

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

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PURPOSE

Systemic to pulmonary vasculature shunting (SPS) is an important finding to identify the probable site of bleeding, especially in multicentric parenchymal lung disease. The purpose of this study was to evaluate the value of imaging findings, which can locate SPS on dual-energy computed tomography angiography (DECTA), and correlate with digital subtraction angiography (DSA), which was considered as a gold standard.

METHODS

Retrospective analysis of 187 patients (148 males, 39 females, mean age: 43.7 ± 15.1 years) between October 2014 and November 2018 who underwent both DECTA and DSA. Computed tomography angiography was performed using dual-source (80 and 140 kV), 2×128 slice equipment, using 50-80 mL iodinated contrast (400 mg iodine/mL). These patients were divided into shunting (group A) and non-shunting groups (group B), based on the presence or absence of signs of shunting on DECTA. Group A had 98 and group B had 89 patients. We analyzed the following imaging signs for identifying SPS: (1) non-tapering pulmonary artery sign, (2) clustering of vessels sign, and (3) significant differential attenuation sign (>25 HU difference in attenuation between segmental pulmonary arteries of shunting side and normal non-shunting side was considered significant). The correlation was done with DSA to identify the presence of SPS.

RESULTS

In 187 patients, 281 lobes were evaluated to look for the signs of shunting from systemic artery to pulmonary vessels on DECTA. A total of 98 patients who showed signs of shunting on DECTA presented 135 lobes with parenchymal, with or without pleural, abnormalities. Of these, 84 patients had one or more aspergilloma in the lobe where shunting was seen. In one patient, a specific artery could not be cannulated due to a tortuous course; hence, all arteries which were seen on CTA causing shunting were also seen on DSA. Non-tapering pulmonary artery segmental branches were seen in 97 (99%) patients, clustering of systemic vessels was seen in 90 (91.8%) patients, and significant attenuation difference was seen in 74 (75.5%) patients. In the rest of the 89 patients, 146 lobes were assessed but no signs of shunting were seen on DECTA. Nine arteries in 8 patients showed shunting on DSA, while the rest did not show any shunting. Digital subtraction angiography correlation showed 96.4%, 100%, 100%, and 93.8% of sensitivity, specificity, positive predictive value, and negative predictive value, respectively, for DECTA in detecting SPS on a per artery basis.

CONCLUSION

The proposed signs on DECTA help in identifying the systemic vessels that cause shunting, and hence, the most likely bleeding site, which aids in planning the endovascular management by targeting specific arteries in case of multicentric disease. Being the gold standard, DSA is an ideal modality for detecting very small SPSs and in classifying the latter.

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Received 10 July 2020; revision requested 21 August 2020; last revision received 6 December 2020; accepted 27 January 2021.

Available online: 16 June 2022.

DOI: 10.5152/dir.2022.20548

Recurrent hemoptysis is a common symptom, especially in developing countries, where the prevalence of tuberculosis (TB) is high. The majority of the patients either have active infection or sequelae of old pulmonary TB often with aspergilloma formation.¹ Multiple lobes/sites are usually affected which are responsible for recurrent hemoptysis.² The source of bleeding originates from the systemic circulation in the majority (90%-95%) of cases.³⁻⁵

You may cite this article as 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.

Bronchial artery embolization (BAE) serves as a life-saving palliative treatment for hemoptysis.^{6,7} In view of variable anatomy of bronchial arteries (BAs), computed tomography angiography (CTA) is helpful, to plan the embolization.⁸ Multidetector CT angiography (MDCTA) enables accurate assessment of the number and location of the BAs as well as non-bronchial systemic arteries (NBSAs), which in turn facilitates the optimal endovascular embolization.⁸⁻¹⁰ Dual-energy CT imaging enables better visualization of vessels even with lower contrast opacification, owing to the low kVp dataset.¹¹

Due to multifocal parenchymal disease in the majority of such patients, it is important to identify the most likely site of active bleed. The parenchymal abnormalities secondary to sequelae of infection include fibrosis, bronchiectasis, and cavities with or without soft tissue within (due to aspergilloma). These parenchymal abnormalities cause neovascularization from systemic arteries (BAs and NBSAs) and the formation of systemic to pulmonary shunts (SPS), which are responsible for the recurrent chronic hemoptysis in these patients.^{12,13} It has been reported that SPS can induce and aggravate bleeding in >30% of patients and is frequently accountable for the recurrence of hemoptysis.¹³

In these patients with multilobar, multifocal disease, it becomes critical to identify the most likely site of bleed and thus

prioritize the culprit arteries for embolization. This is also because these patients not only have multifocal parenchymal disease but also multiple enlarged arteries (both BAs and NBSAs).

