

Clinical applications of

NITINOL VASCULAR PLUGS

CLINICAL APPLICATIONS OF NITINOL VASCULAR PLUGS

Javier Peiró Fernando Camúñez José Urbano

Endorsed by spanish society of vascular and interventional radiology.



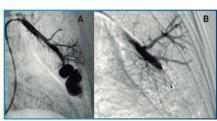


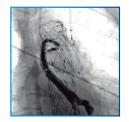
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INTRODUCTION

Kurt Amplatz was a pioneer in the field of vascular and interventional Radiology. He developed new minimally invasive techniques for the treatment of vascular and non-vascular disorders. One of the most amazing and well-known devices crafted by him was the nitinol vascular plug in its different variants indicated for structural cardiology and for peripheral vascular applications. Informally known as "Amplatzer", nitinol vascular plugs or "plugs" are currently an essential tool for endovascular specialists.

All types of "Amplatzer" are self-expandable devices made of single-layer mesh of nitinol wire creating a single or multilobule shape. As nitinol has a poor radiopacity, radiopaque markers in the distal and proximal ends of the plugs allow for a good visibility both folded inside the catheter and expanded in the vessel lumen. They are attached to the delivery wire by a micro-screw that typically has five turns. With this system, they can be recaptured, repositioned and redeployed during a procedure, until the operator is sure that the most effective position of the plug inside the vessel has been achieved. For this reason, nitinol vascular plugs are safe and provide a great precision during embolisation procedures. They can be placed both in veins and in arteries. A diameter oversizing has to be taken into account according to the target vessel. Generally, a 30% oversize for arteries and 50% for veins is required to ensure good apposition in the vessel wall but depending on each case this rule may be modified. There are three different variants of plugs (AVP, AVP II and AVP 4) which are available in a matrix of sizes from 4 to 22 mm. They can be delivered by regular diagnostic catheter, by guide-catheters or by long sheaths. The landing zone must have sufficient length to avoid non-target vessel occlusion. Due to the necessary oversizing of these devices, the implanted device length once inside the vessel, will be marginally longer than the stated nominal length. Although "Amplatzer" plugs provide optimal embolisation with full cross-sectional vessel coverage, the operator has to be aware that many times during the immediate post-deployment control angiography a residual flow across the plug can be appreciated. This is never a problem and in few minutes, occlusion will be definitive and permanent. Tortuous vessels may impact on the pushability of these devices inside the catheter. If this happens, our recommendation is to choose a larger diameter catheter than recommended, for each plug size.

Clinical applications of nitinol vascular plugs are diverse. In general, we could use a plug anywhere where a coil can be used. A clear advantage of plugs is when a high flow vessel or a wide vessel has to be occluded. In those cases, plugs provide reduction in procedure time and reduction in materials used and consequently reduction in radiation exposure and cost. EVAR, TEVAR, interventional oncology, bleeding patients, portal hypertension, pulmonary AVMs, fistulas and gonadal veins are the most popular indications. On the other hand, the learning curve for their use is easy and short,

INTRODUCTION

and together with the safety of the device, making plugs an attractive user-friendly option for endovascular specialists.

This book has been written with the intention to provide further insight specific to the use of vascular plugs in your daily practice. The authors have selected some of their most significant cases which represent a good example of the main indications and applications of the 3 types of "Amplatzer" nitinol vascular plugs. A brief description of the materials and technique is also provided for each case.

GLOSSARY

AAA	Abdominal Aortic Aneurysm
A0	Aorta
ASD	Atrial Septal Defect
AVF	Arteriovenous Fistula
AVP	Amplatzer Vascular Plug
CCLM	Colorectal Cancer Liver Metastasis
CFA	Common Femoral Artery
COPD	Chronic Obstructive Pulmonary Disease
CRP	C-reactive-protein
СТА	Computed Tomography Angiography
DL	Dyslipidemia
EVAR	Endovascular Aneurysm Repair
EVOH	Ethylene Vinyl Alcohol
F	French
FLR	Future Liver Remnat
G-E	Gastro-Esophageal (varices)
GI	Gastrointestinal (bleeding)
НВР	High Blood Pressure
HBV	Hepatitis B Virus
HCC	Hepatocellular Carcinoma
HCV	Hepatitis C Virus
HD	Hemodialysis
IAA	Internal Iliac Artery
IMA	Inferior Mesenteric Artery
IMV	Inferior Mesenteric Vein
IU	International Units

GLOSSARY

IV	Intra Venous			
GDA	Gastroduodenal Artery			
LHL	Left Hepatic Lobe (liver)			
LSA	Left Subclavian Artery			
MDCT	Multidectector Computed tomography			
MI	Miocardial Infaction			
MIP	Maximum Intensity Projection			
MPR	Multiporpose (catheter)			
MRI	Magnetic Resonance Imaging			
NAFLD	Non-Alcoholic Fatty Liver Disease			
PAVM	Pulmonary Arteriovenous Malformation			
PTFE	Polytetrafluoroethylene.			
PEVAR	Percutaneous Endovascular Aneurysm Repair			
PTA	Percutaneous Transluminal Angioplasty			
PTCA	Percutaneous Transluminal Coronary Angioplasty			
PVA	Polivinyl Alcohol (microparticles)			
PVE	Portal Vein Embolisation			
RE	Radioembolisation (Y90)			
SMA	Superior Mesenteric Artery			
SMV	Superior Mesenteric Vein			
TACE	Trans-Arterial Chemoembolisation			
TEVAR	Thoracic Endovascular Aneurysm Repair			
TIPS	Transyugular Intrahepatic Portosystemic Shunt			
UGIB	Upper Gastrointestinal bleeding			
US	Ultrasound			
VR	Volume Rendering			
Y90	Ittrium 90			

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CHAPTER 1 Aorto/arterial

1

AORTO-UNI-ILIAC STENT-GRAFT: AMPLATZER AS AN ANCILLARY TOOL

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MEDICAL HISTORY

65 mm AAA was discovered in an 80-year-old male as an incidental finding in an abdominal CT (Figs. 1 and 2). Considering AAA size and the patient age, endovascular treatment was decided.

13-mm diameter at the bifurcation of the abdominal aorta rule out a bifurcated device. It was decided to place an aorto-uni-iliac stent-graft through the right iliac access and a vascular plug in the left common iliac plus a femorofemoral by-pass.





■ Figures 1 and 2. Abdominal CT in arterial phase with 3D and coronal reformations showing an AAA as well as vascular anatomy for planning stent-graft placement.

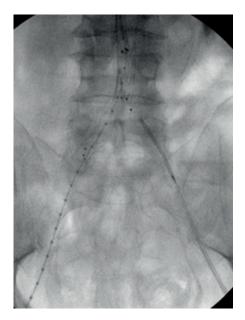
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DESCRIPTION OF THE TECHNIQUE

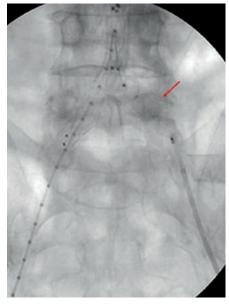
The aortic stetngraft and the iliac extension (Endurant TM - Medtronic TM) were implanted from right femoral cutdown.

Percutaneous puncture of the left femoral artery was performed and a 7-F sheath (Flexor Check Flo® - Cook®) was inserted and advanced to the origin of the common iliac artery. Selective arteriography was performed through the sheath to mark off the common iliac artery and the origin of the hypogastric artery.

Left common iliac artery diameter was 11 mm. A 16 mm diameter AVP II (Abbott) was deployed through the 7-F long sheath in the common iliac artery (Figs. 3 and 4).



■ Figure 3. Deployment of the most proximal part of the Amplatzer in the left common iliac. The vascular plug progresses through the 7-F sheath.



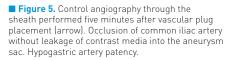
■ Figure 4. Complete deployment of the Amplatzer in the common iliac artery (arrow). It is still attached to the guidewire.

Control arteriography performed five minutes after plug placement revealed occlusion of the common iliac artery without filling the aneurysm sac and maintain internal iliac artery patency (Fig. 5).

The control CT scan one month later shows EVAR patency with no endoleaks. The external iliac and left hypogastric arteries are patent (Fig. 6).

AORTO-UNI-ILIAC STENT-GRAFT: AMPLATZER AS AN ANCILLARY TOOL







■ Figure 6. One month control CT. Volume rendering 3D reformation shows patency of the aorto-uni-iliac EVAR and femorofemoral by pass. No endoleaks are seen.

COMMENTS

Abdominal aortic aneurysms may be treated surgically or endovascularly. EVAR treatment of choice is aorto-bi-iliac stetn-graft placement. There are different scenarios in which bi-iliac technique cannot be performed, such as narrowing of the aortic bifurcation (< 20 mm), small iliac artery (< 7 mm), or severe iliac angulation. In these cases, the standard endovascular treatment is aorto-uni-iliac stent-graft with a femorofemoral by-pass and an occluder in the contralateral common iliac artery. In our experience, Amplatzer nitinol vascular plugs can be used effectively and safely as an occlusion device for the common iliac artery. AVP shows a great precision in its placement and the possibility of repositioning if the device occludes the origin of the ipsilateral hypogastric artery. It is also highly conformable in tortuous iliac arteries

AORTO-UNI-ILIAC STENT-GRAFT: AMPLATZER AS AN ANCILLARY TOOL

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2

VASCULAR PLUG FOR EMBOLISATION OF INTERNAL ILIAC ARTERY PRIOR TO EVAR

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MEDICAL HISTORY

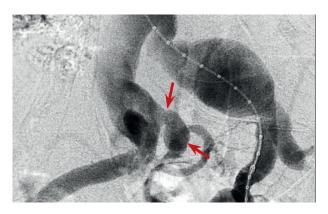
77-year-old male smoker, with pharmacologically controlled HBP and DL. Asymptomatic aorto-bi-iliac aneurysm (Fig. 1a, 1b). EVAR treatment with bifurcated stentgraft was indicated, which should be anchored distally over both external iliac arteries, exceeding the origin of both hypogastric arteries. After analysing the anatomy of the aneurysm and based on the data of the MDCT, it was decided to occlude, using a nitinol vascular plug, the origin of the right internal iliac artery (IIA) to prevent a type II endoleak and place a branch over the left IIA to maintain pelvic irrigation.



■ Figure 1. Preoperative volume rendering reformation in AP and oblique view. Aneurysm affecting both common internal iliac arteries.

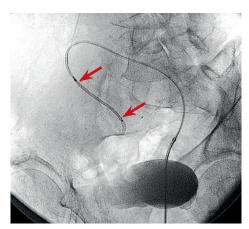
DESCRIPTION OF THE TECHNIQUE

Calibrated angiography of the iliac arteries was performed in frontal and oblique views to decide the best access to the right hypogastric artery (Fig. 2).



■ Figure 2. Calibrated iliac angiography. Left oblique view. The origin and the common trunk of right IIA, where the AVP should be deployed are well depicted [arrows]

The contralateral approach using a crossover technique appeared to be the most appropriate to achieve a stable canalisation of the right IIA trunk. Due to the tortuosity of the iliac arteries, it was decided to use a more flexible guide catheter with better navigability instead of a long introducer to channel the right IIA. Based on CT and arteriography, a 12 mm AVP II (Abbott) was chosen to occlude the right IIA trunk. The purpose was to respect and maintain patent bifurcation of the IIA in its anterior and posterior division. Although the 12 mm AVP II can be theoretically introduced through a 6-F guide catheter, we decided to use a 7-F guide catheter, (Mach® 1, Boston Scientific), in order to have a better navigability and pushability of the vascular plug (Fig. 3). Using a pull back and push manoeuvre, the Amplatzer was deployed. Control was performed through the guide catheter confirming the correct position without occluding the external iliac artery or invading the common iliac artery (Fig. 4).



■ Figure 3. 7-F guide catheter with its tip well placed inside the right IIA. 12 mm AVP II folded inside the catheter (arrows).

VASCULAR PLUG FOR EMBOLISATION OF INTERNAL ILIAC ARTERY PRIOR TO EVAR

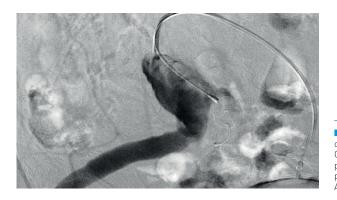


Figure 4. AVP II of 12 mm deployed but not released. Control checking the correct position of the device. At this point we could still recover the Amplatzer if necessary.

The device was released (Fig. 5). The procedure was completed placing a branch in the left IIA and anchoring distally the EVAR legs in both external iliac arteries (Fig. 6).

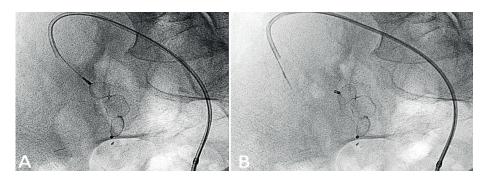


Figure 5. A) AVP II deployed. On pre-release. B) AVP released from its delivery guidewire.

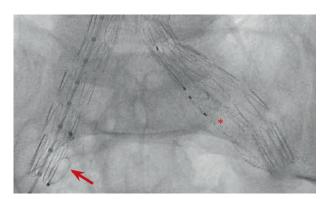


Figure 6. Final stentgraft release with anchoring in both external iliac arteries. The left side shows the branch that will maintain patency of the hypogastric territory (asterisk). The right side shows the vascular plug outside the iliac stent farrow).

32 months later, the patient was asymptomatic, the aneurysm sac had decreased in size both at the iliac and abdominal level, and he did not suffer gluteal claudication.

COMMENTS

A significant proportion of patients undergoing EVAR have common iliac artery aneurysms. Occlusion of the IIA may be necessary prior EVAR to prevent type II endoleak. Although the current trend is to preserve hypogastric arteries using a branch when performing EVAR of aortoiliac aneurysms, sometimes the hostile anatomy or complexity of the procedure do not allow revascularisation of the IIAs that must be sacrificed to successfully treat the aneurysm. On purpose occlusion of the hypogastric artery prior to EVAR has been done since the begining of this technique and is particularly indicated when the origin of the hypogastric artery drains to a common iliac aneurysm. It is desirable to perform an embolisation as proximal as possible in the IIA trunk, avoiding the anterior and posterior IAA divisions bifurcation to promote collateral circulation and prevent secondary ischaemic complications. In general, occlusion of a hypogastric artery is well tolerated without major complications if the contralateral artery remains patent. Buttock claudication and erectile dysfunction occurred in 25% of cases after unilateral IIA sacrifice. Where coils or plugs are technically possible, plugs could be considered preferential to coils, and placed as proximally in the IIA as possible. Nitinol plugs will not provoke streak artefacts in future MDCT controls.

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 53:534-548.

3

VASCULAR PLUG FOR PREVENTION OF TYPE II ENDOLEAK BEFORE EVAR

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CASE 1

MEDICAL HISTORY

70-year-old male, asymptomatic, former smoker, obese, with a history of HBP and hypertriglyceridaemia. Alcoholic chronic liver disease with portal hypertension and hypersplenism. Incidental AAA finding of 56×54 mm. Because of his liver disease and thrombocytopenia, percutaneous endovascular treatment of aneurysm (PEVAR) is indicated

DESCRIPTION OF THE TECHNIQUE

CTA and calibrated arteriography were performed two weeks prior to the procedure showing patency of four lumbar arteries, the inferior mesenteric artery (IMA) and a right inferior renal polar artery supplying the lower pole of the right kidney (RK) that originates in the neck of the aneurysm adjacent to the aneurysm sac (Figs. 1 and 2).

Two weeks later, PEVAR was performed under general anaesthesia, after antibiotic prophylaxis and with preinstallation of percutaneous closure sutures (Prostar®, Abbott Vascular) in both common femoral arteries. An IV bolus of 8000 IU of heparin was administered and right 18-F and left 14-F sheaths were inserted in both femoral arteries. Catheterisation of the right inferior renal polar artery with a 4-F Cobra catheter (Tempo® Aqua Cordis) and embolisation using two 6 mm AVP 4 (Abbott) was performed (Fig. 3). Embolisation of the IMA ostium with fibered coils was also done. Finally a bifurcated stent-graft was deployed (Endurant II®, Medtronic)

Twenty-four months later, the control CTA showed a significant decrease in the aneurysm sac $(43 \times 34 \text{ mm})$ without endoleak. The polar artery is thrombosed and nitinol plugs are visible and do not cause artifacts on CT (Fig. 4).

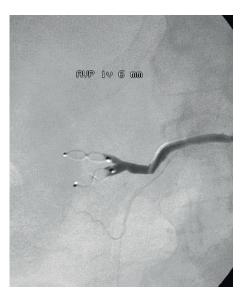
VASCULAR PLUG FOR PREVENTION OF TYPE II ENDOLEAK BEFORE EVAR



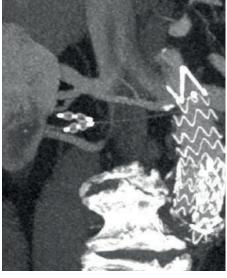
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■ Figure 1. Calibrated aortogram. Right inferior kidney polar artery originating in the aneurysm neck adjacent to the aneurysm sac (arrows).

■ Figure 2. Selective right polar artery arteriography.



■ Figure 3. Occlusion of polar artery with two AVP 4 measuring 6 mm. immediate control arteriography.



■ Figure 4. Control CT at 24 months. Visible AVP 4 without artifacts. Thrombosed polar artery. Small infarction in lower pole of right kidney.

CASE 2

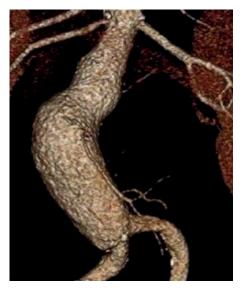
MEDICAL HISTORY

65-year-old, overweight, ex-smoker male with acute promyelocytic leukaemia in complete remission and in maintenance treatment with methotrexate and retinoic acid. Asymptomatic AAA of 65 x 62 mm as incidental finding. CTA showing enlarged IMA (5.5 mm) and several patent lumbar arteries. The aneurysm neck is slightly conical, with no calcium or thrombus. Normal common iliac and femoral arteries (Fig. 5).

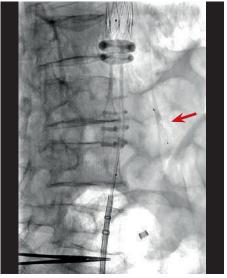
DESCRIPTION OF THE TECHNIQUE

PEVAR was performed using a procedure similar to case A. Methotrexate was discontinued two weeks before. After sheaths and percutaneous sutures were placed, and before stent-graft delivery, the IMA was catheterised with a 4-F Cobra catheter (Tempo® Aqua Cordis) and embolised with an 8 mm AVP 4 nitinol occluder (Fig. 6). Placement of a low profile bifurcaterd stent-graft (Ovation®, Trivascular-Endologix) was performed. Immediate control aortogram with good outcome and no visible endoleaks.

Control CTA one year later revealed aneurysm sac shrinkage and no endoleak.



■ Figure 5. 3D CTA reformation showing the patent IMA arising from the anterior aspect of the AAA.



■ Figure 6.8 mm AVP 4 occluding IMA origing immediately before the stent-graft is released (red arrow).

