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Transcatheter Splenic Artery Occlusion for Treatment of Splenic Artery Steal Syndrome After Orthotopic Liver Transplantation

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Abstract

Purpose: To review some aspects of the problem of splenic artery steal syndrome as cause of ischemia in transplanted livers and treatment by selective splenic artery occlusion.
Materials and Methods: Eleven liver transplant patients from a group of 350 patients, nine men and two women, ranging in age from 40 years to 61 years (mean 52 years), presented with biochemical evidences of liver ischemia and failure, ranging from one to 60 days following orthotopic liver transplantation. Diagnosis of splenic artery steal syndrome was suspected by elevated enzymes, Doppler ultrasound and confirmed by celiac angiogram. Patients with confirmed hepatic artery thrombosis before angiography were excluded from the study. Embolization with Gianturco coils was performed.
Results: All patients were treated by splenic artery embolization with Gianturco coils. The 11 patients improved clinically within 24 hours of the procedure with significant change in the biochemical and clinical parameters. Followup ranged from one month to two years. One of the 11 patient initially improved, but developed hepatic artery thrombosis within 24 hours of the embolic treatment, requiring surgical repair.
Conclusion: Splenic artery steal syndrome following liver transplantation surgery can be diagnosed by celiac angiography, and effectively treated by splenic artery embolization with coils. Embolization is one of the treatments available, it is minimally invasive, and leads to immediate clinical improvement. Hepatic artery thrombosis is a possible complication of the procedure.

Key words: Arteries, therapeutic blockade—Liver transplantation—Liver, interventional procedure—Spleen, diseases—Splenic artery, steal syndrome—Hypertension, portal—Liver ischemia—Hepatic arteries, stenosis or obstruction—Hepatic artery thrombosis

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Hyperdynamic state is a relatively common finding in patients with chronic liver disease and portal hypertension [1]. Increased total splanchnic blood flow towards the spleen results from lowered splenic arteriolar resistance and enlarged splenic artery, as well as, in most cases, causes splenomegaly [2]. Liver transplantation does not result in immediate reduction of the total arterial blood flow in the spleen or splenic parenchymal volume in patients with preexisting increased splenic arterial flow, resulting in diversion of most of the celiac blood flow into the spleen, depriving the liver of a significant amount of arterial blood flow. In fact the condition can be aggravated by such events as preservation injury, rejection, or hepatitis, which usually result in increased intrahepatic arterial resistance, with further diversion of blood flow away from the hepatic artery, into the splenic artery. When there is a significant reduction in intra splenic arterial resistance, associated or not, with some degree of increased hepatic arterial resistance a “steal phenomenon” of the blood into the splenic artery may develop, causing biliary damage and liver failure. This situation of liver failure, reduced hepatic arterial perfusion (without arterial occlusion) and steal of the arterial blood flow into the splenic circulation, has been called “splenic artery steal syndrome” [3]. The splenic artery steal phenomenon has only recently been recognized as a potential threat to transplanted livers and has been already described in case reports or very small series [3–7]. Surgical splenectomy, splenic artery ligation, reanastomosis of the graft hepatic artery with an interposed vascular graft from the aorta, and splenic artery embolization have been proposed for the treatment of the splenic arterial steal [2]. Langer, et al. [3, 4] described the use of emboli-

zation as the treatment of choice to manage splenic arterial steal developing in liver transplant patients. Previous published work, suggested increased hepatic arterial blood flow and improvement in liver function following splenic artery embolization in patients with hyperdynamic splenic circulation and variceal bleeding [8, 9].

Our experience with splenic arterial steal syndrome is limited to a group of 11 patients with suspected liver ischemia (proved to have no hepatic artery thrombosis) from a series of 350 liver transplant patients. We would like to review some aspects of the problem of splenic artery steal syndrome as cause of ischemia in transplanted livers and treatment by selective splenic artery occlusion.

