

Anatomical Level of Main Truncal Splenic Artery Embolization: A Retrospective Cohort Study Using a Pancreatic Arterial Landmark Classification

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Research Article

Keywords: Splenic artery embolization, infarction, trauma, non-operative management, interventional radiology

Posted Date: October 5th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10989070/v1>

Abstract

Background:

To evaluate whether the anatomical level of main truncal splenic artery embolization, classified according to pancreatic arterial landmarks, is associated with post-procedural splenic infarction.

This retrospective single-center study included 28 adult patients who underwent main truncal splenic artery embolization between May 2018 and April 2023 for traumatic ($n = 13$, high-grade blunt splenic injury) or non-traumatic vascular conditions ($n = 15$), with post-procedural imaging follow-up. Embolization sites were classified into three anatomical areas: Area 1 (proximal to the dorsal pancreatic artery), Area 2 (between the dorsal and greater pancreatic arteries), and Area 3 (distal to the greater pancreatic artery). Embolic materials included coils (75%), vascular plugs (18%), and liquid embolic agents (32%). Outcomes included technical success, clinical failure, and post-procedural splenic infarction.

Results:

Technical success was achieved in all procedures, with no rebleeding, splenectomy, or splenic abscess. New or enlarged splenic infarction occurred in 10 of 28 patients (36%), including >50% splenic involvement in 3 (11%). New infarction occurred in 0% (0/3) of Area 1, 17% (1/6) of Area 2, and 32% (6/19) of Area 3 embolizations, without a statistically significant difference between groups (Fisher's exact test, $p = 0.529$).

Conclusions:

Main truncal splenic artery embolization demonstrated excellent technical success and clinical safety across diverse indications. Although not statistically significant, new splenic infarction occurred numerically more often following embolization distal to the greater pancreatic artery. These preliminary findings support prospective validation of this anatomical classification in larger multicenter studies.

1. Background

The spleen is the largest secondary lymphoid organ and plays a central role in immune and hematologic function [1]. Preserving splenic perfusion whenever possible is therefore an important therapeutic objective in patients undergoing splenic artery embolization (SAE), as splenectomy is associated with long-term risks related to impaired immune function, particularly overwhelming post-splenectomy infection [2].

SAE, performed using either selective or main truncal techniques, has become an established minimally invasive treatment for a variety of splenic vascular conditions, including blunt splenic trauma, pseudoaneurysms, aneurysms, pancreatitis-related vascular lesions, and other causes of splenic hemorrhage [3, 4, 5, 6, 7]. In trauma management, SAE is an essential component of nonoperative

management, improving splenic salvage while reducing splenectomy rates in appropriately selected patients [8, 9, 10, 11, 12, 13]. Current management recommendations, including the Society of Interventional Radiology Position Statement on Endovascular Intervention for Trauma [14], emphasize patient selection based on hemodynamic status, injury severity, and imaging findings while acknowledging persistent heterogeneity in embolization strategies [4, 5, 6, 14].

When focal vascular injuries such as active bleeding or pseudoaneurysms are identified, selective intrasplenic embolization is generally recommended. In contrast, main truncal SAE may be performed in the absence of focal lesions or as an adjunct to selective embolization [6, 7, 15]. Although truncal embolization is commonly described as either "proximal" or "distal," these terms are inconsistently defined across the literature. In many studies, "proximal" embolization encompasses anatomically distinct segments of the splenic artery, potentially obscuring differences in collateral perfusion mediated by pancreatic, gastric, and gastroepiploic arterial pathways.

To address this limitation, we classified main truncal SAE according to reproducible pancreatic arterial landmarks into three anatomical areas: proximal to the dorsal pancreatic artery (Area 1), between the dorsal and greater pancreatic arteries (Area 2), and distal to the greater pancreatic artery (Area 3). We hypothesized that this anatomical classification may better characterize the relationship between embolization level and post-procedural splenic infarction than the conventional proximal/distal classification. Accordingly, this study aimed to evaluate the association between embolization level and the occurrence and extent of post-procedural splenic infarction using this novel anatomical classification across traumatic and non-traumatic indications.

2. Method

2.1. Design and population

This retrospective single-center study was approved by the institutional review board (IRB No. 20250508-00011928), and the requirement for written informed consent was waived. Consecutive adult patients who underwent splenic artery embolization between May 2018 and April 2023 were screened for eligibility. Patients were included if they underwent main truncal splenic artery embolization, either alone or in combination with selective intrasplenic embolization. Exclusion criteria were age < 18 years, isolated intrasplenic embolization without concomitant truncal embolization, and absence of post-procedural contrast-enhanced CT follow-up.

