Case Reports

Multiple Venous Thromboses Presenting as Mechanical Low Back Pain in an 18-Year-Old Woman

Andrée-Anne Marchand DC, MSc^{a,*}, Jean-Alexandre Boucher DC^b,
Julie O'Shaughnessy DC, MSc^c

^a PhD Candidate, Département d'anatomie, Université du Québec à Trois-Rivières (UQTR), Trois-Rivières, Québec, Canada

^b PhD Candidate, Département de psychologie, Université du Québec à Trois-Rivières (UQTR), Trois-Rivières, Québec, Canada

^c Full-time Professor, Département de chiropratique, Université du Québec à Trois-Rivières (UQTR), Trois-Rivières, Québec, Canada

Received 30 September 2014; received in revised form 25 March 2015; accepted 6 April 2015

Key indexing terms:

Venous thrombosis;
Low back pain;
Diagnosis;
Case study

Abstract

Objective: The purpose of this case report is to describe a patient who presented with acute musculoskeletal symptoms but was later diagnosed with multiple deep vein thrombosis (DVT).

Clinical Features: An 18-year-old female presented to a chiropractic clinic with left lumbosacral pain with referral into the posterior left thigh. A provisional diagnosis was made of acute myofascial syndrome of the left piriformis and gluteus medius muscles. The patient received 3 chiropractic treatments over 1 week resulting in 80% improvement in pain intensity. Two days later, a sudden onset of severe abdominal pain caused the patient to seek urgent medical attention. A diagnostic ultrasound of the abdomen and pelvis were performed and interpreted as normal. Following this, the patient reported increased pain in her left leg. Evaluation revealed edema of the left calf and decreased left lower limb sensation. A venous Doppler ultrasound was ordered.

Intervention and Outcomes: Doppler ultrasound revealed reduction of the venous flow in the femoral vein area. An additional ultrasonography evaluation revealed an extensive DVTs affecting the left femoral vein and iliac axis extending towards the vena cava. Upon follow-up with a hematologist, the potential diagnosis of May-Thurner syndrome was considered based on the absence of blood dyscrasias and sustained anatomical changes found in the left common iliac vein at its junction with the right common iliac artery. A week following discharge, she presented with chest pain and was diagnosed with venous thromboembolism. The patient was successfully treated with anticoagulation therapy and insertion of a vena cava filter.

* Corresponding author at: Université du Québec à Trois-Rivières, 3351 Boul. Des Forges. C.P 500, Trois-Rivières, Québec, Canada, G9A 5H7. Tel.: +1 819 376 5011x3798.

E-mail address: Andree-anne.marchand@uqtr.ca (A. -A. Marchand).

Conclusion: Although DVTs are common in the general population, presence in low-risk individuals may be overlooked. In the presence of subtle initial clinical signs such as those described in this case report, clinicians should keep a high index of suspicion for a DVT. Rapid identification of such clinical signs in association with a lack of objective examination findings warrants further evaluation due to potentially negative outcomes.
© 2015 National University of Health Sciences.

Introduction

Venous thromboembolism (VTE), a blood clotting process that encompasses both deep vein thrombosis (DVT) and pulmonary embolism (PE), is the third most common vascular disorder in white populations after myocardial infarction and stroke.¹ An individual's absolute lifetime risk of VTE is approximately 11%² and its incidence rate increases exponentially with age, for both women and men.^{1,3,4} Although less frequent than DVT alone (0.93 per 1000 person-years), PE with or without DVT (0.50 per 1000 person-years)¹ has a higher recurrence rate and is often fatal.³ Most cases of DVT (90%) are located in the lower limbs,⁵ particularly within the gastrocnemius and soleus muscles⁶ and become symptomatic when there is proximal vein involvement.⁵ Despite adequate treatment, DVT can recur, and about 10% of patients will develop severe post-thrombotic syndrome within 5 years.⁷ As part of the general chiropractic program, the training on VTE includes education on the usual clinical presentations, such as a "red, swollen, and hot" calf for DVTs and shortness of breath for PEs. Clinicians should be aware of the different clinical presentations of this serious pathology, as faster diagnosis and treatment may lead to a better prognosis.

