Hemostatic performance of SpeedM® under anticoagulated conditions: an in vitro study

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Background

Uncontrolled bleeding remains one of the leading preventable causes of death following trauma [1]. Rapid and effective hemostasis is therefore essential to sustainably reduce morbidity and mortality. This challenge is further amplified by the increasing number of patients receiving long-term anticoagulant therapy [2]. In view of the high and steadily growing global burden of cardiovascular disease — with an estimated 523 million prevalent cases and 18.6 million deaths in 2019 [3] — the use of anticoagulants and antiplatelet agents is substantial and continues to expand. As a consequence, an increasing proportion of trauma patients may present while receiving such medication, where bleeding control is more difficult and time-critical. Modern anticoagulation strategies increasingly rely on direct factor Xa and thrombin inhibitors as well as antiplatelet agents; however, heparins and vitamin K antagonists remain clinically relevant [4]. Through selective inhibition of central hemostatic pathways, anticoagulants and antiplatelet agents prolong clotting time and increase bleeding tendency in a dose-dependent manner. While pharmacological reversal is available, clinical decision-making must weigh restoration of hemostasis against the risk of post-reversal thromboembolic complications [5]. Moreover, these reversal agents may be costly and may not always be immediately accessible in prehospital or ambulatory settings. Locally acting, system-independent therapeutic approaches may therefore represent a valuable adjunct; however, evidence for their effectiveness under anticoagulation remains limited to date.

Objective

This study evaluated the hemostatic performance of the mineral-coated wound dressing SpeedM® in human citrated plasma under defined anticoagulant exposure by comparing clotting time with and without the dressing, and estimated therapeutic reserve from the isoeffective rightward shift of anticoagulant concentration–response curves.

Material & methods

A standardized in vitro plasma coagulation model established at the IDK GmbH (2021) was used to evaluate the hemostatic effects of the HNT mineral-coated wound dressing SpeedM® on dose-response curves of representative anticoagulants. Human citrated platelet-poor plasma pooled from healthy donors was supplemented with clinically used anticoagulants: unfractionated heparin (UFH), enoxaparin (LMWH), rivaroxaban, dabigatran and acetylsalicylic acid. Reference concentrations were estimated from standard clinical doses assuming a circulating blood volume of 5–6 L and complete intravascular distribution and served solely as theoretical starting points for assay calibration; they do not necessarily reflect clinically observed peak or trough plasma concentrations. Two-fold serial dilutions were used to generate dose–response curves spanning concentrations below and above the in vitro activity threshold, tested both in the presence and absence of the hemostatic dressing under otherwise identical conditions. Vitamin K antagonists and P2Y12 inhibitors were not investigated, as their hepatic and platelet-dependent mechanisms cannot be modelled in this plasma-based in vitro model. For testing, 0.5 mL anticoagulated plasma was incubated at a temperature of 37°C with 0.5 cm² dressing samples and coagulation was initiated by recalcification using 25 mmol/L $CaCl_2$. Environmental conditions were set to room temperature (mean temperature 23 °C; relative humidity 46.6%). Clotting time was defined as the interval from recalcification to visible fibrin clot formation and served as the primary endpoint. If no visible clot formation occurred within a predefined observation period, measurements were censored at 120 minutes and recorded as the cut-off value. Therapeutic reserve (functional safety margin) was assessed as the isoeffective rightward shift of the anticoagulant concentration–response curve in the presence of the dressing compared with anticoagulant alone. Literature-reported usual on-therapy DOAC concentration ranges and anti-Xa target ranges for UFH/LMWH were overlaid for contextual interpretation and were not used as strict quantitative reference ranges [6–8]; for acetylsalicylic acid, no plasma therapeutic range was specified, as its primary antithrombotic effect is platelet-mediated and can only be approximated in a platelet-poor plasma model.

