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INTERVENTIONAL RADIOLOGY

ORIGINAL ARTICLE

Hemoptysis recurrence after arterial embolization in lung cancer: a 5-year retrospective cohort study

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PURPOSE

Hemoptysis is a life-threatening complication in patients with lung malignancy. Although arterial embolization provides reliable immediate hemostasis, recurrence rates in the oncologic setting substantially exceed those observed in benign disease. Whether routine baseline clinical and tumor characteristics can identify patients at higher risk of post-embolization rebleeding remains unclear.

METHODS

We conducted a 5-year single-center retrospective cohort study of 126 consecutive patients with histologically confirmed lung cancer treated with arterial embolization for hemoptysis (January 2021–December 2025). The hemoptysis-free interval was estimated using the Kaplan–Meier method and compared across subgroups using the log-rank test. Associations between prespecified covariates and recurrence were assessed using multivariable Cox proportional hazards regression. Embolic materials included metallic microcoils, polyvinyl alcohol particles (300–700 μm), gelatin sponge, and combinations thereof. Five covariates were examined: tumor diameter, cancer stage, histological subtype, age, and sex.

RESULTS

Technical success was achieved in 124 of 126 procedures (98.4%), and clinical success (no hemoptysis recurrence within 30 days) in 121 of 124 technically successful procedures (97.6%). Over a median follow-up of 31.3 months (interquartile range: 14.6–47.8 months), hemoptysis recurrence occurred in 32 patients (25.4%), with a median time to recurrence of 13.9 months. Recurrence proportions across embolic agent groups were considered descriptive only because allocation was not protocolized (log-rank $P = 0.45$). On multivariable Cox analysis, none of the five prespecified covariates showed a statistically detectable association with recurrence: tumor diameter [hazard ratio (HR): 1.00; 95% confidence interval (CI): 0.98–1.01; $P = 0.783$], cancer stage (HR: 1.21; 95% CI: 0.75–1.94; $P = 0.432$), squamous cell vs. other histology (HR: 0.80; 95% CI: 0.36–1.77; $P = 0.585$), age (HR: 0.98; 95% CI: 0.94–1.02; $P = 0.299$), and male sex (HR: 1.12; 95% CI: 0.53–2.36; $P = 0.761$). The events-per-variable ratio was approximately 6.4. Major complications occurred in 2.4% of patients (3/126), with no clinically adjudicated procedure-related mortality or permanent neurological sequelae.

CONCLUSION

In this exploratory single-center cohort, arterial embolization achieved high technical success with an acceptable short-term safety profile. The evaluated baseline clinical and tumor characteristics were not statistically associated with hemoptysis recurrence; however, the limited event count and absence of key angiographic and oncologic treatment variables preclude definitive conclusions regarding their predictive value.

CLINICAL SIGNIFICANCE

Standard tumor burden descriptors alone appeared insufficient for post-embolization recurrence risk stratification in this exploratory cohort. Future models should prioritize angiographic vascular anatomy, collateral supply, prior thoracic radiotherapy, and concurrent systemic anticancer therapy.

KEYWORDS

Hemoptysis, lung neoplasms, bronchial artery embolization, recurrence, embolic agents, time-to-event analysis

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Hemoptysis is reported in approximately 20%–30% of patients with primary or metastatic lung malignancy and may occur at any disease stage. In life-threatening cases, the bronchial circulation is the source in approximately 90% of patients.[1] In the oncologic context, hemoptysis frequently reflects neoplastic invasion of the bronchial or pulmonary vasculature, neovascular erosion at the tumor periphery, or tumor-related pseudoaneurysm formation.[2,3] Massive hemoptysis carries a mortality exceeding 50%, attributable predominantly to acute airway obstruction and asphyxiation rather than hemorrhagic shock alone.[4,5]

Bronchial artery embolization (BAE) has become an accepted first-line interventional strategy for acute hemoptysis control, with immediate technical success rates of 90%–100% across heterogeneous patient populations.[6-8] However, long-term durability in the setting of lung malignancy is substantially inferior to that observed in non-malignant conditions such as bronchiectasis or chronic infectious disease.[9] Recurrence rates following BAE in patients with lung cancer range from 20% to 50% in published series, a pattern attributed to tumor-driven angiogenesis and the recruitment of collateral vascular supply to previously embolized territories.

Despite this recognized recurrence burden, the clinical and procedural determinants of rebleeding after embolization in lung cancer are not well defined. Whether histological subtype predisposes to higher recurrence rates remains unresolved. Han et al. reported a trend toward higher recurrence in squamous cell carcinoma in a cohort of 84 patients,[10] whereas Garcia-Olivé et al.[11] found no significant histological predictor among 40 patients with cancer undergoing BAE for hemoptysis. Whether baseline tumor burden—quantified by tumor diameter or tumor–node–metastasis (TNM) stage—independently predicts interventional failure has not been systematically evaluated in adequately sized contemporary cohorts.[12] In particular, whether larger tumors, which might be expected to harbor more complex neovascularity, are associated with earlier or more frequent recurrence has not been adequately studied.

We therefore evaluated the hemoptysis-free interval (HFI) after arterial embolization in 126 consecutive patients with histologically confirmed lung cancer over 5 years and assessed whether baseline clinical and tumor characteristics—specifically tumor diameter, cancer stage, histological subtype, age, and sex—were associated with recurrence risk. Given the inconsistent findings in the existing literature and the absence of validated risk-stratification tools for this population, clarifying whether these readily available clinical parameters have predictive value is a necessary first step. Should they prove insufficient, future studies will need to investigate procedural and angiographic variables that are more closely related to the pathophysiology of post-embolization rebleeding but are more difficult to collect systematically.

Main points

- Arterial embolization was associated with high technical success and a low major complication rate in this single-center cohort.
- None of the five prespecified baseline covariates showed a statistically detectable association with recurrence, although the analysis was limited by 32 recurrence events.
- Standard oncologic staging descriptors alone appeared insufficient for post-embolization rebleeding risk stratification in this exploratory cohort, supporting future investigation of angiographic and oncologic treatment variables.

