Pharmacokinetic interactions between anticancer drugs and DOACs: clinical implications for safe anticoagulation

Tzu-Fei Wang,[1] Kristen M. Sanfilippo,[2,3] Marc Carrier[1]

[1]Department of Medicine, University of Ottawa at The Ottawa Hospital and Ottawa Hospital Research Institute, Ottawa, ON, Canada; [2]Department of Medicine, Washington University School of Medicine, Saint Louis, MO, USA; [3]John Cochran Saint Louis Veterans Administration Medical Center, Saint Louis, MO, USA

ABSTRACT

Patients with cancer have an increased risk of thrombosis requiring anticoagulation. Direct oral anticoagulants have become the primary anticoagulant of choice in most patients with cancer. Patients with cancer also receive many concomitant medications including anticancer therapies with potential risks of drug-drug interactions with anticoagulants that can lead to concerns of bleeding or recurrent thrombosis. The best management strategies remain unclear. In this article, we aim to review the available literature pertaining to pharmacokinetic interactions between anticancer therapies and direct oral anticoagulants and provide guidance based on the evidence.

Key words: drug-drug interaction; cancer-associated thrombosis; venous thromboembolism; anticoagulation; malignancy.

Corresponding author: Tzu-Fei Wang, The Ottawa Hospital, General Campus, 501 Smyth Road, Box 201A, Ottawa, ON, K1H 8L6, Canada. E-mail: tzwang@toh.ca

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Introduction

Patients with cancer have an increased risk of thromboembolism and are commonly treated with anticoagulant therapy. Multiple recent randomized controlled trials have established the role of direct oral anticoagulants (DOACs) in the treatment and prevention of cancer-associated venous thromboembolism (VTE).[1-6] In addition, DOACs are also the anticoagulant of choice for stroke prevention in patients with atrial fibrillation (AF), including those with cancer.[7] Therefore, DOACs are widely used in patients with cancer given the favorable safety and convenience profiles and the prevalence of the aforementioned indications in cancer. However, potential drug-drug interactions (DDIs) remain a main concern when considering DOAC use and are cautioned in major international guidelines.[8-11]

Polypharmacy is common in patients with cancer with studies citing a median of 9 prescribed oral therapies with medication indications including: anticancer therapies, supportive care therapies to relieve side effects, as well as medications for other co-morbidities.[12] In addition to prescription therapy, many patients also report use of over-the-counter medications and herbal supplements.[12] Polypharmacy increases the risk of DDIs with DOACs which include both pharmacokinetic (PK) and pharmacodynamic (PD) interactions. Pharmacokinetic interactions occur when concomitant medications share metabolic pathways, with one drug causing alteration in drug absorption, distribution, metabolism, or extraction of another drug. Pharmacodynamic interactions happen when the presence of one drug enhances the effect of another drug.

The main pathways involved in PK interactions between DOAC and anticancer therapies involve cytochrome (CYP) 3A4 and/or P-glycoprotein (p-gp) pathways. Among DOACs, rivaroxaban and apixaban are the two most used worldwide and they are metabolized through both CYP3A4 and p-gp pathways, while edoxaban and dabigatran are metabolized through the p-gp path-

way only. Consequently, rivaroxaban and apixaban are frequently cited to carry higher risk of PK interactions than edoxaban or dabigatran, given additional involvement with CYP enzymes. Anticancer therapies that inhibit CYP3A4 or P-gp enzymes can lead to an increase in serum levels of a concurrently used DOAC, which could increase the risk of anticoagulant-related bleeding. Conversely, drugs with enhancing effects of these enzymes could reduce DOAC levels, resulting in an insufficient anticoagulant effect and thus thrombotic complications. Edoxaban is the only DOAC with specific dose adjustment criteria in the setting of DDIs, with recommendation of dose reduction to 30 mg daily (from the standard dose of 60 mg daily) when edoxaban is used concomitantly with a p-gp inhibitor except for amiodarone and verapamil.[13]

On the other hand, anticancer therapies can have important PD interactions with anticoagulants. The prime examples include Bruton's tyrosine kinase (BTK) inhibitors and VEGF (vascular endothelial growth factor) receptor inhibitors. BTK inhibitors have known antiplatelet properties and when used with anticoagulants, have led to concerns of major bleeding complications such as fatal intracranial bleeding in early clinical trials.[14] VEGFR inhibitors have also been reported to potentiate LMWH levels, resulting in an increased risk of bleeding.[15] These all add to the complexity in the consideration of combined anticancer and anticoagulation use.[16]

