Position Statement

Position statement of the FAC Committee on Peripheral Vascular Disease and Stroke on prevention of deep vein thrombosis in prolonged trips.

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ABSTRACT

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are the most common manifestations of venous thromboembolism (VTE). According to published data, VTE affects nearly 10 million people worldwide each year, contributing significantly to the global burden of disease. The pathophysiology of thrombosis is explained through Virchow's triad: presence of blood stasis, damage or dysfunction of the vascular wall, and hypercoagulability. Risk factors for developing DVT include prolonged travel, defined as trips lasting more than 4 hours, which is why VTE is often referred to as "economy class syndrome" or "traveler's thrombosis". However, it is worth noting that travel-related DVT is even more closely linked to people who have other risk factors for developing venous thrombosis. Thus, these individuals are grouped into low, medium/moderate and high risk, with this categorization being very useful when deciding on the best therapeutic option. Given the high morbidity and mortality associated with DVT cases and the resulting PE, related to prolonged immobilization, which is very common in contemporary times during long journeys, a series of measures and recommendations have been proposed to prevent and reduce the risk of developing VTE.

INTRODUCTION

Most DVT affect the veins in the inferior limbs or the pelvis (70-80%). As the symptoms of DVT are unspecific, it is essential to make a fast and standard diagnosis to minimize the risk of PE in the acute phase, and prevent the progression of thrombosis, post-thrombotic syndrome and recurrence of VTE in the long term.[1]

VTE, affecting almost 10 million people of all ethnicities throughout the world and every year, significantly contributes to the world load of morbidity. The yearly incidence of acute venous thromboembolism is 1 to 2 cases per 1000 inhabitants; a figure that increases exponentially with age both for men and women, and is 4 times greater in countries with high income than in those with low income. The risk of VTE over life does not differ according to sex, but women have a higher risk between 20 and 40 years of age, reflecting their exposition to risk factors during reproductive age, while men have a higher risk in other age groups.[2]

Known risk factors for VTE include immobilization, recent surgery, pregnancy, use of oral contraception, obesity, trauma, malignant neoplasia and hereditary thrombophilia. It has been proven that there is an association between long-duration trips (more than 4 hours) and the development of deep venous thrombosis, because of prolonged immobilization.[3]

Taking into account the circumstances and triggering factors that contribute to the development of DVT, in this position statement we present management strategies to prevent the development of DVT in people who take long trips.

PATHOPHYSIOLOGY OF THROMBOSIS

Deep vein thrombosis (DVT) is a disease in which a blood clot develops in the veins; blood vessels that carry blood back to the heart. Venous thromboembolism is a multicausal disease. It is believed to be triggered by interactions between multiple causal factors that could add to each other or be synergic. Some clinical risk factors are stronger and may cause venous thromboembolism in the absence of other risk factors. These causal factors can be transient or persistent. Transient risk factors, such as major surgery, extended immobilization and major trauma, represent approximately 20% of all episodes of venous thromboembolism.[2]

The pathophysiological mechanisms that lead to thrombosis are traditionally explained by Virchow's triad: blood stasis, vas-

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cular wall damage or dysfunction, and hypercoagulability. It is believed that venous thrombi start in the valve pockets of large veins, which are susceptible to blood stasis, particularly during extended immobilization. Stasis and vascular wall damage or dysfunction may lead to hypoxia or inflammation, processes that regulate negatively the natural anticoagulant properties of the endothelium and, therefore, induce a state of hypercoagulability. The activation of procoagulant venous endothelial cells leads to a greater expression of molecules of superficial adhesion, such as P-selectin and von Willebrand factor, which facilitate the subsequent binding of leukocytes, microparticles and circulating platelets. The activated leukocytes express the procoagulant tissue factor, which is believed to initiate the extrinsic pathway of coagulation cascade (the intrinsic pathway, contact pathway, activated through factors XII and XI), leading to factor X activation, production of thrombin, and ending in the formation of thrombi that include fibrin, red blood cells and platelets.[2]