Methods

Ethical approval

This retrospective cohort study was approved by the Institutional Review Board of Jingdezhen Second People's Hospital (approval number: JDZDEY-LC-2023-028, date of approval: May 8, 2023). The approval covered the retrospective review of the medical records of consecutive patients treated at the study institution from January 2021 onward; the record review period was closed in December 2025. The ethics committee waived the requirement for individual written informed consent given the retrospective design and use of fully anonymized records.

Study design and patient selection

A retrospective analysis of 126 consecutive patients with histologically confirmed lung cancer who presented with hemoptysis and underwent arterial embolization at the study institution between January 2021 and December 2025 was performed (Figure 1). Significant hemoptysis was defined as expectoration of ≥ 30 mL of blood within 24 hours or any volume that resulted in hemodynamic or respiratory compromise, including hemodynamic instability or hypoxia attributable to airway blood.[4,5]

The inclusion criteria were as follows: (1) pathologically confirmed primary lung cancer or pulmonary metastasis, (2) significant hemoptysis refractory to conservative medical management, and (3) contrast-enhanced computed tomography angiography (CTA) performed within 48 hours before the procedure. The exclusion criteria were as follows: (1) uncorrectable coagulopathy (international normalized ratio > 2.0 or platelet count < 50,000/μL), (2) severe renal insufficiency (estimated glomerular filtration rate < 30 mL/min/1.73 m^2) precluding iodinated contrast, or (3) terminal illness with an estimated life expectancy of < 30 days.

Pre-procedural imaging

All patients underwent multidetector CTA (≥ 64 detector rows) within 48 hours before the procedure. Images were reviewed by two senior interventional radiologists to characterize tumor location, identify hypertrophied bronchial arteries and non-bronchial systemic collateral (NBSC) feeders, and detect angiographic signs of active hemorrhage, including sentinel vessels and focal hypervascular tumor staining.

Interventional procedure

All procedures were performed in a dedicated digital subtraction angiography (DSA) suite (AlluraClarity; Philips Healthcare, Best, the Netherlands) by operators with ≥ 10 years of experience. Femoral arterial access was obtained via the standard Seldinger technique using a 5-Fr sheath; transradial access has been described as a feasible alternative.[13] A systematic diagnostic survey of the thoracic aorta and its branches was performed using 4–5-Fr catheters (Cobra, Simmons, or pigtail; Terumo Corporation, Tokyo, Japan), targeting bronchial (orthotopic and ectopic), intercostal, internal mammary, and phrenic arteries, as well as the thyrocervical trunk for upper lobe lesions.

Superselective catheterization was achieved using 2.0–2.7-Fr microcatheters (Progreat, Terumo Corporation, Tokyo, Japan; Renegade, Boston Scientific Corporation, Marlborough, MA, USA), advanced as distally as possible while preserving non-target branches, particularly spinal medullary arteries. Culprit vessels were defined angiographically as follows: (a) frank contrast extravasation, (b) aneurysmal dilation or pseudoaneurysm, (c) focal hypervascular tumor blush, or (d) significant vessel tortuosity and enlargement (> 2 mm).

Embolic material was selected according

to vessel caliber and angiographic morphology. Metallic microcoils (Target; Stryker Neurovascular, Fremont, CA, USA) were preferentially selected for vessels ≥ 2 mm in diameter, where proximal flow reduction was the primary technical objective. Polyvinyl alcohol (PVA) particles (300–700 µm; Cook Medical, Bloomington, IN, USA) were used in cases with diffuse tumor blush without an identifiable single culprit vessel. Gelatin sponge (Gelfoam; Pfizer Inc., New York, NY, USA) was used as an adjunct or when temporary occlusion was clinically appropriate. Combined embolization (coils plus a particulate agent in the same session) was employed when both proximal and distal occlusion were required. Embolization proceeded under fluoroscopic monitoring until complete flow stasis and disappearance of pathological tumor blush were achieved on a 3-second delayed angiographic run.

Technical success was defined as angiographic occlusion of all identified culprit vessels to stasis at the end of the procedure. Clinical success was defined as the absence of hemoptysis recurrence within 30 days among technically successful procedures. Adverse events were graded according to the Society of Interventional Radiology (SIR) classification.[14]

Follow-up and outcome definitions

Patients were monitored clinically for 48–72 hours post-procedure and followed at scheduled outpatient visits. The HFI was defined as the time from the index embolization to the first documented episode of hemoptysis requiring medical or interventional management. Patients who died without recurrence were censored at the date of death. Follow-up status was available for all 126 patients through medical records and, when outpatient attendance was incomplete, structured telephone contact with the patient or primary caregiver; no patient was lost to follow-up. All 126 treated patients, including the two in whom technical success was not achieved, were included in the recurrence analysis. No missing data were present for the five covariates included in the Cox model.

