serve as indirect measures of treatment efficacy [20-21].Moreover, the pathophysiological implications of D-Dimer elevation extend beyond coagulation. High levels are associated with systemic inflammation, endothelial activation, and microvascular thrombosis—all of which contribute to tumor progression and therapy resistance. Persistent D-Dimer elevation may indicate ongoing malignant activity, subclinical metastasis, or a tumor microenvironment that is less responsive to standard therapies. Understanding these mechanistic underpinnings provides a rationale for incorporating D-Dimer as a dynamic biomarker in clinical practice, offering a minimally invasive tool to complement imaging and histopathology in monitoring disease course and therapeutic response(Table 1) [22-24].

Table 1: Physiological and Pathophysiological Basis of D-Dimer in Breast Cancer

Aspect	Physiological Role	Pathophysiological Alteration in Breast Cancer	Clinical Implications
Fibrin Formation and Breakdown	Fibrinogen is converted to fibrin to form stable clots; plasmin-mediated degradation produces D-Dimer	Tumor-induced hypercoagulability increases fibrin deposition and turnover, elevating circulating D-Dimer	Elevated D-Dimer indicates active coagulation and tumor-associated thrombotic activity
Coagulation Cascade	Regulates hemostasis and wound repair	Cancer cells express tissue factor and procoagulant microparticles, amplifying thrombin generation	Reflects tumor aggressiveness and systemic prothrombotic state
Inflammatory Mediators	Cytokines (IL-6, TNF-α) support immune response and tissue repair	Chronic inflammation promotes coagulation activation, contributing to elevated D-Dimer	Links tumor progression, chemoresistance, and risk of venous thromboembolism
Tumor Microenvironment	Maintains vascular integrity and cellular homeostasis	Fibrin matrices facilitate tumor cell adhesion, angiogenesis, and immune evasion	Persistent high D-Dimer indicates tumor growth, metastasis, and poor therapy response
Treatment Response Dynamics	Reflects balanced coagulation and fibrinolysis	Effective chemotherapy reduces tumor burden and procoagulant signaling, potentially lowering D-Dimer	Serial D-Dimer monitoring provides non-invasive insights into treatment efficacy and progression

Clinical Evidence and Prognostic Implications

Clinical studies over the past decade have increasingly highlighted the potential of D-Dimer as a biomarker in breast cancer, particularly in predicting treatment response and disease progression. Observational and prospective analyses consistently demonstrate that elevated baseline D-Dimer levels are associated with advanced tumor stage, lymphovascular invasion, and higher metastatic potential. In aggressive subtypes—such as triple-negative and HER2-positive breast cancers—baseline hypercoagulability, reflected by D-Dimer elevation, often correlates with poorer overall survival and disease-free survival, emphasizing its prognostic relevance [25-26].Beyond baseline assessment, serial D-Dimer monitoring provides dynamic insights into therapeutic efficacy. In the neoadjuvant setting, for instance, studies have shown that patients who exhibit early declines in D-Dimer levels during chemotherapy are more likely to achieve a pathological complete response. Conversely, persistently elevated or rising D-Dimer levels during treatment often indicate suboptimal response or early signs of residual disease. Similar trends have been observed in adjuvant and metastatic settings, where changes in D-Dimer levels can precede radiological or clinical evidence of disease progression, potentially offering an early window for intervention [27-28].

D-Dimer's prognostic utility is not limited to response assessment. Elevated levels at diagnosis or postoperatively have been linked to higher risk of recurrence, including distant metastasis, particularly in patients with lymph node involvement or vascular invasion. This suggests that D-Dimer may reflect systemic tumor activity and micrometastatic disease that is not readily detectable through conventional imaging.

Furthermore, integrating D-Dimer assessment with other clinical and laboratory parameters—such as CA 15-3, circulating tumor cells, or inflammatory markers—has been proposed to enhance predictive accuracy and stratify patients based on risk [29-30].While promising, the clinical implementation of D-Dimer monitoring requires standardized protocols. Variability in assay methods, thresholds, and sampling schedules can impact interpretation, underscoring the need for consensus guidelines. Nevertheless, current evidence supports the notion that D-Dimer serves as both a prognostic biomarker and a dynamic indicator of treatment response, offering clinicians a minimally invasive, real-time tool to monitor disease course and guide personalized management in complex breast cancer cases [31-32].

