SURGICAL REVASCULARIZATION IN CHRONIC MESENTERIC ISCHEMIA

R. F. Popa, Cristina Strobescu, Genoveva Baroi, A. Raza
University of Medicine and Pharmacy "Grigore T. Popa"- Iaşi
Faculty of Medicine
Discipline of Surgery
“Sf. Spiridon” Emergency Hospital - Iaşi
Vascular Surgery Department

SURGICAL REVASCULARIZATION IN CHRONIC MESENTERIC ISCHEMIA. CASE REPORTS (Abstract): Chronic mesenteric ischemia (CMI) is a disease causing death either by starvation or enteromesenteric infarction. Diagnosis is often delayed before the patient is referred to a vascular surgery unit. Atherosclerosis is the main cause of CMI. Arteriography is essential in diagnosing CMI and delineating the atherosclerotic lesions. The revascularization procedure consists in an aortomesenteric bypass reconstructing 1-3 visceral arteries. This paper presents two consecutive CMI cases treated at the Vascular Surgery Unit of the Iaşi “Sf. Spiridon” Hospital during 2010. Both patients had symptoms suggestive of mesenteric and aortoiliac diseases. CT angiography revealed specific lesions both for aortoiliac disease and stenotic or occlusive lesions in the celiac trunk and mesenteric arteries. Both cases benefited from aortobifemoral bypass surgery using a synthetic graft associated with aortic-superior mesenteric artery bypass with reversed vein graft (in the first case both mesenteric arteries were revascularized). Immediate and remote results were favorable, with remission of intestinal symptoms and weight gain. Bypass patency was followed-up by CT angiography and Doppler ultrasound. CMI is a diagnostic and therapeutic challenge. Open surgery provides symptom remission in 90% of cases. Permeability at 5 years is 80-90% for open surgery, higher than by endovascular therapy. Average permeability of the two types of intervention is 70% at 5 years, similar to the infraaortic bypasses. Keywords: MESENTERIC BYPASS, CHRONIC MESENTERIC ISCHEMIA, AORTOBIFEMORAL BYPASS

Chronic mesenteric ischemia (CMI) is a disease causing death either by starvation or enteromesenteric infarction. The diagnosis is often delayed prior to patient referral to a vascular surgeon.

The pathophysiological mechanism underlying CMI is the failure to achieve normal postprandial mesenteric hyperemia.

Hemodynamically, significant arterial stenosis causes attenuated postprandial response leading to imbalance between required and available O₂ and metabolites causing postprandial pain (mesenteric angina).

Atherosclerosis is the main cause of visceral artery occlusive disease and consequently the cause of CMI. Other occasional causes are: fibromuscular disease, aortic dissection, neurofibromatosis, rheumatoid arthritis, Takayasu disease, postradiotherapy lesions, Burgers Disease, systemic lupus erythematosus and drug use (cocaine, ergot) (1).
The abdominal digestive tract is supplied by three major arterial axes (celiac trunk, superior and inferior mesenteric arteries) interconnected by an extensive collateral network. Usually, a significant occlusion of 2 or 3 visceral vessels is required for the patient to become symptomatic. But isolated occlusion of the celiac trunk or superior mesenteric artery (SMA) alone can give rise to symptoms when the collateral network is inefficient (1).

There are 3 stages of CMI: chronic intermittent ischemia, permanent ischemia and enteromesenteric infarction (2).

The patient complaints are: intestinal angina (30-60 min postprandial), weight loss caused by food phobia (fear of eating), altered bowel function, bloating, or vague abdominal discomfort, diarrhea or constipation (2).

Patients with CMI frequently have concomitant renal or aortic artery occlusive disease.

Arteriography is essential for confirming the diagnosis and delineating the lesions topography (3).

Late studies are controversial with respect to: the type of surgery (open surgery vs. endovascular surgery), endarterectomy vs. antegrade/retrograde bypass, number of bypassed vessels (1-2-3), the best type of graft (synthetic vs. venous - internal saphenous vein / superficial femoral vein) (1).

Studies in the United States revealed that mesenteric bypass represents less than 0.5% of all peripheral vascular bypasses (1).

**CASE REPORTS**

This paper presents two consecutive cases of CMI treated at the Iaşi, Vascular Surgery Unit, during 2010.

**CASE 1**

G.A., 51-year-old male presented with the following complaints: postprandial abdominal pain, significant weight loss, frequent loose stools, loss of appetite, significant fatigue, intermittent claudication and decreased walking perimeter (< 50m).

Laboratory tests revealed hypoproteinemia (total proteins = 56 g/dl) and hypoalbuminemia (albumin = 2.53 g/dl).

Preoperatively CT angiography confirmed the suspicion of CMI revealing severe celiac trunk stenosis of about 12 mm from the origin and atherothrombotic occlusion in the initial segment of SMA of about 20mm, occlusion of the left internal iliac artery and its branches, and also the atheromatosis of the abdominal aorta, iliac arteries and abdominal aorta branches (fig. 1).

**Fig. 1. Preoperative CT angiography - Patient G.A**

Pre-surgical screening was performed and anticoagulant, vasodilator and hypolipemiant treatment was administered.

The surgical intervention consisted in a deep aortic bifemoral bypass with Dacron graft associated with an aortic–inferior mesenteric artery (IMA ) and aortic- superior mesenteric artery (SMA ) bypass with
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autologous venous graft.

