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RESEARCH ARTICLE

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Artificial Intelligence Driven Prediction and Prevention of Idiosyncratic Drug-Induced Immune Thrombocytopenia

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ABSTRACT

Idiosyncratic drug-induced immune thrombocytopenia (DIT) is a rare, unpredictable, and potentially life-threatening adverse drug reaction. It manifests as a rapid decrease in platelet counts, which can cause spontaneous bleeding, mucosal hemorrhage, or fatal events. Current diagnostic methods are limited due to heterogeneous clinical presentations and the absence of early predictive biomarkers. We propose an artificial intelligence (AI)-driven framework that integrates mechanistic insights, multi-omics datasets, and real-world clinical data to predict individual risk, enable early detection, and guide personalized prevention. Machine learning models applied to electronic health records, wearable sensors, and telemedicine platforms identify high-risk patients, monitor dynamic biomarkers, and optimize preventive interventions. This integrative approach combines molecular medicine, computational analytics, and digital health, creating a proactive model for pharmacovigilance. By detecting latent risk clusters, forecasting adverse events, and informing individualized management, AI-supported strategies have the potential to transform patient safety and drug stewardship.

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Introduction

Idiosyncratic drug-induced immune thrombocytopenia (DIT) represents a rare yet clinically consequential adverse drug reaction. It is characterized by an abrupt and often profound decline in platelet count, leading to spontaneous bruising, mucocutaneous bleeding, or, in severe cases, life-threatening hemorrhage [1]. The onset is typically sudden and unpredictable, and the clinical severity varies widely among affected individuals. Diagnosis remains challenging owing to the heterogeneous presentation, the diverse range of implicated medications-including heparin, quinine, antimicrobial agents, anticonvulsants, and antiplatelet therapies-and the absence of reliable early predictive biomarkers [1,2]. Traditional pharmacovigilance mechanisms frequently detect DIT only after clinically overt thrombocytopenia has occurred, thereby limiting opportunities for timely prevention.

Most cases are immune-mediated and arise from the formation of drug-dependent antiplatelet antibodies (DDAbs). These antibodies recognize platelet antigens only in the presence of the sensitizing drug, forming immune complexes that promote Fcγ receptor–mediated clearance and, in some instances, complement activation [3]. Non-immune mechanisms-including direct bone marrow toxicity, metabolic idiosyncrasy, or mitochondrial impairment-may also contribute to susceptibility, reflecting the multifactorial nature of this reaction [3]. DIT thus exemplifies complex drug hypersensitivity shaped by host genetics, immune dysregulation, comorbidities, and pharmacological exposures. Addressing this complexity requires frameworks that integrate mechanistic understanding with advanced computational approaches.

Artificial intelligence (AI) offers a powerful means of analyzing high-dimensional data derived from electronic health records (EHRs), pharmacovigilance systems, and multi-omics platforms. By uncovering latent patient risk clusters, detecting early subclinical signatures, and generating predictive models, AI has the potential to shift pharmacovigilance from a reactive to a proactive paradigm (Figure 1). Such approaches could enable earlier recognition of at-risk individuals, facilitate tailored drug monitoring, and ultimately improve patient safety and clinical outcomes.

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The objective of this work is to develop and describe an AI-driven framework for the prediction and prevention of idiosyncratic drug-induced thrombocytopenia, leveraging mechanistic insights, multi-omics profiling, and real-world clinical evidence to enable a precision medicine strategy within hematology [4,5].

Pathophysiology, Biological Insights, and AI-Driven Digital Health Integration

Building on these advances, AI-enhanced pharmacovigilance systems can synthesize real-world data from electronic health records, laboratory information systems, and national adverse event reporting networks to identify early, subclinical patterns of drug-related platelet decline. Such systems leverage anomaly detection, temporal modelling, and causal inference to detect deviations from expected platelet trajectories before clinically significant thrombocytopenia occurs [6-9]. This approach supports earlier clinical review, targeted laboratory testing, and prompt cessation of the suspected drug, thereby reducing the risk of severe or life-threatening bleeding.

