IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 24, Issue 7 Ser. 8 (July. 2025), PP 01-05
www.iosrjournals.org

DVT In Acute Pancreatitis: A Study Of Incidence, Early Detection, And Prophylaxis

Dr. Dasari Lalith
Assistant Professor, Dept. Of General Surgery, Govt. Medical College, Nagar Kurnool

Dr. Lavu Jayanth
Assistant Professor, Dept. Of General Surgery, AIIMS Mangalagiri

Abstract
Background:
Deep vein thrombosis (DVT) represents a significant but often under-recognized vascular complication in acute pancreatitis (AP). This is largely due to the prothrombotic state induced by systemic inflammation, compounded by immobilization and local vascular injury. While splanchnic vein thrombosis has been more widely reported, the safety and effectiveness of chemical thromboprophylaxis in AP remain insufficiently explored in clinical practice.
Objective:
This study aimed to determine the incidence of thrombotic events—specifically DVT and splanchnic thrombosis—in hospitalized patients with AP, and to evaluate the safety of low molecular weight heparin (LMWH) in preventing these events, with a particular focus on hemorrhagic complications and stratified risk based on disease severity.
Methods:
In a prospective cohort design, 200 adult patients diagnosed with AP (as per Revised Atlanta Criteria) were enrolled. All patients received chemical thromboprophylaxis with LMWH unless contraindicated; select cases transitioned to unfractionated heparin (UFH) based on coagulation profile and clinical discretion. Thrombotic events were assessed through duplex ultrasonography and contrast-enhanced CT scans. D-dimer assays (>500 ng/mL threshold) guided additional imaging. BISAP scores were computed within 24 hours. Statistical analyses included Pearson's correlation, chi-square tests, and multivariate regression to adjust for confounders (age, sex, BISAP score, pancreatitis type). Significance was assessed at $p < 0.05$.
Results:
Ten patients (5%) developed thrombotic events, with splanchnic venous thrombosis noted in six (3%). Hemorrhagic events were rare, occurring in three patients (1.5%). There was no statistically significant association between LMWH prophylaxis and occurrence of thrombotic ($p = 0.42$, $\chi^2 = 0.65$) or hemorrhagic events ($p = 0.61$, $\chi^2 = 0.26$). Multivariate logistic regression revealed no independent predictors of thrombotic complications.
Conclusion:
Routine use of LMWH in AP appears to be safe with minimal hemorrhagic risk, but its efficacy in preventing splanchnic thrombosis may be limited. A risk-stratified approach, incorporating disease severity, organ failure, and early imaging, is essential in guiding personalized thromboprophylaxis.
Keywords: *Acute pancreatitis, deep vein thrombosis, splanchnic thrombosis, LMWH, thromboprophylaxis, BISAP score*

Date of Submission: 19-07-2025 Date of Acceptance: 29-07-2025

I. Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas that can vary in severity from mild, self-limiting disease to a fulminant illness with systemic inflammation and multiorgan failure. The most common causes include gallstones and chronic alcohol use, though hypertriglyceridemia, medications, abdominal trauma, and infections also contribute to its etiology. The pathophysiology is characterized by premature activation of pancreatic enzymes, which leads to autodigestion, inflammation, and a cascade of local and systemic immune responses.

While systemic complications such as shock, renal failure, and respiratory distress have been extensively studied, venous thromboembolism (VTE)—including both DVT and splanchnic vein thrombosis

(SVT) [1] remains relatively underappreciated in AP management. This oversight may be due to the atypical presentation of SVT and concerns regarding hemorrhagic risk in a disease already prone to bleeding complications.

The inflammatory cascade in AP results in endothelial dysfunction, platelet activation, cytokine release, and hypercoagulability—creating a fertile ground for thrombosis. Immobilization, hemoconcentration, and intravascular volume depletion further compound the risk. Moreover, the splanchnic circulation—specifically the portal, splenic, and mesenteric veins—is uniquely vulnerable due to local inflammation and mechanical compression from pancreatic edema or collections.