In this narrative review, we will focus on PK interactions between anticancer therapies and DOACs. We summarize the evidence of selected anticancer therapies most commonly cited for PK interaction concerns with DOACs (Table 1).[17-21] PD interactions, as well as DDIs with other medications such as antifungal, supportive care drugs, *etc*., are also important considerations in this population but are outside of the scope of this article. We aim to provide a comprehensive review of currently available data and guidance for management based on the available evidence. Selective anticancer therapies are grouped by the type of cancer for which they are most used. Whenever possible, we put emphasis on human studies that provide clinically relevant patient outcomes.

Methods

For this narrative review, we performed a literature search in PubMed database up to December 2025 with combination of search terms including ("name of anticancer therapy") AND ("direct oral anticoagulant" OR "anticoagulation" OR "rivaroxaban" OR "apixaban" OR "edoxaban" OR "dabigatran"). Given limited evidence in many anticancer therapies, to be as comprehensive as possible, we included all types of studies available without restriction to sample size, including randomized controlled trials (if any), prospective and retrospective cohort studies, and case series.

General cancer population

Most conventional chemotherapy agents are substrates of CYP450 or p-gp transporters but not strong inhibitors or inducers of these pathways, and therefore DOACs generally do not have relevant PK interactions with chemotherapy.[16] A secondary analysis of the Caravaggio trial (a randomized controlled trial comparing dalteparin to apixaban in patients with acute cancer-associated VTE) compared patients on anticancer therapies at any time during the study to those not on anticancer therapy, and found no differences in recurrent VTE or bleeding events with either apixaban or dalteparin.[22] Subgroup analyses by class of anticancer therapy or CYP/p-gp involvement (inducers, inhibitors, or none) showed similar results with no differences in outcomes. However, many different anticancer therapies were included, and the number of patients with each individual therapy combination was small, limiting the power of the study.

More recently, we reported our findings in a single center retrospective cohort study of 267 patients treated with anticoagulation for cancer-associated VTE to evaluate the clinical relevance of DDIs.[23] We defined DDI as any drug combination classified as interaction risk C, D, or X in Lexidrug® specified to affect anticoagulant efficacy or safety at any time during the 6-month study period. The incidence of DDIs was high at 41.8%, with more interactions with DOACs compared to low-molecular-weight heparin (LMWH). However, most of the interactions were with supportive care medications (such as anti-nausea medications), with only 4 patients (1.5%) noted to have anticoagulant-anticancer therapy interactions, all due to CYP/p-gp inhibiting or inducing effects with a DOAC. In the entire cohort, there were no statistically significant differences in clinical outcomes, including recurrent VTE or major bleeding events, in those with DDIs compared to those without.

Breast cancer

Tamoxifen

Breast cancer is the most common cancer in women, with 70-80% being hormone receptor positive.[24] Tamoxifen is a commonly used hormonal therapy for estrogen receptor positive (ER+) breast cancer and is a CYP/p-gp inhibitor, leading to cautioned concurrent use with a DOAC in some guidance papers.[17-19,25] To investigate the clinical relevance of this potential DDI, few population-based administrative database studies have been done. In a Taiwanese national cohort study including 13,158 patients with cancer and AF on a DOAC, 147 patients received concurrent tamoxifen and DOAC therapy.[26] When compared to DOAC therapy alone, concurrent use was not associated with an increased risk of major bleeding (rate ratio [RR] 0.94, 95% confidence interval [CI] 0.38-2.33). Another study in Canada included 4,753 patients ≥66 years old with breast cancer and compared the risk of major bleeding leading to emergency room presentation or hospitalization in DOAC-tamoxifen combination therapy to DOAC-aromatase inhibitor (AI) combination therapy.[27] Compared to DOAC-AI users, DOAC-tamoxifen users had no increased risk of major bleeding (weight hazard ratio [wHR] 0.68, 95% CI 0.44-1.06) or any bleeding event (wHR 1.04, 95% CI 0.75-1.43). An additional small, proof-of-concept study enrolled 24 patients with ER+ breast cancer scheduled to start tamoxifen. Patients were given edoxaban 60 mg daily for 4 days, first without concurrent tamoxifen, then with tamoxifen, and a PK model was developed based on serial blood samples.[28] They found no differences in various PK parameters of edoxaban, including concentration max (Cmax), area under the curve (AUC), regardless of the presence or absence of tamoxifen, and concluded that there are no significant DDIs between tamoxifen and edoxaban. These studies sup-

port the safety and effectiveness of continued anticoagulant therapy with a DOAC in patients on concurrent tamoxifen for treatment of breast cancer.