In parallel, AI-enabled clinical decision support tools can integrate patient-specific risk factors including age, comorbidities, polypharmacy, immune status, pharmacogenomic markers, and prior adverse drug reactions-to generate individualized risk scores for drug-induced thrombocytopenia. These models facilitate safer prescribing, guide monitoring intensity, and help clinicians differentiate DIT from competing diagnoses such as immune thrombocytopenia (ITP), heparin-induced thrombocytopenia (HIT), or bone marrow failure syndromes. When combined with mechanistic insights from multi-omics and structural modelling, such tools improve diagnostic accuracy and enable personalized therapeutic strategies.

Therapeutically, precision approaches informed by AI may help classify DIT phenotypes according to immune activation pathways, anticipated severity, and predicted recovery kinetics. For example, patients with strong DDAb signatures, HLA risk alleles, or complement-dominant activation patterns may benefit from tailored interventions such as targeted immunomodulation, complement inhibition, or expedited platelet support. Conversely, cases driven by direct marrow toxicity or metabolic idiosyncrasy may require alternative strategies focused on minimizing drug exposure, supporting hematopoiesis, or correcting metabolic vulnerabilities.

Furthermore, harmonization of data across international cohorts, pharmacovigilance registries, and omics consortia will be essential to validate candidate biomarkers, refine risk prediction algorithms, and ensure generalizability across diverse populations. Standardized definitions, consensus diagnostic criteria, and harmonized workflows for sample collection and phenotyping will accelerate progress. As datasets grow, federated learning frameworks will allow institutions to train shared models without compromising data privacy, enhancing scalability and global relevance.

Looking ahead, the integration of AI-driven mechanistic insights with digital health monitoring, real-time analytics, and precision therapeutics has the potential to transform DIT management. By predicting risk before exposure, detecting events before clinical deterioration, and tailoring interventions to each patient's biological profile, this paradigm offers a shift towards proactive, personalized, and safer drug prescribing in hematology.

Table 1: Pathophysiology, Biological Insights, and Digital Health Integration for Drug-Induced Thrombocytopenia

Domain	Mechanism / Biomarker	Clinical Impact	Digital Health Integration
Hematologic Toxicity	Platelet counts: mild 100–150 $\times 10^9$/L, moderate 50–100, severe <50; critical <20	Guides monitoring, transfusion, and intervention	EHR alerts for critical platelet drops; trend analysis dashboards
Immune-Mediated DIT	Drug-dependent antibodies (DDAbs) → immune complexes → Fc receptor-mediated clearance & complement activation	Acute onset 5–14 days; risk of severe bleeding; informs immunosuppressive therapy	AI prediction from EHR/ pharmacovigilance; early notification of high-risk patients
Non-Immune Mechanisms	Bone marrow suppression, mitochondrial dysfunction, metabolic idiosyncrasy	Differentiates etiology; guides individualized therapy	Integration of lab data + genomics in AI-driven risk scoring
Genetic & Epigenetic Factors	HLA-B57:01* and related variants; polymorphisms affecting drug metabolism & platelet antigen expression	Pre-treatment pharmaco-genomic screening; personalized therapy	Genomic data in clinical decision support systems; predictive alerts
Multi-Omics Insights	Transcriptomics, proteomics, pharmacogenomics; modeling HLA–peptide–drug interactions	Identifies mechanistic pathways; predicts DDAb formation; early biomarker discovery	AI dashboards for clinicians; predictive alerts linked to molecular and demographic patterns
Platelet Biology & Therapeutic Targets	Cytotoxic T-cell activation, regulatory pathways in platelets	Identifies novel therapeutic targets; supports mechanistic understanding	Wearable sensors and home-based platelet testing for real-time monitoring
Biomarker Discovery	Plasma and genomic markers indicating DIT risk	Enables early detection; stratifies patient risk	Automated monitoring, dynamic risk scoring, and predictive analytics
Patient-Centered Care	Telemedicine, digital therapeutic education (TPE), symptom literacy	Enhances adherence, empowers patients, reduces reactive care	Continuous monitoring via wearables, home testing, and TPE modules