The objective of this case report is to present a rare case of multiple thromboembolism events in a young woman, who first presented to a chiropractor's clinic for acute low back pain. An overview of the risk factors, clinical presentation, diagnosis, and management will be discussed.

Case Report

An 18-year-old woman presented to a chiropractic clinic with left lumbosacral pain with referral into the posterior aspect of the left thigh. The pain had started suddenly, 72 hours before the consultation as the patient was pulling a cable out of the swimming pool. The pain occasionally extended below the knee with forward bending of the lumbar spine. However, the patient did not report any numbness, paresthesia, or weakness in the lower extremities. The patient charac-

terized the nature of her complaint as a constant tension with an intensity of 6 of 10 that had gradually increased over time and interfered with her daily activities. There were no other known associated symptoms or red flags.

Inquiry into the patient's personal medical history revealed birth control pills as her only current medication. Her family medical history was unremarkable. She was a full-time student, working part-time as a lifeguard.

Upon examination, the patient's gait (standard, toe and heel walking) was unremarkable. Both active and passive lumbar ranges of motion were full and did not aggravate the pain. Bilateral lower limb neurological examination was normal (intact sensation to light touch, 5/5 motor strength and normal [+2] patellar and Achilles deep tendon reflexes). Lumbar and hip orthopedic examination could not reproduce the chief complaint. Palpation revealed pain and tenderness of the left piriformis and gluteus medius muscles. Joint restrictions were found in her thoracolumbar junction and bilateral sacroiliac joints.

Owing to the inability to reproduce the patient's chief complaint during the physical examination, the decision was made to send the patient for standard lumbar radiographs, which were interpreted as normal.

Based on the previous findings, a provisional diagnosis of acute myofascial syndrome of the left piriformis and gluteus medius muscles was made. For symptomatic relief, a trial consisting of soft tissue therapy to the left gluteus region and manipulative therapy to the thoracic, lumbar and sacroiliac joints was initiated.

The patient received 3 chiropractic treatments over 1 week, which led to an 80% improvement in pain intensity and frequency. Two days later, a sudden onset of severe abdominal pain localized to the left iliac region brought the patient to seek urgent medical attention. In addition, she reported low back pain with a feeling of heaviness and pain into her left thigh. Palpation of the abdominal region revealed pain that was aggravated by palpation of the left iliac and suprapubic regions. Lower limb examination revealed soreness at the lateral aspect of the left gluteal region. Left hip ranges of motion were complete with pain in external-internal rotation and discomfort at the end range of flexion-extension. Neurological testing was within normal limits and dorsalis pedis pulses were present bilaterally.

An ultrasound of the abdominal and pelvic regions and a radiograph of the left hip were performed, and interpreted as normal. Later during the day, the patient reported increased pain and heaviness into her left leg accompanied by stiffness into the left calf. Closer evaluation revealed edema into the left calf and decreased sensation affecting the entire left lower limb. A venous Doppler ultrasound of the left lower limb revealed reduction of the venous flow in the femoral vein area. Based on this particular finding, the decision was made to complete a second ultrasonography evaluation of the pelvis, which revealed an extensive thrombosis affecting the left femoral vein and iliac axis extending towards the vena cava. Blood work findings were consistent with the previous result and yielded high D-dimer level (a fibrin degradation product) (3068 ng/mL, normal = ≤ 243 ng/mL). Hypercoagulable workup excluded presence of factor V Leiden and prothrombin gene mutation. The patient was given heparin as an anticoagulation therapy and was discharged with the recommendation to discontinue oral contraceptive use.

Less than a week following her discharge, the patient presented to the emergency room with pain into the left posterolateral thoracic wall, which had been present for 4 days. A lung scintigraphy revealed hypoperfusion, consistent with bilateral pulmonary embolisms, affecting three different lung segments and obstructing 15% to 20% of the capillary bed. The extent of the lesion raised suspicion regarding a possible adverse reaction to the heparin anticoagulation therapy. Blood work revealed a low platelet level and Heparin-Platelet Factor 4–induced antibodies, confirming the diagnosis of heparin induced-thrombocytopenia. The patient was successfully treated with the insertion of a vena cava filter and Argatroban anticoagulation therapy.

Three months after the onset of symptoms, due to the multiple pulmonary embolisms, the patient still experienced episodes of thoracic pain and the lung scintigraphy revealed sustained lung perfusion defects.

