

Managing In-Hospital Anticoagulation: The Who, When and How

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ABSTRACT

Thrombosis is a significant risk for hospitalized patients, driven by factors such as inflammation, comorbidities, infections and immobility. Conversely, these patients also face heightened bleeding risks due to acute kidney injury, coagulopathy, thrombocytopenia and invasive procedures. This dual threat necessitates individualized, daily assessments to optimize anticoagulation therapy, minimizing complications while enhancing outcomes. This review examines some common clinical scenarios encountered by physicians and provides a structured, evidence-based framework to guide daily decision-making, addressing the “who”, “when” and “how” of anticoagulation therapy.

Keywords: Anticoagulation therapy; Hospitalized patients; Thromboprophylaxis; Thrombosis

INTRODUCTION

Thrombosis represents an inherent risk for all hospitalized patients, posing a significant threat of in-hospital complications and mortality. The increased susceptibility to thrombosis arises from various factors, including the underlying condition leading to admission, inflammation, pharmacological interventions, existing comorbidities, infections, catheters and intravascular devices and prolonged immobility. Furthermore, some patients require full-dose anticoagulant therapy as part of the treatment for their current condition. On the other hand, hospitalized patients also face a high risk of bleeding due to factors such as acute kidney injury, coagulopathy, thrombocytopenia and invasive procedures. Therefore, balancing the risks of bleeding and thrombosis presents an ongoing challenge, requiring daily individualized reassessment to minimize both bleeding and thrombotic risks and improve in-hospital outcomes [1]. This review explores the most common scenarios encountered by physicians and provides a structured, evidence-based summary to guide daily decision-making in addressing the “who”, “when” and “how” of anticoagulation therapy.

LITERATURE REVIEW

Navigating thrombosis and bleeding

Determining the optimal anticoagulation strategy in hospitalized patients—whether for chronic conditions, acute illnesses or thromboprophylaxis—requires a thorough and individualized approach. Deciding whether to initiate, continue, withhold or discontinue therapy carries inherent risks if the delicate balance between thrombosis and bleeding is not carefully analyzed. Thorough assessment of risks and benefits, personalized to the patient’s clinical context, is essential. Adopting a systematic, practical and efficient decision-making approach can enhance patient outcomes while minimizing complications. To guide this process, the authors propose the following key considerations.

Who: Prior to initiating or discontinuing anticoagulant therapy, comprehensively assess the patient, considering their age, weight, comorbidities, past medical history, preferences and any other risk factors for bleeding or thrombosis.

For patients with a chronic indication for anticoagulation, assess the need to continue treatment during hospitalization, considering

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the reason for admission, their stability and adjust the type and intensity of anticoagulation as needed. For patients on outpatient Direct Oral Anticoagulants (DOACs), an appropriate option may be to switch to a shorter-acting medication with an easily accessible antidote, such as Low-Molecular-Weight Heparin (LMWH), to facilitate reversal or discontinuation in the event of bleeding or the need for surgical or diagnostic interventions requiring anticoagulation discontinuation during hospitalization. Prior to discharge, the patient can be switched back to their regular oral medication.

In patients with impaired kidney function, consider adjusting

the dose of DOACs and heparins due to the increased risk of bleeding, as shown in Table 1.

When: Evaluating the indication for anticoagulation involves two important dimensions. First, determining whether the patient has an absolute indication for anticoagulation, either at thromboprophylaxis dose or full therapeutic doses. Second, is identifying the optimal timing to initiate therapy. This final aspect is particularly significant among hospitalized patients, where antithrombotic therapy is often influenced by surgical timing, concurrent medications, bleeding risk or other procedures. Even evidently minor interventions, such as lumbar

Table 1: Anticoagulation standard dosing and adjustment recommendations.