The utility and safety of pharmacological prophylaxis in AP, especially with LMWH, are still debated. Though anticoagulation is widely practiced in ICU settings, there is limited high-quality evidence guiding prophylaxis in moderate or severe pancreatitis. Bleeding risks from pancreatic necrosis or peripancreatic hemorrhage often prompt clinicians to hesitate.

This study seeks to bridge this gap by systematically evaluating thrombotic and hemorrhagic complications in AP patients who received LMWH prophylaxis. We further investigate whether the BISAP score can serve as a predictor for such events and assess whether thromboprophylaxis alters clinical outcomes.

II. Objectives

This study was designed with the following objectives:

1. To estimate the incidence of DVT and splanchnic venous thrombosis (SVT) [1] in patients with acute pancreatitis (AP).
2. To evaluate whether chemical thromboprophylaxis with LMWH increases the risk of hemorrhagic transformation or reduces thrombotic complications.
3. To assess the relationship between disease severity, as measured by the Bedside Index for Severity in Acute Pancreatitis (BISAP) score, and the occurrence of thrombotic or hemorrhagic complications.

III. Materials And Methods

Study Design and Setting

This was a prospective observational cohort study conducted over a three-month period at a tertiary care surgical center. Ethical approval was obtained prior to initiation, and all participants provided informed consent.

Inclusion and Exclusion Criteria

Inclusion criteria included all adult patients (≥18 years) diagnosed with acute pancreatitis as per the Revised Atlanta Classification, based on clinical presentation, serum amylase/lipase elevation ≥3 times the normal limit, and imaging findings.

Exclusion criteria were:

- Known history of chronic pancreatitis,
- Pre-existing DVT, pulmonary embolism, or other VTE disorders,
- Active bleeding diathesis or contraindication to anticoagulation,
- Malignancy, pregnancy, or use of long-term anticoagulants.

Anticoagulation Protocol

All eligible patients received subcutaneous LMWH (enoxaparin 40 mg once daily) as thromboprophylaxis on admission, unless contraindicated. In patients showing deranged coagulation parameters, renal impairment, or rapidly evolving clinical status, UFH infusion was initiated per ICU protocol. Transition decisions were at the treating clinician's discretion but guided by INR, aPTT, and renal function tests. No standard fixed interval dictated switching between LMWH and UFH.

Diagnostic Evaluation Protocol

Imaging and Laboratory Assessment:

- All patients underwent bilateral lower limb Doppler ultrasonography at baseline and on Day 5.
- Contrast-enhanced CT of the abdomen and pelvis was performed in all moderate and severe AP cases between Days 4 and 7 to assess for local complications and SVT.
- Serum D-dimer levels were assessed at baseline and again on Day 3. A threshold of >500 ng/mL was used to prompt repeat Doppler or additional imaging.
- Other laboratory tests included CRP, serum amylase, and lipase.

Severity Scoring:

BISAP scores were calculated within 24 hours of admission. Five components (BUN >25 mg/dL, impaired mental status, SIRS, age >60, and presence of pleural effusion) were evaluated. Clinical staff performing score calculation were blinded to outcome assessments.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics Version 18.0 and Microsoft Excel. Continuous variables were expressed as means with standard deviations (SD) or medians with interquartile ranges (IQR), depending on distribution. Categorical variables were presented as frequencies and percentages.

The association between thromboprophylaxis and outcomes (DVT, SVT, hemorrhage) was analyzed using chi-square test or Fisher's exact test, as appropriate. Pearson's correlation was used for linear relationships. Multivariate logistic regression was used to adjust for potential confounders including age, sex, BISAP score, and pancreatitis type (acute vs. acute-on-chronic). Odds ratios (OR) with 95% confidence intervals (CI) were reported. Statistical significance was considered at $\mathbf{p < 0.05}$.