Lapatinib

Lapatinib, a HER2 (Human Epidermal Growth Factor receptor 2) inhibitor, is a strong inhibitor of both CYP and P-gp pathways.[19] Despite being cautioned against for combination use with DOACs in several guidance articles,[19,29] there are no dedicated clinical studies evaluating outcomes with their combination use, except for a small subgroup in the TacDOAC study.[30] The TacDOAC study is an international registry of patients on concurrent DOAC therapy and select targeted anticancer therapies with DDI potential, including lapatinib.[30] Among the 202 patients included in the study, 4 were taking concurrent lapatinib and DOACs, and no thrombosis or bleeding events were reported within 6 months.

Ribociclib

Ribociclib, a cyclin-dependent kinase 4/6 inhibitor (CDK4/6i), is frequently indicated in patients with hormone receptor positive, HER2 negative breast cancer in both the adjuvant and metastatic settings. In-vitro studies show that it is a moderate to strong CYP3A4 inhibitor as well as a P-gp inhibitor.[31] In a recent in-vivo PK study in rats, ribociclib increased the exposure to rivaroxaban and apixaban (measured by PK parameters including AUC and Cmax) when used concomitantly, with greater impact with rivaroxaban.[32] However, no human studies investigating DDIs between Ribociclib and DOACs are available. Despite the

Table 1. Summary of anti-cancer drugs with potential drug-drug interactions with direct oral anticoagulants.

Anti-cancer drugs	*In vitro* PK effects	Theoretical effect in combination with DOAC	Clinical studies	Level of evidence	Recommendations*
Tamoxifen	CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	No increased risk of bleeding comparing tamoxifen+DOAC to AI+DOAC or DOAC alone[24,25]	Human observational	Likely safe
Lapatinib	Strong CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	No studies	*In vitro*	Monitor
Ribociclib	Strong CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	No studies	*In vitro*	Monitor
Enzalutamide	Strong CYP3A4 inducer	↓ DOAC levels	No increased risk of thrombosis comparing enzalutamide + DOAC *vs* non-DOAC[38]	Human observational	Monitor
Apalutamide	Strong CYP3A4 inducer P-gp inducer	↓ DOAC levels	No increased risk of thrombosis comparing apalutamide + DOAC *vs* non-DOAC[38]	Human observational	Monitor
Abiraterone	Moderate CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	No increased risk of bleeding comparing abiraterone + DOAC *vs* non-DOAC[38]	Human observational	Likely safe
Crizotinib	Moderate CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	Crizotinib + DOAC was associated with an increased risk of bleeding in a small study[40]	Human observational	Monitor
Osimertinib Alectinib	Weak CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	Low 6-month incidence of bleeding (2% major and 2% non-major bleeding) when combined with DOACs[28]	Human observational	Likely safe
Lorlatinib	CYP3A4 inducer P-gp inducer	↓ DOAC levels	No studies	*In vitro*	Monitor
Cabozantinib	Weak CYP3A4 inhibitor P-gp inhibitor	↑ DOAC levels	No differences in major bleeding or VTE events when combined with LMWH or DOAC compared to anticoagulant alone[41]	Human observational	Likely safe
Imatinib Nilotinib Dasatinib	Moderate to strong CYP3A4 inhibitor	↑ DOAC levels	No increased risk of bleeding when combined with DOAC compared to DOAC alone[24]	Human observational	Likely safe
Dabrafenib Vemurafenib	Strong CYP3A4 inducer	↓ DOAC levels	No studies	*In vitro*	Monitor

