

Medical Science

To Cite:

Ziółkowski J, Wagner-Bieleń NK, Zwierzchowska M, Antoniuk A, Jocz AM, Centkowska A, Banaśkiewicz JK, Żyta AM, Dąbrowska GH, Żmijewska MA. Venous thrombosis in athletes - a literature review. *Medical Science* 2025; 29: e171ms3708
doi: <https://doi.org/10.54905/disssi.v29i163.e171ms3708>

Authors' Affiliation:

¹Independent Public Clinical Hospital of Prof. W. Orłowski CMKP, ul. Czerniakowska 231, 00-416 Warsaw, Poland

²Military Institute of Medicine - National Research Institute, ul. Szaserów 128, 04-141 Warsaw, Poland

³Praski Hospital of the Transfiguration of the Lord, al. "Solidarności" 67, 03-401 Warsaw, Poland

⁴Specialist Provincial Hospital in Ciechanów, Powstańców Wielkopolskich 2, 06-400 Ciechanów, Poland

⁵Wolski Hospital of Dr. Anna Gostyńska, ul. Marcina Kasprzaka 17, 01-211 Warsaw, Poland

⁶Independent Public Health Care Complex - Hospital in Iłża, ul. Bodzentyńska 17, 27-100 Iłża, Poland

⁷The Infant Jesus Teaching Hospital, ul. Williama Heerleina Lindleya 4, 02-005 Warsaw, Poland

⁸Faculty of Medicine, Medical University of Warsaw, ul. Żwirki i Wigury 61, 02-091 Warsaw, Poland

*Corresponding author:

Jakub Ziółkowski,
Independent Public Clinical Hospital of Prof. W. Orłowski CMKP
ul. Czerniakowska 231, 00-416 Warsaw, Poland
E-mail: kuba.281299@gmail.com
ORCID: 0009-0008-0027-4315

Peer-Review History

Received: 05 May 2025

Reviewed & Revised: 16/May/2025 to 30/August/2025

Accepted: 09 September 2025

Published: 21 September 2025

Peer-review Method

External peer-review was done through double-blind method.

Medical Science

pISSN 2321-7359; eISSN 2321-7367



© The Author(s) 2025. Open Access. This article is licensed under a [Creative Commons Attribution License 4.0 \(CC BY 4.0\)](https://creativecommons.org/licenses/by/4.0/), which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/>.

Venous thrombosis in athletes - a literature review

Jakub Ziółkowski^{1*}, Natalia Katarzyna Wagner-Bieleń²,
Martyna Zwierzchowska¹, Agata Antoniuk³, Anna Maria Jocz⁴, Anna Centkowska⁵, Joanna Karina Banaśkiewicz⁶,
Aleksandra Maria Żyta¹, Gabriela Helena Dąbrowska⁷, Maria Anna Żmijewska⁸

ABSTRACT

Venous thromboembolism (VTE) is a health concern with serious morbidity and mortality. While athletes are perceived as healthy with a lower VTE incidence than general population, they are exposed to many unique risk factors. VTE in athletes presents unique diagnostic and therapeutic challenges, what impacts their ability to compete. This review brings together information to explore the understanding of VTE in athletes, focusing on its pathophysiology, diagnosis, management and screening. Sportsmen are exposed to sport-acquired risk factors and may have underlying genetic tendencies disrupting the hemostasis. Diagnosis can be difficult as symptoms mimic common sports injuries. Standard therapy prohibited playing contact sports while on anticoagulation. Invention of direct oral anticoagulants led to individualized, intermittent dosing strategies, which allows competing again while lowering bleeding risk. Patients with personal or family history should be considered for screening. Further research is necessary for treatment optimization strategies and better long-term outcomes. For this paper we performed a thorough search of literature by using databases: PubMed, Medline, and Google Scholar, where "Venous thrombosis in athletes" or "Hypercoagulability in athletes" appeared in the title, abstract, or keywords. Athletes, despite their professional physical shape and their overall good health, face specific risks of VTE, often connected to sports discipline. Keeping these factors in mind, being aware of potential misdiagnosis and its consequences, and the approach heavily relying on individualized treatment are crucial for managing VTE in this population and making a return to play possible, while minimizing risks.