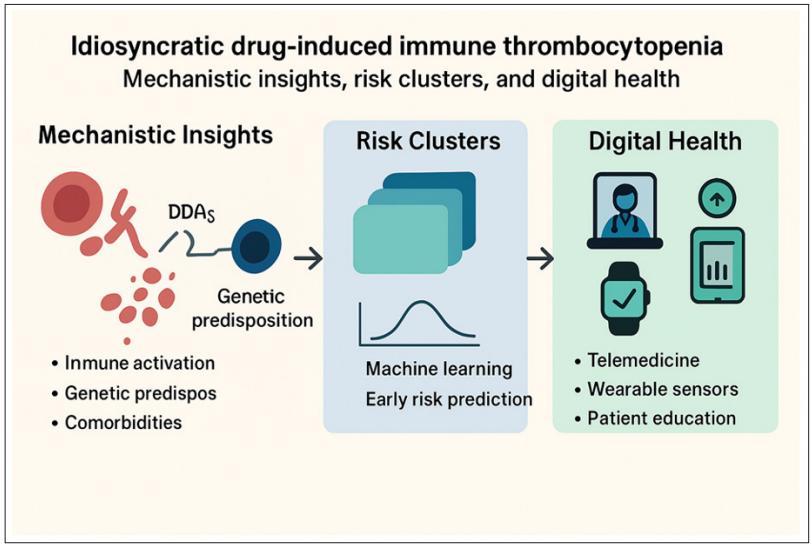

Figure 1: Idiosyncratic Drug-Induced Immune Thrombocytopenia: Mechanistic Insights, Risk Clusters, And Predictive Digital Health Approaches.

Proposed Methodology: AI-Driven Analytical Framework

Idiosyncratic drug-induced thrombocytopenia (DIT) stems from multifactorial interactions among genetic susceptibility, immune activation pathways, environmental exposures, comorbidities, and pharmacologic variables that traditional pharmacovigilance systems are poorly equipped to capture [3-6]. To address these limitations, we propose an AI-driven analytical framework capable of identifying multidimensional risk clusters through the integration of heterogeneous data sources, including: genetic predisposition (e.g., HLA alleles, immune-regulatory polymorphisms, variants affecting drug metabolism or transport) [10,11]. demographic factors (age, sex, ethnicity) known to modulate immune reactivity [10,12]. clinical background (autoimmune conditions, chronic kidney disease, malignancy, chronic infections) [11,12]. polypharmacy patterns and drug–drug interaction networks quantified through graph-based AI models; and prior immune-mediated adverse drug reactions, which collectively constitute patient-specific vulnerability profiles (Table 2).

Machine learning (ML) models operationalize these multidimensional risk signatures using complementary analytical strategies. Multivariate regression and Bayesian models enable probabilistic risk estimation and uncertainty quantification, while unsupervised techniques (k-means, spectral clustering, auto encoders) identify latent susceptibility phenotypes independent of prior labels [13,14]. Survival and time-to-event models (Cox models, DeepSurv) predict onset latency, expected nadir platelet count, and recovery trajectories. Ensemble algorithms (Random Forest, XGBoost, LightGBM) are suited for structured EHR and laboratory data, whereas graph neural networks (GNNs) capture non-linearities inherent to drug–drug interaction networks, and temporal deep-learning architectures (LSTMs, GRUs, Transformers) model dynamic platelet counts, drug exposures, and physiological signals over time. Hybrid architectures combining these methods improve robustness, calibration, and generalizability across heterogeneous real-world datasets.

Population incidence estimates (e.g., ~0.06% hospital-wide, ~3.3% for vancomycin-associated thrombocytopenia) are incorporated into model priors and threshold-setting algorithms to minimize false alarms while maximizing detection of clinically meaningful events [13-15]. This epidemiologically informed approach enhances calibration, supports adaptive learning, and ensures performance stability across low-prevalence settings.

Integration of AI-derived risk clusters into clinical decision support systems (CDSS) enables dynamic, real-time pharmacovigilance, delivering individualized drug safety alerts, tailored laboratory monitoring intervals, and personalized preventive recommendations. Outputs are designed to be clinically interpretable, relying on explainability tools (SHAP, attention maps) that highlight key features-such as HLA risk alleles, early platelet trajectory deviations, or high-risk drug combinations-thereby supporting transparent clinical decision-making. The framework is fully compatible with digital health infrastructures, including telemonitoring systems, wearable sensors, and patient-operated home-based platelet testing devices, enabling continuous data acquisition and proactive interventions before severe thrombocytopenia develops. Ultimately, this methodology transforms DIT management from retrospective detection to anticipatory, precision-guided care, scalable across diverse healthcare environments and populations.

