Available online on 15.11.2025 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Review Article

A Holistic Review of Thrombosis Covering Diagnosis, Conventional Treatment, Nutraceutical Prevention and Advanced Drug Delivery

Mayur S. Jambhulkar*, Pooja R. Hatwar, Dr. Ravindra L. Bakal

Shri Swami Samarth Institute of Pharmacy, At Parsodi, Dhamangaon Rly (444709), Dist. Amravati, Maharashtra, India

Article Info:

Article History:

Received 20 Aug 2025
Reviewed 06 Oct 2025
Accepted 28 Oct 2025
Published 15 Nov 2025

Cite this article as:

Jambhulkar MS, Hatwar PR, Bakal RL, A Holistic Review of Thrombosis Covering Diagnosis, Conventional Treatment, Nutraceutical Prevention and Advanced Drug Delivery, Journal of Drug Delivery and Therapeutics. 2025; 15(11):97-105 *DOI: http://dx.doi.org/10.22270/jddt.v15i11.7448*

*For Correspondence:

Mayur S. Jambhulkar, Shri Swami Samarth Institute of Pharmacy, At Parsodi, Dhamangaon Rly (444709), Dist. Amravati, Maharashtra, India

Abstract

Thrombosis, or the formation of blood clots within blood vessels, is a serious medical condition that can lead to life-threatening complications. It is a complex process involving interactions between hemostatic, hemodynamic, and surface parameters. The risk factors for thrombosis include cancer, surgery, trauma, and genetic conditions. According to research, cancer patients are at a higher risk of developing thrombosis, with venous thromboembolism being the second most common cause of death in these patients. Understanding the causes, diagnosis, and treatment options for thrombosis is crucial for preventing and managing this condition. This review aims to provide an overview of the current knowledge on thrombosis, including its pathophysiology, risk factors, diagnostic tools, and treatment.

Keywords: Thrombosis, Blood clots, Hemostasis, Thrombolytic therapy, Anticoagulation, Cardiovascular disease

1. Introduction:

Thrombosis was defined as "hemostasis in the wrong place" by Oxford University researcher R. Gwynn Macfarlane in a 1977 issue of the British Medical Bulletin that focused on the pathology of blood coagulation [1]. A fibrin clot is the result of the intricate physiological process known as hemostasis [2]. The complex pathophysiological mechanism that causes thrombus development involves interactions between hemostatic (hypercoagulability, inappropriate anticoagulation), hemodynamic (low flow), and surface (poor endothelialization) parameters [3]. Blood hypercoagulability, blood flow abnormalities, and endothelial damage are the three elements of Virchow's triad that interact to upset the equilibrium between prothrombotic and thrombolytic processes, which explains the etiopathogenesis of thrombosis [4]. The primary cause of coronary heart disease, rheumatic heart disease, deep vein thrombosis, peripheral arterial and cerebrovascular illness, and pulmonary embolism is inflammation of the arterial blood vessels [5]. Life-threatening thrombus-related conditions include stroke, pulmonary embolism, and acute myocardial infarction, particularly in industrialized nations [6].

With an incidence of two to three per 100,000 persons annually, the portal vein is the conduit most commonly impacted. Liver cirrhosis is typically the underlying cause of thrombosis [7]. Patients who have cirrhosis or an underlying cancer are more likely to get thrombosis, according to research [8]. In this context, local risk factors include intra-abdominal infections, abdominal trauma, cancer, and intra-abdominal surgery (such as a splenectomy) [9].

A serious consequence is cancer-associated thrombosis, which encompasses both venous and arterial thromboembolism [10]. Venous thromboembolism is the second most common cause of death for cancer patients, after the course of the disease, and cancer is a significant risk factor for the condition on its own [11]. Twenty percent of all cases of venous thromboembolism are caused by active cancer [12].

There is substantial evidence linking the use of estrogen in postmenopausal hormonal replacement therapy or as a contraceptive to a higher risk of venous and arterial thrombosis [13].

A deeper understanding of the mechanisms governing thrombus formation is urgently needed in light of the

dearth of safe and effective treatments for VTE. This understanding could eventually lead to the development of effective preventative medications as well as the identification of novel therapeutic targets[14].

2. Pathophysiology:

Virchow's Triad, which includes changes in blood flow, endothelial damage, and a hypercoagulable condition, has been the primary explanation for thrombosis formation for more than a century [15]. Both superficial veins (closer to the surface) and deep veins (deeper into the body; more dangerous) can develop blood clots [16]. Research on various gene-specific knockout mice has shed a great deal of light on how intrinsic pathway activation contributes to arterial thrombosis and is crucial for the development of thrombi in the venous system [17].

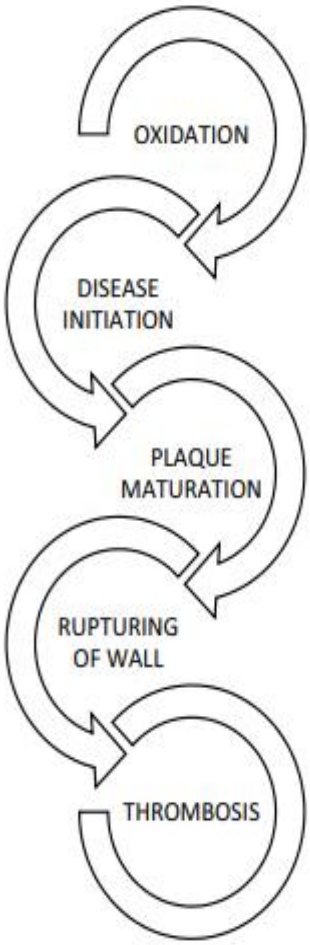

Figure 1: Stepwise progression of the inflammatory process, atherosclerosis, plaque formation and thrombosis [1].