Results

Across all tested agents, clotting time increased in a concentration-dependent manner in recalcified citrated plasma. At higher concentrations, visible clot formation did not occur within the predefined observation period and measurements were therefore censored at the respective upper limit (120 min). In the presence of the hemostatic dressing SpeedM® , clotting time was consistently shorter than in anticoagulated plasma without dressing across all anticoagulants tested, indicating preserved procoagulant activity in this plasma-based model. Comparative concentration–response analysis showed an isoeffective rightward shift of curves in the presence of SpeedM® (Fig. 1), indicating that higher anticoagulant concentrations were required to achieve the same clotting time as with anticoagulant alone. The literature-based concentration ranges highlighted in the plots provide contextual interpretation of this shift and suggest a functional in vitro safety margin of SpeedM® within the experimental system.

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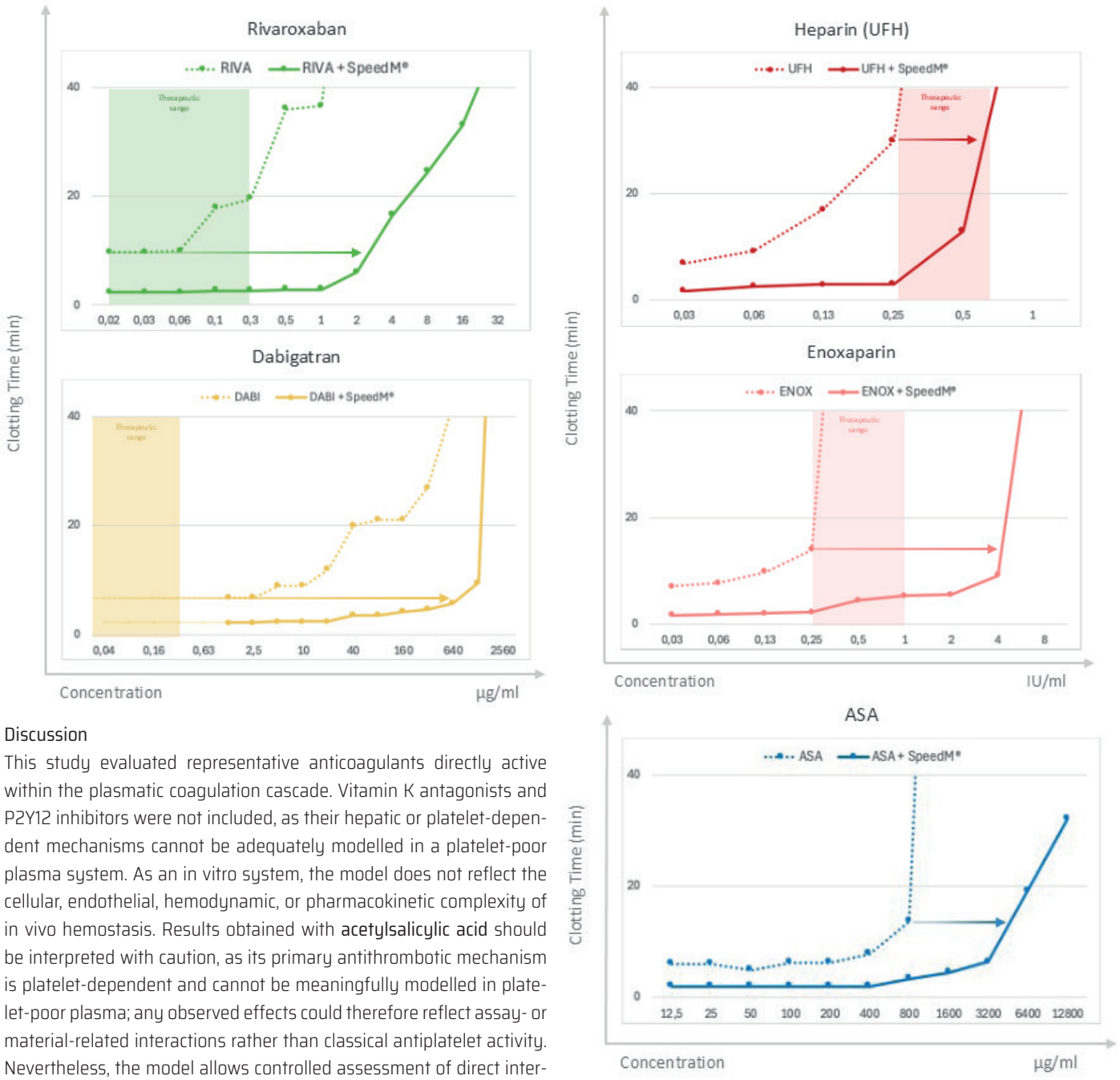

