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Anticoagulation in Elderly Patients with Thrombocytopenia: A Narrative Review and Practical Recommendations

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Abstract

Anticoagulation in elderly patients with thrombocytopenia poses a complex clinical dilemma, requiring a careful balance between thrombotic and bleeding risks. While thrombocytopenia is traditionally viewed as a bleeding risk factor, conditions such as immune thrombocytopenia, cancer, or autoimmune disease may paradoxically increase thrombosis risk. Anticoagulant strategies must consider platelet count thresholds, kinetics, comorbidities, and the nature of the thrombotic indication. Unfractionated heparin remains preferred in acute settings for its reversibility, while LMWHs and DOACs can be used cautiously above specific platelet levels. VKAs are generally avoided below 30 × 109/L, except in patients with mechanical valves. Adjunctive therapies (e.g., steroids, IVIg, thrombopoietin agonists) may allow for temporary therapeutic windows. Recent decision-support tools and multidisciplinary approaches enhance safety and individualization of care. This review summarizes current recommendations, emerging algorithms, and perspectives for optimizing anticoagulation in this high-risk population.

Keywords: Thrombocytopenia; anticoagulation; bleeding; thrombosis; DOACs; geriatrics; internal medicine; clinical guidelines

Introduction

Thrombocytopenia; defined as a platelet count below 150 × 10^9/L; is a common finding in geriatric practice and often results from multiple contributing factors; including infections; hematologic or autoimmune disorders; medications; or consumptive syndromes [1-3]. In older adults; this condition frequently coincides with the need for anticoagulation to prevent or treat thromboembolic diseases such as atrial fibrillation; deep vein thrombosis; or valvular heart disease [4,5]. The coexistence of thrombocytopenia and an indication for anticoagulation presents a major therapeutic challenge; particularly in frail; polymedicated elderly patients. Initiating or continuing anticoagulation in the setting of low platelet counts increases the risk of bleeding—sometimes severe—while withholding anticoagulation in the presence of a significant thrombotic risk may lead to life-threatening events [6,7]. Clinicians are thus faced with complex decision-making; often in the absence of clear; evidence-based guidance [8].

Several professional organizations; including the International Society on Thrombosis and Haemostasis (ISTH); the American Society of Hematology (ASH); and the French Society of Hematology (SFH); have proposed platelet thresholds to guide clinical decision-making based on the balance between bleeding and thrombotic risk. For example; platelet counts ≥50 × 10^9/L are generally considered acceptable for therapeutic anticoagulation; whereas levels <30 × 10^9/L typically contraindicate anticoagulation unless there is a critical thrombotic event [9-11]. However; these recommendations

are often based on limited data and lack validation from large-scale randomized trials [12]. This review aims to provide a comprehensive overview of anticoagulation management in the setting of thrombocytopenia in older adults; drawing upon current pathophysiological knowledge; common etiologies encountered in geriatric care; recent international guidelines; and emerging clinical data.

Special attention will be given to risk stratification (including the use of CHA_2DS_2-VASc and HAS-BLED scores) [13,14]; therapeutic decision thresholds; available treatment options (including transfusions; dose-modified anticoagulation; or thrombopoietin receptor agonists); and high-risk clinical contexts frequently encountered in the elderly population (e.g.; sepsis; malignancy; immune thrombocytopenia; heparin-induced thrombocytopenia) [15-17].

Materials and Methods

This article is a narrative; integrative review based on a comprehensive evaluation of the current literature; clinical guidelines; and expert consensus documents concerning anticoagulation management in elderly patients with thrombocytopenia. A systematic search was conducted in PubMed; Embase; and Cochrane Library databases for publications from January 2010 to May 2025 using the following keywords: thrombocytopenia; anticoagulation; bleeding; thrombosis; direct oral anticoagulants; elderly; frailty; and clinical guidelines. Additional sources included official recommendations from international societies such as the International Society on Thrombosis and Haemostasis (ISTH); the American Society of Hematology (ASH); and the French Society of Hematology (SFH). Abstracts and posters from recent international conferences (ASH 2023-2024; EHA 2024; SFH 2024) were also reviewed to include emerging data and ongoing clinical studies. Only English and French-language articles involving human subjects and relevant to internal medicine; hematology; geriatrics; or cardiovascular care were included. Key inclusion criteria were studies involving elderly patients (≥65 years) with coexisting indications for anticoagulation and documented thrombocytopenia (platelet count <150 $\times 10^9$/L).