Through median laparotomy we entered the left mesenteric–colic space. The abdominal aorta was exposed by dissecting the peritoneum from the ligament of Treitz to the IMA. The left renal vein was identified as a landmark for locating the renal artery and SMA (fig. 2).

The next step was the i.v. administration of 5000 U Heparin. After 5 minutes the infrarenal aorta was clamped proximally and distally. An aorto-bifemoral bypass was performed using a Dacron graft. All anastomoses (synthetic graft to aorta and synthetic graft branches to femoral arteries) were termino-lateral (fig. 3).

The next step was to perform the aortic – IMA and SMA bypass with reversed autologous vein graft previously harvested (fig. 4, 5).
We aimed to adjust the length of the graft to allow its positioning in relaxed “C” position (fig. 6, 7).

The omentum was brought in the aortomesenteric space by coloepiploic detachment. This way the omentum was used as an anchor fringe (fig. 8).

After surgery the patient received the three "A" therapy (antibiotic, anticoagulant, analgesic) associated with vasodilators and hypolipemiant agents.

Immediate and remote results were favorable: warm skin, bilateral popliteal pulse, bilateral Doppler signal along the anterior tibial artery, remission of the abdominal pain, oral feeding after gas transit was resumed, and bowel transit.

A control CT angiography was performed 2 weeks after surgery (fig. 9). It showed that the aortobifemoral and aortomesenteric bypasses were patent. The echo-Doppler follow-up at 1, 2, 3, 6 and 12 months revealed persistent graft patency (fig. 10, 11).
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CASE 2

C.G., 60-year-old, male was admitted to the Vascular Surgery Unit complaining of intermittent claudication of both lower limbs (more intense in the left limb), decreased walking perimeter (<50 m) and significant weight loss.

Laboratory findings revealed the absence of the inflammatory syndrome and presence of hypoproteinemia. Doppler signal was detected from the peripheral arteries of the lower limbs.

Preoperative CT angiography revealed abdominal aorta with multiple calcified atherosclerosis plaques and parietal thrombosis, iliac artery with multiple stenosis, thrombosis of the origin of celiac trunk bifurcation (the remaining vessels being patent) and significant SMA stenosis from the origin (fig. 12).

Fig. 10. Color echo-Doppler graft patency

Fig. 11. Superior mesenteric graft blood flow signal

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Fig. 12. Preoperative CT angiography.
Patient C.G

Fig. 13. Postoperative CT angiography
Patient C.G
The surgical intervention consisted in an aortobifemoral bypass with Dacron graft associated with an aortic – SMA bypass with reversed vein graft (right internal saphenous vein) (fig. 13, 14).

The surgical technique was similar to that used in the first case except that only one mesenteric artery was revascularized (fig. 15).

**DISCUSSION**

Immediate and remote results were favorable: presence of pulse in the lower limb peripheral arteries and resumption of physiologic bowel transit.

This case also benefited from CT angiographic control along with Doppler ultrasound follow-up at 1, 2, 3, 6 and 12 months that revealed the patency of the bypass.

**CONCLUSIONS**

Both our cases were successfully treated, no complications being recorded in the immediate postsurgical period and after a 2-year follow-up. The two patients presented for symptoms of aortoiliac disease and CMI, respectively. With appropriate surgery both problems were solved and the patients were reinserted into their families and society.
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REFERENCES


TREATMENT OF PERI-IMPLANTITIS USING MULTIPLE APPLICATIONS OF CHLORHEXIDINE CHIPS: A DOUBLE-BLIND, RANDOMIZED MULTI-CENTRE CLINICAL TRIAL

Universal strategies for managing peri-implantitis are yet to be adopted. The aim of this study is to examine a protocol of intensive application of chlorhexidine containing chips in sites with peri-implantitis. This multi-centre, randomized, double-blind, parallel, two-arm clinical trial included 60 patients (77 implants) with probing depth (PD) 6-10 mm and bone loss ≥2 mm around 1-2 implants. One to two weeks following scaling and root planing, baseline measurements were made followed by implants' debridement. Patients were randomized to receive matrix chips (MatrixC) or chlorhexidine Chips (PerioC). Measurements and chips placement were repeated at weeks 2, 4, 6, 8, 12 and 18. At 6 months, patients returned for final examination. Probing depth reduction was greater in the PerioC (2.19 ± 0.24 mm) compared with MatrixC (1.59 ± 0.23 mm), p = 0.07. Seventy percentage of the implants in the PerioC and 54% in the MatrixC had PD reduction ≥ 2 mm. Likewise, 40% of the sites (PerioC) and 24% (MatrixC) had PD reduction ≥ 3 mm. Clinical attachment level gains for both groups were significant; however, the changes in the PerioC group were significantly greater than in MatrixC [2.21 ± 0.23 mm. and 1.56 ± 0.25 mm respectively, p = 0.05]. Bleeding on probing was reduced by half in both groups. These results suggest that frequent placement of PerioC and MatrixC together with implants debridement resulted in a substantial improvement in sites with peri-implantitis. Further studies will be required to fully appreciate the mechanism of this treatment (Machtei EE, Frankenthal S, Levi G, Elimelech R, Shoshani E, Rosenfeld O, Tagger-Green N, Shlomi B. Treatment of peri-implantitis using multiple applications of chlorhexidine chips: a double-blind, randomized multi-centre clinical trial. J Clin Periodontol 2012; 39(12): 1198-205).

Gianina Iovan