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REVIEW

Clear Cell Ovarian Carcinoma and Its Distinct Coagulopathy Profile: Molecular Drivers and Clinical Implications

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Abstract: Clear cell ovarian carcinoma is a rare but clinically aggressive subtype of epithelial ovarian cancer characterized by a striking tendency toward hypercoagulability. This narrative review highlights the molecular and clinical underpinnings of CCOC-associated coagulopathy, emphasizing its distinction from other ovarian cancer subtypes. Key drivers include tissue factor overexpression, pro-inflammatory cytokines such as interleukin-6, endothelial dysfunction, and tumor-derived microparticles, all of which converge to activate the coagulation cascade and increase the risk of venous thromboembolism. Comparative data reveal a higher incidence of VTE in CCOC than in serous carcinoma, underscoring the need for histology-specific risk assessment. Current prophylactic strategies rely on standard anticoagulation, but emerging trials targeting coagulation pathways and cytokine signaling show promise for more tailored approaches. Understanding this unique tumor–coagulation interplay is critical for improving early detection, guiding thromboprophylaxis, and informing future therapeutic strategies.

Keywords: clear cell ovarian carcinoma, hypercoagulability, thrombosis, tissue factor, coagulopathy

Introduction

Clear cell ovarian carcinoma (CCOC) is a unique and biologically aggressive subtype of epithelial ovarian cancer. Accounting for around 5–12% of ovarian cancers in Western populations and as much as 25% in East Asia, CCOC is frequently linked to endometriosis and often appears at an earlier stage than high-grade serous carcinoma (HGSC). Even with this earlier identification, advanced-stage CCOC shows a worse prognosis, primarily because of its natural resistance to standard platinum-based chemotherapy and its elevated recurrence rate.[1,2] A clinically important yet often overlooked aspect of CCOC is its close link to coagulopathy and venous thromboembolism (VTE). Multiple studies have indicated unusually elevated rates of VTE in patients with CCOC, with occurrences between 20% to 40%, surpassing those seen in other ovarian cancer types. Crucially, thrombotic occurrences in CCOC frequently happen regardless of tumor size, indicating a fundamental pathophysiological connection tied to tumor biology.[3] Cancer-related thrombosis is multifactorial and commonly known as Trousseau's syndrome—a high-coagulation state induced by interactions between the tumor and the host. In CCOC, this prothrombotic phenotype is especially evident and correlates with heightened expression of procoagulant proteins, raised inflammatory cytokines, and activation of endothelial cells. These characteristics indicate that CCOC is not merely a cancer with specific morphological and molecular properties but also one that uniquely impacts systemic hemostasis.[4]

Historically, CCOC has been recognized as a histologic subtype with a disproportionately high risk of thromboembolic events. Early clinical observations in the 1980s and 1990s first noted the frequent occurrence ofVTE in CCOC patients, often independent of tumor stage or surgical intervention, distinguishing it from serous or endometrioid ovarian cancers.[4] Contemporary epidemiological studies confirm this heightened risk, reporting VTE incidence rates of approximately 27–42% in CCOC, compared with 5–15% in high-grade serous carcinoma and lower rates in other epithelial subtypes. Despite this clear histology-specific risk, current thromboprophylaxis guidelines for ovarian cancer, including

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recommendations from the American Society of Clinical Oncology (ASCO) and the National Comprehensive Cancer Network (NCCN), do not differentiate based on tumor histology, generally advocating prophylactic anticoagulation for high-risk patients based on broader clinical and laboratory risk factors.[5]

