



Splenic Artery Embolisation for the Emergency Treatment of Sinistral Portal Hypertension: A Systematic Review

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Abstract

Objectives Sinistral portal hypertension (SPH) is caused by increased pressure on the left portal system secondary to splenic vein stenosis or occlusion and may lead to gastric varices. The definitive management of SPH is splenectomy, but this is associated with significant mortality and morbidity in the acute setting. In this systematic review, we investigated the efficacy and safety of splenic artery embolisation (SAE) in managing refractory variceal bleeding in patients with SPH.

Methods A comprehensive literature search was conducted using MEDLINE and Embase databases. A qualitative analysis was chosen due to heterogeneity of the studies.

Results Our search yielded 339 articles, 278 of which were unique. After initial screening, 16 articles relevant to our search remained for full text review. Of these, 7 were included in the systematic review. All 7 papers were observational, 6 were retrospective. Between them they described 29 SAE procedures to control variceal bleeding. The technical success rate was 100% and there were no cases of rebleeding during follow up. The most common complication was post-embolisation syndrome. Four major complications occurred, two resulting in death. These deaths were the only 30-day mortalities recorded and were in patients with extensive comorbidities.

Conclusions Although there is a distinct lack of randomized controlled studies comparing SAE to other treatment modalities, it appears to be safe and effective in treating hemorrhage secondary to SPH.

Keywords

- ▶ sinistral portal hypertension
- ▶ splenic artery embolization
- ▶ variceal hemorrhage

Introduction

Sinistral portal hypertension (SPH), also commonly known as left-sided or segmental portal hypertension, is a rare but serious cause of upper gastrointestinal hemorrhage.^{1,2} SPH occurs as a result of splenic venous occlusion and is most commonly

characterized by isolated gastric varices.¹⁻⁴ This occurs in the absence of true portal hypertension and patients have preserved liver function.^{2,5} Several pathologies can result in splenic vein occlusion, including acute or chronic pancreatitis and extrinsic splenic vein compression. As most individuals with the condition are asymptomatic, the incidence of

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Table 1 A table to show the search terms used to perform our literature search

Exposure	AND Intervention
Sinistral	(Splenic arter* AND embol*)
Left side*	OR
Segmental	(Spleen arter* and embol*)
Pre hepatic	OR
Extra hepatic	(Splenic arter* AND inflow AND
Regional	control)
Splenoportal	OR
Local#ed	(Splenic arter* AND occlu*)
Compartmental	
Lineal	
Non cirrho*	
AND	
Portal hypertension	
AND	
Varices	
Varix	
Bleed*	
H?emorrhage	
Mele?na	
H?ematemesis	
H?ematochezia	

Abbreviation: NA, not available.

*Average for the whole cohort, not just the SAE patients.

SPH is widely thought to be underreported.^{2,5} However, Xie et al showed that SPH may affect up to 3% of patients undergoing imaging for acute pancreatitis.⁶

Upper gastrointestinal bleeding is experienced by 18–35% of patients with SPH.^{7,8} Splenectomy is the definitive treatment of symptomatic SPH patients, but it is associated with significant mortality and morbidity in the acute setting.^{8,9} Endoscopic techniques such as balloon tamponade, sclerotherapy, and band ligation may temporarily stop bleeding from varices, but have a high rate of failure.^{10–13} Other non-surgical treatments for variceal hemorrhage secondary to SPH include splenic vein recanalization and splenic artery embolisation (SAE).¹⁴ SAE reduces splenic inflow, thereby reducing perfusion pressure of the gastric varices, and reduces the risk of further hemorrhage. SAE has been used alone, or as a bridging measure to stabilize patients prior to emergency splenectomy.^{15,16} There have been various studies over the years comparing SAE to surgical and other non-surgical treatments for SPH, but the exact role of SAE remains to be fully established in this group of patients.^{5,12,13,17–21} In this systematic review, we investigate the efficacy and safety of SAE in managing refractory gastric variceal bleeding in patients with SPH.

