

“Liver or Let Die”: Percutaneous PFO Closure Through Hepatic Vein Access



Julia Stehli, MD^{a,1}, Michael Michail, MBBS^{a,b,1},
David McGaw, MBBS PhD^a, Richard Harper, MBBS^{a*}

^aDepartment of Cardiology, Monash Health Melbourne, Melbourne, Vic, Australia

^bInstitute of Cardiovascular Science, University College London, London, United Kingdom

Received 8 January 2019; accepted 2 May 2019; online published-ahead-of-print 24 May 2019

A 73-year-old farmer presented with platypnoea-orthodeoxia syndrome (POS). A transoesophageal echocardiogram (TOE) disclosed a patent foramen ovale (PFO) with significant right-to-left shunt on assuming upright posture. An initial attempt at PFO closure through the femoral vein was abandoned due to a completely occluded inferior vena cava. A second attempt through the internal jugular vein was also unsuccessful due to the steep angulation between superior vena cava and septum primum flap. Because of disabling symptoms, an attempt through a hepatic vein (HV) was scheduled and performed under general anaesthesia with TOE guidance. Ultrasound-guided access through an intercostal window to a peripheral HV was performed and the position confirmed with contrast injections. The PFO was easily crossed with a glide wire which was exchanged to a stiffer guide wire. A 25 mm closure device was successfully deployed across the PFO. After retrieval of the delivery system, haemostasis of the HV was attained with a contrast-guided Gelfoam (Pfizer, New York, NY, USA) injection. Unfortunately, the patient had to undergo subsequent emergency coiling to an iatrogenically injured hepatic artery branch leading to full recovery and significant clinical improvement. Subsequent echocardiography demonstrated a well-positioned device with no residual shunt.

This case illustrates that percutaneous PFO closure through a HV is a feasible procedure and should be considered in anatomy that is otherwise prohibitive for conventional approach. Extra care should be taken with initial vascular access into the HV and final haemostasis of the access site.

Keywords

Patent foramen ovale • Percutaneous closure • Alternative access

Case Presentation

A 73-year-old male farmer presented with dyspnoea when sitting up or standing from a recumbent position which increased with minimal exertion. His medical history included a large hiatus hernia and pulmonary embolism in 2016 following which he had an inferior vena cava (IVC) filter insertion due to recurrent bleeding on anticoagulation. His IVC filter was subsequently removed. Due to the extent of his breathlessness, he underwent several investigations, including chest computed tomography (CT) and right heart catheterisation, which were unremarkable. A TOE with bubble study revealed a

patent foramen ovale (PFO) with significant right-to-left shunt on assuming upright posture, leading to a POS diagnosis. This was confirmed with tilt table testing which demonstrated deoxygenation upon inclination.

A percutaneous PFO closure via the right femoral vein was unsuccessful due to complete IVC occlusion. In an attempt via the right internal jugular vein, the device delivery system could not be manoeuvred through the PFO due to the steep angulation between the superior vena cava and septum primum flap, which resulted in recurrent wire prolapse. Since the patient had ongoing debilitating symptoms but declined surgery, a PFO closure via the HV was scheduled.

*Corresponding author at: Director Emeritus Monash Heart, 246 Clayton Road, Clayton, VIC 3168, Australia., Email: richard.harper@monash.edu

¹Shared first authorship.

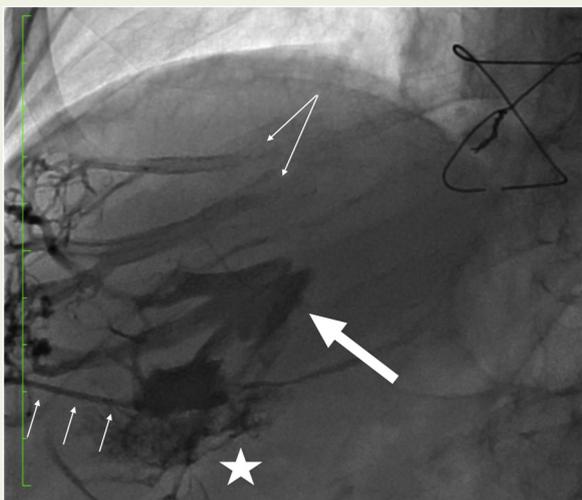


Figure 1 Inadvertent sheath (multiple small arrows) insertion into the portal vein (bold arrow), draining into the hepatic vein (thinner arrows), causing perforation with extravasation of contrast (star).