Most literature on CTA so far has focused on size criteria and tortuosity for the identification of abnormal vessels. While these are immensely useful, they may not be enough. Certain imaging findings on CTA may help in identifying these SPS areas in the lungs and thus enable detection of the likely site of active bleed, but there are very few studies that allude to these.^{12,13} These signs may predict the most likely site of active bleeding in patients with a multifocal parenchymal abnormality on MDCTA and can serve as a guiding tool for BAE. We propose the following 3 signs on MDCTA: (1) non-tapering pulmonary vessels sign, (2) clustering of vessels sign, and (3) significant differential attenuation sign. We validated these signs in our study cohort patients by correlating with the gold standard, that is digital subtraction angiography (DSA). To the best of our knowledge, the 3 signs used in this study are completely novel and have never been reported in any study before.

Thus, the purpose of this study was to evaluate the role of imaging findings that can locate SPSs as predictors of the site of bleeding on dual-energy computed tomography angiography (DECTA) and correlate these with DSA, which was considered as a gold standard.

Methods

Patient population

This study is a retrospective analysis of patients with non-streaky hemoptysis (i.e., moderate to massive hemoptysis or recurrent mild hemoptysis affecting the patient's lifestyle) who underwent both DECTA and BAE at the department of the Radiodiagnosis at our institution for evaluation, between October 2014 and November 2018. The study was approved by the ethics committee of our institution (protocol ref. no.:IESC/T-451/2014, IECGP-557/08.12.2016) and patient informed consent was taken. Figure 1 shows the study design.

Image acquisition

Dual-energy CTA was performed on Siemens Somatom Definition Flash dual-source (80 kVp and Sn140 kVp), dual-energy 2 × 128 slice CT scanner. All patients underwent craniocaudal scanning in the supine position at the end of suspended inspiration during a single breath-hold. The acquisition was performed from the thoracic inlet to the lower border of the second lumbar vertebra, including celiac and inferior phrenic arteries. Collimation of 64 × 0.6 mm was used. All the patients received non-ionic contrast (1.5 mL/kg body weight, maximum: 80 mL, 400 mg I/mL), which was administered through an 18-20 Gauge IV cannula in the antecubital vein, on the side of contralateral to the

Main points

- Multifocal parenchymal disease is common in patients with chronic hemoptysis; thus, it is important to identify the most likely site of active bleed.
- Certain imaging findings on DECTA may help in identifying these systemic to pulmonary shunting (SPSs) areas in the lungs; and thus, enable the detection of the likely site of active bleed in cases of non-streaky chronic hemoptysis.
- This study postulated and validated the signs of SPSs on DECTA in patients with non-streaky hemoptysis. These signs on DECTA guide us during the bronchial artery embolization to target specific arteries on a priority basis that are causing shunting in a particular lobe or segment of the lung; this might reduce procedure time and radiation exposure.

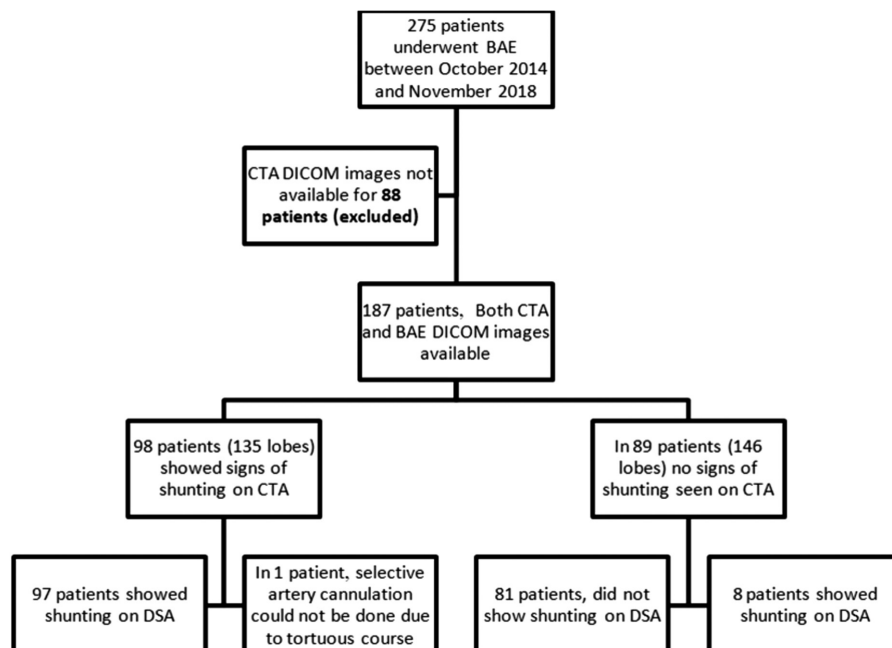


Figure 1. Study design—flow diagram.

