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Venous pathology in athletes
Patologia venoasă la sportivi

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Abstract

Sports activities, at any level, cause great physical stress to the athlete's body. Musculoskeletal injuries are the most common cause of pain and functional impotence in athletes. However, vascular etiology should not be neglected and it should be considered in an athlete with persistent symptoms, refractory to conventional therapy for musculoskeletal pains.

The diagnosis of vascular disease may be overlooked in athletes because they are young, healthy and musculoskeletal impairment may present similar symptoms and signs and seems the most plausible explanation for these.

In this paper, we focused on venous pathology in athletes: deep vein thrombosis and its complications: pulmonary embolism and post-thrombotic syndrome. We reviewed the main risk factors and preventive measures that may be taken; we insisted on warning symptoms and signs that should be considered by the physician and the means of diagnosis; we mentioned the treatment and recovery means for the resumption of sports activities.

Venous pathology in athletes is an important health problem because a delay in diagnosis may have serious consequences for the affected limb, their subsequent sports activity or it may even endanger the athlete's life.

Keywords: deep vein thrombosis, pulmonary embolism, post-thrombotic syndrome, risk factors of deep vein thrombosis in athletes.

Rezumat

Activităţile sportive, la orice nivel, supun organismul sportivului la o mare solicitare fizică. Leziunile musculoscheletale reprezintă cea mai frecventă cauză de durere şi impotenţă funcţională la sportivi. Cu toate acestea, etiologia vasculară nu trebuie neglijată şi trebuie luată în considerare la un sportiv cu simptome persistente, refractare la terapia uzuală pentru suferinţele musculoscheletale.

Diagnosticul de afectare vasculară poate fi trecut cu vederea la sportivi deoarece aceştia sunt tineri, sănătoşi, iar afectarea musculoscheletală poate prezenta simptome şi semne asemănătoare şi pare cea mai plauzibilă explicaţie pentru acestea.

În lucrarea de faţă ne-am oprit asupra patologiei venoase la sportivi: tromboza venoasă profundă şi complicaţiile acesteia, embolia pulmonară şi sindromul posttrombotic. Am făcut o trecere în revistă a principalilor factori de risc şi a măsurilor profilactice ce pot fi luate; am insistat asupra simptomelor şi semnelor de alarmă care trebuie luate în considerare de către medic şi asupra mijloacelor de diagnostic; am precizat tratamentul şi mijloacele de recuperare în vederea reluării activităţii sportive.

Patologia venoasă la sportivi reprezintă o importantă problemă de sănătate, deoarece întârzierea diagnosticului poate avea consecinţe grave asupra membrului afectat, asupra activităţii sportive ulterioare sau chiar poate pune în pericol viaţa sportivului.

Cuvinte cheie: tromboza venoasă profundă la sportivi, embolie pulmonară, sindrom posttrombotic, factori de risc ai trombozei venoase la sportivi.

Introduction

Sports activities of any kind submit athletes to a great physical strain. Many times, their movements and postures push the limits of human anatomy and physiology.

Musculoskeletal lesions are the main cause of pain or functional impairment in athletes. Yet, similar symptoms may also appear in the case of vascular damage. Vascular damage should be suspected in any athlete who presents the following symptoms: pain, paresthesia, early muscular fatigue, oedema, skin discoloration, especially if they are resistant to the usual therapy for musculoskeletal ailments. A thorough medical history check must be carried out for these, as well as a complete clinical exam, which should include specific triggering maneuvers (reproducing the movements specific to the sports type), for a quick and correct diagnosis (Perlowski & Jaff, 2010).

Unfortunately, vascular damage can be overlooked in athletes, because these are young, healthy persons, in whom vascular disease is less likely to exist and a musculoskeletal

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cause to explain the symptoms is more plausible. A delay in diagnosis may have severe consequences on the damaged limb, on the future sports activity and may even threaten the athlete's life (Holzheimer & Stautner-Brückmann, 2008; Perlowski & Jaff, 2010; Hull & Harris, 2013).

In this paper, we focused on venous pathology in athletes: deep venous thrombosis and its complications, pulmonary embolism and post-thrombotic syndrome. We reviewed the main risk factors and prevention measures that can be taken; we focused on the symptoms and alarm signals that need to be considered by the physician, as well as on diagnostic means; we mentioned the treatment and recovery means for the resumption of sports activities.

Deep vein thrombosis

In 1884, Virchow grouped the factors favorable to DVT (deep vein thrombosis) in a triad, which consists of: blood hypercoagulability, venous stasis and vascular wall lesions.