Upon follow-up with a hematologist, the potential diagnosis of May-Thurner syndrome was considered based on the absence of blood dyscrasias and sustained anatomical changes found in the left common iliac vein at its junction with the right common iliac artery. [Table 1](#) highlights the timeline of events and proposed differential diagnoses.

Discussion

In light of the events mentioned above, primary care physicians should recognize the importance of con-

ducting a detailed history and physical examination when vascular peripheral involvement is suspected. The physician should screen the patient's past history for personal or family antecedents of vascular disease and known blood anomalies. Screening for potential risk factors and use of the Wells probability score (detailed in the "Diagnosis" section) should help clinicians estimate the likelihood of a severe vascular event occurring and make appropriate management decisions. Physical examination should include inspection for skin redness or discoloration, presence of edema, measurement for limb asymmetry, quality of pulses (auscultation and palpation), and identification of tenderness to palpation along vascular structures or within their proximity. Prompt detection of a potential vascular event is paramount, as it leads to better clinical outcomes. The following subsections are intended to provide readers with an understanding of the typical VTE patients' portrait and clinical approach to patient care.

Risk Factors

Virchow's Triad, which describes the various factors predisposing an individual to VTE, proposes that venous thromboses occur as a result of blood flow alterations, vascular endothelial injury, or alterations in the constitution of the blood.⁸ When at least 2 of the above factors coexist, it is likely that a VTE will develop. Clinical settings in which these conditions frequently occur are prolonged immobilization, trauma, surgery, infection (including HIV/AIDS) and the postpartum period.⁶ It is now accepted that the combination of stasis and hypercoagulability are crucial for a venous thrombosis to occur.⁹ Most cases of DVT are localized proximally in the left leg,¹ which raises the suspicion as to whether an anatomical component could be responsible for this phenomenon. Occasionally, the right common iliac artery overlies and compresses the left common iliac vein against the lumbar spine, increasing the risk of developing iliofemoral deep venous thrombosis. This anatomical variant has previously been described as the May-Thurner syndrome (MTS).¹⁰ This chronic compression leads to intimal hyperplasia, caused by deposition of elastin and collagen, which creates the potential for venous stasis and subsequent thrombosis.¹¹ Furthermore, a recent case-control study suggested that compression greater than 70% is required in order to be an associated factor for left sided DVTs.¹² Although MTS has been shown to be present in about 20% of the population, it is thought to account for only 2% to 3% of all lower extremity DVTs.¹⁰

Table 1 Timeline of Events and the Differential Diagnosis Suggested

Day	Event	Differential diagnoses according to file recording
1	Onset of left sided back pain	
4	1st visit to the chiropractor	Myofascial syndrome
11	Last visit to the chiropractor	
12	Onset of severe abdominal pain	
13	1st emergency visit	Ovarian cyst Ectopic pregnancy Hip fracture
14	Aggravation of low back and left leg pain Venous Doppler US High D-dimer level Heparin anticoagulation therapy started	Extensive thrombosis of left femoral and iliac veins
16	Patient is discharged from the hospital	
22	2nd Emergency visit for thoracic pain Lung scintigraphy: bilateral pulmonary embolisms Blood work: Identification of low platelet level and Heparin-Platelet Factor 4-induced antibodies Argatroban anticoagulation therapy added Installation of vena cava filter	Heparin-induced thrombocytopenia
34	Patient discharged from the hospital Coumadin anticoagulation therapy added	
53	3rd emergency visit for persistent thoracic pain	Sequelae from previous pulmonary embolisms
106	Hematology follow-up Restoration of normal leg venous flow Persistence of moderate parietal thickening and decreased lumen caliber into the external iliac vein	May-Thurner syndrome
120	Hematology follow-up Sustained lung perfusion defects	

US, ultrasound.

There are over 30 known associated risk factors for the development of the initial VTE, which are classified into genetic, acquired, environmental, and mixed factors.¹³ Recognized clinical risk factors account for about 75% of VTE cases, out of which 50% are factors associated with institutionalization.¹⁴ The proportion of patients with VTE categorized as idiopathic is about 25%,^{14,15} although other risk factors have been identified and reported with inconclusive results (eg, air pollution, high C-protein reactive level, hypertension, diabetes mellitus, smoking).¹³ Oral contraceptive therapy alone has been established as a moderate risk factor (odds ratio; 2-9) of DVT.¹⁶ Table 2 highlights the recognized risk factors for VTE.