Materials and Methods

Eleven patients treated with orthotopic liver transplantation, nine men and two women, ranging in age from 40 years to 61 years (mean 52 years), presented with clinical and biochemical evidences of liver ischemia and failure.

Diagnosis

Diagnosis of possible liver ischemia was based on clinical and laboratory criteria, including failure to thrive, and acute changes in the liver function tests (LFTs) (elevated Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Gamma-Glutamyl Transferase (GGT), unchanged or slightly elevated Alkaline Phosphatase (ALK), and unchanged Bilirubin). Patients with those changes in the LFTs underwent Doppler ultrasound of the hepatic artery. Doppler ultrasound was considered to be abnormal, but non specific, when there was loss of hepatic arterial flow signal, or changes in the spectral waveform, with high-resistance wave pattern with increased resistive index (drop in systolic amplitude and reduced or stopped diastolic flow) and decreased flow velocities. Patients with confirmed hepatic artery thrombosis were excluded from this review. Seven of the patients had a pretransplant diagnosis of posthepatitis cirrhosis (Virus B and/or C), three had a diagnosis of alcoholic cirrhosis and one had a diagnosis of cryptogenic cirrhosis. Signs of liver failure due to possible liver ischemia presented within 1 to 60 days of the transplant.

Angiographic Diagnosis

Emergency arteriography was performed in all patients and a decision for embolization of the splenic artery was made following the study if the diagnostic angiogram was positive for splenic artery steal. The protocol for angiographic diagnosis consisted of selective angiography of the celiac trunk allowing simultaneous visualization the hepatic and splenic arteries, followed by selective superior mesenteric arteriography. Arterial portography was also performed. Forty to 50 ml at 7 ml/s of nonionic contrast (Iohexol, 350 mgI/ml, Nycomed Inc. Princeton, N.J.) were injected into the celiac trunk, during acquisition of digital images at 3 fr/s for 60 s.

Splenic arterial steal was diagnosed when the hepatic artery was patent but presented with sluggish flow, delayed filling of intrahepatic arterial branches by contrast material in comparison with other branches of the celiac trunk, poor peripheral parenchymal perfusion (enhancement), associated with early and abundant filling and of the splenic artery, which also showed increased size and flow. Also filling of the splenic and portal vein was simultaneous or preceded filling of the hepatic artery (Fig. 1).

Treatment

The transcatheter splenic artery occlusion was performed by selective catheterization of the splenic artery, followed by deployment of 3 to 8 metal Gianturco coils, ranging from 5- to 8-mm in diameter (Cook Inc., Bloomington, IN) in the middle part of the splenic artery. The splenic parenchyma was preserved in all cases, as demonstrated by the overall reduced but homogeneous splenic enhancement, and only the main splenic artery was occluded. Antibiotic therapy was always used before, and after the embolization procedure.

A post embolization celiac angiogram was performed to demonstrate changes or improvement in hepatic arterial perfusion. Criteria for improvement in hepatic arterial perfusion included; improvement in the peripheral liver arterial filling, without or reduced delay in comparison with other branches of the celiac trunk, including the splenic artery. Increased liver parenchyma enhancement in the late arterial phase. Increased size of smaller segmental hepatic artery branches. Improvement in hepatic artery timing for opacification in comparison to the portal vein.

The patients were followed clinically, by biochemistry using the LFTs described above, every six hours following treatment, and by Doppler ultrasound observing the protocol described in the M & M section, usually the next day following treatment. No special effort was made to document completeness of splenic artery occlusion. Followup studies for hepatic perfusion changes were performed every month for the first six months and every six months thereafter, or when clinically indicated.

Results

Eleven of 350 patients (3.2%) treated by orthotopic liver transplantation received a diagnosis of splenic artery steal syndrome as the cause of the liver ischemia, based on clinical, laboratory, ultrasonographic and angiographic findings.