2.2. Clinical and radiological outcomes

Pre-procedural contrast-enhanced CT, angiographic, and follow-up CT examinations were independently reviewed by three fellowship-trained radiologists (AB, FL, and TB, with 4, 6, and 12 years of experience in abdominal and interventional imaging, respectively). Assessments of follow-up CT examinations were performed independently before the assignment of the embolization area, and disagreements were resolved by consensus.

Pre-procedural CT examinations were reviewed to record the indication for embolization, AAST injury grade (when applicable), presence of active bleeding, pseudoaneurysm, and pre-existing splenic infarction. Active bleeding was defined as focal contrast extravasation on contrast-enhanced CT. Pre-procedural and follow-up CT examinations were performed on a 64-detector-row CT scanner following intravenous administration of iodinated contrast material, with acquisitions in the arterial, portal venous, and delayed phases.

Angiographic studies were reviewed to determine the anatomical level of main truncal splenic artery embolization, which was classified into three predefined areas (Fig. 1): Area 1, proximal to the origin of the dorsal pancreatic artery; Area 2, between the origins of the dorsal pancreatic artery and the greater pancreatic artery; and Area 3, distal to the origin of the greater pancreatic artery. The choice of embolization level, embolic material (coils, vascular plugs, liquid embolic agents, or combinations thereof), and the need for adjunctive selective embolization were left to the discretion of the treating interventional radiologist, based on the clinical indication, angiographic findings, vascular anatomy, and operator preference. Technical success was defined as complete occlusion of the target splenic artery segment with disappearance of the targeted vascular abnormality on completion angiography.

Follow-up CT examinations were reviewed for persistent or recurrent bleeding, persistent or recurrent pseudoaneurysm, splenic abscess, and new or enlarged splenic infarction. New or persistent bleeding was defined as the appearance of new active contrast extravasation, or the persistence of previously identified active extravasation, on follow-up imaging. The extent of splenic infarction was visually estimated on portal venous phase images as the percentage of newly non-enhancing splenic parenchyma relative to the estimated total splenic volume by comparison with the pre-procedural CT examination. Whenever feasible, traumatic lacerations, intraparenchymal hematomas, regions of transient vasospasm-related hypoperfusion, and pre-existing non-enhancing areas identified before embolization were excluded from this estimation.

2.3. Study outcomes

Study outcomes included technical success, major clinical failure, and post-procedural splenic infarction.

Major clinical failure was defined as any of the following during clinical or imaging follow-up: persistent or recurrent bleeding, persistent or recurrent pseudoaneurysm, repeat splenic artery embolization, secondary splenectomy, or splenic abscess requiring percutaneous drainage.

Post-procedural splenic infarction was categorized according to the estimated percentage of splenic involvement (< 25%, 25–50%, and > 50%). Infarction involving $\geq 25\%$ of the estimated splenic volume was considered clinically relevant for analysis.

2.4. Statistical analysis

Statistical analyses were performed using R software (R Foundation for Statistical Computing, Vienna, Austria). Categorical variables were compared using Fisher's exact test or Pearson's χ^2 test, as

appropriate. Continuous variables were assessed for normality using the Shapiro-Wilk test. Normally distributed variables are presented as mean $\pm$ standard deviation and were compared using Student's t test, whereas non-normally distributed variables are reported as median (interquartile range) and were compared using the Mann–Whitney U test.

Given the limited sample size and the small number of outcome events, no multivariable analyses were performed, and no adjustment for potential confounders (including embolic material, embolization indication, or operator preference) was attempted. Accordingly, all statistical analyses were considered exploratory and hypothesis-generating. All tests were two-sided, and statistical significance was defined as $p < 0.05$.

A generative artificial intelligence tool (Claude, Anthropic) was used to assist with language editing, formatting, and organization of the manuscript. All study design, data acquisition, statistical analysis, and interpretation of results were performed by the authors, who take full responsibility for the content and conclusions of the manuscript.

3. Results

3.1. Population and pre-operative characteristics

Forty-six consecutive patients were screened for eligibility. Eighteen patients were excluded: ten underwent isolated intrasplenic embolization without concomitant main truncal embolization, and eight lacked post-procedural contrast-enhanced CT follow-up. The final study cohort comprised 28 patients (see flowchart, Fig. 2).

Baseline characteristics are summarized in Table 1. The median age was 48 years (interquartile range [IQR], 39–55 years), and 17 patients (61%) were male. Main truncal SAE was performed for blunt splenic trauma (AAST grade ≥ 3) in 13 patients (46%) and for non-traumatic vascular conditions in 15 (54%). Three patients (11%) were receiving anticoagulant therapy, and none were receiving antiplatelet therapy. All patients underwent pre-procedural multiphasic contrast-enhanced CT including arterial, portal venous, and delayed phase acquisitions.