Keywords: athletes, venous thromboembolism, deep vein thrombosis, pulmonary embolism, anticoagulation

1. INTRODUCTION

Venous thromboembolism (VTE) is a medical term which captures both deep vein thrombosis (DVT) and pulmonary embolism (PE). VTE is a important cause of mortality and morbidity through all around the globe. While it is common in the general population, the occurrence of this disease specifically in athletes has not

been extensively studied yet. Sportsmen population is typically younger and healthier than the general, what is naturally related to lower risk of VTE. However, they are repeatedly exposed to specific risk factors linked to training, competition, and athletic lifestyles. Some of these include injuries or surgeries associated with immobilization, frequent exposure to trauma or routine long-distance travels. Additional factors such as physical predispositions, dehydration, repeated intense professional exertion, and the use of certain substances will also affect VTE risk (Berkowitz & Moll, 2017).

When VTE occurs in athletes, it presents distinct challenges. So far athletes on anticoagulants typically were banned from participating in contact sports, due to the risk of injuries and bleeding. Mandatory pause from competing due to a health condition can be a significant psychological and financial detriment for athletes. Progress in medicine and healthcare quality, especially the development of direct oral anticoagulants (DOACs), provided new possibilities to manage VTE in athlete population, allowing for a reconsideration of previous mandatory removal policies. This review aims to consolidate information and to provide a comprehensive overview of venous thromboembolisms in athletes, addressing the interplay between their unique physiology, risk factors, diagnostic considerations, evolving treatment strategies, prevention, and screening (Morrison, 2007).