*Recommendations were made based on literature referenced in the paper, clinical experience, and consensus among the authors in the expert-based framework. As the current evidence remains limited, we are conservative in our recommendations. In those without data in human studies, we recommend "monitor". For Enzalutamide and apalutamide, only one larger study was recently published, and given potential strong interactions noted in PK studies, we conservatively recommend "monitor" in hope for more evidence (as compared to abiraterone where potential interaction is moderate). For crizotinib, there was also only one retrospective study with unclear sample size, therefore "monitor" is recommended. A, aromatase inhibitor; CYP, cytochrome; DOAC, direct oral anticoagulant; PK, pharmacokinetic; P-gp, p-glycoprotein.

lack of in-vivo human data, review papers and expert guidance caution against concurrent use with DOACs, particularly rivaroxaban and apixaban.[29,33]

Prostate cancer

Enzalutamide and apalutamide

Prostate cancer is the most common cancer in males, with close to 50% advanced stage at the time of diagnosis.[34] Enzalutamide, apalutamide, and abiraterone are androgen-receptor pathway inhibitors (ARPI) indicated in most patients with advanced prostate cancer.[34,35] Enzalutamide and apalutamide are strong CYP3A4 inducers, while apalutamide is also a strong p-gp inducer.[36] Therefore, their concurrent use with DOACs had been cautioned against for the risk of reduced effectiveness of DOAC therapy and thereby an increased risk of thrombosis.[17-20] However, these are based on *in vitro* PK data and their clinical implications remain elusive. A *post-hoc* analysis of the SPARTAN and TITAN randomized controlled trials comparing apalutamide to placebo plus androgen deprivation therapy in prostate cancer showed no increased risk of thrombosis in those on concurrent oral anticoagulants and apalutamide compared to anticoagulants plus placebo, but the number of patients on DOACs was small (<20 in each arm of the study).[37] Recently, our group completed an analysis using Ontario and Alberta linked provincial databases to evaluate the outcomes of 1,430 patients with prostate cancer on enzalutamide or apalutamide and anticoagulant therapy.[38] We found no difference in the risk of arterial or venous thrombosis when comparing those taking concurrent enzalutamide or apalutamide plus a DOAC *vs* a non-DOAC anticoagulant (pooled HR 0.83 [95% CI 0.36-1.93]). Subgroup analyses in high-risk groups such as age ≥75, new anticoagulant start, *etc*., were consistent with the main study results.

Abiraterone

Abiraterone is another commonly used ARPI. Different from enzalutamide and apalutamide, it is a moderate CYP3A4 and p-gp inhibitor, leading to a theoretical increased risk of bleeding when used concurrently with DOAC therapy.[17-20] In the above-mentioned population study using Ontario and Alberta provincial data, 1,567 patients on combined abiraterone and anticoagulant therapy had no associated increased risk of hemorrhage in those on DOAC-abiraterone compared to non-DOAC-abiraterone combination (pooled HR 1.16 [95% CI 0.10-13.99]).[38] Although population-based database analysis have inherent limitations, including retrospective study design, potential residual confounding, possibilities of mis-coding, and more, they have the advantage of evaluating clinically relevant outcomes in a large sample size on a data deficit topic and can provide evidence for clinicians.

Lung cancer

Crizotinib

Crizotinib, a tyrosine kinase inhibitor (TKI) of ALK (anaplastic lymphoma kinase) receptor, is a targeted therapy for ALK or ROS1-positive non-small cell lung cancer. Crizotinib is a moderate inhibitor of both CYP and P-gp pathways *in vivo*,[39] and expert consensus statement cautioned against concurrent use with all DOACs.[19] A small prospective study in ambulatory cancer patients (n=108) referred for evaluation of primary thromboprophylaxis revealed that the combination of crizotinib and rivaroxaban or apixaban was associated with an increased risk of bleeding.[40] However, the number of patients on these combinations that the results were derived from, the type and severity of bleeding, as well as the magnitude of the risk were not reported, indicating significant limitation of the results. No other studies are found reporting on the outcomes of concurrent use of crizotinib and DOACs.

Osimertinib, alectinib, lorlatinib

Other ALK and EGFR (epidermal growth factor receptor) inhibitors commonly used in lung cancer include osimertinib (a weak CYP3A4 inhibitor), alectinib (a p-gp inhibitor), and lorlatinib (a CYP3A4 and p-gp inducer). In the TacDOAC study, 42 patients on concurrent DOAC and either osimertinib or alectinib were included, with low incidences of bleeding events at 6 months (major and non-major bleeding 2% each) and no recurrent VTE.[30] No other human studies are found reporting concurrent use of anticoagulants and osimertinib, alectinib, or lorlatinib. Therefore, there remains a significant knowledge gap in the combination use of DOACs and these anticancer therapies.