COMMENTS

Type II endoleaks due to flow re-entry through collateral vessels in the aneurysm sac, once the stent-graft is placed, are generally considered benign. However, once detected, they require close monitoring. If the aneurysm sac grows or the type II endoleak is persistent, it should be treated and the diagnosis of endoleak type I or III should be ruled out. Although rare, cases of AAA rupture due to type II leaks have been reported. A classic paper by Radiology in 2001 postulated that the occurrence of type II leaks is directly related to the number of patent collateral arteries (IMA, lumbar, polar and middle sacral). Physiopathologically, an IMA \rightarrow lumbar circuit is established that perpetuates the leak. This is much more common in anticoaqulated patients. Thus, whenever AAA anatomy allows, we perform prior embolisation of the origin of the IMA to prevent a potential type II leak. This procedure is very well tolerated and has no consequences when the embolisation is performed in the first centimetres of the IMA, before the origin of the left colic artery to allow for a good collateral circulation by the arc of Riolan. This embolisation may be performed a few days before or in the same of EVAR procedure. In our experience, we have found no differences when performing it in one or two stages. The AVP 4 nitinol plug, due to their controlled release, precision, and because they are released through a conventional 4-F catheter and produce no artifacts in the CT, are, in our experience, one of the best possible options for performing this technique.

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4

LEFT SUBCLAVIAN ARTERY COVERAGE WITH VASCULAR PLUG DURING TEVAR

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MEDICAL HISTORY

80-year-old male, ex-smoker since 10 years ago, pharmacologically-controlled HBP and dyslipidemia. Asymptomatic, with good general condition and independent lifestyle. Routine examination with PA and L chest X-rays revealed a mediastinal mass, so chest CTA was indicated. A 64 mm aortic aneurysm was found, located immediately distal to the origin of the left subclavian artery (LSA) (Fig. 1a, 1b). Endovascular treatment with thoracic endograft repair was decided occluding the LSA origin.

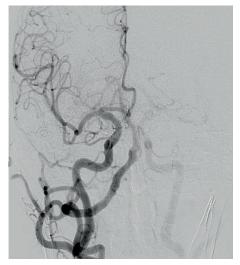




■ Figure 1. A) Preoperative axial CT that show 6.4-cm aneurysm of the descending thoracic aorta. B) MDCT. Volume rendering reformation of the aortic arch. Common axis for both carotid arteries. Aneurysm reaching left subclavian artery origin.

DESCRIPTION OF THE TECHNIQUE

The preoperative study included a carotid Doppler US and brain MRI to rule out any silent ischaemic injuries. A cerebral angiography with selective study of the 4 supraortic vessels was performed several days before surgery (Fig. 2). Cut down of the right CFA from which the stentgraft was inserted. 5-F introducer in left CFA to perform controls with calibrated angiography. Immediately before aortic stentgraft placement, the left subclavian artery was catheterized, and a 6-F and 90 cm long sheath (Destination®, Terumo) was advanced, the tip of which was stable in the origin of the left subclavian artery (Fig. 3).



■ Figure 2. Cerebral arteriography confirming patency of the circle of Willis.

■ Figure 3. 90 cm 6-F sheath (Destination®, Terumo) advanced into LSA. A 12 mm AVP I was deployed in the trunk of LSA but it was not yet released. Control angiography through the sheath ruled out vertebral or mammary arteries occlusion

Due to the short landing zone and the need to oversize at least 30% the vascular plug, we chose an AVP I device. The AVP I, though with less occlusion power than type II, is more adequate when we have a short length landing zone. To maintain collateral circulation it was essential that the left vertebral and mammary arteries remain patent and not covered by the vascular plug (Fig. 4). Finally, the stentgraft was released covering the origin of the LSA. The immediate control arteriography showed a good outcome with the excluded aneurysm, with no endoleak (Fig. 5). In the late phase, supply to the left upper limb was clearly seen thanks to reversal flow through the vertebral artery (Fig. 6).

Four years later, the aneurysm is still excluded, and the patient is asymptomatic without suffering any neurological complication or claudication of the left upper extremity. Surgical revascularisation of the LSA was never necessary in this patient.

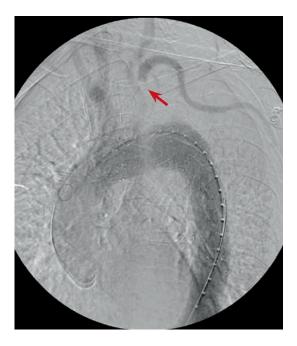
LEFT SUBCLAVIAN ARTERY COVERAGE WITH VASCULAR PLUG DURING TEVAR





■ Figure 4. Control angiography immediately after releasing AVP I. It is seen how the sac of the aneurysm includes the origin of the LSA. Vascular plug (arrow) in the right position without occluding the vertebral or mammary arteries.

■ Figure 5. Early acquisitions of the final control angiography after LSA AVP I occlusion and stentgraft deployment. Aneurysm excluded. LSA is not seen.



■ Figure 6. Final control angiography. Late acquisitions after LSA AVP I occlusion and stentgraft deployment. Retrograde flow from left vertebral artery feeding LSA. There is no endoleak. Plug (arrow).

COMMENTS

When we perform TEVAR and in order to obtain an effective anchorage on a healthy neck of the aorta, sometimes it will be necessary to occlude the origin of the LSA with the fabric of the endograft. If the aneurysm sac or false lumen of a dissection are very close to or include the ostium of the LSA, then type II endoleak due to retrograde re-entry through the subclavian artery is ensured. This is why occlusion of the LSA trunk in the segment between the AO and the origin of the left vertebral artery should be performed either during the same procedure or a few days before or later TEVAR. It is technically a simple procedure for any interventionist. A mechanical embolisation will be used, and although the coils are the most popular, nitinol plugs are, in author's experience, the most advisable because they are simple, controlled release, and also because they do not cause streak artifact in CTA controls that these patients undoubtedly require after treatment.

Occlusion of the LSA is well accepted in emergency cases. In scheduled cases, it is necessary to perform a cerebral arteriography before, assessing vertebral and carotid arteries, confirming the patency of the circle of Willis and, if necessary, perform an occlusion test to establish the patient's tolerance to the LSA occlusion. In most cases, this manoeuvre is well tolerated. Although occlusion of the LSA theoretically increases the risk of stroke and spinal ischaemia, surgical bypass revascularization is not free of complications and the risk-benefit of each procedure should be carefully considered. Pulse deficits in the left arm are common after subclavian artery coverage, but ischemic symptoms are infrequent. LSA coverage without revascularization can be safely performed during TEVAR in patients with bilateral patent vertebrobasilar junctions.

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5

DEEP FEMORAL ARTERY PSEUDOANEURYSM AND ARTERIOVENOUS FISTULA

M. Echenagusía, G. Rodríguez, M. González, A. Álvarez and F. Camúñez

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MEDICAL HISTORY

58-year-old man with medical history of atrial septal defect (ASD) operated in 1990. Mitral valve replacement and tricuspid annuloplasty in 2005. Now he is admitted to the Cardiology Department suffering endocarditis and mitral insufficiency. Cardiac surgery was performed again and postoperatively he had acute renal failure and severe liver insufficiency.

The patient was under oral anticoagulant therapy.

Due to a Shaldon catheter placement in the right femoral vein, the patient suffered a large hematoma in the groin and a pseudoaneurysm of the femoral artery was suspected.



CTA confirmed pseudoaneurysm arising from the deep femoral artery associated with an arteriovenous fistula (Fig. 1).

Given the comorbidity of the patient and the need to maintain anticoagulation therapy, treatment with embolisation was chosen.

[■] Figure 1. CTA. 2 cm pseudoaneurysm in right deep femoral artery and arteriovenous fistula.

DESCRIPTION OF THE TECHNIQUE

Selective right iliofemoral arteriography was performed using a contralateral left femoral approach. A pseudoaneurysm of approximately 2 cm arising from the right deep circumflex femoral artery with arteriovenous fistula was confirmed (Fig. 2).

The affected branch was superselectively canalised using a 4-F multipurpose catheter (Aqua Tempo®, Cordis) (Fig. 3). It was decided to embolise with an Amplatzer™ vascular plug (Abbott) as probably the safest device in the presence of arteriovenous fistula associated to pseudoaneurysm. Two 4 mm AVP 4 were implanted.



■ Figure 2. Right femoral arteriography confirming the suspected diagnosis.

■ Figure 3. Supraselective arteriography of the right deep circumflex femoral artery.

Later control angiography showed a satisfactory result. The pseudoaneurysm was excluded and the arteriovenous fistula was closed (Fig. 4).

Before hospital discharge, a control CTA was performed with evidence of complete pseudoaneurysm and arteriovenous fistula occlusion (Fig. 5). The patient was discharged with preserved distal pulses and no pain in the right lower limb.

DEEP FEMORAL ARTERY PSEUDOANEURYSM AND ARTERIOVENOUS FISTULA







■ Figure 5. Control CTA before patient discharge. AVP 4 devices (arrow) with complete sealing of pseudoaneurysm and fistula.

COMMENTS

The use of vascular plugs for embolisation of traumatic lesions in peripheral arteries provides a fast, effective and safe alternative to other embolisation materials. AVP strengths are their controlled release mechanism, low risk of migration, and speed of implantation. The possibility of using AVP 4 through a 4-F diagnostic catheter is another favourable characteristic for this type of device. In addition, procedure time and radiation exposure is reduced.

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PELVIC TRAUMA

J. Peiró, Á. Iglesias and V. Nacarino

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MEDICAL HISTORY

46-year-old patient, reported to the emergency room after suffering a work accident with pelvic and chest trauma due to a sand avalanche.

Complementary tests performed showed the existence of:

- 1. Pelvic fracture with vertical instability with bilateral fracture of ilio-ischiopubic branches.
- 2. Fracture of the left transverse process of L5.
- 3. Fracture of 7th left rib and 7th and 11th right ribs.
- 4. Left pneumothorax.
- 5. Left perirenal haematoma.

The patient presented hypovolemic shock, and emergency arteriography and embolisation were requested to fix the acute hemorrhage.

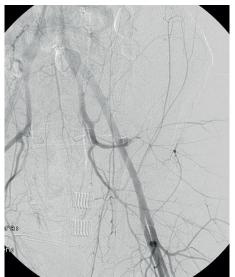
DESCRIPTION OF THE TECHNIQUE

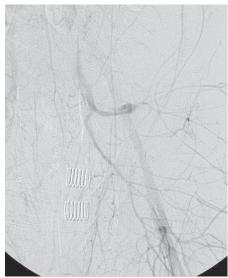
By right femoral access, abdominal aortography and pelvic arteriography were performed, noting the existence of contrast extravasation due to active bleeding in the superior gluteal artery, branch of the left hypogastric artery (Figs. 1 and 2).

The left internal iliac artery was selectively canalized using a 4-F diagnostic regular catheter, and a selective arteriography, confirmed the bleeding due to rupture of the superior gluteal artery (Figs. 3 and 4).

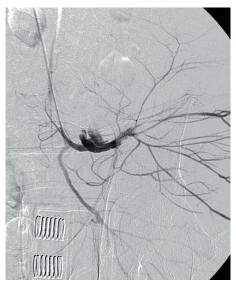
A 4 mm diameter AVP 4 nitinol vascular plug (Abbott) was implanted through the diagnostic catheter, evidencing persistent bleeding in the post-procedure arteriography, performed immediately after implantation, so another 6 mm diameter AVP 4 (Abbott) was implanted. The closure was obtained and cessation of bleeding were confirmed with a new control arteriography post-procedure (Figs. 5 and 6).

PELVIC TRAUMA





■ Figures 1 and 2. Pelvic arteriography showing contrast media extravasation due to active bleeding in the left superior gluteal artery.

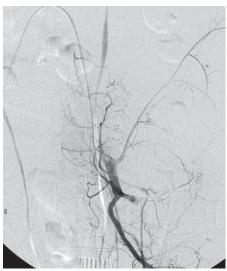




■ Figures 3 and 4. Left hypogastric selective arteriography confirming acute bleeding and laceration of the left superior gluteal artery .

PELVIC TRAUMA





Figures 5 and 6. Cessation of bleeding after closure of the left upper gluteal artery by two AVP 4 of 4 and 6 mm in diameter.

COMMENTS

Pelvic fracture is present in 20% of cases of severe polytrauma. Between 10% and 20% of patients who have suffered pelvic trauma, curse with hemodynamic instability due to acute bleeding, with 30% mortality in these cases. Most cases occur due to traffic accidents, and in this particular case, due to an occupational accident resulting from a sand avalanche.

The use of vascular occluders in acute bleeding episodes is highly effective, though it is important to take into account that it requires time to embolise, which we did not do in this case in which the first occluder would probably have been sufficient, but we placed a second one, as indicated above, due to the patient's clinical status.

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TRAUMATIC SUPRASCAPULAR ARTERIOVENOUS FISTULA

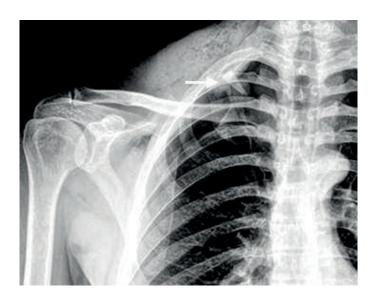
V. Nacarino, Á. Iglesias and J. Peiró

University Hospital Virgen del Rocío, Serville.

MEDICAL HISTORY

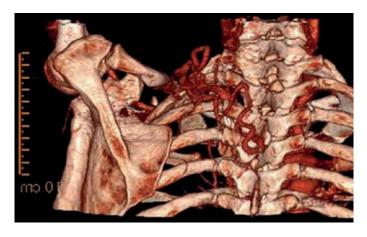
48-year-old man reported to the emergency room after suffering an accidental fall against a glass door, suffering a penetrating wound at suprascapular level. Plain chest X-rays evidenced right pneumothorax, subcutaneous emphysema, pleural effusion, and a foreign body (Fig. 1). Lateral thoracotomy, foreign body extraction, and placement of endothoracic drainage were urgently performed. The patient recovered and was discharged.

At six months, the patient complained of a pulsatile mass in the right supraclavicular region. Contrast-enhanced CT revealed a right suprascapular arteriovenous fistula, dependent on the subclavian artery (Fig. 2). Endovascular treatment was decided.



■ Figure 1. Plain chest X-ray. Subcutaneous emphysema and foreign body located in the right supraclavicular region (arrow).

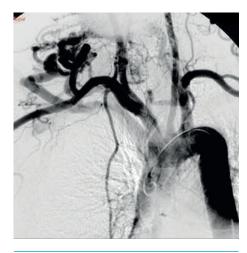
TRAUMATIC SUPRASCAPULAR ARTERIOVENOUS FISTULA



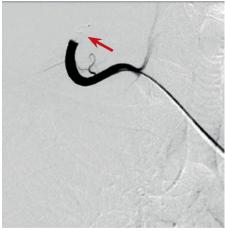
■ Figure 2.3D Volumetric reformation of chest CT. AVF in right suprascapular region

DESCRIPTION OF THE TECHNIQUE

Diagnostic arteriography was performed, visualising a large AVF in the suprascapular and right supraclavicular region, dependent on the costocervical artery, branch of the subclavian artery (Fig. 3) and with development of high-flow collaterals from the thyro-cervical trunk. We selectively catheterised the costocervical artery and an 8 mm diameter AVP 4 (Abbott) was implanted (Fig. 4).



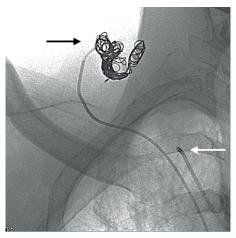
■ Figure 3. Arteriography of supra-aortic trunks. AVF dependent of right costocervical artery and high flow collateral branches of the thyrocervical trunk.

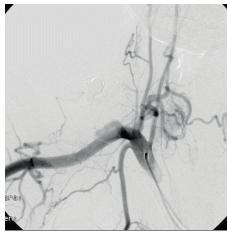


■ Figure 4. Right costocervical artery angiography. Branch occlusion with an 8 mm AVP 4 (Arrow).

TRAUMATIC SUPRASCAPULAR ARTERIOVENOUS FISTULA

We found that the fistula closure had been incomplete, so we decided to access its venous origin (by femoral venous access) and embolise it using fibered microcoils [Interlock® Boston Scientific™] (Fig. 5). Finally, the outcome was satisfactory, with complete closure of the AVF (Fig. 6).





■ Figure 5. Access to the venous end of the AVF and occlusion with fibered coils (black arrow). Right subclavian artery control catheter (white arrow).

■ Figure 6. Control arteriography, checking complete closure of the AVF.

COMMENTS

AVFs of traumatic origin are usually caused by penetrating wounds, and are most commonly found in the extremities. Their effects are both local and systemic: the local include the ischaemic effect and mass effect; Central or systemic effects include increased cardiac output and if persist heart failure can happen.

Traumatic injuries with high-flow arteriovenous fistula are serious and difficult to manage surgically. Endovascular treatment has offered new alternatives, and many materials and techniques are used. The choice will depend of the type of AVF, its location and the possibility of accessing it. In our case, we performed combined treatment to embolise the arteriovenous fistula with the use of fibered coils and vascular occluders and two access, venous and arterial.

TRAUMATIC SUPRASCAPULAR ARTERIOVENOUS FISTULA

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CHAPTER 2 Lung

AVP II CLOSURE OF PERSISTENT ANOMALOUS VERTICAL VEIN BRIDGING THE LEFT SUBCLAVIAN VEIN AND LEFT ATRIUM

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MEDICAL HISTORY

61-year-old male patient with a good general condition but with a significant cardiovascular history. Surgery for aortic coarctation in 1974 when the patient was 19 years old. Bicuspid aortic valve. TEVAR, after carotid-carotid bypass with intentional occlusion of the origin of the LSA, for an 83 mm aneurysm of the descending thoracic aorta 3 years earlier. One year ago, due to frank growth of the thoracic aneurysm, he was treated by our department for a type Ib endoleak by injection of EVOH into the aneurysm sac with a good outcome and current evidence of resolution of the endoleak with reduction of the aneurysm sac in the last CT control. Ascending aorta aneurysm of 53 mm pending of surgical repair (including aortic bicuspid valve repair). The patient also suffered a traffic accident at the age of 42 years with multiple injuries and burns in 40% of his body from which he survived after several months of hospitalisation and rehabilitation. Current smoker of 10 cigarettes/d and alcohol intake of approximately 60 mg/d. The patient is an engineer, with a high sociocultural level and he already warned all of his medical specialists that, after having survived so many things, the ten cigarettes and the daily glass of wine are not negotiable. NO COPD, no stroke, no claudication, echocardiogram with normal ejection fraction, preserved renal function.

One year ago, in an MDCT-angiography during the workup prior to treatment of TEVAR endoleak an asymptomatic right-to-left shunt, not previously known, was discovered. The patient has a vertical vein that connects the left brachiocephalic vein to the left upper pulmonary vein (Fig. 1).

AVP II CLOSURE OF PERSISTENT ANOMALOUS VERTICAL VEIN BRIDGING THE LEFT SUBCLAVIAN VEIN AND LEFT ATRIUM

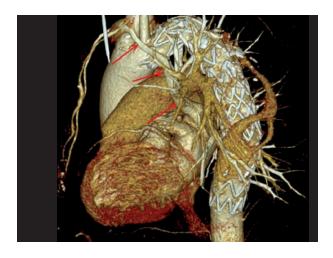
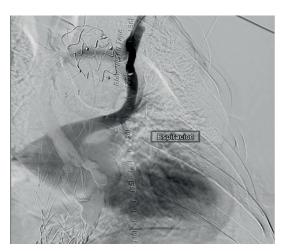


Figure 1. Pre-treatment MDCT. Volume rendering reformation. Abnormal vascular structure originating in the left brachiocephalic trunk is seen, draining into the left atrium: Vertical vein (arrows)

After reviewing the clinical case in the multidisciplinary committee with cardiology and cardiovascular surgery, it was decided that prior to ascending aortic and bicuspid valve surgical replacement, it would be advisable to close this shunt. Given the patient comorbidities, endovascular treatment was chosen over surgical ligation.