Each of these factors may appear in an athlete, alone or combined, in connection to the athlete's activity. The specific risk factors of vein thrombosis in athletes are:

Factors that increase blood coagulability: dehydration and haemoconcentration (triggered by an insufficient intake of liquids during effort), venous wall inflammation, use of estrogen contraceptives during training or competitions, or use of anabolic steroids (Andersen & Spencer, 2003; Bates & Ginsberg, 2004; Goldhaber & Fanikos, 2004; Hull & Harris, 2013; Grabowski et al., 2013; Burrus et al., 2014).

Factors that favor venous stasis: prolonged immobilisation on planes or buses on the way to sports events, bradycardia and low blood pressure (specific to athletes), congenital musculoskeletal malformations (thoracic outlet syndrome that could trigger Paget-Schröetter syndrome) or congenital venous malformations (May-Turner syndrome, narrowing or absence of the inferior vena cava) (Hull & Harris, 2013; Andersen & Spencer, 2003; Goldhaber & Fanikos, 2004; Grabowski et al., 2013; Burrus et al., 2014).

Factors that favor venous wall damage: orthopedic surgery and repeated trauma and microtrauma (Andersen & Spencer, 2003; Goldhaber & Fanikos, 2004; Hull & Harris, 2013; Grabowski et al., 2013).

Thoracic outlet syndrome

Thoracic outlet syndrome (TOS) is a neurovascular compression syndrome of the upper limb. The thoracic duct is delimited by the scalene triangle (composed by the anterior, middle and posterior scalene muscles), the costoclavicular space (between the clavicle and the first rib), and the subcoracoid space (between the pectoral muscle and the coracoid process). At this level the brachial plexus, the subclavian vein and the subclavian artery pass. If nervous compression prevails, neurogenic TOS occurs (90% of cases). If vascular (arterial or venous) compression prevails, vasculogenic TOS appears (3-5% of cases). Vascular compression mostly occurs in the scalene triangle. TOS rarely develops in athletes, but when it does, it can have devastating effects (DiFelice et al., 2002; Laker et al., 2009).

Paget-Schröetter syndrome

Paget-Schröetter syndrome (PSS) is one of the most frequent manifestations of TOC and it represents the spontaneous effort-induced thrombosis of the axillary or subclavian vein. The syndrome was described in 1875 by Paget and in 1884 by Schröetter. It is the most frequent vascular damage in young athletes. As a severe and possibly lethal complication, pulmonary embolism may occur in 36% of the patients, therefore a quick diagnosis is vital. If correctly diagnosed and treated, it allows the prevention of pulmonary embolism and post-thrombotic syndrome, the athlete being able to resume sports activity within several months (Melby et al., 2008; Laker et al., 2009; Perlowski & Jaff, 2010).

PSS occurs mostly in athletes who perform repetitive upper limb overhead motions (basketball, baseball, volleyball, handball, football, tennis, swimming, weightlifting, gymnastics, wrestling, golf, hockey, rowing), through repeated compression of the axillary or subclavian vein. Compression can be localised at the scalene triangle level, through the presence of the cervical rib (0.5-1.5%), between the clavicle and the first rib, or it may be due to specific muscular hypertrophy (swimmers – the small pectoral muscle; weightlifters – the scalene muscle; pitchers, tennis players – the dominant limb muscle) (DiFelice et al., 2002; Treat et al., 2004; Laker et al., 2009, Perlowski & Jaff, 2010).

The conditions of PSS occur in two stages:

1. The stenosis stage: repeated trauma to the vein leads to inflammation, fibrin deposits, progressive narrowing and development of collateral circulation; for this reason, patients are asymptomatic in this stage (Thompson & Driskill, 2008; Perlowski & Jaff, 2010).
2. The thrombosis stage: when the decrease of venous flow reaches a critical point, platelet adherence and aggregation appear, with the formation of thrombi, and the patient becomes symptomatic (Thompson & Driskill, 2008; Perlowski & Jaff, 2010).

Clinically, the patient presents unilateral oedema of the arm, a sensation of "heaviness", upsetting dull pain or intense arm pain, cyanosis, collateral venous circulation (the anterior side of the thoracic wall or the ipsilateral shoulder) or even pulmonary embolism symptoms: dyspnea, chest pain (exacerbated by breathing) radiating to the shoulder, palpitations, cough (with or without blood expectoration), asthenia, dizziness, fever (Perlowski & Jaff, 2010; Hull & Harris, 2013).