more abnormal side on chest radiograph. A single-phase split-bolus injection protocol was used for contrast injection using a pressure injector (Medrad Stellant) version 105.0_SH.¹⁴ Three-fourth of total contrast volume was given at 5 mL/s followed by one-fourth of remaining contrast at 3 mL/s followed by saline (volume equal to one-fourth contrast) at 3 mL/s. The aim of the second bolus of contrast injection was to produce optimal enhancement of the pulmonary vasculature in the same acquisition. Monitoring was automatically initiated 7 seconds after the start of contrast injection. After the attenuation in the region of interest (ROI) reached 100 HU, the scan was triggered automatically after a delay time of 5 seconds.

All patients underwent DSA and BAE for controlling hemoptysis. Two different DSA machines, Siemens Artis Zee and Philips Allura FD 20, were used for the endovascular treatment according to the availability.

Image processing

Axial images of mixed kVp (60% of 80kVp and 40% of 140 kVp) were reconstructed and viewed at standard mediastinal window and lung window settings. Thin sections were evaluated on syngo.via diagnostic workstation version VB10B-HF06 and multiplanar reformats were used. The lung window was also viewed in high-resolution reconstruction. Thick maximum intensity images (MIP) were made by using low kVp (80 kVp) images for CT angiogram evaluation.

DECTA image analysis

The lung parenchyma was evaluated for fibrosis, cavities, aspergillomas, nodules, consolidation, chronic cavitary pulmonary aspergillosis (CCPA), and any mass or mass-like lesion. For diagnosing CCPA, imaging findings, i.e., one or more pulmonary cavities (with either thin or thick walls) containing one or more intraluminal soft tissue contents, with surrounding consolidation, were used.¹⁵ We also assessed vascular (systemic/pulmonary arterial) causes responsible for hemoptysis. Systemic arteries were assessed, and the following parameters were used for the assessment of BAs: (a) number of BAs per side, (b) diameter at origin, and (c) 3-dimensional localization for origin and course of BAs. Bronchial arteries were labeled abnormal if the diameter at origin was ≥ 2 mm and/or tortuous course and/or traceable up to the hilum. Non-bronchial systemic arteries were assessed for diameter and tortuous course toward the parenchymal abnormality. For NBSAs, the size criteria were not taken into account; only subjective criteria were applied, and the contralateral side was used for comparison. Pulmonary arteries were also assessed for any pseudoaneurysm, arteriovenous fistula, and pulmonary embolism.

Systemic to pulmonary shunting was assessed by using the following signs on DECTA (Figure 2). If any of these signs are present on DECTA in any patient who was labeled for the presence of SPS, then the use of the split-bolus technique enables the assessment of SPS on CT.

We propose the following novel signs for the same:

- 1) Non-tapering pulmonary vessels sign: Segmental pulmonary arteries were assessed in the area where thick-walled cavities, aspergilloma, consolidation, or masses were present. Usually, segmental pulmonary arteries taper as they go to the periphery, but in the presence of SPS, the pulmonary arterial branches appear engorged, and thus, this normal tapering of pulmonary vessels will not be seen. Non-tapering sign refers to engorgement due to shunting.
- 2) Cluster of vessels sign: Cluster of vessels can be seen due to recruitment of systemic vessels and shunting into pulmonary vessels in patients with thick-walled cavities with or without aspergilloma. This sign, suggesting the presence of prominent systemic and pulmonary vessels both, would serve to identify the site of SPSs.
- 3) Differential attenuation sign: Differential attenuation sign refers to increased contrast in the pulmonary arterial branches due to shunting. Attenuation of pulmonary artery was evaluated subjectively and objectively to assess the shunting. Subjective assessment was done by comparing the segmental pulmonary arteries of the abnormal side with the normal side; a visible difference in contrast attenuation/enhancement was seen. Objective assessment was done by measuring attenuation value by drawing ROI (≥ 5 mm²) in segmental pulmonary arteries and comparison was done with the contralateral side. A difference of ≥ 25 HU was considered significant.



