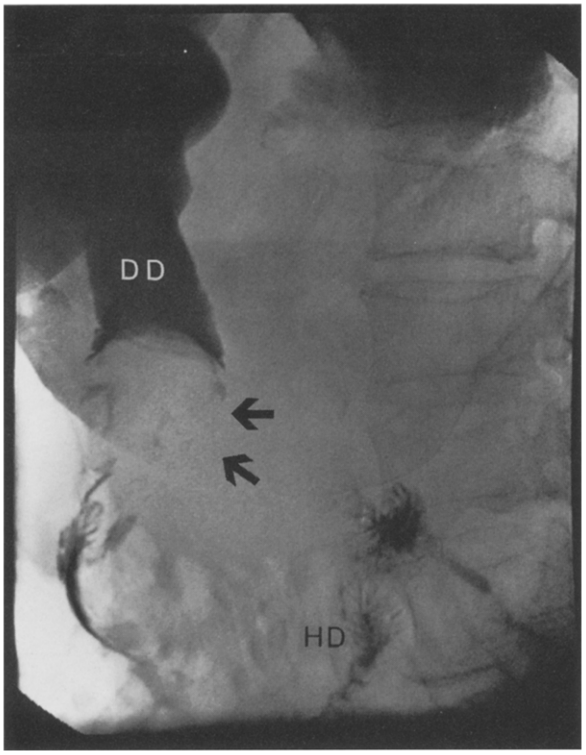

Fig. 2. Barium contrast accumulates in the descending duodenum *(DD)*, and only small amounts pass the submucosal tumor *(arrow)* and reach the horizontal duodenum *(HD)*.

rysm repair is 6.6%. In their series of 472 aortic aneurysmectomies, a small bowel obstruction developed after surgery in only two patients, caused by adherence to the aneurysmal sac and a deep tension suture having pierced the small bowel, respectively. Available data suggest that the obstruction is usually caused by perigraft collagenous adhesions and is probably less likely to occur if the mobilized duodenum is not replaced directly over the aorta during the resuturing of the retroperitoneum.[3]

Duodenal obstruction as a result of compression by a retroperitoneal hematoma is a rare postoperative occurrence and has been described in only two cases,[4,5] in which, however, the hematoma was located in the third part of the duodenum. In those cases, a large retroperitoneal hematoma compressed the third portion of the duodenum anteriorly against the superior mesenteric artery (superior mesenteric artery syndrome). Extensive retroperitoneal dissection extending proximally up to the origins of the renal arteries, implantation of a vascular prosthesis, and the use of heparin constitutes a major potential source of bleeding.[5] In our patient the obstruction was located in the descending part of the duodenum, which has not been reported previously.

Usually a retroperitoneal duodenal hematoma does not require surgery. Continuous nasogastric suction should be employed and total parenteral nutrition initiated. The patient should be reevaluated with upper gastrointestinal contrast studies or gastroscopy at a 7 day interval. Operative exploration and evacuation of the hematoma must be considered after 2 weeks of unsuccessful conservative therapy. In case of a superior mesenteric artery syndrome, end-to-side duodenojejunostomy or a gastroenterostomy are considered to be the method of choice.[5]

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Necrotizing venulitis and in situ saphenous vein bypass

To the Editors:

Residual side branches and concomitant arteriovenous fistula are well known complications associated with in-situ greater saphenous vein bypass.[1] Morphologically they may be divided in two main groups. The first consists of perforators that connect the superficial venous system to the deep one. Physiologically these are true arteriovenous fistulas that sometimes cause a loss of pulsatility in the distal section of the graft, which may result in distal thrombosis.[2] The second group consists of smaller superficial venules related to the skin capillary. They perfuse in a retrograde fashion the capillary network of the dermis. They are not anatomically true arteriovenous fistulas but instead carry through the venous capillary pathway a competitive flow to the dermal arteriolar supply. Consequently, they may cause local venous hypertension with painful cutaneous flares that occasionally result in skin ulcerations.[3] Although the former group normally needs surgical correction to prevent graft hemodynamic failure, the latter is usually self-limited and uneventful, although it may occasionally become quite spectacular and be troublesome for the surgeon who has to deal with it (Fig. 1). Light microscopic examination of these skin lesions reveals that the epidermis is normally preserved