2.1. Platelets and Thrombosis:

The two primary components of venous thrombus are an outside framework made of fibrin and extracellular DNA complexed with histone proteins, and an inner platelet-rich white thrombus that forms the so-called lines of Zahn surrounded by an outer red cell-dense fibrin clot [4]. Venous thrombi are known to contain platelets. GPIIb/IIIa, fibrin, glycophorin A (a particular protein found in red blood cells), and vWF were identified as being localized in the thrombi of patients who died from venous thromboembolism [18].

2.2. Inflammation and Thrombosis:

A rising corpus of research over the past ten years points to inflammation as a key factor in the pathophysiology of VTE, most likely through promoting endothelium damage and the hypercoagulable state [15]. A growing amount of research indicates that lipopolysaccharides (LPS) are linked to atherothrombosis through a number of mechanisms, including binding to macrophages and causing the release of inflammatory cytokines like interleukins 1–6–8–12 and tumor necrosis factor alpha (TNFa), as well as inducing reactive oxidant species that may negatively impact artery walls [19]. According to a study on Gram-negative bacterial-induced inflammatory vesicles and their procoagulant effects, when inflammatory vesicles are activated, macrophages release EVs that contain TF, which subsequently influence thrombogenesis [20].

2.3. Cirrhosis and Thrombosis:

In individuals with cirrhosis, the development of a portosystemic shunt and stagnant portal blood flow from progressive PH are directly linked to the development of portal vein thrombosis [9]. Venous thromboembolism is twice as likely to occur in hospitalized cirrhosis patients as in those without the disease [21].

2.4. Pregnancy and Thrombosis:

Venous thromboembolism is more likely to occur in women who are fertile. Changes in circulating reproductive hormones, especially during pregnancy or hormonal medications (such as oral contraceptives), may be the cause of this. Women with reproductive risk factors are more than five times more likely to experience a first venous thromboembolism than women without such risk factors [11]. The deep veins of the legs and the pulmonary veins are the most often affected areas by thrombosis associated with elevated estrogen levels [13].

2.5. Cancer and Thrombosis:

Tissue factor is a protein that is thought to be essential to cancer-associated thrombosis. It contributes to the development of vein thromboembolism as well as the advancement of cancer. It is produced in excess by cancer cells and functions as an activator of the extrinsic coagulation pathway, which activates factor X and, in turn, activates platelets and fibrin formation [12]. Potential causes of cancer-related hypercoagulability include iatrogenesis from antineoplastic treatment; stasis (direct pressure on blood vessels by the tumor mass); poor performance status and bed rest after surgery; and the release of heparinase from malignant tumors, which causes endogenous heparin to be degraded [22].

2.6. Immunity and Thrombosis:

An unchecked stimulation of the immunothrombosis process would result in the development of dangerous Deep Vein Thrombosis. Leukocytes are recognized to play a role in the pathogenesis of thrombosis [18].

Increased coagulation, reduced fibrinolysis, and immunological responses are the causes of COVID-19-associated prothrombosis [22].

2.7. Hypoxia and Thrombosis:

Hypoxia, or reduced oxygenation, is also a risk factor for thrombosis because systemic or local hypoxia increases the incidence of thrombosis. Numerous molecular and cellular signaling pathways are triggered by reductions in oxygenation, and these pathways may help regulate the development of thrombi [14].

2.8. Pollution and Thrombosis:

The mechanism of increased Vein Thromboembolism risk from air pollution also seems to be mediated by IL-6. Prothrombin time activated partial thromboplastin time, and decreased bleeding time all indicated coagulation activation in a mouse model after 10 μg of particulate matter with a diameter less than 10 μm (PM10) was administered intratracheally [23]. The respiratory epithelium is the point of entry, and endothelial damage may cause alveolar damage, which in turn triggers the production of cytokines and chemokines, attracts immune cells, and triggers coagulation and thrombosis [24].

2.9. Genetic and Thrombosis:

Due to either a compromised inhibition of thrombin or a loss of control over thrombin formation, hereditary thrombophilia increases the risk of VTE. The first genetic condition linked to an elevated risk of thrombosis to be identified was antithrombin deficiency [23].

3. Diagnosis:

Since there is no particular laboratory test that can definitively rule out thrombosis during the acute stage of the illness, blood tests are used to assess coagulation abnormalities such as an inflammatory process, systemic infection, or underlying hypercoagulable state [25]. Researchers in nuclear medicine continue to use a variety of targeting tactics in conjunction with other modalities [26].

3.1. Diagnosis Tools:

3.1.1. Doppler Imaging:

Doppler imaging is particularly crucial when used in conjunction, since it can show significantly lower velocities with aphasic waveforms. In the context of intraluminal filling problems, such observations are suggestive of PVT and portal hypertension [8]. Flow rate is estimated using Doppler [9].