Figure 2. a-c. Signs of shunting. Non-tapering pulmonary vessel sign: Coronal MIP CTA image (a) shows non-tapering of segmental branches of right upper lobe pulmonary artery (*small black arrow*) as compared to the opposite side. In another shunting group patient, coronal MIP CTA image (b) shows "Cluster of vessels sign," formed by the left inferior phrenic artery (*long black arrow*) in the left lower lobe, on the diaphragmatic surface. A differential attenuation sign is seen on coronal MIP CTA (c), which shows increased attenuation (right-sided: 540 HU vs. Left-sided: 399 HU) of the right upper lobe pulmonary artery branch due to SPS (*thick black arrow*). MIP, maximum intensity images; CTA, computed tomography angiography; SPS, systemic to pulmonary vasculature shunting.

DSA image analysis

Bronchial arteries and NBSAs were assessed on DSA. Blush, active extravasation, and SPS were looked for. For the assessment of SPS, each selective artery run was evaluated and shunting was said to be present and abnormal if there was opacification of pulmonary vasculature (either pulmonary veins or pulmonary arteries) within 3 seconds of the start of a specific systemic arterial run, which was at the rate of 3 frames/s. Dual-energy CTA images were reviewed separately, without knowing the results of DSA findings.

According to the previous study,¹³ we classified SPS into 3 types of DSA : (1) bronchial artery–pulmonary artery shunt (BPAS): selective DSA run showed the shunting of contrast from BAs to pulmonary artery branches (centrifugal shunting); (2) bronchial artery–pulmonary vein shunt (BPVS): selective DSA run showed the shunting of contrast from BAs to pulmonary vein and heart (centripetal shunting); (3) non-bronchial systemic artery–pulmonary vasculature (either pulmonary artery or pulmonary vein) shunt (NBPS): selective DSA run showed the shunting of contrast from NBSA to pulmonary vessels.

Statistical analysis

Data analysis was performed using a software package (Statistical Package for the Social Sciences for Windows, Version 15.0.; SPSS Inc.; 2008). The diagnostic accuracy of DECTA was evaluated using chi-square and Fischer exact tests. In all tests, $P < .05$ was considered significant. Sensitivity, specificity, positive predictive value, and negative predictive value of CTA findings for SPS were calculated with DSA as a gold standard.

Results

A total of 187 patients (148 males, 39 females) formed the study cohort, with a mean age of 43.7 ± 15.1 years. In 187 patients, 281 lobes showed parenchymal \pm pleural abnormalities and were evaluated to look for signs of shunting from systemic artery to pulmonary vessels on DECTA. Among these, 135 lobes in 98 patients showed signs of SPS. In the remaining 89 patients, the 146 abnormal lobes had no signs of shunting seen on DECTA. Thus, patients were divided into 2 groups based on DECTA findings: shunting group (group A; 84 males,

14 females, mean age of 43.7 ± 15.1 years) and non-shunting group (group B; 64 males, 25 females, mean age of 42.7 ± 15.7 years). There was no statistically significant difference between the age ($P = .38$) of the two groups; however, a statistically significant difference was noted in sex distribution ($P = .010$) between these groups.

The etiology of hemoptysis in both groups was also evaluated on DECTA. In group A (shunting group), CCPA and sequelae of old infection were found in 87.7% of cases (Figure 3). Eighty-six of the 98 patients in group A (shunting group) had a past history of antitubercular treatment. In group B (non-shunting group), the majority of the patients (58.4%) had bronchiectasis with or without fibrosis. If more than 25% part of a particular lobe was showing fibrosis with or without bronchiectasis, later was labeled as fibrobronchiectasis.

Table 1 shows the etiology of hemoptysis, as ascertained on DECTA, in both groups. The chi-square test revealed a significant difference in the 2 groups in terms of the proportion of patients with CCPA and fibrobronchiectasis (Table 1). While CCPA was the main etiology in the shunting group, bronchiectasis predominated in the non-shunting group. Eighty-four of the 98 patients showing evidence of shunting on DECTA had one or more aspergilloma(s) in the lobe where shunting was seen.

A total of 467 arteries in 187 patients were found as abnormal on DECTA. On a per-patient basis, an average of 2.49 abnormal systemic arteries was seen on DECTA. A minimum of 1 to a maximum of 7 abnormal systemic arteries were noted.

The shunting group (group A) showed a total of 300 abnormal arteries on DECTA, out of which 212 arteries (70.6%) were NBSAs (Figures 4 and 5). In the non-shunting group (group B), 167 systemic arteries were labeled abnormal, with the majority being BAs (106/167; 63.4%). The difference in the proportion of NBSAs between the shunting and non-shunting groups was statistically significant ($P < .001$). Posterior intercostal arteries were the most common NBSAs, comprising 45.7% of all abnormal systemic arteries in the shunting group (group A) on DECTA. While among the BAs, the right intercostobronchial trunk was most common.

