OPEN ACCESS

International Journal of Applied Medical and Health Sciences

Int J Appl Med Health Sci | Vol-1, Iss-1, Jul-Aug., 2026
e-ISSN: XXXX-XXXX
Homepage: https://ioasdpublisher.com/ijamhs

Climate Change and Deep Vein Thrombosis: Emerging Risks and Mechanisms

Emmanuel Ifeanyi Obeagu[1,2,3*]

[1]Department of Biomedical and Laboratory Science, Africa University, Mutare, Zimbabwe
[2]Department of Haematology and Molecular Medicine, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa
[3]Department of Haematology and Immunohaematology, Bahir Dar University, Bahir Dar, Ethiopia

Corresponding Author: Emmanuel Ifeanyi Obeagu **Email:** emmaneulobeagu@yahoo.com

ABSTRACT

Climate change is emerging as a significant driver of cardiovascular and hematologic complications, including deep vein thrombosis (DVT). Rising global temperatures, extreme weather events, and climate-induced environmental stressors contribute to increased thrombotic risk through mechanisms such as hemoconcentration, dehydration, immobilization, systemic inflammation, endothelial dysfunction, and stress-induced hypercoagulability. Vulnerable populations—including the elderly, pregnant women, patients with cardiovascular or hematologic disorders, and displaced individuals—are particularly susceptible to post-disaster or climate-related thrombotic events. Early identification using hematologic, coagulation, and inflammatory biomarkers, combined with preventive strategies such as hydration, mobilization, pharmacologic prophylaxis, and public health interventions, can reduce morbidity and mortality. This narrative review synthesizes current evidence on emerging climate-related thrombotic risks, elucidates underlying mechanisms, and provides insights for monitoring, prevention, and adaptive strategies. Understanding these links is critical for clinical management, disaster preparedness, and public health planning in an increasingly climate-sensitive world.

Keywords: Climate Change, Deep Vein Thrombosis, Hemoconcentration, Hypercoagulability, Environmental Stressors.

Review Article

Article History
Received: 24-06-2026
Accepted: 31-07-2026
Published: 12-08-2026

INTRODUCTION

Climate change is recognized as one of the most pressing global health challenges of the 21st century. Rising temperatures, extreme weather events, and environmental instability are already affecting morbidity and mortality worldwide. While research has predominantly focused on infectious, respiratory, and nutritional outcomes, emerging evidence suggests that climate change also impacts cardiovascular and hematologic health, including the risk of deep vein thrombosis (DVT). Understanding these links is critical for developing effective public health interventions and clinical management strategies [1]. Temperature extremes directly influence blood rheology and coagulation. High ambient temperatures promote dehydration, hemoconcentration, and increased blood viscosity, all of which enhance platelet aggregation and thrombin generation. Conversely, exposure to cold can lead to peripheral vasoconstriction, reduced venous return, and increased blood stasis, which are established risk factors for venous thromboembolism. Both heat and cold extremes, therefore, create physiologic conditions conducive to DVT [2-3].

Extreme weather events, such as heatwaves, floods, and cyclones, often result in prolonged immobility due to evacuation, sheltering, or injury. Immobilization is a well-known contributor to venous stasis, which facilitates thrombus formation in the deep veins of the lower extremities. Disaster-related trauma, stress, and disruptions in daily activity can further exacerbate hypercoagulability, compounding thrombotic risk [4]. Systemic inflammation plays a pivotal role in the development of climate-related thrombosis. Exposure to extreme weather, infections from disrupted sanitation, and physical trauma activate inflammatory pathways, leading to elevated cytokines such as interleukin-6 and tumor necrosis factor-alpha. These inflammatory mediators increase fibrinogen levels, stimulate platelet activity, and activate endothelial cells, creating a pro-thrombotic state [5]. Certain populations are particularly vulnerable to climate-induced DVT. Elderly individuals, pregnant women, patients with cardiovascular or hematologic disorders, children, and those with limited mobility or poor nutrition face heightened risk. Recognizing these groups is essential for targeted monitoring, preventive interventions, and timely clinical management, especially during climate-related disasters or prolonged exposure to extreme temperatures [6]. Biomarkers of hemoconcentration, coagulation, and inflammation—including hematocrit, platelet function, fibrinogen, D-dimer, and C-reactive protein—offer a

means of early detection and risk stratification. Integrating these biomarkers into clinical and disaster-response protocols enables timely intervention and reduces the likelihood of severe thrombotic events in high-risk populations [7-8].

