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FULL PAPER

Efficacy and safety of ultrasound guided percutaneous glue embolization in iatrogenic haemorrhagic complications of paracentesis and thoracocentesis in cirrhotic patients

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Objective: To compare the safety and efficacy of ultrasound guided percutaneous glue (*N*-butyl-2-cyanoacrylate) embolization with transarterial embolization in the management of iatrogenically injured arteries while performing paracentesis or thoracocentesis in patients with chronic liver disease.

Methods: Hospital database was searched for cirrhotic patients having abdominal/thoracic wall haemorrhage following paracentesis/thoracocentesis procedure from January 2011 to June 2016. Doppler ultrasound and/or CT angiography were used to localize the site of haemorrhage and patients were treated by transarterial embolization or ultrasound-guided percutaneous glue embolization. Technical success was defined as cessation of haemorrhage as evidenced by angiography/Doppler ultrasound and clinical success was evaluated in terms of stabilization of the vital signs without the need for further transfusion or pressors, and survival. In both groups, the time to “imaging diagnosis of haemorrhage” and “successful embolization” with the outcome was analysed.

Results: 23 cirrhotic patients had bleeding following a percutaneous procedure, 8 (Group 1) of them

underwent transarterial embolization while 15 (Group 2) underwent ultrasound-guided percutaneous glue embolization. Mean time needed for embolization in Group 1 was 41min while in Group 2 was 9min ($p < 0.001$). Technical success was achieved in all but one case requiring repeat glue embolization. Initial clinical improvement was noted in all cases but the 30-day mortality owing to all causes was not significantly different between treatment groups.

Conclusion: The study and its outcome suggest that ultrasound guided percutaneous glue embolization is a quick and effective treatment for iatrogenic haemorrhage following paracentesis/thoracocentesis in cirrhotic patients with comparable results to transarterial embolotherapy.

Advances in knowledge: This study details an innovative technique of ultrasound guided percutaneous glue embolization of the iatrogenically injured vessel in the management of active extravasation and pseudoaneurysm developing after paracentesis/thoracocentesis in patients with cirrhosis.

INTRODUCTION

Haemorrhagic complications like haemoperitoneum, haemothorax, abdominal/thoracic wall haematoma or pseudoaneurysm are not uncommon complications of paracentesis and thoracocentesis, particularly in patients with end-stage liver disease (ESLD) requiring frequent diagnostic and therapeutic paracentesis and thoracocentesis.

Although these procedures are considered safe, with less than 1% of haemorrhagic complications,

iatrogenic injury to the abdominal/thoracic wall vessels are frequently encountered in daily practice and can be potentially fatal in these cirrhotic patients with severe coagulopathy.

Transarterial embolization is considered the first line in managing these complications, however, valuable time may be lost in mobilizing the patient to the cath lab, which may lead to untoward consequences.

On the basis of a few case reports Based on few case reports describing the use of percutaneous glue (*N*-butyl-2-cyanoacrylate) embolization in abdominal wall pseudoaneurysm¹⁻⁵

We decided to explore the possibility of ultrasound-guided percutaneous glue embolization as a safe, effective and quick method for the treatment of acute vascular injuries in cirrhotic patients with active haemorrhage as compared with standard arterial embolization.

As rapid intervention and timely management of these complications are crucial in cirrhotic patients who are sensitive to even minor haemodynamic changes.

METHODS AND MATERIALS

This study was approved by the institutional review board.

Patients

This was a retrospective study where in the hospital data was searched to identify cirrhotic patients developing abdominal/thoracic wall haemorrhagic complications after paracentesis or thoracocentesis from January 2011 to June 2016. Only cirrhotic patients with iatrogenic abdominal/thoracic wall haemorrhage were included. Out of 189 cirrhotic patients who underwent paracentesis/thoracocentesis with or without ultrasound guidance, 23 patients developed active haemorrhage/progressive haematoma. Eight of these patients presented with haemodynamic instability owing to active haemorrhage requiring, volume replacement, blood transfusion and inotropic support, with or without artificial ventilation, while the rest were haemodynamically stable and presented with growing haematoma and pain at the puncture site with a fall in haemoglobin level by more than 2 gm dl⁻¹ over a 12–24 h period from pre-procedure values.