Mechanistic Insights

The predictive value of D-Dimer in breast cancer treatment response is rooted in the intricate interplay between coagulation, fibrinolysis, and tumor biology. Malignant cells actively manipulate the coagulation cascade, creating a microenvironment conducive to survival, invasion, and metastasis. Tumor-derived tissue factor, microparticles, and pro-inflammatory cytokines initiate thrombin generation, which converts fibrinogen to fibrin. Fibrin not only forms a scaffold supporting tumor architecture but also promotes angiogenesis, protects tumor cells from immune surveillance, and facilitates adhesion to the endothelium during metastatic dissemination [33].D-Dimer reflects the end-product of fibrin degradation, and its circulating levels mirror the degree of tumor-associated coagulation activity. During effective treatment, tumor burden diminishes, reducing procoagulant stimulus and fibrin formation. This leads to decreased fibrin turnover and lower D-Dimer levels. Conversely, persistent tumor activity or therapy resistance sustains the prothrombotic state, resulting in elevated or rising D-Dimer concentrations. In some cases, transient spikes in D-Dimer may occur shortly after initiation of therapy, representing tumor cell apoptosis or necrosis, which increases fibrin degradation without necessarily indicating disease progression [34].

Mechanistically, D-Dimer elevation also intersects with systemic inflammation and endothelial dysfunction, which are hallmarks of aggressive breast cancer. Elevated D-Dimer can indicate microvascular thrombosis, immune cell activation, and release of pro-angiogenic factors, all of which may contribute to therapy resistance and metastatic potential. Additionally, interactions between circulating tumor cells, platelets, and fibrin networks enhance metastatic seeding, providing a direct mechanistic link between coagulation dynamics and disease progression [35].By capturing these complex biological processes, D-Dimer serves as more than a mere marker of coagulation—it functions as an integrated indicator of tumor activity, systemic response, and therapeutic efficacy. Understanding these mechanistic underpinnings provides the rationale for using serial D-Dimer measurements as a minimally invasive tool to monitor treatment response, predict prognosis, and guide timely therapeutic adjustments in complex breast cancer cases [36].

Clinical Translation

The integration of D-Dimer monitoring into clinical practice offers a promising approach to enhance the management of complex breast cancer cases. As a minimally invasive, readily measurable biomarker, D-Dimer provides dynamic information on tumor-associated hypercoagulability, treatment response, and potential early recurrence. Clinically, serial assessment of D-Dimer could complement conventional imaging and laboratory markers, allowing oncologists to detect subclinical disease progression, evaluate therapeutic efficacy in real time, and adapt treatment strategies promptly. For instance, persistently elevated or rising D-Dimer levels during chemotherapy or targeted therapy could trigger closer surveillance, therapy intensification, or early consideration of alternative regimens, potentially improving patient outcomes [37].

Implementation of D-Dimer monitoring in routine practice, however, requires standardization of assay methods, sampling frequency, and clinically meaningful thresholds. Multi-center prospective studies are needed to establish reference ranges specific to breast cancer subtypes and treatment modalities. Integration with other biomarkers, such as circulating tumor DNA, CA 15-3, and inflammatory indices, may further enhance predictive accuracy and risk stratification. Additionally, exploring the relationship between D-Dimer kinetics and emerging therapies, including immunotherapy and novel targeted agents, could inform personalized treatment approaches [37].

Conclusion

D-Dimer has emerged as a promising biomarker in the management of complex breast cancer, offering insights that extend beyond traditional imaging and histopathological assessment. Elevated levels reflect tumor-associated hypercoagulability, systemic inflammation, and disease burden, while serial monitoring provides dynamic information on treatment response and early signs of recurrence. Clinical evidence indicates that reductions in D-Dimer correlate with effective therapy, whereas persistent or rising levels may signal residual disease or treatment resistance. Mechanistically, these changes mirror the intricate interactions between tumor cells, coagulation pathways, and the tumor microenvironment, underscoring D-Dimer's role as an integrated indicator of disease activity.

Incorporating D-Dimer monitoring into clinical practice has the potential to enhance patient stratification, guide adaptive therapeutic decisions, and improve outcomes, particularly in aggressive or treatment-resistant breast cancer subtypes. Standardization of measurement protocols, validation in prospective studies, and integration with complementary biomarkers are essential next steps to fully realize its clinical utility. Overall, D-Dimer represents a minimally invasive, real-time biomarker that bridges mechanistic understanding with practical oncology care, offering a valuable tool for personalized management in complex breast cancer cases.

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