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INTERMEDIATE

CASE REPORT: CLINICAL CASE

Acute Deep Vein Thrombosis in a Cyclist With Iliac Vein Compression From Psoas Muscle Hypertrophy

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ABSTRACT

A 22-year-old avid cyclist presented with 1 month of right lower extremity pain and associated swelling. Subsequent imaging demonstrated an extensive acute deep vein thrombosis (DVT) in the setting of right iliac vein compression from psoas muscle hypertrophy. We present an unusual risk factor for DVT among cyclists. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:1080–1085) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 22-year-old man presented with 1 month of increasing pain and swelling involving his right lower extremity (RLE), from the calf to the groin. He was notably an avid cyclist, biking 250 to 300 miles per week. His initial vital signs included a temperature of 37.1°C, heart rate 84 beats/min, and blood pressure 129/58 mm Hg. On examination, RLE edema from his calf to thigh was noted (**Figure 1**) with intact femoral, popliteal, and pedal pulses, bilaterally equal and there was no significant tenderness on palpation.

LEARNING OBJECTIVES

- To describe a unique case of psoas muscle hypertrophy leading to DVT.
- To describe the differential diagnosis of VTE in young athletes.
- To review the current guideline recommendations for return-to-play in athletes following VTE.
- To describe the challenges associated with anticoagulation in athletes.

PAST MEDICAL HISTORY

The patient reported no significant past medical history, including personal or family history of venous thromboembolism (VTE). He also reported no previous significant edema, injury, or infection in the RLE.

DIFFERENTIAL DIAGNOSIS

Given the significant unilateral RLE edema in an otherwise healthy person, deep vein thrombosis (DVT) was the highest on the differential, but other considerations

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included lymphedema, malignancy, hematoma, and infection.

The differential diagnosis for a VTE in an otherwise healthy young athlete is presented in **Table 1**. Examples of specific risk factors for VTE in athletes include Paget-Schroetter syndrome in athletes with repetitive upper extremity movements (eg, baseball, weightlifting), tissue injury from contact sports, immobilization after injury or orthopedic surgery, polycythemia from altitude training or exogenous red blood cell administration/stimulation (eg, blood doping, erythropoietin use), and possible hypercoagulability from anabolic steroids or compounds in unregulated workout supplements.

INVESTIGATIONS

A RLE venous duplex ultrasound showed extensive occlusive thrombus in the proximal, mid, and distal femoral, popliteal, profunda femoris, peroneal, and posterior tibial veins. A venous-phase computed tomography (CT) scan showed moderate compression of the right common iliac vein (RCIV) between a hypertrophied right psoas muscle, right common iliac artery, and L5 vertebral body (**Figure 2A**). There was also an additional site of distal compression of the RCIV between the right internal iliac artery and psoas muscle (**Figure 2B**), and a filling defect of the right superficial femoral vein (RSFV) (**Figure 2C**). A comparison of venous-phase CT between the current case and an age/sex/weight-matched control is presented in **Figure 3**.

MANAGEMENT

The patient was initially started on anticoagulation with enoxaparin 1 mg/kg twice daily and was transitioned to apixaban 10 mg twice daily for 7 days, followed by apixaban 5 mg twice daily before discharge. He was advised to undergo a "temporary detraining period" from cycling for at least 2 to 3 months given concerns of venous compression from psoas muscle hypertrophy. At 6 months follow-up, the patient reported continual RLE swelling and pain in his leg with exertion when he returned to gradual exercise. A repeat CT abdomen/pelvis at 6 months was notable for unchanged RCIV compression between the hypertrophic psoas muscle and L5 vertebral body with partial recanalization of the RSFV (Supplemental Figure 1). A thrombophilia panel obtained at this time was notable Factor V Leiden gene mutation (homozygous). Other hypercoagulability testing was normal (protein C, antithrombin, prothrombin G20210A gene mutation, anti-B(2) glycoprotein 1 AB immunoglobulin (Ig)M+IgG, anti-cardiolipin AB IgM+IgG and lupus anticoagulant) except for protein S activity (46%, normal 70%-150%). However, the panel was performed while the patient was on apixaban, which could have affected some lab assays (eg, protein S, protein C, lupus anticoagulant), so the implications of these values remain unknown. Therefore, the etiology of the DVT in the current case was thought to be a combination of extrinsic compression from psoas muscle hypertrophy and possible vascular injury from repetitive cycling in a patient with elevated baseline risk from inherited coagulopathies (factor V Leiden).