In group B, a patient had pulmonary artery pseudoaneurysm as a source for hemoptysis. A detail of BAs and NBSAs of both groups of patients on DECTA is given in Table 2.

A total of 387 abnormal arteries were catheterized and embolized during BAE on DSA. All the arteries labeled abnormal on DSA had also been labeled abnormal on DECTA. The decision of embolization of particular systemic arteries was taken on the basis of the severity of parenchymal abnormality on DECTA. Also, in those with more than 5 abnormal arteries, the worst arteries/lobes were prioritized for embolization. A maximum of 4 worst arteries were embolized in a single session which is most likely responsible for hemoptysis and shunting. It was done to avoid a larger volume of contrast (so as to reduce the chances of side effects especially renal dysfunction) as well to avoid excessive radiation exposure to patient and operator. Eighty arteries were not evaluated during DSA due to the decision taken by authors during the procedure to target

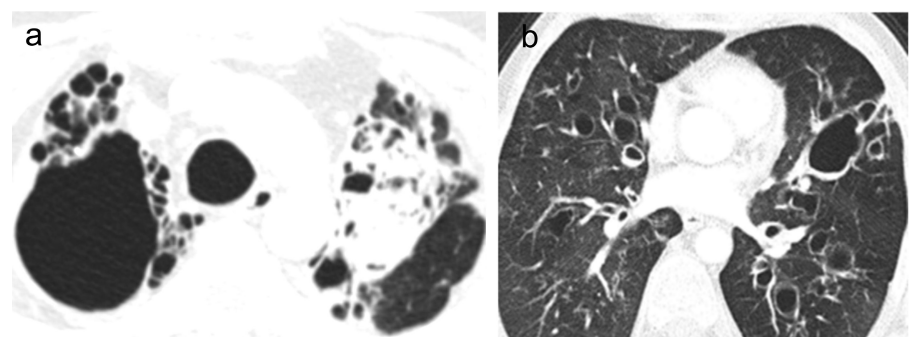


Figure 3. a, b. Etiologies in different patients. Axial lung window image (a) of a 45-year-old male patient of the shunting group shows thick-walled cavities with surrounding consolidation in bilateral upper lobes suggestive of CCPA. Axial lung window image (b) of a 47-year-old female of the non-shunting group, show cystic bronchiectasis with bronchial wall thickening in bilateral lungs. CCPA, chronic cavitary pulmonary aspergillosis.

Table 1. Etiology of hemoptysis in both groups of patients

Etiology	Shunting group, group A (n=98)	Non-shunting group, group B (n=89)	P
Cavity with or without simple aspergilloma	19	24	.22
CCPA	67	03	<.001
Fibrobronchiectasis or bronchiectasis	11	52	<.001
Mass	1	2	.61
Active infection	0	4	.050
Pulmonary artery pseudoaneurysm	0	1	.23
COPD	0	2	.48
Normal	0	1	.48

CCPA, chronic cavitary pulmonary aspergillosis; COPD, chronic obstructive pulmonary disease.

the worst arteries and a few due to difficult/failed cannulation due to unfavorable anatomy. Among 387 abnormal arteries, 241 were seen in group A, while 146 were in group B patients.

In group A (shunting group), 161 arteries (66.8%) were NBSAs, while NBSAs constituted 28.1% of arteries in group B; and this difference was statistically significant ($P < .001$). Posterior intercostals arteries were the major source. In group B, BAs constituted approximately 71.9% (105/146). As on DECTA, there was a statistically significant difference ($P < .001$) in the proportion of BAs in the 2 groups. A detail of BAs and NBSAs of both groups of patients on DSA is given in Table 3.

Among the 3 signs, the non-tapering pulmonary artery sign was the commonest and was seen in 97/98 (99%) patients, while the cluster of vessels sign was seen in 90/98 (91.8%) patients. Significant differences in the attenuation of pulmonary vessels could be seen only in 74 (75.5%) patients.

In a patient in group A, one artery could not be cannulated on DSA; all the other arteries (n=241) which were labeled abnormal and as showing shunting on DECTA also showed shunting on DSA. On the other hand, 9 arteries (in 8 patients) of group B showed shunting on DSA, while no signs of shunting were seen on DECTA and hence had been placed in group B. Out of these, 5 patients had bronchiectasis (Figure 6), with or without fibroparenchymal abnormality in the lung parenchyma. Correlation of DECTA with DSA on a per artery basis showed 96.4%, 100%, 100%, and 93.8% of sensitivity, specificity, positive predictive value, and negative predictive value, respectively, for DECTA in detecting SPS.