Table 1
Baseline characteristics

Overall population	28
Characteristics	
Sex	
Female – no (%)	11 (39)
Male – no (%)	17 (61)
Age – median (IQR)	48 (39–55)
Medication	
Antiplatelet – no (%)	0
Anticoagulant – no (%)	3 (11)
Circumstances of injury and preoperative CT scan analysis	
<i>Traumatic injury – no. (%)</i>	13 (46)
AAST grade's – no. (%)	
3	6 (46)
4	7 (54)
Active bleeding – no. (%)	3 (23)
Pseudoaneurysm – no. (%)	1 (8)
<i>Non-traumatic vascular conditions</i>	15 (54)
Unruptured aneurysm – no (%)	5 (33)
Spontaneous ruptured aneurysm or splenic artery – no (%)	4 (27)
False aneurysm of arterial anomaly of splenic artery due to pancreatitis or neoplasia – no (%)	6 (40)

Table 2
Angiography analysis and postoperative imaging analysis

Overall population	28
Angiography and embolization	
Truncal embolization – no. (%)	28 (100)
Embolization area – no. (%)	
1	3 (11)
2	6 (21)
3	19 (68)
Embolization agent – no. (%)	
Coil	21 (75)
Plug	5 (18)
Liquid agent	9 (32)
Distal intrasplenic embolization – no. (%)	2 (7)
Embolization agent – no. (%)	
Coil	1 (50)
Plug	0
Liquid agent	1 (50)
Technical success – no. (%)	28 (100)
Peroperative complication – no. (%)	0
Postoperative CT scan analysis	
Infarction appearance – no. (%)	10 (36)
< 25%	3 (11)
25–50%	4 (14)
> 50%	3 (11)
Splenic abscess – no. (%)	0
New bleeding or persistent vascular anomaly – no. (%)	0
Follow-up	
Splenectomy – no. (%)	0

Overall population	28
Angiography and embolization	
Spleen drainage – no. (%)	0

3.2. Embolization characteristics

Main truncal embolization was performed in Area 3 in 19 patients (68%), Area 2 in six (21%), and Area 1 in three (11%). Embolic materials included detachable metallic coils in 21 procedures (75%), vascular plugs in five (18%), and liquid embolic agents in nine (32%), used either alone or in combination; all liquid embolic agents were formulations of ethylene-vinyl alcohol copolymer (Onyx 18 or Onyx 34). The distribution of embolic materials varied by embolization area, with liquid embolic agents used in 1 of 3 (33%) Area 1, 0 of 6 (0%) Area 2, and 8 of 19 (42%) Area 3 procedures (Table 3). None of the Area 1 embolizations were performed in the setting of active contrast extravasation on pre-procedural imaging. Adjunctive selective intrasplenic embolization was performed in two patients (7%) for angiographically confirmed active contrast extravasation using detachable coils and ethylene-vinyl alcohol copolymer.

Table 3
Embolic material and pre-procedural active bleeding by embolization area

Embolic material – no. (%)	Area 1 (n = 3)	Area 2 (n = 6)	Area 3 (n = 19)
Coil	1 (33)	5 (83)	15 (79)
Vascular plug	2 (67)	1 (17)	2 (11)
Liquid embolic agent	1 (33)	0 (0)	8 (42)
Active bleeding on pre-procedural imaging – no. (%)	0 (0)	3 (50)	0 (0)
All liquid embolic agents used in this cohort were formulations of ethylene-vinyl alcohol copolymer (Onyx 18 and Onyx 34). Embolic materials could be used alone or in combination; percentages within each column therefore may not sum to 100%. None of the Area 1 (proximal) embolizations were performed in the setting of active contrast extravasation on pre-procedural imaging.			

Technical success was achieved in all procedures (100%), with no intraprocedural complications.

3.3. Clinical and imaging follow-up

The median interval between embolization and follow-up CT was 7 days (IQR, 4–23 days). No patient experienced major clinical failure, including recurrent or persistent hemorrhage, persistent vascular abnormality requiring reintervention, secondary splenectomy, or splenic abscess. In particular, none of the patients with new post-procedural splenic infarction subsequently developed splenic abscess, hemodynamic instability, or a clinical course requiring splenectomy or percutaneous drainage, suggesting that these radiographically detected infarcts were not associated with overt clinical sequelae during the follow-up period.

New or enlarged post-procedural splenic infarction was identified in 10 of 28 patients (36%). Infarction involved < 25% of the estimated splenic volume in three patients (11%), 25–50% in four (14%), and > 50% in three patients (11%). Overall, infarction involving $\geq 25\%$ of the estimated splenic volume occurred in seven patients (25%).

