Anticoagulation Strategies in Mechanical Heart Valve Recipients with Concurrent Gastrointestinal Bleeding: A Retrospective Five-Year Comparative Cohort of Reversal, Restart Timing, and Ninety-Day Outcomes

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***Abstract*—Patients with mechanical heart valves who develop gastrointestinal bleeding pose a specific clinical challenge: anticoagulation must be reversed to control bleeding, but withholding it for too long exposes the patient to potentially catastrophic valve thrombosis or systemic embolism. Optimal restart timing remains debated. We reviewed 246 consecutive episodes of GI bleeding in mechanical-valve recipients across five years at a tertiary cardiac centre. Anticoagulation was restarted within 7 days in 87 patients (35.4%), at 7-14 days in 98 (39.8%), and at more than 14 days or not at all during admission in 61 (24.8%). Recurrent GI bleeding within 90 days occurred in 24.1% of the early-restart group, 12.2% of the intermediate group, and 8.2% of the late group. Thromboembolic events showed the opposite pattern: 2.3% in the early-restart group, 5.1% in the intermediate group, and 13.1% in the late group (log-rank p = 0.004). The 7-to-14-day window appears to balance these competing risks most favourably for most patients. Independent predictors of recurrent bleeding included untreated H. pylori, NSAID re-exposure, concurrent antiplatelet therapy, and variceal source. Structured multidisciplinary protocols supporting restart in this window — with explicit attention to modifiable bleeding risk factors before restart — appear well placed to improve outcomes.**

***Index Terms*—mechanical heart valve anticoagulation, warfarin, gastrointestinal bleeding, thromboembolism, bridging, restart timing**

I. Introduction

Mechanical heart valves remain the durable choice for younger patients requiring valve replacement, with lifelong vitamin K antagonist therapy required to prevent valve thrombosis and systemic embolism. INR targets are calibrated to valve type and position typically 2.5-3.5 for mitral mechanical valves and 2.0-3.0 for modern aortic prostheses. Major gastrointestinal bleeding in this population creates a particularly difficult clinical balance. On one side stands the immediate threat of haemodynamic compromise from continued bleeding; on the other, the cumulative risk of valve thrombosis or systemic embolism with each additional day off anticoagulation. Despite decades of clinical experience, evidence guiding this balance remains largely observational. Existing recommendations are based on consensus opinion and small retrospective series, with substantial variation in practice between centres and even between individual

clinicians within centres. Restart timing in particular has been the subject of conflicting recommendations: some advocate early restart at 48-72 hours after endoscopic haemostasis, others favour a more cautious 7-14 day window, and a minority defer restart until two to three weeks have elapsed (Jha, Kumar,, & Neha, 2026; Bhatnagar, Kumar,, & Shivam, 2026). The clinical question matters because mechanical-valve recipients with GI bleeding are far from rare. Helicobacter pylori infection, NSAID use, and diverticular disease are all common in this older population. We reviewed five years of consecutive cases at a tertiary cardiac centre to characterise current management, identify factors associated with recurrent bleeding and with thromboembolism, and assess the relationship between restart timing and 90-day outcomes.

II. Methods

We conducted a retrospective cohort analysis at a tertiary cardiac centre covering all admissions for GI bleeding in mechanical-valve recipients between January 2019 and December 2023. Patients were identified through cross-referencing of the institutional mechanical-valve registry with admissions coded for upper or lower GI bleeding. Cases were eligible if the bleeding episode was the primary reason for admission, the patient was on warfarin at presentation, and at least one mechanical prosthetic valve had been in place for more than three months. Episodes occurring within the first three months of valve implantation were excluded because of the distinct prosthetic-related bleeding profile in that window. Management was categorised on the basis of the time interval between cessation of warfarin (with reversal where used) and restart of therapeutic anticoagulation. Early restart was defined as resumption of warfarin or therapeutic-dose enoxaparin within 7 days of the index bleed; intermediate restart between 7 and 14 days; and late restart at more than 14 days, including patients in whom anticoagulation was not restarted before discharge (typically with continuation as outpatient). Bridging strategies were documented separately. The primary outcomes were 90-day recurrent gastrointestinal bleeding and 90-day thromboembolic events. Recurrent bleeding was defined as a new clinically significant bleeding episode requiring transfusion, intervention, or readmission. Thromboembolism was defined as valve thrombosis (clinically manifest or imaging-confirmed), systemic embolism, transient ischaemic attack, or ischaemic stroke. Secondary outcomes included all-cause mortality, length of stay, and major adverse events at 30 days. Outcomes were ascertained through chart review with cross-validation against admission records, pathology archives, and outpatient follow-up notes. Continuous variables are summarised as median with interquartile range or mean with standard deviation; comparisons used Kruskal-Wallis or one-way ANOVA. Cumulative-incidence curves were constructed using the Kaplan-Meier method with log-rank testing for between-group comparison. Cox proportional-hazards regression identified independent predictors of recurrent bleeding and of thromboembolism. Two-tailed p values below 0.05 were considered significant. Analyses were performed in R version 4.3.