On DSA in group A (shunting group), BPAS alone was seen in 34/98 (34.7%), while BPVS alone was seen in 6/98 (6.1%) patients. The majority of shunts were from NBPS which constituted 55.1% of all cases (54/98). In 3 patients, more than one type of shunts was seen. On the other hand, in

the non-shunting group on DSA, 8 patients showed shunting, with 6 of them having BPAS and 2 having NBPS.

Discussion

In this retrospective study, we evaluated and validated signs on DECTA which can predict the site of SPS in patients with non-streaky hemoptysis. All of these patients had clinically significant hemoptysis requiring BAE for management. The hypothesis of this study was that the utilization of the 3 proposed signs on CTA could aid in quick identification of maximum vascularization, SPS, and hence, site of hemoptysis on CTA. These signs would aid in quick identification of the likely site of bleed, which is especially important in those with multifocal parenchymal changes. Males constituted the majority of patients overall and also in both groups. The male predominance was reported even more in the shunting group (group A), because the dominant etiology in the shunting group was CCPA, versus fibrobronchiectasis in the non-shunting group (group B), and CCPA is known to have male predominance.¹⁶

Systemic to pulmonary arterial shunting develops in chronic inflammatory states, with both active infections (particularly TB) and secondary to their sequelae. We found that the predominant etiology of hemoptysis in group A was CCPA with underlying sequelae of previous infection (87.7% of patients). On the other hand, in group B, 58.4% of patients had bronchiectasis, with or without fibrosis. Tuberculosis and sequelae of previous infections, with or without aspergilloma, have been reported as the commonest

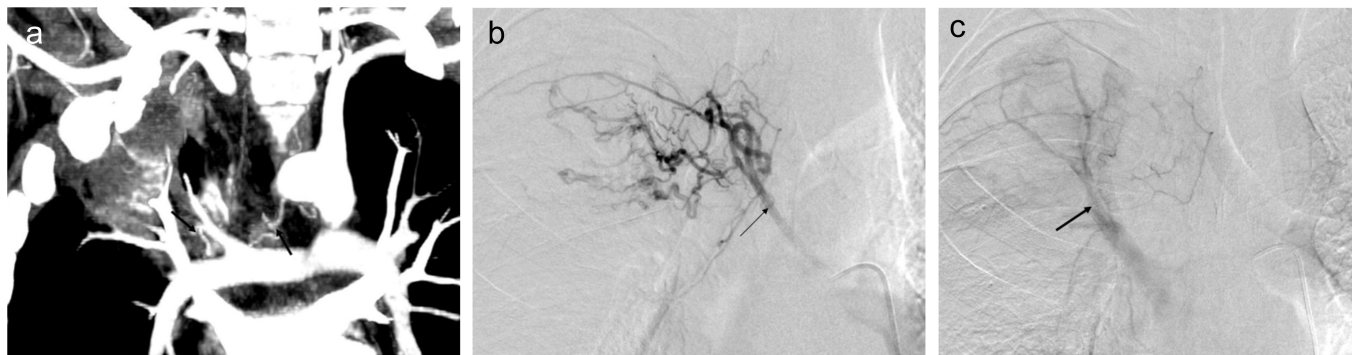


Figure 4. a-c. Abnormal BAs contributing to SPS. Coronal MIP CTA image (a) of patient of shunting group with hemoptysis showing tortuous right bronchial artery which is forming clusters of vessels (*thin black arrows*) adjacent to right superior pulmonary vessels. Corresponding DSA images (b and c) show selective right bronchial artery run (*thin black arrow*) showing early opacification of pulmonary superior pulmonary vein (*thick black arrow*) suggestive of BPVS. MIP, maximum intensity images; CTA, computed tomography angiography; SPS, systemic to pulmonary vasculature shunting; DSA, digital subtraction angiography; BPVS, bronchial artery to pulmonary vein shunting.