A crucial mediator of cancer-related thrombosis in CCOC is tissue factor (TF), a glycoprotein that spans the membrane and starts the extrinsic coagulation cascade. CCOC cells exhibit significantly elevated TF expression compared to other ovarian cancer subtypes. This overexpression boosts thrombin production, fibrin accumulation, and platelet activation, directly aiding in thrombosis development and the formation of a tumor microenvironment conducive to metastasis and immune evasion.[5] Aside from tissue factor, the inflammatory environment of CCOC significantly contributes to the development of coagulopathy. Increased concentrations of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and vascular endothelial growth factor (VEGF) trigger a widespread inflammatory reaction that enhances clotting pathways and compromises vascular integrity. This endothelial dysfunction mediated by cytokines and the production of tumor-derived microparticles increase the likelihood of VTE and disseminated intravascular coagulation (DIC).[6]

The clinical implications of this hypercoagulable condition are extensive. Patients with CCOC face higher incidences of VTE and encounter complications that disrupt cancer care, including postponed surgeries, interruptions in chemotherapy, and elevated perioperative morbidity. Additionally, the existence of coagulopathy has been associated with unfavorable outcomes, positioning it as a possible biomarker for severe disease and reduced overall survival.[7] Considering these findings, there is an urgent necessity for a more comprehensive understanding of the coagulopathy profile in CCOC. This review seeks to investigate the molecular and cellular processes that promote the prothrombotic condition in CCOC, compare them with those in various ovarian cancer subtypes, and examine the clinical repercussions for patient care. This study highlights the distinct characteristics of CCOC within coagulation biology, emphasizing the necessity for subtype-specific approaches in risk evaluation, thromboprophylaxis, and treatment.

Despite growing recognition of the heightened thrombotic risk in ovarian cancer, current thromboprophylaxis recommendations remain largely generalized and do not account for histological subtypes. Existing guidelines, including those from the American Society of Clinical Oncology (ASCO) and the International Society on Thrombosis and Haemostasis (ISTH), advocate risk-based prophylaxis but provide no specific direction for clear cell ovarian carcinoma (CCOC), despite mounting evidence of its disproportionately high incidence of venous thromboembolism (VTE). This lack of histology-focused strategies leaves clinicians reliant on broad clinical factors such as stage, performance status, and treatment modality, potentially overlooking the intrinsic molecular drivers of hypercoagulability unique to CCOC. Addressing this gap is essential, as early identification and tailored prophylaxis could mitigate morbidity and improve survival in this distinct patient population.[5,6]

Aim

This narrative review intends to thoroughly investigate the distinctive coagulopathy characteristics linked to CCOC, emphasizing the molecular, cellular, and clinical processes that set it apart from other ovarian cancer types. The review aims to clarify how elements like tissue factor overexpression, inflammatory cytokines, and endothelial dysfunction play a role in the hypercoagulable condition seen in CCOC. The review seeks to enlighten clinicians and researchers regarding the prognostic importance of thrombosis in CCOC by examining these unique characteristics and highlighting the necessity for specific risk stratification and thromboprophylaxis approaches designed for this aggressive subtype of ovarian cancer.

Methods

This narrative review was performed through a thorough and targeted literature search to collect up-to-date information on the coagulation profile of CCOC. The analysis encompassed research articles released from 2000 to 2025, obtained from prominent biomedical databases such as PubMed, Scopus, Web of Science, and Google Scholar. Keywords employed in the search approach comprised: “clear cell ovarian carcinoma”, “coagulopathy”, “hypercoagulability”, “tissue factor”, “thrombosis”, “venous thromboembolism”, and “cancer-associated thrombosis”. Boolean operators like AND, OR, and NOT were used to narrow down the search. Both experimental and clinical research were included, such

as case series, cohort analyses, reviews, and mechanistic investigations that specifically examined the connection between CCOC and coagulation or thrombotic irregularities. Research concentrating on different histological subtypes of ovarian cancer was included solely when utilized as a comparison to emphasize what distinguishes CCOC. Articles in languages other than English, abstracts lacking full texts, and studies not connected to coagulation mechanisms were removed from the final synthesis.

