Mortality following major gastrointestinal bleeding among patients receiving direct oral anticoagulants

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ABSTRACT

Direct oral anticoagulants (DOACs) are increasingly used for the treatment of cancer-associated venous thromboembolism (CAT). However, concerns remain regarding bleeding complications, particularly gastrointestinal (GI) bleeding, and their associated morbidity and mortality in patients with cancer. This narrative review summarizes available evidence on the epidemiology, predictors, mortality, and management of DOAC-associated major GI bleeding in this high-risk population. Across randomized controlled trials, major bleeding rates with DOACs range from 4 to 7%, with GI bleeding accounting for much of the observed excess bleeding, compared with low molecular weight heparin (LMWH), particularly among patients with unresected upper GI cancers. Observational studies in the cancer population confirm that GI bleeding is the predominant site of major bleeding complications, and it is associated with substantial 30-day mortality rates of 10-20%. Evidence to guide anticoagulation resumption after GI bleeding in patients with cancer is limited. Available data suggest that resumption of anticoagulation reduces thromboembolic events and all-cause mortality at the cost of increased recurrent bleeding risk, with early resumption associated with the highest rates of rebleeding events. In conclusion, GI bleeding is a frequent and clinically important complication of DOAC therapy in patients with cancer, highlighting the importance of individualized, multidisciplinary, and patient-centered management strategies.

Key words: neoplasms; venous thrombosis, venous thromboembolism, hemorrhage; direct oral anticoagulants; heparin, low molecular weight.

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Introduction

Over the past few decades, the landscape of anticoagulation therapy in cancer has evolved dramatically.[1] The early 2000s marked a pivotal shift from vitamin K antagonists (VKA) to low molecular weight heparin (LMWH), driven by multiple randomized controlled trials (RCTs) demonstrating LMWH's superior efficacy in patients with cancer-associated venous thromboembolism (CAT).[2] This transition led to a steady decline in the 30-day hazard ratio (HR) for recurrent venous thromboembolism (VTE) and major bleeding.[1] Approximately 20 years later, in 2020, yet another major transition occurred, this time from LMWH to direct oral anticoagulants (DOACs). Six RCTs compared LMWH to various DOACs, with meta-analyses of these data suggesting that DOACs may be more effective than LMWH (risk difference; RR) 0.67 (95% confidence intervals (CI): 0.52-0.85). However, DOACs are potentially associated with higher bleeding risks: major bleeding (RR: 1.17 - 95%CI: 0.82-1.67) and clinically relevant non-major bleeding (CRNMB) (RR 1.66 - 95% CI: 1.31-2.09).[3] Over the past few years, the convenience of oral administration and favorable efficacy data led to a growing use of DOACs. Nonetheless, observational data suggest that these bleeding risks may be greater than those reported in trials, since only about half of patients encountered in everyday clinical practice would have met the stringent eligibility criteria used in these RCTs.[4] Furthermore, patients with cancer often present with comorbidities such as renal or hepatic impairment and thrombocytopenia, potentially amplifying the underlying bleeding risk associated with anticoagulant therapy.

These findings call for a deeper understanding of DOAC

safety in routine cancer care, especially in patients with gastrointestinal (GI) and genitourinary (GU) malignancies, where bleeding may be associated with increased morbidity and mortality. In this narrative review, we will focus on the morbidity, mortality, and management following major GI bleeding among patients with cancer who receive DOACs.

Prevalence and predictors of major bleeding in patients with cancer-associated venous thromboembolism