Kidney cancer

Cabozantinib

Cabozantinib is a small molecule TKI blocking multiple proteins involved in cancer cell growth and blood vessel formation, and is approved for the treatment of renal and liver cancers.[41] Cabozantinib is reported to be a weak CYP3A4 inhibitor and a p-gp inhibitor, leading to the concern of increasing DOAC concentration during concurrent use. In addition, being a VEGFR (vascular endothelial growth factor receptor) inhibitor, PD interactions leading to potentiation of anticoagulant levels (mainly with LMWH) have been reported.[15] A retrospective multicenter study reported outcomes in 298 patients with advanced renal cell carcinoma on cabozantinib with or without different anticoagulants (including warfarin, LMWH, and DOACs) found no differences in major bleeding or VTE events among all groups regardless of the presence or absence of anticoagulants or the type of anticoagulants.[41]

Chronic myelogenous leukemia (CML)

Imatinib, nilotinib, and dasatinib

BCR-ABL TKIs are the cornerstone for the treatment of CML. Among commonly used BCR-ABL TKIs, imatinib and nilotinib are reported to be moderate to strong CYP3A4 inhibitors and p-gp inhibitors (to a lesser degree, dasatinib). The previously mentioned Taiwanese database study included 33 patients on concurrent imatinib and a DOAC comparing to DOAC alone, and found no increased risk of major bleeding with the combination (RR 1.05, 95% CI 0.36-3.08).[26] In the TacDOAC study, 22 pa-

tients were receiving concurrent DOAC and imatinib or nilotinib, and there were no bleeding events (both major and non-major) nor new VTE during the 6-month follow up.[30] Similarly, another small case series followed 9 patients with CML on concomitant BCR-ABL TKIs and DOACs for a median duration of 8.5 months and reported no major bleeding or symptomatic thrombotic events.[42] Therefore, current clinical evidence has not revealed concerns of concurrent BCR-ABL TKIs and DOAC use, but large studies with more robust data are welcome.

Melanoma

Dabrafenib and vemurafenib

Dabrafenib and vemurafenib are inhibitors of BRAF used for melanoma treatment. *in vitro* studies showed that they are CYP3A4 inducers, leading to the concern of reducing DOAC levels and an increased risk of thrombosis if used concurrently with a DOAC.[17-21] However, there is very little evidence to confirm or dispute these PK findings. In the TacDOAC study, 4 patients were on concurrent DOAC and dabrafenib or vemurafenib.[30] While there were no bleeding events, one out of these 4 patients developed a recurrent VTE within 6 months. More patient data are certainly needed.

Conclusions

In this narrative review, we summarized the current evidence regarding DDIs between DOAC and anticancer therapy, which continues to be sparse and dominated by *in vitro* PK or animal studies. Human studies including clinically relevant outcomes are scarce, resulting in empiric cautions and dosing recommendations that are poorly evidence-based. Although this is not a systematic review which may introduce bias, we searched PubMed database for relevant keywords to ensure inclusion of pertinent studies. We aim to highlight the current knowledge gap in this field as well. Available clinical studies thus far, have largely disputed the concerns of clinically relevant outcomes when combining anticancer therapies and DOACs with potential PK interactions, indicating safe co-administration. Studies evaluating relevant clinical outcomes, preferably in large populations, are desperately needed to allow more accurate assessment of the impact and guidance for safe use of anticoagulation.