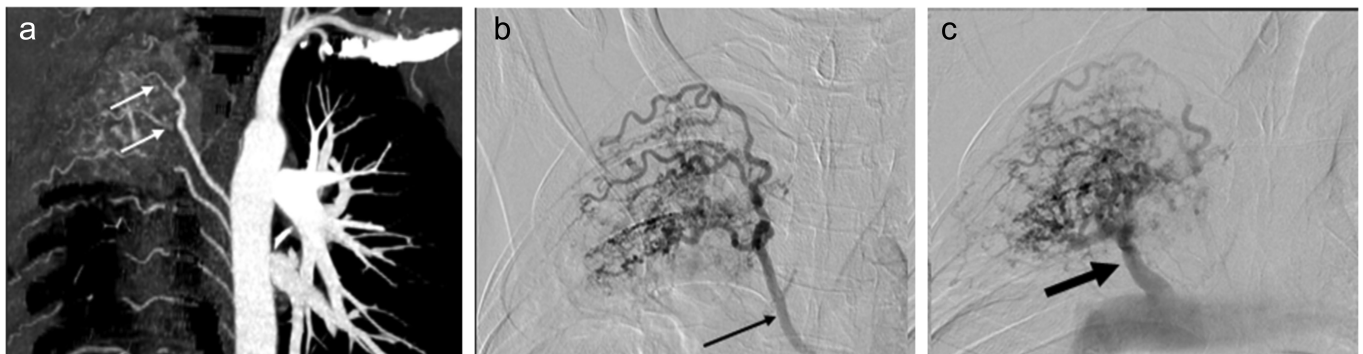


Figure 5 a-c. Abnormal NBSAs contributing to SPS, a-c. Coronal MIP CTA image (a) of patient of shunting group with hemoptysis showing dilated, tortuous right posterior intercostal arteries (white arrows). Corresponding DSA images (b, c) show selective right posterior intercostal artery (c) run (marked by thin black arrows), which shows parenchymal blush with early opacification of right pulmonary artery branches (marked by thick black arrows in image c) suggestive of NBPS. NBSAs, non-bronchial systemic arteries; MIP, maximum intensity images; CTA, computed tomography angiography; DSA, digital subtraction angiography; NBPS, non-bronchial systemic artery to pulmonary vasculature shunt.

cause of hemoptysis, particularly in TB endemic regions.^{2,17} However, the diagnostic criteria of CCPA have only been recently elucidated,¹⁵ and studies on BAE and/or preceding CTA have not addressed this entity adequately. Thus, CCPA is more likely to cause neovascularization, due to which shunts develop between pulmonary vasculature (pulmonary arteries or veins) and systemic arteries.¹³ On the other hand, in bronchiectasis, BAs get enlarged and

supply the dilated bronchioles walls, but SPS is not a dominant feature. This relationship between SPS and chronic inflammatory conditions like CCPA and cavities with aspergillomas has not been reported in the literature.

We found that all the arteries that were found to be abnormal on DSA had also been labeled as such on the pre-procedure DECTA. Further more, in the shunting group (group A), 70.6% of the abnormal

arteries were NBSAs, while these constituted 36.5% of the abnormal arteries in the non-shunting group. The commonest among these were posterior intercostal arteries. Non-bronchial systemic arteries enter the lung parenchyma through inferior pulmonary ligaments or adjacent adherent pleura. In CCPA, there is active, chronic parenchymal inflammation and pleural thickening that surround the chronic cavity housing aspergilloma. This leads to neovascularization and collateral formation from systemic arteries, the majority of these being recruited from posterior intercostal arteries and branches of the subclavian artery.^{12,14} Previous investigators have also stated that underlying chronic lung diseases like pulmonary TB and its sequelae, with or without aspergilloma, have extensive vascular abnormalities.¹⁰ Non-streaky hemoptysis in these patients may be due to new collateral formation and neovascularization due to persistent pulmonary inflammation.^{13,18}

In the non-shunting group (group B), on the other hand, 63.5% of abnormal systemic arteries were BAs, and the majority of these patients had bronchiectasis with or without fibrosis. Bronchiectasis usually occurs secondary to chronic or recurrent infection, aspiration, or airway obstruction. Recurrent airway inflammation leads to bronchial artery dilatation and neovascularization; therefore, BAs are the main source of hemoptysis in bronchiectasis as compared to NBSAs which dominate in chronic parenchymal inflammation.^{10,14,18}

Digital subtraction angiography remains the gold standard, and other studies have shown that CTA may miss few of the arteries which can be normal for CTA, but in our study, all arteries that were labeled

Systemic arteries	Group A (shunting group, n = 98)	Group B (non-shunting group, n = 89)
a) BAs	Total arteries = 88	Total arteries = 106
1) RICBT	41 (46.6%)	42 (39.6%)
2) RBA	8 (9.1%)	11 (10.4%)
3) LBA	16 (18.2%)	34 (32.1%)
4) CBA	23 (26.1%)	19 (17.9%)
b) NBSAs	Total arteries = 212	Total arteries = 61
1) Subclavian artery	2 (0.9%)	0
2) Internal mammary artery	40 (18.8%)	14 (22.9%)
3) Costocervical artery	22 (10.4%)	2 (3.3%)
4) Thyrocervical artery	1 (.5%)	0
5) Lateral thoracic artery	29 (13.7%)	1 (1.6%)
6) Superior thoracic artery	4 (1.9%)	0
7) Inferior phrenic artery	16 (7.6%)	5 (8.2%)
8) Posterior intercostal artery	97 (45.7%)	39 (63.8%)
9) Other*	1 (0.5%)	0