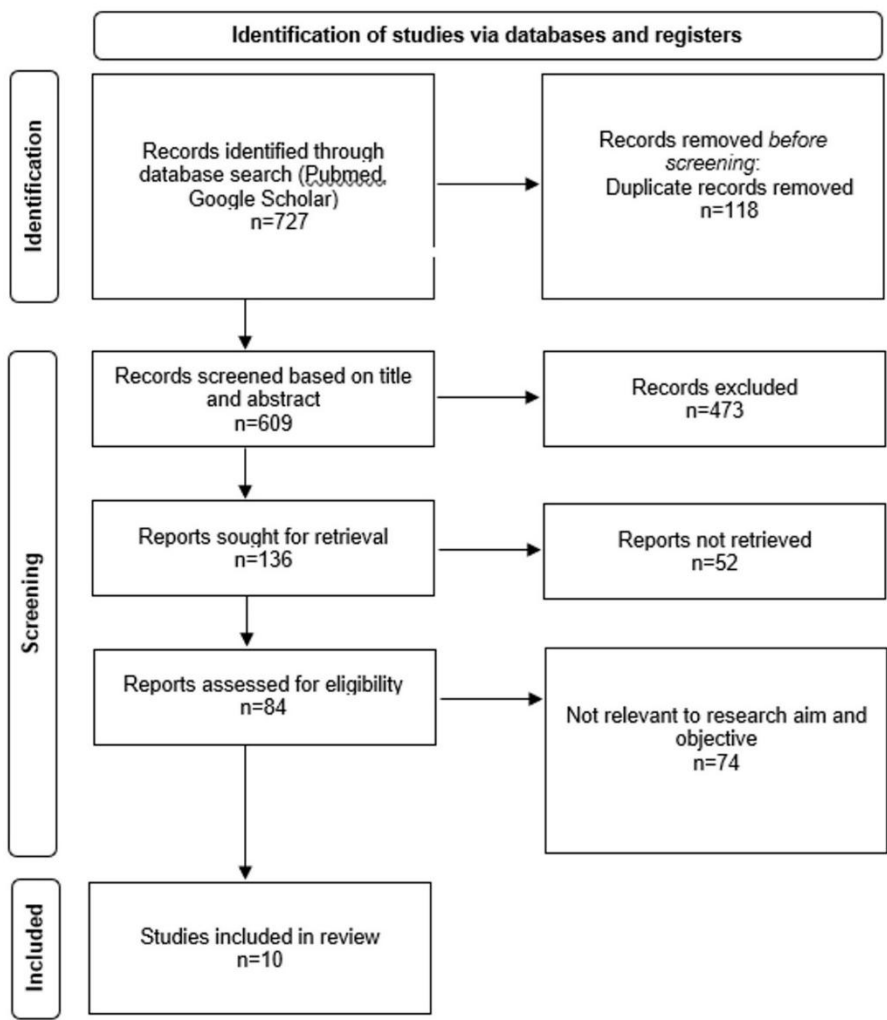


Figure 1. Consort chart

2. REVIEW METHODS

We went through a structured literature search performed in PubMed, Google Scholar, and Medline to identify studies on venous thrombosis in athletes. We browsed for keywords such as “venous thrombosis”, “hypercoagulability”, “deep vein thrombosis”,

“pulmonary embolism”, and “athletes”. Eligible sources consisted of reviews, meta-analyses and peer-reviewed clinical studies available in English that addressed VTE in terms of athletic population.

Inclusion was limited to peer-reviewed clinical or experimental studies, reviews, and meta-analyses in English that examined venous thromboembolism in athletes or sport-related thrombotic risk. We decided to exclude studies which focused solely on non-athlete populations, were lacking methodological detail or were available only as abstracts. Screening was made by sequential review of titles, abstracts, and full texts, with final selection agreed upon by all of the authors. The selection process followed a stepwise approach, as outlined in the CONSORT diagram (Figure 1).

3. RESULTS & DISCUSSION

Pathophysiology

The pathogenesis of venous thromboembolism is traditionally understood through Virchow's triad, which includes venous stasis, endothelial injury, and hypercoagulability. Athletes are exposed to factors that influence all three components of this triad (Menon et al., 2019). Venous stasis in athletes can be a result of each person's physical and lifestyle factors. Intensive training can lead to physiological cardiovascular remodeling resulting in bradycardia and hypotension, potentially causing blood pooling due to reduced flow rates and furthermore increasing the thrombotic risk. Compression of the venous system by hypertrophied muscles is another factor, such as compression of the popliteal vein by an enlarged gastrocnemius in cyclists or the axillary-subclavian vein by muscles in the thoracic outlet in overhead athletes (e.g. baseball, tennis, volleyball players).

Postural requirements of specific sports can also generate increased risk of stasis, including May-Thurner syndrome (iliac vein compression) seen in elite cyclists, which could be related to exceptionally prolonged hyperflexion of the hip. Despite being active, athletes experience periods of immobility during long-distance travel for competition, recovery from injury, or post-surgery, which are well-known risk factors for venous thrombosis (Adams et al., 2018).

Endothelial injury can be caused by direct collision in contact sports, surgery, or repetitive vascular trauma. Lower limb surgery, common after sports injuries, confers an increased risk of DVT. Injuries themselves, including muscle strains and sprains can result in a longer immobilization, a known factor of increased VTE risk. Microtrauma to vessel walls, caused by constantly repeated movement patterns (effort thrombosis), as seen in Paget-Schroetter syndrome in overhead athletes, leads to inflammation, fibrin deposition, and vessel narrowing. The risk of vascular trauma varies by sport, from non-contact to collision games, with higher-risk sports posing a greater potential for vessel injury and thrombotic incident (Grabowski et al., 2013).