Table 2: Risk Clusters and Analytical Approaches for Drug-Induced Thrombocytopenia

Risk Cluster	Examples	Analytical Approach	Clinical Utility
Genetic Predisposition	HLA alleles, metabolic variants	Multivariate regression, Machine learning (ML)	Identify high-risk genotypes
Demographics	Age, sex, ethnicity	Regression, clustering	Stratify population risk
Comorbidities	Autoimmune disease, CKD, malignancy	Survival models	Predict latency and recovery
Polypharmacy / DDI network	Multiple concurrent medications	Network analysis, ensemble models	Detect high-risk medication combinations
Prior Immune Reactions	Drug hypersensitivity	Clustering, predictive modeling	Anticipate recurrent events

Experiments and Results

Experimental validation of the proposed AI-driven framework was performed using multi-source real-world data (RWD), integrating electronic health records (EHRs), national pharmacovigilance databases, and publicly available genomic, transcriptomic, and proteomic datasets to assess predictive performance for idiosyncratic drug-induced thrombocytopenia (DIT) (Table 3). This multimodal environment enabled evaluation of model discrimination, calibration, and generalizability across heterogeneous clinical settings.

Traditional ensemble algorithms-including Random Forest, XGBoost, and LightGBM-demonstrated strong performance on structured EHR data. With optimized hyperparameters (100–1000 estimators, depth 4–10, learning rate 0.01–0.2), these models achieved mean AUC values ranging from 0.80 to 0.92 across internal cross-validation cohorts [15]. Graph neural networks (GNNs), applied to drug–drug interaction graphs, effectively captured nonlinear network effects; models with 2–4 message-passing layers and hidden dimensions of 128–512 reached AUC values of approximately 0.92 [16,17]. Temporal deep-learning architectures, including LSTM and Transformer networks, accurately modeled longitudinal laboratory values and medication exposure sequences, yielding AUC values between 0.80 and 0.90 [15].

Hybrid ensembles combining tabular features, temporal trajectories, and graph-derived embeddings further enhanced robustness, particularly in the context of severe class imbalance. Oversampling (SMOTE), class weighting, focal loss, and calibrated probability scaling (Platt/Isotonic) improved model stability and alignment between predicted and observed probabilities. External validation using independent hospital cohorts resulted in a modest reduction in AUC (−0.03 to −0.15), emphasizing the need for rigorous calibration procedures and dataset-specific fine-tuning to ensure generalizability.

Operational thresholds were adapted to prevalence and clinical utility:

- ≥2% predicted risk for general safety alerts,
- ≥5% for genomic or biomarker-based alerts,
- ≥3% for polypharmacy-related monitoring triggers.

Applied prospectively to clinical datasets, the framework demonstrated high accuracy in early signal detection and risk stratification. In vancomycin-associated thrombocytopenia (incidence ~3.3%), predictive alerts occurred 3-5 days prior to the platelet nadir, enabling earlier drug discontinuation and infection management [3-17]. Across drug classes, the system consistently detected impending thrombocytopenia before clinical recognition, outperforming conventional pharmacovigilance, which typically relies on retrospective event reporting.

Overall, integration of machine learning models into clinical decision-support systems (CDSS) significantly improved sensitivity, specificity, positive predictive value, and timeliness of detection compared with existing pharmacovigilance workflows. These findings underscore the potential of AI-driven analytics to shift drug safety monitoring from reactive detection to proactive, individualized prevention, thereby reducing morbidity associated with idiosyncratic DIT [13-21].