Clinical Presentation

The typical clinical presentation, similar to the patient in this case report, is usually a young adult female with a history of oral contraceptive use, recent pregnancy, or recent prolonged travel.¹⁰ Although DVTs can occur asymptotically, its classic clinical presentation includes swelling, pain, warmth, and redness in the involved extremity. There are no signs or symptoms sensitive or specific to DVT, as trauma, infection,

ruptured Baker cyst, hematoma, and venous insufficiency can all present with similar clinical features.^{3,5} PE does occur when DVT suddenly breaks off and travels in the circulation to the lungs. The most common symptoms and signs of PE include dyspnea, chest pain, tachypnea, syncope, and cough. Less common symptoms and signs include fever, hemoptysis, cyanosis, hypotension, and shock.³ In addition, many patients have concomitant symptoms and signs of DVT and PE.³ There are no specific clinical history details or physical examination tests that health care practitioners can utilize to exclude DVT in a private practice setting. In the case discussed herein, the patient initially presented with low back pain, which raises an important question as to whether or not, what seemed to be mechanical low back pain could indeed have been an early sign of venous obstruction. While pain in the left iliac fossa is not part of the usual clinical presentation of a DVT, it has previously been reported in the literature in a case report of MTS.¹⁷

Diagnosis

Once a DVT is suspected, patients should be referred to the hospital, where more specific medical assessments

Table 2 Risk Factors for VTERisk factors^{9,16,27}

Strong risk (odds ratio >10)	Moderate risk (odds ratio 2–9)	Weak risk (odds ratio <2)
Surgery	Hospital/nursing home confinement	Increasing age
Major trauma	Previous VTE	Pregnancy
Hip/knee replacement	Malignant neoplasm	Immobility due to sitting
	Central venous catheter	Superficial vein thrombosis
	Pacemaker	
	Paresis	
	Oral contraceptive/hormone therapy	
	Thrombophilia	

VTE, venous thromboembolism.

can be performed. Clinical prediction rules based on symptoms, signs and risk factors such as the modified Wells score, have been developed to evaluate patient's pre-test clinical probability of a DVT.¹⁸ The modified Wells score categorizes patients into low probability of a DVT (<2 points; likelihood ratio 0.13), moderate probability of a DVT (2–6 points; likelihood ratio 1.82) and high probability of a DVT (>6 points; likelihood ratio 6.75).¹⁹ The objective diagnostic methods for DVT are venous compression ultrasonography, contrast venography, computed tomography venography or magnetic resonance imaging venography.⁶ When the pretest probability of a DVT is intermediate to high, compression ultrasonography should be the initial test. It is a simplified technique of examination limited to assess the compression of common femoral and popliteal veins.³ Ultrasonography achieves its best sensitivity (89%–96%) and specificity (94%–99%) in symptomatic patients with proximal thrombosis of the lower extremities.²⁰ A negative ultrasonography result alone is insufficient to exclude the diagnosis of a DVT, therefore, a D-dimer level assessment is also recommended.²¹ D-dimer is a small protein fibrin degradation fragment detectable in the blood after a blood clot is degraded by fibrinolysis. The D-dimer level correlates with the size of the thrombus and clot activity.⁵ Other methods such as computed tomography and magnetic resonance imaging are also useful but are generally limited by availability.⁵

The initial evaluation of patients with suspected pulmonary embolism includes a chest radiograph and electrocardiograph.³ Chest radiograph findings associated with pulmonary embolism include plate-like atelectasis, pleural effusion, and elevation of an hemidiaphragm.³ Changes on electrocardiographs may also be seen due to right heart strain.³ Multidetector computed tomography angiography is the diagnostic test of choice for PE. Its clinical validity is

similar to that of conventional pulmonary angiography and ventilation-perfusion scanning with a reported positive predictive value of 92% to 96% in patients with high pre-test probability.³

Hypercoagulable laboratory testing is generally indicated in presence of a primary VTE in patients under 50 years of age, a strong family history of VTE, or recurrent primary VTE and thrombophlebitis.⁶ Identification of genetic risk factors through blood work guides the development of appropriate management strategies.