Fig. 1: **Anticoagulant concentration–response curves showing clotting time with and without SpeedM® in human citrated platelet-poor plasma.** Shaded areas indicate literature-reported on-therapy DOAC concentration ranges and anti-Xa target ranges for UFH/LMWH. Horizontal arrows indicate the isoeffective rightward shift.

Discussion

This study evaluated representative anticoagulants directly active within the plasmatic coagulation cascade. Vitamin K antagonists and P2Y12 inhibitors were not included, as their hepatic or platelet-dependent mechanisms cannot be adequately modelled in a platelet-poor plasma system. As an in vitro system, the model does not reflect the cellular, endothelial, hemodynamic, or pharmacokinetic complexity of in vivo hemostasis. Results obtained with **acetylsalicylic acid** should be interpreted with caution, as its primary antithrombotic mechanism is platelet-dependent and cannot be meaningfully modelled in platelet-poor plasma; any observed effects could therefore reflect assay- or material-related interactions rather than classical antiplatelet activity. Nevertheless, the model allows controlled assessment of direct interactions between anticoagulants and the clot-promoting activity of the dressing. Further validation in extended in vitro systems such as whole-blood viscoelastic assays (e.g., rotational thromboelastometry) and in vivo conditions are recommended to assess translational relevance beyond plasmatic clotting time alone.

Conclusion

SpeedM® reduced clotting time under defined anticoagulated in vitro conditions and maintained clot-promoting activity across multiple anticoagulant classes. The observed rightward shift in dose–response relationships indicates a functional safety margin within the plasma-based model.

References

[1] Jones AR, Miller J, Brown M. Epidemiology of trauma-related hemorrhage and time to definitive care across North America: Making the case for bleeding control education. Prehosp Disaster Med. 2023;38(6):780–783. doi:10.1017/S1049023X23006428.

[2] Grymonprez M, Simoens C, Steurbaut S, De Backer TL, Lahousse L. Worldwide trends in oral anticoagulant use in patients with atrial fibrillation from 2010 to 2018: a systematic review and meta-analysis. Europace. 2022;24(6):887–898. doi:10.1093/europace/euab303

[3] Roth GA, Mensah GA, Johnson CO, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: Update from the GBD 2019 study. J Am Coll Cardiol. 2020;76(25):2982–3021. doi:10.1016/j.jacc.2020.11.010.

[4] Van Gelder IC, et al. 2024 ESC guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2024;45(36):3314–3414. doi:10.1093/eurheartj/ehae176.

[5] Milling TJ Jr, Pollack CV Jr. A review of guidelines on anticoagulation reversal across different clinical scenarios - is there a general consensus? Am J Emerg Med. 2020;38(9):1890–1903. doi:10.1016/j.ajem.2020.05.086.

[6] de Vries TAC, Mallick IU, Bhagirath VC, Eikelboom JW, Gomes C, Yi Q, et al. Usual On-therapy Ranges of Direct Oral Anticoagulant Drug Concentrations for Atrial Fibrillation: A Systematic Review and Meta-analysis. Thromb Haemost. 2025;125(6):563–573. doi:10.1055/a-2446-1348.

[7] Coene KLM, van der Graaf F, van de Kerkhof D. Protocolled Redefinition of the Therapeutic Range for Unfractionated Heparin: Lost in Translation? Clin Appl Thromb Hemost. 2018;24(1):164-171. doi:10.1177/1076029616679508.

[8] Sikes L, Charles K, Antigua A, Patel R, Imboywa S, Cherian P. Anti-Factor Xa Level Monitoring for Enoxaparin Prophylaxis and Treatment in High-Risk Patient Groups. HCA Healthc J Med. 2023;4(2):105-110. doi:10.36518/2689-0216.1464.