AIM

The aim of this narrative review is to synthesize current evidence on how climate change influences the risk, incidence, and mechanisms of DVT. It seeks to elucidate the pathophysiologic pathways linking climate-related environmental exposures—such as extreme temperatures, air pollution, dehydration, infections, and population displacement—to thrombogenesis. Additionally, the review aims to highlight emerging epidemiological trends, identify knowledge gaps, and propose clinical and public health strategies to mitigate climate-associated thrombotic risks.

METHODS

This narrative review was conducted using a structured yet non-systematic approach to identify and synthesize current evidence on the relationship between climate change and DVT. Relevant literature was retrieved from major scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar, covering publications from 2000 to 2025. Search terms included combinations of "climate change," "heat waves," "air pollution," "temperature variability," "deep vein thrombosis," "venous thromboembolism," "hypercoagulability," and "environmental exposure." Both original research articles and review papers were considered. Additional sources, such as reports from the World Health Organization (WHO), Intergovernmental Panel on Climate Change (IPCC), and national health agencies, were used to provide context regarding climate trends and environmental health impacts. Studies not directly related to thrombosis or lacking relevance to climate-associated mechanisms were excluded. The selected literature was analyzed to identify recurring themes, mechanistic pathways, epidemiologic patterns, and clinical implications. Findings were narratively synthesized without quantitative meta-analysis, in line with the objectives of a narrative review. The review emphasizes conceptual integration and critical interpretation rather than statistical pooling.

Mechanisms Linking Climate Change to Deep Vein Thrombosis

The pathogenesis of DVT in the context of climate change is multifactorial, involving a complex interplay of environmental, physiological, and biochemical factors (Table 1) [9].

1. **Hemoconcentration and Dehydration:** Rising global temperatures and heatwaves increase insensible fluid loss through sweating and respiration. Inadequate hydration leads to hemoconcentration, characterized by increased hematocrit and plasma viscosity. Hyperviscous blood slows microvascular flow, elevates shear stress, and promotes platelet aggregation and thrombin generation, creating a pro-thrombotic environment [10].

2. **Immobility and Venous Stasis:** Extreme weather events, including floods and cyclones, often necessitate evacuation, sheltering, or prolonged bedrest. Immobility is a well-established risk factor for venous stasis, a key component of Virchow's triad. Reduced venous return allows accumulation of activated clotting factors, facilitating thrombus formation in the deep veins of the lower extremities [11].

3. **Inflammation and Infection:** Environmental stressors associated with climate change increase susceptibility to infections and tissue injury. Systemic inflammation, characterized by elevated cytokines such as IL-6 and TNF-α, enhances coagulation by upregulating tissue factor expression, increasing fibrinogen levels, and activating platelets. These processes collectively contribute to hypercoagulability and thrombosis risk [12-13].

4. **Endothelial Dysfunction:** Environmental extremes, hypoperfusion, and oxidative stress compromise endothelial integrity. Damaged or activated endothelial cells express adhesion molecules and release von Willebrand factor, promoting platelet adhesion and localized thrombus formation. Endothelial dysfunction, combined with hemoconcentration and stasis, amplifies the risk of DVT [14].

5. **Stress-Induced Hypercoagulability:** Psychological and physiological stress from climate-related events increases circulating catecholamines and cortisol, which modulate platelet function, endothelial activity, and clotting factor expression. This stress-induced hypercoagulability adds another layer of risk, particularly in individuals with pre-existing cardiovascular or hematologic disorders [15].

6. **Trauma and Environmental Injury:** Natural disasters, such as floods and cyclones, can cause blunt trauma, crush injuries, and lacerations. Tissue injury activates the coagulation cascade via tissue factor release and thrombin generation, further increasing thrombotic risk. Severe trauma may also trigger systemic inflammation, enhancing pro-coagulant activity [16-17].