Multiple factors influence hypercoagulability in athletes, including training and its intensity. Strenuous exercise has been shown to be a factor impacting hemostasis, with a temporary increase in blood coagulation, platelet aggregation, and fibrinolytic activity. The degree of the pro-coagulation mediators activation depends on the type, intensity, duration of the workout and individual training status. Exercise leads to increased platelet numbers and activity (intensity-dependent), elevated von Willebrand factor (vWF) and Factor VIII (FVIII) levels, and increased fibrinolytic potential (most evident after short-duration intense activity). Training may ameliorate some of these effects over time. Inadequate fluid intake during a workout may cause dehydration, leading to hemoconcentration, a relative decrease in plasma volume, which increases thrombotic risk. High altitude training may induce a transient increase in hemoglobin concentration and hematocrit, because of hypoxia-stimulated erythropoietin release. This results in increased blood viscosity and hypercoagulability. Oral contraceptive use in female athletes increases VTE risk in the general population and may further increase this risk in athletes, particularly in combination with genetic thrombophilias. Performance-enhancing substances like anabolic androgenic steroids (AASs) and erythropoietin (EPO) have been linked to increased hypercoagulability. AASs affect platelet aggregation and the levels of both plasma coagulation factors and natural anticoagulants, promoting a prothrombotic condition. There are also diurnal rhythms in coagulation and fibrinolysis markers, possibly influencing the risk of thrombotic incident, with a tendency towards hypercoagulability in the morning. The structure of gut microbiota and its metabolites (e.g., TMAO, PAGln) are more often linked to cardiovascular and thrombotic risk. Finally, some hereditary thrombophilias, such as Factor V Leiden mutation, Prothrombin G20210A mutation, or Antithrombin, Protein C, and Protein S deficiencies, even though they aren't more prevalent in athletes than in the general population, these conditions add significant risk when combined with athlete-specific acquired factors (Miele et al., 2024).

Diagnosis

Diagnosing VTE in athletes can be challenging because presenting symptoms such as pain, tenderness, and swelling may be mistaken for general training soreness or common sports injuries. Symptoms of lower extremity DVT (LEDVT) can include unilateral edema, tension, or pain in the calf, moderate fever, and a positive Homans sign. Upper extremity DVT (UEDVT) presents as acute upper limb edema, pain, discoloration, heaviness, and sometimes with dilated superficial collateral veins (Urschel's sign). Another diagnostic difficulties are PE symptoms: dyspnea, chest pain, palpitations, cough, asthenia, and dizziness. Because of their low specificity, they can be mistaken for a common response to exertion. Athletes' higher pain tolerance and potential aversion to reporting "illness" symptoms can also complicate timely diagnosis (Keller et al., 2020).

Diagnostic evaluation follows protocols similar to those of the general population. Venous duplex ultrasound is recommended as the first-line imaging test for DVT. If results are inconclusive and/or clinical suspicion still remains high, further imaging such as computed tomography, venography, or magnetic resonance angiography/venography may be necessary. When suspecting PE, a CT pulmonary angiogram is the diagnostic gold standard, guided by clinical probability scores such as Wells' criteria. D-dimer testing is a biomarker involved in diagnosis, although interpretation can be complex, as levels can be elevated by other conditions like infection, inflammation, or exercise itself. Other biomarkers and global coagulation assays (e.g., PT, aPTT, fibrinogen, thrombin generation assay, thromboelastometry) can provide insights into the hemostatic state but require careful interpretation in the context of recent exercise. The awareness of differences in the athletic population and a high suspicion towards thrombotic incidents are critical for early recognition and accurate diagnosis in patients presenting with unexplained symptoms (Meyering & Howard, 2004).

Treatment

Treatment of acute VTE in athletes generally aligns with principles of treatment in the general population. Anticoagulation is the cornerstone of therapy in terms of preventing thrombus extension, PE, and reducing the risk of post-thrombotic syndrome (PTS). Initial treatment involves low molecular weight heparin (LMWH) or unfractionated heparin, often bridged to an oral anticoagulant (Nazha et al., 2018).

The duration of anticoagulation depends on the nature of the VTE. A VTE resulted from a major, transient risk factor typically requires at least 3 months of uninterrupted anticoagulation. For unprovoked VTE or VTE provoked by persistent risk factors, extended (long-term) anticoagulation is usually considered after at least 3 months of full-dose therapy, involving a risk-benefit discussion regarding recurrent thrombosis versus bleeding risk. Lifetime therapy may be needed for recurrent VTE without pre-existing risk factors or specific high-risk inherited thrombophilias. During the initial 3 months of therapy, pause from competing in contact sports is recommended. Gentle, non-contact exercise may be safe after 4-6 weeks (Swan et al., 2020).