Etiologies of Thrombocytopenia in Older Adults

In geriatric medicine; thrombocytopenia frequently results from a wide range of conditions that may be isolated or multifactorial. Accurate etiological identification is essential for therapeutic decision-making; particularly when anticoagulation is considered [18]. Drug-induced thrombocytopenia is among the most common causes in older adults due to polypharmacy [19]. Frequently implicated agents include heparins; with the risk of heparin-induced thrombocytopenia (HIT); antiplatelet drugs (e.g.; clopidogrel; ticlopidine) [20]; beta-lactam antibiotics (e.g.; penicillins; cephalosporins); anticonvulsants (e.g.; valproate; carbamazepine); chemotherapeutic agents (e.g.; methotrexate; hydroxyurea); and certain herbal supplements such as ginkgo or quinine-containing products [21]. Bone marrow disorders; such as myelodysplastic syndromes (MDS); acute leukemias; or aplastic anemia; are more prevalent with aging and should be suspected in the presence of pancytopenia; macrocytosis; or dysplastic features on blood smear [22,23].

Hematologic malignancies; including chronic lymphocytic leukemia (CLL) and non-Hodgkin lymphoma; may cause thrombocytopenia through bone marrow infiltration; immune-mediated platelet destruction; or splenomegaly [24,25]. Infectious causes; particularly viral infections such as HIV; hepatitis B/C; Epstein-Barr virus; or cytomegalovirus (CMV); can lead to either immune destruction or bone marrow suppression [26,27]. These are especially important in institutionalized or immunocompromised elderly patients. Primary immune thrombocytopenia (PIT); although classically described in younger adults; is increasingly recognized in older populations. It may be primary or secondary to systemic autoimmune conditions such as systemic lupus erythematosus (SLE) or antiphospholipid syndrome (APS) [28]. Nutritional deficiencies; particularly vitamin B12 and folate deficiency; may present with thrombocytopenia in the context of macrocytic anemia; commonly underdiagnosed in elderly patients with cognitive or dietary disorders [29,30].

Hypersplenism; most often associated with cirrhosis or portal hypertension; leads to splenic sequestration of platelets. It may be clinically silent or accompanied by cytopenias and splenomegaly; warranting abdominal imaging when suspected [31]. Consumptive processes such as disseminated intravascular coagulation (DIC); sepsis; or thrombotic microangiopathies (TMA) — including thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS) — must be considered in acute settings with rapid platelet decline; elevated D-dimers; and signs of multiorgan failure [32]. In many elderly patients; the etiology is multifactorial; necessitating a comprehensive clinical evaluation; including a detailed medication review; nutritional assessment; laboratory workup; and; when appropriate; bone marrow aspiration and biopsy [33,34]. Understanding the underlying cause is crucial; especially when balancing thrombotic and hemorrhagic risks during anticoagulant therapy.

Common Indications for Anticoagulation in Elderly Patients

In older adults; the main indications for anticoagulation include non-valvular atrial fibrillation; deep vein thrombosis and pulmonary embolism; mechanical heart valves; and specific conditions such as cancer-associated thrombosis; and secondary prevention of cardio-embolic stroke (Table 1) [35-38]. Indications must always be individualized; taking into account thrombotic risk; bleeding risk; renal function; and the patient's overall frailty and functional status [39,40]. Clinical tools like the CHA_2DS_2-VASc and HAS-BLED scores can help guide decision-making; although their predictive value is limited in polymorbid older adults and was not specifically validated in this population [41]. In atrial fibrillation (AF); anticoagulation is strongly recommended when the CHA_2DS_2-VASc score ≥ 2 in men or ≥ 3 in women; even in the elderly; due to the high risk of ischemic stroke; which disproportionately affects older adults [42]. However; advanced age and comorbidities significantly increase bleeding risk as reflected in HAS-BLED scores ≥ 3; which require closer monitoring but are not absolute contraindications to anticoagulation [43].

Table 1: Common Indications for Anticoagulation in Elderly Patients.