3.1.2. Computed Tomography:

Sonography and/or computed tomography (CT) angiography should be the primary methods used to establish the diagnosis as soon as possible when anamnestic information, risk factors, or clinical symptoms raise the suspicion of thrombosis [7]. Among CT's benefits include its broad availability, clinician-friendly visualization, and capacity to visualize the whole mesenteric venous system [9].

3.1.3. Ultrasound Imaging:

In acute portal vein thrombosis, thrombus appears as hypo- or iso-echoic material occupying the lumen of a modestly dilated vein on ultrasound gray-scale imaging, but in chronic portal vein thrombosis, it appears as hyperechoic material following clot organization [9].

3.1.4. Transthoracic Echocardiography:

To determine the kind of arterial fibrillation and to identify patients at high risk of thrombosis, transthoracic echocardiography offers a thorough assessment of cardiac anatomy and function [27].

3.1.5. Magnetic Resonance Venography:

Acute or subacute, emergency or ambulatory cases benefit from magnetic resonance venography (MRV), which can also be used to confirm suspected cases of deep vein thrombosis when CT venography is either normal or inconclusive [25].

3.1.6. Nanoparticles as Diagnosis Tool:

Together with their superior imaging capabilities, nanoparticles may preferentially diffuse into thrombi. As a result, contrast agents based on nanoparticles that include a targeting molecule may be able to accurately detect thrombosis [28].

Table 1: Briefly the Advantages and Disadvantages of CT and MRV [3,8,7,4,25,28,29].

Techniques	Traits	Description
CT Venography	Advantages	1. Good visualization of major venous sinuses. 2. Simple, less time consuming, and less motion artifacts.
	Disadvantages	1. Ionizing radiation exposure. 2. Diabetes, and CKD patients may develop contrast nephropathy.
MR Venography	Advantages	1. No radiation exposure and good delineation of brain parenchyma. 2. Identify both cortical and deep venous thrombosis.
	Disadvantages	1. Time consuming, unavailability and produce motion artifacts. 2. Risk of gadolinium-induced nephrogenic systemic fibrosis.

4. Prevention:

Prevention of Cardiovascular Diseases with Anti-Inflammatory and Antioxidant Nutraceuticals and Herbal Product.

Table 2: Nutraceuticals and Herbal Products can be used in prevention of cardiovascular diseases [5,30,31,24,32,33].

Nutraceuticals and Herbal Product	Description
Murraya koenigii (Curry leaf)	Reduce cholesterol, phospholipids, LDL (bad cholesterol), very low-density lipoprotein fractions, and increase HDL (good cholesterol)
Curcuma longa (Curcumin)	Increase ECG and serum biomarker and decrease lipid profile
Allium sativum (Garlic)	Reduce serum cholesterol, and LDL cholesterol
Lagenaria siceraria Stand (Bottle gourd)	Increase in HDL levels, reduce hyperlipidemia
Trigonella foenumgraecum (Fenugreek)	Increase heart rate or weight
Beta vulgaris (Beetroot)	Reduce liver harm and inflammation
Allium cepa (Onion)	Slow down fat damage, delay LDL (bad cholesterol) oxidation
Omega-3-fatty acids	Reduce risk of coronary heart disease and sudden death by myocardial infarction
Vitamins C and E	Prolong time to arterial thrombosis
Olive oil	Reduce the risk of heart disease by lowering LDL (bad cholesterol) and in turn leaves high density lipoprotein (HDL) (good cholesterol)
Red Wine and Polyphenols	Protect platelets from a peroxidative stress, decrease platelet aggregation
Polar Lipids	Reduce platelet aggregation, anti-inflammatory effects
n-3 PUFA	Reduce thrombin formation, reduce oxidative stress, anti-inflammatory
Vitamin D	Reduce platelet aggregation and metabolism, anti-inflammatory
Kaempferol	Regulate systolic blood pressure, glycemic levels, and BMI
Catechins	Reduce mean arterial pressure. Improve insulin resistance and LDL, HDL levels

