CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 6 (2015) 55–57

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Upper extremity deep vein thrombosis with tourniquet use

Karan Desai[a,b], Trish P. Dinh[a,b], Susan Chung[a,b], Yvonne N. Pierpont[a,b], Deepak K. Naidu[a,b], Wyatt G. Payne[a,b,*]

[a] Institute for Tissue Regeneration, Repair, & Rehabilitation, Bay Pines VA Health Care System, Bay Pines, FL 33744, United States
[b] Division of Plastic Surgery, University of South Florida, Residency Programs, USF Health (STC), 7th Floor, 2 Tampa General Circle, Tampa, FL 33606, United States

ARTICLE INFO

Article history:
Received 28 August 2014
Received in revised form 9 November 2014
Accepted 12 November 2014
Available online 20 November 2014

Keywords:
Deep vein thrombosis
Tourniquet
Upper extremity
Trauma

ABSTRACT

INTRODUCTION: Upper extremity deep vein thrombosis is an increasingly important clinical finding with significant morbidity and mortality. The condition may be under-diagnosed in trauma and surgery settings.
PRESENTATION OF CASE: We present a case of upper extremity thrombosis with venous congestive symptoms secondary to the use of an operative tourniquet. A literature review and discussion of the causes of upper extremity deep vein thrombosis and the pathophysiological disturbances seen with tourniquet use are presented.
DISCUSSION: Upper extremity deep venous thrombosis is uncommon. In this case the likely cause was operative tourniquet use.
CONCLUSION: Operative tourniquet may be a risk factor in upper extremity deep vein thrombosis.

1. Introduction

Upper extremity (UE) deep venous thrombosis (DVT) accounts for 4–10% of the 5–20 million cases of DVT that occur yearly in the United States.[1] As with DVT of the lower extremity, thrombosis in the upper extremity occurs due to hypercoagulability, venous stasis, or endothelial injury.[2] Higher incidences of pulmonary emboli (PE), as high as 33% in patients with UEDVT, have been reported following an initial thrombus.[3] Iatrogenic UEDVT requires further analysis to correct the offending problem and render proper treatment. The condition may be under-diagnosed in trauma and surgery settings when arm swelling and pain are common. We present a case of UEDVT of the upper extremity as the likely result of prior trauma with subsequent use of upper extremity surgical tourniquet.

2. Presentation of case

An 83 year old male with no prior history of bleeding or clotting disorder presented to the Emergency Department with a limited left forearm hematoma secondary to relatively minor blunt trauma after having his forearm caught in a closing door two weeks prior. The patient noted no significant history of deep vein thrombosis, pulmonary emboli, or any additional medical comorbidities or medications that would increase risk of thromboembolus. Plain film X-ray revealed no fracture. No neurovascular or musculoskeletal deficits were appreciated on physical exam. Local swelling of the left forearm and a 6 cm fluid mass were observed/palpated. No additional swelling of the extremity was noted. Patient was brought to operating room for expanding hematoma evacuation and exploration of upper extremity wound. Under regional Bier block with operative tourniquet in place at 250 mm Hg, surgical exploration revealed subcutaneous old and new hematoma. The hematoma was evacuated with non-specific findings of mild oozing without a specific bleeding point. The forearm was irrigated with pulse lavage and hemostasis was obtained. No other injuries were noted. Total tourniquet time was 35 min, with total sedation time 50 min. The tourniquet was released, hemostasis was assured and the wound was closed in layers over a #10 Jackson-Pratt drain and soft dressing applied. Blood pressure during surgical exploration remained normotensive. During the post-operative period, drain output was minimal (Figs. 1 and 2).

On post-operative day one, the patient was discharged to home with minimal residual swelling of the left upper extremity. On post-operative day 4, the patient returned for follow-up and was noted to have significant left upper extremity (LUE) swelling extending from the palm to the axilla. There did not appear to be free fluid on exam. Venous duplex scan demonstrated evidence of acute thrombosis of the left internal jugular, subclavian and axillary veins. No acute thrombus was appreciated in the lower extremities.