Management

The primary objectives of DVT treatments are to prevent further clot extension, prevent PE and reduce the risk of recurrent thrombosis. Patients with a DVT or PE are acutely treated with an anticoagulant dose of one of the heparins (low-dose, low-molecular-weight heparins or low-dose unfractionated heparin).⁶ Evaluation of clinical risk factors for a VTE is relevant as it helps determine the duration of anticoagulation.⁵ In about 40% to 60% of white patients with a VTE, the thrombotic tendency can be linked to thrombophilia.²² This may be inherited (eg, factor V Leiden, prothrombin gene mutation and deficiencies of protein C, protein S, or antithrombin)²² or acquired (eg, antiphospholipid antibodies).²³ Venous thromboembolisms may be provoked by transient and reversible clinical risk factors such as surgery or estrogen therapy. They are associated with low risk of recurrence and require short-term anticoagulation therapy. On the other hand, long-term and permanent factors, such as antiphospholipid syndrome, hemiparesis from stroke, or an idiopathic VTE have a higher risk of recurrence and are indicative of longer term anticoagulation therapy.^{5,16} In the case of a DVT associated with MTS, systemic anticoagulation alone is not adequate, as it fails to address the underlying

anatomical compression.^{24,25} It is suggested that the anatomical defect should be repaired with the use of stents and balloon venoplasty.¹¹ Patients with a high clot burden should also undergo inferior vena cava filter placement.²⁵

Recurrence/Mortality

VTEs recur frequently, especially within the first 6 (10.1%) to 12 months (12.9%) and continue to recur for at least 10 years after the initial event.²⁶ Patients with a VTE and concomitant diseases such as neurologic diseases, paresis or malignant neoplasms are at increased risk for recurrence, while patients with transient or reversible risk factors are at lesser risk.²⁶ Among non-cancer patients with a VTE, there is an increased risk of mortality within the first 6 months after the event. More specifically, the 30-day and 1-year case-fatality rates have been estimated to 6.8 and 15.5% for a PE and to 1.8 and 11.1% for a DVT, respectively.¹

Limitations

Due to its nature, a case report does not allow for potential associations nor cause and effect. However, it may provide insight into clinical presentations that differ from what is usually expected. The patient experienced sudden pain following a physical effort and did get some relief from conservative chiropractic treatments. It is possible that the occurrence of the deep vein thromboses is purely coincidental and had nothing to do with the initial event and patient presentation. Furthermore, advanced imaging such as venography or magnetic resonance angiography was not performed although it could have strengthened the May-Thurner syndrome diagnosis and informed patient management. The reasons why are unknown but it seems reasonable to think that in the absence of family blood dyscrasias combined with the location of the thrombosis and the parietal thickening and decreased caliber found in the left common iliac vein at its junction with the right common iliac artery, the May-Thurner syndrome was the most probable diagnosis. Another reason may be a limited access to health care or a long waiting period to access imaging resources. In light of these limitations, caution must be made when applying the conclusions of a case report into clinical practice.

Conclusion

Although VTE is a common condition in the general population, its presence in low-risk individuals can be overlooked. In the presence of subtle initial clinical signs such as those the patient in this case report presented with, clinicians should keep a high index of suspicion for a DVT. Rapid identification of such clinical signs in association with a lack of objective examination findings warrants further evaluation due to potentially negative outcomes.

Funding Sources and Conflicts of Interest

No funding sources or conflicts of interest were reported for this study.