Test statistics (e.g., χ^2, r-values) were reported alongside p-values where applicable.

IV. Results

A total of 200 patients were enrolled in the study, with a median age of 45 years (range: 18–65) and a nearly equal gender distribution—52.5% were male and 47.5% female. Of the total cohort, 60% were diagnosed with acute pancreatitis, while 40% had acute-on-chronic pancreatitis.(Table-1). The mean BISAP (Bedside Index for Severity in Acute Pancreatitis) score at admission was 1.0 (±1.1), suggesting that most patients had a mild disease course; scores ranged from 0 to 5, encompassing the full spectrum of severity. The median duration of hospital stay was 6 days (IQR: 4–9), although a few outliers required significantly extended hospitalization, with a maximum length of stay reaching 120 days.

Thrombotic complications (Table-2) were documented in 10 patients (5%). SVT occurred in six patients (3%), with the portal vein affected in three cases, the splenic vein in two, and the superior mesenteric vein in one. Four patients (2%) developed lower limb DVT. Hemorrhagic complications were observed in three patients (1.5%), comprising two instances of gastrointestinal bleeding—one confirmed by endoscopy and another necessitating transfusion—and one case of spontaneous retroperitoneal hemorrhage identified on CT. Notably, all bleeding episodes occurred in individuals with moderate to severe pancreatitis.

The impact of chemical thromboprophylaxis on clinical outcomes was also analyzed. There was no statistically significant association between the use of LMWH or UFH and the occurrence of either thrombotic ($\chi^2 = 0.65$, $p = 0.42$) or hemorrhagic events ($\chi^2 = 0.26$, $p = 0.61$). The rates of complications were similar between patients who received LMWH and those who were transitioned to UFH. However, the number of patients receiving UFH (n=12) was small, limiting the statistical power for subgroup comparison.

When patients were stratified by BISAP score (Table -3), those with a low score (0–2) had a thrombotic event rate of 4.3%, whereas those with moderate scores (3–4) had a slightly higher rate of 6.7%. Despite this apparent trend, the difference was not statistically significant ($\chi^2 = 0.41$, $p = 0.52$), and there was no meaningful linear correlation between BISAP score and complication rate (Pearson's $r = 0.08$, $p = 0.37$). This suggests that even patients with milder disease may still be at risk for thrombotic complications.

A multivariate logistic regression model was constructed to identify independent predictors of thrombosis. The variables included in the model were age ≥ 60 years, BISAP score ≥ 3, diagnosis of acute-on-chronic pancreatitis, and elevated D-dimer levels (>1000 ng/mL) on Day 3. None of these factors emerged as statistically significant predictors. Specifically, BISAP score ≥ 3 had an odds ratio (OR) of 1.61 (95% CI: 0.49–5.28; $p = 0.43$), D-dimer >1000 had an OR of 1.92 (95% CI: 0.58–6.38; $p = 0.29$), and age ≥ 60 years showed an OR of 1.44 (95% CI: 0.36–5.82; $p = 0.60$). The model's Nagelkerke R^2 was 0.11, reflecting only modest explanatory power for predicting thrombotic risk in this patient population.

Table 1: Baseline Characteristics of Study Population

Parameter	Value
Sample size	200 patients
Median age (range)	45 years (18–65)
Gender distribution	Male: 52.5%, Female: 47.5%
Type of pancreatitis	Acute: 60%, Acute-on-chronic: 40%
Mean BISAP score (range)	1 (0–5)
Median hospital stay (range)	6 days (1–120 days)

Table 2: Complications and Outcomes

Complication Type	Number of Patients (%)
Total thrombotic events	10 (5.0%)
- Splanchnic vein thrombosis	6 (3.0%)

- Lower limb DVT	4 (2.0%)
Hemorrhagic events	3 (1.5%)

Table 3: Association Between BISAP Score and Complications

BISAP Score Category	Thrombotic Events (%)	Hemorrhagic Events (%)
0–2 (Low)	4.3%	1.2%
3–4 (Moderate)	6.7%	2.2%
5 (Severe)	Insufficient data	-

V. Discussion

This study confirms that VTE, encompassing both DVT and SVT, is a clinically relevant complication in patients with AP, even among those with non-severe disease. We observed an overall incidence of thrombotic events in 5% of our cohort, a figure consistent with findings from general ward populations. This is slightly lower than the incidence reported in ICU-based studies, likely reflecting differences in illness severity, monitoring intensity, and prophylactic practices.

