International Journal of Gastrointestinal Intervention

journal homepage: www.ijgii.org

Case Report

Transhepatic glue injection for hepatic pseudoaneurysm and arteriohepatic venous fistula after the failure of transarterial embolization

Thanh Dung Le¹, Minh Duc Nguyen^{2,3}, and Van Sy Than^{1,*}

ABSTRACT

The liver is the most frequently injured organ during abdominal trauma. Vascular injuries account for approximately 3% to 25% of all cases of liver trauma and are typically treated by transarterial embolization (TAE). We describe a case of American Association for the Surgery of Trauma grade V liver injury with a very large pseudoaneurysm and an arteriohepatic venous fistula (AHVF), which failed to respond to TAE. This case was successfully treated using a direct transhepatic glue injection without complications. AHVF is an exceedingly rare type of vascular injury in blunt trauma. Direct transhepatic glue injections could represent a reliable alternative to TAE for the treatment of visceral pseudoaneurysms, especially if TAE fails to eliminate the lesion.

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Keywords: Abdominal injuries; Arteriovenous fistula; False aneurysm; Hemoperitoneum; Rupture

Introduction

Liver injuries are the most frequent type of abdominal trauma, and more than 50% of cases are classified as American Association for the Surgery of Trauma (AAST) high-grade injuries (III, IV, and V).¹ High-grade hepatic injuries often include liver vascular injuries, such as pseudoaneurysm (PSA), arterial-venous fistula, and contrast extravasation due to damaged blood vessel walls, typically requiring interventional treatment.^{2–8}

Transarterial embolization (TAE) is the current standard treatment for vascular injuries, with a high success rate and low morbidity.^{6,9–12} The percutaneous direct injection technique has been usually used to treat vascular malformations and in some specific cases, vascular injuries in the head, neck, and limbs,^{13–16} but it has rarely been applied to visceral vascular lesions.^{17–19} We describe a case of grade V liver injury associated with a very large PSA and an arteriohepatic venous fistula (AHVF), which was successfully treated by injecting glue through the hepatic parenchyma into the

PSA following endovascular treatment failure.

Case Report

An 18-year-old male patient was transferred to our institution due to severe abdominal pain following a motorcycle accident. On arrival, the patient was conscious but pale. An examination revealed a pulse rate of 110 bpm and blood pressure of 80/50 mmHg, both of which improved after fluid resuscitation. Whole-body multislice computed tomography showed a right hepatic trauma, classified as AAST grade V, with several lacerations and a large intraparenchymal hematoma measuring 10 cm × 13 cm. A very large PSA was observed, extending from a branch of the anterior segmental artery, measuring 3.2 cm × 5.5 cm, and an AHVF was identified connecting the PSA with the right hepatic vein (Fig. 1). Moderate hemoperitoneum was observed. No other injuries were found at that time. The blood test results revealed the following values: white blood cells, $23 \times 10^9/L$; red blood cells, 3.62

¹Department of Radiology, Viet Duc University Hospital, Hanoi, Vietnam²Department of Radiology, Pham Ngoc Thach University of Medicine, Ho Chi Minh, Vietnam³Department of Radiology, Children's Hospital 2, Ho Chi Minh, Vietnam

Received March 25, 2021; Revised April 12, 2021; Accepted April 12, 2021

* Corresponding author. Department of Radiology, Viet Duc University Hospital, 40 Trang Thi Street, Hang Bong Ward, Hoan Kiem District, Hanoi 100000, Vietnam.
E-mail address: sy.hmu0915@gmail.com (V.S. Than).

Thanh Dung Le, Minh Duc Nguyen, and Van Sy Than contributed equally to this work as first authors.

pISSN 2636-0004 eISSN 2636-0012 <https://doi.org/10.18528/ijgii210022> This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted noncommercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

$\times 10^{12}/L$; hemoglobin, 8.6 mg/dL; hematocrit, 0.308 L/L; platelets, $54 \times 10^9/L$; prothrombin time, 54%; aspartate aminotransferase, 178 IU/L; and alanine aminotransferase, 1,118 IU/L.