Indication	Clinical Context	Preferred Anticoagulant Options	Considerations in the Elderly
Atrial fibrillation (AF)	Non-valvular AF; CHA2DS2-VASc ≥2	DOACs (e.g.; Apixaban; Edoxaban); Warfarin if valvular	High bleeding risk; dose adjustment based on renal function; fall risk assessment
Venous thromboembolism (VTE)	Deep vein thrombosis (DVT); pulmonary embolism (PE)	DOACs; LMWH; Warfarin (in select cases)	Renal monitoring; assess bleeding vs clotting risk
Mechanical heart valve	Mechanical aortic or mitral valve prosthesis	Warfarin (target INR based on valve type)	DOACs not indicated; close INR monitoring; dietary and drug interactions
Bioprosthetic valve or valve repair	Within 3–6 months of surgery	Warfarin (initial); then consider DOAC or Aspirin	Transition to long-term strategy after stabilization
Antiphospholipid syndrome (high-risk)	Triple-positive antibody profile with thrombosis	Warfarin	DOACs may be ineffective; strict INR control needed
Left ventricular thrombus	Post-myocardial infarction or severe systolic dysfunction	Warfarin or DOAC (off-label use)	Monitor thrombus resolution; individualized therapy
Stroke or TIA prevention	Cardioembolic origin or atrial fibrillation	DOACs preferred (if non-valvular)	Consider brain imaging; high hemorrhagic risk post-stroke
Cancer-associated thrombosis	Active malignancy with VTE	LMWH; DOACs (e.g.; Apixaban; Rivaroxaban)	Drug interactions; bleeding risk; thrombocytopenia with chemotherapy
Post-surgical thromboprophylaxis	Orthopedic surgery; prolonged immobilization	LMWH; DOACs; Fondaparinux	Duration of prophylaxis; renal clearance; balance mobility vs thrombosis risk
Peripheral arterial disease	With high-risk features or recent revascularization	Aspirin ± anticoagulation (select cases)	Rare indication for full-dose anticoagulation unless co-existing AF or VTE

In patients with venous thromboembolism (VTE) or cancer-associated thrombosis; direct oral anticoagulants (DOACs) such as apixaban or edoxaban are now preferred over vitamin K antagonists or low molecular weight heparin; including in elderly populations; provided renal and hepatic function are preserved [44,55]. Nevertheless; low-dose regimens or shorter durations may be considered in patients at high bleeding risk; frail individuals; or those with limited life expectancy [46,47]. For patients with mechanical heart valves; vitamin K antagonists (e.g.; warfarin) remain the only validated option; as DOACs have been associated with increased thrombotic risk in this subgroup; particularly in the elderly [48,49]. In all cases; shared decision-making involving the patient; caregivers; and multidisciplinary teams—including geriatricians; hematologists; cardiologists; and pharmacists—is essential; especially when facing therapeutic uncertainty related to thrombocytopenia; polypharmacy; or cognitive impairment [50,51].

It may seem counterintuitive to observe an increased thrombotic risk in patients with thrombocytopenia. However; extensive experimental and clinical evidence shows that a low platelet count does not exclude thrombotic events and may even contribute to them in certain contexts [52,53]. The functional quality of the remaining platelets; their activation status; the presence of procoagulant platelet microparticles; and interactions with the endothelium and leukocytes play a central role in hypercoagulability [54,55]. Animal and human studies have demonstrated that thrombus formation is inhibited below a critical platelet threshold (approximately 10–20 × 10^9/L) [56]. However; at platelet counts between 30 and 50 × 10^9/L; the remaining platelets may be hyperactive and associated with elevated tissue factor levels; promoting a paradoxical prothrombotic state [57].

This paradoxical thrombotic risk in thrombocytopenic patients is particularly observed in several clinical conditions:

a) In PIT; despite low platelet counts; platelet-derived microparticles; elevated platelet turnover; and circulating prothrombotic antibodies contribute to a hypercoagulable state [58,59].

b) In myeloproliferative neoplasms (MPNs) such as essential thrombocythemia or polycythemia vera; platelet function is often qualitatively altered with increased P-selectin expression; platelet-leukocyte aggregates; and enhanced thromboxane generation; increasing thrombotic risk even in the presence of relative thrombocytopenia [60,61].

c) In APS; thrombocytopenia coexists with elevated arterial and venous thrombosis risk; driven by endothelial dysfunction; platelet activation; and prothrombotic autoantibodies [62].

d) In heparin-induced thrombocytopenia (HIT); immune complexes activate platelets via FcγIIa receptors; triggering a prothrombotic cascade despite thrombocytopenia; often leading to limb- or life-threatening thrombotic events [63].

e) Cancer-associated thrombocytopenia can present with paradoxical thrombosis due to tumor-derived tissue factor; NETosis; and inflammatory cytokine release; especially in gastrointestinal and pancreatic malignancies [64,65].