DESCRIPTION OF THE TECHNIQUE

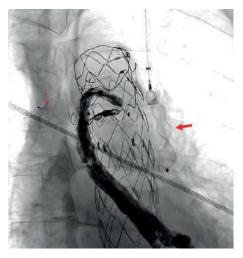
Diagnostic phlebography was obtained 10 days before. From left basilic approach introducing a 4-F Berestein catheter to obtaining a highly precise vascular map of the entire path, speed and direction of flow of the vertical vein over which we will plan the proposed embolisation (Fig. 2).



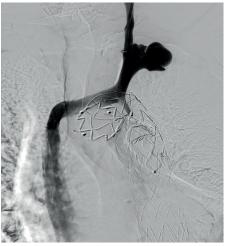
■ Figure 2. Selective venography by approach from the left basilic vein. The presence of a vascular structure consistent with the Vertical vein that communicates the left brachiocephalic vein with the left superior pulmonary vein and causes a R-L shunt is confirmed.

AVP II CLOSURE OF PERSISTENT ANOMALOUS VERTICAL VEIN BRIDGING THE LEFT SURCLAVIAN VEIN AND LEFT ATRIUM

Outpatient procedure. Conscious sedation with 2 cc of midazolam and basic monitoring. PO2 97% at the beginning of the procedure. Antibiotic prophylaxis with 2 g i/v of cefazolin. Systemic anticoagulation with heparin iv bolus (5000 IU, patient weight is 71 kg). Ultrasound-quided puncture of the left jugular vein (carotid-carotid bypass carrier) with a micropuncture system. The vertical vein is easily catheterised with the 0.035" hydrophilic guide. A 6-F 45 cm straight sheath (Flexor®, Cook) was placed in the Vertical Vein. During all these manoeuvres we washed and purged the material very carefully, avoiding any bubbles or small clots that could be detached and end up in the coronary or cerebral flow. Based on the CTA and venography findings, an AVP II measuring 16 mm in size (50% oversize) was chosen. Prior extensive purging by immersing the vascular occluder under saline solution to avoid any air bubbles inside and, once this has been verified, the AVP was advanced through the sheath. Aided by the road map and by pullback of the introducer the AVP II was expanded and centred along the entire vertical vein (Fig. 3). Release and control venography that evidenced good outcome and absence of complications (Fig. 4). The patient was discharged two hours later. One moth later, open surgery was performed with replacement of the AO valve and ascending AO. After 1,5 years of follow up the patient is asymptomatic and is doing well.



■ Figure 3. Closure of the vertical vein by placing a 16 mm AVP II vascular plug (arrow). An AVP IV placed 5 months earlier at the origin of the right bronchial artery for closure of a re-entry during treatment of an endoleak (small arrow). TEVAR, coils in origin of the left bronchial artery and EVOH are also seen as part of previous treatments.



■ Figure 4. Final control venography. Complete vertical vein occlusion and resolution of R-L Shunt are demonstrated.

COMMENTS

The presence of a vertical vein is a congenital malformation usually seen within the context of a total or partial abnormal pulmonary venous connection (PAPVC, or TAPVC). The abnormal pulmonary venous connection (APVC) in a congenital disease characterised by the pulmonary veins not leading to the left atrium as normally occurs, but to the right atrium. This situation usually requires surgery in the neonatal period. The vertical vein is also called levoatriocardinal vein. PAPVR occurs in $\approx 0.5\%$ of the general population. In asymptomatic patients, the condition remains undiagnosed until adulthood in most cases. The left upper pulmonary vein is the most affected vein, accounting for 47% to 79% of the PAPVC cases. The persistent vertical vein should not be confused with a persistent left superior vena cava. Most cases of levoatriocardinal vein have left atrial output failure including multiple levels of obstruction to left-sided outflow, such as mitral atresia, aortic atresia and coarctation. Our patient had been operated on for an Aortic coarctation in 1974. In case of APVC, the vertical vein usually drains blood in a cephalad direction from the left upper lobe to the left innominate vein resulting in a left-to-right shunt. In this patient the direction of the flow was from the innominate vein to left atrium through the upper left pulmonary vein. Although asymptomatic, due to the right-to-left shunt, potential complications such as brain infarction and arterial embolism could happen. The vertical vein was considered to be closed before the planned major cardiac surgery.

With the development of structural cardiology, there are increasing numbers of cases of asymptomatic or oligosymptomatic vertical veins treated endovascularly. The Amplatzer device is the first choice for this condition, since it allows for closing large vessels. As in PAVM, we must be very careful in this condition, avoiding when manipulating that any bubbles or debris are released into the systemic circulation with the consequent risk of stroke, MI or embolism. Before advancing the device into the introducer, the AVP should be removed from its sheath and inserted under saline solution until it is certain that all the air that could have been retained by the nitinol mesh has disappeared.

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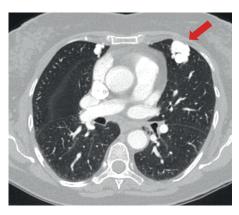
PULMONARY AVM EMBOLISATION

M. González, M. Echenagusía, G. Rodríguez, A. Álvarez and F. Camúñez

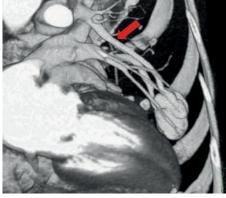
Gregorio Marañón University Hospital, Madrid.

MEDICAL HISTORY

49-year-old patient with Osler-Weber-Rendu syndrome and history of chronic and progressive respiratory failure is presented. Physical and laboratory examinations were normal but basal O2 blood saturation was 85%. Chest CT scan showed two hypervascular nodules, one in the right upper lobe and a larger one in the left lower lobe (Figures 1 and 2). Treatment of the largest lesion by embolisation was decided.



■ Figure 1. Chest CT: Two PAVMs are shown: one in the anterior segment of the right upper lobe and another of larger size in the left lower lobe (arrow).



■ Figure 2. Volume rendering reformation centred on the lingula showing a single feeding artery (arrow).

DESCRIPTION OF THE TECHNIQUE

A selective canalisation of the left lower lobar pulmonary artery was performed from a right femoral vein approach using a 6-F 100 cm Emboy® guide catheter (Cordis) (Figure 3 A). A 10 mm AVP I (Abbott) was then implanted. The AVP I was 40% oversizeed to the embolised vessel (Figure 3 B). Final control arteriography several minutes later demonstrated complete occlusion of the PAVM.

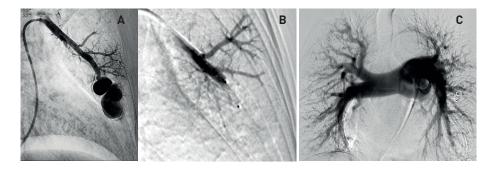


Figure 3. A) Selective left inferior lobar artery arteriography. PAVM is shown. B) Control arteriography immediately after plug implantation, noting absence of filling of the PAVM. C) Final control through the pigtail catheter in the main pulmonary artery confirming complete embolisation of the PAVM.

COMMENTS

Embolisation of PAV fistulas and malformations should be as selective as possible to prevent occlusion of healthy pulmonary arterial branches. In this case, treated in 2006, only the Amplatzer type AVP I was available. A single device of 10 mm in diameter and 7 mm in length was used, which was sufficient for complete occlusion of the arteriovenous malformation. Other Amplatzer Vascular Plugs designed for medium- and high-flow vessels (AVP II and AVP 4, Abbott) may also be used. The three types of currently available vascular plugs provide effective, rapid and safe embolisation, adapted to the calibre of the artery and its anatomical characteristics.

The use of a long vascular sheath or guide catheter allows for angiographic controls and facilitates the procedure. Extreme care must be taken to avoid bubbles or debris escaping through the fistula into the brain or coronary arteries.

PULMONARY AVM EMBOLISATION

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COMBINATION OF EMBOLIC AGENTS FOR THE CONTROL OF MASSIVE HEMOPTYSIS

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MEDICAL HISTORY

78-year-old male admitted to the emergency room for massive hemoptysis lasting two hours. Given the severity of the condition, the vascular radiologist on call was required for emergency embolisation.

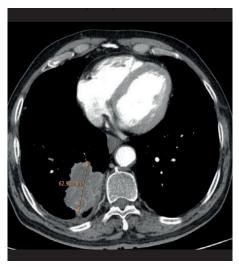
Patient history included HBP, dyslipidemia, ex-smoker, and ischaemic heart disease with stent in the right coronary artery seven years ago. Moderate COPD and anticoagulation with acenocoumarol for atrial fibrillation. He had been admitted three weeks before to the Pneumology Department for mild but repeated episodes of hemoptysis, constitutional syndrome, and weight loss with the recent diagnosis of moderately differentiated squamous cell carcinoma in the lower lobe of the right lung T2N1M0 (Fig. 1). He had been discharged stable and asymptomatic to complete the extension study on an outpatient basis.

DESCRIPTION OF THE TECHNIQUE

Despite the long coagulation times of the patient (INR 1.72), given the severity of the situation, it was decided to start the procedure without waiting to reverse the effect of acenocoumarol. By approaching from the right CFA, using a Michaelson 5-F catheter (Angiodynamics), selective catheterisation of the right bronchial artery was performed, which was angiographically normal with a calibre less than 2 mm and no bleeding findings (Fig. 2). Normal intercostal arteries were also screened selectively. Due to the location of the tumour, examination of the internal mammary artery or lateral thoracic artery was not considered necessary.

Based on the negative findings of the bronchial artery, pulmonary arteriography was considered necessary. By approach from the common femoral vein, the right lower lobe pulmonary artery was selectively catheterised, identifying an amputated posterobasal segmental branch (Fig. 3). Once selectively channelled, it showed clear bleeding with active contrast extravasation towards the airway (Fig. 4).

COMBINATION OF EMBOLIC AGENTS FOR THE CONTROL OF MASSIVE HEMOPTYSIS



■ Figure 1. Contrast-enhanced CT. 6 cm mass in the lower lobe of the right lung.

■ Figure 2. Normal right bronchial artery with no evidence of bleeding.



■ Figure 3. Right pulmonary arteriography.

Amputated posterobasal segmental branch, cut off sign (arrow).

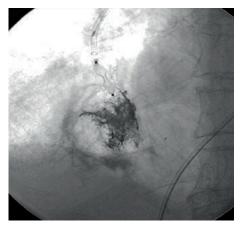


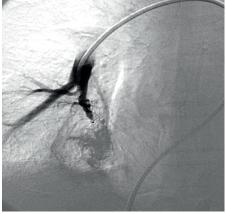
■ Figure 4. Selective arteriography. Active bleeding with contrast extravasation into the airway (arrows).

A 6-F and 90 cm sheath (Destination®, Terumo) was placed at the origin of the amputated artery in order to be able to perform angiographic controls while embolisation was performed. We decided to perform embolisation combining two embolisation materials. The interstitium of the tumour was first embolised using a 0.021" microcatheter

COMBINATION OF EMBOLIC AGENTS FOR THE CONTROL OF MASSIVE HEMOPTYSIS

(Rebar18® Medtronic) and a liquid embolic agent (Onyx®18, Medtronic). Then, through a 4-F multipurpose catheter (Tempo Aqua Cordis®), the amputated arterial branch was sealed with an 8 mm AVP 4 occluder (AbbottTM) (Fig. 5). Control angiography showed a good immediate outcome (Fig. 6) that was confirmed with a good clinical course. The patient could be discharged at 72 hours and remained free of further episodes of hemoptysis until death, due to disease progression, three months later.





■ Figure 5. Post-embolisation image. Tumour-filled EVOH and AVP 4 occluding bleeding artery.

■ Figure 6. Final control angiography with cessation of bleeding.

COMMENTS

Bleeding from a pulmonary artery is not common in patients undergoing embolisation for hemoptysis. However, upon selective angiography of the bronchial arteries of both hemithorax, the intercostal arteries (particularly in pathological areas), the internal mammary arteries and the lateral thoracic arteries, and finding them all to be negative; pulmonary angiography is then indicated.

We report a case of life-threatening massive hemoptysis caused by direct communication between a pulmonary arterial branch and the airway in a patient with bronchogenic carcinoma. This is often fatal due to lack of time for effective treatment. In our case we chose to seal the intratumoural vascularisation a fluid embolisation whose efficacy is not affected by the decoagulation experienced by the patient, which acts re-filling the interstitium of the tumour and which in our experience is effective for achieving haemostasis in tumour tissues with active bleeding. However, in this case it was also very important to be able to seal the bleeding artery eroded by the tumour. Because of its characteristics, amplatzer vascular occluders are particularly useful in sealing high-flow vessels. Its controlled release mechanism also allows for performing the procedure with great precision and safety.

COMBINATION OF EMBOLIC AGENTS FOR THE CONTROL OF MASSIVE HEMOPTYSIS

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ENDOVASCULAR TREATMENT OF ADULT INTRALOBAR PULMONARY SEQUESTRATION IN A HIGH-RISK SURGICAL PATIENT

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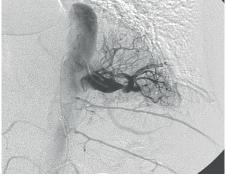
MEDICAL HISTORY

55-year-old male, smoker of two packs a day, with a history of HBP and DL. Ischaemic heart disease treated with PTCA + stent. COPD with moderate flow obstruction. Lung lobectomy of the right upper lobe six weeks before due to giant bulla that compromised the middle lobe. Intralobar sequestration in the left lower lobe was diagnosed pre-operatively. Considering the history and recent surgery in the contralateral lung, surgical treatment of sequestration was ruled out, and it was decided in agreement with the Department of Thoracic Surgery to treat it by embolisation.

Contrast-enhanced CT confirmed the diagnosis and showed the existence of systemic arterial irrigation for the inferomedial portion of the left lung (Fig. 1). Diagnostic arteriography was performed to complete the study and assess treatment options (Fig. 2).



■ Figure 1. Chest contrast-enhanced CT. Large calibre aberrant artery originating in the left aspect of the distal descending thoracic AO.



■ Figure 2. Selective arteriography. Aberrant artery, direct AO branch, irrigating left lower lobe pulmonary parenchyma.

DESCRIPTION OF THE TECHNIQUE

Diagnostic arteriography a few days before embolisation confirmed the diagnosis of intralobar sequestration with venous return to pulmonary veins. The branches of the celiac axis and phrenic arteries were studied and did not show other systemic supplies to sequestration. The nutrient artery of the sequestration was thick (10-11 mm), with a short length and a wide ostium as compared to thoracic AO diameter. Based on these findings, a 14 mm AVP II controlled-release nitinol occluder (Amplatzer, Abbott) was chosen as the ideal and safe device for embolisation. In order to ensure that the occluder is released stable and not receding or protruding into the thoracic AO, a 55-cm curved 8-F sheath (Flexor® - Cook®) needed to be advanced in the secondary branches of the sequestration. This manoeuvre was performed by sliding the introducer coaxially over a Simmons I catheter and a previously positioned amplatz super stiff guidewire (Fig. 3 and 4).



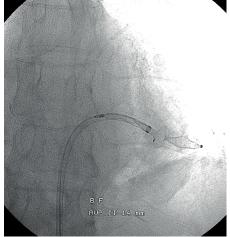


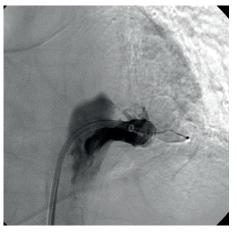
Figure 3. Advance of curved 8-F 55 cm sheath.

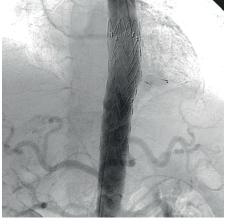
■ Figure 4. Stable and duly advanced sheath within the vessel. Release of AVP II avoiding its roll-back to the AO.

The outcome was satisfactory with cessation of arterial supply to the lung parenchyma (Fig. 5) without the occluder protruding into the aortic lumen. In order to definitively seal the residual arterial stump and prevent its potential growth or rupture, it was decided to insert percutaneouly a short thoracic stentgraft. In the same procedure, after preinstallation of a double percutaneous suture system (Prostar® Abbott Vascular), a 32 mm calibre, 10 cm long, thoracic endograft (Valiant-Captiva® Medtronic) was released and well centred on the residual arterial "diverticulum" (Fig. 6).

Four years afterwards, the patient is asymptomatic, under follow-up by Cardiology and has been discharged by Thoracic Surgery and Pnemology.

ENDOVASCULAR TREATMENT OF ADULT INTRALOBAR PULMONARY SEQUESTRATION IN A HIGH-RISK SURGICAL PATIENT





■ Figure 5. Immediate angiographic control. No lung parenchyma filling.

■ Figure 6. Final control aortogram. AVP II and stent sealing the nutrient artery of the sequestration.

COMMENTS

A pulmonary sequestration is a congenital anomaly in which a portion of the lung is separated from the rest of the lung parenchyma. It is characterised by receiving blood supply from a systemic artery. It is more common in men and is most commonly located in the lower lobe of the left lung (60-90%). Intralobar sequestration is the most common, performs venous drainage into the pulmonary veins, and is enveloped by the same visceral pleura as the rest of the lung. Blood supply may be from one or more systemic arteries originating in the thoracic (74%) and/or abdominal (21%) aorta. In 5% of the cases, the feeding vessels originate in the internal mammary, subclavian, intercostal, inferior pharyngeal, left gastric, renal and coronary arteries. The most common clinical manifestation of lung sequestration is recurrent pneumonia and hemoptysis. Surgery is elective.

Intralobar sequestration embolisation is a therapeutic alternative in adult and pediatric high-risk surgical patients or as a prior step to facilitate surgery. By occluding the abnormal blood supply, the sequestration, which has a normal bronchial tree, will be irrigated from branches of the bronchial arteries and may also be able to provide flow from the pulmonary arteries of the adjacent lung tissue. This explains why no infarction or necrosis occurred in the embolised lung parenchyma. In addition, 15% of intralobar sequestrations share pulmonary and systemic vascularisation.

ENDOVASCULAR TREATMENT OF ADULT INTRALOBAR PULMONARY SEQUESTRATION IN A HIGH-RISK SURGICAL PATIENT

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CHAPTER 3 **Liver/GI Tract**

PREOPERATIVE PORTAL VEIN EMBOLISATION

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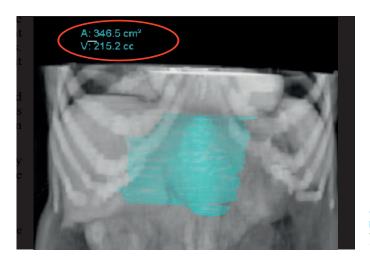
MEDICAL HISTORY

46-year-old patient with a history of three direct relatives diagnosed with colorectal carcinoma. This patient was admitted to the emergency room with two weeks symptoms of nausea, vomiting and postprandial hypogastric pain followed by diarrhoea. Lack of appetite with weight loss of 10 kilograms during the past few weeks.