All 11 patients with a positive angiographic diagnosis of splenic artery steal were treated by transcatheter splenic artery occlusion with Gianturco coils (Fig. 2). All 11 patients improved within 24 hours of the procedure with significant correction of the LFTs and clinical parameters. No symptoms related to the splenic artery embolization were identified. Follow-up ranged from one month to 24 months (mean 12 months). Follow up Doppler ultrasound consistently showed normal hepatic artery flow with improved systolic amplitude and increased diastolic flow. No significant changes in splenic size was observed in any of the patients treated. However, one patient with the diagnosis of splenic artery steal syndrome, initially improved after treatment by transcatheter splenic artery embolization, but 24 hours after the embolization procedure presented dramatic increase of LFTs, suggesting hepatic artery thrombosis (Fig. 3). Repeat Doppler ultrasound did not identify the hepatic artery. In retrospect this patient had a significant stenosis at the origin of the celiac trunk, which was overlooked. Emergency angiogram showed occlusion of the hepatic artery and con-

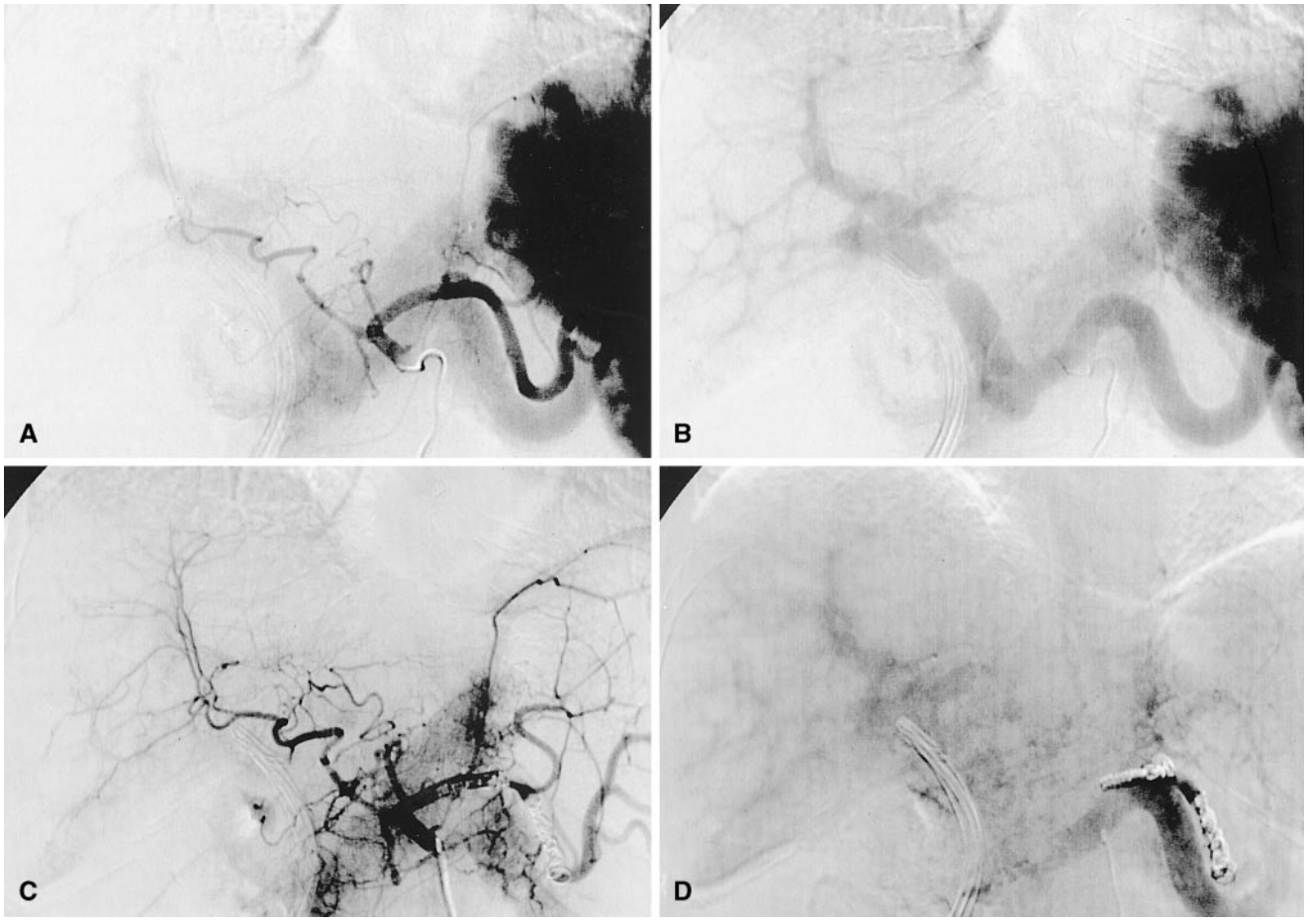


Fig. 1. **A** Selective celiac trunk arteriogram showed a small sized hepatic artery with reduced perfusion into the peripheral arterial circulation of the liver. Note enlarged splenic artery with increased flow and splenomegaly. The portal vein is prematurely opacified, before the hepatic artery filling reaches the periphery of the liver. Note the endoscopic biliary stents. **B** Late phase of the celiac arteriogram showed patency of the portal vein and persistent enhancement of the spleen. **C** Embolization of the mid splenic artery was performed with 8 mm metal Gianturco coils. Post-embolization angiogram showed partial occlusion of the splenic artery with development of collateral circulation through the left gastric artery. There was significant improvement in the hepatic artery perfusion, with dense opacification of the more peripheral hepatic arteries. **D** Post-embolization, late phase celiac angiogram showed, liver enhancement due to the flow in the hepatic artery, and mild portal vein perfusion of the liver. Note the coils within the splenic artery. There was significant clinical improvement after embolization.

firmed an 80% stenosis of the origin of the celiac trunk. Balloon angioplasty of the celiac stricture was performed and unsuccessful attempts of hepatic artery recanalization resulted in dissection at the level of the anastomosis, precluding further angiographic treatment. An interposed vascular graft using the patient's radial artery was performed. The patient slowly improved over a period of several days, eventually overcoming septic complications.

Discussion

In the earlier days of liver transplantation the main concern for liver ischemia and failure was acute thrombosis of the hepatic artery [10], which causes ischemia of the bile ducts and patch necrosis of the graft. Later on, anastomotic stricture of the hepatic artery, reducing the overall arterial flow to the liver, was also recognized as an important cause of liver ischemia [11–13]. Since the transplanted liver is more sensitive to ischemia, due to the reduced chances of developing collaterals, the biliary system is particularly targeted when there is occlusion of the hepatic artery. Hepatic artery thrombosis in this patient population may lead to bile duct necrosis and anatomical disruption with formation of bilomas. Hepatic artery thrombosis may occur in about 10% of the liver recipients, with higher rates in children [2, 14], however, liver ischemia can also be related to rejection or preservation injury, due to increased intrahepatic arterial resistance, following revascularization. An additional cause of graft isch-