Diagnostic evaluation and treatment allocation

Initially, all patients (prior to April 2012) suspected to have arterial injury and active haemorrhage were evaluated with computed tomographic angiography (CTA) for identification of the injured artery and thereafter, transarterial embolization was performed. Later in May 2012, a patient with advanced cirrhosis had post-paracentesis haemorrhage and was too sick to be shifted for CTA. A bedside Doppler ultrasound was performed for evaluation of the active haemorrhage and the bleeding vessel could be identified. So, a bedside ultrasound-guided percutaneous glue embolization was performed in the intensive care unit (ICU) and haemostasis was achieved with stabilization of the vital signs. This successful treatment led to the formation of a new algorithm (Figure 1) for the treatment of post-paracentesis haemorrhage and any patient with suspected vascular injury was evaluated with Doppler ultrasound for 5–10 min for identification of injured vessel. If the active haemorrhage was identified, the patient underwent ultrasound guided percutaneous glue embolization, else a CTA was performed for identification of the vascular injury. Once the point of haemorrhage was identified on CTA, the patient underwent focused Doppler ultrasound of the site recognized on CTA and after identification of injured vessel on Doppler ultrasound, percutaneous glue embolization was performed.

Initially 6 patients were directly sent for CTA without bedside Doppler ultrasound; whereas, remaining 17 patients were first evaluated using bedside Doppler ultrasound for identification and localization of point of haemorrhage/injured vessel. Injured vessel was successfully identified in five patients and ultrasound-guided percutaneous glue embolization was performed without CTA. In remaining 12 patients, point of haemorrhage could not be identified on bedside Doppler and underwent CTA for the diagnosis. Active haemorrhage/pseudoaneurysm was seen on CTA in these 12 patients, so a repeat and focused Doppler ultrasound of the site of injury was done for identification and localization of the point of haemorrhage as seen on CTA. The repeat Doppler ultrasound examination in correlation with CTA was able to identify the culprit bleeding artery in 10 patients who were treated with ultrasound-guided percutaneous glue embolization while in 2 patients point of haemorrhage could not be identified even on repeat Doppler ultrasound and hence, was treated with angiographic embolization. One of the patients who underwent ultrasound-guided percutaneous glue embolization required two sessions of embolization. The first session of ultrasound-guided percutaneous glue embolization was done after localization of bleeding vessel on Doppler ultrasound as identified on CTA and no colour flow was seen in that region after the embolization and the patient had stabilization of vital signs and reduction in serum lactate level. However, the patient required repeated blood transfusions for maintaining haemoglobin level. So, a repeat Doppler ultrasound was done after 24 h of the initial embolization and a small pseudoaneurysm was identified adjacent to the site of prior embolization (this may have developed owing to incomplete embolization of pseudoaneurysm after first session) and a repeat ultrasound guided percutaneous glue embolization was performed successfully.

Inclusion criteria—Cirrhotic patients (CTP- A, B, C) with suspected iatrogenic vascular injury following paracentesis or thoracocentesis on the basis of clinical (Tachycardia, hypotension, puncture site swelling/pain) and haematological (fall in haemoglobin level by more than 2 gm dl⁻¹) parameters were included in the study.

Exclusion criteria—Patients with severe cardiac and renal dysfunction, prior history of severe allergic reactions to iodinated contrast agents, prior history of haemoperitoneum or haemothorax without a definite cause were excluded from this study.

Based on embolization technique, the patients were categorized into two groups, Group 1 consisted of patients who received transarterial embolotherapy (*n* = 8), while Group 2 consisted of patients who underwent ultrasound guided percutaneous glue embolization (*n* = 15).

In this analysis, coagulopathy was defined as having the international normalized ratio (INR) more than 1.5 and/or the platelet count less than 50,000 µl⁻¹. Patient's diagnosis and disease status (Childs status and MELD score) are detailed in Table 1.

Figure 1. Algorithm showing management protocol for iatrogenic haemorrhage post-paracentesis/thoracocentesis.

Procedure technique

Transarterial embolization

For an abdominal wall haemorrhage, contralateral femoral arterial access was obtained and 5F cobra-2 catheter (Cook, Inc., Bloomington, IN) was used to cross to the opposite side. The catheter was advanced and placed into the external iliac artery and 2.7F Progreat microcatheter (Terumo Corp, Tokyo, Japan) was used to cannulate inferior epigastric artery and deep circumflex iliac artery after identifying the bleeding branch, embolization was done using embolization coils or glue mixed with Lipiodol in the ratio of 1:2. Similarly, right femoral access was obtained for thoracic wall haemorrhage and the injured intercostal artery was identified and coil embolization on either side of the injured vessel was done.