Methods

Institutional review board approval is not required to conduct this type of study.

Literature Search

MEDLINE and Embase databases were searched up until 1st September 2020 for randomized controlled trials, pilot, cohort or case-control studies investigating the outcomes of SAE in patients with gastric variceal bleeding due to SPH.

Search terms included synonymous names for SPH, “gastric varices”, “variceal hemorrhage” and “SAE” (displayed in ►Table 1). The search algorithm retrieved 339 papers overall (see ►Figure 1). After a title and abstract screen, 262 papers were excluded due to the type of procedure or indication for intervention, paediatric or non-human populations or study design. Upon performing a full text review of the remaining 16 papers, those with insufficient data regarding the procedural success, recurrence of variceal bleeding and post procedural complications were excluded. A total of 7 papers were included in the systematic review. The 7 articles included were published between 1981 and 2014. With the exception of one prospective observational study, all the remaining articles were retrospective observational studies.

Outcomes

The primary outcomes were technical and clinical success. These were defined as cessation of splenic arterial blood flow on completion angiography and cessation of variceal bleeding, respectively. Secondary outcomes were procedural complications and mortality to assess the safety of SAE. Peri-procedural complications were classified as minor or major according to the Society of Interventional Radiology criteria.²²

Eligibility Criteria

The titles and abstracts of the publications retrieved by the search were reviewed. Studies were excluded from further review if SPH was not the exposure of interest (e.g., cirrhotic portal hypertension, splenic artery aneurysm, thrombocytopenia and splenic trauma), intervention was not SAE, or the population was not human adults. Additionally, all case reports, review articles, letters to the editor and commentaries were excluded. We performed a full text review of the remaining studies. Studies assessing the efficacy and safety of SAE as a treatment for gastric variceal bleeding due to SPH were included.

Data Extraction

From each paper included in this study, we extracted details of the study design, population, exposure of interest, SAE and outcomes. Regarding the studies themselves, we extracted: the name of the first author, year of publication, study design and whether it was a single or multi-center study. In terms of the study population, we extracted: the number of participants, age range, gender and length of follow-up. For the exposure, we extracted the etiologies of SPH and the number of participants with acute bleeding. SAE data extracted were the number of SAE procedures, embolic agent(s) used, whether failed endoscopy preceded the SAE, whether participants had a splenectomy post-SAE. Lastly, we extracted the rate of technical success, recurrence of bleeding in the SAE group and control group if present, complications and mortality post-SAE.

Data Analysis

A meta-analysis was not performed due to heterogeneity of the studies. A qualitative analysis was therefore chosen.