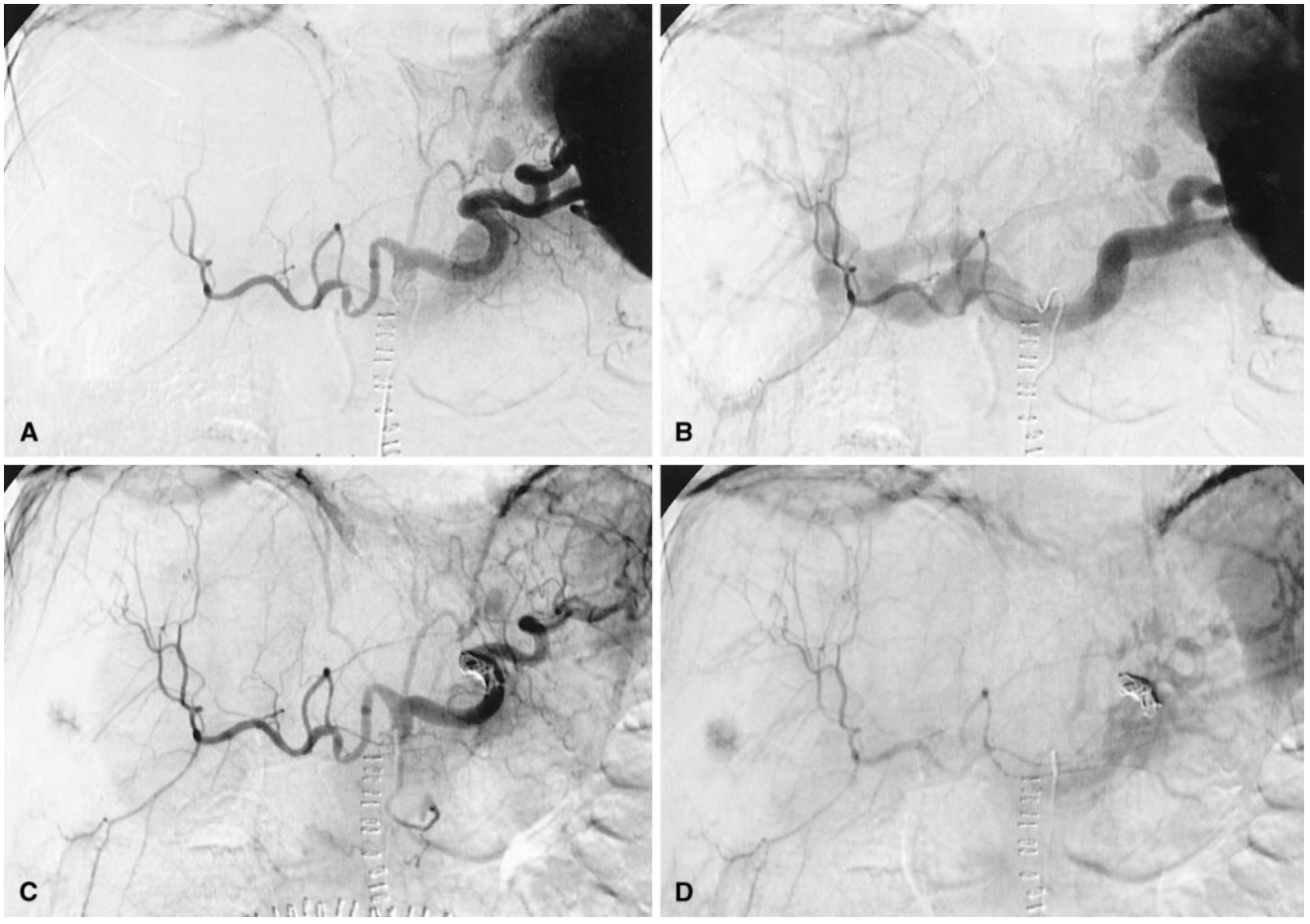