Ultrasound guided percutaneous glue embolization

A detailed and careful examination of the abdominal/thoracic wall was performed using a high frequency linear array ultrasound probe (5–12 MHz, Xario, Toshiba Medical Systems

Corporation, Otawara, Japan), revealed a pseudoaneurysm on greyscale and Doppler within the abdominal/thoracic wall or as a bleeding point identified by continuous stream of echoes leaking from the peritoneal/pleural surface puncture site into the ascites on greyscale which is demonstrated as focal colour spill into the peritoneal/thoracic cavity on Doppler evaluation. (Figures 2–4). Doppler evaluation to demonstrate colour spill was carried out at a low PRF setting of 1–5 kHz and colour gain of 20–30 dB.

A 23G spinal needle was used to target the injured vessel on ultrasound and the tip of the needle was placed exactly at the point leading to the colour spill from abdominal/thoracic wall into the peritoneal/pleural cavity. In case of pseudoaneurysm, the needle tip was positioned within the aneurysmal sac. Once the needle was in position, the embolizing agent [glue (*N*-butyl-2-cyanoacrylate) mixed with Lipiodol (Guerbet, Paris, France) in the ratio of 1:1] was injected in small aliquots (0.5 ml) till the time haemostasis was achieved. The volume of glue

Table 1. Diagnosis, disease score, haemodynamic status, comparison of time needed for the identification of haemorrhage and time needed for embolization and status at 30 days

Group	Pt no	Diagnosis	Disease score		Haemodynamic status	Time needed for the identification of haemorrhage (minute)			Mean time needed for identification of haemorrhage	Time interval between identification of haemorrhage and embolization (minute)			Mean time needed for embolization	Status at 30 days
			CTP	MELD		Doppler ultrasound	CTA (time includes shifting to CT suite)	Total time		Time needed for repeat Doppler ultrasound	Time needed for embolization	Total time		
1	1	ACLF-ethanol related	13	33	Compromised	Not done	60	60	58 min	Not done	30	30	37 min	Live and discharged
	2	Ethanol-related CLD	12	36	Compromised	Not done	30	30		Not done	28	28		Dead
	3	Cryptogenic CLD	14	18	Stable	Not done	90	90		Not done	32	32		Live and discharged
	4	ACLF-ATT induced	9	24	Stable	Not done	45	45		Not done	30	30		Live and discharged
	5	ACLF cryptogenic	14	29	Compromised	Not done	45	45		Not done	40	40		Dead 9 days later
	6	Ethanol-related CLD	11	34	Stable	Not done	60	60		Not done	50	50		Live and discharged
	7	Autoimmune-related CLD	13	39	Stable	6	90	96		5	35	40		Dead
	8	Hepatitis B-related CLD	12	18	Stable	7	30	37		6	40	46		Live and discharged
2	1	Cryptogenic CLD	12	19	Compromised	5	Not done	5	41 min	Not done	4	4	9 min	LDLT
	2	Ethanol-related CLD	12	36	Compromised	6	Not done	6		Not done	8	8		Live and discharged
	3	ACLF-Ethanol related	14	33	Compromised	8	60	68		5	3	8		Dead
	4	ACLF-cryptogenic	13	33	Compromised	5	90	95		6	3	9		Dead 15 days later

(Continued)

Table 1. (Continued)

Group	Pt no	Diagnosis	Disease score		Haemodynamic status	Time needed for the identification of haemorrhage (minute)			Mean time needed for identification of haemorrhage	Time interval between identification of haemorrhage and embolization (minute)			Mean time needed for embolization	Status at 30 days
			CTP	MELD		Doppler ultrasound	CTA (time includes shifting to CT suite)	Total time		Time needed for repeat Doppler ultrasound	Time needed for embolization	Total time		
2	5	ACLF-ethanol related	15	43	Compromised	7	45	52	41 min	6	4	10	9 min	Dead
	6	ACLF-ATT induced	12	26	Compromised	6	Not done	6		Not done	5	5		Live and discharged
	7	Ethanol-related CLD	11	27	Compromised	6	30	36		8	5	13		Live and discharged
	8	Hepatitis B-related CLD	10	21	Stable	5	90	95		7	3	10		Live and discharged
	9	Cryptogenic CLD	8	18	Stable	8	Not done	8		Not done	6	6		Live and discharged
	10	CKD with CLD	9	27	Stable	7	45	52		9	7	16		Live and discharged
	11	Hepatitis B-related CLD	10	18	Stable	5	30	35		5	5	10		Live and discharged
	12	Ethanol-related CLD	8	24	Stable	7	Not done	7		Not done	9	9		Live and discharged
	13	Hepatitis B-related CLD	11	30	Compromised	5	30	35		6	5	11		Live and discharged
	14	ACLF – hepatitis B and ethanol related	13	35	Compromised	8	45	53		6	4	10		Dead 10 days later
	15	Hepatitis B-related CLD	10	32	Stable	6	60	66		5	7	12		Live and discharged