Table 1: Mechanisms Linking Climate Change to Deep Vein Thrombosis (DVT)

Climate-Related Factor	Mechanistic Pathway	Impact on Thrombosis
Extreme Heat	Heat stress causes dehydration, hemoconcentration, increased blood viscosity, and reduced plasma volume.	Promotes venous stasis and hypercoagulability, increasing risk of DVT.
Air Pollution (e.g., PM2.5)	Inhaled particles trigger systemic inflammation, oxidative stress, endothelial dysfunction, and elevated cytokines (IL-6, TNF-α).	Enhances coagulation activation and platelet aggregation; increases DVT and VTE incidence.
Extreme Cold	Vasoconstriction increases blood pressure, platelet activation, and reduces fibrinolysis.	Facilitates thrombus formation, contributing to seasonal DVT peaks.
Wildfire Smoke Exposure	High particulate load increases oxidative stress, endothelial activation, and systemic inflammatory response.	Elevates short-term risk of DVT and PE during wildfire seasons.
Immobility During Climate Events	Heat waves, floods, storms, and evacuations restrict mobility and encourage prolonged sitting or sheltering.	Venous stasis increases DVT risk, similar to travel-related thrombosis.
Climate-Driven Infections	Climate change expands vector-borne and respiratory infections, many of which induce systemic inflammatory and hypercoagulable states.	Infection-mediated inflammation and coagulation activation elevate DVT risk.
Population Displacement	Migration due to storms, sea-level rise, or drought leads to immobility, dehydration, stress, and limited healthcare access.	Increases vulnerability to unrecognized or untreated DVT.

Vulnerable Populations

Not all individuals exposed to climate-related environmental stressors are equally susceptible to DVT. Physiological, demographic, and socio-economic factors combine to create higher risk in certain groups, making identification of vulnerable populations crucial for targeted prevention and early intervention [18].

1. **Elderly Individuals:** Aging is associated with reduced mobility, impaired thermoregulation, and a higher prevalence of cardiovascular disease. During heatwaves or disaster-related displacement, elderly individuals are particularly prone to dehydration, hemoconcentration, and venous stasis, increasing the likelihood of DVT [19].

2. **Pregnant Women:** Pregnancy is inherently a hypercoagulable state due to elevated fibrinogen and clotting factor levels, decreased fibrinolysis, and increased venous pressure. Environmental stressors such as heat, immobility, or limited access to care during climate events further elevate thrombotic risk, potentially compromising maternal and fetal outcomes [20].

3. **Individuals with Pre-existing Cardiovascular or Hematologic Disorders:** Patients with heart failure, coronary artery disease, polycythemia, or known coagulation disorders are at heightened risk. Climate-related dehydration, immobility, and inflammatory stress exacerbate their pro-thrombotic states, necessitating closer monitoring and preventive measures [21].

4. **Displaced or Shelter-Confined Populations:** Evacuees and those residing in temporary shelters often experience prolonged immobility, overcrowding, inadequate nutrition, and limited hydration. These factors promote stasis, hemoconcentration, and endothelial activation, all of which contribute to increased DVT risk [22].

5. **Children and Malnourished Individuals:** Nutritional deficiencies and immature or compromised immune systems make children and malnourished populations more susceptible to infection-induced inflammation, endothelial dysfunction, and coagulation abnormalities. Exposure to extreme temperatures or disaster conditions may further amplify these risks [22].

6. **Individuals with Limited Access to Healthcare:** Populations in low-resource settings or regions where healthcare infrastructure is disrupted face delayed diagnosis, limited access to prophylactic anticoagulation, and inadequate monitoring. This amplifies morbidity and mortality associated with DVT during climate-related events [23].

Potential Biomarkers and Monitoring

Early detection of individuals at risk for climate change-related DVT relies on careful monitoring of hematologic, coagulation, and inflammatory biomarkers. These markers help identify hypercoagulable states, hemoconcentration, and endothelial dysfunction, enabling timely intervention to prevent thrombotic events [24-25].

1. **Hematocrit and Hemoglobin:** Elevated hematocrit and hemoglobin levels indicate hemoconcentration, often resulting from dehydration due to heat stress or limited fluid access during extreme weather events. Monitoring these parameters allows clinicians to identify individuals at increased risk of DVT and implement hydration or other preventive measures [26-27].

2. **Platelet Count and Function:** Platelets are central to thrombus formation. Hemoconcentration increases platelet density, while environmental stress and inflammation can activate platelets, promoting aggregation. Functional assays, including platelet aggregation studies or flow cytometry for activation markers like P-selectin, can provide early warning of a pro-thrombotic state [28].

3. **Coagulation Factors:** Fibrinogen and von Willebrand factor are key indicators of hypercoagulability. Elevated fibrinogen increases blood viscosity and enhances clot stability, while von Willebrand factor promotes platelet adhesion and thrombus formation. Regular assessment of these factors can help detect individuals at heightened thrombotic risk [29].