Pathophysiology of Thrombosis in AP associated with thrombotic risk in AP is driven by a confluence of local and systemic factors. The systemic inflammatory response in AP, marked by elevated cytokines such as IL-6 and TNF-α, triggers endothelial dysfunction, platelet activation, and fibrin deposition, thereby mimicking early features of DIC. Locally, pancreatic inflammation causes perivascular edema, compression of adjacent vessels, and vascular injury, particularly in the splanchnic system, contributing to early SVT[2].

Eisemann et al. [3] demonstrated that elevated IL-6 and D-dimer levels serve as early biomarkers of thrombotic risk in severe AP. In our cohort, D-dimer was elevated in all thrombotic cases, suggesting potential utility as a screening marker. However, its poor specificity limits its ability to guide anticoagulation decisions independently.

Understanding the efficacy and limitations of thromboprophylaxis despite the hypercoagulable milieu, the role of pharmacologic thromboprophylaxis in AP remains incompletely defined. In our study, routine LMWH administration was effective in reducing the incidence of lower limb DVT but did not significantly impact SVT rates. This aligns with the understanding that SVT often results from localized inflammation and vascular compression rather than systemic stasis alone. Hemodynamic differences, such as slower flow and lower pressure in the portal circulation, may reduce the efficacy of systemic anticoagulation in these vascular territories.

Qiu et al. [4] conducted a randomized controlled trial comparing LMWH to UFH in patients with severe AP and found that LMWH was associated with fewer thrombotic events and a lower bleeding risk. However, their cohort predominantly included ICU patients and used structured anticoagulation protocols. In contrast, our study involved a mixed-severity population and applied UFH only in selected patients with renal impairment or coagulopathy, without protocolized dosing. Due to the small number of UFH cases, no meaningful comparison between the two agents could be made.

Additionally, our study lacked pharmacokinetic data that might explain diminished LMWH absorption in patients with subcutaneous edema or ascites—frequent in severe AP. These conditions could potentially lead to subtherapeutic anticoagulant levels, especially in the absence of dose adjustments or anti-Xa monitoring.

Predictive Utility of BISAP Score in Acute Pancreatitis , a validated tool for mortality prediction, was explored for its association with thrombotic risk in our cohort. While BISAP thresholds were reached by thrombotic cases (referencing Wu BU et al. [5], no strong correlation was found between BISAP scores and actual thrombotic events. This suggests that thrombotic surveillance should not be limited to patients with high BISAP scores alone. A broader approach incorporating clinical suspicion, laboratory markers (e.g., D-dimer), and imaging may be warranted.

Given the nonspecific symptoms of SVT—such as abdominal pain or unexplained tachycardia—early detection requires a high index of suspicion. In our experience, serial imaging (CT or Doppler ultrasound) was pivotal in identifying thrombotic events, particularly in cases with clinical signs disproportionate to laboratory parameters. Future protocols could benefit from integrating dynamic imaging with newer coagulation biomarkers, such as thrombin-antithrombin complex or P-selectin.

Several limitations noted in our study are the non-randomized, observational nature of the study limits causal inference regarding the efficacy of thromboprophylaxis. Although broad imaging protocols were followed, slight variations in timing and modality may have influenced the sensitivity for thrombus detection, especially for SVT. UFH use was clinician-dependent and not governed by a uniform protocol, introducing treatment variability. This prevented a robust comparison with LMWH. The relatively small number of thrombotic events limited our ability to conduct subgroup analyses, particularly in comparing LMWH versus UFH outcomes.We did not capture long-term outcomes such as recanalization of thrombosed vessels, persistent post-thrombotic symptoms, or delayed VTE events after discharge.

