Interdisciplinary Management of Deep Vein Thrombosis During Rehabilitation of Acute Rupture of the Anterior Cruciate Ligament: A Case Report

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Abstract

Objective: The purpose of this case report is to describe a patient who experienced deep venous thrombosis (DVT) during pre-operative rehabilitation of an acute rupture of an anterior cruciate ligament (ACL) reconstruction graft, to increase awareness of DVT occurring in a healthy individual after periodic immobilization, and to describe the interdisciplinary management for this patient.

Clinical features: A 30-year-old male was referred to a chiropractic clinic for presurgical treatment of a left ACL rupture and medial meniscus tear confirmed at magnetic resonance imaging. During the course of preoperative rehabilitation, the patient became limited in ambulation and presented for a routine rehabilitation visit. During this visit, he experienced increased leg swelling, pain and tenderness. The patient was assessed for DVT and was referred to the local emergency department for further evaluation where multiple DVTs were found in the left popliteal, posterior tibial, and peroneal veins.

Intervention/outcome: The patient was treated with a 17-week course of warfarin during which time the clinical signs and symptoms of DVT resolved. Meanwhile, the patient completed the rehabilitation treatment plan in preparation for ACL reconstruction without further complications.

Conclusions: This case raises awareness that DVT may occur in a healthy individual after periodic immobilization. While there may be controversy regarding the appropriate application of pharmaceutical anticoagulants in patients with DVT of the leg, the most risk averse strategy is for a short duration prescription medication with compression stockings. Through interdisciplinary management, the patient experienced a successful outcome.

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Introduction

Deep vein thrombosis (DVT) is a common disorder that occurs within the spectrum of venous thromboembolism (VTE).¹ DVT is especially common in older individuals and in certain predispositions such as

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thrombophilic disorders and malignancy.² DVT is often clinically asymptomatic,^{1,3,4} can masquerade as musculoskeletal conditions, and are known to occur after trauma and immobilization.

There is a high incidence after acute rupture of the Achilles tendon and after major orthopedic surgery.^{3,5-7} Occurrence during preparation for surgical repair of anterior cruciate ligament (ACL) has, to the best of our knowledge, not been reported. DVT have a penchant for chronicity and may have dire complications, such as post-thrombotic syndrome, pulmonary embolism (PE), or death,¹ if left inappropriately managed. Death occurs in approximately 6% of DVT cases and 12% of PE cases within 1 month of diagnosis.¹ Idiopathic DVT is particularly problematic as they are often perpetual and require extended and sometimes indefinite anticoagulation therapy.^{1-4,8} The paradigm for treating various forms of DVT has undergone a radical change since the late 1990s as it is recognized that different forms of venous thromboembolism can accompany different risk factors.⁸ For instance, isolated distal DVT (IDDVT) is not associated with the existence of thrombophilic disorders and some authors question the value of anticoagulation therapy in these cases.⁴

At present, there are no known case reports of interdisciplinary management including chiropractic care of DVT during pre-surgical treatment of an ACL rupture. Therefore, purpose of this case is to describe the occurrence of DVT in a healthy individual after a history of episodic immobilization, describe the importance of timely urgent care, present risk factors, and current management schemes.

Case Report

A 30-year-old male was referred to a chiropractic clinic for pre-surgical treatment of a left ACL rupture and medial meniscus tear confirmed at magnetic resonance imaging. The injury was sustained after jumping from a boat onto a stable platform. On presentation 2 weeks after incident the knee was drained by an orthopedist for extensive effusion (3+). The patient complained of considerable left knee discomfort, swelling, and reduced range of motion. Pain was reported to radiate inferiorly and superiorly along the left calf and rated 7/10 on Numerical Pain Scale. The patient was ambulating with crutches and maintained a leg flexion angle of 25°. He denied nausea, vomiting, dyspnea, and back and/or chest pain. The patient had a 10 pack-year history of tobacco use and arthroscopic ACL repair 10 years prior. Medication included Vicodin for pain relief. The clinical

exam revealed an effusion of the left knee and moderate ankle swelling without warmth or erythema. Active range of motion (AROM) was 10° of extension and 70° of flexion with pain at both end ranges. The patient reported significant tenderness at the medial knee joint line. Neurovascular deficits were not present. Active contractibility of the quadriceps was minimal (grade 1) with notable diffuse quadriceps atrophy. There was no calf atrophy noted. Straight leg raise (SLR) revealed significant hyperextension lag. Patellofemoral glide was within normal limits and tolerated without pain or restriction. Orthopedic exam was not performed due to his confirmed diagnosis of medial meniscus tear and ACL rupture. The chest was clear without wheezes, rales, or rhonchi.