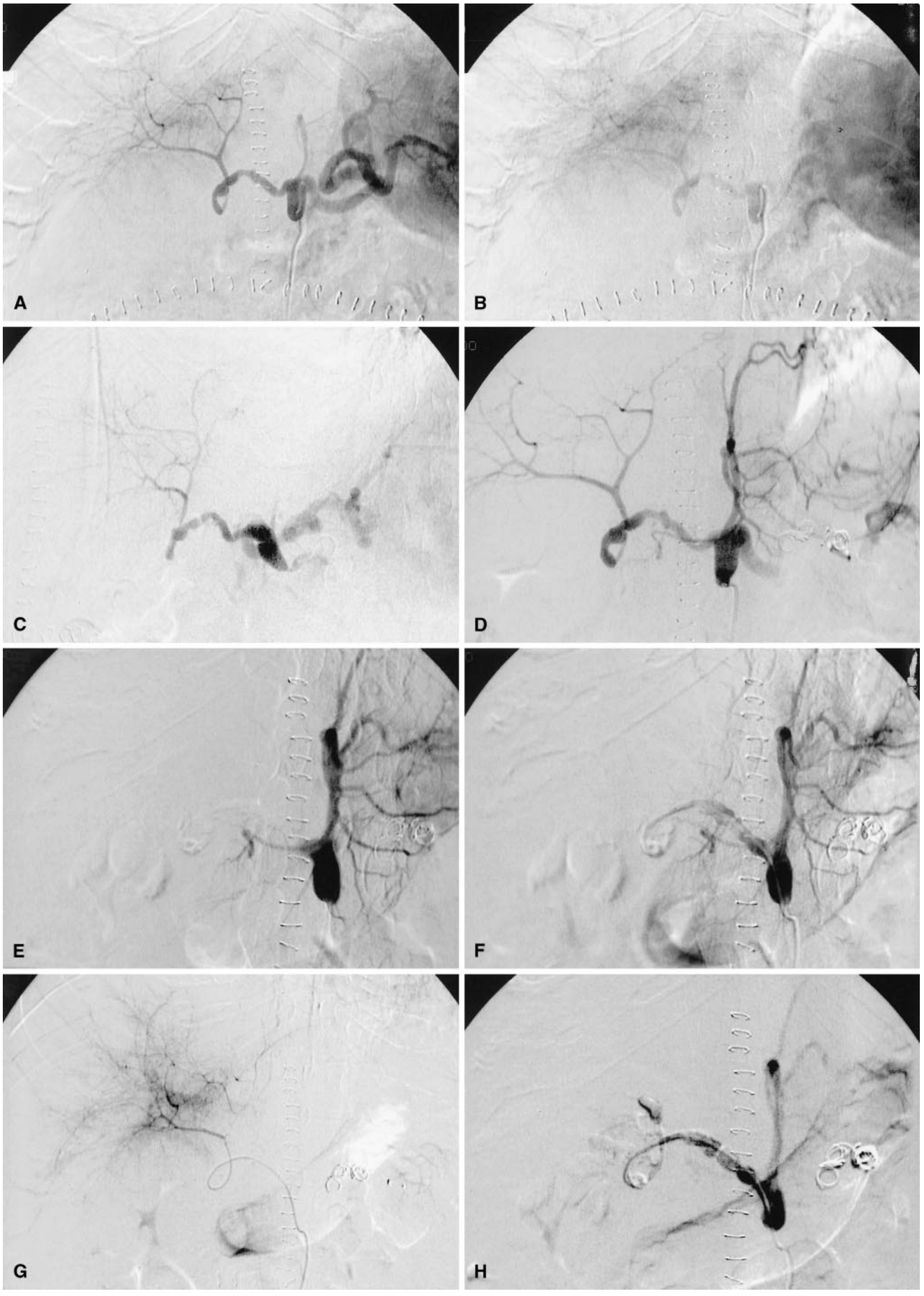