Diagnosis is based on the above mentioned clinical manifestations, occurring suddenly in a young and apparently healthy athlete on physical examination and complementary explorations.

Physical examination can evidence unilateral swelling of the arm, cyanosis, collateral circulation, with unchanged peripheral pulse of the affected upper limb. Maneuvers for the diagnosis of TOS should also be performed: the Adson test and the Wright test. The Adson test is performed by palpating the radial pulse with the arm in supination and abduction at 15 degrees, while the neck is turned towards the affected area; the test is positive if the pulse disappears in deep inspiration. The Wright test is performed by palpating the radial pulse with the shoulder in abduction and external rotation; the test is positive if the pulse disappears (Gilard et al., 2001; Laker et al., 2009; Perlowski & Jaff, 2010).

The following complementary exams are required for

diagnosis: color and 2D Doppler ultrasound (the quickest and most accessible method), which show the presence of thrombi. Computed tomography (CT), CT angiography or magnetic resonance angiography (MRA) (with the upper limb lifted above the head) can be useful because, in addition to showing the presence of thrombi, it also provides anatomical information (the presence of the cervical rib or muscular hypertrophy). The exam of choice is contrast venography, which allows the localisation of the thrombus, assesses its surface and the state of collateral circulation and, if needed, allows to perform thrombolysis. Coagulation tests must also be carried out (Protein C, Protein S, factor V Leiden, lupus anticoagulant, cardiolipin antibodies, antithrombin III, ANA) in order to exclude a precoagulant state with secondary thrombosis. In PSS, these are most frequently within normal parameters (Laker et al., 2009; Perlowski & Jaff, 2010).

Regarding treatment, unfortunately, until today there is no international agreement. It must be individualised, prompt and aggressive. One could either use classic heparin or LMWH (low molecular weight heparin) in order to prevent the propagation of the thrombus and embolisation, or intravenous thrombolysis with or without thrombectomy, followed by anticoagulant treatment. Eventually, if there is significant narrowing by fibrosis, temporary balloon expansion could be attempted (although there is a high risk of restenosis). Inserting a stent is not recommended by most authors, due to a high risk of thrombosis recurrence (Molina et al., 2007; Melby et al., 2008; Laker et al., 2009; Perlowski & Jaff, 2010).

The duration of anticoagulant treatment with classic heparin or LMWH, followed by anticoagulant treatment administered orally, such as Coumadin, warfarin, varies according to each author: 3-6 months (Perlowski & Jaff, 2010); 8 weeks (Laker et al., 2009).

Unfortunately, these patients have a high recurrence risk. This is why surgery must be taken into account, which needs a multidisciplinary approach. The aim of surgery is the mechanical decompression of the subclavian vein (through venotomy and/or venoplasty) (Laker et al., 2009; Perlowski & Jaff, 2010).

A follow-up of these patients is mandatory and is carried out by 2D Doppler ultrasound and venography. Sports activity can be resumed only after the complete disappearance of symptoms and after the completion of anticoagulant treatment (Laker et al., 2009; Perlowski & Jaff, 2010).

Lower limb deep vein thrombosis (DVT)

DVT appears post-trauma mostly at the level of the popliteal vein, posterior tibial vein and peroneal vein, either through direct trauma, or as a consequence of a sudden hyperextension movement of the knee, of knee dislocation or lower limb torsion during kicking or approaching movements (Echlin et al., 2004; Casey et al., 2009).

Just like in the case of PSS, cases of non-traumatic lower limb deep vein thrombosis, induced by effort, have also been described. These are found in skiers and marathon runners. Although it is generally known that physical exercise prevents the formation of thrombi, an extremely intense effort may lead to a decrease in venous flow and vascular wall micro-traumas, which, in turn, lead to the appearance of thrombi. Non-traumatic thrombosis may also occur in the case of popliteal vein compression syndrome, through the median end of the gastrocnemius muscle. This syndrome is specific to athletes prone to gastrocnemius muscle hypertrophy, such as rugby and football players (Ehsan et al., 2004; Casey et al., 2009; Perlowski & Jaff, 2010).

The clinical presentation shows unilateral oedema of the lower limb, particularly visible on the calf and ankle, "tension" or pain in the calf (which does not disappear after the application of ice, after extension or pain killers). Immobilisation can exacerbate the pain, while movement can alleviate it. Clinical examination shows: oedema, warm skin, redness or cyanosis (more pronounced after warm baths), moderate fever and positive Homans sign (calf pain on leg dorsiflexion) (Goodacre et al., 2005; Casey et al., 2009; Burrus et al., 2014).