The prevalence and predictors of major bleeding, CRNMB, and clinically relevant bleeding (the composite of major bleeding and CRNMB) in patients with CAT have been characterized in several meta-analyses and cohort studies. In a recent meta-analysis including over 96,000 patients across 33 studies, the pooled incidence of clinically relevant bleeding during anticoagulation ranged between 3% and 10% within the first 6-12 months, depending on cancer type and treatment intensity.[5] Across studies, several predictors of clinically relevant bleeding were identified. High-certainty evidence indicated that a prior history of bleeding, poor performance status (ECOG, Eastern Cooperative Oncology Group, ≥2), and advanced or metastatic cancer are important risk factors. Cancer site also strongly influenced bleeding risk, with brain, GI, GU, and prostate malignancies associated with significantly higher rates of anticoagulant-related bleeding.[5] Additional factors with moderate evidence included anemia, liver dysfunction, and ongoing cancer therapy, particularly chemotherapy. Interestingly, bleeding risks appear higher in patients with upper GI cancers and in those treated with DOACs, reflecting tumor-related mucosal fragility and drug exposure.[5] Overall, clinically relevant bleeding in patients with CAT receiving anticoagulation is frequent, occurring in approximately one in ten patients within a year, with the highest risk observed among those with advanced disease, prior bleeding, or GI and GU cancers. These findings highlight the need for individualized anticoagulation strategies and the incorporation of validated predictors into bleeding-risk models to optimize the balance between recurrent thrombosis prevention and safety in this high-risk population.

Bleeding complications are associated with significant morbidity and mortality in patients with cancer-associated venous thromboembolism

Major bleeding complications are associated with significant morbidity and a sustained decline in quality of life (QoL) among patients receiving anticoagulation therapy.[6] Although few studies have quantified this relationship directly, available evidence indicates that major GI bleeds can significantly impair QoL for up to nine months post-event, reflecting the enduring physical, psychological, and functional consequences of these episodes.[7] Beyond their immediate clinical impact, bleeding events frequently lead to hospitalizations, transfusions, and interruption or discontinuation of anticoagulation, thereby increasing the risk of subsequent thrombotic events and overall clinical deterioration.

In patients with cancer, the burden of bleeding is particularly severe. Compared with non-cancer populations, patients with active cancer experience more frequent and clinically significant bleeding events, with approximately 1% resulting in fatal bleeding compared to 0.3% among patient without cancer.[8] Those with advanced age (≥75 years), GI or GU lesions, or concurrent mucosal disease are especially prone to life-threatening bleeding complications during anticoagulation with either LMWH or DOACs. Across studies, the case-fatality rate of oral anticoagulant-related bleeding in patients with cancer ranges from 8% to 13% which is consistent with a pooled global estimate of 8.9% (95% CI: 3.5-21.1%).[9] These rates highlight the serious and often life-threatening nature of anticoagulant-related bleeding events. Overall, major bleeding remains a leading cause of morbidity and mortality during anticoagulation, with outcomes particularly poor among patients with cancer. The high mortality rates highlight the need for careful monitoring, early recognition, and individualized management strategies to mitigate bleeding-related complications in this high-risk population.

Direct oral anticoagulant-related gastrointestinal bleeding episodes are common in patients with cancer

RCTs evaluating DOACs for CAT have consistently demonstrated similar or modestly increased rates of major bleeding compared with LMWH, with GI bleeding emerging as the principal driver of between-trial heterogeneity. Across the pivotal RCTs, the absolute rates of major bleeding at 6 months in DOAC-treated patients ranged from approximately 4% to 7%, compared with 3% to 4% with dalteparin (Table 1). In the HOKUSAI VTE Cancer and SELECT-D trials, this translated into higher major bleeding rates with edoxaban and rivaroxaban, respectively, whereas CARAVAGGIO demonstrated comparable major bleeding with apixaban and dalteparin (3.8% *vs* 4.0%).[10-12] Importantly, severe or fatal bleeding events were uncommon across all trials, indicating that the higher risk of bleeding with DOACs is largely from non-fatal but clinically relevant bleeding episodes.

