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244 Letters to the Editor

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Regarding "A spontaneous rupture of the external iliac vein revealed as a phlegmasia cerulea dolens with acute lower limb ischemia: Case report and review of the literature"

In their case report and review of the literature, Jazayeri et al (J Vasc Surg 2002;35:999-1002) analyzed etiology, diagnostic, and therapeutic problems of spontaneous rupture of the iliac vein.

In this case, ultrasound scan showed occlusion of iliac vein and a suspected neoplastic mass in the left iliac fossa; a retroperitoneal hematoma was ultimately recognized during emergency laparotomy.

They concluded that the cause remains obscure and the diagnosis should be considered particularly in female patients with sudden-onset lower abdominal pain and hypotension.

We recently had an opportunity to treat two patients, one man and one woman, who had spontaneous rupture of iliac vein.¹

Contrast-enhanced abdominal CT scan was able to detect venous thrombosis and retroperitoneal hemorrhage in both our patients. Although no direct sign of vein leakage was exhibited by CT scanning, we agree with Lin et al,² who postulated that this technique should be considered in differential diagnosis in stabilized patients.

To investigate the pathogenesis of vein rupture, pathologic examination was performed in a few cases and aspecific inflammatory changes were noted.³⁻⁶

In our own patients, tissue samples of the edges of the venous tear were fixed in 10% buffered formaldehyde and embedded in paraffin; part of the serial sections (5 μ m) was prepared for histochemical study (hematoxylin-cosin, Weigert-elastin, van Gieson), the remaining part for immunocytochemistry, using the avidinbiotin peroxidase method and the cell type–specific monoclonal antibodies to T lymphocytes (CD 43), B lymphocytes (CD 20), and macrophages (CD 68; Dako, Inc,).

Histologic examination of the edges of the tear revealed inflammatory cell infiltrate, consisting of macrophages, T and B lymphocytes, with fragmentation of elastic fibers and presence of areas of necrosis (Figure).

Findings of infiltration of cells producing cytokines with high elastolytic and collagenase activities support the hypothesis that significant modifications of parietal structure, such as elastin and collagen degradation, may result in weakening of the vein wall, leading ultimately, in association with venous outflow obstruction, to its rupture.

Treatment options for vein repair include ligation or suture; in one of our patients, a large erosion on the lateral wall of the left external iliac vein was found.

The edges of the vein defect were excised, and reconstruction was performed with an ePTFE patch angioplasty.

The patient was treated with oral anticoagulants and compressing stockings.

During a 5-year follow-up, duplex surveillance revealed no venous occlusion.

On the contrary, despite trombectomy and postoperative anticoagulation treatment, a high incidence of vein obstruction after repair with a continuous suture has been reported by Jazayeri and colleagues, requiring in their case a crossover saphenous vein bypass grafting with an arteriovenous fistula.

Moreover, an approximately 50% compromise in the lumen was noted by Brown, Sanchez, and Marrix⁵ after vein direct repair and possibly predisposed to venous thrombosis.

The result in our patient suggests that patch angioplasty could represent a proper option in case of iliac vein rupture with a large parietal defect.

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A, H & E staining of vein wall shows area of necrosis and inflammatory infiltration ($250 \times$). **B**, Transverse section of vein immunostained for macrophages (CD 68) shows positive staining ($250 \times$). **C**, Vein tissue that has been stained with Weigert-elastin. Note fragmentation of elastin fibers ($250 \times$).

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