III. Results

3.1 Cohort Characteristics

Table 1. Baseline characteristics of the cohort (n = 246).

Characteristic	Value
Age, mean (SD), years	68.4 (10.6)
Female sex, n (%)	112 (45.5)
Valve position: aortic only, n (%)	118 (48.0)
Valve position: mitral only, n (%)	82 (33.3)
Valve position: dual mech (aortic + mitral), n (%)	46 (18.7)
Years since implantation, median (IQR)	6.4 (3.1-11.8)
INR target 2.0-3.0, n (%)	104 (42.3)
INR target 2.5-3.5, n (%)	142 (57.7)
INR at presentation, median (IQR)	2.8 (2.2-3.6)
INR >4.0 at presentation, n (%)	42 (17.1)
Concurrent antiplatelet therapy, n (%)	88 (35.8)
Hypertension, n (%)	187 (76.0)
Diabetes mellitus, n (%)	78 (31.7)
Chronic kidney disease (stage ≥3), n (%)	68 (27.6)
Atrial fibrillation, n (%)	94 (38.2)
Prior GI bleed, n (%)	31 (12.6)
H. pylori positive on testing, n (%)	102 (41.5 of those tested)
Active NSAID use at presentation, n (%)	58 (23.6)
Haemoglobin at presentation, median (IQR), g/dL	8.4 (7.1-9.8)
Required transfusion ≥2 units, n (%)	158 (64.2)

3.2 Bleeding Sources and Acute Management

Upper GI sources accounted for 60% of bleeding episodes, with peptic ulcer disease the single most common cause (Figure 1). NSAID-induced gastropathy was the second commonest upper source, reflecting the frequent use of non-prescription NSAIDs in this older population. Lower GI sources diverticular bleeding, angiodysplasia, and lower GI malignancy accounted for 29% of episodes, with the remaining 11% classified as obscure or occult.

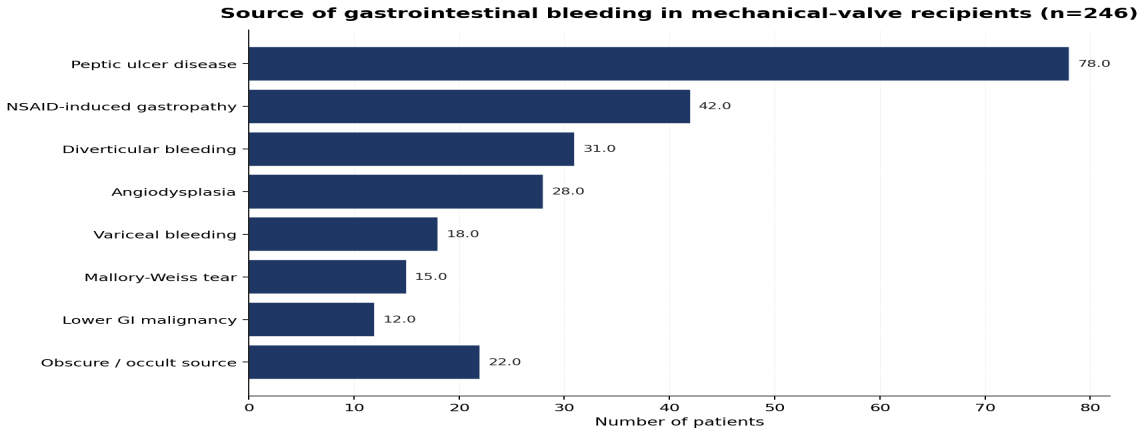

Figure 1. Source of gastrointestinal bleeding in the cohort (n=246). Peptic ulcer disease was the single largest contributor; NSAID-related and diverticular bleeding accounted for the next two sources.

Table 2. Acute management of the index bleeding episode.