The emergence of direct oral anticoagulants has fundamentally changed treatment options due to their rapid onset and clearance in patient ("fast on/fast off") and additionally they've got predictable pharmacokinetics, and shorter half-lives compared to vitamin K antagonists (VKAs) like warfarin. DOACs (e.g. apixaban, rivaroxaban, dabigatran, edoxaban) are similarly as effective as than VKAs, but they are generally safer and easier to manage for VTE treatment. Individualized, intermittent anticoagulation strategies using DOACs are being proposed to allow athletes to return to play while minimizing bleeding risk during competition. This involves timing the drug dosing based on an individual athlete's pharmacokinetic/pharmacodynamic (PK/PD) profile to ensure minimal plasma drug concentration during competition and re-establishing therapeutic level quickly afterward.

PK/PD studies determine the drug's elimination half-life and the time it takes to reach a minimal plasma concentration, which is desirable in time of sport competition. Repetitive measurements may be beneficial due to intra-individual variability. Coming back to safe participation should result from considering current treatment, PK/PD profile, and personal activity. Basic strategies include skipping doses before high-risk activities and resuming quickly after. Warfarin is generally undesirable for in-season athletes, because of requirement to monitor the INR level and relatively long drugs half-life. LMWH offers a shorter half-life and predictable kinetics, potentially useful for intermittent dosing, but it involves injections.

Specific reversal agents are available for specific DOACs (idarucizumab for dabigatran, andexanet alfa for both apixaban and rivaroxaban) and for LMWH (protamine sulfate), which can be necessary for managing bleeding complications. Also, the prothrombin complex concentrate (PCC) can reverse the anti-Xa action of DOACs.

Thrombolysis (Catheter-Directed Thrombolysis - CDT) may be considered, particularly for proximal (iliofemoral or subclavian/axillary) DVT, to reduce the risk or severity of PTS. However, its role in the general population is debated. Its efficacy may be time-dependent.

We should keep in mind a surgical intervention as a possible treatment option in specific conditions, such as first rib resection, which may be required for VTE caused by anatomical compression in the thoracic outlet (Paget-Schroetter syndrome). This procedure aims to relieve compression and reduce the risk of recurrent VTE. Venous reconstruction may also be needed in some cases.

Patient autonomy and shared decision-making are most important for balancing safety with the athlete's desire to compete and potential career implications. Practical considerations should include interaction with performance enhancers, effects of athletic intensity on metabolism, potential heavy menstrual bleeding with DOACs, and use of NSAIDs. Athletes on anticoagulation should carry alert cards; also, special padding and protective equipment may be considered.

Prevention

Challenging the modifiable risk factors, targeting them and using specific measures in high-risk situations are paramount for preventing VTE in athletes. The correct connection between risk factors and management strategies (Figure 2) is critical for successful prevention.