Data were gathered and thematically structured to uncover patterns in pathophysiological processes, occurrence of thrombotic incidents, manifestation of procoagulant markers, and clinical consequences. The results were consolidated into main thematic categories covering epidemiology, molecular triggers of coagulopathy, clinical implications, comparative assessment with other subtypes, and new therapeutic insights. The aim was to offer a comprehensive understanding of the thrombotic phenotype of CCOC and to emphasize areas needing future research and clinical focus.

Epidemiology and Clinical Characteristics of CCOC

CCOC is a specific histological variant of epithelial ovarian cancer, representing around 5–12% of cases in Western nations and as much as 25% in East Asian demographics, especially in Japan. The significant geographic and ethnic differences in prevalence indicate a possible interaction between genetic factors and environmental or hormonal influences, like endometriosis, which is more commonly observed in East Asian women and is closely linked to the development of CCOC. CCOC usually occurs in perimenopausal women and is frequently identified at an earlier phase (FIGO stage I or II) compared to high-grade serous carcinoma (HGSC), the prevalent subtype.[1,8] Clinically, CCOC notably contrasts with other ovarian carcinomas regarding its behavior and treatment response. Even with regular early-stage diagnoses, advanced or recurrent CCOC has a bleak outlook because of its resistance to conventional platinum-based chemotherapy treatments. The tumor is frequently marked by sizable, unilateral pelvic masses, occasionally accompanied by thromboembolic incidents or paraneoplastic syndromes like hypercalcemia or thrombotic microangiopathy. Moreover, CCOC displays distinctive morphological characteristics, featuring transparent cytoplasm, hobnail cells, and a tubulocystic or papillary structure.[9–11]

A notable clinical characteristic of CCOC is its significant link to hypercoagulability. Numerous retrospective and prospective studies have indicated a specifically high occurrence of VTE in individuals with CCOC, varying between 20% and 40%. Significantly, VTE can manifest in patients with early-stage disease and limited tumor burden, suggesting that the hypercoagulable condition is influenced by inherent tumor biology instead of merely by size or metastasis. These thrombotic complications pose serious risks to life and greatly affect treatment strategies, timing of surgery, and overall survival, highlighting the clinical necessity for prompt identification and intervention.[12]

Pathophysiology of Coagulopathy in CCOC

The coagulopathy linked to CCOC is complex and fundamentally connected to the tumor's inherent biology. In contrast to other ovarian cancer subtypes, where thrombosis typically relates to disease severity or advanced progression, CCOC demonstrates a hypercoagulable condition even during early-stage disease. This indicates a key function of molecular and cellular processes specific to CCOC in causing systemic coagulation issues.[13,14] Central to this process is the heightened expression of TF, a transmembrane glycoprotein crucial for starting the extrinsic coagulation pathway. In CCOC, TF is highly expressed in tumor cells and is additionally increased in circulating microparticles derived from tumors. These microparticles enter the blood and serve as powerful procoagulant surfaces, initiating extensive thrombin production and fibrin accumulation. Elevated TF expression is not simply a consequence of cancer; it is frequently increased in reaction to low oxygen levels in the tumor microenvironment, influenced by transcription factors like hypoxia-inducible factor-1 alpha (HIF-1α). This establishes a harmful cycle in which hypoxia, coagulation, and tumor advancement enhance one another.[15,16]

Simultaneously, inflammatory signaling within the CCOC microenvironment intensifies coagulopathy. Increased concentrations of pro-inflammatory cytokines—especially interleukin-6 (IL-6), interleukin-1β (IL-1β), and TNF-α—promote the liver's synthesis of acute-phase proteins like fibrinogen and C-reactive protein. These cytokines further promote endothelial cell activation, resulting in the increased expression of adhesion molecules and strengthened interactions between leukocytes and platelets. This inflammatory process fosters a prothrombotic

environment that not only aids in VTE development but may also help tumor cells escape immune detection and spread through blood vessels.[17,18] Endothelial dysfunction intensifies the hypercoagulable condition in CCOC. Factors secreted by tumors and systemic inflammation impair endothelial integrity, leading to the exposure of subendothelial matrix proteins and the recruitment of platelets. Endothelial cells and platelets that have been activated release more microparticles abundant in TF and phosphatidylserine, enhancing the coagulation response. Moreover, certain research indicates that CCOC might disrupt the body's natural anticoagulant processes, such as decreasing thrombomodulin levels and protein C function, which could further favor thrombosis (Table 1).[19]