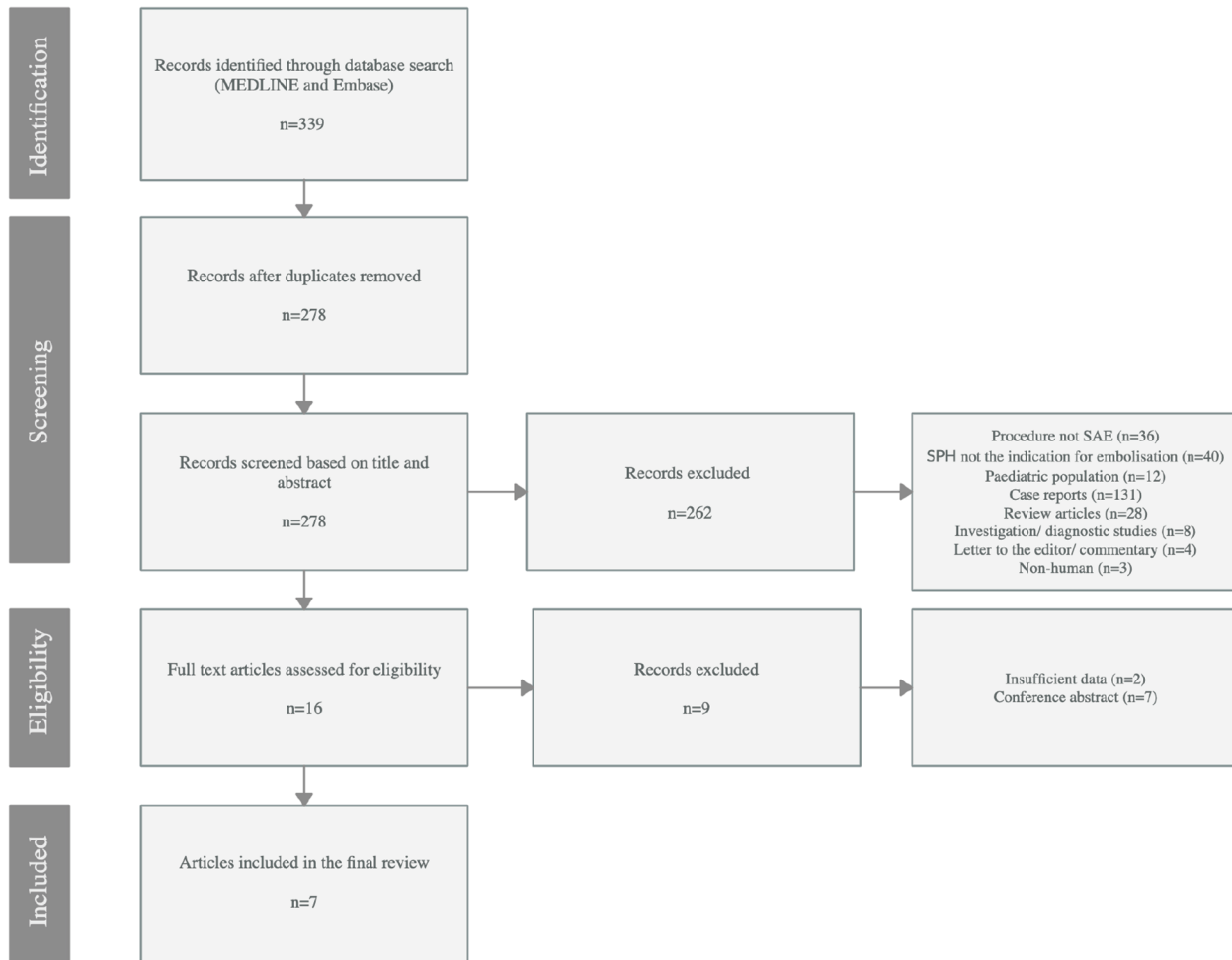


Fig. 1 Study selection flow chart.

A formal assessment of bias was not completed as all of the studies bar one were retrospective.

Results

The 7 articles included were published between 1981 and 2014. All of the articles were retrospective observational studies with the exception of one prospective observational study. The data extracted from these papers is summarized in ►Table 2. The average number of patients included in each study was 16, with an average of 4 embolisations to control variceal bleeding per study. Gender distribution was not included in one paper,²⁰ however of the remaining studies, 74% of participants were male. Follow-up periods varied considerably between the different papers, with a range of 0–14 years.

The details of patient baseline characteristics (summarized in ►Table 3) are limited, providing little insight into their clinical status at the time of intervention. Two papers described their SAE patient population to be too unwell or prevented by underlying disease from having surgery^{12,17} Goldman et al reported that each patient received an average of 12 blood transfusions in the 5 days pre- and

post-embolisation. They also clarified previous interventions, such as exploratory laparotomy in one patient, distal splenorenal shunts in 2 patients and portocaval shunts in 2 patients.

Embolisation techniques varied across the studies. Of the 29 SAE procedures, the embolisation technique / embolic agent was unknown in 7 patients. Polyvinyl alcohol particles (PVA) were used in 3 patients, and coils were used in 7 patients. Combined embolisation with PVA followed by coils was performed in 7 patients, 3 of which were completed at the same session and 4 completed 1 month apart. *N*-butyl cyanoacrylate (Bucrylate) was used in 4 patients, and a combination of an Amplatz occluder and gelfoam in one patient. Information on the proportion of the spleen embolised was limited. When using PVA particles as part of their combined embolisation, Wang et al aimed for embolisation of 60–70% of splenic volume.