Chiropractic treatment consisted of 4 visits in which low load long duration (LLLD) extension stretches were employed to achieve terminal extension, decrease effusion and return quadriceps neuromuscular control. LLLD stretches were performed with the heel propped and an 8-lb weight placed on the quadriceps for 10 to 12 min. Pulsed electric muscle stimulation on the quadriceps with short arc biofeedback contractions and icing were also provided. Joint mobilization techniques such as low-grade femorotibial AP/PA glide, internal/external rotation and multiplanar patellofemoral glides were employed to reduce adhesions and prevent infrapatellar contracture syndrome. No high-velocity low amplitude was utilized on the knee joints. He was placed on a home care exercise plan that included straight leg raises (SLRs) × 4, supine isometric quadriceps activation, gluteus sets (clams, side-lying hip abduction, prone hip extension) (Fig 1), heel props, ankle pumps, and icing. He was encouraged to reduce his usage of crutches as tolerated. His AROM by the fourth visit had improved to 5° of extension and 80° of flexion. After LLLD stretch terminal extension (0°) was obtained and 90° of passive flexion was obtained with joint mobilization techniques as described previously. His quadriceps contractibility had improved to grade 3. SLR was performed without lag post treatment. The patient continued to use crutches for ambulation. He had begun working again, however, remained as sedentary as possible. His job required him to sit for prolonged periods, up to 6 hours per day. On his fifth visit he presented with significant discomfort in his knee and entire leg/ankle. Upon inspection he continued to demonstrate a knee effusion but also had signs of left ankle and leg effusion as well. He denied any new injury or incident. He did not display erythema or visible hypervascularity. Left-sided posterior tibial and dorsalis pedis pulses were 1+. Homan's and Pratt's signs were positive on the left. Suspicion for DVT was noted and the patient was immediately referred to the orthopedic

Fig 1. Composite image demonstrating prescribed quadriceps and gluteal exercises. Image A demonstrates the relaxed quadriceps while image B shows the isometric contraction. Image C and D demonstrate the clam exercise for gluteus medius activation. Image E and F show the prone hip extension exercise to promote gluteus maximus activation. All exercises were instructed to be held for 3 seconds.

surgeon. The patient was then transported to the local Emergency Department for evaluation and treatment.

Diagnostic workup at the Emergency Department included color duplex sonography. Compressibility was lost in the popliteal and calf veins without normal phasic flow patterns distally (Fig 2). DVT was diagnosed in the left popliteal, posterior tibial and peroneal veins and warfarin was prescribed. The patient was seen 2 weeks later in the chiropractic office to resume therapy. At that point his condition was under control with anticoagula-

tion. The patient was not ambulating with crutches but had significant gait compensation. He stated his left knee felt weak and sore. His AROM was 5° of extension and 85° of flexion. He proceeded with 6 more weeks of therapy and regained full AROM (0-140°). Normal joint play and passive motion were restored without pain. SLR was performed without lag. His left lower extremity strength returned to 90% and no muscle atrophy was noted. Activities of daily living were performed without difficulty and gait resumed to normal. He was released to

Fig 2. Transverse ultrasound image of the left posterior tibial vein. Uncompressed transverse image (A) demonstrates the vascular bundle as round, apposed, hypoechoic structures (white open arrow). Transverse image with transducer compression (B) demonstrates non-compressibility of the veins, the ultrasound hallmark of deep vein thrombosis. Notice that in the compression view (B), the vascular structures appear more superficial, evidence of an adequate compressive attempt.

perform unsupervised resistance exercises in the gym with only closed kinetic chain exercises at safe/comfortable flexion angles. He reported no further complications and followed up with the orthopedic surgeon for ACL and meniscus repair.

Discussion

Incidence and Risk Factors

Deep-vein thrombosis has an estimated annual incidence of 67 to 100 per 100 000 among the general population.^{1,9,10} The incidence of first time venous thromboembolism rises above 100/100,000 at about the age of 55 with no significant difference in sex.¹ About 25% to 50% of first time DVT are thought to be idiopathic¹; however, in 50% of these, a thrombophilic factor such as activated protein C resistance (Factor V Leiden), protein S or C deficiency could be identified.^{2,11} Generally, VTE events may be unprovoked (spontaneous or idiopathic) or secondary to other factors such as trauma or surgery.^{1,11} Approximately 2-18% VTE are associated with trauma.¹ In the setting of trauma, it is thought that endothelial injury initiates the thrombotic events.² Additional predispositions include increasing age, immobilization, hospital or nursing home confinement, malignancy, neurologic disease with extremity paresis, certain types of oral contraception and hormone replacement therapy, current or recent central venous catheter, long-distance flights, obesity, smoking, thrombophilic defects.^{2,9,12} The use of oral contraceptives in women with BMI >25 increased the risk of VTE 10-fold.¹²