The higher bleeding rates observed in DOAC trials seems to be largely attributable to GI bleeding. In the HOKUSAI VTE Cancer trial, major GI bleeding occurred in approximately 4% of edoxaban-treated patients compared with approximately only 1% of those receiving dalteparin, accounting for nearly all the additional major bleeding events.[13] This risk was particularly elevated in patients with active GI cancers, in whom the cumulative incidence of major bleeding reached 13% with edoxaban compared to 4% with dalteparin, predominantly from upper GI sources.[13] In contrast, the CARAVAGGIO trial did not demonstrate an excess risk of GI bleeding with apixaban, even among patients with GI cancers, who comprised approximately one-third of the study population.[12] Absolute rates of major GI bleeding were low (approximately 2%) and comparable between apixaban and dalteparin, with no reported increase in upper or lower GI bleeding and no major bleeding events observed in patients with resected GI tumors.[14] Interestingly, patients with GI cancers receiving apixaban seem to have more CRNMB events compared to those receiving dalteparin (37% *vs* 22%).[14] However, these bleeding events occurred mainly in the GU and upper

Table 1. Rates of major bleeding and gastrointestinal bleeding across direct oral anticoagulant trials

Study (year)	DOAC	Design	Comparator	Major bleeding (%)	GI bleeding
HOKUSAI VTE Cancer (2018)[10]	Edoxaban	RCT	Dalteparin	6.9% *vs* 4.0% (12 mo)	3 to 4% major GI bleeding; excess with edoxaban
SELECT-D (2018)[11]	Rivaroxaban	RCT (pilot)	Dalteparin	6% *vs* 4% (6 mo)	Excess GI bleeding with rivaroxaban in esophageal or gastroesophageal cancers
ADAM VTE (2020)[22]	Apixaban	RCT	Dalteparin	0% *vs* 1.4% (6 mo)	Very low GI bleeding rates with both anticoagulants
CARAV	Apixaban	RCT	Dalteparin	3.8% *vs* 4.0% (6 mo)	No excess major GI bleeding between the two anticoagulants
CASTA-DIVA (2022)[23]	Rivaroxaban	RCT	Dalteparin	1.4% *vs* 3.7% (3 mo)	No excess major bleeding between the two anticoagulants
CANVAS (2023)[24]	Apixaban/ Rivaroxaban	Pragmatic RCT	LMWH	5.2% *vs* 5.6% (6 mo)	No clear apixaban–rivaroxaban difference

GI, gastro-intestinal; LMWH, low-molecular weight heparin; mo, month; RCT, randomized controlled trial.

respiratory tracts. Whether the absence of higher major GI bleeding in CARAVAGGIO reflects more stringent patient selection, with fewer patients with tumor-related mucosal fragility, or a more favorable safety profile of apixaban relative to other factor Xa inhibitors remains uncertain.

These RCTs and *post-hoc* data suggest that the risks of GI bleeding with DOACs in CAT are not uniform, but rather potentially reflect complex interactions between drug-specific pharmacologic properties, the presence of tumor-related mucosal fragility, and trial inclusion criteria. However, the structure of RCTs limits the ability to evaluate how individualized risk assessment and treatment selection may mitigate bleeding in routine clinical care. In a large prospective institutional cohort from Memorial Sloan Kettering Cancer Center (MSKCC), rivaroxaban was associated with a relatively low absolute risk of GI bleeding in patients with CAT when used within a risk-adapted clinical pathway that discouraged DOAC use in patients with tumor-related mucosal fragility or active luminal GI lesions.[15] Among 1,072 patients treated with rivaroxaban, the 6-month cumulative incidence of major bleeding was 2.2% (95% CI: 1.1-3.2%), with GI bleeding accounting for 73% of the major bleeding episodes, corresponding to an absolute major GI bleeding risk of approximately 1.5%.[15] Importantly, most GI bleeding events occurred in the presence of identifiable anatomic lesions (e.g., active tumor involvement, ulcers, *etc.*), and patients with GI cancers were not disproportionately represented among major bleeding events, with only one major bleeding episode reported each among 100 colorectal and 40 gastroesophageal cancer patients.[15] These findings are consistent with recent data from the COMMAND registry, which specifically examined DOAC-associated bleeding in patients with GI cancer.[16] In this multicenter Japanese registry including 1,149 patients with CAT treated with DOACs, the cumulative 5-year incidence of major bleeding was higher in patients with upper GI cancers (22.4%) compared with those with lower GI cancers (15.4%) and non-GI cancers (11.6%), with GI bleeding accounting for over half of all major bleeding events.[16] After multivariable adjustment, upper GI cancer, but not lower GI cancer, remained independently associated with major bleeding. Furthermore, bleeding events seem to occur early after CAT diagnosis (within 90 days) and predominantly originated from the upper GI tract. Patients with non-resected upper GI tumors experienced the highest bleeding rates, whereas those with resected upper GI cancers had bleeding risks comparable to non-GI cancers, highlighting the importance of active luminal disease or tumor-related mucosal fragility rather than GI cancer location *per se*.[16] Therefore, the MSKCC and COMMAND registries reinforce the findings observed in the RCTs and meta-analyses, demonstrating that the excess GI bleeding associated with DOACs is largely driven by upper GI tumor activity and unresected luminal disease, highlighting the importance of careful patient selection when considering DOACs in this patient population.