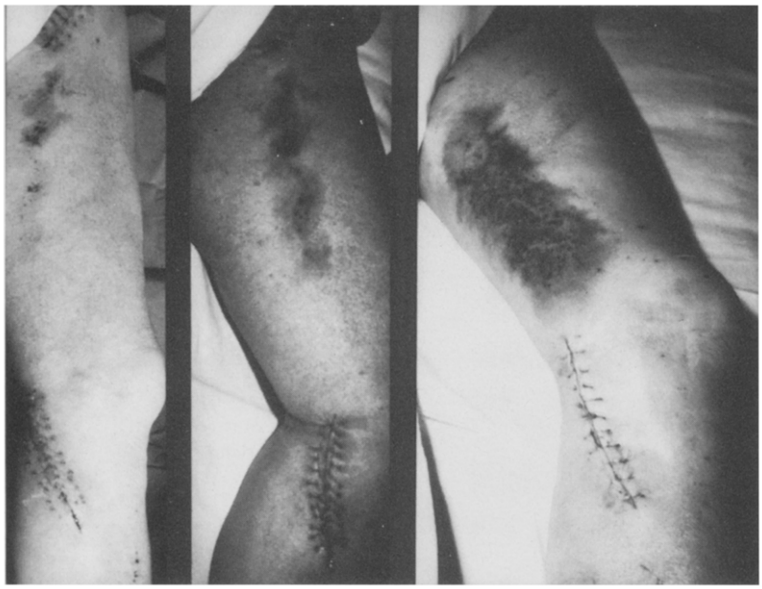

Fig. 1. *Right panel,* Severe skin reaction over internal saphenous pathway 7 days after in-situ femoropopliteal bypass was performed. *Middle and left panels,* Milder reaction evolving from day 6 *(middle)* and 12 *(left)* after the surgery, showing the self-limiting aspect of the reaction.

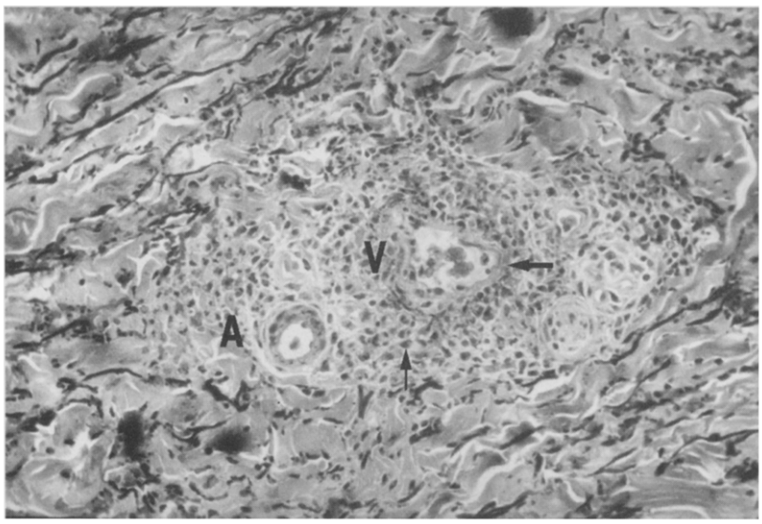

Fig. 2. Photomicrograph (hematoxylin and eosin; original magnification, ×125) of a skin biopsy harvested during acute phase of skin congestion. At junction of dermis and hypodermis, venules *(V)* show deposition of fibrinoid material around their wall *(horizontal arrow)* surrounded by severe inflammatory infiltrate of neutrophils *(vertical arrow).* Arterioles *(A)* are not affected by the process.

during this congestive reaction. On deeper section, however, an extravasation of red blood cells through the connective tissue of the dermis is observed. The mild dermis, especially at the dermis-hypodermis junction, shows numerous venule deposits of fibrinoid material around their walls with a severe infiltrate of neutrophils with fragmented nuclei. Interestingly, the arterioles around these venules are left intact by the inflammatory process (Fig. 2).

This fibrinoid necrosis results from the systemic arterial pressure transmitted through the side branches of the in-situ graft to the venous system. The subcutaneous venules (in opposition to the deep arteriovenous fistula) do not beneficiate of any muscular coating. They are surrounded by connective and fatty tissue, making the advent

of a brutal systemic perfusion a major physiologic event explaining the necrotizing venulitis aspect observed in this particular situation.

Necrotizing venulitis has not been described as a specific complication of in-situ venous bypasses. Although the clinical manifestation has been known for many years, the histologic aspect has been neglected. Intimal hyperplasia is well recognized as a common complication of venous conduit exposed to systemic pressure and characterizes long-term adaptation of the venous conduit to systemic pressure. Necrotizing venulitis represents early failure to this adaptation. The vascular lesions displayed resemble the alterations observed in arterioles of the small bowel after correction of coarctation of the aorta in rare cases of "necrotizing arteriolitis," as described by J.J. Perez-Alvarez in 1955.[4] He reported the case of a 5-year-old boy who died of visceral ischemia after a thoracic aortic coarctation repair. The postmortem examination revealed necrotizing arteriolitis characterized by a fibrinoid necrosis involving the entire arteriolar wall. Referring to Bing's work on arch coarctation, he attributed this phenomenon to the sudden overflow through the visceral arterial system. The lack of surrounding muscular coating makes them more susceptible to overstretching due to unaccustomed high pressure.[5] In our clinical experience, this cutaneous and subcutaneous necrotizing venulitis is a self-limited phenomenon. The skin congestion normally resumes within 10 to 12 days and should not be mistaken for cellulitis, deep venous thrombosis, or graft failure. Occasionally the reaction may be quite extensive and require skin-graft surgery. By dividing the largest side branches at the time of the surgery, the surgeon can prevent or attenuate the occurrence of this complication. Nevertheless, conservative management of this complication is advocated, especially early after surgery to avoid iatrogenic damage to the venous conduit. The majority of the skin lesions will spontaneously heal in less than 2 weeks without any permanent deficit.

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