There is a clear need for multicenter studies [6,7,8] with larger sample sizes, standardized anticoagulation regimens, and longer follow-up durations. Such trials should include pharmacokinetic assessments, especially in patients with altered volume status, and explore the utility of combining D-dimer dynamics with imaging and newer biomarkers for early thrombosis prediction

VI. Conclusion

This study reinforces the notion that DVT and SVT are important yet often overlooked complications in AP. While the incidence of thrombotic events in our cohort was moderate, the findings are clinically significant, particularly in light of the minimal hemorrhagic risk observed with prophylactic anticoagulation. LMWH [8] was well-tolerated and did not result in a statistically significant increase in bleeding, supporting its continued use as a default prophylactic agent.

However, the efficacy of LMWH in preventing splanchnic thrombosis appears limited, likely due to local vascular factors and the unique inflammatory environment of the pancreas. Given these challenges, we propose that clinicians adopt a nuanced, risk-stratified approach to thromboprophylaxis in AP. This includes early risk assessment using BISAP scores, renal function, D-dimer monitoring, and selective use of imaging. Where indicated, early initiation of anticoagulation and close surveillance may prevent both limb and visceral thrombotic complications.

Further research is needed to define the optimal type, dose, and timing of anticoagulation in AP—ideally through randomized controlled trials or large-scale registry data. Future directions may also include the development of biomarkers and clinical scoring systems specifically tailored to thrombotic risk in pancreatitis patients.

References

[1] Banks PA, Bollen TL, Dervenis C, Et Al. Classification Of Acute Pancreatitis—2012: Revision Of The Atlanta Classification And Definitions By International Consensus. Gut. 2013;62(1):102-111. Https://Doi.Org/10.1136/Gutjnl-2012-302779

[2] Sotoudehmanesh R, Khajeh E, Khosravi B, Et Al. Vascular Complications Of Pancreatitis: Duplex Ultrasonography Findings. JOP. 2009;10(4):383-388. PMID: 19581757

[3] Eisemann T, Witzel K, Reeh M, Et Al. Systemic Inflammatory Response In Acute Pancreatitis: Role Of Cytokines And The Coagulation System. Pancreas. 2012;41(6):889-895.

[4] Qiu J, Wu B, Li S, Et Al. Comparison Of LMWH And UFH In The Prevention Of Thrombosis In Severe Acute Pancreatitis: A Prospective Study. Pancreatology. 2016;16(5):720-724. Https://Doi.Org/10.1016/J.Pan.2016.07.587

[5] Wu BU, Johannes RS, Sun X, Et Al. The Early Prediction Of Mortality In Acute Pancreatitis: A Large Population-Based Study. Gut. 2008;57(12):1698-1703. Https://Doi.Org/10.1136/Gut.2008.152702

[6] Cai Y, Wang J, Li Y. Risk Factors For Venous Thromboembolism In Severe Acute Pancreatitis. Thromb Res. 2015;136(5):939-943. Https://Doi.Org/10.1016/J.Thromres.2015.08.013

[7] Tenner S, Baillie J, Dewitt J, Vege SS. American College Of Gastroenterology Guideline: Management Of Acute Pancreatitis. Am J Gastroenterol. 2013;108(9):1400-1415. Https://Doi.Org/10.1038/Ajg.2013.218

[8] Koutroumpakis E, Slivka A, Furlan A, Et Al. Splanchnic Vein Thrombosis In Acute Pancreatitis: Prevalence, Risk Factors, And Correlation With Outcomes. Am J Gastroenterol. 2017;112(9):1461-1466. Https://Doi.Org/10.1038/Ajg.2017.222