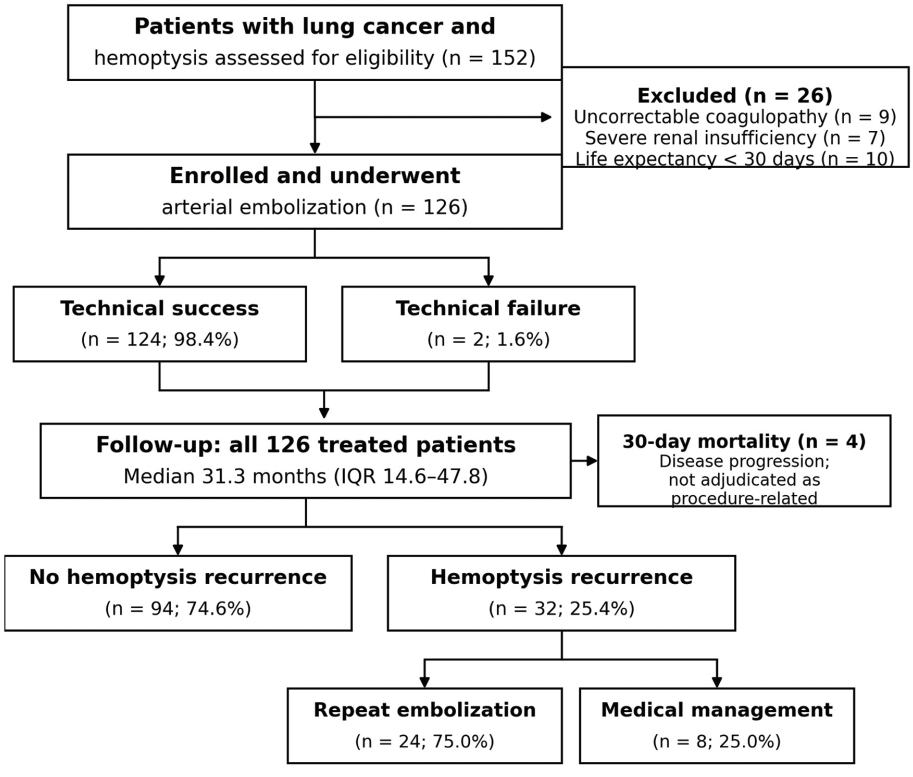

Figure 1. Patient enrollment flowchart. Of 152 patients assessed for eligibility, 26 were excluded: uncorrectable coagulopathy (international normalized ratio > 2.0 or platelet count < 50,000/µL; n = 9), severe renal insufficiency (estimated glomerular filtration rate < 30 mL/min/1.73 m^2; n = 7), or life expectancy < 30 days (n = 10), leaving 126 patients in the final analysis. Technical success was achieved in 124 patients (98.4%); the two patients with technical failure were followed up and included in the recurrence analysis. All 126 patients were followed (median 31.3 months); 32 patients (25.4%) experienced hemoptysis recurrence. IQR, interquartile range.

Statistical analysis

Continuous variables are reported as the mean (SD) or median [interquartile range (IQR)]; categorical variables are reported as frequencies and percentages. The HFI was estimated using the Kaplan–Meier method, and group differences were compared using the log-rank test. Multivariable analysis used a Cox proportional hazards model with five covariates entered a priori based on clinical relevance: age (continuous, per year), sex, tumor diameter (continuous, per mm), cancer stage (ordinal, per unit), and squamous cell carcinoma vs. other histology. With 32 recurrence events and five model covariates, the events-per-variable ratio was approximately 6.4. The multivariable Cox model was therefore considered exploratory and potentially vulnerable to overfitting, and the resulting estimates are reported with this limitation in mind. The proportional hazards assumption was assessed using scaled Schoenfeld residuals, and no covariate showed evidence of violation (all $P > 0.05$). Results are expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). Tumor diameter was additionally compared between recurrence groups using the Mann–Whitney U test. The correlation between tumor diameter and fluoroscopy time was assessed using Pearson's r as a secondary exploratory analysis. All analyses were performed using R version 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria). A two-sided $P < 0.05$ was considered statistically significant. This study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement; a completed STROBE checklist is provided as a Supplementary File.

Results

Baseline characteristics and technical outcomes

Baseline characteristics are summarized in Table 1. The cohort comprised 126 patients (mean age: 64.3 ± 10.4 years; 62.7% men). The predominant histological subtypes were adenocarcinoma (38.1%) and squamous cell carcinoma (34.1%). The majority of patients had advanced disease: Stage III in 41.3% and Stage IV in 30.2% (combined Stage III/IV: 71.4%). Metallic microcoils were the embolic agent in 50.0% of cases, gelatin sponge in 32.5%, PVA particles in 7.1%, and combined embolization in 10.3%.

Active hemorrhage signs on pre-procedural CTA—including sentinel vessels or focal hypervascular staining consistent with active

Table 1. Baseline characteristics of the study population stratified by recurrence status

Characteristic	Overall (n = 126)	No recurrence (n = 94)	Recurrence (n = 32)
Age (years)			
Mean (SD)	64.3 (10.4)	64.8 (10.0)	62.8 (11.7)
Median (IQR)	63 (56–71)	63 (57–71)	63 (54–69)
Range	40–89	45–89	40–86
Sex, n (%)			
Male	79 (62.7)	58 (61.7)	21 (65.6)
Female	47 (37.3)	36 (38.3)	11 (34.4)
Tumor diameter (mm)			
Mean (SD)	53.9 (26.4)	53.6 (26.4)	54.7 (26.7)
Median (IQR)	49.8 (35.0–66.3)	50.0 (35.0–67.8)	49.4 (40.0–61.7)
Range	12.2–120.0	12.2–120.0	17.2–120.0
Cancer stage, n (%)			
Stage I	6 (4.8)	6 (6.4)	0 (0.0)
Stage II	30 (23.8)	20 (21.3)	10 (31.3)
Stage III	52 (41.3)	40 (42.6)	12 (37.5)
Stage IV	38 (30.2)	28 (29.8)	10 (31.3)
Histological subtype, n (%)			
Adenocarcinoma	48 (38.1)	33 (35.1)	15 (46.9)
Squamous cell carcinoma	43 (34.1)	34 (36.2)	9 (28.1)
Small cell carcinoma	17 (13.5)	13 (13.8)	4 (12.5)
Other	18 (14.3)	14 (14.9)	4 (12.5)
Embolic agent, n (%)[a]			
Metallic microcoils (coils only)	63 (50.0)	49 (52.1)	14 (43.8)
Gelatin sponge (sponge only)	41 (32.5)	29 (30.9)	12 (37.5)
PVA particles (PVA only)	9 (7.1)	8 (8.5)	1 (3.1)
Combined (coils + particulate, same session)	13 (10.3)	8 (8.5)	5 (15.6)

[a]See Methods for embolic agent selection criteria. Values are n (%) unless otherwise stated. IQR, interquartile range; PVA, polyvinyl alcohol; SD, standard deviation.

extravasation—were identified in 38 patients (30.2%). Frank contrast extravasation on initial DSA was documented in 19 patients (15.1%); the remaining cases met the culprit vessel criteria based on tumor blush, vessel hypertrophy, or pseudoaneurysm formation. NBSC arteries—most commonly intercostal, internal mammary, and inferior phrenic branches—were embolized in a subset of patients when they were demonstrated to contribute to the culprit vascular supply. However, the number of NBSC feeders embolized per patient was not systematically recorded and could not be analyzed.