References

1. Naess IA, Christiansen SC, Romundstad P, Cannegieter SC, Rosendaal FR, Hammerstrom J. Incidence and mortality of venous thrombosis: a population-based study. *J Thromb Haemost* 2007;5:692–9.
2. Heit JA, Silverstein MD, Mohr DN, et al. The epidemiology of venous thromboembolism in the community. *Thromb Haemost* 2001;86:452–63.
3. Wilbur J, Shian B. Diagnosis of deep venous thrombosis and pulmonary embolism. *Am Fam Physician* 2012;86:913–9.
4. Fowkes FJ, Price JF, Fowkes FG. Incidence of diagnosed deep vein thrombosis in the general population: systematic review. *Eur J Vasc Endovasc Surg* 2003;25:1–5.
5. Ho WK. Deep vein thrombosis—risks and diagnosis. *Aust Fam Physician* 2010;39:468–74.
6. Veller MG, Pillai J. Lower-limb venous thrombosis. *CME* 2009;27:306–11.
7. Kearon C. Natural history of venous thromboembolism. *Circulation* 2003;107:122–30.
8. Dickson BC. Venous thrombosis: on the history of Virchow's triad. *Univ Tor Med J* 2004;81:166–71.
9. Martinelli I, Bucciarelli P, Mannucci PM. Thrombotic risk factors: basic pathophysiology. *Crit Care Med* 2010;38:S3–9.
10. Peters M, Syed RK, Katz M, et al. May-Thurner syndrome: a not so uncommon cause of a common condition. *Proc (Bayl Univ Med Cent)* 2012;25:231–3.
11. Kalu S, Shah P, Natarajan A, Nwankwo N, Mustafa U, Hussain N. May-Thurner syndrome: a case report and review of the literature. *Case Rep Vasc Med* 2013;2013:740182.
12. Narayan A, Eng J, Carmi L, et al. Iliac vein compression as risk factor for left- versus right-sided deep venous thrombosis: case-control study. *Radiology* 2012;265:949–57.
13. Lijfering WM, Rosendaal FR, Cannegieter SC. Risk factors for venous thrombosis—current understanding from an epidemiological point of view. *Br J Haematol* 2010;149:824–33.

14. Heit JA, O'Fallon WM, Petterson TM, et al. Relative impact of risk factors for deep vein thrombosis and pulmonary embolism: a population-based study. *Arch Intern Med* 2002;162:1245–8.
15. White RH. The epidemiology of venous thromboembolism. *Circulation* 2003;107:I4–8.
16. Anderson Jr FA, Spencer FA. Risk factors for venous thromboembolism. *Circulation* 2003;107:I9–I16.
17. De Bast Y, Dahin L. May-Thurner syndrome will be completed? *Thromb Res* 2009;123:498–502.
18. Wells PS, Anderson DR, Rodger M, et al. Evaluation of D-dimer in the diagnosis of suspected deep-vein thrombosis. *N Engl J Med* 2003;349:1227–35.
19. Wells PS, Anderson DR, Rodger M, et al. Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. *Thromb Haemost* 2000;83:416–20.
20. Wells PS, Lensing AW, Davidson BL, Prins MH, Hirsh J. Accuracy of ultrasound for the diagnosis of deep venous thrombosis in asymptomatic patients after orthopedic surgery. A meta-analysis. *Ann Intern Med* 1995;122:47–53.
21. Johnson SA, Stevens SM, Woller SC, et al. Risk of deep vein thrombosis following a single negative whole-leg compression ultrasound: a systematic review and meta-analysis. *JAMA* 2010;303:438–45.
22. Lensing AW, Prandoni P, Prins MH, Buller HR. Deep-vein thrombosis. *Lancet* 1999;353:479–85.
23. Wahl DG, Guillemin F, de Maistre E, Perret-Guillaume C, Lecompte T, Thibaut G. Meta-analysis of the risk of venous thrombosis in individuals with antiphospholipid antibodies without underlying autoimmune disease or previous thrombosis. *Lupus* 1998;7:15–22.
24. Suwanabol PA, Tefera G, Schwarze ML. Syndromes associated with the deep veins: phlegmasia cerulea dolens, May-Thurner syndrome, and nutcracker syndrome. *Perspect Vasc Surg Endovasc Ther* 2010;22:223–30.
25. Moudgill N, Hager E, Gonsalves C, Larson R, Lombardi J, DiMuzio P. May-Thurner syndrome: case report and review of the literature involving modern endovascular therapy. *Vascular* 2009;17:330–5.
26. Heit JA, Mohr DN, Silverstein MD, Petterson TM, O'Fallon WM, Melton 3rd LJ. Predictors of recurrence after deep vein thrombosis and pulmonary embolism: a population-based cohort study. *Arch Intern Med* 2000;160:761–8.
27. Heit JA, Silverstein MD, Mohr DN, Petterson TM, O'Fallon WM, Melton III LJ. Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study. *Arch Intern Med* 2000;160:809–15.