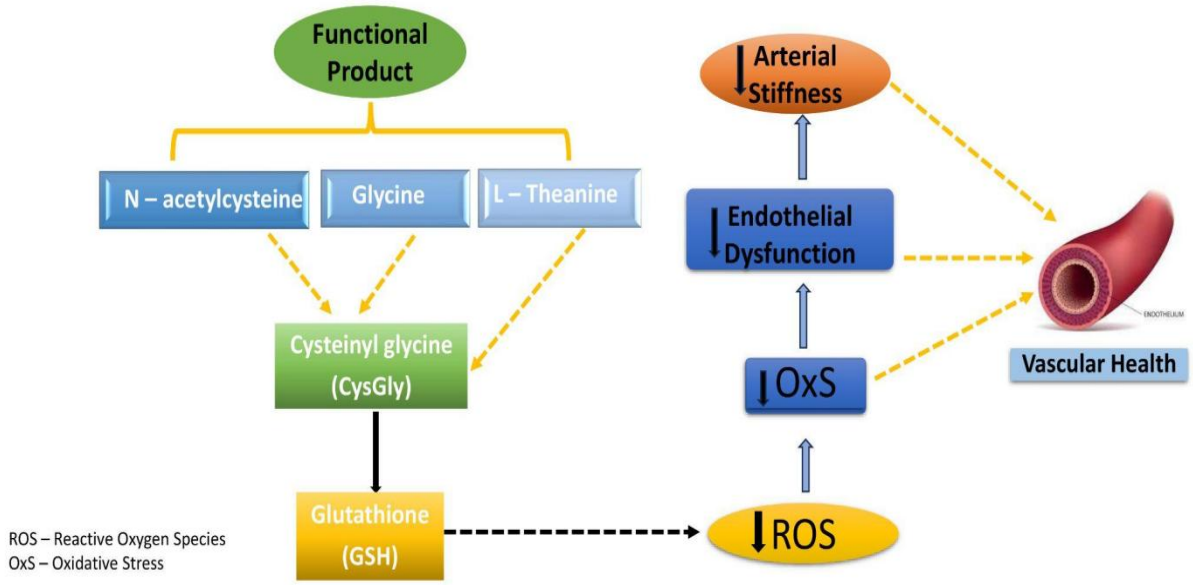

Figure 2: Potential mechanism of action of N-acetylcysteine, Glycine, and L-theanine in preservation of vasculature and cardiovascular health in humans [34].

5. Treatment and Management:

Surgery, thrombolytic therapy, and enhanced anticoagulation are available treatments for thrombosis [3]. Anticoagulation is the first-line treatment for thrombosis, according to practical guidelines [8]. Anticoagulation stops thrombosis from getting worse, reduces the chance of organ problems like liver failure and intestine infarction, and permits thrombosis to recanalize to stop portal hypertension from developing as a result of ongoing thrombotic occlusion [7]. Patients with thrombosis who were relative contraindications to both thrombolytic treatment and surgery may benefit from unfractionated heparin and low-molecular-weight heparin [3,4]. For many years, vitamin K antagonists have been utilized as anticoagulants [4]. In patients with nonvalvular atrial fibrillation, novel oral anticoagulants are currently regarded as the first-line treatment for long-term stroke prevention because they are safe and effective substitutes for VKA medication [27].

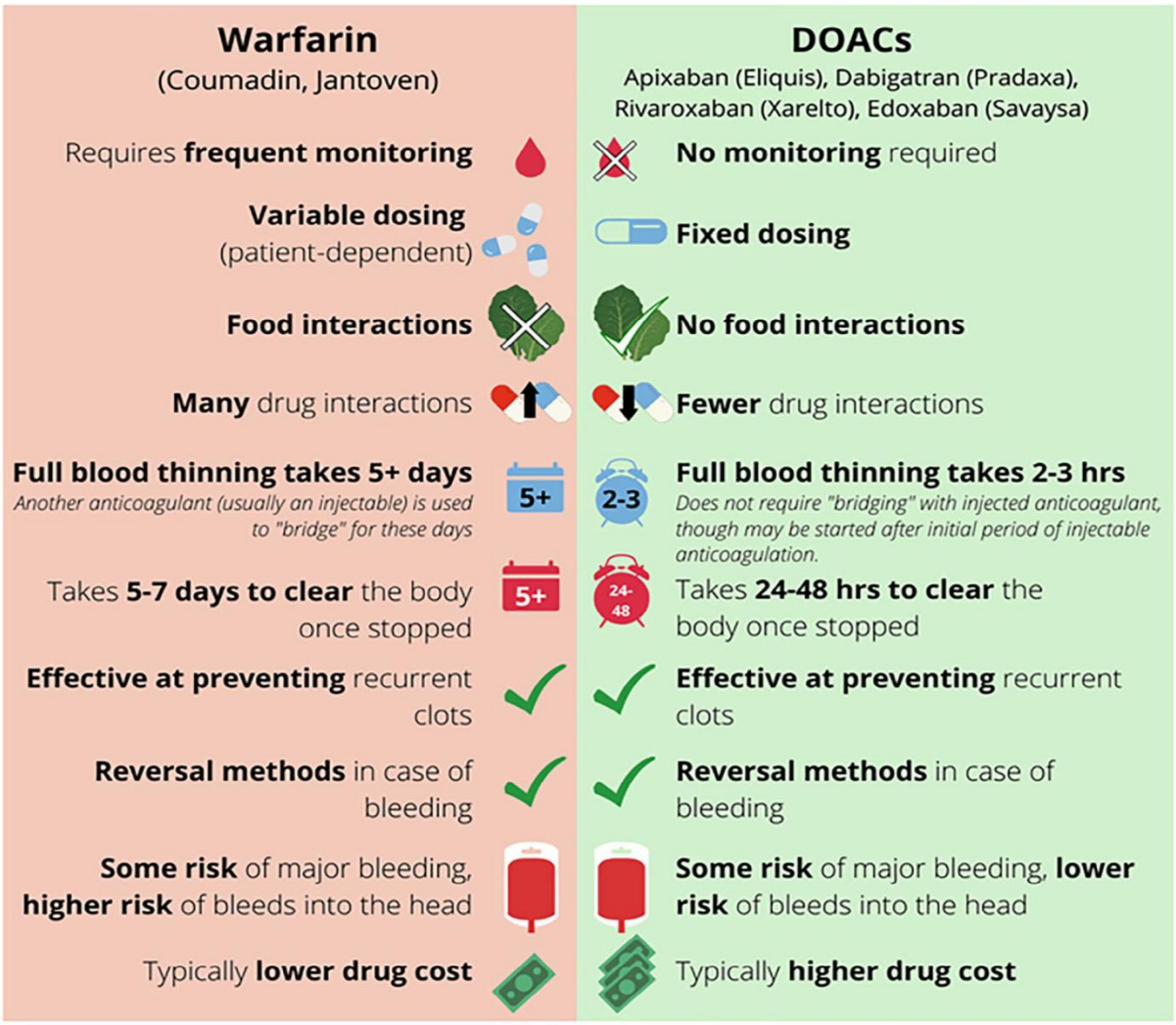

Figure 3: The similarities and differences between the two types of commonly prescribed blood thinners: warfarin and direct oral anticoagulants (DOACs) [16].