Technical success was achieved in 124 of 126 procedures (98.4%). The two failures were attributable to the inability to achieve stable catheterization of severely tortuous ectopic bronchial arteries. Both patients were followed up and included in the recurrence analysis; no recurrent episode requiring medical or interventional management was recorded for either patient. Clinical success was achieved in 121 of 124 technically successful procedures (97.6%). The 30-day mortality rate was 3.2% (4/126); these four patients were not judged to have an estimated life expectancy of < 30 days at the time of pre-procedural eligibility assessment, but rapid oncologic deterioration occurred after embolization. No death was clinically adjudicated as procedure-related, although autopsy confirmation was not available.

Hemoptysis-free interval (Figure 2)

Over a median follow-up of 31.3 months (IQR: 14.6–47.8 months), hemoptysis recurrence requiring medical or interventional treatment occurred in 32 patients (25.4%). The median time to recurrence among those who rebled was 13.9 months. Of the 32 patients with recurrence, 24 (75.0%) underwent repeat embolization, and 8 (25.0%) were managed medically (hemostatic pharmacotherapy, bronchoscopic intervention, or conservative care) according to clinical assessment and patient preference.

Kaplan–Meier HFI curves stratified by embolic agent, with a numbers-at-risk table, are shown in Figure 2. Because embolic material was selected according to vessel caliber and angiographic morphology rather than by a protocolized allocation strategy, recurrence proportions by embolic agent were considered descriptive only. Observed recurrence proportions were 22.2% for metallic microcoils (14/63), 29.3% for gelatin sponge (12/41), 11.1% for PVA particles (1/9), and 38.5% for combined embolization (5/13). The unadjusted log-rank comparison across the four groups yielded $P = 0.45$, but these unadjusted proportions should not be interpreted as estimates of comparative efficacy.

Predictors of hemoptysis recurrence (Figures 3 and 4)

Tumor diameter distributions were similar between patients who experienced recurrence (median: 49.4 mm; IQR: 40.0–61.7 mm) and those who did not (median: 50.0 mm; IQR: 35.0–67.8 mm; Mann–Whitney U $P = 0.9285$), as illustrated in Figure 3.

On multivariable Cox proportional hazards analysis (Figure 4), none of the five prespecified covariates showed a statistically detectable association with hemoptysis recurrence. Tumor diameter per mm (HR: 1.00; 95% CI: 0.98–1.01; $P = 0.783$), cancer stage per unit (HR: 1.21; 95% CI: 0.75–1.94; $P = 0.432$), squamous cell vs. other histology (HR: 0.80; 95% CI: 0.36–1.77; $P = 0.585$), age per year (HR: 0.98; 95% CI: 0.94–1.02; $P = 0.299$), and male sex (HR: 1.12; 95% CI: 0.53–2.36; $P = 0.761$) showed no statistically detectable associations with recurrence. Given the events-per-variable ratio of approximately 6.4, the resulting estimates are reported as exploratory.

Procedural safety

Complications occurred in 18 patients (14.3%). Minor complications (SIR Grade 1–2) were observed in 15 patients (11.9%): post-embolization syndrome (fever ≥ 38°C and/or pleuritic chest pain lasting > 24 hours) in 12 patients (9.5%) and access-site hematoma in 3 patients (2.4%). Major complications (SIR Grade 3–4) occurred in 3 patients (2.4%): 2 cases of localized bronchial wall necrosis—both in patients who had received prior thoracic radiotherapy and were treated with gelatin sponge—and 1 case of transient paraparesis that resolved completely within

72 hours. In this patient, no anterior spinal artery or radiculomedullary contribution to the embolized territory had been identified on pre-procedural CTA or initial diagnostic angiography, and the paraparesis was therefore considered an unanticipated intraprocedural event rather than a recognized pre-procedural risk. No permanent spinal cord ischemia was recorded. No death was clinically adjudicated as procedure-related, although autopsy confirmation was not available. Tumor diameter showed no measurable correlation with fluoroscopy time (Pearson's r: 0.039; $P = 0.661$).

Illustrative case (Figure 5)

Figure 5 presents a representative case of a 62-year-old man with squamous cell carcinoma and massive hemoptysis. On initial DSA (Panel a), the right bronchial artery exhibited marked hypertrophy (diameter approximately 3 mm) and tortuosity with intense peritumoral staining; metallic microcoils were selected to achieve proximal flow reduction in this large-caliber vessel. Panels b and c confirm pathological tumor blush and irregular neovascularization on superselective angiography. Complete flow stasis after coil deployment is shown in Panel d; durable devascularization without residual tumor blush is confirmed in Panels e and f. The patient remained hemoptysis-free at the 24-month follow-up.

Discussion

This study evaluated the HFI and its potential predictors in 126 consecutive patients with lung cancer treated with arterial embolization over 5 years. First, the procedure achieved technical success in 98.4% and 30-day clinical success in 97.6% of cases, with a major complication rate of 2.4%, compared with the pooled technical success of 98.1% and immediate clinical success of 94.0% reported in the most recent systematic review of BAE for hemoptysis.[15] Second, none of the five prespecified covariates—tumor diameter, cancer stage, histological subtype, age, or sex—showed a statistically detectable association with hemoptysis recurrence on multivariable analysis. Third, recurrence proportions varied across embolic agent groups, but these observations were descriptive only because of group size imbalance and non-protocolized allocation.

