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ROLE OF ANTITHROMBOTICS IN CANCER SURVIVAL

CLINICAL IMPLICATIONS OF ANTICOAGULANT DRUG INTERACTIONS WITH TARGETED AND IMMUNE THERAPIES IN ONCOLOGY: A CONTEMPORARY RISK STRATIFICATION FRAMEWORK

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Background. Anticoagulation in patients with cancer is challenging due to competing risks of thrombosis and bleeding. The expanding use of targeted therapies and immune checkpoint inhibitors further complicates management, as many agents interact with commonly used anticoagulants. Recent guidance from the American Society of Clinical Oncology and American Heart Association along with emerging registry and pharmacokinetic data, provides an opportunity to refine anticoagulation strategies in this population.

Aims. This review evaluates drug–drug interactions (DDIs) between anticoagulants and cancer-directed therapies using pharmacokinetic, clinical, and registry data. We propose a practical, risk-based framework to guide anticoagulant selection.

Methods. We reviewed registry studies, systematic reviews, expert consensus guidelines, and pharmacologic data addressing DDIs between anticoagulants and cancer therapies. Interaction risk was categorized and incorporated into a clinical framework for selecting direct oral anticoagulants (DOACs), low molecular weight heparin (LMWH), or vitamin K antagonists (VKAs) in patients receiving VEGF, BTK, EGFR, ALK, HER2, and immune checkpoint inhibitors.

Results. Targeted therapies demonstrate variable DDI profiles with DOACs, largely mediated by CYP3A4 and P-glycoprotein pathways. BTK inhibitors, particularly ibrutinib, are associated with increased bleeding risk due to combined pharmacokinetic, antiplatelet, and arrhythmogenic effects. VEGF inhibitors also confer elevated bleeding risk when combined with certain DOACs. In contrast, most immune checkpoint inhibitors lack clinically significant pharmacokinetic DDIs, though immune-mediated toxicities may contribute to bleeding. A structured classification system was developed to guide anticoagulant selection. LMWH is preferred in patients receiving strong CYP3A4/P-gp modulators or with GI/GU malignancies, while VKAs are often deprioritized due to unpredictable effects. Registry data suggest bleeding events exceed recurrent thrombosis in select high-risk pairings.

Conclusions. We present a clinically actionable framework for anticoagulation management in cancer patients receiving targeted and immune therapies. Risk-based anticoagulant selection, informed by pharmacologic and clinical data, can reduce adverse events while maintaining efficacy.

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Agent/Class	CYP3A4/P-gp Modulation	Clinical DDI Risk with DOACs	Recommendation
BTK Inhibitors (e.g., ibrutinib, acalabrutinib)	Yes (CYP3A4 inducer)	High (↑ bleeding, ↓ efficacy of DOACs)	Avoid DOACs; prefer LMWH
VEGF Inhibitors (e.g., bevacizumab)	No significant data	Intermediate (↑ bleeding risk, esp. with DOACs)	Consider LMWH if bleeding risk is high
EGFR/ALK Inhibitors (e.g., osimertinib, alectinib)	Minimal	Low	DOACs acceptable with monitoring
HER2 Inhibitor (e.g., neratinib)	Likely (CYP3A4 substrate)	High (pharmacovigilance signal for bleeding)	Avoid DOACs; prefer LMWH
Checkpoint Inhibitors (e.g., nivolumab, ipilimumab)	No	Very Low / None	DOACs or LMWH acceptable
Warfarin (with cancer agents)	Warfarin is CYP-metabolized	High (↑ bleeding with tamoxifen, capecitabine)	Avoid if possible; prefer LMWH or DOACs