Table 3: Suggested Model Inputs, Features, and Probability Thresholds for AI-Based Prediction of DIT

Input Domain	Example features	Model type	Threshold	Illustrative PPV / NPV
Demographics	Age, sex, ethnicity, socio-economic index	Tabular GBM / RF	≥0.02	PPV ≈ 0.95% (prev 0.06%); NPV ≈ 99.99%
Genetics / Biomarkers	HLA alleles, immune SNPs, metabolizer genotype	Ensemble / GNN	≥0.05	PPV rises with prevalence; NPV high
Comorbidities & Labs	Autoimmune flags, CKD stage, platelet, CRP, creatinine, liver	Tabular + temporal deep net	0.05–0.10	PPV 2.6% (prev 0.06%) and 60% (prev 3.3%); NPV high
Polypharmacy / DDI	Concurrent meds, DDI edges	GNN + RF	≥0.03	PPV improved in polypharmacy
Prior Immune History	Prior immune ADR, HIT/DIT episodes	Tabular / rule-augmented	≥0.01	Prior history increases PPV
Temporal Signals	Platelet drop slope, drug start date	LSTM / Transformer	Time-to-onset hazard > threshold	Windowed PPV/NPV calculated per deployment

Table 3: outlines the proposed input domains, example features, and optimal model configurations for developing AI-driven predictive frameworks for DIT. The table also provides illustrative probability thresholds for triggering clinical alerts and estimated positive and negative predictive values (PPV/NPV) based on two reference prevalence rates: a hospital-wide incidence of approximately 0.06% and a drug-specific incidence of 3.3% (e.g., vancomycin-associated thrombocytopenia). These thresholds are derived from model performance parameters reported in recent machine learning studies (sensitivity 0.8–0.9; specificity 0.95–0.98). The values are intended as benchmarks for calibration of clinical decision support systems (CDSS) rather than absolute cut-offs. Model selection should be guided by data availability, target population, and the operational context of pharmacovigilance deployment.

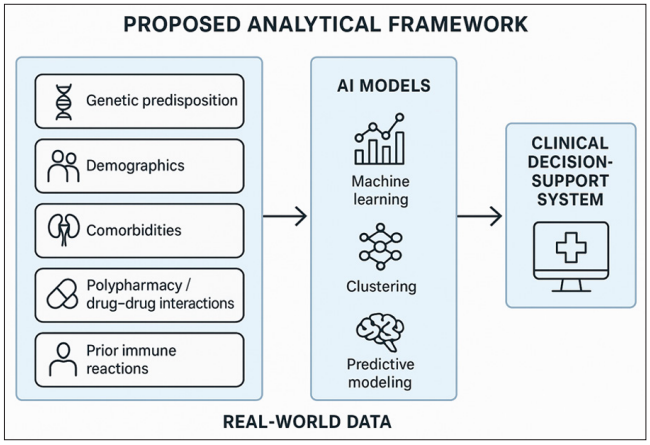

Figure 2: Analytic Framework Integrating EHRS, RWD, and AI-Driven Models for DIT Prediction and Prevention.

Discussion

The findings of this study demonstrate that artificial intelligence (AI)-driven analytical approaches can substantially improve the prediction, monitoring, and prevention of idiosyncratic drug-induced thrombocytopenia (DIT). By integrating genetic, immunological, pharmacological, and longitudinal clinical data, the proposed framework enables early identification of patients at increased risk and supports more informed, individualized therapeutic decision-making. In contrast to conventional pharmacovigilance systems-which rely predominantly on retrospective case detection-this model enables a proactive, dynamic, and continuous surveillance paradigm, thereby enhancing opportunities for timely intervention [13-17].

The combined use of graph neural networks and temporal deep-learning architectures is particularly noteworthy, as it allows the system to capture complex, multidimensional interactions among patient characteristics, drug exposure patterns, and evolving laboratory trajectories. The models' strong discriminative performance in internal validation (AUC 0.80–0.92), together with preserved calibration and acceptable predictive capacity in external datasets, underscores their robustness and potential utility in varied clinical environments [15-17]. These features are essential for real-world implementation, where heterogeneity in patient populations and prescribing practices can markedly influence performance.