Pathophysiology

The pathogenesis of VTE is associated with the triad of interdependent risk factors described by Virchow, consisting of hemodynamic imbalance (blood stasis), endothelial vessel wall damage and a local or systemic state of hypercoagulability. Blood stasis is particularly apparent in situations of physical inactivity such as bed rest and during air travel, where a lack of pumping action of the large muscles of the calf causes decreased blood flow or even stasis. It has been suggested that blood pooling leads to vessel distention and activation of the coagulation system, both contributing to a state of local hypercoagulability.² When the homeostatic balance between pro- and anticoagulation forces shifts in favor of coagulation, the risk for VTE increases. If this imbalance is due to an inherited defect, this risk exists for

life.¹ In the setting of a transient factor, this risk exists only for as long as the risk factor is present.^{1,2} Damage to the vessel wall is another essential component of venous thrombogenesis. After injury, certain changes develop in the clotting system, particularly an increase in von Willebrand factor and platelet aggregability.² Venous thrombi consist mainly of fibrin and red blood cells as opposed to arterial thrombi which consist mainly of platelets.²

Anatomical Considerations of Lower Extremity DVT

DVT is one disease within the spectrum of venous thromboembolism (VTE). The most common form of symptomatic VTE is DVT of the leg.¹⁻³ The proximal veins of the thigh are considered separately from the distal veins of the leg. Proximal veins are defined as the common femoral vein caudally to the popliteal vein.¹⁰ Distal veins are sometimes referred to as the deep calf veins and include all infra-popliteal deep veins.¹⁰ Isolated calf muscle vein thrombosis (ICMVT) and deep calf vein thrombosis (DCVT) have been used to describe DVT within the distal veins of the leg. Isolated distal DVT is the composite of ICMVT and DCVT – occurring either in isolation or in combination.⁴ Isolated distal deep vein thrombosis account for 31-56% of all diagnosed leg DVTs.⁴ Despite its frequency, IDVT currently is one of the most debated issues in the field of venous thromboembolism.⁴ There is growing evidence that the risk factor profile of IDVT is different from proximal DVT or PE. A recent study, involving more than 11,000 patients with confirmed leg DVT revealed that IDVT was less prevalent in elderly patients (greater than or equal to 75 years), in women during pregnancy or puerperium, and in other chronic states, such as previous VTE or active cancer. IDVT was associated with transient risk factors such as hospitalization, recent surgery or trauma, recent travel and the presence of leg varicosities.⁴ The presence of inherited thrombophilic alterations had no effect on the prevalence of IDVT.⁴ Systemic etiologies or prothrombotic conditions are seen more frequently when there is bilateral distal DVT.⁴

Clinical Diagnosis

A clinical prediction rule that takes into account signs, symptoms and risk factors can be accurately applied to categorize patients as having low, moderate, or high probability of DVT (Table 1).^{10,13} Over 14 studies have validated the reproducibility of this model.¹⁰ Patients with low pretest probability can have DVT safely excluded on the basis of a single negative ultrasound

result and thus can avoid the inconvenience of serial ultrasound imaging.¹⁰

Laboratory Diagnosis

D-dimer is a degradation product of a cross-linked fibrin blood clot. Conditions that elevate D-dimer levels include acute venous thromboembolism and a variety of non-thrombotic conditions (recent major surgery, hemorrhage, trauma, pregnancy or cancer). D-dimer assays are sensitive but non-specific markers of DVT.¹⁰ Incorporation of D-dimer testing into diagnostic algorithms simplifies the management of a patient presenting with suspected DVT.¹⁰ The patient should be referred for urgent laboratory evaluation if there is suspicion of DVT and the patient is stratified into either a moderate or high pretest probability for DVT.