Endovascular thrombolysis or surgery are required if the thrombus worsens in spite of medication treatment or if there are signs of an impending intestinal infarction [9]. By integrating catheter-mounted devices that provide extra energy (mechanical, ultrasonic) into venous thrombus, catheter-directed thrombolysis has improved thrombus clearance and decreased the requirement for higher dosages of fibrinolytic medications [35].

Figure 4: Devices for Venous Thrombolysis and Thrombectomy [35].

Proenzymes known as plasminogen activators (PAs) are the forerunners of a class of proteolytic enzymes and are widely employed as thrombolytic agents to treat thrombi-induced obstructions [6].

Types of Thrombolysis Agents [6,36,37,38,39].

- Streptokinase
- Urokinase
- Staphylokinase
- Prourokinase
- Acylated Plasminogen-Streptokinase Activator Complex
- Alteplase
- Reteplase
- Tenecteplase
- Monteplase
- Lanoteplase
- Vampire Bat Plasminogen Activator
- Heparin

A potent collection of nano-engineered devices for diagnostic and/or therapeutic uses is combined in nanomedicine, a medical application of nanotechnology [40].

Nano-based Therapeutic Strategies [36]

- Targeted delivery of anti-inflammatory agents (eg, NO donors, corticosteroids) Targeted release of antiplatelet drugs (eg, aspirin, ligustrazine).
- Thrombin-responsive drug delivery systems (eg, chitosan-heparin nanocarriers) Fibrin-responsive release of fibrinolytic agents (eg, tPA-loaded nanogels).
- Microenvironment-responsive delivery of gases or antioxidants.
- Controlled-release platforms integrated with real-time imaging feedback.
- Long-acting anticoagulant delivery (eg, rivaroxaban-loaded nanocarriers).

Table 3: Drugs used in treatment of Thrombosis and their Nanocarriers [38,28].

Drug	**Nanocarriers**
Streptokinase	Liposome encapsulated & microencapsulated
Streptokinase	Platelet derived Microparticles—inspired nanovesicles
D-phenylalanyl-L-prolylL-arginyl chloromethyl ketone	Semipermeant perfluorocarbon core nanoparticles
tPlasminogen Activator	Chitosan magnetic nanoparticles
Recombinant tissue plasminogen activator	Magnetofluorescent nanoparticle