These situations highlight the importance of assessing platelet function; procoagulant markers; and overall coagulation status; rather than relying solely on absolute platelet count;

when evaluating thrombotic risk in elderly thrombocytopenic patients [66]. More recently; the emergence of vaccine-induced immune thrombotic thrombocytopenia (VITT) related to COVID-19 vaccination has highlighted another pathophysiological model of thrombosis associated with severe thrombocytopenia [67,68]. The mechanism; similar to HIT; involves the formation of anti-PF4 antibodies in the absence of heparin exposure. Clinically; VITT is characterized by profound thrombocytopenia (often < 30 × 10^9/L) and extensive thromboses; particularly cerebral venous sinus thrombosis (CVST) or splanchnic vein thrombosis; with significant morbidity and mortality [69]. Table 2 list the major thrombotic and hemorrhagic risk scores in elderly patients.

Table 2: Major Thrombotic and Hemorrhagic Risk Scores in Elderly Patients.

Score	Type	Key Parameters	Purpose	Clinical Utility in Geriatrics
CHA2DS2-VASc	Thrombotic	Congestive heart failure; hypertension; age ≥75 (2 points); diabetes; stroke/TIA; vascular disease; female sex	Estimate stroke risk in atrial fibrillation (AF)	Guides anticoagulation decisions for stroke prevention in elderly AF patients
HAS-BLED	Hemorrhagic	Hypertension; abnormal renal/liver function; stroke history; bleeding history; labile INR; age >65; drugs/alcohol use	Assess bleeding risk in patients on anticoagulation	Identifies modifiable bleeding risk factors to optimize safety in older adults
ATRIA	Hemorrhagic	Anemia; severe renal disease; age >75; hypertension; prior bleeding	Predict major bleeding risk	Alternative to HAS-BLED with good validation in elderly populations
HEMORR2HAGES	Hemorrhagic	Hypertension; abnormal liver/renal function; stroke; bleeding history; advanced age; anemia	Predict severe bleeding risk	Useful in complex; multimorbid elderly patients on anticoagulants
S2TOP-BLEED	Hemorrhagic	Female sex; renal impairment; heart failure; prior bleeding; hypertension	Predict bleeding risk in patients on direct oral anticoagulants (DOACs)	Specifically developed for DOAC-treated elderly patients
IMPROVE VTE Score	Thrombotic	Prior venous thromboembolism (VTE); active cancer; immobilization >7 days; thrombophilia; age ≥60	Assess risk of venous thromboembolism in hospitalized patients	Helps guide thromboprophylaxis decisions in hospitalized elderly
ORBIT Bleeding Score	Hemorrhagic	Older age (≥74); reduced hemoglobin/hematocrit; bleeding history; renal dysfunction; antiplatelet therapy	Assess bleeding risk in patients with AF	Validated in elderly; includes anemia and antiplatelet use
PADUA Prediction Score	Thrombotic	Active cancer; previous VTE; reduced mobility; thrombophilia; trauma or surgery; age ≥70	Predict risk of hospital-acquired VTE	Useful in medical elderly patients to stratify thrombosis risk

Risk Stratification and Platelet Thresholds for Anticoagulation in Older Adults

In elderly patients with thrombocytopenia requiring anticoagulation; individualized risk stratification is essential to balance the competing risks of thrombosis and bleeding. This balance is particularly delicate in geriatric populations due to frailty; multiple comorbidities; renal and hepatic dysfunction; and increased pharmacologic sensitivity [70]. Several expert societies have proposed platelet count thresholds below which anticoagulation should be modified; reduced; or withheld. These thresholds are not supported by randomized clinical trials but rely on expert consensus; observational data; and retrospective case series published by major organizations such as the International Society on Thrombosis and Haemostasis (ISTH); American Society of Hematology (ASH); and Société Française d'Hématologie (SFH) [71-73].

In general; the following pragmatic thresholds are proposed:

a) >50 × 10^9/L: Full-dose anticoagulation is considered acceptable in most patients; including the elderly [74].

b) 30–50 × 10^9/L: A dose reduction (e.g.; prophylactic or intermediate dose) may be considered; especially in patients with high thrombotic risk (e.g.; active cancer; recent thromboembolism).

c) <30 × 10^9/L: Anticoagulation is generally contraindicated; unless there is ongoing thrombosis; in which case a case-by-case approach involving platelet transfusions or TPO-receptor agonists may be used as bridging strategies [75,76].