Unique Coagulopathy Profile of Clear Cell Ovarian Carcinoma

CCOC demonstrates a coagulopathy profile that differs significantly from other ovarian cancer types, featuring an unusually elevated risk of thrombosis that frequently appears early in the progression of the disease. This distinct profile is mainly influenced by the tumor's inherent biological traits, such as the heightened expression of TF, which acts as a strong trigger for the extrinsic coagulation cascade. Increased TF levels are present not only on the surface of CCOC tumor cells but also on circulating tumor-derived microparticles, which enhance systemic coagulation activation beyond the localized tumor setting.[13,20] Alongside TF-mediated coagulation, CCOC is linked to a significant inflammatory environment marked by increased levels of cytokines like IL-6 and TNF-α. These inflammatory substances boost coagulation by elevating liver production of clotting factors and stimulating endothelial cell activation, which leads to heightened expression of adhesion molecules and platelet clustering. Endothelial dysfunction additionally plays a role by revealing procoagulant surfaces and emitting microparticles abundant in prothrombotic elements. Collectively, these mechanisms induce a hypercoagulable condition that is more severe and enduring than that observed in other types of ovarian cancer.[21]

This unique coagulopathy not only makes patients more susceptible to VTE but also relates to more aggressive tumor behavior and worse clinical outcomes. In contrast to other ovarian cancers where thrombosis usually occurs as a result of advanced disease or treatment, the distinctive coagulation issues in CCOC are closely associated with its molecular pathology and may directly contribute to tumor progression and chemoresistance. Grasping this distinct coagulopathy profile is crucial for crafting specific prevention and treatment approaches that tackle both thrombotic risk and tumor biology in CCOC patients (Figure 1).[22]

Table 1 Summarizing the Pathophysiology of Coagulopathy in Clear Cell Ovarian Carcinoma (CCOC)

Pathophysiological Component	Description	Role in Coagulopathy
Tissue Factor (TF) Overexpression	High levels of TF expressed on tumor cells and tumor-derived microparticles	Initiates the extrinsic coagulation cascade, leading to thrombin generation and fibrin formation
Hypoxia and HIF-1α Activation	Tumor hypoxia induces HIF-1α expression, which upregulates TF and other procoagulant factors	Amplifies coagulation activation by promoting TF expression and sustaining a prothrombotic microenvironment
Pro-inflammatory Cytokines (IL-6, IL-1β, TNF-α)	Elevated systemic and local cytokine levels secreted by tumor and immune cells	Stimulate hepatic production of clotting factors, activate endothelial cells, and promote platelet adhesion
Endothelial Dysfunction	Damage and activation of endothelial cells by tumor factors and inflammation	Exposure of subendothelial matrix and release of procoagulant microparticles enhance platelet aggregation and clot formation
Microparticles	Circulating membrane vesicles derived from tumor cells, platelets, and endothelial cells	Provide a phospholipid surface enriched with TF, facilitating coagulation cascade amplification
Reduced Natural Anticoagulants	Downregulation of thrombomodulin and protein C pathways	Impairs anticoagulant mechanisms, tipping hemostatic balance toward thrombosis
Platelet Activation a		

