Treatment of acute venous thrombosis in a female thriathlete

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Introduction

Acute Deep Vein Thrombosis (DVT) has become an important public health problem as it affects more than 20 million individuals in the United States (Bluth, Benson & Ralls et al., 2008). Estimates of the incidence of DVT range between 100 and 180 per 100, 000 persons every year in the US (Jacobson, 2001). DVT is a manifestation of thromboembolism (VTE). PE and DVT result in numerous preventable deaths every year. Death from DVT-associated massive pulmonary embolism (PE) caused about 300, 000 deaths every year in the United States (Tapson, 2008). DVT is the formation of a thrombus or a blood clot in a deep vein, most commonly in the lower leg. The blood clot restricts circulation of blood through the blocked area causing symptoms such as swelling, pain, surface veins dilation, and redness of the leg (Morrison & Ray, 2007; Theiss, Fink, & Gerber, 2011). A third of DVT patients usually develop pulmonary embolism (PE), which is a critical condition. PE develops when blood clot pieces dislodge and move through blood circulation to the heart and into the lungs. This causes partial or initial blocking of pulmonary artery (Ramz & Leeper, 2004). PE symptoms include chest pains when breathing, breathing difficulties, and circulatory instability. Most of PE is caused by deep vein thrombosis of the proximal lower margin veins like popliteal, iliac, and femoral veins (Bulger, Jacobs, & Patel, 2004). There are numerous risk factors for deep vein thrombosis. They include surgery, congestive heart failure, restricted mobility, infectious disease, obesity/overweight, cancer, respiratory failure, family history of DVT, and demanding exercises among others. Strenuous athletic episodes usually

contribute numerous risk factors to DVT development in athletes (Cerneca, Simeone & Bruno et al., 2005).

The factors that increase the risk for an athlete to develop DVT are dehydration, traveling long distances, significant trauma, family history, major surgery or bone fracture, existence of a congenital deformity of veins structure, May-Thurner Syndrome, and Thoraic outlet obstruction (Hyers, 2003; Oger, 2000). Mechanisms, which underlie DVT, are hypercoagulability, endothelial cell injury, and venous stasis (these symptoms are known as Virchow’s triad). In athletes, common symptoms of DVT are pain, discoloration, and swelling of the affected area. Physical examinations normally expose a clear cord of a vein and edema, warmth, and tenderness in the affected area (Bulger, Jacobs, & Patel, 2004). Other symptoms include tachycardia, shortness of breath, sweating, anxiety, and a cough that leads to blood production (ChanLee et al., 2013).

Female triathletes usually engage in multiple-stage competition, which entails completion of three sequential and continuous events. They contend for the best overall course completion time counting timed transition between the person running components (Arco & Olcott, 2003). As a result, these endurance athletes are at risk of numerous physical causal aspects, which contribute to DVT experiencing endothelial harm, recurring trauma, and dehydration during competitions, followed by periods of immobility, sluggishness, and stasis while moving from and to and/or recuperating from an athletic incident (Arco et al., 2001).

When it comes to treatment of DVT, it entails prevention the blood clot from enlarging, ensuring that the clot does not break lose to prevent pulmonary embolism, preventing or reducing the chances of DVT reoccurrence, alleviating the acute leg symptoms like swelling and pain, and preventing the post-thrombotic condition and minimizing post-thrombotic indications (Weitz et al., 2004). For most patients, treatment objectives are realized through offering ample anticoagulant management, at first therapy and continuous long-term for three months or longer (Chang, 2004). Treatment options include blood thinners, filters, clot busters, and compression stockings. Blood thinners or anticoagulants are medications that are used in treating DVT and decrease the ability of the blood to clot (Levine et al., 2001). They prevent existing blood clots from enlarging and reduce the risk of developing additional clots. Nonetheless, they do not break up existing blood clots. According to Van Beek, Buller, & Oudkerk, (2009, p. 245), “ adequate anticoagulant treatment reduces the incidence of symptomatic extension and/or recurrence of thromboembolism during the first three months after diagnosis from 25% of more to 4% or less”. Physicians normally give an infusion or shot of the blood thinning heparin for a number of days after which they administer blood thinnersin tablet forms (Wells et al., 2005). Medications are taken for duration of three months or longer. Clot busters are used to treat serious cases of DVT or PE or in case other forms of medicines are not effective. Thrombolytics are a group of medications, for example, tissue plasminogen activators, which are offered to patients using an intravenous line to disintegrate blood clotting (Hoppensteadt et al., 2003). Van Beek et al. (2008, p. 476) argued, “ thrombolytic therapy, either by systemic infusion or catheter-directed infusion, is currently indicated only for

