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A giant portal vein thrombosis as a complication of cryotherapy

Kriyoterapinin bir komplikasyonu olarak dev portal ven trombozu

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ABSTRACT

Portal vein thrombosis (PVT) is frequently observed as a complication of certain malignant or benign conditions. In this article, we report a 42-year-old male patient with no history of previous illness who applied to our emergency department due to severe abdominal pain in all four quadrants. One week prior to the incident, the patient had started whole-body cryotherapy as a remedy for shoulder pain. Routine and detailed etiology-oriented laboratory tests showed no abnormalities. The patient was diagnosed with acute PVT based on abdominal computed tomography (CT) scan findings. The patient was started on intravenous heparin infusion, after which the state of thrombosis was reassessed with follow-up CT scans. When the thrombus receded, oral intake was started. The patient was followed-up for several days and discharged with instructions for the use of low-molecular-weight heparin. Portal vein thrombosis may be an acute complication of whole-body cryotherapy, as discussed in this case.

Keywords: Abdominal pain; portal vein thrombosis; shoulder pain; whole-body cryotherapy.

ÖZ

Portal ven trombozu (PVT) sıklıkla belirli malign veya benign durumların bir komplikasyonu olarak görülür. Bu yazıda, geçmiş hastalık öyküsü olmayan, dört kadranın tümünde şiddetli abdominal ağrı nedeniyle acil servisimize başvuran 42 yaşında bir erkek hasta bildirildi. Olaydan bir hafta önce, hasta omuz ağrısına çare olarak tüm beden kriyoterapisine başlamıştı. Rutin ve ayrıntılı etyoloji odaklı laboratuvar testleri anormallik göstermedi. Abdominal bilgisayarlı tomografi (BT) taraması bulgularına dayanılarak hastaya akut PVT tanısı konuldu. Hastada intravenöz heparin infüzyonu başlatıldı ve sonrasında trombozun durumu takip BT taramalarıyla yeniden değerlendirildi. Tromboz azaldığında, oral alım başlatıldı. Hasta birkaç gün takip edildi ve düşük moleküler ağırlıklı heparin kullanımı için yönergeler ile taburcu edildi. Bu olguda tartışıldığı üzere, PVT tüm beden kriyoterapisinin akut bir komplikasyonu olabilir.

Anahtar sözcükler: Abdominal ağrı; portal ven trombozu; omuz ağrısı; tüm beden kriyoterapisi.

Portal vein thrombosis (PVT), frequently observed as a complication of certain malignant or benign conditions (liver cirrhosis, malignancies, myeloproliferative diseases, certain infectious diseases, congenital or acquired prothrombotic diseases, abdominal trauma),[1-4] occurs due to unknown etiology in 8 to 15% of cases.[4,5] Whole-body cryotherapy is a sports medicine procedure used by athletes to decrease inflammatory and pain-inducing effects of before or after high-intensity training. It provides improved pain control and is used to increase regeneration rate.[6] As per standard protocol, a minimally dressed subject enters a vestibule

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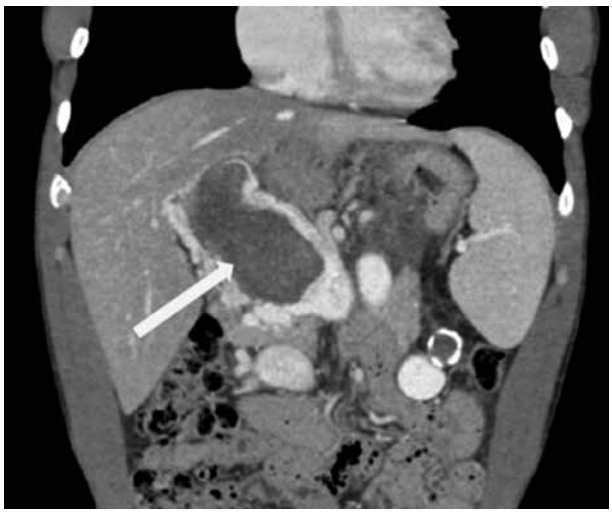

Figure 1. Giant cryotherapy-induced portal vein thrombosis in portal computed tomography scan.