Gastrointestinal bleeding episodes are associated with high mortality in patients with cancer

As noted above in the MSKCC and COMMAND registry, GI is consistently the dominant location of bleeding across different cancer populations.[15,16] Despite the frequency of GI bleeding, data specifically quantifying early mortality after DOAC-associated GI bleeding in patients with cancer remain relatively limited. A recent systematic review and meta-analysis of DOAC-treated patients with major GI bleeding (20 studies; 3,987 major GI bleeds, not specific to cancer) reported a pooled 30-day all-cause mortality of 8.4% (95% CI 4.9-12.5%).[17] In subgroup analyses, the 30-day all-cause mortality was 10.3 % (95 % CI 6.5-14.7%) in prospective studies (9 studies, 675 major GI bleeds) and 7.3% (95 % CI 2.2-14.4%) in retrospective studies.[17] These findings highlight that major GI bleeding is not only common but associated with significant morbidity and mortality. In patient with cancer specifically, a large cohort study of 9,326 patients with solid tumors and CAT (24% treated with a DOAC) reported a 12-month cumulative incidence of clinically significant bleeding of 9.1% (95% CI 8.4-9.7%), with GI as the most frequent site of bleeding (56.2%). Following any bleeding event, the 30-day cumulative incidence of mortality was 19.4% and remained high specifically after GI bleeding (20.9%).[18] Similarly, in the COMMAND registry, fatal bleeding occurred in 10 of 119 major bleeds (8.4% case-fatality), and among fatal bleeding events, the most frequent site was GI (70%).[16] Additionally, in

patients with hematological malignancies receiving anticoagulation for CAT, GI bleeding is not only common but is strongly associated with early mortality.[19] In a nationwide cohort, GI bleeding accounted for nearly 60% of clinically relevant bleeding events and was associated with a 30-day mortality of 10%, as well as a more than four-fold increased risk of death compared with patients without bleeding.[19] Any anticoagulant-related bleeding tripled the 12-month mortality and was associated with a substantially reduced overall survival, with these adverse outcomes observed across all anticoagulant classes (e.g., DOACs, LMWH, *etc*.). Hence, GI bleeding is the leading site of clinically relevant bleeding complications and a main driver of short-term morbidity and mortality. These observations support tailoring of anticoagulation (e.g., identifying luminal lesions, reassessing concomitant antiplatelets or nonsteroidal anti-inflammatory drugs (NSAIDs), *etc*.) for patients with CAT.

When to resume anticoagulation following a clinically relevant gastrointestinal bleeding episode?

Decisions regarding resumption of anticoagulation after GI bleeding in patients with cancer, particularly those receiving DOACs, are complex and should be individualized within a multidisciplinary, patient-centered framework. Post-bleed management should begin once hemostasis is achieved (e.g., gastric irradiation in patient with gastric cancer, *etc*.) and incorporate a structured assessment of competing risks, balancing the absolute risk and clinical consequences of recurrent thrombosis against the risk, source, and severity of recurrent bleeding, while explicitly addressing modifiable risk factors such as concomitant antiplatelet or NSAID use, anticoagulant choice and dose, drug-to-drug interactions, and untreated luminal lesions.[20] Importantly, because health-care providers and patients may differ in their tolerance for short- and long-term risks, treatment decisions should meaningfully integrate patient values and preferences through shared decision-making. This framework highlights that resumption of anticoagulation is not a binary decision, but a nuanced process informed by interdisciplinary input (e.g., hematology, gastroenterology, oncology), clinical course, and the best available evidence on thrombotic and bleeding outcomes.