ABBREVIATIONS AND ACRONYMS

CT = computed tomography
DVT = deep vein thrombosis
Ig = immunoglobulin
PE = pulmonary embolism
RCIV = right common iliac vein
RLE = right lower extremity
VTE = venous thromboembolism

Given his continual symptoms, the decision was made to pursue invasive contrast venography with intravascular ultrasound. His pre-intervention venogram was notable for a smooth tapered stenosis of the RCIV (**Figure 4A,** Video 1), and intravascular ultrasound confirmed a slit-like lesion of the RCIV thought to be secondary to extrinsic compression (**Figure 4B**). Serial balloon dilation was performed with a 12 × 40-mm and then 14 × 40-mm balloon (**Figure 4C**). A post-intervention venogram showed minimal improvement in the RCIV stenosis (**Figure 4D,** Video 2). The etiology of his stenosis was thus attributed to extrinsic compression from excessive psoas muscle hypertrophy. A venous stent was not placed at the time of intervention given the potential morbidity related to stent complications (eg, compression by the hypertrophied muscle) in a young athlete. The patient was restarted on therapeutic anticoagulation, with a plan to reassess the risks and benefits of venous intervention if symptoms did not improve in the foreseeable future.

DISCUSSION

We describe the first reported case of DVT in the setting of iliac compression from psoas muscle hypertrophy in a young competitive cyclist. There have been rare prior reports among older athletes (>50 years of age); however, both were in the left lower extremity with unclear underlying mechanisms.[1,2] Young cyclists often develop psoas muscle hypertrophy from training,[3] and therefore this should be considered in the differential for this patient population if they present with DVT.

In athletes who develop a VTE, there are currently no discrete recommendations provided for return-to-play in VTE guidelines[4] or among specific return-to-play guidelines in athletes with cardiovascular conditions.[5] This remains an important void in the

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FIGURE 1 Significant Lower Extremity Edema of the Right Thigh in the Setting of Acute Deep Vein Thrombosis

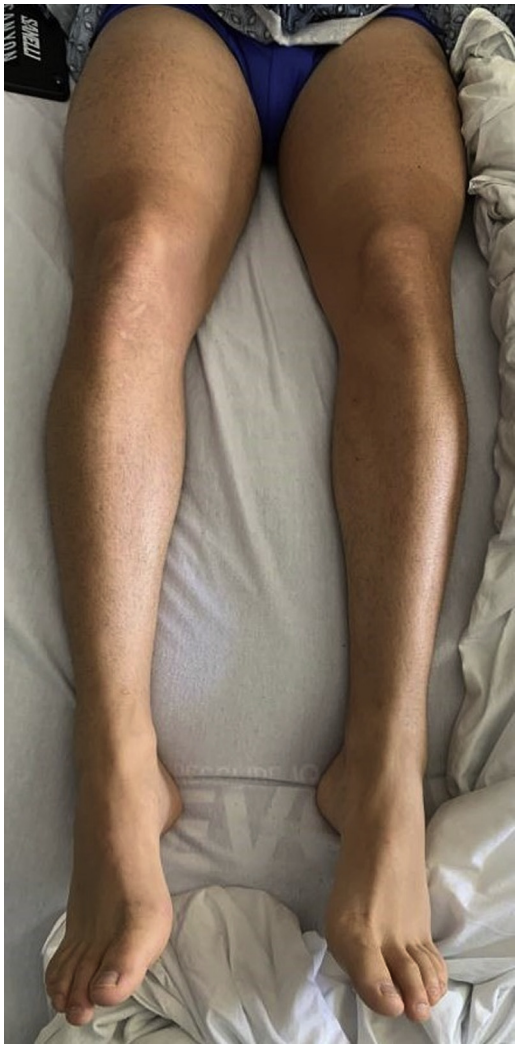

TABLE 1 Risk Factors for Venous Thromboembolism in Athletes

Stasis
Anatomic abnormalities (eg, May-Thurner syndrome, Paget-Schroetter syndrome)
Prolonged immobility (eg, prolonged travel to sporting events)
Polycythemia (eg, altitude training, dehydration, exogenous erythropoietin use, blood doping)
Hypercoaguability
Inherited thrombophilia disorders (eg, Factor V Leiden, prothrombin gene mutation, protein C and S deficiency)
Acquired hypercoagulability
Medication effect (eg, oral contraceptives, performance-enhancing supplements)
Obesity
Pregnancy
Malignancy
Autoimmune disease
Endothelial damage
Surgery
Trauma/tissue injury
Smoking
Medical disorders (eg, sickle cell disease)