4. **D-dimer and Thrombin Generation:** D-dimer is a sensitive marker of active fibrin formation and degradation, reflecting ongoing clot formation. Thrombin generation assays provide dynamic insight into coagulation activity. Both are valuable for detecting subclinical or emerging thrombosis in populations exposed to climate stressors [30].

5. **Inflammatory Biomarkers:** Cytokines such as IL-6, TNF-α, and acute-phase reactants like C-reactive protein (CRP) reflect systemic inflammation, which drives coagulation activation. Monitoring these markers is particularly relevant in post-disaster or extreme-weather contexts where infection, trauma, or stress-induced inflammation may elevate thrombotic risk [31].

6. **Endothelial Activation Markers:** Vascular endothelial health is critical in thrombosis prevention. Circulating markers such as soluble ICAM-1, VCAM-1, tissue factor, and endothelial microparticles indicate endothelial activation or injury, providing additional predictive information for DVT risk assessment [32].

7. **Monitoring Strategies:** In climate-affected populations, rapid and practical monitoring is essential. Point-of-care testing, portable hematology devices, and wearable biosensors can deliver real-time information on hematologic and coagulation status, particularly for vulnerable or displaced individuals. Combined with clinical assessment of hydration, mobility, and trauma exposure, these tools enable timely interventions and risk mitigation [33].

Preventive and Adaptive Strategies

Reducing the risk of climate change-related DVT requires a multifaceted approach that addresses individual health, clinical management, and broader public health measures. These strategies aim to minimize hemoconcentration, venous stasis, and hypercoagulability, particularly in vulnerable populations [34].

1. **Hydration and Electrolyte Management:** Maintaining adequate hydration is fundamental in preventing hemoconcentration and hyperviscosity. Populations exposed to heatwaves or limited water access benefit from oral rehydration solutions, electrolyte supplementation, and education on proper fluid intake, especially the elderly, children, and pregnant women [35].

2. **Mobilization and Physical Activity:** Prolonged immobility contributes significantly to venous stasis. Encouraging periodic leg exercises, walking, or movement within shelters or homes can improve venous return and reduce DVT risk. In disaster settings, designated activity areas or structured mobilization programs can be highly beneficial [36].

3. **Pharmacologic Prophylaxis:** High-risk individuals—such as those with pre-existing cardiovascular or hematologic disorders, or prolonged immobility—may benefit from anticoagulant or antiplatelet therapy under clinical supervision. Low-molecular-weight heparin or other thromboprophylactic agents can prevent thrombus formation but must be balanced against bleeding risk, particularly in trauma-exposed or pregnant populations [37].

4. **Shelter and Environmental Adaptation:** Proper shelter design can mitigate DVT risk by ensuring space for mobility, adequate ventilation, temperature control, and safe access to fluids. Overcrowding, extreme temperatures, and limited sanitation exacerbate risk factors for hypercoagulability and should be addressed in disaster preparedness planning [38].

5. **Public Health Education:** Awareness campaigns on hydration, movement, and early recognition of DVT symptoms (e.g., limb swelling, pain, or redness) empower communities to take preventive measures. Education targeting caregivers, shelter staff, and healthcare providers is particularly effective in high-risk settings [39].

6. **Monitoring and Early Detection:** Routine assessment of hematologic, coagulation, and inflammatory biomarkers allows for early identification of at-risk individuals. Point-of-care devices, portable testing kits, and wearable sensors can provide rapid, field-friendly monitoring in extreme weather or disaster-affected areas [40].

7. **Integrated Disaster and Climate Response:** Incorporating DVT risk mitigation into broader climate adaptation and disaster response frameworks ensures coordinated preventive efforts. Policies should address hydration infrastructure, mobility facilitation, pharmacologic access, and health surveillance to reduce thrombotic morbidity during climate-related events [41].

Future Directions

The emerging links between climate change and DVT highlight a critical need for further research and innovation in both clinical and public health domains. Addressing these gaps will improve understanding, prevention, and management of thrombotic events in climate-affected populations [42].

1. **Mechanistic Research:** Although hemoconcentration, immobilization, inflammation, endothelial dysfunction, and stress-induced hypercoagulability are recognized contributors, the precise molecular and cellular pathways remain incompletely understood. Future studies should investigate endothelial signaling, platelet activation, coagulation cascade modulation, and stress hormone interactions under climate-induced environmental stressors [43].