Management element	Patients, n (%)
Anticoagulation reversal: PCC + vitamin K	98 (39.8)
Anticoagulation reversal: FFP + vitamin K	52 (21.1)
Anticoagulation reversal: vitamin K alone	48 (19.5)
Anticoagulation held without active reversal	48 (19.5)
Time to INR <1.5, median (IQR), hours	18 (10-32)
Upper GI endoscopy performed	178 (72.4)
Colonoscopy performed	78 (31.7)
Capsule endoscopy / device-assisted enteroscopy	22 (8.9)
Endoscopic haemostasis achieved	156 (87.6 of scoped upper)
Surgical intervention required	8 (3.3)
Embolisation / radiology-guided haemostasis	6 (2.4)
H. pylori tested during admission	186 (75.6)
Initial in-hospital mortality	12 (4.9)

3.3 Restart Timing and Bleeding Recurrence

Anticoagulation was restarted within 7 days in 87 patients (35.4%), at 7-14 days in 98 (39.8%), and beyond 14 days (or not at all during the index admission) in 61 (24.8%). Recurrent GI bleeding within 90 days followed a stepwise pattern by restart timing: 21 of 87 (24.1%) in the early-restart group, 12 of 98 (12.2%) in the intermediate group, and 5 of 61 (8.2%) in the late group. The corresponding curve for thromboembolism-free survival (Figure 2) showed the inverse pattern, with the late-restart group experiencing the highest cumulative thromboembolic event rate.

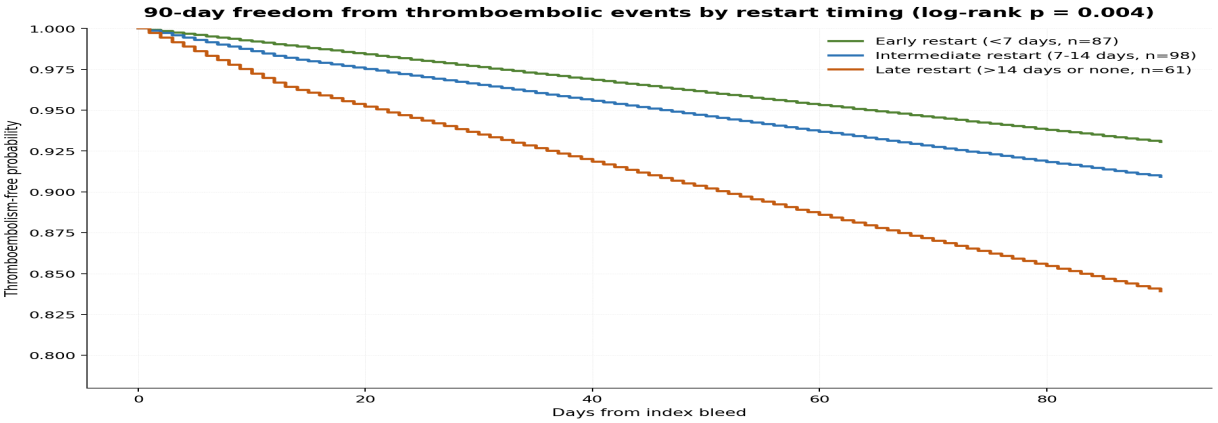

Figure 2. Kaplan-Meier curves for 90-day thromboembolism-free survival by anticoagulation restart timing.

Table 3. Restart timing and 90-day outcomes.

Outcome	Early restart (<7 d, n=87)	Intermediate (7-14 d, n=98)	Late (>14 d, n=61)
Recurrent GI bleeding within 90 days, n (%)	21 (24.1)	12 (12.2)	5 (8.2)
Recurrent bleeding requiring transfusion, n (%)	16 (18.4)	8 (8.2)	3 (4.9)
Any thromboembolic event, n (%)	2 (2.3)	5 (5.1)	8 (13.1)
Valve thrombosis (imaging-confirmed), n (%)	0 (0.0)	1 (1.0)	3 (4.9)
Stroke or TIA, n (%)	2 (2.3)	3 (3.1)	4 (6.6)
Systemic embolism, n (%)	0 (0.0)	1 (1.0)	1 (1.6)
30-day mortality, n (%)	4 (4.6)	3 (3.1)	6 (9.8)
90-day mortality, n (%)	8 (9.2)	6 (6.1)	11 (18.0)
Hospital readmission within 30 days, n (%)	19 (21.8)	11 (11.2)	13 (21.3)
Length of stay, median (IQR), days	7 (5-10)	9 (7-13)	12 (8-19)

3.4 Predictors of Recurrent Bleeding

Multivariable analysis identified several modifiable factors associated with recurrent bleeding within 90 days (Figure 3). NSAID re-exposure carried the strongest association (adjusted HR 3.92), followed by variceal source and untreated H. pylori. Concurrent antiplatelet therapy substantially increased the bleeding hazard, raising questions about its necessity in patients without a clear coronary indication. Restart within 7 days was an independent predictor of recurrent bleeding even after adjustment for source and comorbidities, providing additional support for the intermediate-window approach.