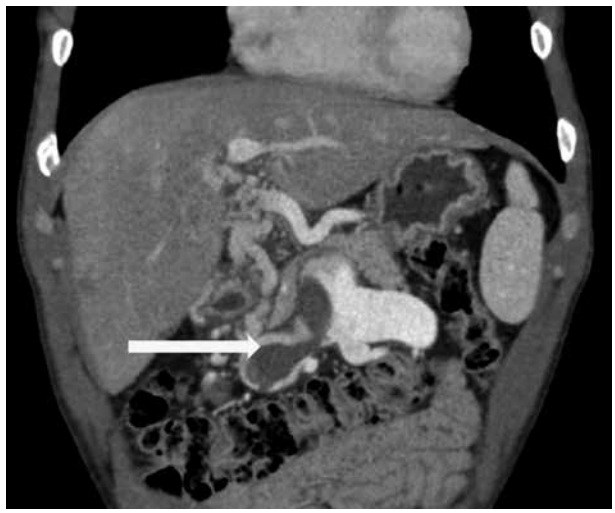

Figure 2. Giant portal vein thrombosis leading to superior mesenteric vein in computed tomography scan.

chamber at -60°C, where he stays for about 30 seconds for body adaptation and then passes into a cryochamber at -110° to -140°C, where he or she remains for no more than three minutes.[6] Although stent thrombosis has been previously reported as a complication of cryotherapy, occurrence of PVT has not been described in the literature yet. This case represents a case of acute PVT in a previously healthy young male athlete with no known risk factors.

CASE REPORT

A 42-year-old male patient with no history of previous illness, who had been training professionally as a kick-boxer for the past 25 years, applied to our emergency department due to severe abdominal pain in all four quadrants (severity score of 10 out of 10). One week prior to the incident, the patient had started whole-body cryotherapy as remedy for shoulder pain. Complaints of severe abdominal pain had started following a total of four cryotherapy sessions performed once in every two days. Physical examination revealed no icterus, telangiectasia, tremor, hepatosplenomegaly, or ascites. Intestinal sound were normoactive with auscultation. Patient had no history of alcohol, tobacco, medication use; neither there was a history of previous surgery or family history of related disease. Routine laboratory tests showed no abnormalities. Abdominal computed tomography (CT) scan, however, revealed the following finding: Pre-contrast images showed widening in splenic vein, portal confluence, and portal vein, with increased density in portal branches. After intravenous (IV) contrast administration, abdominal aorta and branches were visualized as normal in the arterial series. Two consecutive venous series revealed diameter enlargement and thrombotic filling defects in portal confluences, areas adjacent to splenic-superior mesenteric veins and the entire portal vein. Filling of the intrahepatic portal veins was not observed. Minimal collaterals developed in the liver, with no enlargement of spleen observed. A cyst of 8 mm in diameter was noticed in segment 4B of the liver. Two neighboring lesions of 16 mm and 20 mm in size in the hilus in segment 4, and one with a size of 14 mm in segment 5, observed as isodense in pre-contrast series and low density in venous series, were noted (Figure 1). A written informed consent was obtained from the patient.

Anti-nuclear antibody screening: <1:100 negative. Indirect coombs: negative, immunoglobulin (Ig) E: 17.59 IU mL, anti hepatitis B virus core IgG antibody (anti HBc IgG): negative, anti hepatitis B virus envelope Ag (anti-HBe): negative, anti hepatitis A virus antibody (anti-HAV) IgG: negative, cytomegalovirus IgM enzyme-linked immunosorbent assay: negative, Epstein-Barr virus viral capsid antigen IgG: negative,

hepatitis B envelope antigen: negative, EBV IgM negative, EBV IgG: positive, anti-beta-2 glycoprotein-1 IgG: 2.1 IU/mL (<20 IU/mL), anti-cardiolipin IgM: <2, anti-cardiolipin IgG: 2.8 U/mL, homocysteine 6.9 µmol/L (5.4-16.2), lactate dehydrogenase (LDH): 194 U/dL, urine analysis: normal, cancer (CA) 19-9 <0.600 U/mL, carcinoembryonic antigen: 1.8 ng/mL, total cholesterol: 6 mg/dL, low-density lipoprotein (LDL) cholesterol: 103 mg/dL, very LDL: 1.83 ng/mL, iron: 94 µg/dL, iron binding capacity: 263 mcg/dL, ferritin: 75.08 ng/mL, vitamin D total (25-hydroxyvitamin D): 39.09 ng/mL, antithrombin III: 100.7% (79-112), protein C activity: 98% (70-140), protein S activity: 89% (60-130), hemoglobin A1c (HbA1c): 5.3%, amylase: 27, lipase: 33, creatinine 1.1 mg/dL, hemoglobin: 14.2 g/dL, hematocrit (Htc): 40.7%, mean corpuscular volume (MCV): 81.1 fL, white blood count: 5.12x10^3/L, platelet: 201×10^3/L, anti-phospholipid IgG: 0.75 (negative), anti-phospholipid IgM: 0.41 (negative), glucose (fasting) 131 mg/dL, blood urea nitrogen (BUN): 18 mg/dL, creatinine: 1.1 mg/dL, uric acid: 4.00, alkaline phosphatase: 56 U/L, aspartate aminotransferase (AST): 25 U/L, alanine aminotransferase (ALT): 29 U/L, gamma-glutamyltransferase: 27 U/L, bilirubin (total): 0.63 mg/dL, bilirubin (direct): 0.29 mg/dL, sodium: 139 mEq/L, potassium: 3.9 mEq/L, magnesium: 1.9 mg/dL, albumin: 4.1 g/dL, total protein: 6.2 g/dL, C-reactive protein: 0.82 mg/dL, ammonia: 84 µg/dL, activated partial thromboplastin time (aPTT): 30.3 seconds, prothrombin time: 12.3 seconds (86.5% activated clotting time [ACT]), and international normalized ratios (INR): 1.07.