selected patients with DVT as it prevents post-thrombotic symptoms". Nonetheless, it has to be followed by sufficient anticoagulant treatment to avert recurring venous thromboembolism. Filters are medicines, which are put in a large vein, usually the venacava, in patient's abdomen in order to prevent blood clots, which dislodge from lodging in the lungs (Vedantham et al., 2006). Compression stockings normally assist in preventing swelling, which results from deep vein thrombosis (Muir et al., 2000; Allan, Williams & Bolton, 2005). Patients usually wear stockings on their legs from the foot to the knee level. The pressure created by the stocking prevents and/or reduces the changes of blood pooling and clotting (Allan et al., 2005). " Using properly fitted graded compression stocking with an ankle pressure gradient of 3—40 mmHg, applied as soon after diagnosis as the patient symptoms will allow and continued for at least 2 years, is effective in reducing the incidence of post-thrombotic symptoms, including moderate-to-severe symptoms" (Van Beek et al., 2008, p. 476).

Literature Review

There are various case studies on athletes who have suffered from deep vein thrombosis or pulmonary embolism after physical activity or competition. Tao & Davenport (2008) case report was about a female triathlete diagnosed with deep vein thrombosis as well as pulmonary embolism after she competed in a triathlon. She took part in a triathlon and the following morning traveled by car for five hours, after which she suffered left lower margin pain symptoms and swelling and afterward lightheadedness and dyspnea upon physical exertion. DVT was appropriately diagnosed and treated after visiting the emergency department 3 weeks later. Meignan et

al. (2000) also reported a case of PE and DVT occurrence after jogging and running. Mackie & Webster (1981) described the case of two marathon runners, who developed DVT and PE about seven days after taking part in a marathon. In both instances, physicians misdiagnosed DVT at first either as a Baker’s cyst or as muscle strain. DVT thus occurs in female thriathlete presenting with lower extremity swelling or pain after engaging in strenuous activity. Athletes are often exposed to many diverse factors, which lead to endothelial injury or venous stasis, hence increasing the risk for DVT (Difelice et al., 2002). Numerous treatment options are available for treating acute venous thrombosis. Despite the effectiveness of the natural antithrombotic system, patients like triathletes require therapeutic agents to treat thrombosis and to prevent over activation of the clotting system. Triathletes engage in an endurance sport activity, which exposes them to various factors, which increase the risk of clot formation. Tao & Davenport (2008) report that DVT in athletes is mostly misdiagnosed and this leads to delayed treatment even in cases where symptoms are severe. The most frequently applied test for testing to diagnose DVT in athletes is Doppler ultrasonography in the affected area. It is rather costly to undertake and requires special equipment as well as skilled vascular specialists and technicians. According to Tao & Davenport (2008), physicians are usually uncertain about making the assessment in athletes who have symptoms, which are similar to musculoskeletal pain after an exercise and those who fail to show tachycardia with pulmonary embolism because of baseline bradycardia. Nonetheless, after diagnosis a number of treatment options are available for acute venous thrombosis.

The anticoagulation medicines warfarin and heparin are usually used to treat deep vein thrombosis on its initial stages (Kearon, 2003). Anticoagulation treatment is the first treatment for numerous patients with deep vein thrombosis. The therapy is very effective in decreasing thromboembolism risk, and it is the treatment of choice for almost all cases of acute lower-extremity DVT. Heparin is one of the important anticoagulants used for therapy. It is a highly sulfated mucopolysaccharide, which is negatively charged. Its molecular weight lies between 6, 000 and 25, 000 daltons. It has to be given by injeection under the skin (subcutaneous) or into the veins (intravenous) as it is not usually absorbed from the gastrointestinal tract. Heparin usually exerts its anticoagulant effect through activating antithrombin III, which is a natural anticoagulant (Morris, 2003; Valenstein, Walsh & Meier, 2004).