The recurrence rate of 25.4% over a median follow-up exceeding 31 months is consistent with published data from malignant hemoptysis cohorts. Syha et al.[16] reported

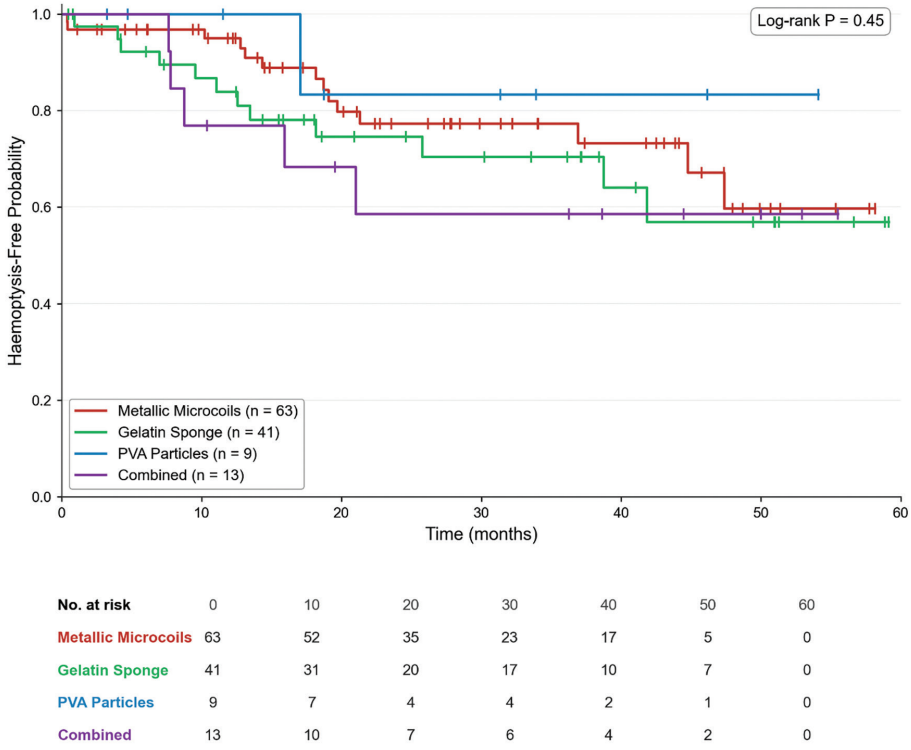

Figure 2. Kaplan–Meier estimates of the hemoptysis-free interval stratified by embolic agent (metallic microcoils, n = 63; gelatin sponge, n = 41; PVA particles, n = 9; combined, n = 13). Log-rank $P = 0.45$. Numbers at risk are shown at 10-month intervals below the curves. Tick marks indicate censored observations. Because embolic material was not allocated by protocol, the embolic-agent subgroups are shown for descriptive purposes only and should not be interpreted as comparative efficacy results. PVA, polyvinyl alcohol.

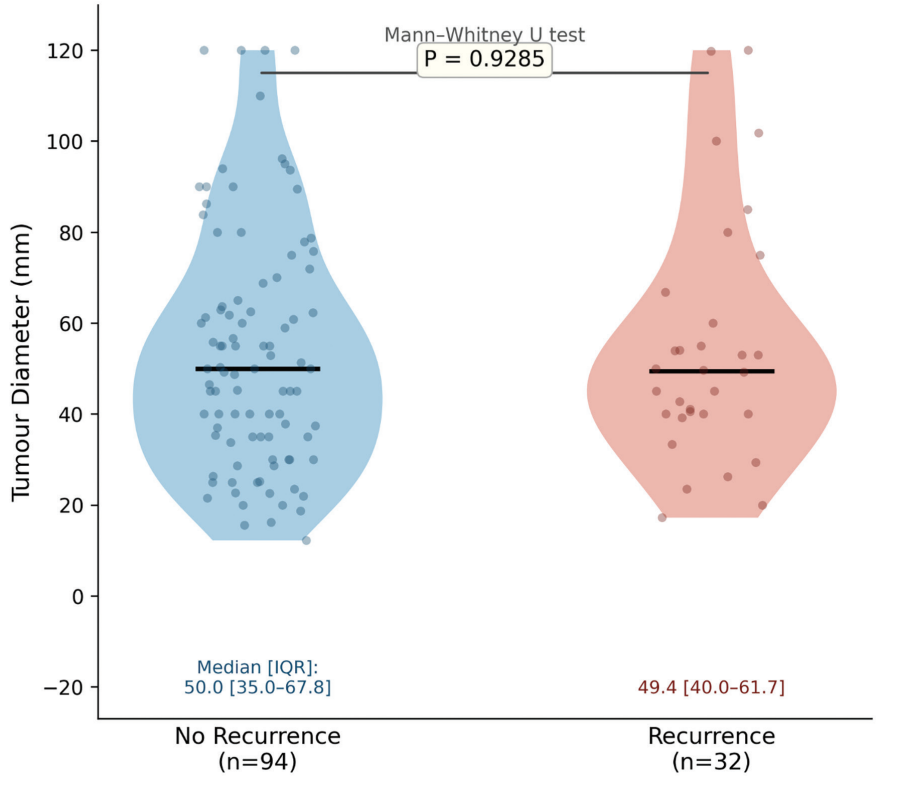

Figure 3. Distribution of tumor diameter by recurrence status. Violin plots with individual data points (jittered). Horizontal bars represent medians. Median (IQR) values were 50.0 mm (35.0–67.8 mm) for no recurrence and 49.4 mm (40.0–61.7 mm) for recurrence. Mann–Whitney U test $P = 0.9285$. IQR, interquartile range.

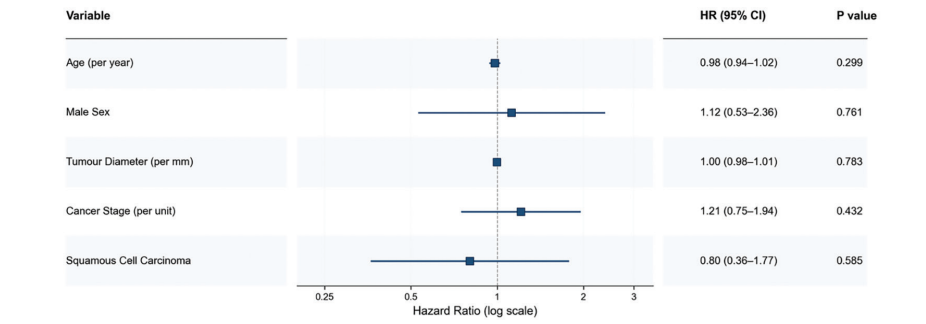

Figure 4. Forest plot of multivariable Cox proportional hazards regression. Hazard ratios (HRs) with 95% confidence intervals (CIs) displayed on a logarithmic scale. No statistically detectable association was observed for the five prespecified covariates in this exploratory multivariable Cox model.