The rate of technical success across all 7 studies was 100%. Coil embolisation of the celiac trunk was performed in one patient due to tumor involvement of the proximal splenic artery and the presence of a pseudoaneurysm.¹³ There was no recurrence of gastric variceal bleeding during follow up, therefore, the overall clinical success rate was 100%. Seven

Table 2 Summary of the data extracted from eligible papers

First author, year	Type of Study	Single or multicentre	Total number of patients	Number of patients receiving SAE to control gastric variceal bleeding, n (% of total participants)	Technical success of embolisation, n (%)	Embolic agent	Clinical success of completed embolisations, n (%)	30-day mortality following embolisation, n (%)	Major post-embolisation complications, n (%)	Controls, n
1 Goldman 1981 ¹⁷	Prospective	Single center	15 (11 male, 4 female)	4 (27)	4 (100)	Bucrylate (N-butyl cyanoacrylate)	4 (100)	1 (25)	2 (50)	N/A
2 Evans 1990 ¹²	Retrospective	Single center	12 (7 male, 5 female)	2 (17)	2 (100)	Not stated	2† (100)	1 (50)	1 (50)	10
3 Liu 2014 ¹³	Retrospective	Single center	21 (14 male, 7 female)	6 (29)	6* (100)	Coils only	6† (100)	0	0	15
4 Fernandes 2015 ¹⁸	Retrospective	Multicentre	22 (17 male, 5 female)	5 (23)	5 (100)	Not stated	5† (100)	Unknown*	0	11
5 Wang 2016 ¹⁹	Retrospective	Single center	14 (12 male, 2 female)	7** (50)	7 (100)	Polyvinyl alcohol (PVA) particles then coils	7 (100)	0	1 (14)	N/A
6 Petermann 2012 ²⁰	Retrospective	Single center	17	2 (12)	2 (100)	Coils only (1) Amplatz® Occluder & resorbable gelatin (1)	2 (100)	0	0	N/A
7 Luo 2014 ²¹	Retrospective	Single center	11 (9 male, 2 female)	3 (27)	3 (100)	PVA	3† (100)	0	0	8

Abbreviations: SAE: splenic artery embolization.

* Fernandes et al. noted that two participants died, however it is not noted which treatment they had (SAE, splenectomy or endoscopy) and how long after the treatment they died.

** 8 patients in the study group had gastric variceal bleeding, however one patient was lost during the follow up after receiving only splenic embolisation with PVA particles (half of the treatment).

† Seven patients went on to have a splenectomy post-SAE. One patient (Evans 19Abbreviation90) had a splenectomy 48 hours later. For the rest (3 patients in Luo et al, 2 in Fernandes et al and 1 in Liu et al), the timing of the splenectomies are unclear.

x In one patient the celiac artery trunk was embolised instead due to tumor involvement of the proximal splenic artery and the presence of a pseudoaneurysm.

Table 3 Summary of patient characteristics from the selected papers

	First author, year	Number of patients receiving SAE	Gender	Average age	Etiology of SPH	Number of patients bleeding acutely
1	Goldman 1981 ¹⁷	4	4 males	47	Acute and chronic pancreatitis (1) Thrombosed splenorenal shunt (2) Cirrhosis (1)	4
2	Evans 1990 ¹²	2	1 male 1 female	43	Pancreatic pseudocyst (1) Pancreatic adenocarcinoma (1)	2
3	Liu 2014 ¹³	6	NA	47*	Advanced pancreatic tumors (6)	6
4	Fernandes 2015 ¹⁸	5	NA	60*	NA	NA
5	Wang 2016 ¹⁹	7	NA	NA	Acute pancreatitis (4) Chronic pancreatitis (3)	7
6	Petermann 2012 ²⁰	2	NA	61*	Pancreatitis (1) Metastatic adenopathies (1)	2
7	Luo 2014 ²¹	3	2 males 1 female	45	Isolated pancreatic tuberculosis (1) Chronic pancreatitis (2)	3

out of 29 patients (24%) had splenectomies following SAE. The time that elapsed between SAE and splenectomy was unclear for 6 patients, but one splenectomy was performed 48 hours post-SAE.¹²