current guidelines given DVT/pulmonary embolism (PE) can occur in both amateur and professional athletes, and because PE can be a cause of sudden cardiac death in young athletic populations.

The management of DVT/PE in athletes is also challenging given the inherent risks of anticoagulation with contact and noncontact sports associated with a possibility of incurring bodily injury (eg, cycling, climbing, hang gliding). In these cases, shared-decision making between the athlete and their providers is imperative to convey the risks and the benefits of anticoagulation and continued exercise. Previous studies have proposed regimens of intermittent anticoagulation for athletes at risk for thromboembolism[6]; however, the long-term efficacy of these strategies remains unknown.

FOLLOW-UP

The patient continues on anticoagulation consisting of apixaban 5 mg twice daily. His symptoms have markedly improved with decreasing his frequency of cycling, other than mild RLE edema.

CONCLUSIONS

We present a case of DVT in the setting of RCIV compression from psoas muscle hypertrophy in an avid cyclist. Given the implications of DVT/PE in this population, this potential risk factor should be considered in cyclists and other athletes presenting with VTE without a clear cause.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Baggish has received funding from the National Institutes of Health/National Heart, Lung, and Blood Institute, the National Football Players Association, and the American Heart Association and receives compensation for his role as team cardiologist from the U.S. Olympic Committee/US Olympic Training Centers, U.S. Soccer, U.S. Rowing, the New England Patriots, the Boston Bruins, the New England Revolution, and Harvard University. Dr Weinberg is a consultant for Magneto thrombectomy solutions and principal investigator for STRIKE-PE- Penumbra, Inc. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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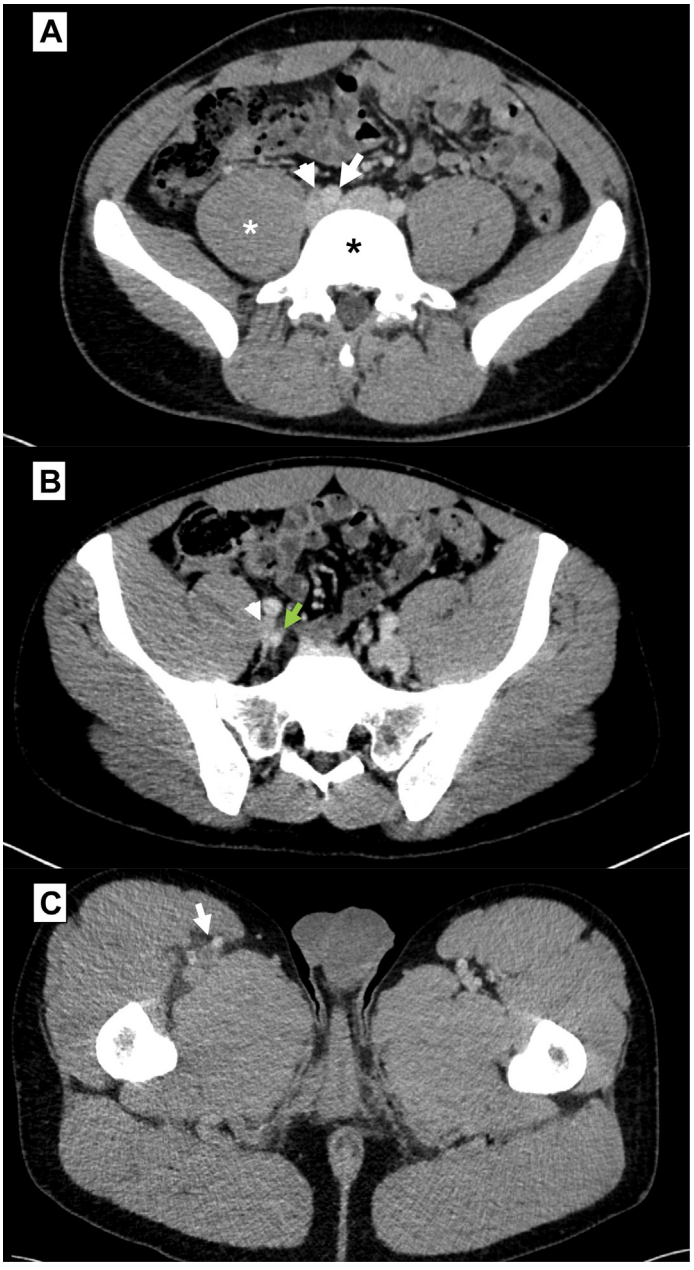
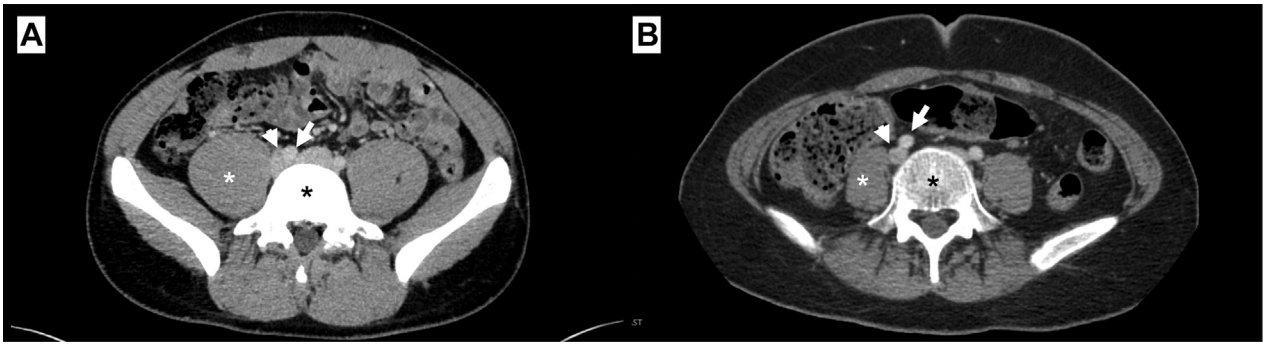
FIGURE 2 **Computed Tomography Scan Demonstrating Right Common Iliac Vein Compression From Psoas Muscle Hypertrophy**