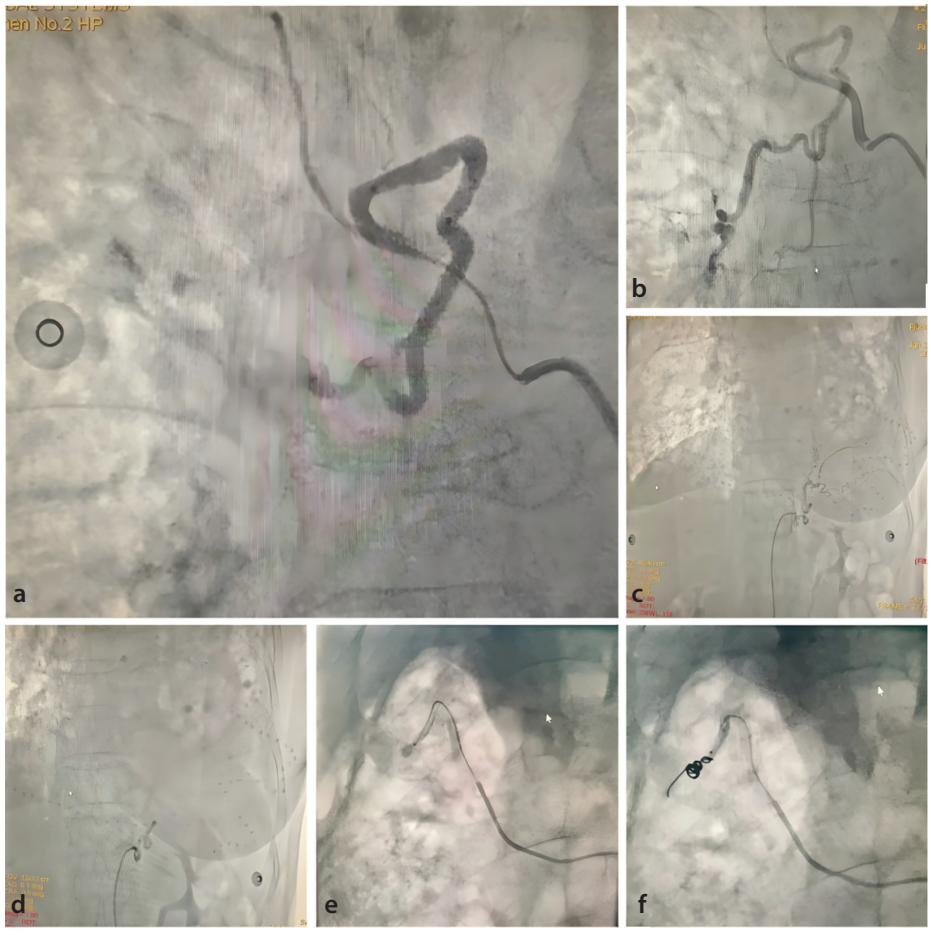

Figure 5. Arterial embolization in a 62-year-old man with squamous cell carcinoma and massive hemoptysis. **(a)** Initial DSA: hypertrophied, tortuous right bronchial artery with intense peritumoral tumor staining. **(b)** Superselective angiography: pathological tumor blush in culprit branch. **(c)** Irregular neovascularization within tumor bed. **(d)** Complete flow stasis after microcoil deployment. **(e)** Completion angiography: total occlusion of target vessel. **(f)** Durable devascularization without residual tumor blush. DSA, digital subtraction angiography.

a 10-year cumulative recurrence rate of 42.8% in patients with malignant etiologies compared with 16.5% in those with benign disease, whereas Ittrich et al.[17] estimated malignant recurrence at 25%–40%, depending on histology and follow-up duration. Our recurrence rate falls within this range and is consistent with both the pooled estimates of 23.0% at 1 year and 34.6% at 4–5 years derived from the most recent systematic review[15] and the observation that malignant hemoptysis carries approximately twice the rebleeding risk of non-malignant disease. The elevated recurrence burden in the oncologic setting has been attributed in the literature to ongoing tumor-related angiogenesis and the recruitment of collateral arterial supply to previously embolized territories, although these mechanisms have not been directly demonstrated in clinical studies, and their relative contributions remain speculative.

The absence of a statistically demonstrable association between tumor diameter and recurrence (Figures 3 and 4) warrants cautious interpretation. One might expect larger tumors to harbor more complex vascularity and a higher rebleeding risk; however, our data did not support this assumption. This observation is consistent with the report by Mehta et al.,[18] which states that tumor size did not show a statistically detectable association with technical failure or early recurrence in a single-institution malignant hemoptysis series. It has been suggested in the literature that hemoptysis in lung cancer may arise from focal vascular events, such as erosion of a single culprit vessel or pseudoaneurysm formation, rather than from diffuse hemorrhage proportional to tumor volume. If so, tumor diameter alone may not adequately capture bleeding risk. Our data cannot address this mechanistic question directly. The negligible Pearson's r correlation between tumor diameter and fluoroscopy time (r: 0.039) is reported as a secondary exploratory observation and should not be overinterpreted. Given the limited number of recurrence events (n = 32) and the wide CIs observed, the study was likely underpowered to detect a moderate association; the negative finding should therefore not be interpreted as evidence that tumor diameter is irrelevant to recurrence risk. A recent multivariable analysis of 144 patients with lung cancer identified massive hemoptysis volume and pulmonary artery injury—but not tumor diameter—as independent predictors of 1-month rebleeding.[19]

Similarly, histological subtype showed no statistically detectable association with recurrence in this cohort (squamous cell vs. other: HR: 0.80; $P = 0.585$). This adds to an inconclusive body of literature. Han et al. observed a non-significant trend toward higher recurrence in squamous cell carcinoma (30.0% vs. 19.4%; $P = 0.21$),[10] whereas Garcia-Olivé et al.[11] found no histological predictor in a smaller cancer-specific cohort (n = 40). Our data do not resolve this question, but the consistency of non-significant findings across multiple studies may indicate that histological classification alone provides insufficient resolution for stratifying post-embolization rebleeding risk or that all studies to date—including the present one—have lacked adequate statistical power to detect a real but moderate effect.