* Corresponding author at: BPVAMC/Surgical Service, 112, PO Box 5005, 10,000 Bay Pines Blvd, Bay Pines, FL 33744, United States. Tel.: +1 727 398 9385; fax: +1 727 398 9584.
E-mail address: Wyatt.Payne@va.gov (W.G. Payne).

http://dx.doi.org/10.1016/j.ijscr.2014.11.055

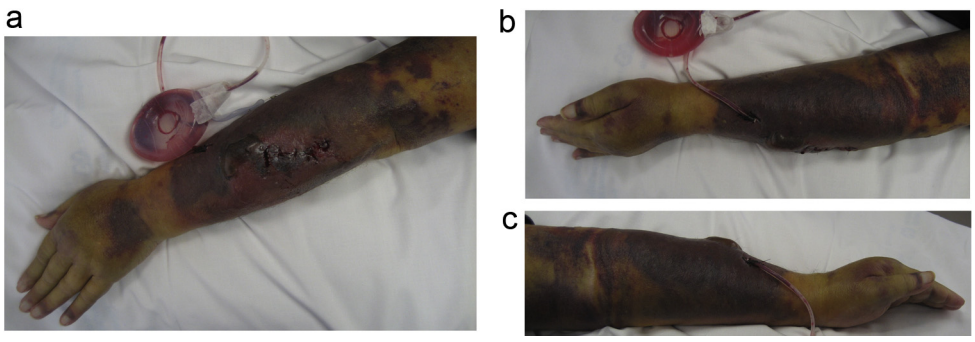

Fig. 1. (a–c): Photos taken at time of presentation with upper extremity deep vein thrombosis demonstrating significant diffuse swelling of upper extremity. No clinical evidence of recurrent hematoma.

Hypercoagulable workup, including platelet associated antibody panel, antithrombin III, Protein C, Protein S, haptoglobin, lupus anticoagulant, cardiolipin antibodies, factor V Leiden and prothrombin gene mutation, was negative. The patient exhibited no signs or symptomatology suspicious for pulmonary embolus (PE). Treatment included heparin drip followed by transition to coumadin. Healing progressed without difficulty. The edema subsided over the course of the following 3 weeks. Coumadin was continued for 6 months and then discontinued. Follow-up over the ensuing 18 months revealed no further issues.

3. Discussion

Upper extremity deep vein thrombosis (UEDVT) commonly refers to thrombosis of the axillary and/or subclavian veins.[4] Based on the etiology of thrombus formation, UEDVT is either classified as primary or secondary. Primary UEDVT is a rare disorder, resulting from thrombosis of the arm veins without evident predisposing factors in the patient's history.[5] Pathogenesis of primary UEDVT are venous thoracic outlet syndrome, effort-related thrombosis (Paget-Schroetter Syndrome), and idiopathic.[6]

Approximately 66–80% of all reported UEDVT cases are secondary in nature.[6] UEDVT is classified as secondary when endogenous or exogenous risk factors are known.[5] Origins such as central venous catheters (CVC), surgery or trauma of the arm or shoulder, and hypercoagulable states can usually be identified.[1,3,4,6] Both malignancy-related and genetic hypercoagulable states have been known to be etiologic factors.[2]

Multiple authors have reported correlations between recent upper extremity operative procedures and upper extremity DVT.[1,3,7,8] However, a large registry of UEDVT discovered that major surgical procedures, a conventional risk factor for lower-extremity DVT, did not predispose upper extremity DVT in patients who did not have a central venous catheter.[9]

Arterial tourniquets are commonly used to achieve a bloodless operative field. Deep venous thrombosis has been reported at a higher incidence in a series of patients when lower extremity tourniquets were used to achieve a bloodless surgical field.[10]

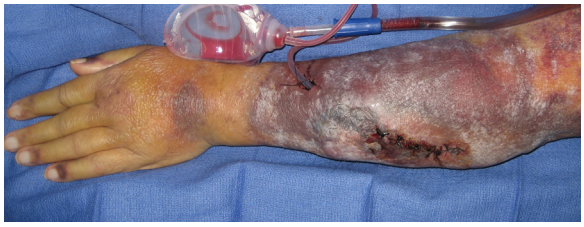

Fig. 2. One week after initiation of anticoagulation therapy for upper extremity deep being thrombosis. Photo demonstrates significant improvement in upper extremity edema.

Additionally, a number of reports describe new onset DVT and pulmonary emboli (PE) as attributed to surgical tourniquet use on the lower extremities.[11] Because these reported patients suffered fatal consequences secondary to DVT/PE, the use of lower extremity tourniquet in high risk patients, such as those with lower extremity trauma, prolonged immobilization, or history of venous thromboembolism, may be contraindicated.[12] No reports of UEDVT as a consequence of upper extremity traumatic injury and use of operative tourniquet were found upon review of the literature.