According to embolization area, new or enlarged splenic infarction of any extent occurred in 0 of 3 patients (0%) following Area 1 embolization, 1 of 6 (17%) following Area 2 embolization, and 9 of 19 (47%) following Area 3 embolization (Fisher's exact test, $p = 0.221$). When the analysis was restricted to infarctions involving $\geq 25\%$ of the estimated splenic volume — the pre-specified clinically relevant threshold — the corresponding rates were 0%, 17%, and 32%, respectively (Fisher's exact test, $p = 0.529$).

Although infarction occurred numerically more frequently following embolization distal to the greater pancreatic artery, the observed differences did not reach statistical significance (Fig. 3 and Fig. 4).

4. Discussion

Main truncal splenic artery embolization achieved excellent early clinical outcomes in this cohort, with 100% technical success and no cases of recurrent hemorrhage, persistent vascular abnormalities, secondary splenectomy, or splenic abscess during follow-up. These findings are consistent with previous series reporting high technical success and low rates of rebleeding and splenectomy following truncal SAE in appropriately selected patients [6, 11, 13, 16, 17]. Although embolization distal to the greater pancreatic artery was associated with a numerically higher frequency of post-procedural splenic infarction, these differences did not reach statistical significance. Rather than identifying an optimal embolization site, our findings suggest that pancreatic arterial landmarks provide a more anatomically meaningful framework for describing truncal splenic artery embolization and investigating collateral preservation.

The physiological rationale for truncal embolization is to reduce intrasplenic arterial pressure sufficiently to promote hemostasis while preserving collateral perfusion to maintain splenic viability [7, 15]. This concept is reflected in current trauma management guidelines and the Society of Interventional Radiology position statement, which recognize proximal splenic artery embolization as an effective strategy for splenic preservation in appropriately selected patients [4, 5, 6, 14]. Splenic collateral circulation arises primarily through the pancreatic arterial arcade—including the dorsal pancreatic, transverse pancreatic, and greater pancreatic arteries—as well as the short gastric and gastroepiploic arteries [15]. Consequently, embolization performed distal to these pancreatic branches may interrupt important collateral pathways and increase the extent of ischemic parenchymal injury. In addition, the physical characteristics and hemodynamic behavior of the embolic agent may influence collateral preservation. Whereas coils and vascular plugs generally produce focal arterial occlusion, liquid embolic agents may extend beyond the intended landing zone and compromise collateral circulation independently of embolization level. Because embolic material was selected at the operator's discretion, its distribution across anatomical zones may also have contributed to the observed infarction pattern:

liquid embolic agents (ethylene-vinyl alcohol copolymer) were used in 42% (8/19) of Area 3 procedures, versus 0% (0/6) of Area 2 and 33% (1/3) of Area 1 procedures (Table 3), a plausible, non-mutually-exclusive alternative to embolization level itself. Although our study was not powered to demonstrate statistically significant differences, the observed gradient of post-procedural splenic infarction is anatomically consistent with the collateral preservation model.

Previous studies comparing proximal and distal splenic artery embolization have generally reported similar rates of splenic salvage and procedure-related complications despite substantial variability in embolization techniques and anatomical definitions [6, 7, 11, 16]. One potential explanation for these inconsistent findings is the absence of a standardized anatomical definition of "proximal" embolization. In many published series, embolization performed either proximal to the dorsal pancreatic artery or between the dorsal and greater pancreatic arteries has been grouped into a single category despite representing distinct anatomical situations with potentially different patterns of collateral preservation. Such anatomical heterogeneity may have diluted differences between embolization strategies and contributed to the inconsistent results reported across the literature.

The principal contribution of the present study is therefore not the demonstration of superiority of one embolization site over another, but rather the proposal of a reproducible anatomical classification based on pancreatic arterial landmarks (Figs. 1 and 4). By distinguishing three truncal embolization zones, this classification separates anatomical scenarios that are frequently combined within the conventional proximal category and may facilitate more meaningful comparisons among future clinical studies. If prospectively validated in larger multicenter cohorts, this approach could contribute to a more standardized description and reporting of truncal splenic artery embolization techniques.

Importantly, the differences observed in this study were driven exclusively by radiographic splenic infarction identified on early follow-up CT. No patient experienced major clinical failure, including recurrent hemorrhage, secondary splenectomy, or infectious complications. Therefore, the clinical significance of early CT-detected splenic infarction remains uncertain, particularly because splenic immune function and long-term hematologic outcomes were not assessed. Previous studies suggest that radiographic infarction does not necessarily correlate with clinically significant loss of splenic function, and preservation of splenic perfusion should not be considered a surrogate for preserved immunologic function without dedicated functional assessment [2, 9, 11].