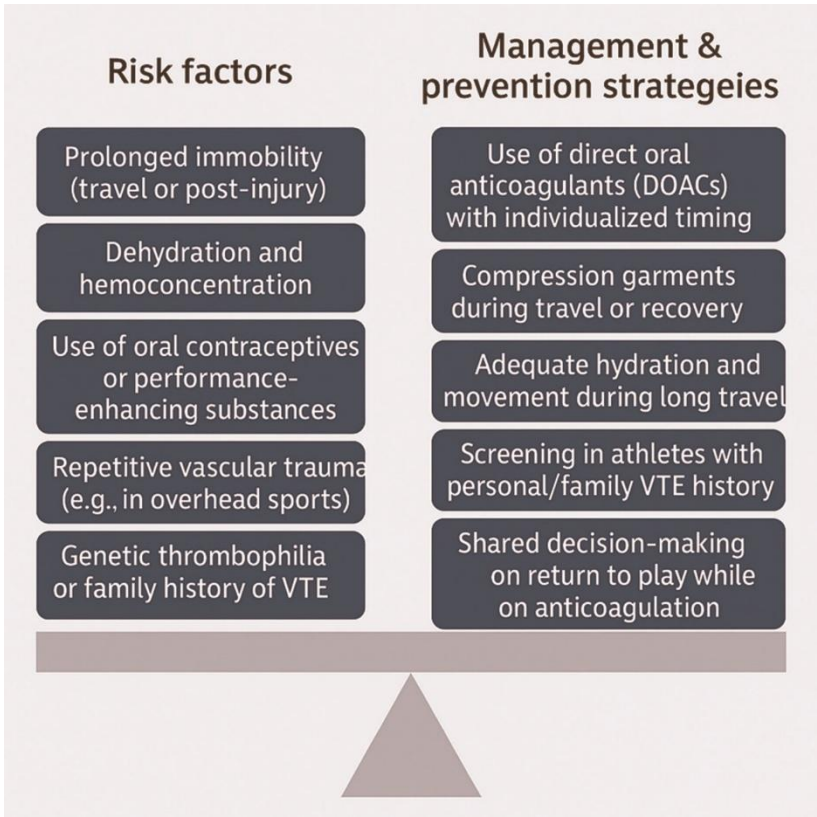


Figure 2. Risk and Management Considerations of Venous Thrombosis in Athletes

Generally, prevention comprises adequate hydration during exercise to avoid hemoconcentration, maintaining mobility (e.g., frequent short walks, leg exercises while seated, stretching) during long travel or flight, choosing a comfortable seating position, and wearing loose clothing to reduce venous stasis and thrombotic risk. Avoiding flying immediately after significant workout exertion may also be beneficial.

The use of compression garments/stockings is a preventative measure that has been shown to reduce the incidence of asymptomatic DVT during air travel in the general population and may be considered by athletes. However, definitive clinical benefit data in this specific population are limited. Compression garments can help improve venous blood flow rate and reduce venous pooling.

We should consider prophylactic anticoagulation in high-risk situations. For athletes with actually increased VTE risk (e.g., history of VTE, strong family history, known thrombophilia, flying for over 4 hours), prophylactic LMWH or potentially a DOAC dose may be discussed. Adequate thromboprophylaxis for an appropriate duration should be considered post-operatively, especially for lower limb procedures. Athletes who are immobilized for more than 3 days and for whom rapid mobilization is not anticipated should receive prophylactic anticoagulation in addition to physical measures.

Avoiding the use of performance-enhancing drugs and awareness of their thrombogenic risks is crucial in this population. Athletes engaging in high-altitude training should be monitored for early symptoms of VTE. Female athletes with thrombophilia should act carefully with oral contraceptive intake. For sports involving repetitive motion that can cause compression (like Paget-Schroetter), good posture, strengthening, and stretching exercises may help prevent compression and injury.

Screening

Screening for thrombotic risk factors in athletes is important, particularly in those with a suggestive history. Routine screening for hereditary thrombophilia in athletes without a significant personal or family history of VTE is generally not recommended.

Nevertheless, a thorough history should be taken routinely during preparticipation physicals and preoperative examinations. This should include questioning about:

- Personal history of VTE.
- Family history of VTE or clotting disorders.
- Known hypercoagulable disorders.
- Use of prothrombotic drugs such as oral contraceptives, hormone replacement therapy, or performance enhancers (EPO, anabolic steroids). Female athletes should also be questioned about current or recent pregnancy.
- Unusual training practices such as high-altitude training or simulation.
- History of specific sport-related trauma or repetitive motion injuries associated with conditions like Paget-Schroetter syndrome or popliteal vein entrapment.

For athletes with a positive personal or family history suggestive of hereditary thrombophilia, further evaluation is warranted. This typically involves screening for the most common hereditary thrombophilias:

- Factor V Leiden mutation.
- Prothrombin G20210A mutation (Factor II).
- Deficiencies of Antithrombin III, Protein C, and Protein S.
- Presence of antiphospholipid antibodies and homocysteine levels may also be checked.
- Sick cell trait is another risk factor to consider, particularly in African American athletes.

The incidence of Factor V Leiden and Prothrombin 20210A mutations is lower in non-Caucasian populations. When a thrombophilic disorder is found during diagnostics, we should thoroughly explain the risk to the athlete and implement early appropriate prevention and management strategies (e.g., including potential prophylactic anticoagulation in high-risk situations). Being aware of these conditions and their influence on athletic activity is essential for appropriate screening and further counseling.