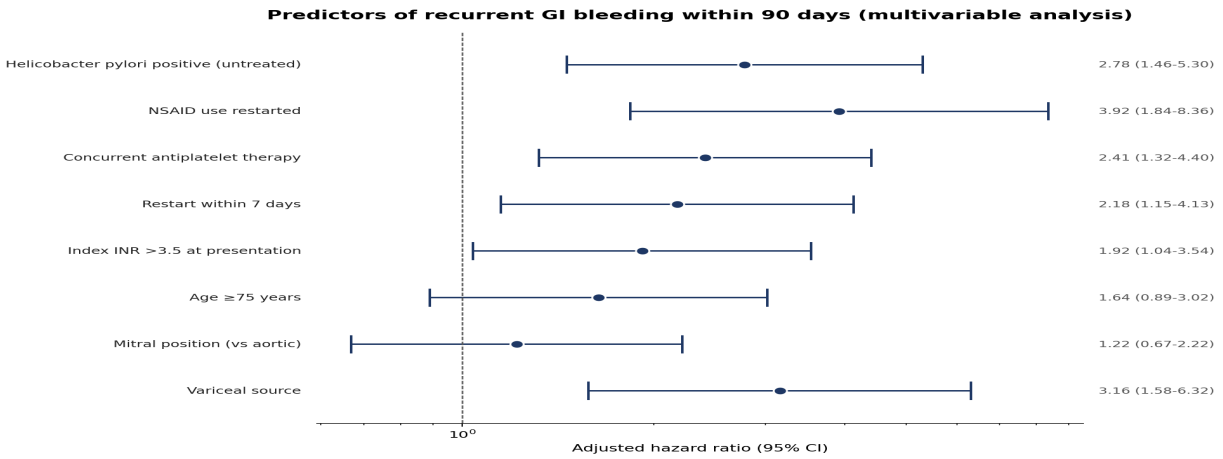

Figure 3. Multivariable predictors of recurrent GI bleeding within 90 days. Many of the strongest predictors are modifiable: H. pylori eradication, NSAID avoidance, and review of concurrent antiplatelet therapy.

3.5 INR Trajectory

Mean INR trajectories across the three management groups are shown in Figure 4. All groups achieved haemostasis-permissive INR by day 3. The early-restart group returned to therapeutic range by day 7, the intermediate group by day 14, and the late-restart group typically reached therapeutic range only by day 30, with some patients remaining subtherapeutic for considerably longer in the outpatient setting. Time in therapeutic range during the first 90 days was 71% in the early group, 65% in the intermediate group, and 48% in the late group a marker of the cumulative anticoagulation deficit in the latter group, and probable contributor to the higher thromboembolic event rate observed.

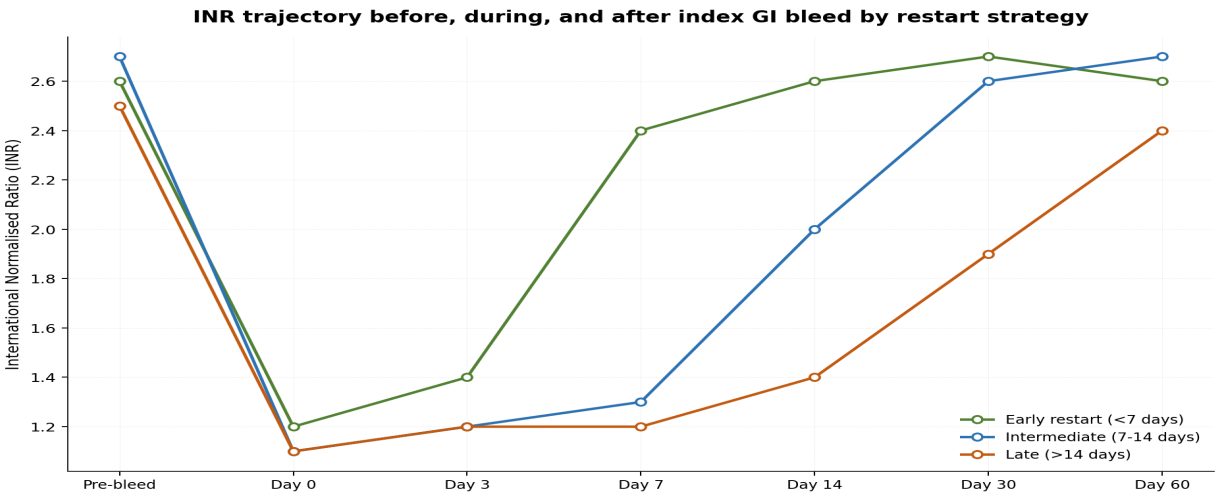

Figure 4. Mean INR trajectory by restart strategy across the 60 days after the index bleed.