The patient was diagnosed with acute PVT based on abdominal CT scan findings. He was started on IV heparin infusion. Daily liver function tests, complete blood count (CBC) follow-up, and aPTT&ACT follow-up every six hours were performed. Oral intake was stopped and the pain score was assessed frequently. State of thrombosis was reassessed with follow-up CT (Figure 2). When the thrombus receded, patient's oral intake was started, followed by hospital follow-up for a few more days. Patient was discharged with instructions for the use of low-molecular-weight heparin.

DISCUSSION

Prothrombotic effects of hypothermic treatments have been demonstrated in preclinical and clinical studies.[7] In a study by Rosillo et al.,[8] it has been shown that therapeutic hypothermia increases the risk of thrombosis in coronary artery stents. This case is unique, however, in that it represents acute PVT development after whole-body cryotherapy treatment, which, to our knowledge, has never been reported before.

Portal vein thrombosis is defined as thrombus obstruction in the portal vein and its branches. Acute venous thrombosis is defined as the occurrence and identification of thrombotic symptoms in a period of less than 60 days. Occlusion may be partial or total.[9] The most common causes of PVT in adults are cirrhosis, neoplasms (hepatocellular and pancreatic carcinomas), infections, inflammatory events, myeloproliferative diseases, and idiopathic causes. Less common causes include congenital hypercoagulability disorders (protein C and/or protein S deficiency, antithrombin III deficiency), acquired hypercoagulability (pregnancy, oral contraceptive use, presence of lupus anticoagulant, inflammatory bowel disease, systemic lupus erythematosus, Behçet's disease, scleroderma, idiopathic pulmonary hypertension, paroxysmal nocturnal hemoglobinuria, nephrotic syndrome), and other causes (non-cirrhotic portal fibrosis, hepatoportal fibrosis, blunt trauma, abdominal surgery, splenectomy and functional hyposplenism, distal splenorenal shunt surgery, liver transplantation, endoscopic varicose sclerotherapy).[1,2,9] The purpose of PVT therapy is to provide obstructive venous recanalization in order to prevent portal hypertension and intestinal infarction. In cirrhotic and non-malignant acute PVT, anticoagulation therapy is recommended.[10-13] Following six months of anticoagulant treatment, complete recanalization was observed in 50%, partial recanalization in 40%, and no response in 10% of the patients. In acute PVT, anticoagulant therapy is recommended for at least three months, with life-long treatment if prothrombotic test positivity is present.[14] Other treatment modalities (surgical thrombectomy, thrombolysis, transjugular intrahepatic portosystemic shunting) have limited utility in the treatment of acute PVT.[14,15]

Invasive procedures do not seem to be more effective and have more risks compared to anticoagulant therapy alone.[16] Thrombectomy or thrombolysis is usually recommended when clinical deterioration, progression of PVT, intestinal necrosis or infarction is observed despite anticoagulation therapy.[17]

Interestingly, our patient was a healthy athlete who started all-body cryotherapy treatment for shoulder pain, did not have any complaints after three sessions of treatment, applied to the hospital with severe abdominal pain following the fourth session, and received the diagnosis of PVT based on biochemical test panel and abdominal CT. Anticoagulant treatment with heparin was started immediately, and the dosages were adjusted according to ACT & aPTT follow-up with six-hour intervals. CBC, AST, ALT, total and direct bilirubin, creatinine, BUN, and LDH values were reassessed daily. Physical examination with attention to the development of acute abdomen and follow-up CT scans were performed. Paracetamol 3×1 g IV, tramadol 50 mg 3×1 IV, and pethidine hydrochloride 25 mg subcutaneous, as needed, were administered for pain control. Oral intake was stopped; IV fluids were administered. After PVT regression was observed on follow-up CT scans, oral intake was reintroduced and the patient was discharged with low-molecular-weight heparin.

In the reported case, the development of acute PVT after whole-body cryotherapy, with all etiological testing proven negative, leads to the presumption that cryotherapy may be a probable cause of acute PVT, the occurrence of which has not been previously reported in the literature.

Declaration of conflicting interests

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