4. CONCLUSION

Venous thromboembolism in the athlete population presents a challenging clinical problem due to unique risk factors associated with their professional training, exertion, and activities, as well as diagnostic difficulties stemming from the overlap of symptoms with common sports injuries and fatigue. Modern management strategies no longer rely on mandatory removal from participation, instead focusing on individualized, intermittent dosing of direct oral anticoagulants, which allows players to compete while minimizing the bleeding risk. Future research should prioritize standardized dosages and include clearly defined outcome measures. Until better evidence is available, physicians should focus on matching the right prevention or management strategies to corresponding risk factors. In consideration of a professional sports career, shared decision-making, patient autonomy, and balancing risks and benefits are crucial for achieving positive long-term outcomes.

Acknowledgments

The authors have no acknowledgments to disclose.

Author's Contribution

Conceptualization: Jakub Ziółkowski

Methodology: Agata Antoniak

Software: Joanna Karina Banaśkiewicz

Check: Anna Maria Jocz

Formal analysis: Garbiela Hanna Dąbrowska

Investigation: Martyna Zwierzchowska

Resources: Natalia Katarzyna Wagner-Bieleń

Data curation: Maria Anna Żmijewska

Writing-rough preparation: Aleksandra Maria Żyta

Writing-review and editing: Agata Antoniak

Supervision: Anna Centkowska

Project administration: Jakub Ziółkowski

All authors have read and agreed with the published version of the manuscript.

Informed consent

Not applicable.

Ethical approval

Not applicable.

Funding

This study has not received any external funding.

Conflict of interest

The authors declare that there is no conflict of interest.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES

1. Adams M, Kitic C, Wu S, Fell J, Zadow E. Acquired and genetic thrombotic risk factors in the athlete. *Semin Thromb Hemost* 2018;44(08):723-733. doi:10.1055/s-0038-1673625
2. Berkowitz JN, Moll S. Athletes and blood clots: individualized, intermittent anticoagulation management. *J Thromb Haemost* 2017;15(6):1051-1054. doi:10.1111/jth.13676
3. Grabowski G, Whiteside W, Kanwisher M, American Academy of Orthopaedic Surgeons. Venous thrombosis in athletes. *J Am Acad of Orthop Surg* 2013;21:108-117. doi:10.5435/JAAOS-21-02-108
4. Keller RE, Croswell DP, Medina GIS, Cheng TTW, Oh LS. Paget-Schroetter syndrome in athletes: a comprehensive and systematic review. *J Shoulder Elbow Surg* 2020;29(11):2417-2425. doi:10.1016/j.jse.2020.05.015
5. Menon D, Onida S, Davies AH. Overview of venous pathology related to repetitive vascular trauma in athletes. *J Vasc Surg Venous and Lym Dis* 2019;7(5):756-762. doi:10.1016/j.jvsv.2019.03.012
6. Meyering C, Howard T. Hypercoagulability in athletes. *Curr Sports Med Rep* 2004;3(2), 77-83. doi:10.1249/00149619-200404000-00005
7. Miele C, Mennitti C, Gentile A, Veneruso I, Scarano C, Vastola A, La Monica I, Uomo F, Iafusco F, Capasso F, Pero R, D'Argenio V, Lombardo B, Tinto N, Di Micco P, Scudiero O, Frisso G, Mazzaccara C. Thrombosis and Thrombotic Risk in Athletes. *J Clin Med* 2024;13,4881. doi:10.3390/jcm13164881
8. Morrison GC. Blood Clots and the Athlete: A Review of Deep Vein Thrombosis in Sports. *Athl Ther Today* 2007;12(4), 2-4. doi:10.1123/ATT.12.4.2
9. Nazha B, Pandya B, Spyropoulos A, Kessler C. Treatment of venous thromboembolism in Elite Athletes: A Suggested approach to Individualized Anticoagulation. *Semin Thromb Hemost* 2018;44(08):813-822. doi:10.1055/s-0038-1673690
10. Swan D, Carter-Brzezinski L, Thachil J. Management of venous thromboembolism in athletes. *Blood Rev* 2020;47:100780. doi:10.1016/j.blre.2020.100780