The patient underwent emergency TAE to treat vascular injuries. The anterior segmental hepatic artery was embolized using a 3 mm $\times$ 50 mm fiber coil (Cirrus coil; BALT, Montmorency, France) (Fig. 2). However, on subsequent angiography, the PSA continued to display perfusion by small collaterals from the sixth subsegmental branch, which was completely embolized using a glue mixture consisting of *n*-butyl 2-cyanoacrylate (NBCA) and lipiodol (1 : 3 ratio) (Fig. 2, 3). On the next angiography, the PSA remained visible. The seventh segmental artery and the remaining anterior segmental branch were occluded using gelfoam, but the PSA could not be excluded (Fig. 3B). Therefore, we advanced an angiocath (16 G $\times$ 5.25"; Angiocath™ BD, Sandy, UT, USA) needle directly into the PSA, under ultrasonography (US) and digital subtraction angiography guidance, and a 5 mL mixture of NBCA and lipiodol (1 : 1 ratio) was injected into the PSA (Fig. 3C). The final angiography showed total PSA obliteration (Fig. 3D). Neither

nontarget vessel occlusion nor distal embolism was found. After the procedure, the patient's hemodynamics remained stable, with no signs of blood loss and no complications. Two units of blood were transfused. The intraparenchymal hematoma was drained on day 12. The patient recovered and was discharged from the hospital on day 15. At 1-month and 3-month follow-up visits, clinical examinations, liver function tests, and abdominal echography showed normal liver function, and no complications were found.

Consent for publication was obtained for the use of the patient's data included in the study during the post-withdrawal visit, as noted in the patient's paper medical record.

Discussion

Hepatic vascular injuries occur in approximately 3% to 25% of all liver trauma cases,^{1,2} and conservative management is associated with a high probability of failure. The most common hepatic vascular injuries in blunt trauma are PSA, contrast extravasation, or arterioportal venous fistula while AHVF is quite

Fig. 1. Multislice computed tomography images from the maximum intensity projection of the post-contrast phase (A, B) show a very large pseudoaneurysm of the anterior segmental hepatic artery (black arrows), and arteriohepatic venous fistula (white arrows).

Fig. 2. Angiographic images from the celiac trunk (A) and selective anterior segmental hepatic artery (B) show the pseudoaneurysm (black arrow in A) and arteriohepatic venous fistula draining into the right hepatic vein (white arrow), confirming the multislice computed tomography diagnosis. A 3 mm $\times$ 60 mm fiber coil was deployed at the neck of the pseudoaneurysm. On a post-coiling embolization angiogram (C, D), the pseudoaneurysm was still perfused via collaterals (black arrows).

Fig. 3. Selective sixth subsegmental hepatic artery (A) angiogram showing small collaterals feeding the pseudoaneurysm (black arrows). An angiogram obtained after embolizing the sixth and seventh subsegmental hepatic arteries and the other branches of the anterior segmental artery (B) illustrates the opacification of the pseudoaneurysm. (C) The pseudoaneurysm was cannulated by an angiocath 16 G needle (white arrowhead) through the hepatic parenchyma. Contrast material was injected through the needle, confirming the location of the pseudoaneurysm and arteriohepatic venous fistula (white arrow). The pseudoaneurysm was embolized using a mixture of *n*-butyl 2-cyanoacrylate and lipiodol (1:1 ratio). (D) The final angiography examination shows the complete occlusion of the pseudoaneurysm and arteriohepatic venous fistula (black arrowhead).

rare.^{20–22} We found fewer than 10 case reports about AHVF, three of which occurred in blunt trauma situations.^{20–22}

Owing to the rapid development of interventional radiology in recent decades, TAE has become the standard treatment for hepatic trauma with vascular injuries, and it is associated with minimized complications and improved treatment outcomes.^{4,5,8,10,11} Currently, primary surgery is only necessary to treat severe hepatic injury cases associated with hemodynamic instability.^{10,11,23,24}

Our patient presented with a grade V liver injury. Although the patient's hemodynamics were initially unstable, the patient responded to fluid resuscitation, enabling hemostatic treatment by interventional radiology. After nearly all branches of the right hepatic artery were occluded, the PSA continued to be fed by previously undetected collaterals branches. The broader application of embolization is associated with the risk of post-intervention complications, such as biliary ischemia, cholecystitis, or gallbladder necrosis.^{25,26} Therefore, alternative embolization approaches remain necessary to avoid complications and prevent the need for secondary surgery due to residual PSA.