2. **Predictive Modeling and Risk Stratification:** Developing predictive algorithms that integrate environmental, demographic, physiologic, and behavioral factors could enhance early identification of individuals at risk for climate-related DVT. Such models can guide targeted monitoring, resource allocation, and prophylactic interventions, particularly during heatwaves, floods, or other extreme events [44].

3. **Innovative Monitoring Technologies:** Portable point-of-care devices, wearable biosensors, and telemedicine platforms offer opportunities for real-time assessment of hematologic, coagulation, and inflammatory parameters in vulnerable populations. Future research should validate and optimize these tools for use in extreme weather conditions and disaster scenarios [45-46].

4. **Optimized Therapeutic Strategies:** Research is needed to determine the efficacy, timing, and safety of pharmacologic prophylaxis in climate-affected populations. Studies should evaluate anticoagulant dosing protocols, risk-benefit balances, and integration with hydration and mobilization strategies, especially for elderly, pregnant, and comorbid patients [47].

5. **Public Health and Policy Integration:** Investigating the effectiveness of climate-adapted public health interventions—including hydration programs, shelter design, mobility protocols, and education campaigns—will inform evidence-based policies. Embedding DVT prevention into disaster preparedness and climate adaptation plans is essential for reducing morbidity and mortality [48].

6. **Climate-Resilient Healthcare Systems:** Multidisciplinary research bridging climatology, hematology, emergency medicine, and public health is required to build resilient healthcare strategies. Integrating thrombotic risk management into broader disaster and climate response frameworks will improve preparedness and outcomes in vulnerable populations [48].

CONCLUSION

Climate change is increasingly recognized as a significant contributor to DVT risk. Rising temperatures, extreme weather events, and environmental stressors promote hemoconcentration, venous stasis, systemic inflammation, endothelial dysfunction, and stress-induced hypercoagulability, all of which contribute to thrombus formation. Vulnerable populations—including the elderly, pregnant women, patients with cardiovascular or hematologic disorders, children, and displaced individuals—are particularly susceptible. Preventive and adaptive strategies, such as maintaining hydration, encouraging mobility, implementing pharmacologic prophylaxis where indicated, optimizing shelter environments, providing public health education, and monitoring relevant biomarkers, are essential for reducing DVT incidence and associated morbidity. Early identification of at-risk individuals enables timely interventions and improved outcomes. Future research should focus on elucidating mechanistic pathways, developing predictive models, validating innovative monitoring technologies, optimizing therapeutic strategies, and integrating thrombotic risk mitigation into public health and disaster preparedness frameworks. By addressing these priorities, healthcare systems and

communities can better anticipate, prevent, and manage DVT in the context of a changing climate. Understanding and mitigating climate-related thrombotic risks is therefore critical for protecting vulnerable populations and strengthening public health resilience.