Abdominal CT: tumor in the ascending hepatic flexure of the colon, perforated, with an adjacent multiloculated collection consistent with abscess. Mesenteric fat infiltration. Hypodense nodules in right liver lobe consistent with metastases.

Liver biopsy (metastasis of moderately differentiated adenocarcinoma). Right hemicolectomy was performed. Pathology diagnosis of stage IV colon adenocarcinoma.

Right hepatectomy was planned, however volumetric of the future liver remnant (FLR) was insufficient (Fig.1), so a preoperative portal vein embolisation (PVE) was requested.



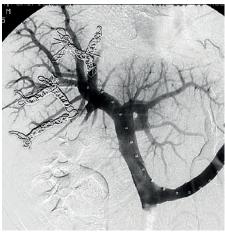
■ Figure 1. Previous liver volumetry showing FLR to be insufficient.

DESCRIPTION OF THE TECHNIQUE

Puncture in the right axillary midline was made to transhepatic gain access to the right portal branch. Reaching the splenic vein to perform splenoportography, which confirmed the permeability of the splenoportal axis and of the intrahepatic portal vascularisation. The presence of two right portal branches close to the portal bifurcation was found (Fig. 2). These proximal portal branches were selectively catheterised using a Simmons I catheter, and they were embolised by perfusion of PVA microspheres followed by implantation of fibered coils (Fig. 3).



■ Figure 2. Splenoportography showing patency of the splenoportal axis and of the intrahepatic portal vascularisation. Two right portal branches near bifurcation (arrows).



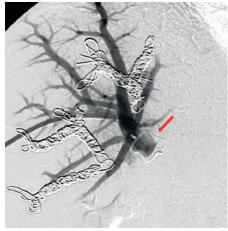
■ Figure 3. Embolisation of the two proximal right portal branches using PVA microspheres + fibered microcoils.

PVA microspheres were then infused from the main trunk of the right portal branch, followed by implantation of a 16 mm diameter AVP (Abbott) (Fig. 4), confirming the adequate embolisation obtained by performing the post-procedure venography from the introducer, showing retention of the contrast medium due to lack of washing (Fig. 5).

Good clinical outcome with marked hypertrophy of the LHL in the volumetry performed four weeks later (Fig. 6).

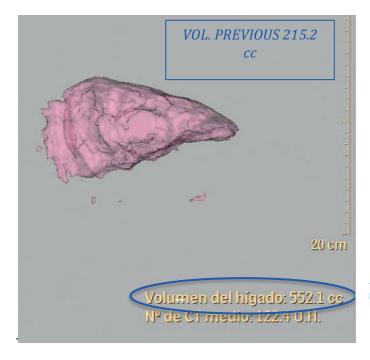
PREOPERATIVE PORTAL VEIN EMBOLISATION





■ Figure 4. Embolisation of the main right portal branch, after infusion of PVA microspheres, with a 16 mm diameter AVP.

■ Figure 5. Post-procedural control phlebography from the introducer, observing a retention of the contrast medium due to lack of washing. AVP in the main right portal vein (arrow).



■ Figure 6. Volumetry four weeks after PVE. Marked LHL liver hypertrophy (215.2 cc vs. 552.1 cc).

COMMENTS

PPVE consists of embolisation of the portal vascularisation of the hepatic lobe affected by metastasic lesions, to be able to perform hepatectomy of this lobe four weeks later, when the contralateral lobe has become hypertrophic.

It is indicated in patients who are candidates for liver lobectomy in whom an insufficient future remnant liver is expected. This is calculated by dividing this future remnant liver by total liver volume, minus the tumour volume:

(Future remnant liver volume) X 100%
(Total liver volume-Tumour volume)

The FLR is considered insufficient when is less than 20% in healthy livers, 30% in livers after chemotherapy or 40% in cirrhotic livers.

PVE can be performed from ipsilateral or contralateral approach. Although catheterisation of the portal branches is easier when accessed from the contralateral lobe, many authors advocate avoid puncturing it to prevent complications in the FI R

Different embolic materials have been used for pre-operative PVE, including coils, PVA microspheres, liquid embolics and nitinol vascular plugs. We report a case in which, after accessing through the RHL, two proximal right portal branches were embolised with coils to subsequently, after infusion of PVA microspheres, occlude the right main portal branch with AVP. AVP I is very useful when, as in this case, the landing zone for the plug is short.

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RADIOEMBOLISATION

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MEDICAL HISTORY

Three case reports are briefly discussed.

CASE 1

49-year-old male with HCV presenting a 10 cm HCC mass in the right liver lobe. He had tumoral thrombosis of the main portal vein and two smaller nodules in the left liver lobe. Y90 treatment was programmed. He had accessory tumoral feeding arteries arising from left liver artery. Left liver artery arises just in front of GDA and the risk of Y90 reflux into GDA was high. GDA was embolised using a 6 mm AVP 4 (Figs. 1 and 2).



■ Figure 1. Selective celiac axis angiography. Large mass in the right lobe of the liver. Since part of the treatment had to be performed from the left hepatic artery (arrow) and there was no safety margin in the event of Y90 reflux, GDA was occluded.



■ Figure 2. Pre-radioembolisation GDA occlusion with a 6 mm AVP 4 plug (arrow). A right gastric artery stump is seen and should also be embolised.

RADIOEMBOLISATION

CASE 2

76-year-old male with 4 cm HCC located in segment IV. Tumor feeding arteries arised from the right and middle hepatic artery. The left hepatic artery was branch of left gastric artery. Tumoral portal vein thrombosis was also present (Figs. 3 and 4).

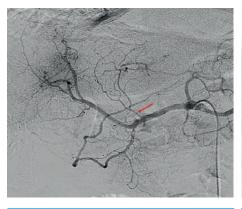




Figure 3. Celiac axis angiography. In this case, treatment will be performed from the middle (arrow) and right hepatic arteries. GDA is found to be between both with high risk of reflux.

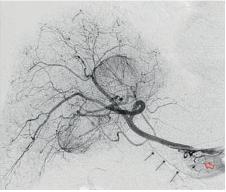
■ Figure 4. Arteriography from celiac axis. 6 mm AVP 4 plug GDA occlusion was verified. 50% plug oversize (arrow). Supraduodenal and right gastric artery had been embolised using microcoils.

CASE 3

Tumor progression in a 66-year-old HBV male with four HCC nodules in the right liver lobe previously treated by chemoembolisation (Figs. 5 and 6).



Figure 5. Celiac axis angiography. In this case, right lobar radioembolisation will be performed. The right hepatic artery is long and reflux into GDA is unlikely to happen, despite that GDA embolisation was decided.



■ Figure 6. Selective right hepatic arteriography. This ttempts to illustrate what may occur after GDA occlusion. Fine secondary tiny branches feeding the duodenal area appear and should require embolisation (arrow head). Right gastric artery is now visible (blacks arrows).

DESCRIPTION OF THE TECHNIQUE

In these three cases, during the pre-radioembolisation work-up, it was estimated that there was a potential risk of Y90 microspheres reflux into the GDA, so it was decided to embolise it.

AVP 4 (Abbott) can be deployment through a regular 4-F or 5-F diagnostic catheter, the same used for hepatic angiographic study, adding simplicity and speed to the procedure. The possibility of controlled release also adds safety and comfort to the patient and to the operator.

AVP II may also be used if a 5-6-F guide catheter is preferred for the angiographic study; the presumed advantage of this type of vascular plug subtype is that its cylindrical morphology once achieves a greater contact area with arterial circumference and could prevent persistence of some proximal collateral branches that sometimes arise once the device is implanted and complicate the procedure. Effectiveness in occlusion is high and the recanalisation rate is minimal. The AmplatzerTM device family , as in other territories, have to be oversized adequately in each case, while a diameter of 6 mm should be the most widely used according to our experience in the GDA.

COMMENTS

Radioembolisation is a therapeutic treatment indicated in cases of intermediate-advanced HCC that are poor candidates for chemoembolisation (absence of hepatic portal irrigation, bulky masses or large number of nodules). RE is a part of the therapeutic regimen in metastatic liver disease in combination with chemotherapy (neuroendocrine tumours, melanoma, CCLM among others). The therapeutic agent is yttrium-90 isotope incorporated to 30-60 microns microspheres.

The angiographic work-up of a patient involves embolisation of the possible extrahepatic branches at risk for reflux of microspheres, either because they are located in the territory to be embolised (often the right gastric artery), or because they are too close to the infusion site (< 1-2 cm) (Figs. 1 to 4). The caliber and tortuosity in the access to some of these arteries often require a microcatheter and microcoils. However, vascular plugs are an advisable alternative for occlusion of larger vessels like GDA. It should not be overlooked that occlusion of this artery may provoke the angiographic visualization of other tiny branches like right gastric or surpaduodenal arteries. Therefore, regardless of the embolisation material used, the convenience of occlusion should always be assessed by estimating the risk-benefit ratio in each particular case (case 3: Figs. 5 and 6).

RADIOEMBOLISATION

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SUCCESSFUL CLOSURE WITH AVP 4 OF AN INTRAHEPATIC ARTERIOPORTAL FISTULA THAT PREVENTED Y-90 TREATMENT

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MEDICAL HISTORY

68-year-old male with compensated alcoholic liver cirrhosis, diagnosed three years before with hepatocellular carcinoma of 7 cm in stage B according to the BCLC classification. A radiofrequency session combined with TACE was initially performed and completed with a second TACE session alone five weeks later. The patient did well, and a complete radiological response was obtained.

Twenty-two months later, local and distant recurrence occurred, so three new chemoembolisation sessions were performed in a four-month period despite which tumour persisted. The patient was asymptomatic with Child A-5 liver function. It was then decided to perform radioembolisation with yttrium 90, but when the workup study was performed, an intrahepatic arterioportal fistula in the tumor area and a 20.3% hepatopulmonary shunt that contraindicate radioembolisation treatment was founded (Figs. 1 and 2). It was decided to attempt fistula closure and re-evaluate the possibility of radioembolisation.

SUCCESSFUL CLOSURE WITH AVP 4 OF AN INTRAHEPATIC ARTERIOPORTAL FISTULA THAT PREVENTED Y-90 TREATMENT





■ Figure 1. Selective arteriography of the right hepatic artery. Simultaneous enhancement of the right portal vein.

■ Figure 2. Selective arteriography of the right hepatic artery. Fistula details (arrow).

DESCRIPTION OF THE TECHNIQUE

A few days later, with the tip of the catheter located in different sites along the right hepatic artery, selective studies were performed in oblique views to find and confirm that the fistulous tract was unique. Then with a 4-F Cobra catheter (Aqua Tempo® Cordis) and aided by a 0.035" hydrophilic guide, it could be easily passed through the fistula tract from the right segmental hepatic artery into the right portal vein. It was necessary to close the fistula but to keep the arterial segment distal to the fistula patent. An Amplatzer AVP 4 device (Abbott) was chosen. A 4 mm occluder was advanced over the Cobra catheter located through the fistulous tract. After many attempts, this occluder did not remain stable over the fistulous tract, so it was necessary to remove it. Then it was chosen a bigger one, 8 mm, which remained stable across the fistula (Figs. 3 and 4).

The control arteriography confirmed complete fistula closure (Fig. 5). The Tc-99 gammagraphy simulation study was conducted again, and, in this case, the hepatopulmonary shunt had decreased to 7.2%. Radioembolisation therapy with yttrium-90 one week later could therefore be performed without problems.

The patient outcome was favourable, and in the controls with CT the fistula persisted occluded (Fig. 6). The patient survived 16 months after radioembolisation.

SUCCESSFUL CLOSURE WITH AVP 4 OF AN INTRAHEPATIC ARTERIOPORTAL FISTULA THAT PREVENTED Y-90 TREATMENT



HUP IV 8 mm

■ Figure 3. 8 mm AVP 4 before release (arrow). Microcoil previously placed along cystic artery is also shown.

■ Figure 4. AVP 4 of 8 mm, released and stable within fistulous tract.



■ Figure 5. Control hepatic arteriography. No fistula. AVP 4 (arrow) without occluding arterial branch.



■ Figure 6. Pre and post arterial phase enhanced CT. Strong portal enhancement (red arrow) simultaneous to aortic enhancement. CT control nine months later, no portal enhancement in the arterial phase. AVP 4 without artifact creation.

COMMENTS

Hepatic radioembolisation with yttrium-90 microspheres is an accepted treatment for intermediate-stage HCC in patients who do not respond to TACE. In the case reported, this treatment was not possible initially because, due to intrahepatic arterioportal fistula, yttrium-90 would not selectively reach the tumour, and also the dose potentially deposited in the pulmonary parenchyma exceeded the accepted limits. It was therefore necessary to previously close the fistula. The simplest technique would have been to occlude, using coils or plugs, the segmental hepatic artery causing the problem. However, this was not advisable because it would have also prevented the yttrium-90 microspheres from subsequently reaching the tumour tissue, located distal to the fistula and supplied by the segmental hepatic artery.

The AVP 4 vascular plug, because of its compatibility with 4-F catheters and its design, appeared to be appropriate for this particular case. Leaving a lobe of the occluder in each end of the fistulous tract was a good option both for closing the fistula and for keeping the distal portion of the artery patent. The controlled release system of these devices was essential, allowing controls to be performed until the proper positioning was achieved. Although the fistulous tract measured only a few millimetres, it finally had to be oversized and an 8 mm occluder released, since the smaller tracts did not remain stable and migrated to the portal lumen. This oversize involved a risk of also occluding the arterial end, which fortunately did not occur. The absence of streak artifact in CT controls is a significant advantage to be considered in cancer patients who require multiple follow-up CTs to assess liver response.

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TIPS FLOW REDUCTION

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MEDICAL HISTORY

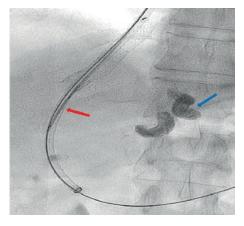
59-year-old male with chronic liver disease and HIV and HCV co-infection. Former intravenous drug addict. Recurrent episodes of variceal UGIB refractory to medical and endoscopic treatment, so 4 months earlier a TIPS (Viator® 10 mm x 7 cm) and embolisation of large G-E varices using cyanoacrylate had been performed at another site. Since then, no further episodes of GI bleeding. The patient was admitted to our hospital for a third episode of hepatic encephalopathy since the creation of the TIPS. Child C10 on admission. Ammonia at admission was 73 mmol/l. A brain CT scan was performed, which was normal, and after ruling out other possible causes of encephalopathy, it was decided to attempt endovascular treatment to reduce flow through the TIPS without completely occluding it.

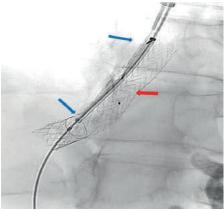
DESCRIPTION OF THE TECHNIQUE

General anaesthesia was performed using a laryngeal mask. Antibiotic prophylaxis with 2 g iv cefazolin and approach from the right internal jugular vein. Trans-TIPS canalisation of the splenic vein and direct splenoportography confirming hepatopetal flow with permeability of the portal vein and TIPS with no evidence of restenosis. Absence of G-E varices or other type of spontaneous portosystemic shunt. Measurement of direct portal pressure was 17 mmHg before TIPS flow reduction procedure.

A 12-F and 45 cm hydrophilic sheath (Flexor® Cook) was placed through the TIPS. An 80 cm MPR 4-F catheter (Tempo®, Cordis) was advanced. Through this 4-F catheter a 6 mm AVP 4 (Abbott) nitinol vascular occluder was positioned in the center of the TIPS chanel (Fig. 1). In parallel and also through the 12-F sheath, a 10 mm x 4 cm bared self-expanding nitinol stent (Absolute®, Abbott Vascular) was advanced, centring it from the AVP 4 occluder, and all within the 10 mm Viator prosthesis placed 4 months earlier. The AVP 4 occluder is first deployed within the TIPS by "pull-back" of the 4-F catheter and the 12-F sheath, but it is not yet released from its delivery guidewire. We confirm that the occluder had been properly expanded inside the Viator lumen (Fig. 2).

TIPS FLOW REDUCTION





■ Figure 1. Right jugular approach. Trans-TIPS Flexor® 12-F sheath. 6 mm vascular occluder of nitinol AVP 4 (red arrow) inside the Flexor. Radiopaque material related to cyanocrylate (blue arrow) due to prior embolisation of coronary-stomal vein.

■ Figure 2. The AVP-4 occluder is first deployed within the TIPS by pull-back of the sheath and the 4-F catheter (red arrow). In parallel, we centred from AVP-4 the self-expanding nitinol stent of 10 mm x 4 cm that was positioned and pending release (blue arrows).

The second manoeuvre was to release the coaxial bare nitinol stent into the Viator, which was well expanded in its proximal and distal ends, but in an hourglass shaped manner, since the AVP 4 prevent complete stent expansion in its central portion, thus causing a partial reduction in the calibre and flow of the TIPS (Fig. 3). At this point we released the plug from its delivery guidewire. A post-reduction control portography of the TIPS showed patency of the main portal with no filling of portosystemic collaterals (Fig. 4).



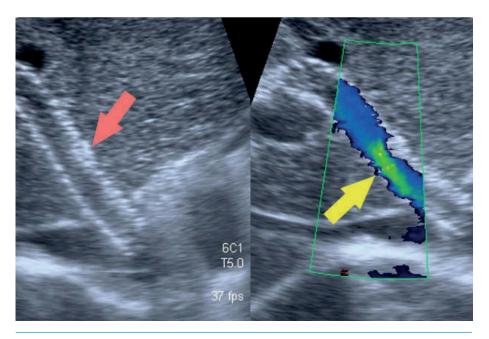
Figure 3. Release of the nitinol stent expanded in the form of an hourglass due to the presence of AVP-4 that prevents complete expansion of its central area, thus causing a partial reduction in the calibre and flow of the TIPS. Nitinol stent end marks [red arrows].



■ Figure 4. Post-reduction control portography of TIPS. Permeability of main portal and TIPS without filling of portosystemic collaterals.

TIPS FLOW REDUCTION

Hemodynamic study was repeated, which now evidenced a portosystemic gradient of 13 mmHg with direct portal pressure, from 17 (pre) to 23 mmHg (post). Thirty-six hours afterwards he was awake, with no clinical signs of encephalopathy, and ammonia blood level had fallen to 31 mmol/L. A control Doppler ultrasound was performed 48 hours after the procedure, confirming the clinical and hemodynamic result. An intraprosthetic stenotic notch caused by the occluder was observed on Doppler US (Fig. 5).



■ Figure 5. Doppler ultrasonography 48 h after treatment: Focal reduction of the internal lumen of the TIPS prosthesis by the AVP-4 vascular occluder (red arrow) with increased intraprosthetic flow rate from 120 cm/s pre-procedure to 150 cm/s post-procedure with appearance of aliasing effect (yellow arrow).

COMMENTS

TIPS is an established treatment for UGIB of variceal origin when standard pharmacological and endoscopic measures are insufficient. The TIPS is indicated for both bleeding control and to prevent rebleeding in patients with severe portal HT. With the current PTFE-coated TIPS-specific stentsgraft, TIPS dysfunction has almost passed into the history and few to very few TIPS become occluded. However, encephalopathy secondary to the creation of a TIPS is still a challenge in the clinical management of these patients and remains an issue pending a definitive solution. Hyperammonemia secondary to spontaneous or acquired portosystemic shunt is known to cause neurotoxicity and is ultimately responsible for encephalopathy. If splanchnic flow does not pass through the liver, ammonia transformation to urea is deficient and problems arise.