Two weeks prior to the tourniquet use, the patient sustained a crush injury resulting in a local hematoma. Crush injuries result in possible muscle injury, endothelial damage, and bleeding.[13] The loss of vascular integrity exposes the collagen-rich wall to platelet-aggregation factors, activating the clotting cascade and causing the release of procoagulant proteins. These proteins include tissue factor (TF), cytokines, and surface adhesion molecules that promote leukocyte adhesion and thrombosis.

A number of physiologic disturbances that take place with tourniquet usage may predispose to a thrombotic state. Application of a tourniquet leads to venous stasis from the hand to the arm, violating the third aspect of Virchow's triad.[14] Circulating TF, released by activation of leukocytes, accumulate in areas of stasis.[13] Tourniquet use then induces an increase in circulating thrombotic markers of plasmin D-dimer, tissue plasminogen activator, angiotensin-converting enzyme, antithrombin-III and protein C.[15] Furthermore, the activation pathways and feedback regulation shared by inflammation and hemostasis are intensified by tourniquet use. Technical errors during tourniquet placement may exacerbate the problem as well. Slow inflation rates of pneumatic tourniquets can lead to cessation of venous outflow while concomitantly allowing arterial inflow, resulting in venous congestion. The same phenomenon can also occur during deflation of the tourniquet, which should also be performed as quickly as possible.[8] These effects promote systemic hypercoagulability.

Numerous disturbances of the coagulation cascade have been described both intraoperatively and after deflation of a tourniquet. In animal studies, a hypercoagulable state was observed 60 min after placement of a circumferential Esmarch bandage.[16] Platelet aggregation was significantly elevated due to tissue compression of the limb. Human studies have shown that the pain induced by initial tourniquet inflation increases levels of catecholamines and serotonin intravascularly, thereby increasing platelet aggregation, without an increase in fibrinolysis.[17] The combination of these factors predisposes to a procoagulant state in the compressed extremity and likely contributes to DVT formation.

There is no consensus on a "safe" period of time for upper extremity tourniquet, although a commonly utilized guideline suggests compression for no longer than 120 min, mostly to avoid excessive warm ischemia time. This may also minimize stasis time before allowing reperfusion.[18]

Our patient had no previous history of DVT or prior risk factors for hypercoagulability and did not have a central venous catheter insertion. His Caprini Risk Assessment model (RAM) score was 5; age > 75, 3 points + major surgery, 2 points; under the venous RAM score for consideration of chemical prophylaxis. During the time line of forearm injury until surgical intervention (two weeks), the patient did not exhibit signs or symptoms of upper extremity DVT. The presentation with an extensive DVT four days post-operatively appears to be due to the use of the intra-operative tourniquet. The initial crush injury, though relatively minor in severity, may have led to a predisposed hypercoagulable state exacerbated by tourniquet use. Surgeons should be aware of the potential risk of upper extremity tourniquet use in patients, especially those with recent traumatic injury. This combination may lead to DVT occurrence and potential risks of DVT associated complications.

4. Conclusion

Deep vein thrombosis is an increasing problem in the United States. There are a number of mechanisms, which cause secondary thrombosis including malignancy, trauma, central venous catheters, and circumferential compression, as with surgical tourniquets. The various pro-coagulant disturbances in the coagulation cascade and venous stasis experienced with the use of a tourniquet necessitate greater attention to patient factors which could increase the risk of upper extremity DVT. If tourniquet use is desirable or unavoidable in patients with a history of hypercoagulability, cancer, operative area trauma, or indwelling intravenous catheters, a high index of suspicion for DVT and its symptoms should be maintained. If not necessary, tourniquet use should be minimized, especially with these risk factors. While the risk of thrombosis may be a minor consideration when compared to the ease of operation in a bloodless field, surgeons cannot invariably deny that upper extremity tourniquet usage may lead to VTE in high-risk patients.

Caprini VTE Risk Assessment scores should be utilized as an indicator of those at significant risk for VTE.[19,20] Though the model/score does not assess tourniquet use or upper extremity surgical procedures specifically (other than central venous access), perhaps these factors should be considered in each individual's risk assessment.

We report here a case of upper extremity deep vein thrombosis, status posttraumatic crush injury with use of operative tourniquet. Our literature search revealed no prior reports of similar cases.

Conflict of interest

None.

Funding

This material is the result of work supported with resources and the use of facilities at the Bay Pines VA Healthcare System. *Disclaimers*: The contents of this work do not represent the views of the Department of Veterans Affairs or the United States Government.

Ethical approval

Consent for surgery was obtained. There is no identifying factors used in this case report.