Fig. 2. **A** Selective celiac trunk arteriogram showed splenomegaly and enlarged splenic artery. Note delayed filling of the hepatic artery, in comparison with the splenic artery and celiac branches, while the splenic vein is already visible. **B** Late phase celiac arteriogram showed intrahepatic filling of the portal vein and right and left hepatic arteries with sparse filling of the peripheral hepatic artery branches. Splenic artery occlusion with Gianturco coils was performed. **C** Celiac trunk arteriogram after splenic artery embolization, showed partial obstruction of the splenic artery by the coils, with reduced perfusion of the splenic circulation and improved filling of the hepatic artery. **D** Later phase angiogram showed improved filling of peripheral hepatic artery branches and a small hypervascular lesion in the right lobe, that may have been related to previous needle biopsy. The portal vein is faintly visualized. There was immediate clinical improvement following embolization.

emia, which was recognized in 1990, is the "splenic artery steal syndrome" [3]. In those cases, liver ischemia is related to reduced perfusion or a situation of low flow status, through the hepatic artery, rather than obstruction. The most likely explanation for the reduced perfusion in the hepatic artery is the sump effect of an often enlarged splenic artery with increased flow, diverting the blood flow away from the hepatic artery [3, 4, 6, 7]. It is conceivable that the hyperdynamic state may act to the detriment of the patients in the postoperative period because the precapillary vascular resistance of the liver may remain normal while the increased blood flow in these patients may be shunted to structures such as the gut and spleen [1]. Other causes of transient or permanent increased intra hepatic arterial resistance, such as rejection or preservation injury, may worsen the effects of the splenic artery steal [2].