Low molecular weight heparin has been derived from heparin and it acts in the same way as heparin but with fewer side effects and also requires less frequent injections (Liapis, Daskalopoulos, & Daskalopoulos, 2002; Bernardi & Prandoni, 2003; Garon, 2003; Hoppensteadt, Walenga & Fareed et al 2003; White & Ginsberg, 2003). Low molecular weight heparin (LMWH) has become the deep vein thrombosis care standard as it is safe and effective unfractionated heparin, however, it is considered more suitable to apply (Bates & Ginsberg, 2004; Hirsh & Rashke, 2004). It has a predictable anticoagulant reaction such that it is offered in a set weight attuned subcutaneous dosage without laboratory monitoring for the majority of patients. This enables out of hospital management in over 80% of individuals suffering from acute deep vein thrombosis (Eikelboom & Baker, 2001). There

are patients who need to be admitted to the hospital, for instance, patients with obesity, renal impairment, or those who are pregnant. LMWH also has a limited risk of causing heparin-induced thrombocytopenia (Bates & Ginsberg, 2004). Nonetheless, it is not used in treating heparin-induced thrombocytopenia as antibodies of unfractionated heparin cross-react with LMWH. LMWH is also preferred because it is less likely to cause osteoporosis compared to unfractionated heparin.

Unfractionated heparin is mostly used to treat patients who are undergoing invasive procedures, those at high risk of renal failure, and bleeding as it has reversibility with protamine sulfate, shorter half life as well as extra-renal metabolism. Warfarin is also a commonly used oral anticoagulant in treatment of DVT (Krsihnan & Streiff, 2003; Beattini, Vedovati, & Agnelli, 2007). It obstructs production of vitamin K dependent procoagulants (factors X, IX, VII, II) in the liver through hindering oxidized vitamin K reduction. Warfarin does not affect functional circulation clotting factors, and hence a week of oral anticoagulation is needed to attain the optimum restorative impact. When the patient is offered the correct dosage and is monitored well, warfarin is effective for prophylaxis of thrombosis (Chain & Macik, 2002; Gabe & Fihn, 2000; Shcafer, 2003; Aberegg, 2003). Excessive anticoagulation causes serious bleeding complications whereas inadequate coagulation leads to thromboembolic complications. As a result, proper laboratory measurement of prothrombin time (PT) is very important while managing patients receiving oral anticoagulation (Riley, Rowe, & Fisher, 2000).

Although anticoagulation is effective in preventing clinically significant pulmonary embolism, it is not effective for clot removal in most cases, and complete resolution of DVT is only accomplished in about 10% of patients (Konstantinides & Geibel, 2002). Lack of effectiveness of short-term anticoagulation leads to prolonged acute symptoms of discomfort and swelling. As a result, long-term anticoagulation is required for most patients. Warfarin is usually the ideal anticoagulant for long-term DVT management for nearly all patients. It is preferred as it is highly effective, can be given orally, and reduces the risk of recurring DVT by 80%-90% during its management (Kearon, 2004; Ansell et al., 2004). Warfarin treatment is usually continued for 3 months. Lee, Levine & Baker (2003) noted than LMWH therapy is also effective in long-term therapy.

Another important treatment method of acute venous thrombosis in a female triathlete is thrombolytic therapy as well as surgical embolectomy (Dong, Hao, & Yue, 2009). Watson & Armon (2004) noted that thrombolysis improves the patency of veins and lessens post-thrombotic syndrome risk. However, it raises the bleeding risks and there is a lack of indication of a net proven advantage. The vena cava filter is also an important treatment method as it prevents PE in DVT patients not eligible for anticoagulant treatment and those who experience PE in spite of sufficient anticoagulation (Streiff, 2000). The filters do not reduce the need for anticoagulationas it leads to an increased DVT recurrent risk. Graduated compression stockings are also important in treatment of DVT. Geerts et al. (2004) notes that graduated compression stockings normally decrease post-thrombotic

syndrome risk and should be used when there are no contra-indications (extensive varicosities or existing leg ulceration).