In this patient, TIPS was very effective for definitive control of portal hypertension and variceal GIB. However, the shunt created resulted in the development of disabling encephalopathy. This is a complex clinical situation where the balance between bleeding risk and the development of encephalopathy should be sought. In extreme cases, occlusion of the TIPS is necessary. Occluding TIPS using an endovascular procedure is much easier than creating it and is usually performed using an oversized AVP II nitinol plug released within the stent. What is technically more difficult, but undoubtedly is more useful for these patients, is to perform a partial occlusion of the lumen of the TIPS, reducing its flow and thus allowing for a greater portal supply to the liver parenchyma that allows for increased detoxification of systemic ammonia. With partial occlusion, we will maintain control over the risk of bleeding while reducing the risk of encephalopathy. The parallel technique for partial closure of TIPS has been widely described, and in this case is reported one of its possible variants.

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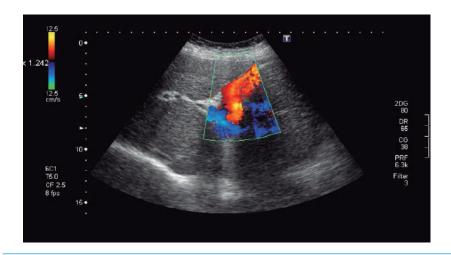
PARAUMBILICAL PORTOSYSTEMIC SHUNT OCCLUSION

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MEDICAL HISTORY

62-year-old woman with HCV well-compensated chronic liver disease (Child-Pugh score A6) is presented. She had portal hypertension with grade I oesophageal varices in primary prophylaxis with beta-blockers. She suffered from recurrent episodes of liver encephalopathy with frequent hospital admissions affecting patient's autonomy. She had developed a large paraumbilical portosystemic shunt (from left portal branch to left common femoral vein) (Figs. 1 and 2). No ascites was present. Episodes of encephalopathy were initially masked by concomitant depression. An encephalogram was performed and the encephalographic pattern was consistent with hepatic encephalopathy. In view of the radiological and clinical findings, it was decided to close the paraumbilical portosystemic shunt to try to cure or reduce the neurological signs of the patient.



■ Figure 1. Doppler ultrasound. 17 mm left portal vein dilatation and umbilical vein recanalization.



■ Figure 2. CT MIP reformation.
Portosystemic shunt from the left portal vein into the left common femoral vein is clearly shown.

DESCRIPTION OF THE TECHNIQUE

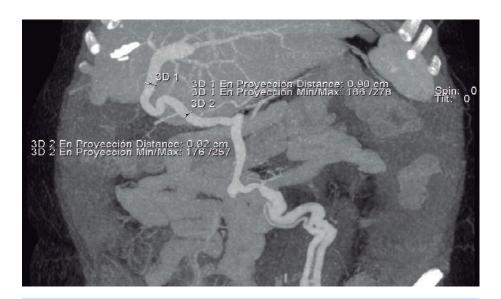
From left common femoral vein approach the paraumbilical vein was canalised. Using a 4-F multipurpose catheter (Aqua Tempo Cordis®) and a hydrophilic guidewire (0.035 Glide, Terumo®) the shunt was canalised up to its origin in the left portal vein. A venography was acquired demonstrating hepatofugal flow (Fig. 3). Despite the tortuosity of the shunt an exchange to a 6-F multipurpose guide catheter (Envoy®, Cordis) could be made. Through the guide catheter a 12 mm AVP II (Abbott) nitinol vascular plug was smoothly implanted.

Unlike to other embolic agents (coils) a vascular plug was chosen for this particular case due to the target vessel features: high flow, large calibre and a straight and long proximal venous segment (landing zone). Amplatzer controlled release also reduced the risk of nontarget portal vein embolisation during the procedure. A 12 mm AVP II nitinol plug was selected since the diameter of the vein in its proximal segment was 9 mm (measurement performed on the CT - Fig. 4), calculating an oversize of 30-35%. Once implanted a progressive flow slowdown was appreciated until complete shunt closure (Fig. 5).

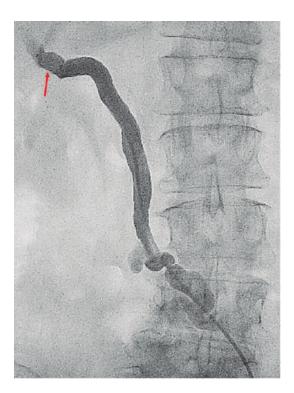
The patient improved and after two years of clinical follow-up, neurological improvement, attention, short-term memory and walking, was demonstrated.



■ Figure 3. Umbilical vein venography. Access was gained from the left common femoral vein with a 4-F multipurpose catheter and a hydrophilic guide, obtaining catheterisation of the venous shunt to its origin: hepatofugal flow was verified. A good correlation to the CT image revealed the tortuosity of the shunt's middle segment.



■ Figure 4. Measurement of the diameter of the umbilical vein (9 mm). An AVP II of 12 mm, of high occlusive capacity, is selected in order to oversize 30-35% of vessel diameter.



■ Figure 5. Umbilical vein venography. Image immediately after plug implantation (arrow). Flow slowdown is shown

COMMENTS

Liver encephalopathy includes a wide range of potentially reversible neuropsy-chiatric symptoms that may be due to acute liver failure, chronic liver impairment, or the presence of portosystemic shunt. Clinical presentation is variable, from minimal or mild to severe, episodic or recurrent encephalopathy, with or without recognisable triggering factors. The main disorders include impact on sleep pattern (insomnia/hypersomnia) and state of consciousness, appearance of extrapyramidal disorders, and intellectual and behavioural impairment. Hyperammonemia, electroencephalographic pattern, and evoked potentials are some of the parameters that may be affected. Liver encephalopathy diagnosis continues to be established based mainly on clinical parameters. It is important to rule out other organic causes leading to a similar condition by performing brain CT/MRI and other metabolic disorders. In the case discussed, the disease was persistent and severe, and was associated with cirrhosis with portal hypertension and the development of a large portosystemic shunt.

Embolisation of portosystemic collaterals may be considered in an attempt to reduce or correct this clinical condition that may be achieved in more than half of the cases. Indication for portosystemic shunt occlusion are a significant single or dominant shunt, good liver function (MELD <11) and no gastro-oesophageal varices at risk of bleeding.

From a technical point of view, the approach used to treat these shunts is transhepatic versus femoral that has less morbidity and is less invasive. The key point is to advance a guide catheter to the landing zone that will provide good navigability for the AVP II plug despite the tortuosity of the central segment of this shunt.

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CONGENITAL INTRAHEPATIC PORTOSYSTEMIC SHUNT

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MEDICAL HISTORY

Patient with Down's syndrome and a congenital intrahepatic portosystemic shunt discovered as an incidental finding on a postnatal US examination is presented. A fistula between right portal vein and the inferior vena cava (Park Type I) was found. Interatrial communication and a left superior vena cava were also observed.

Two years later, a new US examination and liver MRI revealed a mild enlargement of the portosystemic shunt, without laboratory abnormalities. Imaging showed that there was a small direct connection between the portal vein and inferior vena cava. Shunt length was of few millimetres (Fig.1-2).





■ Figures 1 and 2. Liver US and MRI that shows intrahepatic portosystemic shunt (arrow). The length of the shunt is a few millimetres.

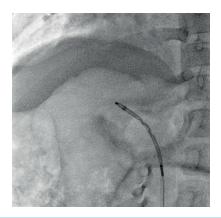
DESCRIPTION OF THE TECHNIQUE

The shunt was canalised (CR 5-F catheter) from a right femoral vein approach. A transfistula hemodynamic study was done using an occlusion balloon (pressure in portal vein with shunt occlusion: 18 mmHg; free portal vein direct pressure: 12 mmHg). Subsequently, a portography confirming the MRI-described findings was performed (Fig. 3)



■ Figure 3. Portography after catheterisation of the shunt from the right femoral vein access.

A 5-F sheath was advanced into the right portal vein using the femoral vein approach (Fig 4). A 12 mm vascular plug (Amplatzer II, Abbott) was deployed. (Fig. 5). The distal disc of the plug was released into the portal vein and the proximal disc into the inferior vena cava (Fig 6).





■ Figures 4 and 5. After advancing the tip of the 5-F sheath into the right portal vein, the Amplatzer plug II was released.

CONGENITAL INTRAHEPATIC PORTOSYSTEMIC SHUNT



■ Figure 6. Cavography from the sheath showing right placement of the vascular plug (distal lobule into the portal vein and proximal lobule into the vena cava) and vena cava patency.

Follow-up with Doppler ultrasound revealed complete occlusion of the portosystemic shunt (Fig 7).



■ Figure 7. Post-treatment US that shows occlusive lobule into the portal vein side. No portosystemic shunt was observed.

COMMENTS

Intrahepatic congenital shunts are abnormal communications between portal branches and suprahepatic or vena cava veins. According to Park's classification there are 4 types of shunts:

- **Type 1.** Most common: Single tract that connects the right portal vein to the inferior vena cava.
- **Type 2.** Single or multiple peripheral shunts between portal vein branches and suprahepatic veins in a single liver segment.
- **Type 3.** Portal vein connection with systemic circulation via aneurysmal dilatation.
- **Type 4.** Multiple and diffuse communications between peripheral portal branches and suprahepatic veins in both liver lobes.

Type 1 portosystemic spontaneous shunts reports are more commonly associated to liver cirrhosis and portal hypertension. Liver encephalopathy may develop during its course. This shunt could be caused by the persistence of a high flow communication between the omphalomesenteric system from which the portal vein is derived, with the right horn of the venous sinus, originating the inferior vena cava.

The management of portosystemic congenital liver shunts may be surgical or endovascular. In the present case, shunt length is very short, so the use of regular embolisation devices is not advisable due to the high risk of distal migration. Better than other embolic devices, it was decided to deploy a nitinol vascular plug for ductus arteriosus occlusion (ADO type II) that allows an effective closure with greater stability and no risk of distal migration.

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DUODENAL VARICES

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MEDICAL HISTORY

63-year-old male diagnosed of alcoholic liver cirrhosis, Child-Pugh score C11 and MELD score 17 with signs of portal hypertension.

The patient was admitted due to acute gastrointestinal bleeding and presyncopal status. An emergency endoscopy showed absence of gastro-oesophageal varices and a large varix in the second duodenal portion with active bleeding points, successfully treated with ethanolamine oleate direct injection. Referred for TIPS at 24 hours.

DESCRIPTION OF THE TECHNIQUE

By right internal jugular access, the right suprahepatic vein was selectively catheterised, and intrahepatic puncture was performed. Portal vein was accessed at it's bifurcation between the main right and left branches. Splenoportography was performed (Figs. 1 and 2).





Figures 1 and 2. Splenoportography before TIPS showing large varices dependent on a duodenal vein.

DUODENAL VARICES

The intrahepatic tract was subsequently dilated and the length was measured with a calibrated catheter from the entry into the portal vein to the origin of the suprahepatic vein, implanting a specific coated stent (Viatorr® GORE), which also required post-dilatation.

Post-procedural splenoportography was performed, confirming the proper TIPS function, though residual filling of the duodenal varices was still observed (Fig. 3).

Based on these findings, a 6-F guide catheter (Guider Soft®, Boston Scientific) was advanced trans-TIPS into the afferent branch to the duodenal varices and a 10 mm AVP II occluder (Abbott) was deployed through it (Fig. 4).

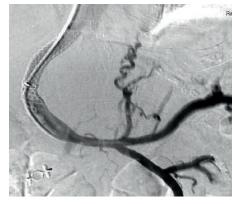
Post-procedural splenoportography was performed after implantation of the occluder, evidencing a lack of opacification of these duodenal varices (Figs. 5 and 6).

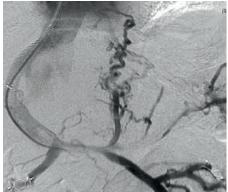


■ Figure 3. Persistence opacification of duodenal varices after TIPS implantation.



■ Figure 4. A 10 mm AVP II occluder implanted in the afferent branch towards duodenal varices, still fixed without release.





■ Figure 5 and 6. Good TIPS performance with lack of duodenal varix opacification.

The patient progressed well, and was discharged with no new episode of gastrointestinal bleeding.

DUODENAL VARICES

COMMENTS

Gastrointestinal varices may appear in any place of the gastrointestinal tract, but the most common are gastro-oesophageal varices occurring in up to 50% of cases. Duodenal varices are much less common, occurring in 4% of patients, and are rarely the cause of gastrointestinal bleeding.

Embolisation of gastrointestinal varices, after TIPS, is a controversial issue. Some authors always recommend performing it, based on published studies showing a comparative re-bleeding rate of 32% vs 19% in favour of embolisation, but other authors condition it to the post-procedural pressure gradient. Although there is no consensus, embolisation should be performed when varices continue to opacify in angiographic control after TIPS creation.

Different types of materials have been used for endovascular embolisation of gastrointestinal varicose veins, such as fibered coils, liquid embolics and vascular plugs, either alone or in combination. In our experience, the implantation of vascular occluders for embolisation of gastro-oesophageal and duodenal varices persisting after TIPS is an alternative to other embolic materials due to their good outcomes.

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MESENTERIC-GONADAL PORTOSYSTEMIC SHUNT OCCLUSION

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MEDICAL HISTORY

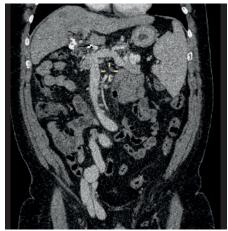
62-year-old patient with no relevant family history, no alcohol consumption. NAFLD cirrhosis known since two years before with evidence of portal HT without oesophageal varicose veins.

Hepatocellular carcinoma measuring 7 cm in segment IV and treated with two TACE sessions. Complete portal vein thrombosis was found, so he was excluded from the transplant list.

In the last control CT, a filiform portal vein was found, with collaterals in right flank, dependent of the superior mesenteric vein. Collateral circulation with dilatation of the right gonadal vein by spontaneous mesenteric-gonadal shunt (Figs. 1 and 2).



Figure 1. Axial CT image showing right varicocele.



■ Figure 2. Coronal image shows superior mesenteric vein dilatation and connection to gonadal vein.

MESENTERIC-GONADAL PORTOSYSTEMIC SHUNT OCCLUSION

The patient showed refractory hepatic encephalopathy, so embolisation of the mesenteric-gonadal shunt was planned to try to improve his neurological status. It was decided to perform the embolisation and a TIPS during the same procedure, to try to prevent the risk of bleeding from varicose veins in the future.

DESCRIPTION OF THE TECHNIQUE

Right internal jugular vein approach was made and a TIPS was performed according to the standard technique, placing a 10 mm diameter and 7 cm covered stentgraft (Viatorr®, GORE). Main portal vein recanalization was done using a 12 mm x 6 cm selfex-pandable bare stent (Wallstent®, Boston Scientific), since the main portal trunk showed a very reduced caliber (Figs. 3 and 4).



Figure 3. Direct splenoportography with hepatofugal flow towards SMV and mesenteric-gonadal shunt.



■ Figure 4. TIPS and portal vein patent but hepatofugal flow is still present. High retrograde flow into SMV.

The verification of the function of the TIPS showed persistence of hepatofugal flow towards the superior mesenteric vein and mesenteric-gonadal shunt. Using a multipurpose catheter, the right gonadal vein was accessed from the inferior vena cava and a 22 mm diameter AVP II (Abbott) was placed using a 90 cm 7-F sheath (Destination®, Terumo) (Figs. 5 and 6).

We performed a new splenoportography finding a clear flow improvement towards the TIPS with decreased hepatofugal flow.

MESENTERIC-GONADAL PORTOSYSTEMIC SHUNT OCCLUSION





■ Figure 5. Contrast injection in the varicocele accessing to it through the right gonadal vein through the inferior vena cava.

■ Figure 6. Varicocele occlusion and closure gonadal mesenteric shunt with a 22 mm AVP II.

COMMENTS

Hepatic encephalopathy is a reversible state of impaired cognitive function that may occur in patients with liver disease or with portosystemic shunts. These shunts help reduce portal pressure, but they may also promote the occurrence of hepatic encephalopathy. The most common spontaneous by-passes are splenorenal and gastrorenal, but there are other possibilities, such as the mesenteric-gonadal that we present here. Closure of these shunts causes improvement of encephalopathy due to reversion of hepatic flow. However, portal pressure will increase, so these patients should be monitored for the risk of development of gastro-oesophageal varices and/ or ascites. Nitinol vascular plugs are a good option for closure of portosystemic shunts due to their simple management and lower risk of migration than other embolisation agents.

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NITINOL PLUGS AS ANCILLARY TOOL FOR LIQUID EMBOLIC INJECTION IN CARDIOFUNDAL VARICES

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CASE 1

MEDICAL HISTORY

53-year-old male with alcoholic cirrhosis. Two recent admissions for variceal UGIB that resolved with endoscopic treatment. The patient reported to the emergency room with a new episode of UGIB, new endoscopic treatment despite which anaemia persisted. TIPS and embolisation of gastrooesophageal varices using cyanoacrylate were indicated.

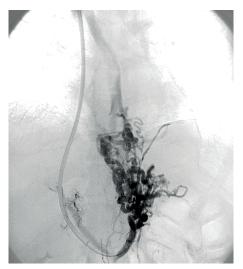
Five days after TIPS, the patient suffered a left frontoparietal haematoma causing death. He had no further episodes of UGIB or required transfusion after embolisation.

DESCRIPTION OF THE TECHNIQUE

A 12-F and 45-cm radiopaque tip hydrophilic sheath (Flexor® Ansel, Cook®) through the TIPS with its tip in the main portal vein. Stomatal coronary vein catheterisation with 4-F MPR catheter (Tempo® Aqua Cordis) and venography showing gastrooesophageal varices (Fig. 1). In parallel, through the sheath, a second MPR catheter with similar characteristics was placed in the ostium of the stomatal coronary vein, and an 8 mm AVP 4 occluder (Abbott) was advanced through it, which was expanded to block the trunk of the vein. The first catheter is now trapped by AVP 4 and its tip at the origin of the varices (Fig. 2). The occluder will protect from the potential reflux of cyanoacrylate into the portal vein.

3 cc of cyanoacrylate (Glubran® GEM) mixed with 6 cc of lipiodol (33% dilution formula) were injected through the 4-F catheter to occlude the entire variceal bed. The catheter was removed and AVP 4 was released as the last step (Fig. 3).

NITINOL PLUGS AS ANCILLARY TOOL FOR LIQUID EMBOLIC INJECTION IN CARDIOFUNDAL VARICES



■ Figure 1. Pre-embolisation venography. Gross gastro-oesophageal varices dependent on the stomatal coronary vein with distal drainage toward the azygos vein.



■ Figure 2. Detail prior to injection of cyanoacrylate. AVP 4 occlusion blocking the coronary vein origin (arrow). Second 4-F catheter in parallel with the tip distal to the occluder (arrow head).



■ Figure 3. Final control portography.
Varices excluded. Distal to AVP 4 (arrow)
the cyanoacrylate filling the varicose veins.