The splenic artery steal syndrome was reported to occur in about 4% of the patients with liver transplantation in a recently small published series [2] and occurred in 3.2% of our series of 350 patients with liver transplants. The splenic artery steal syndrome has been perceived as a problem only when more experience was obtained in dealing with the complications of liver transplantation. The ischemia of the liver graft in cases of splenic artery steal syndrome is usually acute, but in general more relenting and progressive than acute hepatic artery thrombosis. The differential diagnosis with graft rejection or ischemia related to preservation injury is difficult and usually requires a high degree of clinical suspicion and a more proactive diagnostic methodology. The onset of the splenic artery steal syndrome is variable and may occur in the first few hours after liver transplantation or can occur as late as several weeks following transplantation. It is impossible to anticipate how the hemodynamic of the celiac artery and splenic circulation will readjust to the new flow pattern following liver transplantation. Early cases of splenic artery steal syndrome probably occur in patients with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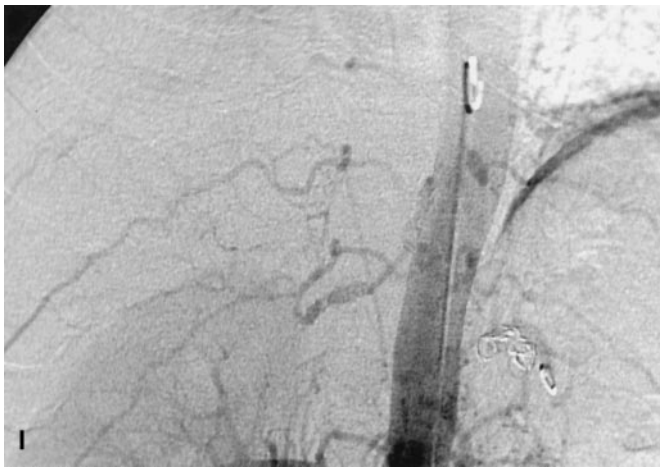

Fig. 3. **A** Celiac trunk arteriogram showed an enlarged splenic artery with splenomegaly. The hepatic artery showed reduced perfusion, with multiple irregularities in the walls, at the level of the anastomoses. **B** Late phase arteriogram showed mild liver enhancement and poor filling of the portal vein. **C** Oblique view of the celiac injection showed severe stenosis of the celiac trunk orifice. The stenosis was overlooked at the time of the procedure. Embolization of the splenic artery was carried out with Gianturco coils. **D** Angiogram of the celiac artery showed improvement in the hepatic artery perfusion, better visualized in the full angiographic series (not shown) with improved liver enhancement. There was immediate clinical improvement. **E** Within 24 hours following embolization the patient developed dramatic biochemical changes. Emergency angiogram showed thrombosis of the hepatic artery and occlusion of the splenic artery. Note persistent perfusion of the left gastric artery. **F** Attempts of hepatic artery recanalization were performed but failed. Note stagnant flow into the hepatic artery during contrast injection. **G** An 1.8 French microcatheter was advanced through the occluded artery and an angiogram demonstrated patency of the intrahepatic artery, which prompted surgical intervention decision to save the graft. **H**Subsequently dissection and extravasation developed, precluding further attempts of recanalization. **I** The patient was treated with the interposition of a radial artery connecting the hepatic artery to the aorta, which was followed by clinical improvement over a period of several days.