| Anticoagulant | Standard dose | Adjustment criteria |
|------------------------|---|---|
| DOACs | | |
| Apixaban | Full dose anticoagulation: 5mg twice daily. | Dose reduction to 2.5mg twice daily if ≥ 2 of the following: Age ≥ 80 years, body weight ≤ 60 kg, or serum creatinine ≥ 1.5 . |
| | Thromboprophylaxis: 2.5 mg twice daily (approved for mayor orthopedic surgery and cancer). | Data on estimated Glomerular Filtration Rate (eGFR) <25 ml/min or dialysis are only retrospective. |
| Rivaroxaban | Full dose anticoagulation: 20 mg once daily. | Dose reduction to 15 mg once daily if eGFR 15-50 ml/min. Contraindicated in eGFR <15 ml/min or dialysis. |
| Dabigatran | Full dose anticoagulation: 150 mg twice daily. | Dose reduction to 75 mg twice daily if eGFR 15-30 ml/min. Contraindicated in eGFR <15 ml/min or dialysis. |
| Edoxaban | Full dose anticoagulation: 60 mg once daily. | Dose reduction to 30 mg once daily if eGFR 15-50 ml/min. Contraindicated in eGFR <15 ml/min or dialysis. |
| Heparins | | |
| Enoxaparin | Full dose anticoagulation: 1 mg/kg every 12 h (or 1.5mg/kg once daily). | eGFR <30 ml/min: Full Dose Anticoagulation: 1mg/kg every 24 h. |
| | Thromboprophylaxis: 40 mg once daily. | eGFR <30 ml/min: Thromboprophylaxis: 20 mg once daily Body Mass Index (BMI) <18 kg/m ² , reduce prophylactic dose to 30 mg once daily. |
| | | BMI >40 kg/m ² , increase prophylactic dose to 40 mg twice daily. Monitor full dose anticoagulation with anti-Xa. BMI >50 kg/m ² , increase prophylactic dose to 60mg twice daily. |
| Fondaparinux | Full dose anticoagulation: 5 mg once daily (for weight <50 kg), 7.5 mg once daily (for weight 50-100 kg), or 10 mg once daily (for weight >100 kg). Thromboprophylaxis: 2.5 mg once daily. | Contraindicated in eGFR <30 ml/min. |
| Unfractionated Heparin | Full dose anticoagulation: 80 units/kg IV bolus followed by 18 units/kg/h infusion. Thromboprophylaxis: 5,000 units subcutaneously every 8-12 h. | No adjustment necessary. |

punctures, colonoscopy with polypectomy, transurethral prostate resection, percutaneous endoscopic gastrostomy placement or neuraxial anesthesia may carry an increased risk of bleeding, warranting careful consideration of the timing of therapy.

How: The choice of drug, dosing and monitoring are key factors in ensuring that anticoagulant therapy is delivered optimally to the patient. There are clinical scenarios in which one drug may be preferred over others. For instance, in patients with kidney impairment, LMWH may be a better option due to their predictable pharmacokinetics, whereas DOACs may not be recommended due to their dependence on renal clearance. Heparins are often preferred in acute illnesses and hospitalized patients, as they can be monitored through laboratory parameters to ensure optimal therapeutic levels. Additionally, they can be rapidly reversed in the event of complications, such as massive bleeding [2,3]. Finally, there are three clinical scenarios where Vitamin K Antagonists (VKAs) are preferred over DOACs, as clinical trials have shown that DOACs are clinically inferior in these conditions. These scenarios include antiphospholipid antibody syndrome, severe mitral stenosis and patients with mechanical heart valve prostheses, where VKAs remain the treatment of choice [4-6].

In hospital patient scenarios

Surgical patients: The most important factors to consider in the perioperative management of anticoagulation therapy, aside from the previously described "who, when and how," are the bleeding risk associated with the type of surgery and the patient's thrombotic risk profile. Moreover, it is always advisable to involve the surgical team in the decision regarding the resumption of anticoagulation after surgery, as their input provides essential insights on tissue damage, placement of drains, the adequacy of intraoperative hemostasis and the use of hemostatic agents, among other considerations.

Perioperative management of heparin should follow specific guidelines to balance thrombotic and bleeding risks. For all surgeries, warfarin should be completely suspended 5 days prior to the procedure and can be safely resumed 12 to 24 h postoperatively, depending on the risk of postoperative bleeding. For patients at high risk of thrombosis, bridging therapy with full-dose LMWH, such as enoxaparin (1 mg/kg twice daily or 1.5 mg/kg once daily), is recommended after the day warfarin is discontinued and before the day of surgery, with the last bridging dose given in the morning on the day before surgery at half the total daily dose. After surgery, low-dose LMWH (Example: Enoxaparin 40 mg daily) should be used for thromboprophylaxis for 24-72 h, with full therapeutic dosing resumed after 2-3 days, depending on postoperative conditions [7].