References

1. Raskob GE, van Es N, Verhamme P, et al. Edoxaban for the treatment of cancer-associated venous thromboembolism. N Engl J Med 2018;378:615-24.
2. Young AM, Marshall A, Thirlwall J, et al. Comparison of an oral factor Xa Inhibitor with low molecular weight heparin in patients with cancer with venous thromboembolism: results of a randomized trial (SELECT-D). J Clin Oncol 2018;36:2017-23.
3. Agnelli G, Becattini C, Meyer G, et al. Apixaban for the treatment of venous thromboembolism associated with cancer. N Engl J Med 2020;382:1599-607.
4. Frere C, Farge D, Schrag D, et al. Direct oral anticoagulant versus low molecular weight heparin for the treatment of cancer-associated venous thromboembolism: 2022 updated systematic review and meta-analysis of randomized controlled trials. J Hematol Oncol 2022;15:69.
5. Carrier M, Abou-Nassar K, Mallick R, et al. Apixaban to prevent venous thromboembolism in patients with cancer. N Engl J Med 2019;380:711-9.
6. Khorana AA, Soff GA, Kakkar AK, et al. Rivaroxaban for thromboprophylaxis in high-risk ambulatory patients with cancer. N Engl J Med 2019;380:720-8.
7. Delluc A, Wang TF, Yap ES, et al. Anticoagulation of cancer patients with non-valvular atrial fibrillation receiving chemotherapy: guidance from the SSC of the ISTH. J Thromb Haemost 2019;17:1247-52.
8. Khorana AA, Noble S, Lee AYY, et al. Role of direct oral anticoagulants in the treatment of cancer-associated venous thromboembolism: guidance from the SSC of the ISTH. J Thromb Haemost 2018;16:1891-4.
9. Key NS, Khorana AA, Kuderer NM, et al. Venous thromboembolism prophylaxis and treatment in patients with cancer: ASCO guideline update. J Clin Oncol 2023;41:3063-71.
10. Lyman GH, Carrier M, Ay C, et al. American Society of Hematology 2021 guidelines for management of venous thromboembolism: prevention and treatment in patients with cancer. Blood Adv 2021;5927-74.
11. Farge D, Frere C, Connors JM, et al. 2019 international clinical practice guidelines for the treatment and prophylaxis of venous thromboembolism in patients with cancer. Lancet Oncol 2019;20e566-e81.
12. Lees J, Chan A. Polypharmacy in elderly patients with cancer: clinical implications and management. Lancet Oncol 2011;12:1249-57.
13. Daiichi Sankyo, Inc. Savaysa (edoxaban) prescribing information. Basking Ridge, Daiichi Sankyo, Inc.; 2024.
14. Wang ML, Rule S, Martin P, et al. Targeting BTK with ibrutinib in relapsed or refractory mantle-cell lymphoma. N Engl J Med 2013;369:507-16.
15. Patel SH, George TL, Wang TF, et al. Increased bleeding risk associated with concurrent vascular endothelial growth factor receptor tyrosine kinase inhibitors and low-molecular-weight heparin. Cancer 2021;127:938-45.
16. Vrotniakaite-Bajerciene K, Tsang C, Wang TF, Carrier M. Drug-drug interactions in anticoagulant therapy: a focus on oncology patients. Expert Rev Clin Pharmacol 2025;18:847-59.
17. Kraaijpoel N, Carrier M. How I treat cancer-associated venous thromboembolism. Blood 2019;133:291-8.
18. Short NJ, Connors JM. New oral anticoagulants and the cancer patient. Oncologist 2014;19:82-93.
19. Musgrave KM, Power K, Laffan M, et al. Practical treatment guidance for cancer-associated thrombosis - Managing the challenging patient: A consensus statement. Crit Rev Oncol Hematol 2022;171:103599.
20. Bellesoeur A, Thomas-Schoemann A, Allard M, et al. Pharmacokinetic variability of anticoagulants in patients with cancer-associated thrombosis: Clinical consequences. Crit Rev Oncol Hematol 2018;129:102-12.
21. Bolek H, Urun Y. Cancer-associated thrombosis and drug-drug interactions of antithrombotic and antineoplastic agents. Cancer 2023;129:3216-29.