Several limitations should be acknowledged. First, the retrospective single-center design introduces selection bias and limits generalizability. Second, the small sample size, particularly the limited number of Area 1 embolizations ($n = 3$), precluded multivariable analysis and definitive assessment of the independent relationship between embolization level and infarction; all statistical analyses should therefore be considered exploratory and hypothesis-generating. Third, restricting the analysis to patients with post-procedural CT follow-up may have introduced selection bias, as patients with an uncomplicated course may have been less likely to undergo repeat imaging. Fourth, as discussed above, heterogeneity in embolic material distribution across embolization areas (Table 3) is an important

potential confounder that cannot be disentangled from embolization level in this small cohort. Fifth, radiologists were not formally blinded to clinical presentation or embolization technique, and interobserver agreement for embolization area assignment and infarction scoring was not calculated. Finally, the median imaging follow-up of seven days precluded assessment of delayed complications, splenic remodeling, or immune function.

5. Conclusions

Main truncal splenic artery embolization provided excellent early clinical outcomes across a heterogeneous cohort of traumatic and non-traumatic splenic vascular conditions. Although embolization distal to the greater pancreatic artery was associated with numerically higher rates of post-procedural splenic infarction, the limited sample size precludes definitive conclusions regarding the optimal embolization level. The proposed pancreatic landmark-based classification offers a reproducible and anatomically meaningful framework for the characterization and reporting of truncal splenic artery embolization, and may facilitate standardized comparisons across future prospective multicenter studies evaluating the relationship between embolization level, collateral preservation, and clinically relevant splenic outcomes.

Abbreviations

AAST

American Association for the Surgery of Trauma

CT

computed tomography

SAE

Splenic artery embolization

Declarations

Availability of data and materials:

The datasets generated and/or analysed during the current study are not publicly available due to patient privacy but are available from the corresponding author on reasonable request.