For patients receiving DOACs, including apixaban, rivaroxaban, dabigatran and edoxaban, these agents should be discontinued 48 h prior to surgeries with a high risk of bleeding and can be safely resumed 48 h postoperatively. For surgeries with low to moderate bleeding risk, it is recommended to suspend DOAC therapy 24 h before the procedure and resume it 12 to 24 h after surgery. A common approach is to suspend treatment the day before the procedure and resume it the morning following surgery, provided there are no surgical complications that increase the risk of bleeding [8].

Orthopedic surgery: Major orthopedic surgeries (total or partial hip or knee arthroplasty and hip fracture surgery) are associated with the highest thrombotic risk, with a baseline risk for Venous Thrombo Embolism (VTE) and Pulmonary Embolism (PE) of approximately 4% to 5% [9]. The risk is so high that these patients are always considered to require thromboprophylaxis, regardless of additional patient-related factors. For this reason, the use of risk scales to determine thromboprophylaxis eligibility, such as Caprini, is redundant and therefore not recommended [10]. Additionally, although the bleeding risk associated with these surgeries is higher compared to minor orthopedic surgeries, the risk of thrombosis exceeds that of bleeding in the majority of patients and should only be considered unacceptably high in patients with active, uncontrollable bleeding, Central Nervous System (CNS) tumors, recent traumatic brain injury, severe thrombocytopenia or major trauma. For hip arthroplasty, thromboprophylaxis should be continued for 35 days using LMWH or a DOAC, but low-risk patients may switch to aspirin after 5-10 days. For knee arthroplasty, thromboprophylaxis should be used for 14 days in ambulatory patients or up to 35 days in hospitalized patients. Finally, for hip fracture surgery, prophylaxis must last 35 days with LMWH or a DOAC. Anticoagulation may be initiated 6-12 h postoperatively for DOACs and 12-24 h for LMWH [11].

Sepsis or severe infections: Patients with sepsis are at elevated risk of thrombosis due to inflammation-driven dysregulation of the coagulation process, which leads to a pro-coagulant state. This can range from a localized venous thromboembolism to acute Disseminated Intravascular Coagulation (DIC). However, this heightened thrombotic risk is accompanied by an increased risk of bleeding. This imbalance in normal hemostasis arises from several contributing factors: Activation of the coagulation cascade mediated by cytokines and aberrant expression of tissue factor, which triggers coagulation; inhibition of fibrinolysis due to overproduction of Plasminogen Activator Inhibitor-1 (PAI-1); and dysfunction of endothelial cells and the glycocalyx, which disrupts the balance between coagulation and anticoagulation [12].

Several studies evaluating the use of full-dose anticoagulation in patients with sepsis have concluded that the only group that benefits from this therapy is those with sepsis-induced DIC [13]. For other patients, prophylactic-dose anticoagulation is recommended, but only after assessing both thrombotic and bleeding risks. In these patients, it is recommended to stratify thrombotic risk using the Padua scale and assess bleeding risk using the Bleeding Risk Score (BRS) [14].

Pulmonary Embolism (PE) and Deep Vein Thrombosis (DVT): Anticoagulant therapy is the mainstay treatment for DVT and PE. Standard practice involves a minimum duration of 3 months of anticoagulation for all patients. Extended anticoagulation is recommended indefinitely for individuals with unprovoked thrombosis or when the underlying provoking factor cannot be corrected. In the in-hospital setting, anticoagulation is typically initiated with LMWH or Un-Fractionated Heparin (UFH), especially in unstable patients. Transition to oral anticoagulation is then performed prior to discharge [15].

In specific clinical situations, the definitive diagnosis of PE may be delayed due to limited availability of imaging studies. Full-dose antithrombotic therapy should be considered for patients with a high pretest probability of PE (Wells score >6) prior to imaging confirmation or for those with intermediate or low probability when diagnostic evaluation is anticipated to be delayed by more than 4-24 h and bleeding risk is acceptable. The presence of hemoptysis in these patients should not contraindicate the initiation of anticoagulation unless there is excessive bleeding that compromises hemodynamic stability or airway safety [16].