The most common minor complication reported was post-embolisation syndrome, characterized by left upper quadrant abdominal pain and fever. Nine patients (31%) described by 3 articles experienced either pain, fever or both.^{17,20,21} Wang et al noted that post-embolisation syndrome was the most frequent complication but did not quantify this. The remaining 13 patients (45%) included in a further 3 articles experienced no minor complications.^{12,13,18} Other minor complications included 4 transient, reactive left pleural effusions,²⁰ 3 significantly raised platelet counts managed with aspirin and constipation in 5 patients.¹⁹ However, it is unclear whether these complications occurred in the patients of interest to this review as these papers included patients receiving SAE for other etiologies, such as hypersplenism.

The overall major complication and 30-day mortality rates were 14% and 8%, respectively. There were 4 major procedural complications, 2 of which resulted in death. Two post-embolisation infections were observed. One infection occurred after PVA and coil embolisation and was due to splenic necrosis confirmed by CT.¹⁹ The second was a left upper quadrant abdominal abscess, which was drained 3 weeks post-SAE.¹⁷ This was following distal embolisation with N-butyl-cyano-acrylate.

The 2 deaths observed were in patients with multiple co-morbidities, however, both experienced SAE-related pulmonary complications and so were recorded as major procedural complications. One death was 3 weeks post-embolisation with N-butyl-cyano-acrylate and was attributed to a bleeding duodenal ulcer, hepatorenal syndrome and bronchopneumonia.¹⁷ This patient had received 33 units of blood during their admission prior to embolisation. Post-mortem examination showed no evidence of

splenic infection or inflammation. Another death occurred 7 days after SAE in a patient with a background of pancreatic carcinoma, malignant ascites, pneumonia and a pleural effusion.¹² Autopsy found a pulmonary embolus, a perforated gastric ulcer, and a pancreatic carcinoma occluding the splenic vein. Fernandes et al reported two deaths during the median follow up period of 24 months. However, it was unclear whether these patients had received SAE (this study also included endoscopic management and splenectomy) and how long after the procedure these deaths occurred.

Discussion

SPH is a rare but serious cause of upper gastrointestinal hemorrhage and is most commonly characterized by isolated gastric varices in the absence of true portal hypertension. Splenectomy is accepted as the definitive treatment of symptomatic SPH patients, but it is associated with significant mortality and morbidity in the acute setting.^{8,9} Splenic artery embolisation is a minimally invasive alternative that may be used alone or as an adjunct to surgery.

Risk of Developing Varices in SPH

The most common complication of SPH is bleeding, which occurs as a result of varices.^{3,7,8} Splenic vein occlusion results in increased venous pressure in local venous collaterals that act as venous outflow to the spleen such as the short gastric, gastroepiploic and coronary veins. The increased pressure in these venous collaterals results in dilatation of the veins in the gastric wall and can develop into gastric and sometimes oesophageal varices.^{3,23} Although common, not every individual with SPH is equally likely to develop varices.⁵ This is thought to be due to anatomic variations which may not allow the pressure diversion mechanisms to take place. Additionally, the risk of variceal bleeding also varies between individuals, most likely due to the degree of non-variceal collateral flow formation.⁵

Treatment Options for SPH

As variceal bleeding occurs in up to 35% of cases, control of bleeding is the most common aim in symptomatic patients in addition to removing the primary cause.^{7,8,11} Endoscopic measures to control bleeding in SPH patients include endoscopic sclerotherapy, balloon tamponade, band ligation and cyanoacrylate injection, which are associated with a high recurrence rate and are not without complication.^{2,24} Splenectomy has widely been considered to be the definitive management in symptomatic SPH patients.²⁵ This is thought to work by both decreasing the venous outflow from the collateral circulation as well as decompressing the existing collaterals, thus decreasing the risk of further hemorrhage.²⁶⁻²⁸ Splenectomy is associated with considerable morbidity and mortality in the acute setting. It carries a 10% risk of thromboembolic events, 1–2% lifetime risk of post-splenectomy infection and up to 15% perioperative mortality, most commonly due to bleeding.²⁹ Furthermore, the use of splenectomy in treating asymptomatic SPH patients prophylactically is difficult to justify, and watchful waiting is commonly the practice of choice in these patients.^{1,2}