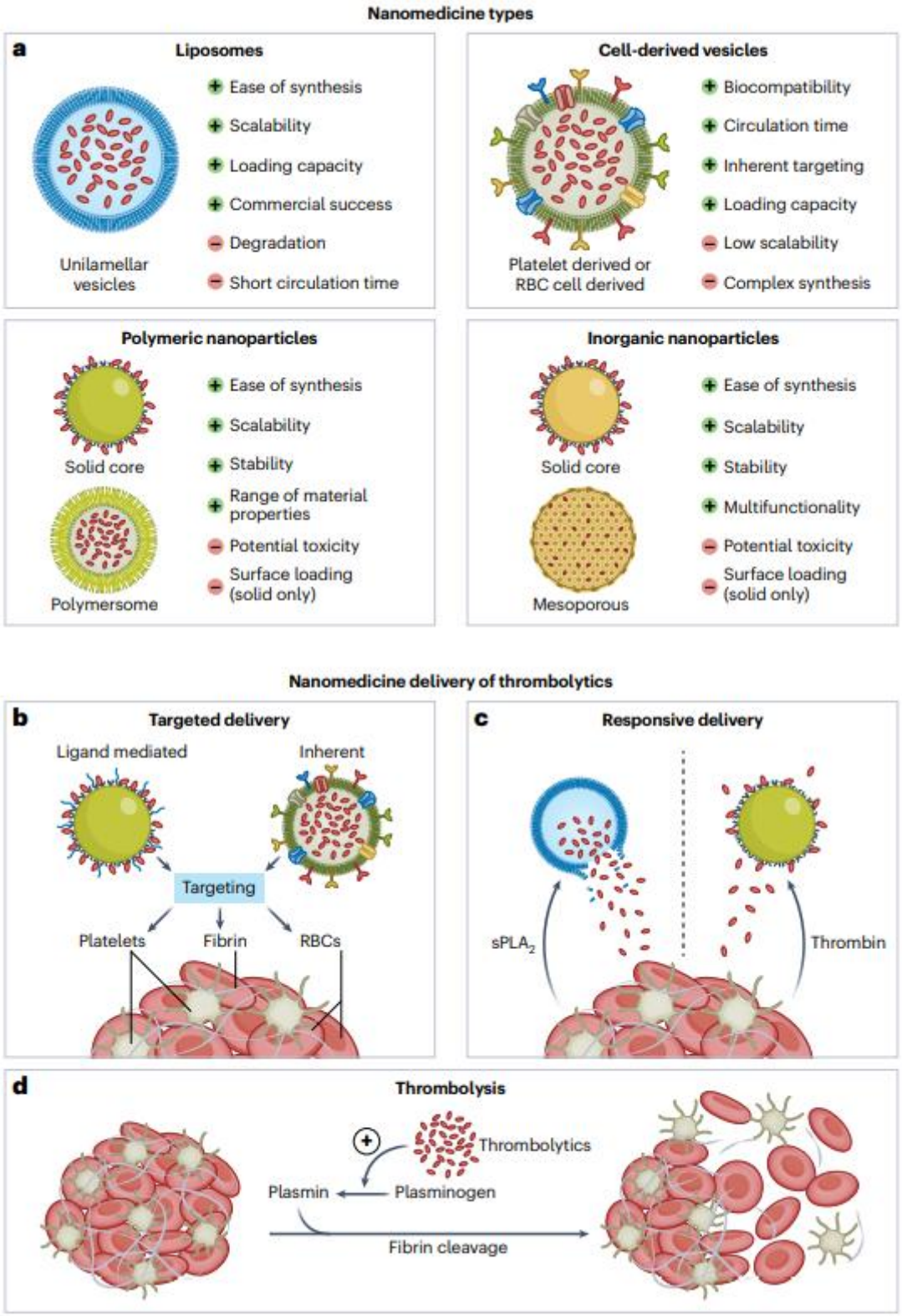

Figure 5: Nanomedicine types and their delivery as thrombolytics [41].

Conclusion:

Thrombosis is a complex and multifactorial condition that requires a comprehensive approach to prevention and treatment. Understanding the underlying causes and risk factors is essential for developing effective strategies to manage thrombosis. Diagnostic tools such as Doppler imaging, computed tomography, and magnetic resonance venography play a crucial role in detecting thrombosis. Treatment including anticoagulation, thrombolytic therapy, and surgery, aim to prevent thrombus growth, reduce complications, and restore blood flow. Further research is needed to develop new and effective treatments for thrombosis, particularly in patients with cancer and other high-risk conditions. By understanding the mechanisms of thrombosis and

developing targeted therapies, we can improve patient outcomes and reduce the burden of this disease.

Conflict of Interest: The authors declare no potential conflict of interest concerning the contents, authorship, and/or publication of this article.

Author Contributions: All authors have equal contributions in the preparation of the manuscript and compilation.

Source of Support: Nil

Funding: The authors declared that this study has received no financial support.

Informed Consent Statement: Not applicable.

Data Availability Statement: The data supporting this paper are available in the cited references.

Ethical approval: Not applicable.

References:

1. Gailani D, Gruber A. Targeting factor XI and factor XIa to prevent thrombosis. Blood. 2024 Apr 11;143(15):1465-1475. https://doi.org/10.1182/blood.2023020722 PMid:38142404 PMCid:PMC11033593

2. Burton AG, Jandrey KE. Use of Thromboelastography in Clinical Practice. Vet Clin North Am Small Anim Pract. 2020;50(6):1397-1409. https://doi.org/10.1016/j.cvsm.2020.08.001 PMid:32981595

3. Gündüz S, Kalçık M, Gürsoy MO, Güner A, Özkan M. Diagnosis, treatment & management of prosthetic valve thrombosis: the key considerations. Expert Rev Med Devices. 2020;17(3):209-221. https://doi.org/10.1080/17434440.2020.1733972 PMid:32085683

4. Morais MB, Aguiar de Sousa D. Pathophysiology and Management of Cerebral Venous Thrombosis. Hamostaseologie. 2025 Aug;45(4):324-334. https://doi.org/10.1055/a-2518-9103 PMid:40199511

5. Jain S, Buttar HS, Chintameneni M, Kaur G. Prevention of Cardiovascular Diseases with Anti-Inflammatory and Anti-Oxidant Nutraceuticals and Herbal Products: An Overview of Pre-Clinical and Clinical Studies. Recent Pat Inflamm Allergy Drug Discov. 2018;12(2):145-157. https://doi.org/10.2174/1872213X12666180815144803 PMid:30109827

6. Hassanpour S, Kim HJ, Saadati A, Tebon P, Xue C, van den Dolder FW, Thakor J, Baradaran B, Mosafer J, Baghbanzadeh A, de Barros NR, Hashemzaei M, Lee KJ, Lee J, Zhang S, Sun W, Cho HJ, Ahadian S, Ashammakhi N, Dokmeci MR, Mokhtarzadeh A, Khademhosseini A. Thrombolytic Agents: Nanocarriers in Controlled Release. Small. 2020 Oct;16(40):e2001647. https://doi.org/10.1002/smll.202001647 PMid:32790000 PMCid:PMC7702193

7. Mühlberg KS. Diagnosis and Therapy of Visceral Vein Thrombosis: An Update Based on the Revised AWMF S2k Guideline. Hamostaseologie. 2024 Apr;44(2):135-142. https://doi.org/10.1055/a-2178-6670 PMid:37992729

8. Ju C, Li X, Gadani S, Kapoor B, Partovi S. Portal Vein Thrombosis: Diagnosis and Endovascular Management. Rofo. 2022 Feb;194(2):169-180. English. https://doi.org/10.1055/a-1642-0990 PMid:34649289