The complementary exams required for diagnosis are color or 2D venous Doppler ultrasound, which show the presence of the thrombus. Determination of D-dimer in blood is useful because its increased value significantly increases the suspicion of DVT. In order to precisely locate the thrombus, to assess its extension and collateral circulation, CT or MRI angiography can also be carried out. Coagulation tests must also be performed in this case (Protein C, Protein S, factor V Leiden, lupus anticoagulant, cardiolipin antibodies, antithrombin III, ANA) in order to exclude a precoagulant state; generally, they are within normal parameters. If the thrombus cannot be highlighted through the previously mentioned methods, but there is a high DVT suspicion, intravenous venography can also be carried out (Hyers, 2003; Casey et al., 2009; Burrus et al., 2014).

The potential complications are pulmonary embolism and post-thrombotic syndrome (PTS). Pulmonary embolism may occur in 50% of untreated DVT patients and may cause death in 2.1% of cases. It manifests through dyspnea, chest pain (exacerbated by breathing) radiating to the shoulder, palpitations, cough (with or without bloody expectoration), asthenia, dizziness, fever (Perlowski & Jaff, 2010; Hull & Harris, 2013; Boden et al., 2013; Burrus et al., 2014).

Post-thrombotic syndrome occurs in 20-50% of DVT patients in whom complete thrombus resorption has not been reached; there is also the risk that resuming training too early may lead to thrombus recurrence. It clinically manifests by chronic pain, paresthesia, chronic oedema, stasis dermatitis, pigmentation, ulceration. All these manifestations lead to a decrease in the quality of life and sports performance of the athlete (Kahn & Ginsberg, 2002; Echlin et al., 2004; Casey et al., 2009).

The two complications can be prevented through early and correct treatment.

Treatment consists of anticoagulation with classic heparin or LMWH, followed by oral anticoagulant treatment such as Coumadin or warfarin (3-6 months). Some authors also recommend thrombolysis, followed by anticoagulant treatment, which would preserve valve function and prevent PTS. Wearing elastic compression is also very important (Kahn & Ginsberg, 2002; Echlin et al.,

2004; Casey et al., 2009).

In terms of returning to sports activity, a close collaboration between the athlete and the attending physician is necessary. Activity will be resumed gradually, after 4-6 weeks and after the complete disappearance of signs and symptoms. During anticoagulant treatment, sports with a high risk of trauma are forbidden; the athlete may resume light jogging and swimming. Resuming training and attending sport competitions can only be done after the completion of anticoagulant treatment. Wearing compression stockings is mandatory for the prevention of PTS. It is also important to avoid, as much as possible, the risk factors (Hull & Harris, 2013; Burrus et al., 2014).

DVT prophylaxis

Given that it is much easier to prevent than to treat, we can mention some prophylactic measures to prevent DVT in athletes.

Athletes must pay attention to thirst as a sign of dehydration. High caffeine or alcohol intake will be avoided and water electrolyte rehydration will be done during and after practice.

During long travelling, pauses must be taken to mobilize the limbs and body; cramped positions and crossed leg positions must be avoided and elastic compression must be worn.

Female athletes must see a family planning specialist in order to use contraceptives that do not contain estrogens.

The prophylactic use of aspirin can be taken into account, but only as a medical recommendation.

Most importantly, the athlete must "listen to his/her body". If they feel something is not right, they must stop training and ask for a medical consult.

The identification and the management of any hereditary procoagulant condition are necessary in the case of athletes with a history of DVT, in close collaboration with the attending physician (Andersen & Spencer, 2003; Goldhaber & Fanikos, 2004; Hull & Harris, 2013; Burrus et. al., 2014).

Practical considerations

From everything presented above, the following must be kept in mind:

1. DVT can occur in athletes who may have risk factors (sometimes associated) for it.

2. DVT must be taken into account in any trauma episode, especially if there are problems with the differential diagnosis of musculoskeletal lesions, but it can also appear without obvious trauma.

3. DVT can develop anywhere, including in the upper limbs.

4. Due to physical condition, bradycardia and high pain tolerance, athletes may present atypical DVT symptoms.

5. If untreated, DVT may have devastating effects on the affected limb, on sports activity and it may even endanger the athlete's life.

6. Despite the athletes' physical condition and psychological determination, we must not forget that: *To be fit does not mean to be healthy*.

Conflicts of interest

Nothing to declare.

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