Conflicts of Interest

The author declares no conflict of interest

Sources of Funding

No fund was received to write this review paper

Ethical Approval

Not applicable

Consent

Not applicable

REFERENCES

1. Ebi, K. L., Vanos, J., Baldwin, J. W., Bell, J. E., Hondula, D. M., Errett, N. A., Hayes, K., Reid, C. E., Saha, S., Spector, J., & Berry, P. (2021). Extreme weather and climate change: Population health and health system implications. *Annual Review of Public Health, 42*, 293–315. https://doi.org/10.1146/annurev-publhealth-012420-105026
2. Gao, Y., Liu, Y., He, J., Zhang, Y., Wang, T., Wu, L., Sun, N., Fang, T., Mao, H., Tang, N. J., & Chen, X. (2024). Effects of heat waves and cold spells on blood parameters: A cohort study of blood donors in Tianjin, China. *Environmental Health and Preventive Medicine, 29*, 25. https://doi.org/10.1265/ehpm.24-00023
3. Buono, M. J., Krippes, T., Kolkhorst, F. W., Williams, A. T., & Cabrales, P. (2016). Increases in core temperature counterbalance effects of haemoconcentration on blood viscosity during prolonged exercise in the heat. *Experimental Physiology, 101*(2), 332–342. https://doi.org/10.1113/EP085504
4. Obeagu, E. I., & Obeagu, G. U. (2024). Adapting to the shifting landscape: Implications of climate change for malaria control: A review. *Medicine, 103*(29), e39010. https://doi.org/10.1097/MD.0000000000039010
5. Dewi, S. P., Kasim, R., Sutarsa, I. N., & Dykgraaf, S. H. (2024). A scoping review of the impact of extreme weather events on health outcomes and healthcare utilization in rural and remote areas. *BMC Health Services Research, 24*(1), 1333. https://doi.org/10.1186/s12913-024-11695-5
6. Nahari, M. A., Ibrahim, H. S., Rambo, H. Y., & Alfa, T. A. (2026). Plant-Based Phytoremediation of Hydrocarbon-Contaminated Soils; A Review. *IOASD Journal of Medical and Pharmaceutical Sciences*, *3*(1), 7-11.
7. Obeagu, E. I. (2025). Climate change and red blood cell disorders: A review of risks and adaptations for pregnant women and children. *Medicine, 104*(40), e45129. https://doi.org/10.1097/MD.0000000000045129
8. Hong, L. Z., Shou, Z. X., Zheng, D. M., & Jin, X. (2021). The most important biomarker associated with coagulation and inflammation among COVID-19 patients. *Molecular and Cellular Biochemistry, 476*(7), 2877–2885. https://doi.org/10.1007/s11010-021-04122-4
9. Damnjanović, Z., Jovanović, M., & Stojanović, M. (2013). Correlation between the climatic factors and the pathogenesis of deep vein thrombosis. *Hippokratia, 17*(3), 203–206.
10. Rostomily, K. A., Jones, D. M., Pautz, C. M., Ito, D. W., & Buono, M. J. (2020). Haemoconcentration, not decreased blood temperature, increases blood viscosity during cold water immersion. *Diving and Hyperbaric Medicine, 50*(1), 24–27. https://doi.org/10.28920/dhm50.1.24-27
11. Ye, F., Bell, L. N., Mazza, J., Lee, A., & Yale, S. H. (2018). Variation in definitions of immobility in pharmacological thromboprophylaxis clinical trials in medical inpatients: A systematic review. *Clinical and Applied Thrombosis/Hemostasis, 24*(1), 13–21. https://doi.org/10.1177/1076029616677802
12. Chen, L., Deng, H., Cui, H., Fang, J., Zuo, Z., Deng, J., Li, Y., Wang, X., & Zhao, L. (2017). Inflammatory responses and inflammation-associated diseases in organs. *Oncotarget, 9*(6), 7204–7218. https://doi.org/10.18632/oncotarget.23208
13. Wang, X., & He, B. (2024). Endothelial dysfunction: Molecular mechanisms and clinical implications. *MedComm, 5*(8), e651. https://doi.org/10.1002/mco2.651
14. Obeagu, E. I. (2025). Stress-induced hemostasis: Mechanisms and implications for health. *Annals of Medicine and Surgery, 87*(6), 3300–3309. https://doi.org/10.1097/MS9.0000000000003012
15. Savioli, G., Ceresa, I. F., Caneva, L., Gerosa, S., & Ricevuti, G. (2021). Trauma-induced coagulopathy: Overview of an emerging medical problem from pathophysiology to outcomes. *Medicines, 8*(4), 16. https://doi.org/10.3390/medicines8040016
16. Obeagu, E. I., Isiko, I., & Obeagu, G. U. (2025). Climate change and HIV prevention: Towards sustainable solutions—A narrative review. *Medicine, 104*(16), e42198. https://doi.org/10.1097/MD.0000000000042198
17. Baccarelli, A., Martinelli, I., Zanobetti, A., Grillo, P., Hou, L. F., Bertazzi, P. A., Mannucci, P. M., & Schwartz, J. (2008). Exposure to particulate air pollution and risk of deep vein thrombosis. *Archives of Internal Medicine, 168*(9), 920–927. https://doi.org/10.1001/archinte.168.9.920
18. Millyard, A., Layden, J. D., Pyne, D. B., Edwards, A. M., & Bloxham, S. R. (2020). Impairments to thermoregulation in the elderly during heat exposure events. *Gerontology and Geriatric Medicine, 6*, 2333721420932432. https://doi.org/10.1177/2333721420932432