Imaging Diagnosis

Compression ultrasonography is currently the imaging test of choice to diagnose and characterize DVT.¹⁰ Diagnostic criteria used include lack of compressibility of a venous segment. Doppler ultrasound can be used to accurately identify vessels and to confirm the compressibility of a particular segment. Sensitivity for the diagnosis of DVT in the proximal veins is 97% while in the distal veins is only 73%. Given this low sensitivity and a negative result, serial scanning of proximal veins (to

evaluate for proximal extension of a previously undetected distal thrombus) at 1 week has been recommended, however, a series of studies has shown that a single complete ultrasound in symptomatic patients has comparable clinical results as serial proximal ultrasound.^{10,4} Ultrasound can also characterize DVT and delineate between acute, chronic and recurrent DVT.¹⁰ Acute DVT is usually occlusive, not echogenic and tends to be continuous.¹⁰ Chronic DVT tends to be echogenic, non-occlusive and non-continuous.¹⁰ It is important that chronicity, or the lack thereof, be established as the treatment strategies between acute and chronic DVT varies. Recurrence is suggested by clot extension or when an existing thrombus increases diameter by 4 mm.¹⁰ Venography may be utilized in equivocal cases.¹⁰

Treatment

Currently the goal of therapy for lower-extremity DVT is to prevent the extension and embolization of thrombus in the short-term and to prevent recurrent events in the long-term.¹⁰ Standard anticoagulation prevents clot extension and embolization but does not lyse the acute thrombus; in many cases, only partial clearance of thrombus occurs.¹⁴ Residual thrombosis can be detected for at least 1 year after a symptomatic episode.³ Therefore, idiopathic DVT often becomes a chronic process where guidelines suggest an indefinite duration of anticoagulation; DVT associated with trauma (or other transient risk factors) does not require an indefinite duration of therapy.⁸ There are guidelines established for treatment with anticoagulation therapy for proximal DVT.¹⁰ In the presence of a transient risk factor, a limited duration of 3 months is adequate.¹⁰ When ongoing risk factors exist, the duration of anticoagulation therapy should be at least 6 months and in some cases indefinite.^{8,10} When considering prolonging anticoagulation therapy beyond 6 months, the risks of bleeding must be weighed against the potential benefits of preventing recurrent thrombosis.¹⁰ Major hemorrhage occurs in approximately 2-4% of patients on long term anticoagulation but is usually non-fatal.^{8,10} But there is little evidence for guidelines specifically concerning IDVT and optimal treatment is still controversial. There is no conclusive proof that all IDVTs need to be diagnosed and anticoagulated and, when diagnosed, there is still a good deal of uncertainty on the type and duration of anticoagulation needed.⁴ However, in most current clinical practices IDVT are typically treated with a 6- to 12-week course of anticoagulation therapy.^{4,8} Studies with small sample sizes suggest that over 90% IDVT have

Table 1 Clinical Prediction Rule for Pretest Probability of Deep Venous Thrombosis[†]

Clinical Feature	Score
Active cancer (treatment ongoing or within previous 6 months or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden >3 days or major surgery within 4 weeks	1
Localized tenderness along distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling >3 cm when compared with asymptomatic leg (measured 10 cm below tibial tuberosity)	1
Pitting edema (greater in the symptomatic leg)	1
Collateral superficial veins (non-varicose)	1
Alternative diagnosis as likely or greater than that of deep-vein thrombosis	-2

[†] A score of 3 or higher indicates high probability of deep venous thrombosis; a score of 1 to 2 indicates that the probability is moderate; a score of less than 1 indicates low probability.

complete resolution when treated without anticoagulation but with Class-II-calf elastic compression stockings when only transient risk factors are present.⁴ Criteria to identify subjects at higher risk of complications from IDDVT are lacking.⁴

Complications

DVT is a condition that can result in morbidity and mortality if undiagnosed or inadequately treated. Mild-to-moderate venous valvular insufficiency can be found in approximately one-third of patients after IDDVT.⁴ Valvular insufficiency is often temporary.³ The rate of proximal extension of IDDVT is a clinically important issue, linked with the differing demands of managing proximal DVT and the possibility of developing symptomatic pulmonary embolism. Identification of patients with IDDVT at risk for proximal extension using predictive factors is an unresolved question.⁴ The rate of proximal extension of untreated IDDVT seems to be less than 10%.⁴

IDDVTs are associated with a lower risk of recurrence versus other VTEs and less late sequelae than proximal DVT.⁴ Generally, first year recurrence rate of VTE is about 10%. Most VTE events recur within the region of initial diagnosis.¹ Recurrence of proximal DVT is approximately 25% after 5 years and 30% after 10 years.⁸ Patients with cancer show the highest rate of recurrence.¹ Elevated levels of factor VIII and homocysteine are independent risk factors that increase the risk of recurrence of VTE.¹¹ Recurrence rates are lower when transient risk factors are present, such as trauma and patients who had undergone surgery.^{1,15} Without treatment, about 50% of patients with symptomatic proximal DVT or PE have recurrent thrombosis within 3 months.³