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Figures

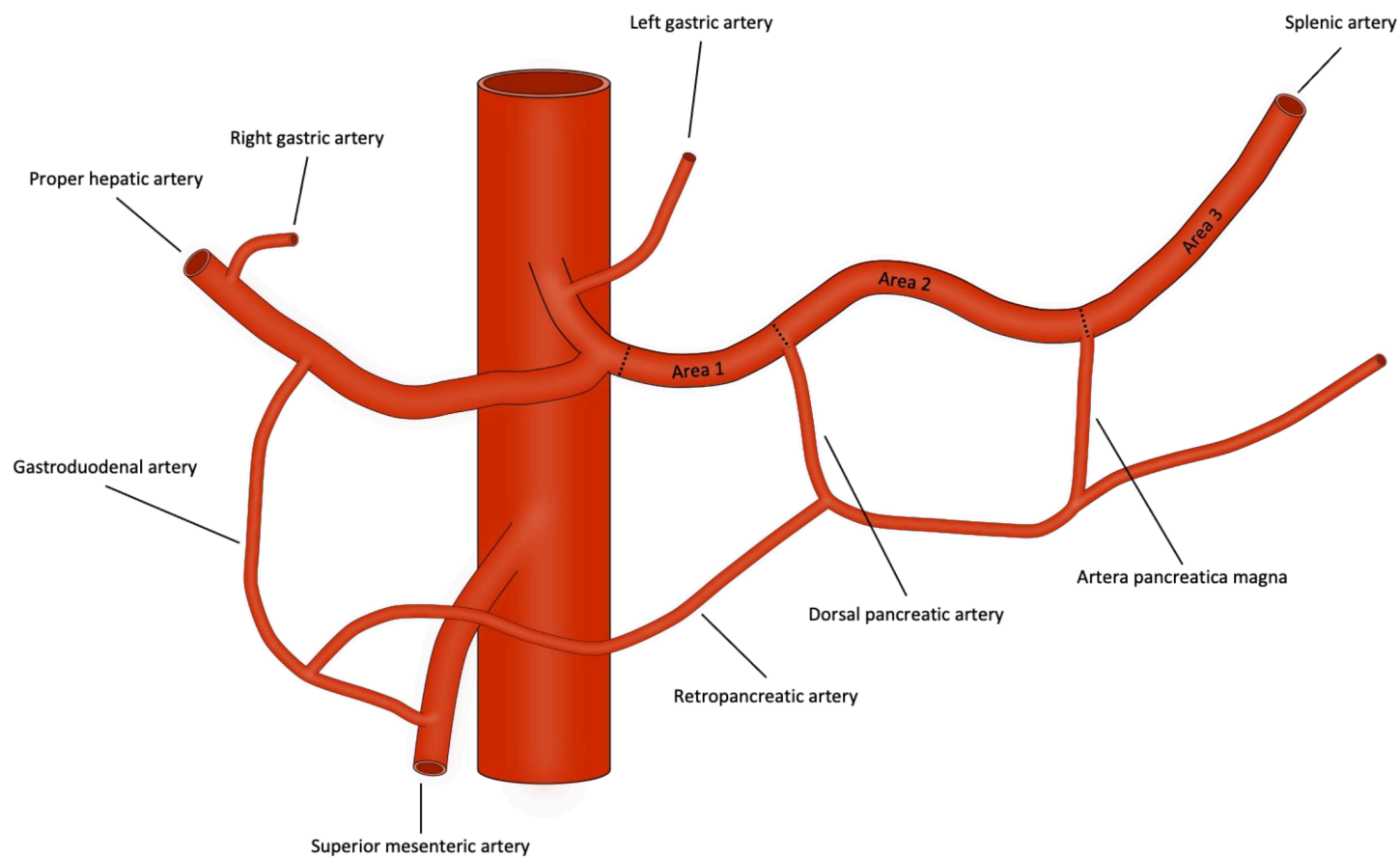


Figure 1
Splenic artery anatomy and embolization areas

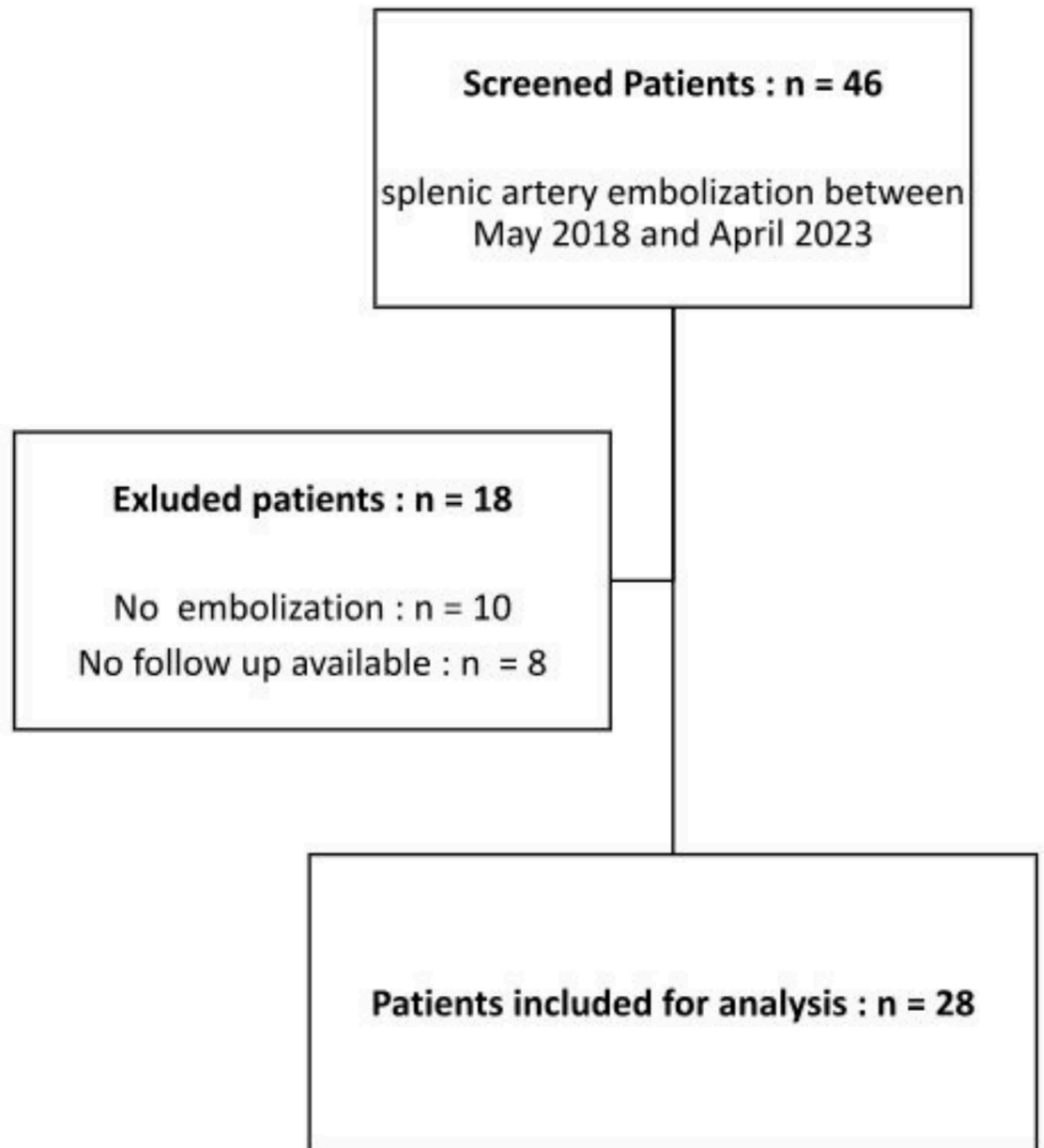


Figure 2

Flow chart

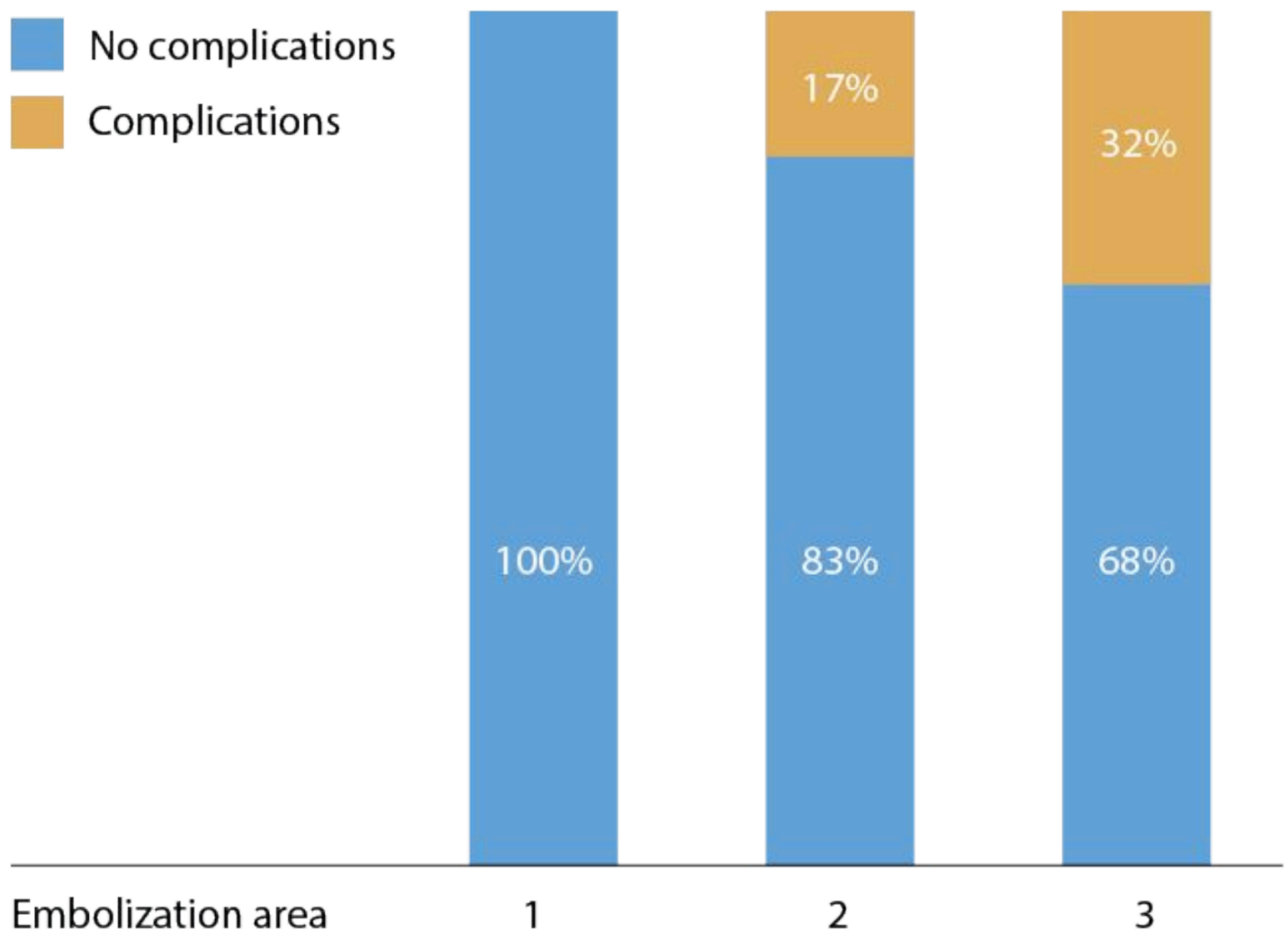


Figure 3

New splenic infarction rates according to embolization area. Given the small number of patients in Areas 1 ($n = 3$) and 2 ($n = 6$) and the absence of a statistically significant difference between groups (Fisher's exact test, $p = 0.529$), these findings should be interpreted as hypothesis-generating.