Recent evidence suggests a complex interaction between coagulation and inflammation, by which the coagulation cascade activation triggers the immunology system and, in turn, innate immune cells contribute to the formation of thrombi, in a process called immunothrombosis. In fact, drugs with anti-inflammatory properties, such as statins, may reduce incident and recurrent venous thromboembolism. Activated leukocytes are the main source of positive procoagulant microparticles for tissue factor, which may stimulate the formation and growth of thrombi. Neutrophils, the most abundant leukocytes, produce neutrophil extracellular traps (NET), that are composed by DNA, histones and antimicrobial proteins. NETs provide a structure for red blood cells, platelets and procoagulant molecules to promote thrombi formation. The concentrations of microparticles positive for circulating tissue factor and NET markers (e.g.: extracellular DNA) increase in patients with venous thromboembolism, but their use as biomarkers for VTE has not been established yet.[2]

Platelets help to induce the formation of NET and increase procoagulant activity in innate immune cells. This could be the mechanism by which aspirin reduces the risk of incident and recurrent venous thromboembolism. The genetic contribution to the risk of venous thromboembolism is not completely characterized. Traditional hereditary thrombophilia, included protein C, protein S and antithrombin deficiency, the gene mutation of prothrombin and factor V Leiden, is associated to genes of the coagulation system. However, a meta-analysis of studies of association of the whole genome identified several genes associated with inflammation, red blood cells and platelets, that were also associated with the risk of venous thromboembolism.2

RELATIONSHIP WITH PROLONGED TRIPS

Venous thromboembolism (VTE) is an ailment that has also been known as the "economy class syndrome" or "traveler's thrombosis" due to its incidence in individuals who have underwent a long period of traveling (more than 4 hours), whether by plane, train, car, truck or bus, within the 8 weeks before diagnosis.[4]

In year 1944, professor John Homans described the sign that carries its name, which is characterized by presenting pain in the calf muscles when performing foot dorsiflexion. The appearance of DVT after traveling by plane was first reported by John Homans himself, who published the relationship between the development of venous thrombosis and immobilization due to long trips in 1954. Initially, it was called the "economy class syndrome", as it was considered that the risk of presenting DVT increased by the narrow or compact configuration of the economy class seats. Later, PE occurred in business class seats, which include additional space for the legs. This suggested that a reduced movement of passengers in long distance flights is the most important determinant factor. However, a significant point is that DVT associated to trips is even more related with people with other risk factors to develop venous thrombosis.[5]

It has been proposed to group these individuals into people with low, medium and high risk of suffering traveler's thrombosis, in such a way that people in high risk are those with personal history of VTE, neoplasia or recent major surgery; people with low to medium risk of VTE have a risk of 0-2% of developing DVT associated with long distance trips. This increases to 5% for people with high risk, as those with history of DVT or states of hypercoagulability, including factor V Leiden mutation and obesity.[6] Another specific factor that may increase the risk of trips is pregnancy. Pregnancy and puerperium are periods known to be of high risk of VTE. An estimated risk of 0.03%-0.1% has been suggested for this population, and is included within the group of medium risk travelers.[6]

Several definitions of long-distance or prolonged trips are used. According to evidence, these vary from trips of more than 4 hours to more than 8 hours. A dose-response or exposure-response relationship has been proven, in the sense that for every 2 hours of additional flight trip, it is estimated that there is a 26% increase in the risk of VTE. Although the risk of VTE is greater during the first week after the trip, the risk can remain elevated for up to 8 weeks after a single long-distance flight. The risk of VTE related to traveling is based, in part, to a passanger's profile.[7] A meta-analysis found an 18% increase in risk for every 2 hours of increase in the duration of trips by any means (car, truck, bus, train), with a higher increase, 26%, associated to flights (due to the hypobaric environment that may induce hypercoagulability).[8] Furthermore, the more flights made, the greater the risk, which generates a greater risk for frequent travelers or business travelers. The risk tripled after five or more long-distance trips in a study, and each additional flight increase the risk by 1.4 times.[9]

DVT PREVENTION STRATEGIES

Because of the known risk of developing DVT and PE as a consequence of immobilization during prolonged periods of time related to trips, several strategies have been proposed to prevent and decrease risk. The recommendation of such strategies will depend on the individual risk of presenting VTE (low, medium or high risk), considering mechanical methods and other that include performing physical movements (non-pharmacological methods), besides pharmacological methods.