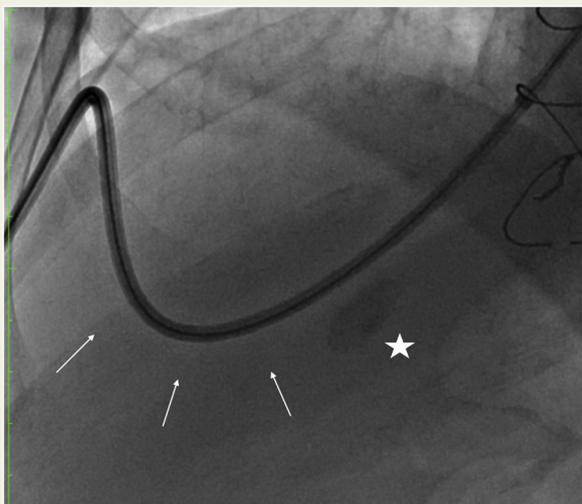


Figure 2 Bending of the delivery system upon passage through the hepatic vein (small arrows).

The procedure was undertaken under general anaesthesia with TOE guidance. Ultrasound guided vascular access was performed through an intercostal window with a micropuncture needle, over which contrast was injected to confirm position in a HV. However, despite appropriate diligence, the initial vascular access attempt resulted in the inadvertent insertion of a 6 F sheath into a portal vein (Figure 1, Video 1). Hepatic vein access was successfully attained on second attempt and 5,000 IU of unfractionated heparin was administered. The PFO was traversed with ease with a Terumo Glidewire (Terumo, Tokyo, Japan) directed by a JR4 catheter. The Glidewire was subsequently exchanged for a standard 0.035" wire once the catheter was placed in the pulmonary

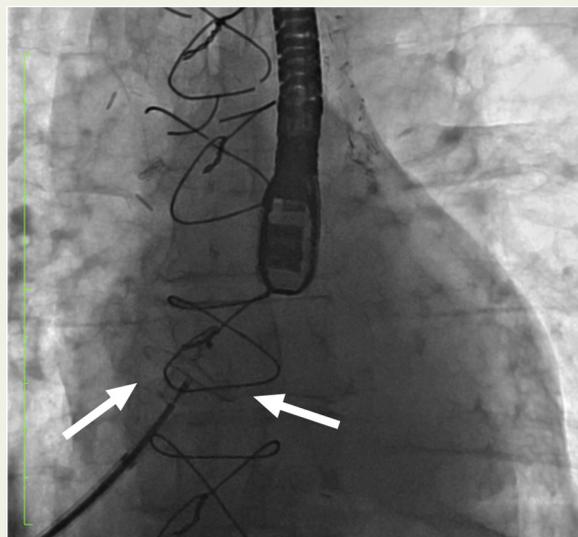


Figure 3 25 mm Amplatzer Occluder device (arrows) across patent foramen ovale (PFO).

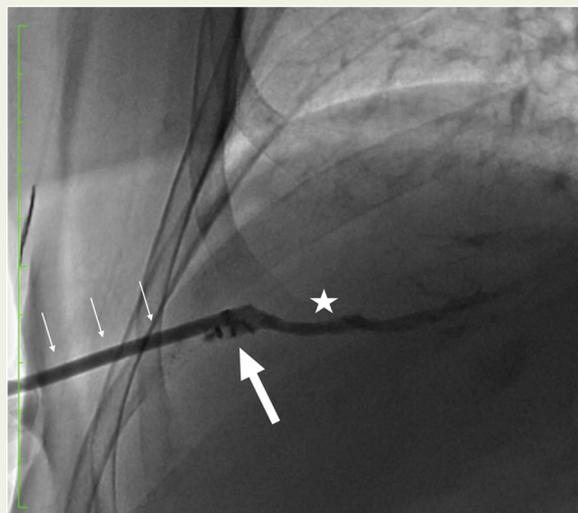


Figure 4 Withdrawal of the delivery system (multiple small arrows) from the hepatic vein branch (star) with simultaneous Gelfoam injection (bold arrow).

vein. Due to difficulties in advancing the 8F Amplatzer delivery system (Abbott, Abbott Park, IL, USA) through the liver (Figure 2) the wire was further exchanged for a 0.035" Amplatzer Superstiff guidewire (Boston Scientific, Marlborough, MA, USA) and then to a Lunderquist Extra-Stiff guidewire (Cook Medical, Bloomington, IN, USA). The delivery system was then advanced slowly along the guidewire and positioned in the left atrium. A 25 mm Amplatzer PFO Occluder device (Abbott) was deployed across the PFO with no notable issues (Figure 3). Following device release, the delivery system was withdrawn back into the HV. Haemostasis was attained by contrast-guided Gelfoam injection

(Pfizer, NY, USA) contemporaneously to the delivery system being withdrawn (Figure 4, Video 2 and 3).