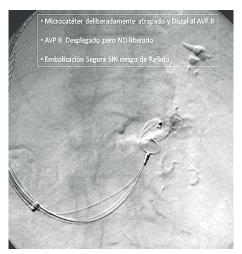
CASE 2

MEDICAL HISTORY

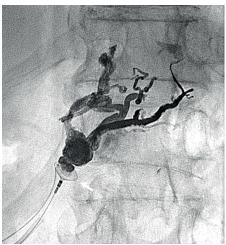
74-year-old patient with alcoholic cirrhosis, complaining of a third episode of UGIB secondary to gastrooesophageal varices. Despite endoscopic treatment, bleeding persists. TIPS indicated. Gastrooesophageal varices persist after the TIPS was created. Embolisation of the varices was indicated for which non-adhesive liquid embolic ethylene vinyl alcohol copolymer (EVOH-Onyx® 18, Medtronic) was chosen. The patient died 24 months later due to liver disease progression with no further episodes of UGIB.

DESCRIPTION OF THE TECHNIQUE

Trans-TIPS 12-F 45 cm sheath (Flexor® Ansel. CooK) in the main portal vein. With a 4-F multipurpose catheter, the stomatal coronary vein was catheterised. A microcatheter (Rebar® 14, Medtronic) was coaxially inserted, the distal end of which lies within the oesophageal varices. Simultaneously, in parallel to the multipurpose catheter, a 6-F and 90-cm guide catheter (Guider Soft® Boston Scientific) was introduced to also channel the coronary vein. Through the 6-F guide catheter, we advance a 12-mm AVP II nitinol occluder (Abbott) in the trunk of the coronary vein. The microcatheter is then trapped on purpose by AVP II (Fig. 4). We were then ready to start the embolisation of the varices with EVOH at stopped flow and without the risk of liquid embolic reflux back into the portal vein, as it is prevented by AVP II (Fig. 5).



■ Figure 4. Details of the position of the microcatheter, AVP II, guide catheter and Flexor® sheath immediately before starting EVOH embolisation.



■ Figure 5. EVOH injection by blocking technique. AVP II occludes flow allowing EVOH to advance into varicose cords.

NITINOL PLUGS AS ANCILLARY TOOL FOR LIQUID EMBOLIC INJECTION IN CARDIOFUNDAL VARICES

After embolisation was completed, we pulled back the microcatheter that came out without problems despite AVP II. We released AVP II as the final step (Fig. 6).



■ Figure 6. Final control portography. AVP II in the somatic coronary vein trunk preventing reflux of EVOH (arrow).

COMMENTS

Although embolisation of gastroesophageal varices may potentially exacerbate portal hypertension, it has been shown to reduce re-bleeding in these patients. Proximal embolisation of the somatic coronary vein with coils does not prevent future recanalization of varices. This is partly due to the thrombocytopenia and coagulopathy suffered by these patients. Liquid embolics, cyanoacrylates and EVOH, are of special interest in this territory because they are not influenced by the coagulation status of the patient. In addition, they are not limited to occluding the origin of the vein, but to filling and thrombosing most of the variceal bed. EVOH is safe and controlled release, but, as a technical difficulty, it has problems preventing reflux. Cyanoacrylate is a highly effective but uncontrolled-release product, so the risk of unwanted embolisation is greater. We present an AVP + Glubran®/ Onyx® combined technique, in which the Amplatzer occluders are essential for safe and effective embolisation with liquid materials.

NITINOL PLUGS AS ANCILLARY TOOL FOR LIQUID EMBOLIC INJECTION IN CARDIOFUNDAL VARICES

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MESOCAVAL PORTOSYSTEMIC SHUNT OCCLUSION WITH AMPLATZER NITINOL PLUGS

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MEDICAL HISTORY

57-year-old male with ortotopic liver transplantation four years before. Chronic rejection and reinfection with HCV. Asymptomatic until admission in our centre for encephalopathy, with hyperammonemia of 221 mcg/dL. Conservative management with medical treatment was initially done without achieving clinical improvement.

Liver Doppler ultrasound revealed a thin patent portal vein, and the abdominal CT examination revealed massive spontaneous mesocaval portosystemic shunt (Fig. 1). Shunt occlusion was considered by embolisation as part of the treatment for encephalopathy.



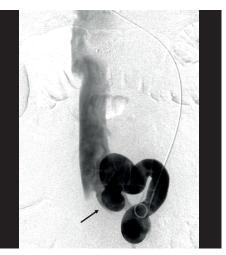
■ Figure 1. Abdominal CT VR reconstruction. IMV with a 15-mm caliber (white arrow), a tortuous portion and direct connection to the iliac vein (arrowheads). Inferior vena cava (hollow arrow).

DESCRIPTION OF THE TECHNIQUE

Transhepatic portal vein approach with direct ultrasound-guided puncture and transhepatic placement of a 23-cm 6-F sheath (Brite TIP, Cordis) to the main portal vein of the graft. Direct spleno-portography confirming the CT findings. Hepatic flow in splanchnic territory with a significant reduction of the intrahepatic portal perfusion (Fig. 2 and 3). Direct portography from inferior mesenteric vein (IMV) and superior mesenteric vein (SMV) showing mesocaval shunt. No other significant portosystemic shunt were seen.



■ Figure 2. Direct transhepatic portography. Poor intrahepatic portal flow (arrow head). Hepatofugal flow by hypertrophic IMV (arrows) that drains directly to the IVC.



■ Figure 3. Catheter venography at the distal end of the IMV with retrograde flow and significant mesocaval shunt (arrow).

The thranshepatic 6-F sheath was exchanged for an 8-F, 45-cm sheath (Flexor®, Cook®) compatible with the use of bigger AVP II nitinol plugs (Amplatzer, Abbott). Two of these devices, 22 mm and 20 mm respectively, were released in tandem and were anchored in the middle third of the IMV (Fig. 4). The post-procedural portography study showed a satisfactory outcome with complete shunt occlusion and hepatopetal redistribution of the portal flow, resulting in a significant increase of the intrahepatic perfusion (Fig. 5). Occlusion of the transhepatic access tract was done using fibered coils.

Blood tests 12 hours after the procedure showed normalisation of blood ammonia (27 mcg/dL), which preceded clinical improvement of the patient 24 hours later. The abdominal CT control at 48 hours confirmed complete shunt closure (Fig. 6).

Potential complications of this procedure by transhepatic approach include: intraperitoneal bleeding, right pleural effusion, biliary leakage, and impaired liver function as a result of an iatrogenic arterio-venous fistula.

MESOCAVAL PORTOSYSTEMIC SHUNT OCCLUSION WITH AMPLATZER NITINOL PLUGS

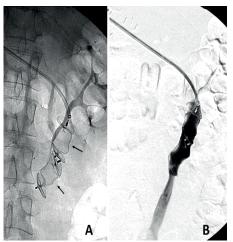
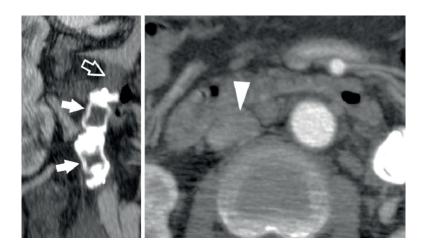


Figure 4. A) Release of two 20 mm and 22 mm AVP II in the middle third of the IMV (arrows).

B) Immediate control venography.

■ Figure 5. Final control portography. Marked increase in intrahepatic portal perfusion (pre-treatment portal perfusion detail in lower left corner), and occlusion of IMV.



■ Figure 6. Control CT at 48 h. Correct position of AVP II (arrows). Thrombosis of the IMV (hollow arrow). Disappearance of early enhancement in IVC (arrow head).

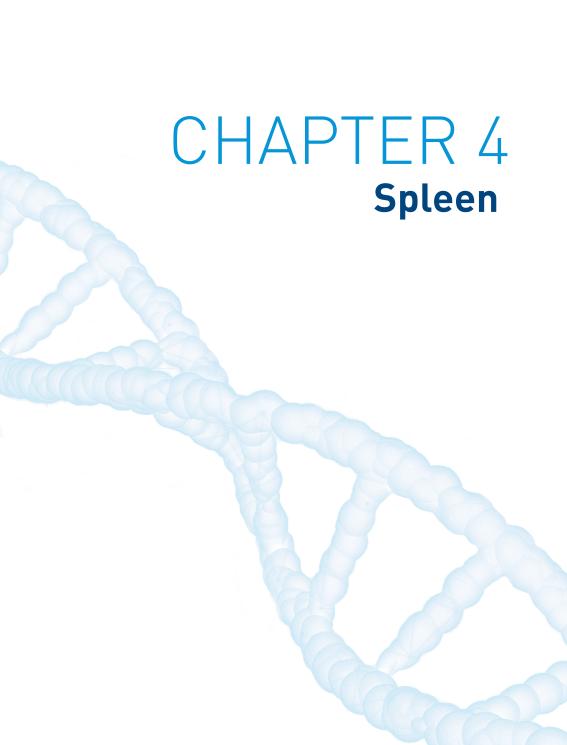
There was no procedure complications and the patient presented no further episodes of encephalopathy. He died seven months later due to brain haemorrhage unrelated to his liver condition.

COMMENTS

Mesocaval portosystemic shunt in cirrhotic patients is less common than splenorenal, gastroesophageal varices or umbilical vein recanalization that are the usual spontaneous shunts in the setting of portal hypertension. Mesocaval portosystemic shunts develop from pre-existent subclinical shunts between SMV or IMV and ICV that enlarge due to severe portal hypertension. In some situations, these shunts lead to hepatic encephalopathy. In the present case, the portosystemic shunt was established in the context of the native liver cirrhosis, and this shunt had remained active despite liver transplantation.

Initial treatment for encephalopathy is medical and conservative. The development of new embolisation materials has allowed for effective and safe percutaneous treatment of these shunts when medical treatment fails or is insufficient. Vascular plugs, compared to coils as the traditionally used material, have several advantages. They embolise large-calibre, high-flow vessels, cause rapid occlusion and are of controlled-release with the possibility of repositioning prior to their final release, allowing accurate and safe positioning of the device within the vessel.

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IATROGENIC RUPTURE OF A SPLENIC ANEURYSM

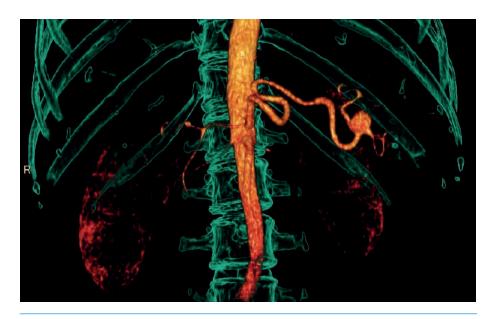
G. Rodríguez R., M. González L., A. Álvarez L., M. Echenagusía and F. Camúñez

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MEDICAL HISTORY

53-year-old woman with liver transplant due to fulminant liver failure secondary to HBV in 2011. A 20 mm splenic aneurysm close to the spleen hilum was fond during a follow-up CT scan (Fig. 1).

Although surgery is a therapeutic option, due to the patient's comorbidity and high surgical risk, endovascular treatment of the aneurysm was decided. Bare stent placement with microcoils was the initial therapeutic plan.

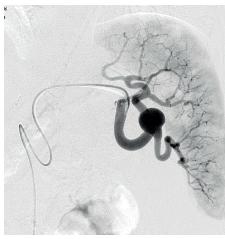


■ Figure 1. CTA 3D VR reformation. 20 mm splenic artery aneurysm in the splenic hilum.

DESCRIPTION OF THE TECHNIQUE

Selective arteriography of the celiac axis and splenic artery were performed from right femoral approach. A highly tortuous splenic artery is shown and a 20 mm aneurysm is confirmed in the distal segment of the splenic artery with two efferent arteries arising from the aneurysm (Figs. 2 and 3).





■ Figures 2 and 3. Selective arteriograms of the celiac trunk and splenic artery confirming the CTA diagnosis.

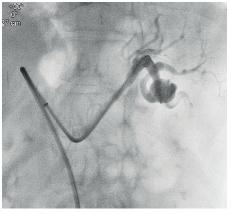
After diagnostic angiography, an 8-F long sheath (Flexor® - Cook®) was advanced into the proximal splenic artery to have permanent access to the celiac axis and to have a greater support which was considered necessary due to the tortuosity of the artery. A 7-F guiding catheter was advanced coaxially through the sheath to reach the aneurysm (Fig. 4).

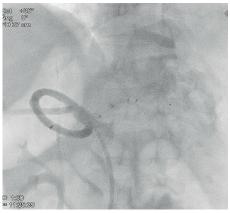
During this aneurism access operation, contrast extravasation was observed due to aneurysm wall rupture. Hypovolemic shock was detected and quick treatment was mandatory. Due to the emergency situation and thanks to the 7-F guide catheter was in position, the splenic artery main trunk was embolised using several Amplatzer plugs (AVP, AVP II and AVP4 – Abbott) to control the bleeding as soon as possible. The types and sizes of the devices were chosen based on the size of the artery segment and the tortuosity of the artery.

The final control angiography shows complete occlusion of the splenic artery with no evidence of bleeding (Fig. 5).

When this emergency embolisation was completed the patient recovered and she was hemodynamically stable. However after discussing the case with surgery and ICU Services she was transferred to the operating room for splenectomy and resection of the splenic artery proximal to the implanted vascular plugs.

IATROGENIC RUPTURE OF A SPLENIC ANEURYSM





■ Figure 4. Angiographic control through the 8-F long sheath after catheterisation of aneurysm.

■ Figure 5. Control arteriography after embolisation. Vascular plugs in the main splenic artery with complete occlusion and absence of bleeding.

COMMENTS

It is difficult to decide which embolisation material should be used in an emergency case like this. Clearly the goal is to occlude the splenic artery as quickly and safely as possible to control massive bleeding. No material available for occlusion of medium-sized arteries ensures immediate and safe hemostasis under all circumstances. In this case, the patient developed hypovolemic shock due to aneurysm rupture during catheterisation manoeuvres. As discussed, complete and swift embolisation of the splenic artery was the key target for bleeding control. Since there was a guide catheter adjacent to the rupture, it was decided, as the best option, to deploy in tandem several Amplatzers occluders until bleeding was controlled, which fortunately was achieved very fast.

The easy implantation of the Amplatzer Vascular Plugs and their high haemostatic efficacy when compacting several of them, may be an effective alternative for these type of emergencies.

IATROGENIC RUPTURE OF A SPLENIC ANEURYSM

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IATROGENIC RUPTURE OF SPLENIC ARTERY

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MEDICAL HISTORY

78-year-old male with high blood pressure and no other relevant medical history. Asymptomatic, with good general condition and active lifestyle. Incidentally, in a plain abdominal X-ray, a calcification was detected in the left flank. An abdominal CT was performed and on the distal portion of the splenic artery a 32 mm saccular aneurysm, with calcified wall, was found. Endovascular treatment was proposed to the patient and he accepted. The initial therapeutic purpose was to exclude aneurysm by packaging it with hydrocoils (Terumo) assisted by a self-expanding bare stent that would have kept the artery patent and would prevent the coils from moving outside the aneurysm into the arterial lumen.

DESCRIPTION OF THE TECHNIQUE

General anaesthesia and antibiotic prophylaxis with cefazolin and gentamicin. Approach from the right common femoral artery and catheterisation of the celiac axis. Non-selective arteriography showing the branches of the celiac axis as well as the aneurysm (Fig. 1). Progreat® 2.7 microcatheter (Terumo) was advanced through the splenic artery, crossing the aneurysm and reaching intrasplenic distal segmental branches. A 0.018" steel guidewire (Steel Core®, Abbott) was advanced over this microcatheter. Based on this stiff microwire, Simmons 4-F catheter (Tempo® Aqua, Cordis) could be advanced to the splenic artery distal to the aneurysm.

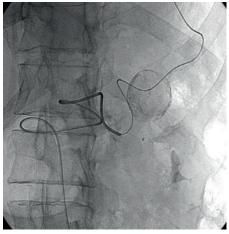
An exchange to the Amplatz super stiff 0.035" guidewire was performed (Boston ScientificTM) (Fig. 2). A 8-F 55 cm sheath (Flexor® Cook®) was advanced over this guide until surpassing the first loop of the splenic artery. Then an attempt was made to advance a self-expanding nitinol stent of 6 mm in calibre by 8 cm in length (Misago®, Terumo) with monorail system to cover the wide neck of the aneurysm and then be able to insert the coils through its mesh. During this manoeuvre, perforation of the splenic artery occurred proximal to the aneurysm with severe bleeding and intra-abdominal hematoma (Fig. 3). Given the life-threatening emergency situation created, it was decided to

IATROGENIC RUPTURE OF SPLENIC ARTERY

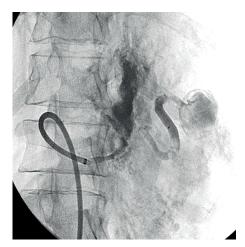
embolise the splenic artery using a 10 mm AmplatzerTM AVP II nitinol vascular occluder (Abbott), which could be quickly positioned to control the bleeding (Fig. 4).



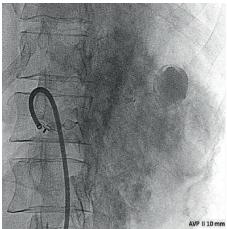
■ Figure 1. Celiac axis and its branches. Tortuous splenic artery with saccular aneurysm.



■ Figure 2. 0.035" Amplatz guidewire across the aneurysm reaching the most distal branches of the splenic artery.



■ Figure 3. Perforation of the splenic artery proximal to the aneurysm. Massive bleeding.



■ Figure 4. Proximal splenic artery embolisation with AVP II 10.

IATROGENIC RUPTURE OF SPLENIC ARTERY

Control angiography showed cessation of bleeding and the procedure was terminated (Fig. 5). Despite bleeding, the patient remained hemodynamically stable. He received transfusion of two red cell packs and was monitored at the intensive care unit for 24 hours after which he was discharged to the ward with no incidents. Six days later, the patient was discharged from the hospital and one year later he was doing well, with no splenic infarction and controlled aneurysm (Fig. 6).



Figure 5. Immediate control angiography with

cessation of bleeding. AVP II deployed (arrow).



■ Figure 6. Control CT three months later. AVP II deployed. There is no splenic infarction and the aneurysm is excluded.

COMMENTS

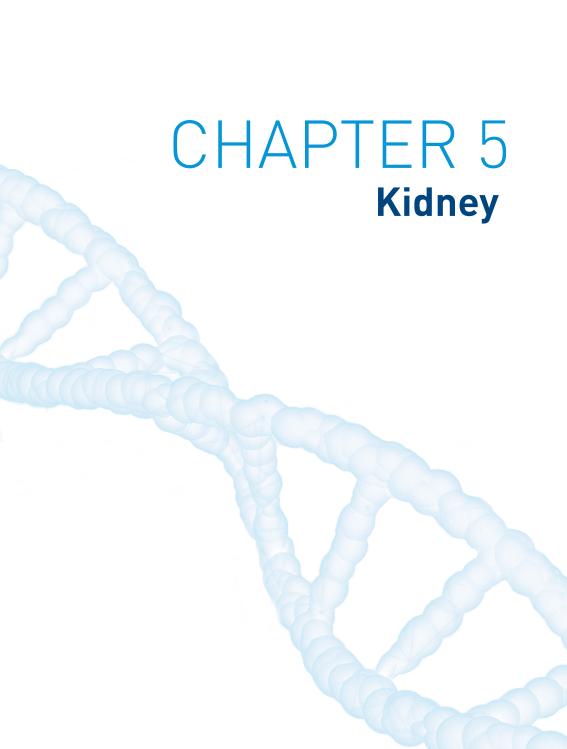
Incidental diagnosis of asymptomatic aneurysms in visceral arteries has increased in recent years due to the popularization of CT and MRI as diagnostic tests. As in aortic aneurysms, the most common cause is atherosclerotic degeneration, and the splenic artery is the visceral artery most commonly affected. Endovascular treatment is currently considered to be of choice because it is less invasive. However, a visceral aneurysm is always a challenge, particularly when considering patency of the artery distal to the aneurysm. Advancing bare or covered stents along the loops of the splenic arteries is not an easy task and as, shown in our case, it involves some risks.