ACLF, acute on chronic liver failure; ATT, antitubercular drug therapy; CLD, chronic liver disease; CKD, chronic kidney disease; CTA, computed tomographic angiography; CTP, Child-Turcotte-Pugh; MELD, modified end-stage liver disease.

Figure 2. CTA image (a) axial and (b) coronal showing large pelvic haematoma with active contrast extravasation (white arrows). Ultrasound Doppler images (c) showing arterial waveform from the bleeding vessel, (d) showing closure of the active leak. Post-procedure non-contrast CT image (e) showing Lipiodol cast (black arrow) at injection site. CTA, computed tomographic angiography.

(with Lipiodol) injected ranged from 0.5 to 3 ml depending on the site and size of bleeder. Post glue embolization, Doppler ultrasound was performed after a time interval of 30 min to confirm the cessation of active bleeding/obliteration of the aneurysm (Figures 2–4). Follow-up Doppler ultrasound was done every 24 h for the next 2 days.

CTA and ultrasound-guided percutaneous glue embolization were performed by an interventional radiologist with over 10 years of experience.

Outcome measures

The outcome in the form of technical and clinical success was obtained from hospital data and discharge summary or by

telephonic survey. Detailed comparison was done for variables consisting of (a) the time needed for the identification of haemorrhage (time when haemorrhage suspected to imaging confirmation, using Doppler ultrasound and/or CTA), (b) time needed for embolization (time to imaging confirmation of haemorrhage and completion of embolization), (c) 30-day mortality owing to all causes in both the groups.

Endpoints

Technical success

Technical success was defined as cessation of active haemorrhage/embolization of injured vessel or pseudoaneurysm on Doppler ultrasound or angiography.

Figure 3. Doppler ultrasound image (a) showing small abdominal wall pseudoaneurysm (yin-yang colour flow—black arrow) with adjacent haematoma (b) showing needle being positioned within the pseudoaneurysm (white arrows) (c) showing complete embolization of pseudoaneurysm with no flow (white arrow heads).

Clinical success

Clinical success was evaluated in terms of stabilization of the vital signs as well as improvement in the haemoglobin, serum lactate levels of the patients and no further requirement for blood transfusion.

Statistical analysis

Data were described as proportions or median with interquartile range as required. The medians were compared in two groups using Mann–Whitney *U* test and proportions were compared by Fisher's exact test. $p < 0.05$ was considered as significant. All statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) version 22.0.

Figure 4. Doppler ultrasound image (a) showing an abdominal wall bleeder following paracentesis seen as a focal colour spill and a stream of echoes on greyscale leaking from the peritoneal surface puncture site into the ascites (white arrows). (b) Showing glue casting (white arrow heads) in the abdominal wall at the site of bleeder following percutaneous glue embolization.

RESULTS

20 patients developing active haemorrhage underwent paracentesis/thoracocentesis in ward or ICU without ultrasound guidance, while 3 patients developed haemorrhage after ultrasound-guided paracentesis/thoracocentesis. On the day of procedure, eight patients ($n = 1$, Group 1; $n = 7$, Group 2) had platelet count less than $50,000 \mu\text{l}^{-1}$. Similarly, 17 patients ($n = 5$, Group 1; $n = 12$, Group 2) had INR more than 1.5. Patients with low platelets were infused with platelet concentrate and patients with elevated INR were infused with fresh frozen plasma prior to the paracentesis/thoracocentesis.

Both the groups had patients who were haemodynamically stable ($n = 11$) and unstable ($n = 12$). Patients with compromised haemodynamic status at presentation had significant tachycardia, hypotension, tachypnea and blood gas analysis suggested of rising lactate and fall in haemoglobin. These patients required active resuscitation with blood transfusion owing to active bleeding and rapid fall in blood pressure and whenever possible were preferred for a bedside therapy in ICU (ultrasound-guided percutaneous glue embolization). Table 1 shows the comparison of the time needed for the identification of haemorrhage and time needed for embolization.