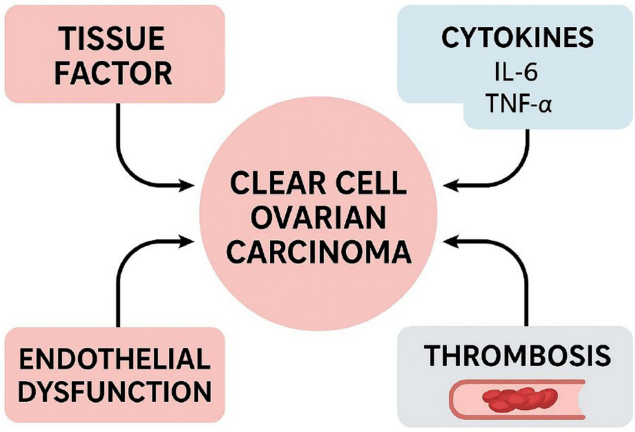

Figure 1 A Schematic Figure Illustrating the Interplay Between Tissue Factor, Cytokines, Endothelial Dysfunction, And Thrombosis in CCOC.

What Sets Clear Cell Ovarian Carcinoma Apart

CCOC stands out from other ovarian cancer types due to its distinct biological, clinical, and coagulopathic characteristics. In contrast to HGSC, the most common subtype, CCOC displays a unique molecular profile marked by significant overexpression ofTF and a pronounced inflammatory microenvironment that induce a hypercoagulable state early in disease advancement. This inherent tendency for coagulation issues arises even in early tumors with minimal spread, distinguishing CCOC as a cancer where thrombosis is a core aspect of its biology rather than just a complication of progressed disease.[23,24] Clinically, patients with CCOC exhibit a much greater occurrence of VTE, frequently showing thrombotic events that occur before or at the same time as cancer diagnosis. This differs from other ovarian cancer subtypes, in which thrombosis is often associated with advanced disease or the effects of treatment. Moreover, the thrombotic risk in CCOC stays high even with standard anticoagulation, highlighting the aggressive and enduring aspect of its coagulopathy. This distinctive interaction between tumor biology and coagulation affects patient morbidity and mortality, while also complicating treatment management, underscoring the need for histology-specific risk evaluation and therapeutic approaches.[25]

At the junction of molecular and clinical aspects, CCOC's unique coagulopathy is not just a paraneoplastic effect; it plays a crucial role in tumor development, immune evasion, and resistance to chemotherapy. The combination of hypoxia-induced transcription factor expression, inflammatory cytokine pathways, and endothelial dysfunction establishes a self-reinforcing prothrombotic milieu that supports tumor development and metastasis. This distinguishes CCOC as a model where handling coagulopathy is crucial not just to avert dangerous thrombotic incidents but also possibly to enhance oncologic outcomes via specific strategies targeting the coagulation-cancer relationship.[25]

Clinical Implications and Prognostic Significance

The distinct coagulopathy characteristics of CCOC have significant clinical implications that go beyond thrombotic occurrences alone. VTE, which consists of deep vein thrombosis and pulmonary embolism, occurs considerably more frequently in patients with CCOC compared to those with other histological subtypes. Crucially, these occurrences are not mere incidental discoveries; they often represent the initial clinical indication of cancer or may arise in the early phases of therapy. The early and unequal risk of thrombosis renders VTE an important indicator in the clinical course of CCOC, frequently warning clinicians about aggressive tumor biology underneath.[13] From a predictive perspective, the occurrence of thrombotic complications in CCOC correlates with poorer results. Research indicates that CCOC patients who experience VTE have lower overall survival rates and progression-free survival compared to those who do not have thrombotic incidents. This relationship might indicate the biological aggressiveness of tumors that are more likely to initiate systemic coagulation, or it could arise from the clinical repercussions of the thrombosis itself—including treatment delays, surgical postponements, or issues associated with anticoagulation therapy. Additionally, VTE in

CCOC patients may reoccur despite prophylactic or therapeutic anticoagulation, indicating that standard thromboprophylaxis might be inadequate in this group without customized risk assessment approaches.[26]