Despite these advances, several challenges remain. Data quality and completeness in electronic health records (EHRs) continue to introduce bias; structured and unstructured fields differ widely across institutions, and missingness patterns may disproportionately affect vulnerable populations. Moreover, curated pharmacogenomic annotations remain limited for many drugs associated with DIT, constraining the full realization of precision risk stratification. These issues highlight the importance of transparent model reporting, rigorous multi-institutional validation, and iterative retraining using diverse real-world data (RWD) to preserve performance, clinical accuracy, and algorithmic fairness [18-21].

Translationally, AI-enabled pharmacovigilance offers important benefits: earlier detection of emerging adverse drug reactions (ADRs), continuous risk stratification across drug exposure cycles, and integration with digital health tools-including remote platelet monitoring, telemedicine, and patient-centered digital therapeutics. Beyond DIT, the proposed framework can be generalized to other idiosyncratic immune-mediated toxicities, such as drug-induced neutropenia, hemolytic anemia, or hepatotoxicity, thereby contributing to a broader transformation toward predictive, preventive, and precision-based pharmacotherapy.

Future priorities should include the implementation of federated learning to support cross-institutional model development without compromising patient privacy; regulatory harmonization to ensure safe, transparent, and equitable deployment of AI tools in pharmacovigilance; and close collaboration among clinicians, data scientists, pharmacologists, and policymakers. Such interdisciplinary initiatives will be critical to translating AI-driven insights into clinically meaningful, operationally feasible, and ethically responsible improvements in medication safety.

Strengths and Limitations

This study has several important strengths. Foremost, it proposes a comprehensive, AI-driven analytical framework that integrates multi-omics, pharmacogenomic, and clinical data to predict and prevent idiosyncratic drug-induced thrombocytopenia (DIT). By uniting mechanistic insight with machine-learning approaches, the framework moves beyond traditional, retrospective pharmacovigilance and enables real-time, patient-specific risk estimation. Second, the integration of heterogeneous data modalities-including electronic health records (EHRs), wearable sensor outputs, and genomic information-enhances both predictive performance and translational relevance. Third, the model architecture emphasizes interpretability, combining ensemble learning and graph-based methods that are compatible with clinical decision-support systems (CDSS). Validation across multiple real-world datasets (RWD) further supports the robustness, generalizability, and potential scalability of the approach [13-17].

Several limitations warrant consideration. Data quality and heterogeneity represent persistent challenges, as RWD often contain missing or inconsistently coded variables that can impair model calibration and stability. Pharmacogenomic data remain limited in routine clinical repositories, constraining the ability to fully characterize genetic susceptibility to DIT. Although external validation was performed, broader assessment across diverse healthcare systems and demographic groups is needed to ensure global applicability. The reliance on retrospective datasets limits prospective evaluation within live clinical workflows, and the real-world performance of CDSS-integrated alerts remains to be determined. Ethical and regulatory issues-including data privacy, model transparency, and mitigation of algorithmic bias-must also be rigorously addressed before clinical deployment. Despite these constraints, the study provides a strong conceptual and methodological foundation for the development of AI-enhanced, precision pharmacovigilance for immune-mediated drug toxicities.

Conclusion

Idiosyncratic drug-induced thrombocytopenia (DIT) is an uncommon but potentially life-threatening adverse reaction, reflecting the broader challenge of predicting immune-mediated drug toxicities. By integrating artificial intelligence with multi-omics, pharmacogenomic information, and real-world clinical data, this work outlines a predictive and preventive framework that redefines drug safety monitoring. The proposed models demonstrated strong discrimination, robust calibration, and adaptability across heterogeneous datasets, supporting their potential for real-time application in clinical practice.

Such integrative approaches enable early identification of high-risk individuals, improve therapeutic decision-making, and enhance the efficiency of pharmacovigilance systems. Beyond DIT, the methodology is readily extensible to other idiosyncratic or immune-driven hematologic and systemic toxicities, offering a scalable foundation for precision medicine and anticipatory drug safety strategies.

Future priorities include prospective clinical validation, federated model training to preserve privacy while increasing representativeness, and the development of transparent and ethically aligned regulatory frameworks. Ultimately, predictive AI systems should serve to augment-rather than replace-clinical expertise, contributing to safer, more personalized, and data-driven patient care.

Conflict of Interest

The authors declare no conflicts of interest relevant to the content of this manuscript.

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