Technical success was 100% in Group 1 (8 patients—transarterial embolotherapy) and 93% in Group 2 (15 patients—ultrasound-guided percutaneous glue embolization) as 1 patient in the Group 2 required repeat percutaneous glue embolization owing to probable incomplete embolization with the first session. Complete cessation of blood flow with obliteration of the pseudoaneurysm was achieved in this patient following second session of percutaneous glue embolization.

A single session of percutaneous glue embolization was adequate to achieve technical and clinical success in rest of the 14 patients in Group 2.

Within 30 min to 2 h of embolization, all haemodynamically unstable patients showed signs of improvement (patients started responding to fluid resuscitation and blood products transfusion with reduction in inotropic support, stabilization of blood pressure and heart rate). Serum lactate levels showed a declining trend after 6 and 24 h with improvement in haemoglobin level.

Mean time of diagnosis of haemorrhage in Group 1 was 58 min as compared to 41 min in Group 2, as CTA was not needed in five cases in this group (Table 1), however, the values were not statistically significant ($p = 0.48$).

The mean time needed to embolization in our study is defined by the time interval between identification of haemorrhage by Doppler or CTA till cessation of active haemorrhage/embolization of injured vessel or pseudoaneurysm.

In our study, we found that the mean time to embolize the injured artery using angioembolization (Group 1) was 41.3 min as compared with 9.4 min with ultrasound-guided percutaneous glue embolization in Group 2 (Table 1). This difference in the time to embolize was statistically significant ($p < 0.001$).

Once the injured vessel was identified on ultrasound, percutaneous glue embolization was performed with ultrasound guidance using spinal needle and injection of glue. The procedure had an added advantage of bedside performance (owing to portability offered by ultrasound) as against the time lost in mobilizing the patient to the cath lab for angioembolization.

Mortality

18 patients had coagulopathy on the day of interventional procedure. All of them received transfusions of fresh frozen plasma and/or platelet concentrates in an attempt to correct the coagulopathy. The number of transfused units ranged from 2 to 22.

A total of 9/15 (60%) patients in Group 2 were haemodynamically compromised as compared with 3/8 (37.5%) in Group 1 ($p = 0.40$). This may be probably owing to selection bias as patients with haemodynamic compromise were preferred for bedside ultrasound guided percutaneous glue embolization.

Although the 30-day mortality in Group 2 was less (4/15, 26.7%) as compared with Group 1 (3/8, 37.5%), the same did not translate to statistical significance ($p < 0.66$).

Four patients having MELD (model for end-stage liver disease) score of over 27, developed disseminated intravascular coagulation (DIC) and multiorgan failure and died within 72 h despite successful embolization of active bleeding vessel. Three patients having acute-on-chronic liver failure, (Child C, MELD 29/33/35) died on 9th, 10th and 15th day mostly owing to liver failure complicated by sepsis. One patient underwent liver transplantation post-embolization (Table 1).

DISCUSSION

Treatment options for refractory ascites and hepatic hydrothorax include repeated large-volume paracentesis/thoracocentesis

or transjugular intrahepatic portosystemic shunts.^{6–8} Overall complication rate after paracentesis is around 1%, so it is considered to be a safe procedure.⁹ However, sometimes major haemorrhage can occur following diagnostic paracentesis/thoracocentesis.^{10–12}

Haemorrhagic complications following paracentesis/thoracocentesis can be divided into three groups: abdominal/chest wall haematoma, haemoperitoneum/haemothorax, pseudoaneurysm formation.¹³ These complications can occur alone or in combination. More often, paracentesis-related complications become symptomatic during first 6 to 48 h after the procedure, however, few cases may present with late onset of symptoms up to 1 week after paracentesis.¹⁴

Inferior epigastric artery injury is the most common iatrogenic injury in ESLD patients after paracentesis.¹⁵ Similarly, intercostal arteries are the commonest to get injured post-thoracocentesis. Active haemorrhage from injury to abdominal/chest wall vessels may lead to significant drop in haemoglobin with haemodynamic compromise and requirement of blood or blood products transfusion.¹⁵ Patients with low platelet count, Child-Pugh Stage C or significant renal dysfunction are at higher risk for paracentesis-related haemorrhage.^{16,17} All our patients were in Child-Pugh class C, barring one patient in Group 1 and three patients in Group 2. Similarly, our patients had a MELD score of 18 or more with a maximum of 43.