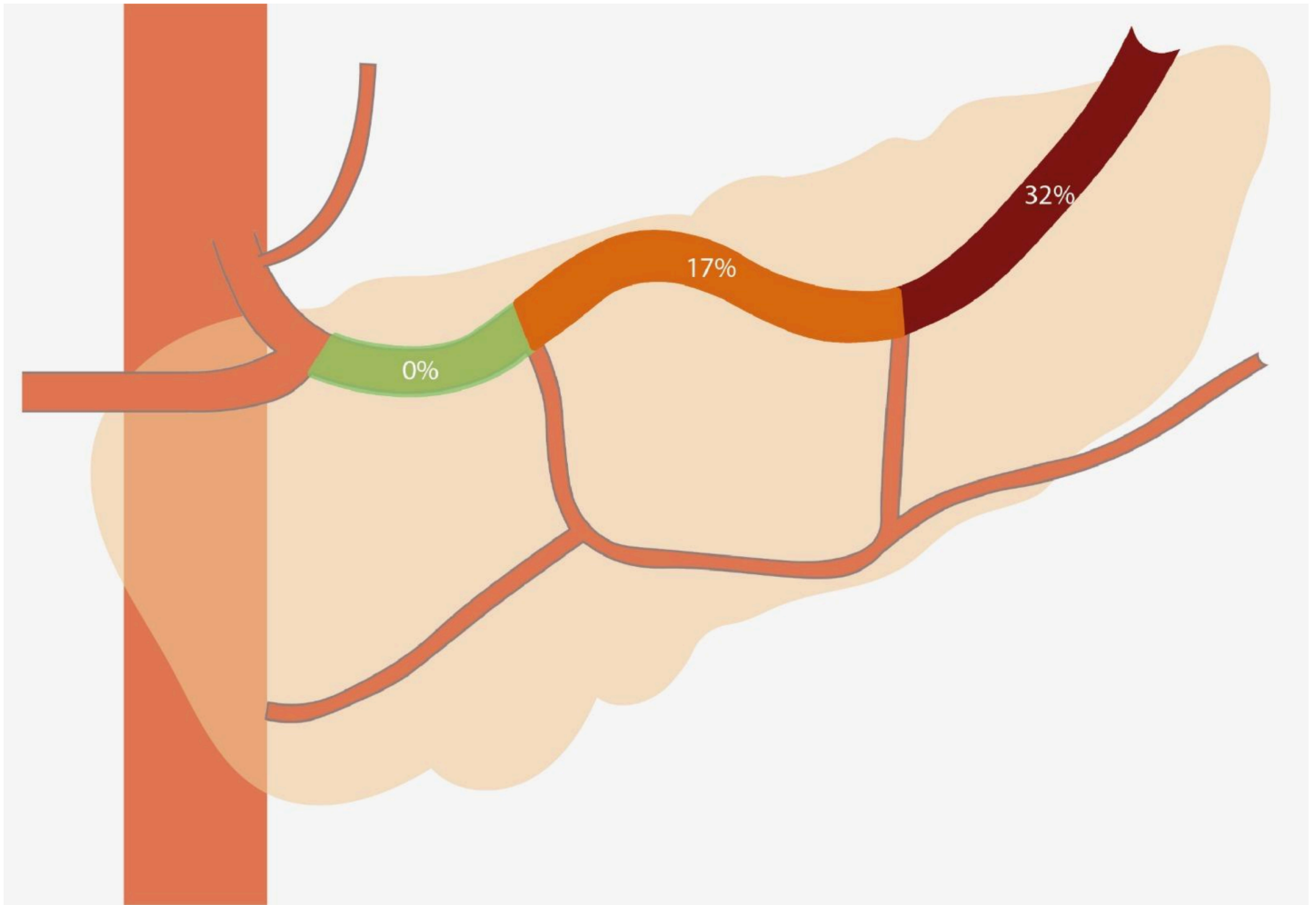


Figure 4

Diagram of the splenic artery depicting infarction rates stratified by embolization site. Proximal embolization (green, Area 1) was associated with no reported new infarction (0%), whereas mid (orange, Area 2) and distal (dark red, Area 3) embolization demonstrated numerically higher infarction rates (17% and 32%, respectively). These differences were not statistically significant, and Areas 1 and 2 comprised only 3 and 6 patients, respectively; this figure should therefore be interpreted as hypothesis-generating rather than as evidence of a causal relationship between embolization site and infarction risk.

Supplementary Files

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