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Portal vein thrombosis after total knee replacement: a case report

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ABSTRACT

We present a 74-year-old woman who developed a portal vein thrombosis following an elective total knee replacement. She had atrial fibrillation for which she was taking warfarin for anticoagulation. Seven days prior to surgery, she was instructed to discontinue warfarin and replace it with prophylactic low-molecular-weight heparin. On postoperative day 1, routine blood tests revealed deranged hepatic synthetic function, despite standard anticoagulation management. Doppler ultrasonography confirmed a portal vein thrombosis. She was treated with therapeutic doses of low-molecular-weight heparin until her international normalised ratio reached therapeutic levels. Her liver function results had normalised 2 weeks later. Portal vein thrombosis is a potentially fatal complication that is reversible if identified and treated early.

Key words: *arthroplasty, replacement, knee; heparin, low-molecular-weight; portal vein; venous thrombosis; warfarin*

INTRODUCTION

Orthopaedic surgery is associated with a high incidence of venous thromboembolism (VTE). Contributing factors include direct vessel trauma, venous stasis of the limb, and hypercoagulability of the blood secondary to ageing and immobility. In the absence of thromboprophylaxis, the incidence of venographically proved VTE after total knee replacement (TKR) ranges from 40 to 84%.[1] As the population ages, the number of patients having co-morbidities requiring anticoagulation therapy (for deep venous thrombosis, pulmonary embolism, atrial fibrillation, artificial heart valves, thrombophilia and cardiac bypass surgery or stenting) increases. Guidelines for perioperative management of these patients recommend discontinuing warfarin and replacing it with prophylactic or therapeutic low-molecular-weight heparin (depending on the underlying pathology) while the prothrombin time normalises, then resuming warfarin therapy soon after surgery. Concerns with this practice include increased peri-operative blood loss, haematomas, impaired wound healing, and fluctuations of the international normalised ratio (INR) while trying to regain therapeutic anticoagulation.[2]

Warfarin selectively inhibits vitamin K-dependent Factors II, VII, IX, and X as well as Protein C and S. This action on Protein C and S, prior to its inhibition of the vitamin K-dependent clotting factors, has a prothrombotic effect. Cessation of anticoagulation in patients on long-term warfarin poses a greater risk

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of VTE compared with the background risk owing to this rebound hypercoagulable state, in addition to the prothrombotic stress effect of the surgery itself.[3] Portal vein thrombosis shares a common pathogenesis with VTE after TKR. We present a 74-year-old woman who developed a portal vein thrombosis following an elective TKR.

CASE REPORT

In November 2010, a 74-year-old woman presented with a long history of progressively worsening anteromedial knee pain that was exacerbated by physical activity. The pain was worse at the end of the day and limited her mobility. She had used a walking stick and was taking strong analgesia to facilitate daily activities.

On examination, she walked with an antalgic gait and had a correctable varus deformity and weak quadriceps. She had tenderness over the medial joint line, and her active range of movement was 5º to 90º of flexion. Weight-bearing radiographs of the knee confirmed tri-compartmental degenerative changes, particularly in the medial tibiofemoral compartment. She decided to undergo a TKR.

At the pre-assessment clinic, she was noted to have hypertension, type-2 diabetes, hypercholesterolaemia, and atrial fibrillation for which she was prescribed warfarin for anticoagulation. She was advised to discontinue warfarin 7 days prior to surgery during which she self-administered subcutaneous injections of low-molecular-weight heparin (5000 units of dalteparin sodium once a day) until she could be fully anticoagulated postoperatively. All preoperative tests including liver function tests were within normal limits.

Her INR was checked on the morning of surgery and it was deemed safe to proceed. She underwent a standard cruciate-retaining TKR under tourniquet control. On postoperative day 1, prophylactic doses of low-molecular-weight heparin injections were re-commenced. Thromboembolic deterrent stockings were applied to both legs, and a loading dose of warfarin was administered. Routine blood tests revealed deranged hepatic synthetic function: alanine transaminase of 1061 (normal range, 40–129) U/l, alkaline phosphatase of 317 (normal range, 0–40) U/l, total bilirubin of 27 (normal range, 1–17) μmol/l, albumin of 30 (normal range, 35–50) g/l, INR of 1.6 (normal, 1), prothrombin time of 21.0 (normal range, 9.1–11.4) seconds, and activated partial thromboplastin time of 25.0 (normal range, 21–29) seconds. Nonetheless, the patient was asymptomatic and systemically well. Her abdomen was soft and non-tender, with no signs of hepatosplenomegaly. On day 2, repeat liver function tests showed even greater derangement and she underwent a full thrombophilia screen, autoantibody screen, hepatitis screen, and ultrasound scanning of her liver. All other blood tests were within normal limits. Doppler ultrasonography of the liver demonstrated no significant colour flow within the portal vein, whereas spectral Doppler ultrasonography showed minimal flow within the vein, indicating portal vein thrombosis (Fig.). She was treated with therapeutic doses of low-molecular-weight heparin until her INR reached therapeutic levels. Serial liver function tests peaked on day 3 and then gradually returned to normal. Her liver function results had normalised 2 weeks later.

DISCUSSION

Most portal vein thromboses are asymptomatic.