Elderly patients with underlying hematologic conditions; such as primary immune thrombocytopenia or myelodysplastic syndromes; pose additional challenges. In these populations; platelet function and clinical bleeding history may be more informative than platelet count alone [77]. Furthermore; concomitant use of antiplatelet agents; NSAIDs; or SSRIs—common in geriatric care—can significantly amplify bleeding risk and should be considered when assessing treatment safety [78]. Some experts recommend dynamic platelet thresholds; adjusted for baseline bleeding risk; comorbidities; and the indication for anticoagulation (e.g.; atrial fibrillation vs. mechanical valve vs. acute VTE) [79]. Several institutional protocols and recent guidelines suggest temporary interruption or bridging strategies (e.g.; low-dose anticoagulation with platelet support) in severe thrombocytopenia; particularly in high-risk thrombotic settings such as recent stroke; left ventricular thrombus; or cancer-associated VTE [80,81].

Despite these proposals; robust prospective data are lacking; and decisions often depend on clinician judgment; multidisciplinary input; and patient-centered factors. Future research in elderly and thrombocytopenic populations is critically needed to validate these thresholds and to develop predictive models for hemorrhagic and thrombotic complications [82] (Table 3). These thresholds are context-dependent. In cases of high thrombotic risk (e.g.; mechanical heart valves; recent VTE; severe atrial fibrillation with CHA_2DS_2-VASc ≥ 5); some authors recommend continuing anticoagulation at reduced doses with close monitoring; even at lower platelet counts [83,84]. Conversely; in situations of high bleeding risk (e.g.; recent intracranial hemorrhage; active gastrointestinal bleeding); anticoagulation may be delayed or withheld; regardless of platelet count [85]. In hospitalized geriatric patients; tools such as the HAS-BLED score for bleeding risk and the CHA_2DS_2-VASc score for thrombotic risk in atrial fibrillation may aid in decision-making. However; their predictive value is limited in older populations with multimorbidity; polypharmacy; and functional impairment (Table 2) [86,87].

Table 3: General guidance based on platelet count.

Platelet count ($\times 10^9$/L)	Suggested anticoagulation strategy
> 50	Full-dose anticoagulation generally safe
30–50	Consider reduced-dose anticoagulation or close monitoring
20–30	Individualized decision; low-dose prophylactic anticoagulation may be considered with caution
< 20	Avoid anticoagulation except in life-threatening thrombosis

The PLASMIC score; developed to assess the probability of TTP; is also useful in the geriatric context when thrombotic microangiopathy is suspected. This tool integrates clinical and biological variables (platelet count; hemolysis; creatinine; MCV; INR; etc.) and demonstrates good performance even in older patients [88,89]. New decision-making algorithms incorporating platelet counts; consumption markers (e.g.; D-dimers; fibrinogen); and hemodynamic instability are currently under development or clinical validation; especially in intensive care and hematology-oncology settings [90]. In all cases; the dynamic nature of platelet counts should be considered. A rapidly falling platelet count or evidence of consumptive coagulopathy (e.g.; DIC; sepsis-induced coagulopathy) may necessitate urgent reassessment of anticoagulant therapy [91]. Regular re-evaluation is essential; especially in response to changes in clinical status; renal function; or concomitant medications that may interact with anticoagulant metabolism or bleeding risk [92].

Therapeutic Options for Anticoagulation in Thrombocytopenic Older Adults

Platelet Kinetics and Its Impact on Anticoagulation Management

The dynamics of platelet count changes—referred to as platelet kinetics—have emerged as a critical factor guiding anticoagulation strategies in patients with thrombocytopenia. Unlike static thresholds; the rate of platlet decline or recovery provides valuable insight into bleeding risk and therapeutic windows [93]. For instance; in PIT; patients who exhibit a progressive rise in platelet counts may be safely considered for temporary anticoagulation; especially in the presence of high thrombotic risk. This is often combined with immunomodulatory therapies (e.g.; corticosteroids; IVIG) or thrombopoietin receptor agonists (TPO-RAs) such as eltrombopag and romiplostim; which accelerate platelet recovery and enhance hemostatic stability [94]. In contrast; patients with aplastic anemia undergoing hematopoietic stem cell transplantation or intensive chemotherapy often experience prolonged and profound thrombocytopenia.