19. Varrias, D., Spanos, M., Kokkinidis, D. G., Zoumpourlis, P., & Kalaitzopoulos, D. R. (2023). Venous thromboembolism in pregnancy: Challenges and solutions. *Vascular Health and Risk Management, 19*, 469–484. https://doi.org/10.2147/VHRM.S404537
20. Hsieh, C. C., Kuo, F. H., & Liao, C. T. (2022). Heart failure and polycythemia: A case report and literature review. *Acta Cardiologica Sinica, 38*(4), 532–535. https://doi.org/10.6515/ACS.202207_38(4).20220221C
21. Obeagu, E. I., & Obeagu, G. U. (2024). Implications of climatic change on sickle cell anemia: A review. *Medicine, 103*(6), e37127. https://doi.org/10.1097/MD.0000000000037127
22. Puttarak, K., Angchaisuksiri, P., & Boonyawat, K. (2025). Delayed diagnosis and treatment of deep vein thrombosis—An underrecognized factor for its related outcomes? *Thrombosis Journal, 23*(1), 85. https://doi.org/10.1186/s12959-025-00769-x
23. Chang, Y., Lu, J., & Chen, C. (2025). Biomarkers in venous thrombosis: Diagnostic potential and limitations. *Biology, 14*(7), 800. https://doi.org/10.3390/biology14070800
24. Memon, A. A., Sundquist, K., PirouziFard, M., Elf, J. L., Strandberg, K., Svensson, P. J., Sundquist, J., & Zöller, B. (2018). Identification of novel diagnostic biomarkers for deep venous thrombosis. *British Journal of Haematology, 181*(3), 378–385. https://doi.org/10.1111/bjh.15206
25. Hjaouj, K., Ahmed, T. C., Mekkaoui, M., Elhafi, Z., Arkoubi, Z., Bencheikh, R., ... & Essakalli, L. (2026). A Parotid Mass Mimicking a Benign Tumor: Acinic Cell Carcinoma in an Adolescent. *IOASD Journal of Medical and Pharmaceutical Sciences*, *3*(1), 19-23.
26. Obeagu, E. I., & Bolo, B. (2025). Climate change and medical laboratory operations: Impacts, challenges, and adaptation strategies: A narrative review. *Medicine, 104*(23), e42718. https://doi.org/10.1097/MD.0000000000042718
27. Scridon, A. (2022). Platelets and their role in hemostasis and thrombosis—From physiology to pathophysiology and therapeutic implications. *International Journal of Molecular Sciences, 23*(21), 12772. https://doi.org/10.3390/ijms232112772
28. Palta, S., Saroa, R., & Palta, A. (2014). Overview of the coagulation system. *Indian Journal of Anaesthesia, 58*(5), 515–523. https://doi.org/10.4103/0019-5049.144643
29. Wang, H., Rosendaal, F. R., Cushman, M., & van Hylckama Vlieg, A. (2021). D-dimer, thrombin generation, and risk of a first venous thrombosis in the elderly. *Research and Practice in Thrombosis and Haemostasis, 5*(5), e12536. https://doi.org/10.1002/rth2.12536
30. Stec-Martyna, E., Wojtczak, K., Nowak, D., & Stawski, R. (2025). Battle of the biomarkers of systemic inflammation. *Biology, 14*(4), 438. https://doi.org/10.3390/biology14040438
31. Maneta, E., Aivalioti, E., Tual-Chalot, S., Emini Veseli, B., Gatsiou, A., Stamatelopoulos, K., & Stellos, K. (2023). Endothelial dysfunction and immunothrombosis in sepsis. *Frontiers in Immunology, 14*, 1144229. https://doi.org/10.3389/fimmu.2023.1144229
32. Lippi, G., Pighi, L., & Mattiuzzi, C. (2024). Update on patient self-testing with portable and wearable devices: Advantages and limitations. *Diagnostics, 14*(18), 2037. https://doi.org/10.3390/diagnostics14182037
33. Pradhan, S., Devi, W. A., & Bhutia, K. D. (2026). Effectiveness of warm water foot bath on fatigue among chronic renal failure patients undergoing hemodialysis in hospitals of Sikkim. *IOASD Journal of Medical and Pharmaceutical Sciences*, *3*(2), 45-50.
34. Sawka, M. N., & Montain, S. J. (2000). Fluid and electrolyte supplementation for exercise heat stress. *The American Journal of Clinical Nutrition, 72*(2 Suppl.), 564S–572S. https://doi.org/10.1093/ajcn/72.2.564S
35. Kunutsor, S. K., Mäkikallio, T. H., Seidu, S., de Araújo, C. G. S., Dey, R. S., Blom, A. W., & Laukkanen, J. A. (2020). Physical activity and risk of venous thromboembolism: Systematic review and meta-analysis of prospective cohort studies. *European Journal of Epidemiology, 35*(5), 431–442. https://doi.org/10.1007/s10654-019-00579-2
36. Alsuhebany, N., Alfehaid, L., Alodhaibi, D., Mahdali, M., Almohareb, S., & Alshaya, A. (2025). Venous thromboembolism prophylaxis practices in hematology/oncology patients admitted to neurological intensive care units. *Frontiers in Cardiovascular Medicine, 12*, 1573080. https://doi.org/10.3389/fcvm.2025.1573080
37. Conzatti, A., Kershaw, T., Copping, A., & Coley, D. (2022). A review of the impact of shelter design on the health of displaced populations. *Journal of International Humanitarian Action, 7*(1), 18. https://doi.org/10.1186/s41018-022-00123-0
38. Almodaimegh, H., Alfehaid, L., Alsuhebany, N., Bustami, R., Alharbi, S., Alkatheri, A., & Albekairy, A. (2017). Awareness of venous thromboembolism and thromboprophylaxis among hospitalized patients: A cross-sectional study. *Thrombosis Journal, 15*, 19. https://doi.org/10.1186/s12959-017-0144-2
39. Haghayegh, F., Norouziazad, A., Haghani, E., Feygin, A. A., Rahimi, R. H., Ghavamabadi, H. A., Sadighbayan, D., Madhoun, F., Papagelis, M., Felfeli, T., & Salahandish, R. (2024). Revolutionary point-of-care wearable diagnostics for early disease detection and biomarker discovery through intelligent technologies. *Advanced Science, 11*(36), e2400595. https://doi.org/10.1002/advs.202400595
40. Etongo, D., Bristol, U., Cetoupe, D., Landry, J., & Labrosse, J. C. (2025). Integrating disaster risk