The hypercoagulable condition in CCOC further complicates treatment management. Anticoagulation is essential but increases the risk of bleeding, especially during cytoreductive surgery or chemotherapy when blood parameters can vary. Moreover, thrombotic complications may result in the placement of inferior vena cava filters, extended hospital stays, or adjustments in doses of chemotherapy drugs, which can all undermine treatment effectiveness. These complexities highlight the necessity of early identification of thrombotic risk, evaluation of prophylactic anticoagulation for high-risk patients, and inclusion of coagulation status in comprehensive treatment strategies. Ultimately, acknowledging coagulopathy as a key feature of CCOC not only helps in risk evaluation and prognosis but also paves the way for incorporating antithrombotic approaches into the cancer treatment of impacted patients.[7]

Comparing Coagulopathy Across Ovarian Cancer Subtypes

Though coagulopathy is an acknowledged complication in all types of ovarian cancer, CCOC exhibits a notably aggressive and unique thrombotic profile in comparison to other subtypes. HGSC, the most prevalent and researched type, is linked to VTE, although the rates and clinical presentations vary. In HGSC, thromboembolic incidents are more strongly associated with advanced disease progression, significant tumor load, and peritoneal carcinomatosis. Conversely, CCOC patients might develop VTE even during early-stage disease, indicating that thrombotic risk in CCOC is more closely associated with the tumor's inherent biology rather than tumor volume or disease severity.[13] At the molecular level, CCOC displays significantly increased expression of TF, the main initiator of the extrinsic coagulation pathway. Although TF expression is present in HGSC and other subtypes, it tends to be less prominent and not as consistently linked to thrombotic events. Moreover, CCOC is marked by increased levels of circulating procoagulant microparticles and more significant inflammatory cytokine signaling, featuring heightened IL-6 and TNF-α, which further activate coagulation pathways. In contrast to mucinous or endometrioid ovarian carcinomas, coagulopathy is infrequent and not extensively researched, and when it occurs, it is typically linked to advanced stages of the disease.[15,27]

These disparities have important clinical consequences. In CCOC, the heightened thrombotic risk demands an aggressive strategy for thromboprophylaxis, possibly starting in early-stage or perioperative situations. In contrast, thromboprophylaxis in HGSC is typically limited to advanced cases or hospital stays. Additionally, the increased occurrence of VTE in CCOC despite anticoagulation indicates that conventional risk models, primarily based on diverse histologic populations, might undervalue thrombotic risk in this cohort. Consequently, it may be essential to have histology-specific risk assessment tools and management guidelines. Acknowledging the diversity of coagulation profiles among ovarian cancer subtypes is crucial for individualized treatment and may enhance outcomes via prompt intervention and focused therapeutic approaches.[28,29]

Emerging Strategies in Management

The identification of a unique and severe coagulopathy pattern in CCOC has spurred increasing interest in creating customized approaches for risk reduction and treatment strategies. Conventional methods of thromboprophylaxis—like low-molecular-weight heparin (LMWH) given perioperatively or throughout hospitalization—might be insufficient for patients with CCOC, considering their early and ongoing thrombotic risk. Consequently, healthcare providers and scientists are investigating bolder and personalized approaches, such as prolonged anticoagulant therapy and proactive risk assessment frameworks tailored for this at-risk population.[30] A highly promising field of advancement is the application of risk prediction driven by biomarkers. Increased plasma concentrations of D-dimer, tissue factor, and pro-inflammatory cytokines like interleukin-6 (IL-6) have been suggested as potential markers for identifying patients with the greatest risk of thrombosis. These biomarkers might be incorporated into new risk evaluation models that consider tumor histology and personal coagulation profiles. These models could facilitate the earlier start of thromboprophylaxis in patients with recently diagnosed CCOC, even in outpatient or early-stage contexts, thus decreasing the occurrence of severe thromboembolic complications.[31]