Splenic vein recanalization via a percutaneous transhepatic or splenic approach is another treatment option for isolated gastric varices secondary to SPH.³⁰ A retrospective analysis of 11 patients who underwent endovascular recanalization found a technical success rate of 73% (8/11) with no cases of rebleeding in those patients.²¹ 6 patients had splenic vein stenosis rather than occlusion however. Post-procedural complications were observed in 25% of patients and in-stent stenosis occurred in 25% of patients at 9 and 15 months. The durability of splenic vein recanalization and stenting are drawbacks to the technique, particularly in benign disease.

Various studies have explored SAE as a viable treatment option for upper gastrointestinal hemorrhage secondary to SPH. However, the use of SAE as a definitive treatment in place of splenectomy remains widely debated.^{5,15} SAE can cause serious complications, such as splenic abscess, pleural effusions, pneumonia, atelectasis, pulmonary emboli, and portal vein thrombosis.¹⁶ The disadvantage of SAE compared with splenectomy is the loss of opportunity to surgically treat pancreatic pathology, such as removing tumors or cysts, particularly as this is a major etiology of SPH in addition to leaving potentially ischemic tissue (spleen) in situ.^{10,31} Pancreatic pathology may cause local inflammation and adhesions, making splenectomy more technically difficult however.³² Advantages of SAE over splenectomy include being less invasive with shorter operative time and the ability to perform the procedure under local anesthesia.³³ Currently, SAE is predominantly used as a temporising measure in patients who are unable to undergo splenectomy due to hemodynamic instability.^{12,34} However, the results of this systematic review show that SAE as a standalone treatment controlled gastric variceal bleeding in 100% of cases. Additionally, there was no rebleeding during follow up with 7 patients proceeding to splenectomy during this time. There was an overall major complication rate of 14% (4/29) and (excluding Fernandes et al) a 30-day mortality rate of 8% (2/24). This is comparable to

a previous review of 79 splenectomies for the treatment of SPH between 1969 and 1984, in which the clinical success rate was 92% and post-operative complication and mortality rates were 8%.²⁶ The slightly higher rate of major complications in this review is to be expected as SAE is often performed in high-risk patients who are unlikely to survive an operation.¹² Similar complication rates have been observed in SAE for the treatment of hypersplenism and blunt splenic trauma, varying between 4–13% in hypersplenism and averaging 20% in splenic trauma.³⁵⁻³⁷ SAE use in trauma is usually either proximal or superselective, which rarely result in complete splenic infarction. Furthermore, only partial embolization of the spleen is usually performed in hypersplenism. The safety and efficacy of the SAE for these two indications cannot, therefore, be extrapolated to SPH.

A variety of embolic agents may be used for splenic artery embolisation, including coils, vascular plugs, cyanoacrylate, particles and gelfoam. Particulate and glue embolic agents achieve distal embolisation, where splenic inflow is completely obliterated thereby reducing splenic outflow into the varices.³⁹ On the other hand, proximal embolisation, using coils, reduces splenic inflow but does not obliterate it due to collaterals from the dorsal pancreatic and short gastric arteries.⁴⁰ If patients continue to bleed following proximal embolisation, further embolisation may be difficult if not impossible.⁴¹

Splenic artery embolisation is an effective treatment for gastric variceal bleeding secondary to sinistral portal hypertension. Further prospective studies are needed to ascertain optimal embolisation strategies and material and whether a staged approach (SAE followed by splenectomy) improves outcomes.

Conflict of Interest

None declared.

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