reduction and climate change adaptation in Seychelles: Challenges and proposed strategies. *Jàmbá: Journal of Disaster Risk Studies, 17*(2), 1808. https://doi.org/10.4102/jamba.v17i2.1808

41. Excel, D. K., Chisom, O. A., Chinyere, E. T. D., Nwadikor, U. U., Nneka, O. I., & Ihuarulam, O. C. (2025). Microbial Analysis of Three-Leaf Yam (Dioscorea dumetorum): Microbiological Quality and Spoilage Dynamics During Storage. *IOASD Journal of Medical and Pharmaceutical Sciences*, *2*(4), 161-164.
42. Nnamdi, N. J., Achukwu, C. E., Eze-Onuegbu, N. P., Eluwa, C. G., & Obeagu, E. I. (2026). Factors Influencing the Acceptance of Immunization Among Mothers Who Attend Federal Medical Centre Umuahia, Abia State. *IOASD Journal of Medical and Pharmaceutical Sciences*, *3*(3), 94-103.
43. Xu, L., & Da, M. (2025). Incidence and risk factors of lower limb deep vein thrombosis in psychiatric inpatients by applying machine learning to electronic health records: A retrospective cohort study. *Clinical Epidemiology, 17*, 197–209. https://doi.org/10.2147/CLEP.S501062
44. Vo, D. K., & Trinh, K. T. L. (2024). Advances in wearable biosensors for healthcare: Current trends, applications, and future perspectives. *Biosensors, 14*(11), 560. https://doi.org/10.3390/bios14110560
45. Liao, Y., Thompson, C., Peterson, S., Mandrola, J., & Beg, M. S. (2019). The future of wearable technologies and remote monitoring in health care. *American Society of Clinical Oncology Educational Book, 39*, 115–121. https://doi.org/10.1200/EDBK_238919
46. Teichman, A. L., Cotton, B. A., Byrne, J., Dhillon, N. K., Berndtson, A. E., Price, M. A., Johns, T. J., Ley, E. J., Costantini, T., & Haut, E. R. (2023). Approaches for optimizing venous thromboembolism prevention in injured patients: Findings from the consensus conference to implement optimal venous thromboembolism prophylaxis in trauma. *Journal of Trauma and Acute Care Surgery, 94*(3), 469–478. https://doi.org/10.1097/TA.0000000000003854
47. Fox, M., Zuidema, C., Bauman, B., Burke, T., & Sheehan, M. (2019). Integrating public health into climate change policy and planning: State of practice update. *International Journal of Environmental Research and Public Health, 16*(18), 3232. https://doi.org/10.3390/ijerph16183232
48. Mosadeghrad, A. M., Isfahani, P., Eslambolchi, L., Zahmatkesh, M., & Afshari, M. (2023). Strategies to strengthen a climate-resilient health system: A scoping review. *Globalization and Health, 19*(1), 62. https://doi.org/10.1186/s12992-023-00965-2