In terms of therapy, focusing on the coagulation pathway itself offers a novel approach in managing CCOC. Antibodies against tissue factor, direct thrombin inhibitors, and factor Xa inhibitors are being studied for their combined

effects in preventing thrombosis and possibly hindering tumor growth, since thrombin signaling has been linked to tumor invasion and angiogenesis. Additionally, anti-inflammatory medications aimed at cytokine pathways—like IL-6 inhibitors—could potentially reduce the prothrombotic inflammatory environment that defines the CCOC microenvironment. These new treatments not only provide promise for improved management of thrombotic issues but might also function as supplements to address the chemoresistance frequently observed in CCOC. As the knowledge of the molecular interactions between coagulation and cancer biology expands, incorporating antithrombotic approaches into the typical cancer treatment for CCOC patients could enhance survival rates and quality of life.[32]

Ongoing and Emerging Clinical Strategies Targeting Coagulation in Ovarian Cancer

The recognition of hypercoagulability as a hallmark of clear cell ovarian carcinoma (CCOC) has prompted a growing interest in refining anticoagulation strategies and exploring targeted therapies that modulate the tumor–coagulation axis. Standard prophylaxis with low-molecular-weight heparin (LMWH) has been the mainstay in ovarian cancer, yet its use is largely guided by general risk assessment models rather than histology-specific evidence. Recent and ongoing clinical trials aim to bridge this gap by evaluating both conventional anticoagulants and novel agents targeting molecular drivers of thrombosis.[33] One area of active investigation involves direct oral anticoagulants (DOACs), which offer the advantages of oral administration and predictable pharmacokinetics. Trials such as Hokusai VTE Cancer and SELECT-D have included ovarian cancer cohorts, demonstrating comparable efficacy to LMWH for preventing recurrent venous thromboembolism (VTE), though subgroup analyses for CCOC remain limited. These studies underscore the potential for integrating DOACs into routine thromboprophylaxis for high-risk histologies, while highlighting the need for stratification based on molecular risk factors.[34]

Beyond conventional anticoagulation, targeted approaches are emerging to address tumor-specific procoagulant mechanisms. Tissue factor (TF) inhibitors are of particular interest, given TF overexpression in CCOC and its central role in activating the extrinsic coagulation pathway. Preclinical studies and early-phase trials with TF-blocking monoclonal antibodies or recombinant inhibitors have demonstrated reduced thrombin generation and attenuated tumor growth in xenograft models, suggesting dual benefits for thrombosis prevention and tumor progression control.[35,36] Parallel efforts focus on cytokine-targeted therapies, particularly inhibitors of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which mediate inflammation-induced hypercoagulability. Early-phase clinical trials of IL-6 blockade in solid tumors, including ovarian cancer, have reported reductions in systemic inflammatory markers and circulating procoagulant microparticles. While data specific to CCOC are limited, these interventions offer a mechanistically rational approach to mitigating the hypercoagulable state inherent to this histology.[18,37] Anti-angiogenic therapies, such as bevacizumab, have demonstrated benefits beyond tumor growth inhibition, including partial normalization of tumor vasculature and reduction of endothelial activation. Retrospective analyses suggest a potential decrease in thrombotic complications, though prospective evaluation in CCOC-specific cohorts is warranted.[38–40]

Conclusion

CCOC is a distinct subtype of ovarian cancer recognized for its aggressive coagulopathy characteristics and significant risk of venous thromboembolism, even at early stages of the disease. This hypercoagulable condition is influenced by a complex interaction of tumor-specific elements, such as increased tissue factor expression, intensified inflammatory signaling, and endothelial impairment. These mechanisms not only heighten the risk of thrombosis but also aid tumor growth and resistance to standard treatments, highlighting the crucial role of coagulation disorders in CCOC pathophysiology. In clinical practice, the higher occurrence of thrombotic complications in CCOC correlates with worse prognosis, complicating treatment approaches and requiring increased awareness. Existing risk assessment models and preventive anticoagulation protocols, mainly derived from general ovarian cancer populations, might not adequately address the needs of this high-risk group. Consequently, there is an urgent necessity for histology-focused methods that integrate molecular biomarkers to facilitate early detection and targeted treatment of coagulopathy in CCOC patients.

Disclosure

The author reports no conflicts of interest in this work.

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