As non-pharmacological measures, using compression socks during long distance flights, which may help to reduce the risk of developing DVT, as well as moving the lower limbs, standing up or walking occasionally during the flights, and appropriate hydration. Pharmacological methods encompass from a preventive use of aspirin to low doses of low-molecular-weight heparin.[10]

Several scientific trials have researched these methods to reduce the risk of VTE. The LONFLIT-2 and 5 trials explored the use of compression socks, showing a statistically significant difference between the control group and those using socks, 4.5% and 5.8% of controls developed DVT in the LONFLIT-2 and 5, respectively, in comparison with 0.24% and 0.97% of those using compression socks.[11,12] Current reviews coincide in that there is evidence that compression socks reduce the risk of VTE related to trips, and moreover, help reduce lower limb edema in low risk people.[6]

The pharmacological prophylaxis of DVT has been tested, including using aspirin and low-molecular-weight heparin (LMWH). So much so, that the LONFLIT-3 study showed that aspirin reduced DVT rates significantly; however, the risk was abolished in the group treated with LMWH.[13] Another study evaluated using pirokinase in 150 mg doses (a fibrinolytic agent of oral administration) in individuals in high risk, showing that no thrombotic events occurred in the treated group in comparison with the control group, who presented DVT in 5.4% and a lower number of cases with superficial venous thrombosis (SVT).[14]

It is necessary to consider the potential use of new oral anticoagulants, such as apixaban. Their short half-life, its fast onset of action and its oral administration turns them into an attractive option for prophylaxis of DVT in high risk travelers.

However, trials are necessary to examine their safety and efficacy in the prevention of travelers' thrombosis.[7]

The function of thromboprophylaxis with LMWH during pregnancy varies and will depend on the threshold of risk of VTE used during pregnancy exceeds 1 or 3%, the more recent guidelines recommend a risk threshold of 2% during pregnancy and a risk threshold of 1% in the postpartum period to guide the use of thromboprophylaxis.[8]

Performing exercises with the lower limbs during the trip decreases the risk of developing VTE. These exercises include: feet movements with active contraction of feet and calves muscles (activating the muscle pump), feet exercises against moderate resistance (plantar flexion and active dorsiflexion made against a flat, non-rotating board), feet movements against a higher resistance using a rotating pedal connected to a resistance band. All these exercises proved to provide a maximum contraction of the feet and calves muscles, thus activating the muscle pump while in a sitting position; so much so, that in a series of cases it was proven that the flow of blood volume in the popliteal vein was reduced in almost 40% with individuals sitting and immobilized, and nearly twice that when sitting, immobilized and with their feet not touching the ground. Feet exercise against a greater resistance positively improved the flow of venous blood volume.[15]

Therefore, and in brief, this committee recommends:

- Doing, whenever possible, short trips, of less than 4 hours.
- If long and uninterrupted trips are made in transportation means (bus, car, plane, and so on), increase the seat tilt as much as possible, during a long journey.
- Moving before the fourth consecutive hour of an uninterrupted trip, whether within the transportation means (bus, train, plane) or if traveling by car, stopping it (in a safe place) and move outside of it.
- Ingest abundant water to ensure proper hydration.
- Use loose clothes during and after the trip.
- During the immobilization period, perform regular exercises, at least every hour, extending both legs and moving both feet forward and backward in a circular motion; besides placing the feet on the ground and pointing the fingers upward (activating the calves muscle pump).
- For people with inherited or acquired prothrombotic disease, or with risk factors, reinforce or enhance the previous recommendations (that is to say, do not remain sitting in a car for more than 2 uninterrupted hours), use compression socks during and immediately after the trip, do not forget to take anticoagulation medication (in patients already in treatment).
- Consider using LMWH according to the thrombotic risk of each individual.
- In any case, consult a physician if signs or symptoms of VTE appear, within 4 to 8 weeks after a long trip and prolonged immobilization.