In our experience also, the bleeding vessel was mostly a branch vessel of inferior epigastric artery rather than the main trunk, similar to study by Sobkin et al.¹⁵ Conventionally, transarterial embolization has been frequently used to treat inferior epigastric artery injury.^{15,18–20} In the present study, two different embolization techniques, transarterial embolization and ultrasound-guided percutaneous glue embolization were performed for the treatment of injured vessel and the outcomes were compared. Ultrasound-guided percutaneous glue embolization is an effective technique for embolization of an injured blood vessel with active haemorrhage, identified on Doppler ultrasound. This technique scores over transarterial embolization as it can be performed bedside with significantly short procedure times.

A similar concept of percutaneous glue embolization has been used by Del Corso et al.² in which they have treated 93 cases of iatrogenic pseudoaneurysm in peripheral locations (femoral artery, brachial artery, radial artery and axillary artery).² Although peripheral pseudoaneurysms may cause neuropathy, venous compression or tissue necrosis, these are rarely life threatening.² As far as the concept of study is concerned, our study has similar concept, however, the difference lies in the clinical scenarios. In our study, active haemorrhage with growing haematoma/haemoperitoneum/haemothorax leading to clinical deterioration and haemodynamic instability were treated, while in the previously mentioned study peripheral pseudoaneurysm/haematoma were treated. Active haemorrhage itself becomes an emergent situation requiring urgent intervention. Furthermore, patients with cirrhosis are already a vulnerable group and any continuous bleed may turn fatal owing to low hepatic reserve. So, it is a run against time, earlier the haemorrhage

is controlled, the better the outcome. Therefore, ultrasound guided percutaneous glue injection in this setting quickly attains the haemostasis which may be lifesaving.

Another series by Sobkin et al¹⁵ included six patients with seven inferior epigastric artery injury suffering from ESLD and coagulopathy, all treated by transarterial embolization, three patients (50%) died at the follow-up of 30 days while three patients were alive. In our study, 30-day mortality for patients treated with transarterial embolization was 37%. Despite having more serious patients in Group 2, the survival was better (73 vs 63%). However, this finding could not reach statistical significance owing to smaller number of patients in each group. Further studies with larger cohort are needed to generate better evidence for the same.

Although a complete embolization was achieved with both the techniques and initial transient improvement was noted in all patients, high 30-day mortality was seen in both groups. This high mortality may be attributed to the extremely advanced liver disease associated coagulopathy as correlated by their higher MELD score (ranged from 27 to 43) and Child-Pugh class C (CTP score ranged from 11 to 15). The reason of death was DIC and multiorgan failure in four patients and liver failure complicated by sepsis in another three patients.

Ultrasound-guided percutaneous embolization offers a simple but effective treatment (with minimal hardware requirement) for iatrogenic vessel injury following paracentesis/thoracocentesis in patients with cirrhosis with an ease of bedside treatment even in ICU. Other advantages of this technique are minimal hardware requirement consisting of spinal needle, glue and lipiodol, whereas for transarterial embolization, arterial access devices along with sheaths and catheters are required with embolizing materials. Transarterial embolotherapy requires an angiography unit and administration of iodinated contrast media and exposure to ionizing radiation. At times transarterial embolization

may be technically challenging and time consuming owing to unfavourable anatomy/vasospasm.

Ultrasound-guided percutaneous glue embolization is a relatively easy procedure, similar to the several routine ultrasound-guided procedures performed by an interventional radiologist. The operator is required to place the needle tip precisely at the bleeding point under ultrasound guidance as identified on Doppler ultrasound. The possibility of incomplete percutaneous glue embolization may arise, however, this can be managed by repeating the process and achieving haemostasis. Another potential reported drawback of the use of glue is the possibility of reflux in the non-target vessels which can lead to ischaemia of the organs supplied. However, we did not encounter this complication in our study cohort.

The limitations of this study are its small patient group and inability to perform ultrasound-guided percutaneous glue embolization in cases where site of injury is not properly identified on Doppler ultrasound. Being a retrospective study, randomization was not done during allocation of groups along with selection bias. Despite these limitations, this study documents a real world scenario for diagnosis and management of haemorrhage after a paracentesis/thoracocentesis in patients with ESLD which is a rare event and conducting prospective studies may be a time taking process.

CONCLUSIONS

In this small sample, ultrasound guided percutaneous glue embolization seems a feasible option in the management of haemorrhagic complications following paracentesis/thoracocentesis with a good and comparable outcome to transarterial embolotherapy. Other advantages of percutaneous glue embolization include relative ease of bedside performance, quick to perform, no radiation exposure and no requirement of iodinated contrast.

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