Direct percutaneous embolization has been used to treat arteriovenous malformations in the head, neck, and extremities, which are difficult to access transvascularily.^{13,14,27} Visceral PSAs that can be delineated by US can be accessed percutaneously under US guidance. The literature describes the use of this technique to treat visceral aneurysms and PSAs in select circumstances associated with favorable outcomes.^{17–19,28}

Direct percutaneous embolization of vascular malformations and PSAs using gelfoam, thrombin, glue, and coils have previously been reported.^{18,19,28,29} Thrombin, also known as coagulation factor IIa, was initially used to initiate thrombosis, which is visible on ultrasound without long-term mass effects. A very high success rate of thrombin injection for treatment of peripheral PSAs was reported (up to 96%, according to Pezzullo et al.).³⁰ However, Gorski et al.¹⁷ reported a technical success rate of only 53% when using thrombin to treat 19 visceral PSAs. Large PSAs and PSAs

with wide necks were described as unfavorable factors for this technique.¹⁷ Allergic reactions and the risk of end-organ infarctions are negative factors associated with thrombin use. Gelfoam is an accessible agent with worldwide popularity, but there is a high risk of recurrence following gelfoam absorption after a few weeks. In our experience, gelfoam injections are more difficult to control compared with other materials, especially in PSAs with large necks or combined with arteriovenous fistulas (as in our case). The coil is a reliable, permanent embolization agent with a high success rate and a high safety profile, even in high-flow arteriovenous fistula cases. However, in our patient, the PSA was very large, and the necessary quantity of coils was too expensive for the patient.

NBCA is a commonly used embolic agent with many advantages, including a permanent occlusion effect, a low recurrence rate, and minimal tissue toxicity. Combining NBCA with lipiodol increases the polymerization time and allows the mixture to be visualized under fluoroscopy, improving injection control. Ideally, the glue should be injected into the PSA sac until the PSA is filled to the neck.¹⁸ In the present case, after hepatic artery embolization, the low residual flow in the AHVF allowed the use of highly concentrated glue without the risk of distal embolism via the hepatic vein. In a study by Gorski et al.,¹⁸ the use of this technique in 21 visceral PSAs was only associated with one case of minimal reflux to the parent artery (intercostal artery) without any symptoms.

However, direct percutaneous glue injection is also associated with several disadvantages. First, because the glue is a liquid agent, injection is associated with the risk of reflux into the parent artery, which can lead to downstream infarction or residual PSA. Fortunately, these effects can be minimized by the slow injection of the agent under fluoroscopic guidance. In cases of residual PSA, the procedure can be repeated quickly until the PSA sac is totally eliminated.^{19,29} Second, although this technique is performed under imaging guidance, the risk of further organ

damage remains a possibility due to the needle passing through the parenchyma. In previous reports, no needle pathway-related complications were recorded,^{18,19,29} however, further evaluations in larger patient populations remain necessary to evaluate the safety and efficacy of this method.

In conclusion, we described a case of AAST grade V liver trauma associated with a very large PSA and AHVF that could not be completely eliminated using TAE. A direct percutaneous glue injection could be a reliable and safe supplementary or alternative method for endovascular embolization, helping to avoid high-risk surgery.

Funding

None.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

ORCID

Thanh Dung Le, <https://orcid.org/0000-0001-5257-2092>
 Minh Duc Nguyen, <https://orcid.org/0000-0001-5411-1492>
 Van Sy Than, <https://orcid.org/0000-0001-5162-5631>

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