Author contributions

Karan Desai, MD – Case Report authorship, writing of paper; Trish Dinh, BS – Research of references, writing of paper; Susan Chung, MD – Writing of paper, addition of references, study concept; Yvonne Pierpont, MD – Writing of paper, addition of references, study concept; Deepak K. Naidu, MD – Case Report authorship, writing of paper; Wyatt G Payne, MD – Study concept/design, edits.

References

1. Spiezia L, Simioni P. Upper extremity deep vein thrombosis. *Intern Emerg Med* 2010;**5**(2):103–9.
2. Martinelli I, Bucciarelli P, Mannucci PM. Thrombotic risk factors: basic pathophysiology. *Crit Care Med* 2010;**38**(2 Suppl.):S3–9.
3. Prandoni P, Polistena P, Bernardi E, Cogo A, Casara D, Verlato F, et al. Upper-extremity deep vein thrombosis. Risk factors, diagnosis, and complications. *Arch Intern Med* 1997;**157**(1):57–62.
4. Joffe HV, Goldhaber SZ. Upper-extremity deep vein thrombosis. *Circulation* 2002;**106**(14):1874–80.
5. Czihal M, Hoffman U. Upper extremity deep venous thrombosis. *Vasc Med* 2011;**16**(3):191–202.
6. Kucher N. Clinical practice. Deep-vein thrombosis of the upper extremities. *N Engl J Med* 2011;**364**(9):861–9.
7. Blom JW, Doggen CJ, Osanto S, Rosendaal FR. Old and new risk factors for upper extremity deep venous thrombosis. *J Thromb Haemost* 2005;**3**(11):2471–2.
8. Marinella MA, Kathula SK, Markert RJ. Spectrum of upper-extremity deep venous thrombosis in a community teaching hospital. *Heart Lung* 2000;**29**(2):113–7.
9. Joffe HV, Kucher N, Tapson VF, Goldhaber SZ. Upper-extremity deep vein thrombosis: a prospective registry of 592 patients. *Circulation* 2004;**110**(12):1605–11.
10. Abdel-Salam A, Eyres KS. Effects of tourniquet during total knee arthroplasty. A prospective randomised study. *J Bone Jt Surg Br* 1995;**77**(2):250–3.
11. McGrath BJ, Hsia J, Epstein B. Massive pulmonary embolism following tourniquet deflation. *Anesthesiology* 1991;**74**(3):618–20.
12. Kroese AJ, Stiris G. The risk of deep-vein thrombosis after operations on a bloodless lower limb. A venographic study. *Injury* 1976;**7**(4):271–3.
13. Saha P, Humpheries J, Modarai B, Mattock K, Waltham M, Evans CE, et al. Leukocytes and the natural history of deep vein thrombosis: current concepts and future directions. *Arterioscler Thromb Vasc Biol* 2011;**31**(3):506–12.
14. Hall IR, Lo TS, Nolan J. Deep vein thrombosis in the arm following transradial cardiac catheterization: an unusual complication related to hemostatic technique. *Catheter Cardiovasc Interv* 2004;**62**(3):346–8.
15. Estebe JP, Davies JM, Richebe P. The pneumatic tourniquet: mechanical, ischaemia-reperfusion and systemic effects. *Eur J Anaesthesiol* 2011;**28**(6):404–11.
16. Yoshida WB, Thomazini IA, Carvalho I, Lastória S, Curi PR, Maffei FH. Platelet activation following application of an Esmarch bandage and tourniquet in rabbits. *Braz J Med Biol Res* 1989;**22**(9):1091–3.
17. Kohro S, Yamakage M, Arakawa J, Kotaki M, Omote T, Namiki A. Surgical/tourniquet pain accelerates blood coagulability but not fibrinolysis. *Br J Anaesth* 1998;**80**(4):460–3.
18. Cox C, Yao J. Tourniquet usage in upper extremity surgery. *J Hand Surg Am* 2010;**35**(8):1360–1.
19. Pannucci CJ, Bailey SH, Dreszer G, Fisher Wachtman C, Zumsteg JW, Jaber RM, et al. Validation of the Caprini risk assessment model in plastic and reconstructive surgery. *J Am Coll Surg* 2011;**212**(1):105–12.
20. Pannucci CJ, Barta RJ, Portschy PR, Dreszer G, Hoxworth RE, Kalliainen LK, et al. Assessment of postoperative venous thromboembolism risk in plastic surgery patients using the 2005 and 2010 Caprini risk score. *Plast Reconstr Surg* 2012;**130**(2):343–53.