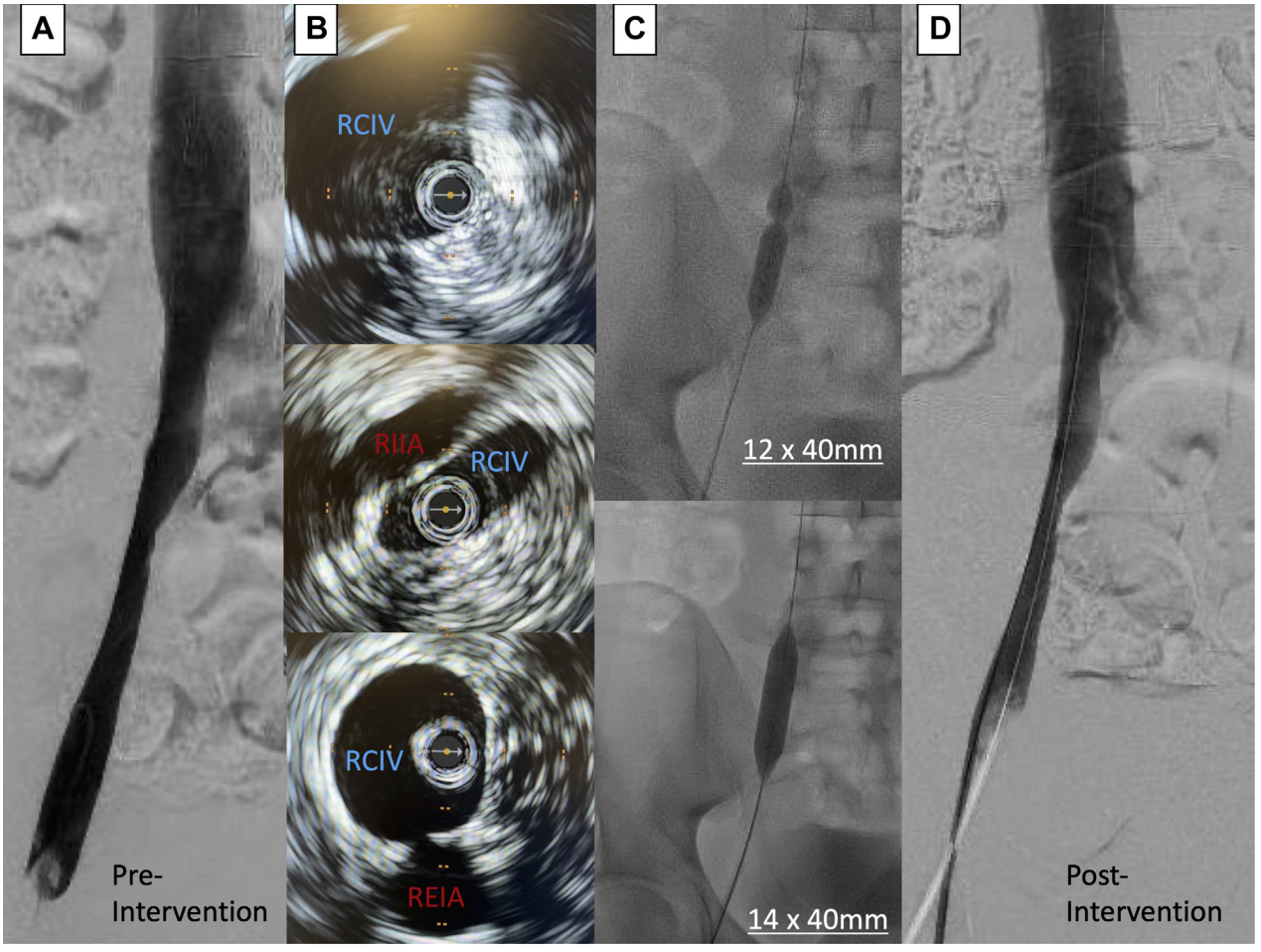
Venous phase contrast-enhanced computed tomography images in axial plane at the time of initial presentation **(A to C)** demonstrate moderate compression of the right common iliac vein **(A) (arrowhead)** between the hypertrophic psoas muscle **(white asterisk)**, right common iliac artery **(arrow)**, and the L5 vertebral body **(black asterisk)**. **(B)** There was another site of distal right common iliac vein **(arrowhead)** compression between the right internal iliac artery **(green arrow)** and psoas muscle. Acute deep vein thrombosis **(C)** was seen as a filling defect within the expanded right superficial femoral vein **(arrow)** with adjacent perivenular fat stranding.