9. Intagliata NM, Caldwell SH, Tripodi A. Diagnosis, Development, and Treatment of Portal Vein Thrombosis in Patients With and Without Cirrhosis. Gastroenterology. 2019 May;156(6):1582-1599.e1. https://doi.org/10.1053/j.gastro.2019.01.265 PMid:30771355

10. Kim AS, Khorana AA, McCrae KR. Mechanisms and biomarkers of cancer-associated thrombosis. Transl Res. 2020 Nov;225:33-53. https://doi.org/10.1016/j.trsl.2020.06.012 PMid:32645431 PMCid:PMC8020882

11. Farge D, Le Maignan C, Doucet L, Frere C. Women, thrombosis, and cancer. Thromb Res. 2019 Sep;181 Suppl 1:S47-S53. https://doi.org/10.1016/S0049-3848(19)30367-6 PMid:31477228

12. Fernandes CJ, Morinaga LTK, Alves JL Jr, Castro MA, Calderaro D, Jardim CVP, Souza R. Cancer-associated thrombosis: the when, how and why. Eur Respir Rev. 2019 Mar 27;28(151):180119. https://doi.org/10.1183/16000617.0119-2018 PMid:30918022 PMCid:PMC9488553

13. Abou-Ismail MY, Citla Sridhar D, Nayak L. Estrogen and thrombosis: A bench to bedside review. Thromb Res. 2020 Aug;192:40-51. https://doi.org/10.1016/j.thromres.2020.05.008 PMid:32450447 PMCid:PMC7341440

14. Gupta N, Zhao YY, Evans CE. The stimulation of thrombosis by hypoxia. Thromb Res. 2019 Sep;181:77-83. https://doi.org/10.1016/j.thromres.2019.07.013 PMid:31376606

15. Branchford BR, Carpenter SL. The Role of Inflammation in Venous Thromboembolism. Front Pediatr. 2018 May 23;6:142. https://doi.org/10.3389/fped.2018.00142 PMid:29876337 PMCid:PMC5974100

16. Frank C, Ratchford EV, Moll S. Vascular Disease Patient Information Page: A guide for patients with newly diagnosed deep vein thrombosis or pulmonary embolism. Vasc Med. 2023 Oct;28(5):481-486. https://doi.org/10.1177/1358863X231154756 PMid:37401771 PMCid:PMC10559639

17. Grover SP, Mackman N. Intrinsic Pathway of Coagulation and Thrombosis. Arterioscler Thromb Vasc Biol. 2019 Mar;39(3):331-338. https://doi.org/10.1161/ATVBAHA.118.312130 PMid:30700128 PMCid:PMC12578993

18. Navarrete S, Solar C, Tapia R, Pereira J, Fuentes E, Palomo I. Pathophysiology of deep vein thrombosis. Clin Exp Med. 2023 Jul;23(3):645-654. https://doi.org/10.1007/s10238-022-00829-w PMid:35471714

19. Carnevale R, Sciarretta S, Valenti V, di Nonno F, Calvieri C, Nocella C, Frati G, Forte M, d'Amati G, Pignataro MG, Severino A, Cangemi R, Arrivi A, Dominici M, Mangieri E, Gaudio C, Tanzilli G, Violi F. Low-grade endotoxaemia enhances artery thrombus growth via Toll-like receptor 4: implication for myocardial infarction. Eur Heart J. 2020 Sep 1;41(33):3156-3165. https://doi.org/10.1093/eurheartj/ehz893 PMid:31898723

20. He Y, Wu Q. The Effect of Extracellular Vesicles on Thrombosis. J Cardiovasc Transl Res. 2023 Jun;16(3):682-697. https://doi.org/10.1007/s12265-022-10342-w PMid:36441349 PMCid:PMC9707253

21. Van den Boom BP, Lisman T. Pathophysiology and management of bleeding and thrombosis in patients with liver disease. Int J Lab Hematol. 2022 Sep;44 Suppl 1(Suppl 1):79-88. https://doi.org/10.1111/ijlh.13856 PMid:35446468 PMCid:PMC9540811

22. Brito-Dellan N, Tsoukalas N, Font C. Thrombosis, cancer, and COVID-19. Support Care Cancer. 2022 Oct;30(10):8491-8500. https://doi.org/10.1007/s00520-022-07098-zm PMid:35567609 PMCid:PMC9106567

23. Schulman S, Makatsariya A, Khizroeva J, Bitsadze V, Kapanadze D. The Basic Principles of Pathophysiology of Venous Thrombosis. Int J Mol Sci. 2024 Oct 24;25(21):11447. https://doi.org/10.3390/ijms252111447 PMid:39519000 PMCid:PMC11547114

24. Tsoupras A, Lordan R, Zabetakis I. Thrombosis and COVID-19: The Potential Role of Nutrition. Front Nutr. 2020 Sep 25;7:583080. https://doi.org/10.3389/fnut.2020.583080 PMid:33102511 PMCid:PMC7545367

25. Ranjan R, Ken-Dror G, Sharma P. Pathophysiology, diagnosis and management of cerebral venous thrombosis: A comprehensive

review. Medicine (Baltimore). 2023 Dec 1;102(48):e36366. https://doi.org/10.1097/MD.0000000000036366 PMid:38050259 PMCid:PMC10695550