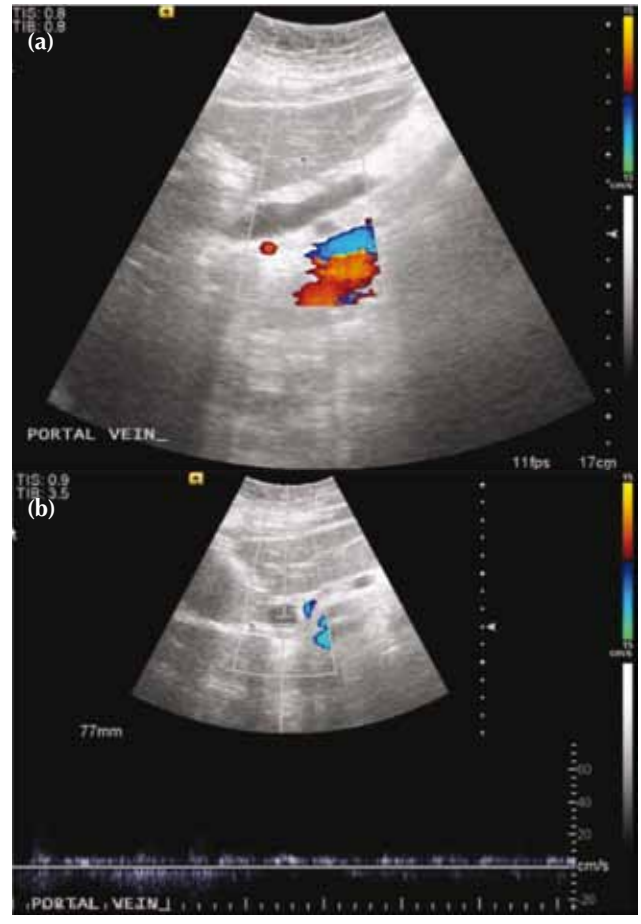

Figure Portal vein thrombosis is indicated by **(a)** lack of significant colour flow within the vein on Doppler ultrasonography and **(b)** minimal flow within the vein on spectral Doppler ultrasonography.

Discontinuation of warfarin therapy prior to elective surgery leads to a transient hypercoagulable state, which increases the risk of thromboembolic complications, despite the replacement of warfarin with prophylactic doses of low-molecular-weight heparin.

The portal vein originates from the superior mesenteric and splenic veins and accounts for approximately 75% of the blood supply to the liver. Portal vein thrombosis is the complete or partial obstruction of blood flow within the extra-hepatic portion of the portal vein, owing to thrombus within the vessel lumen.[4] Its presentation can be acute or chronic. It can be classified according to the extent of the thrombosis, development of collateral vessels, and involvement of other mesenteric vessels (which is associated with a considerably higher risk of bowel ischaemia and mortality).[5,6]

Portal vein thrombosis affects approximately 1% of the general population.[7] It is more common among cirrhotic patients with a prevalence of 4.4 to 15%, and is responsible for 5 to 10% of portal hypertension cases.[8] Its prognosis is good in non-cirrhotic patients without associated neoplastic disease, with an overall mortality of <10% and a 5-year survival of 85%, attributing to early diagnosis, the advent of antibiotics, early surgical intervention, and use of anticoagulation.[9,10]

The aetiology of portal vein thrombosis is associated with conditions that cause a prothrombotic state and other local factors.[6,9–12] Inherited prothrombotic disorders associated with portal vein thrombosis include Protein C and Protein S deficiencies, anti-thrombin III deficiency and Factor V Leiden and Factor II gene mutations. Acquired prothrombotic disorders include anti-phospholipid syndrome, paroxysmal nocturnal haemoglobinuria, and other inflammatory conditions. Local factors associated with the development of portal vein thrombosis include portal vein injury following splenectomy, laparoscopic abdominal surgery, abdominal trauma, infection, and localised inflammatory lesions such as pancreatitis and cholecystitis. Malignancy is also a key factor, with the incidence of neoplasm-related portal vein thrombosis being 21 to 24% owing to prothrombotic changes, direct tumour invasion, and local tumour mass effects.[6,9–12]

Clinical manifestations depend on the extent of thrombosis, speed of onset, and development of compensatory collateral vessels.[6] Patients may be asymptomatic or present with life-threatening complications. Common symptoms include abdominal pain or distension, vomiting, fever, anorexia and nausea, whereas more severe cases may manifest with mesenteric ischaemia, infarction, and peritonitis.[6]

Under spectral Doppler ultrasonography, portal vein thrombosis is shown as hyperechoic materials within the lumen of the vessel, and absence of blood flow. Colour Doppler ultrasonography has a diagnostic sensitivity of 66 to 100% and a negative predictive value of 98%.[4,10] Ultrasonography is less reliable at examining thrombus spread into the mesenteric circulation. Computed tomography and magnetic resonance imaging can be used to assess thrombus extension and identify damage in surrounding organs and evidence of bowel ischaemia. Once the diagnosis is established, a full thrombophilia screen and exclusion of local factors is advisable prior to commencing anticoagulation therapy.[10]

If this potentially fatal complication is identified and treated early, it is reversible and may be preventable. Liver function tests should be requested as part of standard blood test monitoring during the immediate postoperative period.

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