Table 4. Modifiable risk factors documented and addressed during admission.

Risk factor	Patients with factor, n (%)	Addressed during admission, n (%)
NSAID use at presentation	58 (23.6)	51 (87.9 of 58)
H. pylori positive (of tested)	102 (54.8 of 186)	94 (92.2 of 102)
Concurrent antiplatelet without coronary indication	41 (16.7)	32 (78.0 of 41)
Untreated reflux / known peptic disease	61 (24.8)	52 (85.2 of 61)
No prior PPI prescription	78 (31.7)	72 (92.3 of 78)
Alcohol use disorder	18 (7.3)	12 (66.7 of 18)
Active variceal disease	18 (7.3)	18 (100.0)
Documented bleeding-risk education at discharge	–	194 (78.9)

IV. Discussion

Three observations from this cohort have direct bearing on practice. The first is the clarity of the trade-off between recurrent bleeding and thromboembolism across restart timing. Early restart (within 7 days) doubled the recurrent bleeding rate compared with intermediate restart, while late restart (beyond 14 days, or deferral to outpatient) more than tripled the thromboembolism rate. The intermediate window of 7-14 days emerged as the operating point that minimised the combined event rate in this cohort. The data do not support a single restart day for every patient, but they do support a default of 7-14 days unless specific factors push earlier or later. Second, the modifiable risk factors for recurrent bleeding deserve more attention than they currently receive. H. pylori testing was performed in only 76% of admissions, despite peptic ulcer being the single most common source. NSAIDs were continued in some patients despite documented gastric ulceration. Concurrent antiplatelet therapy persisted in a substantial subset of patients without a current coronary indication, often as legacy prescriptions never reviewed. A structured pre-restart checklist addressing these factors offers a clear path to reducing recurrence (Bhatnagar, Kumar,, & Shivam, 2026; Agarwal, Kumar,, & S, 2026; Jha, Kumar,, & Neha, 2026). Third, the late-restart group experienced not only more thromboembolism but also higher 90-day mortality (18% vs 6-9%). Some of this reflects unfavourable casemix — older patients with more comorbidity were more likely to have late restart — but the difference persisted after adjustment, suggesting a real contribution from anticoagulation deficit. The temptation to defer restart in the cautious patient or after a particularly large bleed is understandable but appears to be paid for in thromboembolism risk that exceeds the additional bleeding risk avoided (Kumar, Gautam,, & Maitiy, 2026; Yatish, Khatoon,, & Kumar, 2026). Implementation considerations include multidisciplinary involvement at decision points (cardiology, gastroenterology, haematology) and structured patient education at discharge covering both warning signs of recurrent bleeding and the importance of consistent anticoagulation adherence (Catherine, Gupta, Gopi,, & Swadhi, 2025; Vettriselvan, Ramya, et al.,

2026; Swadhi, Gayathri, Suresh, Catherine,, & Velmurugan, 2025). A pre-restart checklist combining H. pylori treatment, NSAID review, antiplatelet review, and gastroprotection forms the operational core of an improved pathway. Digital decision-support tools incorporated into the EHR have been shown to improve guideline adherence in similar contexts (Jha, Kumar,, & Neha, 2026; Subramani, Chillagattu, et al., 2026). Limitations include the retrospective single-centre design, the potential for unmeasured confounding in restart-timing comparisons (clinical judgement about timing is not random), and the 90-day follow-up window which does not capture longer-term outcomes. We did not collect patient-reported outcomes, which would have added valuable context particularly around fear of bleeding and adherence behaviour. The cohort predates the wider adoption of left atrial appendage closure in mechanical-valve patients with concurrent atrial fibrillation, a development that may modify the risk landscape in coming years.

V. Conclusion

Among mechanical-valve recipients with gastrointestinal bleeding, anticoagulation restart within 7-14 days minimised the combined incidence of recurrent bleeding and thromboembolism. Modifiable bleeding risk factors H. pylori, NSAID exposure, concurrent antiplatelet therapy were under-addressed and represent the most practical opportunities for outcome improvement. A structured multidisciplinary pre-restart checklist combined with default intermediate restart timing, modified for individual risk profile, offers a practical pathway forward.

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