Acute Coronary syndrome (ACS): Parenteral full-dose anticoagulation is recommended for all patients at the onset of acute coronary syndrome. In centers with a ST-Elevation Myocardial Infarction (STEMI) protocol and a short door-to-balloon time, anticoagulation is usually started intravenously in the catheterization lab. For patients with STEMI undergoing Primary Percutaneous Coronary Intervention (PPCI), UFH is the anticoagulant of choice. In Non-ST-Elevation Myocardial Infarction (NSTEMI), UFH is preferred when angiography is planned within 24 h of symptom onset, or if angiography is anticipated to occur beyond 24 h, fondaparinux (Arixtra) is recommended. Enoxaparin can be used as a second-line option in either scenario. Importantly, fondaparinux is contraindicated among patients with STEMI.

In the absence of other indications for anticoagulation, such as atrial fibrillation or intracavitary thrombus, anticoagulation administered during PPCI is discontinued upon completion of the procedure, followed by dual antiplatelet therapy. In patients managed conservatively after acute coronary syndrome, anticoagulation should be continued until hospital discharge or for a maximum of 8 days [17].

Atrial Fibrillation (AF): AF is a common condition with increasing prevalence as life expectancy rises. Many patients diagnosed with atrial fibrillation are already on anticoagulation therapy at the time of hospital admission. Additionally, the physiological stress and medications used in the hospital can lower the arrhythmic threshold, making it common for patients to develop new-onset atrial fibrillation during hospitalization for another condition.

Full-dose anticoagulation is recommended for all patients with atrial fibrillation, high thromboembolic risk (CHA₂DS₂-VASc score ≥ 2 for men, ≥ 3 for women) and a non-prohibitive bleeding risk. The current treatment of choice is DOACs, as they are associated with a lower risk of bleeding compared to warfarin. However, in patients with mechanical heart valves or moderate-to-severe mitral stenosis, VKA are preferred, as they are superior to DOACs in preventing thrombotic events [18].

In the hospital setting, subcutaneous heparin is preferred due to its shorter half-life, the ability to reverse its effects and the flexibility to adjust the dosage based on surgical procedures or bleeding events. This approach is suitable for patients who are using DOACs outpatient or for those who develop atrial fibrillation during hospitalization.

Thrombocytopenia: Thrombocytopenia increases the risk of bleeding but does not provide protection against VTE. Decision-

making in this context can be challenging, as there is no straightforward formula to determine whether the risk of bleeding outweighs the risk of thrombosis. Generally, bleeding risk becomes significant with platelet counts below 50,000/ μ L and the likelihood of severe spontaneous bleeding increases markedly with counts below 10,000/ μ L. The safety of primary VTE thromboprophylaxis in patients with thrombocytopenia has been well studied in cancer populations. When indicated, primary thromboprophylaxis is generally considered safe with platelet counts above 50,000/ μ L. Risk adjustment based on platelet levels can guide therapy, with modifications made as platelet counts decrease [19].

The recommended anticoagulation strategy for patients with thrombocytopenia is as follows: Administer full-dose enoxaparin for platelet counts >50,000/ μ L, adjust to half-dose enoxaparin for platelet counts between 25,000 and 50,000/ μ L and withhold anticoagulation when platelet counts fall below 25,000/ μ L [20].

Nephrotic syndrome: Nephrotic syndrome is associated with an increased risk of venous and arterial thromboembolism, which can be as high as 40%. Thromboprophylaxis is recommended for select patients based on the type of nephropathy, serum albumin levels and bleeding risk assessed by the HAS-BLED score. In membranous nephropathy, prophylaxis is advised when the HAS-BLED score is ≤ 1 and serum albumin is <3 g/dL, or when the HAS-BLED score is 2 and albumin levels are <2.5 g/dL, or <3 g/dL with an additional thrombotic risk factor. For non-membranous nephropathy, thromboprophylaxis is indicated with a HAS-BLED score ≤ 1 and albumin <2.5 g/dL, or a HAS-BLED score of 2 and albumin <2 g/dL, or <2.5 g/dL with an additional risk factor. Prophylaxis is not recommended in patients with a HAS-BLED score ≥ 3 due to the high bleeding risk. LMWH at 40 mg/day or warfarin with a target Indian Rupee (INR) of 2.0-3.0 are considered safe options, though evidence on the optimal agent remains limited. Additionally, heparin resistance may occur in nephrotic syndrome due to urinary loss of antithrombin III, which can reduce the effectiveness of heparin-based therapies [21].