Within an hour of returning to the ward, the patient became hypotensive and tachycardic. Urgent abdominal CT demonstrated a subcapsular collection of blood with active bleeding on arterial and portal venous phase imaging, suggestive of a hepatic arterial injury. The patient was subsequently taken for emergency coiling. Injection of contrast through a micro catheter confirmed persistent bleeding from a hepatic arterial branch, over which coils were placed, resulting in complete haemostasis.

The patient required blood transfusion but made a full recovery and demonstrated significant clinical improvement. Subsequent echocardiography showed no residual shunt. The patient was discharged after 48 hours and was well at 3-month follow-up.

Discussion

This case illustrates two important points: Firstly, it serves as a reminder that POS should be considered as a differential diagnosis for unexplained dyspnoea in the context of a PFO. Secondly, percutaneous PFO closure through a HV is a feasible, albeit higher risk alternative when the more conventional femoral or internal jugular vein approaches are not possible.

Platypnoea-orthodeoxia syndrome is characterised by right-to-left shunting of deoxygenated blood across a PFO when changing from a recumbent to an upright position, leading to hypoxaemia [1]. It is a rare, but disabling condition which is difficult to diagnose and therefore likely under-recognised. Patients often undergo numerous investigations before a diagnosis is made. This is best achieved with echocardiography with bubble study, left pulmonary venous sampling through the PFO or tilt table testing with simultaneous oxygen measurements [2].

Patent foramen ovale closure in POS prevents right-to-left shunting of deoxygenated blood across the interatrial septum. The evidence is scanty, however, such patients gain significant symptomatic relief from a condition which can otherwise cause debilitating symptoms in approximately 90% of cases [2].

The procedure is conventionally performed percutaneously via femoral venous access. The internal jugular vein can be used as alternative access although this is often procedurally challenging since the PFO septum primum flap is so angled as to favour an inferior approach. The hepatic venous approach, instead, provides almost direct access to the PFO at a favourable angle for passage of the delivery system. The major disadvantage, as illustrated here, is the

increased risk for access site bleeding at a site where direct compression is not possible. Ultrasound guidance and the use of micropuncture needles can reduce the likelihood of such complications. In our case, more accurate interpretation of the contrast injections through the micropuncture needle may have avoided the inadvertent sheath insertion into the portal vein. This lies in close proximity to the hepatic artery (Glisson's triad), which must have also been iatrogenically injured and the likely cause for the contrast extravasation and subcapsular collection of blood. Consideration should have been given to the use of coiling for the inadvertent portal vein puncture prior to withdrawing the sheath. Additionally, we advocate considering the use of upfront femoral or radial arterial access to facilitate subtraction angiography of the hepatic arterial system at the end of the procedure to confirm no active extravasation. Importantly, surgical PFO closure should always remain a consideration when the anatomy is difficult and procedural risk is accentuated.

Conclusion

Patent foramen ovale closure through a HV may be considered in anatomy that is otherwise prohibitive for a conventional approach. The transhepatic approach is feasible but meticulous attention to detail is required to minimise the risk of access site complications.

Acknowledgement

We would like to acknowledge Dr James Burnes for his essential role in the clinical care of this patient.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.hlc.2019.05.096>.

References

- [1] Saver JL, Carroll JD, Thaler DE, Smalling RW, MacDonald LA, Marks DS, Tirschwell DL. Long-term outcomes of patent foramen ovale closure or medical therapy after stroke. *N Engl J Med* 2017;377(11):1022–32.
- [2] Shah AH, Osten M, Leventhal A, Bach Y, Yoo D, Mansour D, Benson L, Wilson WM, Horlick E. Percutaneous intervention to treat platypnea-orthodeoxia syndrome: the Toronto experience. *JACC Cardiovasc Interv* 2016;9(18):1928–38.