IATROGENIC RUPTURE OF SPLENIC ARTERY

We know from the experience in blunt abdominal trauma that splenic bleeding can be effectively controlled by proximal embolisation of the splenic artery. There is a significant reduction in perfusion, allowing the bleeding site to spontaneously hemostasis while the collaterality of the spleen through the gastroepiploic artery and the left gastric artery will protect the spleen from massive infarction.

We report a case with a very serious and life-threatening complication that was solved with endovascular treatment. It is advisable that vascular radiology departments have nitinol plugs available that can be used at any time and urgently to treat bleeding in large, high-flow arteries.

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RENAL ARTERIOVENOUS FISTULA

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MEDICAL HISTORY

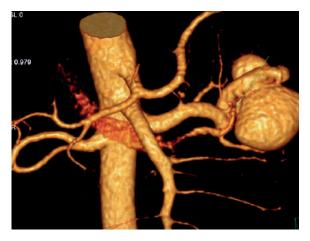
40-year-old male referred by the urology department of another hospital, complaining of hematuria and mild pain in the left flank lasting for two years, with no other relevant history. An initial ultrasound examination showed a cystic mass in the middle third of the left kidney, but Doppler US detected flow inside the cyst.

The abdominal CT scan showed a bilobar vascular mass (6 x 5 x 3 cm in size) with eggshell calcification located in the middle third of the left kidney. These CT findings were related to the dilated and arterialised proximal venous segment due to a fistulous tract between the distal segment of the left renal artery and a single renal vein outflow (Fig. 1- 2). The described mass compressed the upper calicial system, which was slightly enlarged and it was associated with mild parenchymal atrophy of the upper pole and middle third of the kidney. The main renal artery was enlarged, reaching 9 mm in diameter (Fig. 3).

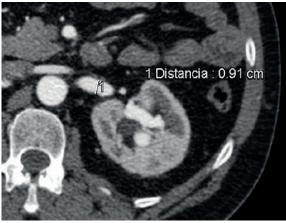
Given the findings, it was decided to perform a left renal arteriography and fistula embolisation.



■ Figure 1. Axial CT scan. A fistulous tract is defined (arrow), and the arterial and venous ends are noted.



■ Figure 2. CT volume rendering reformation. Hypertrophy of the left renal artery, "vascular mass" and venous efferent.



■ Figure 3. Measurement of the left renal artery diameter on CT to calculate the diameter of the vascular plug to be used after calculating the oversize (approx. 30%)

DESCRIPTION OF THE TECHNIQUE

From right femoral artery access, the left renal artery was canalised using a 4-F cobra 2 catheter (Tempo® Cordis). A selective left kidney angiography confirms CT findings: a bilobed vascular mass and early filling of the renal vein consistent with arteriovenous fistula (Fig. 4).

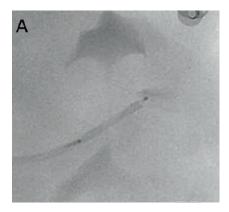
Subsequently, using a coaxial system consisting of a 7-F multipurpose Envoy® guide catheter and a 4-F cobra 2 angiographic catheter (Cordis), the renal artery was catheterised. 7-F guide catheter tip was located distally to the origin of the segmental parenchymal branches, reaching the fistulous tract in a slightly tortuous pathway over a hypertrophic main renal artery (9 mm). The vascular plug chosen was a 12 mm AVP I (Abbott), attempting to adapt oversize, occlusive capacity and post-implantation length to allow the closest parenchymal branches to remain patent. The constrained

plug was advanced, maintaining constant tension over the guide catheter (after removing the diagnostic 4-F cobra catheter) due to the tendency to retract over the curve of the artery in its segment immediately proximal to the fistulous tract (Fig. 5). Once the plug was implanted, complete occlusion of the fistula was shown a few minutes later. Patency of the healthy parenchymal arterial branches, presence of contrast retained in the aneurysmal segment of the fistula and in the upper caliceal system, as a result of compression caused by the fistula was shown (Fig. 6).

Patient follow-up was satisfactory, and complete fistula occlusion persists in imaging controls three years later.



■ Figure 4. Left kidney selective angiography. Good correlation is seen with the CT image: feeding artery hypertrophy, bilobed aneurysmal dilatation corresponding to fistulous tract and early renal vein drainage.



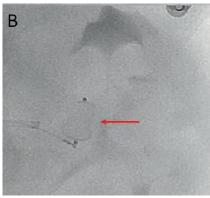


Figure 5. A) AVP I inside the 7-F guiding catheter before plug implantation. **B)** Plug already deployed but not yet released. Typical bilobed morphology is identified (arrow).

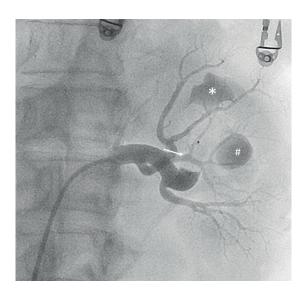


Figure 6. Arteriography ten minutes after vascular plug implantation. Complete fistula occlusion is confirmed. Normal kidney arterial segmental branches are preserved. Some contrast remnant is seen in the aneurysmal dilatation of the fistula (hash) and ectasia in the upper caliceal system due to extrinsic compression caused (asterisk).

COMMENTS

80% of kidney arteriovenous fistulas are acquired secondary to prior surgery, injuries or tumoural lesions. Their anatomy is usually simple with a single afferent and efferent vessel. Congenital fistulas may also occur but the configuration is often more complex, sometimes representing a true vascular malformation. There is another small percentage of kidney fistula with no clear cause, as in this case. They may be asymptomatic, but usually large kidney fistula cause haematuria, flank discomfort or pain (as in this patient), hypertension or hyperdynamic heart failure.

Embolisation is the treatment of choice in most cases, because it involves less morbidity and a shorter recovery time. However, this procedure may be technically challenging, given the high-flow conditions induced by the arteriovenous fistula, with the associated risk of potential migration of the embolisation material. Multiple embolisation agents and technical variants have been used, including the simultaneous transvenous approach, or the use of different filtering devices. The use of vascular plugs, if the fistulous tract can be reached and if there is an adequate implantation segment, is an excellent alternative and is particularly indicated given its volumetric configuration, control at release and high effectiveness. AVP I is very useful when the landing zone is too short.

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POST-TRAUMATIC RENAL PSEUDOANEURYSM

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MEDICAL HISTORY

50-year-old patient diagnosed in May 1993 of urothelial neoplasm. Treatment included left nephrectomy, radical cystoprostatectomy, and right uretero-ileostomy to the ileal loop (Bricker). Right hydronephrosis due to stenosis of the pyeloureteral junction with recurrent urinary tract infections, and right nephrectomy performed in May 1994. Hemodialysis until March 1998, when a kidney transplantation was performed, that functions normally to date.

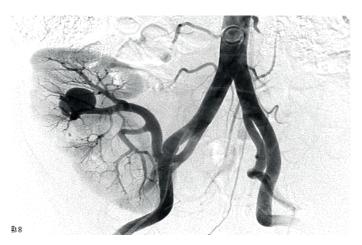
Urgent admission due to hematuria after suffering a traffic accident, performing Doppler ultrasonography and enhanced pelvic CT, which evidenced post-traumatic renal pseudoaneurysm (Fig. 1).



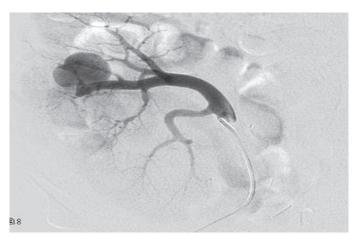
■ Figure 1. Angio-CT.
Pseudoaneurysm in right renal graft.

DESCRIPTION OF THE TECHNIQUE

We performed a pelvic and selective arteriography of the right renal graft by ipsilateral femoral access (Fig. 2), that confirmed the existence of a pseudoaneurysm, visualizing the laceration of a small arterial branch with ejection jet of contrast medium into the false aneurysm cavity (Fig. 3) and retention of it inside, until the later stages of the examination.



■ Figure 2. Pelvic arteriography confirming the existence of a pseudoaneurysmal cavity.



■ Figure 3. Selective arteriography of the renal graft. Laceration of a small arterial branch with ejection jet into the cavity.

We catheterised the branch that had the laceration, initially attempting to embolise it with coils (Fig. 4A) and, as it was not stable, an 8 mm diameter AVP 4 nitinol vascular plug (Abbott) was implanted (Fig. 4B), obtaining thrombosis of the pseudoaneurysm, which was confirmed through post-procedural arteriography (Fig. 5).

POST-TRAUMATIC RENAL PSEUDOANEURYSM

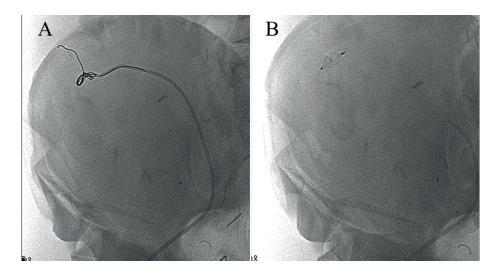
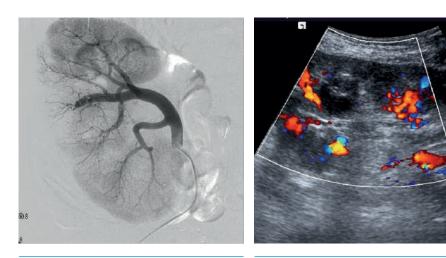


Figure 4. A) Initial attempt of embolisation with coils, not stable. B) Embolisation with an 8 mm diameter AVP 4.

The patient was discharged with no new hematuria, followed-up with regular Doppler ultrasonography, which confirmed the persistence of pseudoaneurysm thrombosis (Fig. 6).



■ Figure 5. Post-embolisation arteriography that evidenced exclusion of pseudoaneurysm.

■ Figure 6. Control Doppler ultrasound confirming pseudoaneurysm thrombosis.

POST-TRAUMATIC RENAL PSEUDOANEURYSM

COMMENTS

Renal pseudoaneurysms are usually caused by penetrating injuries, including surgical or percutaneous (iatrogenic) procedures. When they occur after a non-penetrating injury, the cause is usually sudden deceleration (traffic accidents).

The most common symptoms are flank pain, hypotension, and particularly hematuria which, though they usually yield, persist in 10% of cases.

Our case was a pseudoaneurysm secondary to a non-penetrating injury, in which embolisation with a vascular occluder was rapid, effective and reliable, and maintained over time as confirmed by control Doppler ultrasound.

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RENAL GRAFT INTOLERANCE SYNDROME

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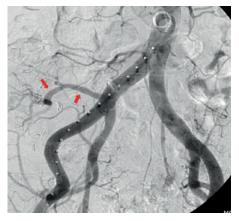
MEDICAL HISTORY

57-year-old patient with a kidney transplant due to chronic renal faliure related to tubulointerstitial nephropathy. The graft failed and a transplantectomy was required three years later due to graft intolerance. A new kidney transplant was performed, which was not functional at any time, so the patient returned to dialysis. The patient was admitted due to three weeks of fever. After multiple tests, graft intolerance syndrome was again diagnosed, and transplant embolisation was requested.

DESCRIPTION OF THE TECHNIQUE

From right femoral artery approach, we performed pelvic arteriography (Fig. 1), visualising renal graft in right pelvis, with anastomosis in the right common iliac artery. We selectively catheterised the renal artery, seeing a significant decrease of intrarenal vascularisation (Fig. 2).

RENAL GRAFT INTOLERANCE SYNDROME

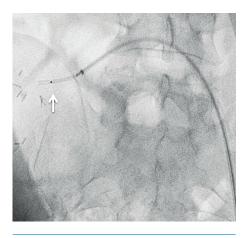




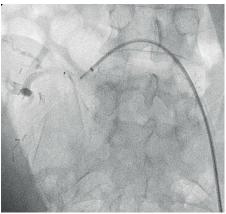
■ Figure 1. Pelvic arteriography. Renal graft (arrows).

Figure 2. Selective renal graft arteriography. Decrease of intrarenal vascularisation.

To ensure the stability of AVP (Abbott) placement and facilitate navigation, we decided to access via the left femoral route. We placed a 6-F curved introducer in the common iliac artery and a 6-F guide catheter in the renal graft artery and through it inserted the 10 mm diameter AVP II (Fig. 3). We removed the guide catheter a few millimetres and checked that the AVP II was properly positioned (Fig. 4). Finally, we released AVP II and performed control arteriography, verifying that treatment was successful, achieving complete occlusion of the renal graft artery (Figs. 5 and 6).



■ Figure 3. 6-F curved introducer in the common iliac artery and 6-F guiding catheter in renal graft artery. Punctiform mark of the AVP II (arrow)



■ Figure 4. Check adequate positioning of AVP II before release.

RENAL GRAFT INTOLERANCE SYNDROME

The patient had a post-embolisation syndrome, with low-grade fever and mild pain, which subsided with medical treatment at 24 hours. Four days later, the laboratory parameters of chronic inflammation had improved and the patient was free of fever.



-1943 H BA/195-05 -2005

■ Figure 5. Common iliac arteriography, visualising complete renal graft artery occlusion and absence of opacification of renal graft vascularisation.

■ Figure 6. Image without digital subtraction to be able to assess AVP II (arrow).

COMMENTS

Immunologic intolerance to a failed renal allograft left in situ is referred to as "graft intolerance syndrome". Treatment by transcatheter vascular embolisation has been reported to be less invasive than transplantectomy. This syndrome may occur in patients who have lost the functionality of the transplanted kidney and who have symptoms such as pain, fever, haematuria and swelling in the graft area. These symptoms are caused by a graft-versus-host immune reaction. Sometimes there are no clinical signs, and only laboratory findings included anaemia, hypoalbuminaemia and elevated ESR and CRP can be detected.

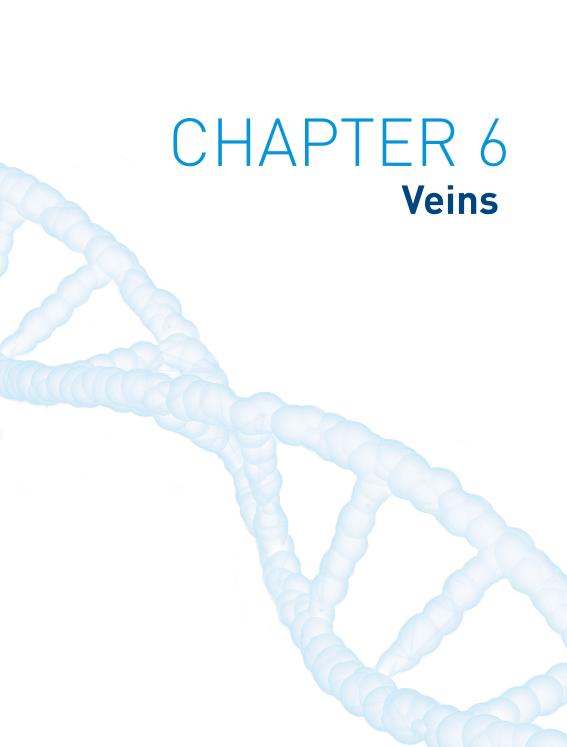
If graft intolerance cannot be resolved by medical management, a more aggressive treatment should be considered by nephrectomy or graft embolisation. Surgery is not free of risks, and embolisation is currently considered the procedure of choice in these cases.

There are many materials available for proper embolisation. In our case, because of the clear scarce of intrarenal vasculature, we decided to embolise the graft using only an AVP II, obtaining a complete occlusion.

RENAL GRAFT INTOLERANCE SYNDROME

The most common complication is post-embolisation syndrome, occurring in 50%-90% of cases, characterised by fever, local pain, haematuria, nausea, and vomiting, which subside within 48-72 hours.

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PELVIC CONGESTION SYNDROME

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MEDICAL HISTORY

29-year-old patient with no relevant family history, reporting varicose veins in the lower limbs since 14 years of age with worsening since her last childbirth and vulvar varices with recent occasional bleeding.

Doppler ultrasound of the lower extremities was performed, showing venous insufficiency with absence of valve closure after performing a Valsalva manoeuvre in both internal saphenous veins. In the deep venous system, there was a delay in valve closure, though it finally occurs completely.

The patient was referred for pelvic venography, for diagnostic confirmation of the suspected pelvic congestive syndrome and possible endovascular treatment with embolisation.

DESCRIPTION OF THE TECHNIQUE

By puncture of a superficial vein in the right antecubital fossa, both gonadal veins were selectively catheterised. The location of the ovarian veins was usual, the left flowed into the ipsilateral renal vein (Figs. 1-2) and the right directly into the inferior vena cava, just inferior to the right renal vein. The Valsalva manoeuvre may be useful for locating gonadal veins, particularly the right one, whose catheterisation tends to be more laborious.

Once the presence of pelvic varicose veins was confirmed, a microcatheter was advanced as distally as possible, and fibered microcoils (Interlock®, Boston Scientific) ranging from 4 to 8 mm in size were inserted, obtaining occlusion of the viewed pelvic varices.

After that a 8 mm diameter AVP 4 (Abbott) were placed in the middle third of the ovarian veins to prevent the persistence of reflux and the possibility of varicose veins recurring by this pathway.

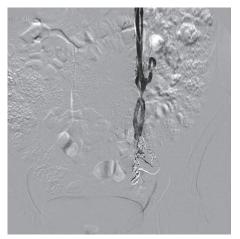
Collaterals are often present, as in this case in the left side, where two AVP devices were placed (Figs. 3 and 4).

PELVIC CONGESTION SYNDROME

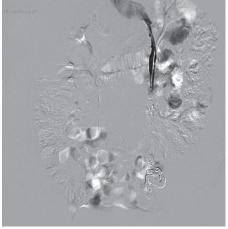


■ Figure 1. Phlebography of the left gonadal vein showing reverse flow and varicose veins.

■ Figure 2. Filling of varicose veins and right gonadal vein by injecting from the left.



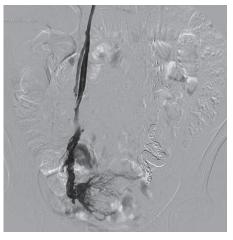
■ Figure 3. Embolisation of varicose veins with 0.018" fibered metallic microcoils.

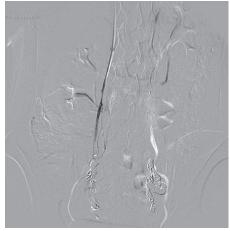


■ Figure 4. AVP 4 in middle third of ovarian vein. Two devices were placed due to duplicity of the vein.

The result was immediately satisfactory, with confirmed disappearance of reverse flow in both ovarian veins (Figs. 5 and 6), and the symptoms disappeared within one month, as confirmed in the follow up.

PELVIC CONGESTION SYNDROME





■ Figure 5. Selective right ovarian vein phlebography.

■ Figure 6. Final outcome on the right side after inserting coils and AVP 4.

COMMENTS

Pelvic congestion syndrome is characterised by chronic, non-cyclic pelvic pain lasting more than six months, which may also be associated with multiple symptoms such as post-coital pain, dysmenorrhoea, uterine bleeding, low-back pain, etc.