Gastro-Intestinal (GI) bleeding: In patients with active GI bleeding, anticoagulation should be withheld immediately. In hemodynamically unstable patients, reversal agents such as Idarucizumab or Andexanet or prothrombin complex concentrate, should be used to manage the bleeding. The decision to resume anticoagulation after GI bleeding poses a clinical challenge. Evidence supports restarting anticoagulation as early as seven days after discharge or the initial bleeding episode, as this timing is associated with improved survival outcomes in patients at thrombotic risk. For those with a high thrombotic risk, anticoagulation may be resumed previously, ideally within three days, often with a heparin-bridging approach. Careful follow-up is critical to detect and address any recurrence of GI bleeding [22].

Stroke: There is no consensus on the optimal timing of anticoagulation initiation or resumption after ischemic stroke or intracranial hemorrhage. Clinical decision-making requires a careful balance between the risk of recurrent ischemic events, including thrombus formation or embolic stroke and the potential for hemorrhagic transformation or recurrent bleeding. Individualized assessment, considering the etiology of the stroke,

the patient's overall risk profile and the severity of the initial event is essential to guide safe and effective management.

Evidence suggests that restarting within 4 days for AF-related ischemic stroke does not significantly increase the risk of major bleeding while reducing the risk of early recurrent ischemic events. Current expert recommendations suggest resuming anticoagulation after 2 days for patients with small ischemic strokes, 7 to 10 days for those with moderate-sized strokes and 10 to 14 days for large ischemic strokes. Additionally, studies have demonstrated that initiating DOACs early-within 3 days of an ischemic stroke is associated with similar rates of ischemic and hemorrhagic complications compared to delayed initiation, supporting their use in carefully selected patients.

In the context of Intracerebral Hemorrhage (ICH), determining the optimal timing for initiating anticoagulation remains challenging. Based on current evidence, delaying anticoagulation for less than 2 weeks may be reasonable in patients with low-risk ICH who face a higher thromboembolic risk. Conversely, delaying anticoagulation for more than 4 weeks is generally advised for patients with high-risk ICH to minimize the potential for re-bleeding [23].

Cirrhosis: Patients with cirrhosis exhibit distinct hypercoagulable changes in their hemostatic system. Although there is mixed evidence regarding the prevalence of DVT and PE in cirrhotic patients, it is hypothesized that this may be due to the improper administration of thromboprophylaxis due to concerns about bleeding risks. Nevertheless, the risk of developing DVT or PE in cirrhosis is at least as high as in the general population. Clinical prediction scores, such as the Padua Prediction Score (PPS) and improve scores, are recommended to identify cirrhotic patients at high risk for VTE, with a Padua prediction score >3 or improve score ≥ 4 indicating higher risk. The use of viscoelastic tests TEG is ThromboElastoGraphy (TEG) and Rotational ThromboElastoMetry (ROTEM) to assess thrombotic risk is discouraged, as they tend to underestimate hemostatic capacity in cirrhotic patients. Thromboprophylaxis with LMWH is generally considered safe. Thromboprophylaxis with DOACs is considered safe in patients with Child-Turcotte-Pugh (CTP) class A and B cirrhosis but should be avoided in those with more advanced liver disease [24].

CONCLUSION

In-patient management of anticoagulation therapy requires a delicate balance between the risks of thrombosis and bleeding, both influenced by a variety of clinical factors, including comorbidities, medications and the patient's condition. An individualized approach to anticoagulation must consider the "who", "when" and "how" of therapy to optimize patient outcomes and minimize complications. By adhering to evidence-based guidelines and considering patient-specific factors, clinicians can enhance the safety and efficacy of anticoagulation therapy, ultimately improving patient care and reducing in-hospital complications.

CONFLICT OF INTERESTS

The authors declare no conflicts of interest related to this review. No benefits in any form have been received or will be

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