22. Verso M, Munoz A, Bauersachs R, et al. Effects of concomitant administration of anticancer agents and apixaban or dalteparin on recurrence and bleeding in patients with cancer-associated venous thromboembolism. Eur J Cancer 2021;148:371-81.
23. Wang TF, Hill M, Mallick R, et al. The prevalence of relevant drug-drug interactions and associated clinical outcomes in patients with cancer-associated thrombosis on concurrent anticoagulation and anticancer or supportive care therapies. Thromb Res 2023;231:128-34.
24. Anderson WF, Katki HA, Rosenberg PS. Incidence of breast cancer in the United States: current and future trends. J Natl Cancer Inst 2011;103:1397-402.
25. Bekaii-Saab TS, Perloff MD, Weemhoff JL, et al. Interactions of tamoxifen, N-desmethyltamoxifen and 4-hydroxytamoxifen with P-glycoprotein and CYP3A. Biopharm Drug Dispos 2004;25:283-9.
26. Wang CL, Wu VC, Tu HT, et al. Risk of major bleeding associated with concomitant use of anticancer drugs and direct oral anticoagulant in patients with cancer and atrial fibrillation. J Thromb Thrombolysis 2022;53633-45.
27. Wang TF, Clarke AE, Awan AA, et al. Hemorrhage risk among patients with breast cancer receiving concurrent direct oral anticoagulants with tamoxifen vs aromatase inhibitors. JAMA Netw Open 2022;5:e2219128.
28. Bosch F, Mulder F, Franken L, et al. Effect of the P-glycoprotein inhibitor tamoxifen on edoxaban plasma levels in women with breast cancer. Thromb Res 2023;228:46-53.
29. Beavers CJ, Rodgers JE, Bagnola AJ, et al. Cardio-oncology drug interactions: a scientific statement from the American Heart Association. Circulation 2022;145:e811-e38.
30. Wang TF, Baumann Kreuziger L, Leader A, et al. Characteristics and outcomes of patients on concurrent direct oral anticoagulants and targeted anticancer therapies-TacDOAC registry: Communication from the ISTH SSC Subcommittee on Hemostasis and Malignancy. J Thromb Haemost 2021;19:2068-81.
31. Samant TS, Huth F, Umehara K, et al. Ribociclib drug-drug interactions: clinical evaluations and physiologically-based pharmacokinetic modeling to guide drug labeling. Clin Pharmacol Ther 2020;108:575-85.
32. Liu Z, Du W, Wang Q, et al. In vivo pharmacokinetic interactions of ribociclib with rivaroxaban and apixaban in rats: implications for increased drug exposure and dose adjustments. Front Pharmacol 2025;16:1530806.
33. Sahin TK, Kavgaci G, Guven DC, Aksoy S. Drug-drug interactions and special considerations in breast cancer patients treated with CDK4/6 inhibitors: A comprehensive review. Cancer Treat Rev 2025;137:102956.
34. Leigh J, Qureshi D, Sucha E, et al. A population-based study of factors associated with systemic treatment in advanced prostate cancer decedents. Cancer Med 2023;12:5569-79.
35. National Comprehensive Cancer Network. Clinical Practice Guidelines in Oncology, Prostate Cancer, version 2.2020. Plymouth Meeting, National Comprehensive Cancer Networ; 2020.
36. Boujonnier F, Lemaitre F, Scailteux LM. Pharmacokinetic interactions between abiraterone, apalutamide, darolutamide or enzalutamide and antithrombotic drugs: prediction of clinical events and review of pharmacological information. Cardiovasc Drugs Ther 2024;38:757-67.
37. Potdar R, Gartrell BA, Given R, et al. Concomitant use of oral anticoagulants in patients with advanced prostate cancer receiving apalutamide: A post-hoc analysis of TITAN and SPARTAN studies. Am J Cancer Res 2022;12:445-50.
38. Wang TF, Clarke A, Rath M, et al. Risks of thrombosis and hemorrhage in concurrent use of anticoagulants and potential interacting prostate cancer agents. Cancer 2026;132:e70266.
39. Lafaie L, Hodin S, Saib S, et al. Tyrosine kinase inhibitors and direct oral anticoagulants: In vitro evaluation of drug-drug interaction mediated by P-glycoprotein. Fundam Clin Pharmacol 2022;36:860-8.
40. Ng HK, Rogala BG, Ades S, et al. Prospective evaluation of drug-drug interactions in ambulatory cancer patients initiated on prophylactic anticoagulation. J Oncol Pharm Pract 2020;26:1637-42.
41. Shayeb AM, Urman D, Dizman N, et al. Evaluation of cabozantinib in combination with diret oral anticoagulants or low molecualr weight heparin in renal cell carcinoma. J Clin Oncol 2021;39:291.
42. Serrao A, Scalzulli E, Fiori L, et al. Concomitant Administration of direct oral anticoagulants in chronic phase chronic myeloid leukemia patients treated with tyrosine kinase inhibitors. Clin Drug Investig 2020;40:1177-81.