DVT and PE are distinct but related aspects of VTE; most PE are the result of emboli from DVT.^{2,3} Autopsy data report a large number of pulmonary embolisms with DVT, although it is unclear how many of these PE are symptomatic.¹ Current data estimate that the incidence of symptomatic PE in patients with IDDVT is about 1.6-6.3% with no fatalities reported; however, this is based on a small number of untreated patients.^{4,8} A systematic review concluded that the prevalence of asymptomatic PE in IDDVT was about 13%.⁴ The real risk of IDDVT-associated PE is not well established.⁴ 50% of patients with untreated proximal DVT will develop symptomatic PE within 3 months.³ Approximately 4% of patients with anticoagulant treated proximal DVT develop further proximal extension, to include PE.³ Mortality associated with DVT is strongly associated with age and

other risk factors such as cancer and the presence of cardiovascular disease.¹

Postphlebitic or post-thrombotic syndrome (PTS) is a frequent complication of DVT.^{10,14} This condition results from chronic thrombosis and residual venous obstruction.³ Increased risk of PTS is evident with proximal DVT, previous ipsilateral venous thrombosis, higher body mass index, and older age.¹⁴ Incomplete resolution of leg signs and symptoms of DVT at 1 month strongly predicts development of PTS.^{10,14} The use of graduated elastic compression stockings reduces the risk of PTS after proximal DVT.¹⁴ The diagnosis of PTS is made clinically after the pain and swelling of acute DVT resolve, which can persist for a few months in some patients, without further diagnostic testing.¹⁴ Symptoms of PTS include pain, heaviness and swelling of the leg exacerbated by standing or walking.³ Severe PTS manifests as varicose eczema, subcutaneous atrophy, hyperpigmentation and chronic skin ulceration.³ PTS develops less frequently in patients with IDDVT (5% at 5 years) versus proximal DVT (28% at 5 years).^{3,4} There is limited evidence currently for the use of medications to treat PTS.¹⁴ Medications to treat PTS are only as effective as ECS and, currently, no added benefit has been derived from combining the 2 therapies.¹⁴ Horse chestnut seed extract has been shown, in the short term, to be effective in managing leg pain and edema associated with PTS. Long term data on the effectiveness and safety of this extract does not exist.¹⁴ PTS is managed with compression, leg elevation and elastic compression stockings.¹⁴ Symptoms resolve in over half, regardless of initial severity.³

There are a few reports of thromboembolic complications following various forms of manual therapy such as aggressive massage.¹⁶⁻¹⁹ These reports are rare, and in some instances, difficult to ascertain causality in the presence of other risk factors.^{18,19} In one case, a chest CT revealed a fractured, migrated stent within the right atrium, discovered during trauma workup after the patient fell off a ladder and suffered rib fractures and other soft tissue injuries. The stent was originally placed to treat a thrombus in the left common femoral vein approximately 3 years prior.¹⁹ The author attributed the stent migration and fracture to episodes of massage directly to the stent site (left thigh). While this is plausible, it seems equally plausible for the stent to have fractured and migrated during the fall from the ladder. In another case, a pregnant woman with a known hereditary thrombophilic defect (Factor V Leiden mutation) who recently quit smoking presented with acute-onset right neck swelling hours after receiving rotatory cervical manipulation (C2-C3) for neck and

shoulder pain. The manipulation was only described as rotatory and it is unknown whether the internal jugular vein was directly contacted. Workup revealed a right-sided internal jugular vein thrombosis. The authors acknowledged the difficulty with determining the role of the manipulation in this case.¹⁸ Nonetheless, it seems prudent to avoid aggressive manipulation and/or contact of major vessels in the presence of multiple thrombophilic risk factors or at sites of known or suspected VTE. DVT is thought to be a relative contraindication to local manipulative and manual therapy.²⁰

Limitations

Limitations of this case report include the retrospective observational nature of the case study and the lack of long-term follow-up. Our patient was not screened for inherited thrombophilic factors. Additionally, it cannot be determined whether the DVT was a response to the ACL rupture, the periodic occupational immobilization, or both.

Conclusion

Deep vein thrombosis is a condition that may result in morbidity and mortality if left undiagnosed or inadequately treated. DVT are known to present after trauma and immobilization. While there may still be some disagreement regarding the appropriate application of pharmaceutical anticoagulants in patients with DVT of the leg, the most risk averse strategy is for a short duration prescription combined with compression stockings. The patient in this case was treated with a 17-week course of anticoagulants and the clinical signs and symptoms of DVT resolved. Meanwhile, the patient continued and completed the rehabilitation treatment plan in preparation for ACL reconstruction without further complications.

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