The comparison of embolic agents requires cautious interpretation. The substantial imbalance in group sizes—63 patients receiving coils vs. 9 receiving PVA particles—means that the log-rank analysis was insufficiently powered to support comparative conclusions regarding embolic materials. The observed recurrence rates (coils, 22.2%; gelatin sponge, 29.3%; PVA particles, 11.1%; combined embolization, 38.5%) should be interpreted as descriptive data only and cannot be taken as evidence of differential efficacy. In addition, the distinction between "permanent" and "temporary" embolic agents is an oversimplification: PVA particles are subject to tissue incorporation, and their occlusive effect is not reliably permanent, whereas coils are susceptible to distal recanalization and collateralization over time.[20,21] The high proportion of coil use in this cohort (50%) reflects an institutional practice pattern—coils were preferentially selected for vessels ≥ 2 mm requiring proximal flow reduction—rather than a protocol-driven choice, and this heterogeneity limits comparability with series using standardized particulate protocols.

The safety profile compares favorably with the published literature. The major complication rate of 2.4% is within the published range (median 0.1%; 0%–6.6%) reported in pooled BAE series.[2] The absence of permanent spinal cord ischemia reflects rigorous superselective microcatheter technique, permitting embolization distal to anterior spinal artery contributions.[22,23] Notably, the single episode of transient paraparesis occurred despite no spinal arterial contribution having been demonstrated on pre-procedural imaging or initial angiography, underscoring that part of the spinal ischemic risk may not be predictable from pre-procedural studies and that the possibility of neurological injury should be addressed during informed consent even when at-risk anatomy is not identified in advance. Both cases of bronchial wall necrosis occurred in patients with prior thoracic irradiation; prior radiotherapy has been proposed as a risk factor for post-embolization ischemic injury, and its presence may warrant discussion with the treating team before embolization.

Pre-procedural contrast-enhanced CTA played a useful planning role in this cohort, allowing identification of active hemorrhage signs—sentinel vessels or focal hypervascular staining consistent with active extravasation—in 30.2% of patients and characterization of bronchial and ectopic feeder anatomy before angiography. Recent comparative work using split-bolus dual-energy CT angiography has shown that CTA features may predict the angiographic bleeding site,[24] supporting CTA's expanding role in pre-procedural planning. CTA can also depict NBSC feeders that contribute to post-embolization recurrence. However, systematic mapping and quantification of CTA-derived collateral burden were not performed in this retrospective series, and the prognostic value of structured CTA-based vascular mapping remains a target for prospective evaluation.

This study has several limitations inherent to its retrospective single-center design. First, the restricted covariate set and limited number of recurrence events (n = 32) relative to the five model covariates yield an events-per-variable ratio of approximately 6.4, below the commonly recommended threshold of 10. This may result in imprecise HR estimates, increase the risk of model overfitting, and limit the study's ability to rule out moderate associations. The absence of statistically detectable associations should therefore be interpreted as a failure to detect—not as confirmation of the absence of—predictive relationships. Second, important procedural and angiographic variables—including the number of culprit vessels per patient, vessel tortuosity scores, NBSC involvement, and the completeness of collateral vessel mapping—were not systematically recorded and could not be evaluated. NBSC supply, which is recognized as a contributor to recurrence after BAE, may be a particularly important unmeasured variable. Third, concurrent systemic anticancer therapy—including chemotherapy, targeted therapy, immunotherapy, and antiangiogenic agents that may directly modulate tumor vascularity and bleeding risk—was not systematically captured and could not be evaluated as a potential confounder or effect modifier. Fourth, prior thoracic radiotherapy status, although clinically relevant to ischemic complications and observed in both cases of post-embolization bronchial wall necrosis in this cohort, was not systematically recorded as a candidate predictor of recurrence and could not be entered into the multivariable model. Therefore, the observed absence of statistically detectable associations may reflect limited statistical power and unmeasured confounding rather than a true absence of a predictive signal. Fifth, recurrence was defined as hemoptysis requiring medical or interventional management, which may introduce treatment-threshold bias because management decisions can vary by clinician, patient condition, and time period. In addition, hemoptysis volume was based on clinical estimation rather than a standardized quantitative measurement protocol, introducing the potential for misclassification. A sensitivity analysis using an objective recurrence definition (any documented hemoptysis episode regardless of management) was not feasible because untreated episodes were not consistently recorded in the source documentation. Sixth, the attribution of 30-day deaths as not procedure-related was based on clinical adjudication without autopsy confirmation. Seventh, the substantial group size imbalance across embolic agents, particularly the small PVA cohort (n = 9), and the non-protocolized, operator-dependent allocation of embolic material preclude meaningful conclusions regarding comparative agent efficacy. Eighth, as a single-center study, the generalizability of our institutional practice patterns—particularly the high proportion of coil embolization—to centers with different embolic agent preferences is uncertain.

Despite these limitations, the findings have implications for clinical practice and future research. In this exploratory single-center cohort of 126 patients—among the larger single-center lung cancer embolization series reported to date—the routine baseline tumor parameters most readily available at the time of procedure planning (tumor diameter, TNM stage, and histological subtype) showed no statistically detectable associations with post-embolization hemoptysis recurrence. Although this observation requires confirmation in adequately powered prospective studies, it is consistent with the hypothesis that future efforts to predict BAE durability in lung cancer may need to focus less on standard oncologic staging descriptors and more on variables that directly reflect the vascular anatomy of the embolization target. Variables worth investigating include the number and caliber of culprit vessels identified at angiography, the extent of NBSC contribution, the completeness of pre-procedural collateral vessel identification on CTA, prior thoracic radiotherapy status, and the type and timing of concurrent systemic anticancer therapy. Until such predictors are validated, close post-procedural surveillance for all patients—rather than risk-stratified follow-up based on tumor burden alone—remains the most reasonable clinical approach.

In this 5-year single-center retrospective cohort of 126 patients with lung cancer, arterial embolization achieved high technical success with a low major complication rate. The evaluated baseline clinical and tumor characteristics were not statistically associat-

ed with hemoptysis recurrence; however, the limited number of recurrence events, potential model overfitting, non-protocolized embolic material selection, and the absence of key angiographic and oncologic treatment variables preclude definitive conclusions regarding their predictive value. Future adequately powered studies should systematically capture vascular anatomy, NBSC supply, prior thoracic radiotherapy, and concurrent systemic therapy to clarify the determinants of durable bleeding control after embolization.