In such cases; carefully scheduled platelet transfusions are coordinated to maintain adequate levels during high-risk periods (e.g.; catheter-related thrombosis; recent pulmonary embolism). This approach allows creation of therapeutic windows where anticoagulation can be administered with minimized bleeding risk [95]. These individualized strategies are increasingly supported by data from large hematology registries and incorporated into the 2024 updated guidelines of the Société Française d'Hématologie (SFH) for managing thrombocytopenia in complex settings [96]. Integrating platelet kinetics into clinical decision-making represents a significant advancement in balancing thrombotic and hemorrhagic risks; particularly in frail elderly populations with frequent treatment fluctuations [97].

Anticoagulation Strategy in Elderly Patients with Thrombocytopenia

In elderly patients with thrombocytopenia and an indication for anticoagulation; the choice of anticoagulant must consider multiple factors: platelet count; renal and hepatic function; bleeding

history; and comorbidity burden [98]. Therapeutic options must be tailored individually and reassessed regularly as the clinical condition evolves. When appropriate; direct oral anticoagulants (DOACs) may be preferred due to predictable pharmacokinetics; shorter half-lives; and a lower risk of intracranial hemorrhage compared to vitamin K antagonists—especially in older patients without contraindications [99]. In patients with persistent thrombocytopenia (<30–50 × 10^9/L); prophylactic anticoagulation or dose-adjusted regimens may be considered if thrombotic risk is high; ideally combined with supportive strategies such as TPO-RAs or platelet transfusion protocols. Figure 1 proposes a simple clinical decision-making algorithm for anticoagulation in elderly patients with thrombocytopenia; based on current evidence; expert consensus; and clinical context.

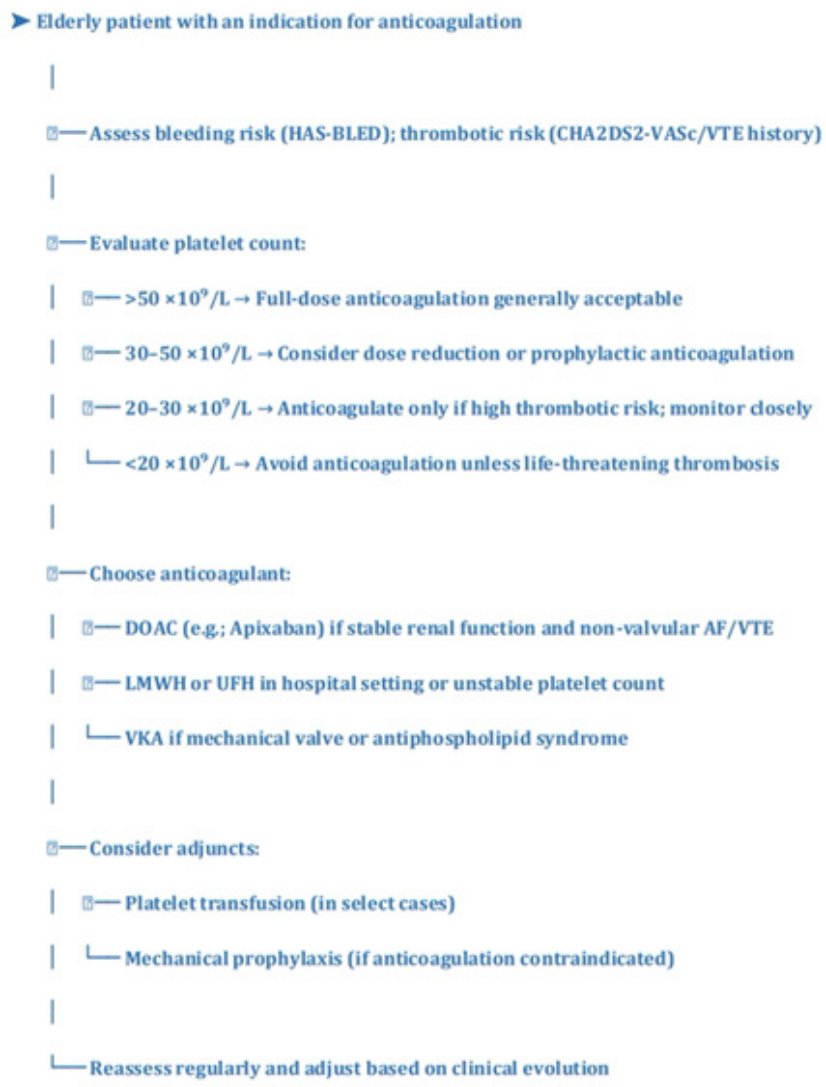

Figure 1: Anticoagulation Strategy in Elderly Patients with Thrombocytopenia.