previous hyperdynamic states, enlarged spleen, reduced splenic arterial resistance and increased splenic arterial flow preexistent to the time of the transplant. In some early cases of splenic artery steal syndrome, the liver swell developed due to preservation injury may be an important factor in the equation, while later cases may be more related to progressive increase of the splenic arterial flow due to the preexisting hyperdynamic state, with or without development of clinical hypersplenism. Later occurrence of splenic artery steal syndrome might also be related to a preexisting condition of increased splenic arterial flow, not clinically significant at the time of the transplant and, aggravated by rejection of the graft or viral hepatitis. In our series of patients the symptoms developed within 1–60 days following liver transplantation. Curiously, only one of the three patients with late splenic artery steal syndrome (>30 days from surgery) presented with rejection and none with hepatitis reinfection.

We were surprised with the occurrence of hepatic artery thrombosis, in one patient, following splenic artery embolization. In this case the presence of an ostial stenosis of the celiac trunk was initially overlooked, and apparently the celiac artery, without the sumping action of the enlarged splenic artery, did not maintain enough blood flow or pressure within the celiac arterial system and the hepatic artery eventually thrombosed.

It is also possible to speculate that unrecognized initial splenic artery steal syndrome may be the cause of many of the real hepatic artery thrombosis seen in the liver transplant population, however, we could not prove that assumption based in our experience. It is important, however, to remark that angiography, besides a high degree of clinical suspicion, should be considered the most important tools for the diagnosis of splenic artery steal syndrome, mainly because Doppler ultrasound may not be sensitive enough to differentiate between hepatic artery strictures and hepatic artery low flow states or occlusion, as demonstrated in some cases in the series.

Proposed treatments may include splenectomy, splenic artery ligation, reanastomosis of the graft hepatic artery directly with the aorta and more recently transcatheter splenic artery occlusion. We have had experience to use angiography for the diagnosis of splenic artery steal syndrome in a number of patients with clinical and biochemical evidences of hepatic ischemia, which eventually resulted in a diagnosis of a hepatic artery low flow state. Angiography allowed the diagnosis and the access for transcatheter splenic artery occlusion in the same procedure, sparing the patients an additional open surgical procedure. It is our position, at this point, that splenic artery embolization, to treat splenic artery steal, should be carried on in the middle part of the splenic artery, in order to avoid splenic parenchymatous ablation and hopefully reduce the risks of pain and infection. The embolization occlusion may not be necessarily immediate, and in some cases delayed or progressive obstruction was perfectly adequate. Besides preventive antibiotic therapy, no other special pre or post procedure care was necessary in our series. Embolization may become the therapeutic procedure of choice, for treating splenic artery steal syndrome, as suggested by Langer et al. [3, 4, 6, 7] and by our own experience. However, there is still controversy in the scarce literature available, and reanastomosis of the hepatic artery of the graft to the receptor's aorta with an interposed vascular graft is the preferred treatment in the experience of some authors [2]. Intraoperative measurement of graft blood flow may accurately predict an impaired liver graft flow that may result in damage to the function of the liver [15]. And, indeed, when a hyperdynamic state in the splenic circulation is evidenced during transplantation procedure, arterial reconstruction should be carried out with the interposition of a

graft directly to the aorta, thus excluding the splenic circulation from the hepatic artery anastomosis, and therefore avoiding the possible occurrence of the steal phenomenon in the postoperative course. However, an adequate conduit may not be immediately available for the interposition graft. Intraoperative ligation of the splenic artery may also be carried out to prevent splenic artery steal syndrome. However, when a splenic hyperdynamic state is not identified previous to transplantation surgery, or develops during the postoperative course, the more central splenic artery embolization with coils may be an adequate treatment, because it may be able to save the graft, it is minimally invasive and very effective, as demonstrated in our series. In addition to that, middle splenic artery embolization, as used in our series, produced no significant post-embolization syndrome with pain or the infectious complications observed with the partial splenic embolization, when used for treating hypersplenism syndrome. Hepatic artery thrombosis, however, is a possible complication of the procedure.

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