26. Samridhi, Setia A, Mehata AK, Priya V, Pradhan A, Prasanna P, Mohan S, Muthu MS. Nanoparticles for Thrombus Diagnosis and Therapy: Emerging Trends in Thrombus-theranostics. Nanotheranostics. 2024 Jan 1;8(2):127-149. https://doi.org/10.7150/ntno.92184 PMid:38328614 PMCid:PMC10845253

27. Melillo E, Palmiero G, Ferro A, Mocavero PE, Monda V, Ascione L. Diagnosis and Management of Left Atrium Appendage Thrombosis in Atrial Fibrillation Patients Undergoing Cardioversion. Medicina (Kaunas). 2019 Aug 21;55(9):511. https://doi.org/10.3390/medicina55090511 PMid:31438560 PMCid:PMC6780583

28. Su M, Dai Q, Chen C, Zeng Y, Chu C, Liu G. Nano-Medicine for Thrombosis: A Precise Diagnosis and Treatment Strategy. Nanomicro Lett. 2020 Apr 20;12(1):96. https://doi.org/10.1007/s40820-020-00434-0 PMid:34138079 PMCid:PMC7770919

29. Shahinian JH, Chan V, Pislaru SV. Chronic thrombosis of bioprostheses: Diagnosis and management. Prog Cardiovasc Dis. 2022 May-Jun;72:15-20. https://doi.org/10.1016/j.pcad.2022.06.008 PMid:35764124

30. Banning M. The role of omega-3-fatty acids in the prevention of cardiac events. Br J Nurs. 2005 May 12-25;14(9):503-8. https://doi.org/10.12968/bjon.2005.14.9.18075 PMid:15924000

31. Chakrabarti S, Freedman JE. Review: Nutriceuticals as antithrombotic agents. Cardiovasc Ther. 2010 Aug;28(4):227-35. https://doi.org/10.1111/j.1755-5922.2010.00161.x PMid:20553290

32. Caffrey C, Leamy A, O'Sullivan E, Zabetakis I, Lordan R, Nasopoulou C. Cardiovascular Diseases and Marine Oils: A Focus on Omega-3 Polyunsaturated Fatty Acids and Polar Lipids. Mar Drugs. 2023 Oct 24;21(11):549. https://doi.org/10.3390/md21110549 PMid:37999373 PMCid:PMC10672651

33. Khan J, Deb PK, Priya S, Medina KD, Devi R, Walode SG, Rudrapal M. Dietary Flavonoids: Cardioprotective Potential with Antioxidant Effects and Their Pharmacokinetic, Toxicological and Therapeutic Concerns. Molecules. 2021 Jun 30;26(13):4021. https://doi.org/10.3390/molecules26134021 PMid:34209338 PMCid:PMC8272101

34. Meegaswatte H, Speer K, McKune AJ, Naumovski N. Functional Foods and Nutraceuticals for the Management of Cardiovascular Disease Risk in Postmenopausal Women. Rev Cardiovasc Med. 2024 Dec 25;25(12):460. https://doi.org/10.31083/j.rcm2512460 PMid:39742223 PMCid:PMC11683719

35. Goldhaber SZ, Magnuson EA, Chinnakondepalli KM, Cohen DJ, Vedantham S. Catheter-directed thrombolysis for deep vein thrombosis: 2021 update. Vasc Med. 2021 Dec;26(6):662-669. https://doi.org/10.1177/1358863X211042930 PMid:34606385 PMCid:PMC9009765

36. Wu J, Zhang Y, Chen W, Hao T, Ran C, Zhou Y, Shen Y, You W, Wang T. Smart Thrombosis Care: The Rise of Closed-Loop Diagnosis-to-Treatment Nano Systems. Int J Nanomedicine. 2025 Jun 19;20:7851-7868. https://doi.org/10.2147/IJN.S530884 PMid:40551977 PMCid:PMC12184709

37. Palazzolo JS, Westein E, Hagemeyer CE, Wang TY. Targeting Nanotechnologies for the Treatment of Thrombosis and Cardiovascular Disease. Semin Thromb Hemost. 2020 Jul;46(5):606-621. https://doi.org/10.1055/s-0039-1697946 PMid:31614390

38. Manners N, Priya V, Mehata AK, Rawat M, Mohan S, Makeen HA, Albratty M, Albarrati A, Meraya AM, Muthu MS. Theranostic Nanomedicines for the Treatment of Cardiovascular and Related Diseases: Current Strategies and Future Perspectives. Pharmaceuticals (Basel). 2022 Apr 1;15(4):441. https://doi.org/10.3390/ph15040441 PMid:35455438 PMCid:PMC9029632

39. Russell P, Hagemeyer CE, Esser L, Voelcker NH. Theranostic nanoparticles for the management of thrombosis. Theranostics. 2022 Mar 14;12(6):2773-2800. https://doi.org/10.7150/thno.70001 PMid:35401833 PMCid:PMC8965493

40. Zenych A, Fournier L, Chauvierre C. Nanomedicine progress in thrombolytic therapy. Biomaterials. 2020 Nov;258:120297. https://doi.org/10.1016/j.biomaterials.2020.120297 PMid:32818824

41. Russell P, Esser L, Hagemeyer CE, Voelcker NH. The potential impact of nanomedicine on COVID-19-induced thrombosis. Nat Nanotechnol. 2023 Jan;18(1):11-22. https://doi.org/10.1038/s41565-022-01270-6 PMid:36536042