Unfractionated Heparin (UFH)

Heparin remains a cornerstone of anticoagulant therapy; especially in acute settings. Unfractionated heparin (UFH) is often preferred in thrombocytopenic patients because it allows rapid dose adjustments; close monitoring (via activated partial thromboplastin time or anti-Xa activity); and reversal with protamine sulfate in case of bleeding [100]. UFH is particularly used in hospital and intensive care environments; especially in unstable patients or those with fluctuating platelet counts [101]. UFH's short half-life and reversibility make it suitable for critically ill patients. However; heparin-induced thrombocytopenia (HIT) remains a concern; appropriate HIT testing (PF4 antibodies; functional assays) should be performed if thrombocytopenia develops during therapy [102].

Low Molecular Weight Heparins (LMWH)

LMWHs (e.g.; enoxaparin; tinzaparin) are frequently used in hospitalized older adults due to their predictable pharmacokinetics; subcutaneous administration; and reduced need for laboratory

monitoring [103]. They are preferred in temporary or moderate thrombocytopenia (platelet count > 30 × 10^9/L) with high thrombotic risk. Dose adjustment is essential in renal impairment; which is common in the elderly [104].

In patients with platelets < 30 × 10^9/L; prophylactic doses may be used with caution or withheld; depending on the bleeding risk. The 2022 ISTH guidelines suggest:

a) Full dose LMWH above 50 × 10^9/L;

b) Half dose between 30–50 × 10^9/L;

c) Avoid LMWH below 30 × 10^9/L [105].

In cancer-associated thrombocytopenia; some teams have proposed a dose-adjusted LMWH protocol based on body weight and platelet count; enabling safe anticoagulation down to 25 × 10^9/L in select patients. This protocol was discussed at the 2024 SFH Congress [106].

Direct Oral Anticoagulants (DOACs)

DOACs (e.g.; apixaban; rivaroxaban; edoxaban) are increasingly used in elderly patients for stroke prevention and VTE treatment; offering fixed oral dosing; no need for INR monitoring; and fewer drug–food interactions [107]. Apixaban may be preferred in older adults due to a favorable bleeding profile and relative safety in moderate thrombocytopenia (> 50 × 10^9/L) [108]. However; DOACs are not recommended in severe thrombocytopenia (< 30 × 10^9/L) and in advanced renal insufficiency. Moreover; reversal agents (e.g.; andexanet alfa; idarucizumab) are not always available or affordable [109].

Vitamin K Antagonists (VKAs)

VKAs (e.g.; warfarin) require frequent INR monitoring and are subject to numerous interactions with drugs and diet. This limits their practicality in older; polymedicated patients [110]. In moderate thrombocytopenia (40–50 × 10^9/L); VKAs may be continued with close surveillance (INR; bleeding signs). Below 30 × 10^9/L; VKAs are generally discontinued due to elevated bleeding risk [111]. In patients with mechanical heart valves; temporary bridging with UFH and platelet transfusion may be considered on a case-by-case basis. VKAs remain standard for valvular atrial fibrillation and APS; but maintaining INR stability in thrombocytopenic states is often difficult [112,113].

Non-Pharmacological Alternatives

In patients with severe thrombocytopenia or contraindications to anticoagulation; mechanical thromboprophylaxis (e.g.; compression stockings; intermittent pneumatic compression) can be considered as a temporary alternative. Although not equivalent to pharmacologic anticoagulation; they are useful when bleeding risk outweighs thrombotic risk [114].

Platelet Transfusion Strategy and Adjunctive Therapies

In selected high-risk cases: platelet transfusions may be used to temporarily increase platelet counts and allow safe anticoagulation initiation. This strategy must be employed with caution in chronic thrombocytopenia due to the risk of alloimmunization and transfusion-related adverse events [115]. Adjunctive therapies: such as corticosteroids; IVIg; or TPO receptor agonists (eltrombopag; avatrombopag); are increasingly used to raise platelet counts; particularly in ITP or SLE-associated thrombocytopenia [116,117]. Recently: digital applications and clinical decision-support platforms—such as Thrombopenia Care and Platelet Risk Calc—have been developed to assist real-time decision-making. These tools integrate clinical variables; lab data; and treatment history; generating individualized recommendations. Validated in pilot studies; they show promise in standardizing practices and reducing complications [118,119] (Table 4).

Table 4: Digital Decision-Support Tools for Thrombocytopenia and Anticoagulation Management.