CONCLUSIONS

Given the proven relationship between the incidence of venous thromboembolism and prolonged trips, there is a quite reasonable weight to support effective measures to prevent thrombosis related to long-distance trips, also taking into account the fact that most travelers, even flight passengers, have a deficient knowledge of the thrombotic risk associated to long-distance trips/flights.

BIBLIOGRAPHY

1. Arriba AM, Gallardo LS, Günther RM, et al. Natalidad y Mortalidad 2022, sín- Birgit Linnemann, Beyer-Westendorf J, Espinola-Klein C, et al. Management of Deep Vein Thrombosis: An Update Based on the Revised AWMF S2k Guideline. Hamostaseologie **2024**; 44: 97 - 110.
2. Khan F, Tritschler T, Kan SR, et al. Venous thromboembolism. Lancet **2021**; 398: 64 - 77.
3. McKerrow Johnson I, Shatzel J, Olson S, et Al. Travel-Associated Venous Thromboembolism. Wilderness Environ Med **2022**; 33: 169 - 178.
4. G. Lippi G, Favaloro EJ. Car Travel-Related Thrombosis: Fact or Fiction? Semin Thromb Hemost **2018**; 44: 327 - 333.
5. Byard TW. Deep venous thrombosis, pulmonary embolism and long-distance flights. Forensic Sci Med Pathol **2019**; 15: 122 - 124.
6. Schobersberger W, Toff WD, Eklof B, et al. Traveller's thrombosis: International consensus statement. Vasa **2008**; 37: 311 - 317.
7. Karsanji DJ, Bates SM, Skeith L. The risk and prevention of venous thromboembolism in the pregnant traveller. J Travel Med **2020**; 21: taz091.
8. Chandra D, Parisini E, Mozaffarian D. Meta-analysis: travel and risk for venous thromboembolism. Ann Intern Med **2009**; 151: 180 - 190.
9. Kuipers S, Cannegieter SC, Middeldorp S, et al. The absolute risk of venous thrombosis after air travel: a cohort study of 8,755 employees of international organisations. PLoS Med **2007**; 4: e290.

10. Clarke MJ, Broderick C, Hopewell S, et al. Compression stockings for preventing deep vein thrombosis in airline passengers. Cochrane Database Syst Rev **2021**; 4 CD004002.
11. Belcaro G, Geroulakos G, Nicolaides AN, et al. Venous thromboembolism from air travel: the LONFLIT study. Angiology **2001**; 52: 369 - 374.
12. Belcaro G, Cesarone MR, Nicolaides AN, et al. Prevention of venous thrombosis with elastic stockings during long-haul flights: the LONFLIT 5 JAP study. Clin Appl Thromb Hemost **2003**; 9: 197 - 201.
13. Cesarone MR, Belcaro G, Nicolaides AN, et al. Venous thrombosis from air travel: the LONFLIT3 study-prevention with aspirin vs low-molecular-weight heparin (LMWH) in high-risk subjects: a randomized trial. Angiology **2002**; 53: 1 - 6.
14. Cesarone M, Belcaro G, Nicolaides AN, et al. Prevention of venous thrombosis in long-haul flights with Flite Tabs: the LONFLIT-FLITE randomized, controlled trial. Angiology **2003**; 54: 531 -539.
15. Hitos K, Cannon M, Cannon S, et al. Effect of leg exercises on popliteal venous blood flow during prolonged immobility of seated subjects: implications for prevention of travel-related deep vein thrombosis. J Thromb Haemost **2007**; 5: 1890 - 1895.