FIGURE 3 Comparison of Psoas Muscle and Femoral Vessel Anatomy Between the Current Case and a Matched Control



Venous phase contrast-enhanced computed tomography images in axial plane comparing the current case **(A)** with an age/sex/weight-matched control **(B)**. The current case **(A)** showing moderate compression of the right common iliac vein (RCIV) **(arrowhead)** between the hypertrophic psoas muscle **(white asterisk)**, right common iliac artery (RCIA) **(arrow)**, and the L5 vertebral body **(black asterisk)**. The age/sex/weight-matched control **(B)** showing a normal psoas anatomy **(white asterisk)** without compression of the RCIV **(arrowhead)**, RCIA **(arrow)**, and L5 vertebral body **(black asterisk)**.

FIGURE 4 Images From Right Common Iliac Venogram With Intravascular Ultrasound and Balloon Dilation



(A) Pre-intervention venogram showing a slit-like stenosis of the RCIV. **(B) Top** = proximal RCIV proximal to the stenosis, **middle** = mid RCIV with slit-like stenosis from extrinsic compression, **bottom** = distal RCIV. **(C) Top** = balloon venoplasty using 12 × 40-mm balloon. **Bottom** = balloon venoplasty using 14 × 40-mm balloon. **(D)** Post-intervention venogram showing persistent stenosis of the RCIV. RCIV = right common iliac vein; REIA = right external iliac artery; RIIA = right internal iliac artery.

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KEY WORDS anticoagulation, athlete, deep vein thrombosis, exercise, venous thromboembolism

APPENDIX For supplemental videos and a figure, please see the online version of this paper.