Tool / Application	Main Features	Clinical Input Parameters	Target Users	Validation / Status
ThrombopénieCare®	Real-time risk stratification for bleeding and thrombosis	Platelet count trend; anticoagulation indication; bleeding history	Hospital physicians; hematologists	Pilot study in tertiary care centers (France; 2023)
PlateletRiskCalc®	Automated therapeutic recommendations based on integrated score models	Platelet level; INR; renal function; comorbidities; recent thrombosis	Internal medicine; oncology	Preliminary validation at Mayo Clinic (ASH 2023)
eTMA Decision Suite	Algorithmic support for suspected thrombotic microangiopathy (TMA)	PLASMIC score; ADAMTS13; LDH; hemodynamic status	Hematology; ICU	Used in prospective registry studies
BleedSafe AITM	AI-assisted bleeding risk calculator for anticoagulated elderly patients	Age; frailty index; renal/liver function; medications	Geriatrics; cardiology	Multicenter validation planned 2024–2025
HemoGuard Clinical App	Integrates transfusion thresholds; anticoagulation dose adjustments and alerts	Platelets; anticoagulant used; bleeding signs; transfusion availability	Hematology; emergency medicine	In development phase (EHA 2024 poster)

A multidisciplinary approach is essential; particularly in complex clinical scenarios; and should involve:

a) The geriatrics; as the coordinator of overall patient care;

b) The internist or hematologist; to evaluate and manage complex causes of thrombocytopenia;

c) The cardiologist and/or pulmonologist; for the management of thromboembolic events;

d) The clinical pharmacist; to optimize dosing and prevent drug–drug interactions.

This collaborative model enables continuous adjustment of the benefit–risk balance; especially in high-risk or rapidly evolving cases.

Conclusion

Managing anticoagulation in elderly patients with thrombocytopenia remains a complex clinical challenge that requires individualized assessment and multidisciplinary coordination. The geriatric population is particularly vulnerable to both thrombotic events and hemorrhagic complications; due to age-related physiological changes; polypharmacy; comorbidities; and functional decline. While expert guidelines provide platelet thresholds and general recommendations; clinical decisions must always consider patient-specific factors such as frailty; renal and hepatic function; nutritional status; and the dynamic nature of thrombocytopenia. In the absence of robust evidence from randomized controlled trials; physicians must rely on clinical judgment; risk stratification tools; and shared decision-making with patients and families. Therapeutic strategies should balance efficacy and safety; with dose adjustments; choice of anticoagulant; or even temporary treatment suspension in severe thrombocytopenia. In selected cases; mechanical prophylaxis or platelet transfusions may offer short-term alternatives.

As the evidence base grows; future research should focus on defining optimal anticoagulation strategies in thrombocytopenic patients; particularly in the growing population of older adults with complex multimorbidity. Ultimately; safe and effective anticoagulation in thrombocytopenic elderly patients relies on personalized medicine; close clinical monitoring; and ongoing reassessment of risks and benefits. In this context; several leading academic centers have recently proposed practical algorithms to guide anticoagulation management in patients with thrombocytopenia; addressing the complex balance between thrombotic and bleeding risks. At the 2023 ASH Annual Meeting; a team from the Mayo Clinic introduced a structured three-step approach incorporating: (1) an assessment of immediate thrombotic risk using a modified risk scoring system tailored for thrombocytopenic patients; (2) close monitoring of platelet count levels and their temporal trends; and (3) evaluation of hemorrhagic comorbidities; including portal hypertension; prior bleeding history; and the presence of varices.

These components allow for dynamic adjustment of anticoagulation strategies based on evolving patient status; rather than relying solely on fixed platelet thresholds. Prospective multicenter studies such as TAPAS (Thrombocytopenia and Anticoagulation Prospective Study) and PLATE-Tx (Platelet-Guided Therapy in Thrombocytopenic Patients) are currently underway to validate the safety and efficacy of these individualized protocols in high-risk cohorts. Preliminary findings presented at the 2024 European Hematology Association (EHA) Congress suggest that this adaptive; multidisciplinary approach significantly reduces hemorrhagic complications without compromising thrombotic event prevention. By integrating platelet kinetics; risk stratification; and comprehensive clinical assessment; these algorithms represent a promising advancement towards personalized anticoagulation care in thrombocytopenic patients; especially within the elderly and frail population where clinical decisions are particularly challenging.

Conflict of Interest Statement

Professor Emmanuel Andres has received honoraria or consulting fees from pharmaceutical companies marketing the following anticoagulant agents: Apixaban; Rivaroxaban; and Dabigatran.

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