Footnotes

Conflict of interest disclosure

The authors declared no conflicts of interest.

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Supplementary: https://d2v96fxpocvxx.cloudfront.net/337910e4-2271-4883-88bd-db7abaf5adda/content-images/b24a0c7e-0432-41b4-b8ab-dd4a3c5a03d1.pdf

References

1. Yoon W, Kim JK, Kim YH, Chung TW, Kang HK. Bronchial and nonbronchial systemic artery embolization for life-threatening hemoptysis: a comprehensive review. *Radiographics*. 2002;22(6):1395-1409. [Crossref]
2. Panda A, Bhalla AS, Goyal A. Bronchial artery embolization in hemoptysis: a systematic review. *Diagn Interv Radiol*. 2017;23(4):307-317. [Crossref]
3. Radchenko C, Alraiyes AH, Shojaee S. A systematic approach to the management of massive hemoptysis. *J Thorac Dis*. 2017;9(Suppl 10):S1069-S1086. [Crossref]
4. Sakr L, Dutau H. Massive hemoptysis: an update on the role of bronchoscopy in diagnosis and management. *Respiration*. 2010;80(1):38-58. [Crossref]
5. Davidson K, Shojaee S. Managing massive hemoptysis. *Chest*. 2020;157(1):77-88. [Crossref]
6. Sopko DR, Smith TP. Bronchial artery embolization for hemoptysis. *Semin Intervent Radiol*. 2011;28(1):48-62. [Crossref]
7. Fruchter O, Schneer S, Rusanov V, Belenky A, Kramer MR. Bronchial artery embolization for massive hemoptysis: long-term follow-up. *Asian Cardiovasc Thorac Ann*. 2015;23(1):55-60. [Crossref]
8. Chun JY, Morgan R, Belli AM. Radiological management of hemoptysis: a comprehensive review of diagnostic imaging and bronchial arterial embolization. *Cardiovasc Intervent Radiol*. 2010;33(2):240-250. [Crossref]
9. Chen J, Chen LA, Liang ZX, et al. Immediate and long-term results of bronchial artery embolization for hemoptysis due to benign versus malignant pulmonary diseases. *Am J Med Sci*. 2014;348(3):204-209. [Crossref]
10. Han K, Yoon KW, Kim JH, Kim GM. Bronchial artery embolization for hemoptysis in primary lung cancer: a retrospective review of 84 patients. *J Vasc Interv Radiol*. 2019;30(3):428-434. [Crossref]
11. Garcia-Olivé I, Sanz-Santos J, Centeno C, et al. Results of bronchial artery embolization for the treatment of hemoptysis caused by neoplasm. *J Vasc Interv Radiol*. 2014;25(2):221-228. [Crossref]
12. Wang GR, Ensor JE, Gupta S, Hicks ME, Tam AL. Bronchial artery embolization for the management of hemoptysis in oncology patients: utility and prognostic factors. *J Vasc Interv Radiol*. 2009;20(6):722-729. [Crossref]
13. Lee Y, Lee M, Hur S, et al. Bronchial and nonbronchial systemic artery embolization with transradial access in patients with hemoptysis. *Diagn Interv Radiol*. 2022;28(4):359-363. [Crossref]
14. Khalilzadeh O, Baerlocher MO, Shyn PB, et al. Proposal of a new adverse event classification by the Society of Interventional Radiology Standards of Practice Committee. *J Vasc Interv Radiol*. 2017;28(10):1432-1437.e3. [Crossref]. Erratum in: *J Vasc Interv Radiol*. 2018;29(1):146. [Crossref]
15. Ravetta P, Bruyneel M, Vouche M. New insights on Bronchial Artery Embolization (BAE) for hemoptysis: a systematic review. *BMC Pulm Med*. 2025;25(1):541. [Crossref]
16. Syha R, Benz T, Hetzel J, et al. Bronchial artery embolization in hemoptysis: 10-year survival and recurrence-free survival in benign and malignant etiologies - a retrospective study. *Rofo*. 2016;188(11):1061-1066. [Crossref]
17. Ittrich H, Klose H, Adam G. Radiologic management of haemoptysis: diagnostic and interventional bronchial arterial embolisation. *Rofo*. 2015;187(4):248-259. [Crossref]
18. Mehta AS, Ahmed O, Jilani D, et al. Bronchial artery embolization for malignant hemoptysis: a single institutional experience. *J Thorac Dis*. 2015;7(8):1406-1413. [Crossref]
19. Hanotin C, Salvayre R, Lassalle L, et al. Predictive factors for recurrent hemoptysis after bronchial artery embolization in patients with lung cancer. *J Vasc Interv Radiol*. 2024;35(9):1296-1303. [Crossref]
20. Woo S, Yoon CJ, Chung JW, et al. Bronchial artery embolization to control hemoptysis: comparison of N-butyl-2-cyanoacrylate and polyvinyl alcohol particles. *Radiology*. 2013;269(2):594-602. [Crossref]
21. Makoto TT, Daniele P, Francesco P, et al. Bronchial artery embolization for the treatment of hemoptysis: permanent versus temporary embolic materials, a single center study. *CVIR Endovasc*. 2025;8(1):40. [Crossref]
22. Tanaka N, Yamakado K, Murashima S, et al. Superselective bronchial artery embolization for hemoptysis with a coaxial microcatheter system. *J Vasc Interv Radiol*. 1997;8(1 Pt 1):65-70. [Crossref]
23. Ishikawa H, Ohbe H, Omachi N, Morita K, Yasunaga H. Spinal cord infarction after bronchial artery embolization for hemoptysis: a nationwide observational study in Japan. *Radiology*. 2021;298(3):673-679. [Crossref]
24. Meena P, Bhalla AS, Goyal A, Naranje P, Kumar KP. Imaging findings as predictors of the site of bleeding in patients with hemoptysis: comparison between split-bolus dual-energy CT angiography and digital subtraction angiography. *Diagn Interv Radiol*. 2022;28(4):344-351. [Crossref]