Spontaneous rectus sheath haematoma in a deceased donor renal transplant recipient: a rare complication

Abstract

Rectus sheath haematoma (RSH) is rarely thought of as a cause of abdominal pain in renal transplant recipients. A 36-year-old woman, a post-deceased donor renal allograft transplant recipient for chronic interstitial nephritis, on triple drug immunosuppression (tacrolimus, mycophenolate mofetil and prednisolone) with basiliximab induction, developed acute vascular rejection and acute tubular injury with suspected antibody-mediated rejection. While on plasmapheresis and haemodialysis for delayed graft function, she developed acute left lower abdominal pain on the 16th postoperative day with tender swelling in the left paraumbilical region. CT of the abdomen showed a large haematoma in the left rectus sheath with no extension. The patient underwent haematoma evacuation through a left paramedian incision and had an uneventful recovery. Serum creatinine stabilised at 0.8 mg/dL and she is on regular follow-up with excellent graft function at 6 months. Diagnosis requires a high index of suspicion, and prompt treatment prevents morbidity and can expedite patient recovery.

Background

Rectus sheath haematoma (RSH) is a rare cause of abdominal pain in the renal transplant recipient. Predisposing factors include female gender, older age, corticosteroids and postoperative status. Anticoagulation is also a predisposing factor.1 Straining and pregnancy have also been identified as risk factors. However, RSH in the renal transplant recipient is rare. We report a case of a 36-year-old woman post-deceased donor renal transplant recipient who developed RSH in the immediate postoperative period.

Case presentation

A 36-year-old woman underwent a deceased donor renal allograft transplant for chronic interstitial nephritis, and was started on triple drug immunosuppression (tacrolimus, mycophenolate mofetil and prednisolone) and induction with basiliximab. She was on neither anticoagulants nor antiplatelets. She had acute vascular rejection and acute tubular injury, and suspected antibody-mediated rejection. She improved with plasmapheresis and haemodialysis. On the 16th postoperative day, she developed acute left lower abdominal pain after twisting her torso in bed. On examination, she had an acute tender swelling measuring 6×5 cm in the left paramedian region.

Investigations

CT of the abdomen showed a large haematoma measuring 7×6×4 cm in the left rectus sheath infra-umbilically An external file that holds a picture, illustration, etc. Object name is bcr2015214144f01.jpg

Treatment

The patient underwent haematoma evacuation and ligation of the inferior epigastric artery through a left paramedian incision, under short general anaesthesia (GA). Postoperative recovery was uneventful and sutures were removed on the 10th postoperative day.

Outcome and follow-up

The patient's creatinine stabilised to 0.8 mg/dL at discharge. She is doing well with excellent graft function at 6 months (figure (figure22).

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Discussion

RSH is a relatively uncommon cause of abdominal pain in a transplant recipient. RSH occurs due to damage to superior or inferior epigastric arteries (SEA or IEA) or their branches into the rectus sheath, or due to a direct tear of the rectus muscle.1 Based on a Mayo Clinic series, the major risk factors identified were female gender, the elderly, anticoagulation, cough and abdominal trauma. Pregnancy is also a risk factor.1 Patients with renal failure are at risk due to platelet/coagulation abnormalities.2 RSH has been reported following insertion of peritoneal dialysis catheters and as the first manifestation of post-renal transplant lymphoproliferative disease.3 4

Spontaneous RSH in renal transplant recipient was first described by Nikolina et al in 2010, and bilateral RSH by Feizzadeh Kerigh et al in 2013.1–5 The risk factors included abrupt change in position, anticoagulation, coagulation disorder, postoperative status, steroids and amyloidosis. In our patient, RSH developed 16 days after transplant. The association between steroids and anticoagulants and its time-duration with the incidence of RSH is not clearly known due to the rarity of this condition. The immediate cause of the rupture may be external trauma to the abdominal wall, iatrogenic trauma from surgery, or excessive vigorous contractions of the rectus muscle.6 Because the arteries supply the recti from behind, most haematomas are posterior to the muscle, making diagnosis by means of palpation more difficult. The incidence is thought to be on the rise, with the increased use of oral anticoagulation drugs and low-molecular-weight heparins.6

The rectus sheath is composed of the rectus abdominis muscles, enveloped by a fascial sheath, and is supplied via the epigastric arteries and veins. The SEA after its origin from the external thoracic artery, enters the sheath from behind the seventh costal cartilage and descends between the rectus abdominis muscle and the posterior rectus sheath. The IEA, after originating from the external iliac artery, ascends loosely between the rectus muscle and posterior rectus sheath. During contractions, the length of the rectus muscle varies and the artery glides with the muscle to avert tearing. This combination of loose attachment of IEA and stabilisation of its perforators fixed to the muscle belly makes the artery prone to shearing stresses at the branching sites during strong muscular contraction. The SEA and IEA have rich microscopic anastomoses near the umbilicus, which help to diminish the likelihood of trauma to the vessels during muscular contraction.1

Most RSHs can be treated conservatively with bed rest, analgaesia and treatment of predisposing conditions, and by discontinuing anticoagulation. However, RSH can lead to significant morbidity and has an overall mortality of up to 4%, which is as high as 25% if on anticoagulants. The morbidity of RSH is primarily due to incorrect diagnosis and unnecessary exploratory laparotomy or delay in cessation of anticoagulant therapy and, to avoid this, ultrasonography (US) and CT are used.1 7 8 CT is more sensitive

and specific than US, and has the added advantage of ruling out an intra-abdominal pathology.9 10 Active bleeding can be managed either surgically, by evacuating the haematoma and ligating the bleeding vessels, or radiologically, with catheter embolisation.11 12 We identified female gender, corticosteroids, postoperative status, plasmapheresis and haemodialysis as risk factors for spontaneous RSH in our patient, and the indication for surgical evacuation was an expanding haematoma. Invasive haemorrhage control of RSHs should be considered in haemodynamically unstable patients who are not responding to fluid resuscitation, in the form of angiography and embolisation or surgical ligation of bleeding vessels.13 After the surgical procedure, our patient had good recovery with respect to graft function as well, and was discharged home, with a serum creatinine value of 0.8 mg/dL.