The optimal timing for resumption of anticoagulation after GI bleeding remains uncertain, particularly in patients with cancer receiving DOACs, as available evidence is derived almost exclusively from observational studies with high risk of bias. In a recent systematic review and meta-analysis including 12 observational studies and 3,098 patients with anticoagulant-associated GI bleeding, resumption of oral anticoagulation was associated with a higher absolute risk of recurrent GI bleeding (13.1% *vs* 5.9%), but significantly lower risks of thromboembolism (7.6% *vs* 15.4%) and all-cause mortality (21.5% *vs* 31.6%) over follow-up.[7] Resuming anticoagulation was associated with approximately 46 fewer thromboembolic events and 155 fewer deaths per 1,000 patients, at the cost of 53 additional recurrent GI bleeding.[7] However, all included studies were judged to be at serious risk of bias, primarily due to baseline confounding, as patients selected for anticoagulation resumption were often healthier, had lower perceived bleeding risk, or higher thrombotic risk. Data on timing were heterogeneous and limited, but early resumption (i.e., within the first 7 days) seemed to be consistently associated with higher rebleeding risk, whereas delayed resumption appeared safer. In a risk-modelling analysis of patients with anticoagulant-associated upper GI bleeding, the lowest combined risk of recurrent bleeding and thromboembolism was observed when anticoagulation was resumed between 3 and 6 weeks after the index bleed.[20] However, this finding needs to be validated in clinical studies. Importantly, patients with cancer and those receiving DOACs were underrepresented in these analyses, limiting their applicability. Therefore, while available data support eventual resumption of anticoagulation in most patients after GI bleeding, the timing, particularly in cancer populations, should remain individualized, integrating bleeding source control, thrombotic risk, cancer status, and patient preferences.

Evidence to guide the optimal choice of anticoagulant after GI bleeding in patients with cancer is limited and largely extrapolated from RCTs and observational data in the pre-bleed setting. Expert consensus and observational practice patterns support a stepwise and risk-adapted approach.[7,20] This often includes initial resumption with LMWH frequently at prophylactic or intermediate dosing, followed by escalation to full therapeutic dosing once hemostasis is achieved and bleeding risk has stabilized. For patients in whom DOAC resumption is considered appropriate, strategies such as delayed reinitiation, temporary dose reduction, or selection of agents with more favorable GI bleeding profiles may be reasonable, although these approaches have not been prospectively validated. Importantly, dose reduction should not be viewed as benign or permanent. Subtherapeutic anticoagulation may expose patients to recurrent thrombosis without clearly mitigating bleeding risk, highlighting the need for ongoing reassessment. Hence, the choice of agent and dose after GI bleeding in patients with cancer should be individualized, incorporating the bleeding source and its definitive management, cancer type and activity (particularly upper *vs* lower GI involvement), anticipated duration of anticoagulation, and patient preferences.

Conclusions

GI bleeding is the most frequent and clinically impactful bleeding complication among patients with cancer receiving DOAC therapy for CAT. Accumulating evidence from RCTs and observational cohorts demonstrates that bleeding risks are strongly influenced by cancer location, the presence of unresected luminal disease or tumor-related mucosal fragility, patient comorbidity, and treatment selection. Importantly, major GI bleeding is associated with substantial short-term morbidity and mortality, highlighting that these events are not benign complications but clinically important outcomes that affect survival and quality of life. Decisions regarding anticoagulant selection and resumption after GI bleeding remain challenging and are largely informed by expert opinions and observational data. While resumption of anticoagulation appears to reduce thromboembolic events and mortality, it carries a measurable risk of recurrent bleeding. Therefore, management strategies should be individualized, multidisciplinary, and patient-centered and incorporate bleeding source control, cancer status, thrombotic risk, and patient values. Future research should prioritize prospective studies

focused on post-bleed anticoagulation strategies in patients with cancer, including optimal timing of resumption, agent selection, and dosing.

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