In addition, it is usually associated with varicose veins in the lower limbs. Treatment of pelvic varicose veins may improve the symptoms in the lower limbs and decrease the possibility of recurrence after surgery.

The materials used for embolisation of the pelvic varicose veins may be diverse, but we believe that the use of the nitinol vascular plugs in the middle part of the gonadal veins is greatly helpful by preventing the persistence of backward flow.

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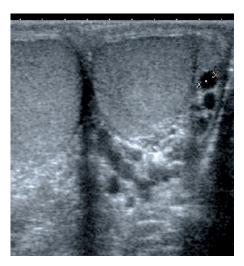
VARICOCELE

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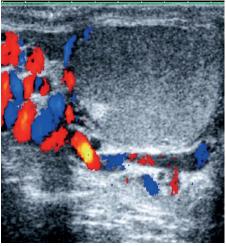
MEDICAL HISTORY

24-year-old male with pain in the left testicle lasting for several months. A testicular Doppler ultrasound was performed (Figs. 1 and 2), showing the existence of a left varicocele.

The patient was referred for endovascular treatment.



■ Figure 1. Ultrasonography. Dilatation of the spermatic cord veins of the left testis.



■ **Figure 2.** Doppler ultrasonography: Venous flow in varicose dilatations, which increase with Valsalva manoeuvre.

DESCRIPTION OF THE TECHNIQUE

By puncture of peripheral vein in the antecubital region of the right arm, we catheterished the left gonadal vein using a multipurpose catheter (Aqua Tempo®, Cordis) and performed selective phlebography, visualising dilation and tortuosity of the left spermatic vein, with no evidence of collaterals (Fig. 3). The spermatic vein measured approximately 6 mm, selecting an 8 mm AVP 4 (Abbott) which was implanted in the distal pelvic portion of the vein (Fig. 4).

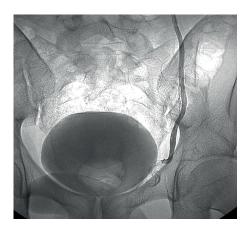
Post-procedural venography shows absence of filling of the varicocele (Fig. 5).



■ Figure 3. Phlebography of the left spermatic vein.

Dilation of the spermatic vein and pampiniform plexus.

Figure 4. Placement of an 8-mm AVP 4.



■ Figure 5. Final check. No filling of varicocele

The patient was followed-up at the urology clinic and control ultrasonography was performed after six months, showing absence of varicocele and disappearance of clinical signs.

VARICOCELE

COMMENTS

Varicocele is the varicose dilatation of the spermatic vein and the pampiniform plexus. It arises as a result of a malfunction or absence of the semilunar valves of the spermatic vein, being more common on the left side (10:1). It affects 15% of the male population and up to 40% of sterile men. The most affected age group is 15-35 years of age.

Clinically, it usually presents as testicular pain or discomfort and even testicular atrophy. Diagnosis is confirmed by Doppler ultrasound, where venous flow, direction, resistance index, and even testicular volume can also be assessed.

Treatment may be surgical (ligation of the spermatic vessels) or through selective percutaneous embolisation. According to different series, the recurrence rate is lower with embolisation (5%) than with surgical treatment (5-20%). In our case, since no collaterals were found, we decided to perform embolisation using only an AVP 4, obtaining a complete occlusion of the varicocele.

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AMPLATZER VASCULAR PLUG IN VARICOCELE AND PELVIC CONGESTION SYNDROME

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CASE 1

MEDICAL HISTORY

29-year-old male with no relevant history. For several months he has reported persistent left scrotal tenderness. A testicular Doppler US confirmed grade III left varicocele. Embolisation and phlebosclerosis of the gonadal vein was performed with cyanoacrylate monomer, (Glubran®, GEM), aided by an Amplatzer vascular occluder (Abbott). Six months later, the patient was asymptomatic, and the Doppler US evidenced resolution of the varicocele.

DESCRIPTION OF THE TECHNIQUE

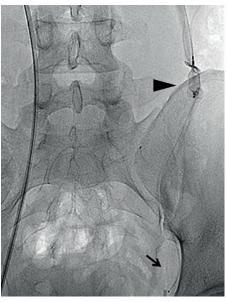
Outpatient procedure under conscious sedation and antibiotic prophylaxis. Access from the right common femoral vein and catheterisation of the left gonadal vein which was insufficient (Fig. 1). An 8-F 70 cm long sheath (Flexor®, Cook) was advanced into the middle third of the gonadal vein through which a 0.021" microcatheter (Renegade STC, Boston Scientific) and a 12-mm AVP II occluder were introduced in parallel. The microcatheter tip was positioned in the most distal part of the gonadal vein, and AVP II expanded at the level of the iliac crest (Fig. 2).

3 cc of cyanoacrylate emulsified with 3 cc of lipiodol (50% formula) were injected through the microcatheter to fill the gonadal vein and its collaterals while pulling back the microcatheter to the occluder where the cyanoacrylate column would be stopped (Fig. 3). The microcatheter was removed across the vascular plug without any problem and then the plug was released.

AMPLATZER VASCULAR PLUG IN VARICOCELE AND PELVIC CONGESTION SYNDROME



■ Figure 1. Gonadal venography. Thick, avalvulated vein. Marked reverse flow on Valsalva. Venous insufficiency confirmation. Duplicate vein with collaterals can be seen in the lower part of the gonadal vein (arrow)



■ Figure 2. AVP II expanded and unreleased in parallel with the microcatheter (arrowhead). Microcatheter tip in the most distal part of the gonadal vein (arrow).



■ Figure 3. Final image. Cyanoacrylate column filling gonadal vein and stopped by AVP II (arrow). Microcatheter removed without problems.

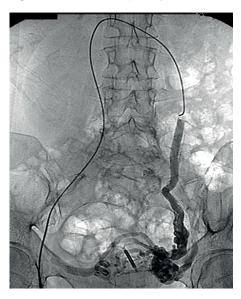
CASE 2

MEDICAL HISTORY

42-year-old woman with chronic pelvic pain that began years before. An abdominal CT was performed for another reason, and thick pelvic varices were diagnosed. The patient was sent to Vascular Radiology for assessment and treatment. Embolisation of pelvic varices was performed by injection with cyanoacrylate aided by an Amplatzer occluder. One year later she is asymptomatic.

DESCRIPTION OF THE TECHNIQUE

Conscious sedation and antibiotic prophylaxis. Approach from the right common femoral vein. Left gonadal venography with 4-F Cobra catheter (GlideCath® Terumo) confirming venous insufficiency and periuterine varices (Fig. 4).

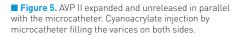


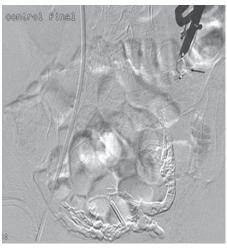
■ Figure 4. Left ovarian venography. Large and insufficient vessel. Refilling of left and right periuterine varices. Leak to left hypogastric vein.

To achieve bilateral embolisation and to ensure that cyanoacrylate diffuses from the left-side to the right-side varices, we diluted it to 33%. In addition, immediately before injection, we abundantly washed the target vessels with 5% glucose. With these manoeuvres, polymerisation would be delayed allowing cyanoacrylate to diffuse into the desired areas. However, these manoeuvres could also cause migration through the gonadal vein. To resolve this problem, a long 8-F sheath was placed in the gonadal vein and a microcatheter was inserted in parallel, the end of which was within the varices, and a 10 mm AVP II occluder was expanded, sealing the distal portion of the vein (Fig. 5). Once the microcatheter was removed, AVP II was released to consolidate the embolisation treatment. Control venography with good outcome (Fig. 6).

AMPLATZER VASCULAR PLUG IN VARICOCELE AND PELVIC CONGESTION SYNDROME







■ Figure 6. Final control from the introducer. Bilateral pelvic distribution of cyanoacrylate. AVP II occluding the ovarian vein (arrow).

COMMENTS

Varicocele in males and pelvic congestion syndrome in females originate from gonadal vein insufficiency and may cause chronic, scrotal or hypogastric pain, depending on sex. In males, it may also be associated with decreased fertility.

Embolisation therapy is currently the first choice for symptomatic patients. Embolisation with coils is the treatment commonly used. However, recent studies have shown that embolisation with liquid agents, which, in addition to the embolic effect, produce also vein sclerosis, are more effective. Fluids and foams have the advantage of distributing and treating not only the main vein but also collaterals that often cause late relapse. The potential risk of liquid and foam agents is their migration causing non-target embolisations. In our experience, when the gonadal vein is large and requires a significant volume of sclerosing fluid, nitinol vascular plugs have been very effective in blocking the origin of the vein during fluid injection. This technique allows us to perform the procedure with the reassurance that the embolising/sclerosing agent cannot migrate outside the gonadal vein. We may increase the volume of sclerosing agent used and also increase its contact time with the venous endothelium. We have had no problems of microcatheter entrapment between the vein and the occluder in any of our cases.

AMPLATZER VASCULAR PLUG IN VARICOCELE AND PELVIC CONGESTION SYNDROME

- 1. Urbano J, Cabrera M, Alonso-Burgos A. Sclerosis and varicocele embolization with n-butyl cyanoacrylate: experience in 41 patients. Acta Radiol. 2014; 55:179-85.
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CLOSURE OF HEMODIALYSIS ARTERIOVENOUS FISTULA WITH "STEAL SYNDROME"

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MEDICAL HISTORY

54-year-old patient with a history of hemodialysis AVF in the left upper extremity, which was virtually unable to be used due to puncture difficulties, which was also suspected a "steal syndrome" due to the presence of coldness, pallor, paraesthesia and pain in fingers.

DESCRIPTION OF THE TECHNIQUE

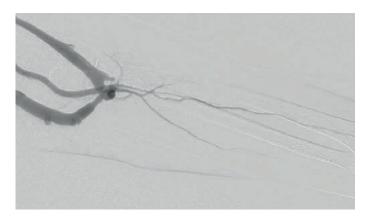
A study of the hemodialysis AVF was initially performed by direct puncture of the basilic vein, with basal series and with compression by blood pressure cuff.

The first series showed filling of both cephalic and basilic veins with no abnormalities, and no changes were seen either in the axillary or subclavian veins, the left brachiocephalic venous trunk or the superior vena cava. In the series performed by cuff compressor, which was inflated above patient's systolic pressure, the arteriovenous anastomosis was adequately filled, evidencing a very decreased flow in the forearm and hand, with very dim opacification of both radial and ulnar arteries (Figs. 1 and 2).

Subsequently, in order to adequately assess distal arterial flow, brachial artery catheterisation was performed (Fig. 3) which adequately showed the artery (A), anastomosis (An), drainage vein (V) and filling of both basilic (Ba) and cephalic (Ce) veins. The particular interest is that the arterial flow travel mainly to the AVF and, minimally, to the distal arterial territory, which is known as "steal syndrome".

Given the reported clinical manifestations of the patient and the non-functional nature of the hemodialysis AVF, it was decided by consensus with the Nephrology Department to close it.

CLOSURE OF HEMODIALYSIS ARTERIOVENOUS FISTULA WITH "STEAL SYNDROME"



■ Figure 1. Hemodialysis AVF study by direct puncture of the basilic vein. Compression with a blood presume cuff inflated above systolic pressure.

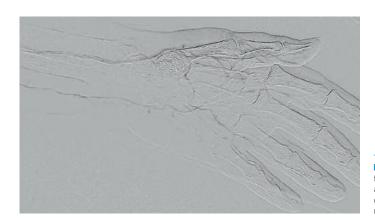
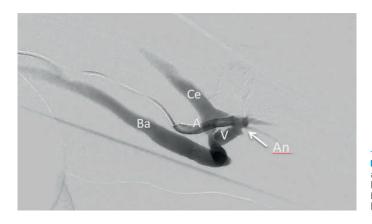


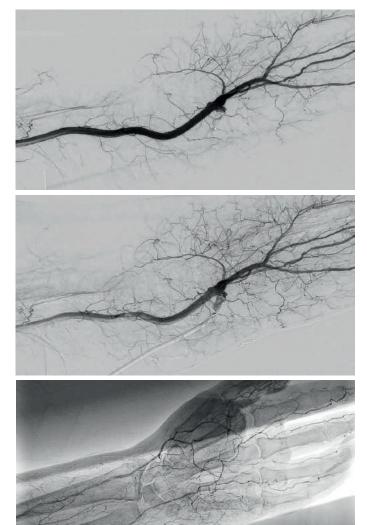
Figure 2. Very low filling of the distal arterial bed with very dim opacification of the radial and ulnar arteries.



■ Figure 3. Humeral artery (A), Anastomosis (An), drainage vein (V), basilic (Ba) and cephalic (Ce) veins.

CLOSURE OF HEMODIALYSIS ARTERIOVENOUS FISTULA WITH "STEAL SYNDROME"

After accessing the basilic vein, an angled hydrophilic guidewire was inserted and a 5-F introducer placed over it. A short 5-F Berenstein catheter was used for the passage through the anastomosis to the distal arterial territory. The catheter was removed and the introducer was advanced into the drainage vein (V) of the AVF. A 10 mm AVP vascular occluder (Abbott) was selected, which was passed through the introducer, verifying its adequate positioning using both venous and arterial injection of contrast medium, and subsequently deploying it by removing the introducer. Finally, after re-checking, the occluder was released by counter-clockwise rotation of the fixation system.



■ Figures 4, 5 and 6.

Arteriography confirming closure of the AVF, with vascular occluder closing the drainage vein and marked increase in distal arterial vascularization.

CLOSURE OF HEMODIALYSIS ARTERIOVENOUS FISTULA WITH "STEAL SYNDROME"

For this type of device, a few minutes are needed before the final verification is done, which is performed by arteriography (Figs. 4, 5 and 6), where it can see the complete closure of the hemodialysis AVF and a marked increase in distal arterial vascularization. The patient improved since the start of closure, with immediate disappearance of the paraesthesias.

COMMENTS

"Steal syndrome" in hemodialysis AVF is due to the "excessive" flow passage through the surgical anastomosis into the drainage vein, leading to a decrease in this flow to the distal arterial bed, resulting in coldness, paraesthesia, pain and even necrotic lesions in cases of severe ischaemia.

The classical treatment was surgical closure of the AVF. We present, as an alternative option, closure of the drainage vein using a vascular plug with an excellent result and immediate disappearance of the clinical manifestations of the patient.

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HEMODIALYSIS VASCULAR ACCESS RESCUE

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MEDICAL HISTORY

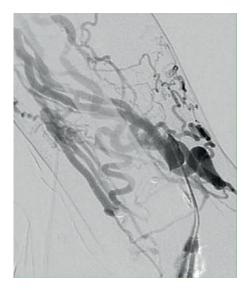
47-year-old woman on hemodialysis. Prosthetic vascular access with 6 mm left brachio-axillary Gore-Tex performed 68 months before. When attending her regular HD session, the access was thrombosed. Physical examination revealed a beat in the first centimetres of the graft and palpable aneurysmal dilatation located at approximately 10 cm of the AV anastomosis. From this point on, the graft has no pulse or thrill. The vascular radiologist on call was requested for access repair.

DESCRIPTION OF THE TECHNIQUE

Direct puncture venography at the most proximal end of the graft with the following findings. Patency of the proximal third of access with thrombosis and obstruction of the distal two thirds. Focal rupture of the Gore-Tex in its proximal third and formation of a pseudoaneurysm that had spontaneously fistulised over two large veins located adjacent to it, through which all drainage of fistula flow with the formation of extensive venous collaterals in the left arm (Figs. 1 to 3). The left axillary-subclavian vein and central veins were patent and normal.

A catheter was advanced to the distal third of the graft (thrombosed) and venography was performed confirming a focal critical stenosis in the venous anastomosis. An angioplasty was first performed with Armada® 0.035^{TM} balloons (Abbott Vascular) of 6 and 8 mm by 4 cm x 80 cm, with complete resolution of the stenosis. Then mechanical thrombectomy was performed: first, with an Arrow-Trerotola® (TeleflexTM) anterograde device. Secondly, from a retrograde approach, through the right common femoral vein, we reached the left humeral artery through the access and inserted a 260 cm Amplatz guidewire. Over the guide, a low-pressure inflated balloon was displaced three times from the brachial artery to the axillary vein, with full-access pulse recovered. This manoeuvre was aided by a 90 cm 6-F sheath (Brite Tip, Cordis) and using a 6 mm x 2 cm x 135 mm Armada (Abbott VascularTM) balloon. Control venography showed recanalization of the vascular access but persistence of large collateral veins (Figs. 4 and 2).

HEMODIALYSIS VASCULAR ACCESS RESCUE



Graft drainage through mative collaterals

AVP 4.

Graft drainage through mative collaterals

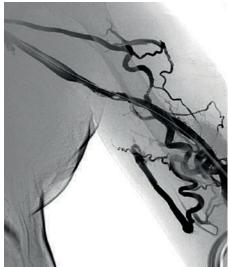
Fatent proximal graft thumbrail drain through collaterals

■ Figure 1. Fistulography from a 18-gauge needle inserted into the proximal lumen of the Gore-Tex. Only collateral veins are seen. Thrombosed access.

■ Figure 2. Illustrative scheme of fistulography findings.



■ Figure 3. Guide through the thrombosed access (arrows). 4-F Berestein catheter labelled on one of the collaterals.



■ Figure 4. PTA on the venous anastomosis and effective percutaneous thrombectomy was performed. Vascular access now is patent but with great steal due to collaterals.

HEMODIALYSIS VASCULAR ACCESS RESCUE

Third, collaterals were closed at origin. Using a 40 cm 4-F Berestein catheter with 0.035" lumen (Soft-Vu® Angiodynamics), we advanced through the rupture of the Gore-Tex, to the origin of each of the collateral veins, deploying and releasing two 6 mm AVP 4 nitinol plugs (Abbott) that in a few minutes caused complete collateral occlusion and stopped the access steal (Fig. 5).

Procedure under conscious sedation and under local anaesthesia. Heparin or thrombolitycs were not used as standard practice at our centre. The patient was dialysed normally through the recently repaired access. It was stressed that it was compulsory not to puncture the graft in the pseudoaneurysm area for six weeks.

Fifteen months later the access required PTA of the venous end. Besides this, the access remains patent, with normal function and with completely resolved pseudoaneurysm rupture (Fig. 6).



■ Figure 5. Guide wire across the recanalised graft. 4-F Berestein released in one of the collaterals with AVP 4 deployed but not released (red arrow). AVP 4 released in the other collateral (yellow arrow).

Figure 6. Control at 15 months. Occluder details.

COMMENTS

Hemodialysis grafts are very common today. Over time, they develop hyperplasia in the anastomosis with the axillary vein causing a progressive stenosis that, if an angioplasty is not performed in time, eventually triggers thrombosis and failure of the vascular access. The reported case is unusual, since spontaneous fistulisation between focal Gore-Tex rupture and native veins in the patient's arm (presumably caused by repetitive punctures) allowed for maintaining the outflow of the fistula and thus prevented its complete thrombosis. It was necessary to understand what was happening to decide to perform thrombectomy. Once percutaneous thrombectomy have been effective, anomalous connection between the graft and the collateral veins were causing a severe steal syndrome and hemodialysis access dysfunction. The problem was fixed with two AVP 4 plugs. Placement of a coaxial coated stent to repair the hemodialysis graft rupture while sealing the collateral leak was not considered in this particular case because of the need for thrombectomy with Trerotola® and balloon dragging.

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