BAs, bronchial arteries; NBSAs, non-bronchial systemic arteries; DECTA, dual-energy computed tomography angiography; RICBT, right intercostobronchial artery; RBA, right bronchial artery; LBA, left bronchial artery; CBA, common bronchial artery.
*Collateral arising from common hepatic artery and supplying the left lower lobe bronchiectasis.

Table 3. Abnormal BAs and NBSAs on DSA in both groups

Systemic arteries	Group A (shunting group, n = 98)	Group B (non-shunting group, n = 89)
a) BAs	Total arteries = 80	Total arteries = 105
1) RICBT	41 (51.2%)	44 (41.9%)
2) RBA	7 (8.8%)	11 (10.5%)
3) LBA	9 (11.2%)	32 (30.5%)
4) CBA	23 (28.8%)	18 (17.1%)
b) NBSAs	Total arteries = 161	Total arteries = 41
1) Subclavian artery	1 (.6%)	0
2) Internal mammary artery	36 (22.4%)	11 (26.8%)
3) Costocervical artery	19 (11.8%)	1 (2.4%)
4) Thyrocervical artery	1 (0.6%)	0
5) Lateral thoracic artery	22 (13.7%)	0
6) Inferior phrenic artery	5 (3.1%)	1 (2.4%)
7) Posterior intercostal artery	77 (47.8%)	28 (68.4%)

BA, bronchial artery; NBSA, non-bronchial systemic artery; DSA, digital subtraction angiography; RICBT, right intercostobronchial artery; RBA, right bronchial artery; LBA, left bronchial artery; CBA, common bronchial artery.

abnormal on DSA were also labeled abnormal on DECTA. However, the presence of SPS was missed in 9 (2.3%) arteries on DECTA. On DSA, abnormal parenchymal blush refers to the increased caliber of branches of the systemic artery supplying abnormal lung parenchyma. It was the most common abnormality seen in 100% of the patients in the shunting group and 75.3% of those in the non-shunting group. In those patients with SPS, there is the retrograde filling of pulmonary arteries/veins from systemic arteries (BAs or NBSAs). This occurs due to the difference in pressure gradient between systemic

and pulmonary circulations, with systemic circulation being the high-pressure system and hence “pushing” the blood. All (except one) arteries that were labeled as contributing to SPS in group A of our study also demonstrated shunting on DSA. One artery could not be cannulated during DSA, due to its tortuous course. However, in the non-shunting group, 9 arteries (in 8 patients) showed SPS on DSA, which had not been predicted on DECTA. We could not find any of the 3 signs on DECTA in these 8 patients, possibly the shunts were very small. Thus, selective arterial runs on DSA are the most accurate method of

assessing SPS and classifying them into BPAS, BPVS, and NBPS.

While due to neovascularization secondary to chronicity of disease and persistent inflammation, multiple vessels get recruited and form a cluster of vessels, which form some micro fistulae with pulmonary vessels of that lobe, that leads to SPS. These SPSs have often been held responsible for the reoccurrence of hemoptysis, identifying these SPSs further guides us to target arteries during BAE on a priority basis.^{10,12,19,20}

Non-tapering pulmonary vessels and cluster of systemic vessel signs were most frequently seen in patients of the shunting group on DECTA. Differential attenuation sign was also seen in more than two-thirds of patients in the shunting group. Identifying these signs of SPS on DECTA will help in identifying the likely site of the bleed. Several previous studies have shown signs of SPS on DSA, but to the best of our knowledge, no previous study has shown or described any sign of SPSs on CT angiography.^{13,21,22}

Recurrent hemoptysis leads to increased morbidity and mortality in patients with SPSs. Recurrent hemoptysis is caused either by the progression of underlying disease, recanalization, or previously embolized arteries or neovascularization due to chronic inflammatory conditions like CCPA.¹³ Recruitment of new vessels leads to formations of shunts with pulmonary vasculature.^{12,13} Although CTA accurately depicts the origin and numbers of abnormal systemic vessels responsible for hemoptysis, shunting on CT has not been reported.^{4,8,14} Systemic to pulmonary vasculature shunting and site of shunting are easily seen on DSA, while it has not been previously reported in the literature on CTA.

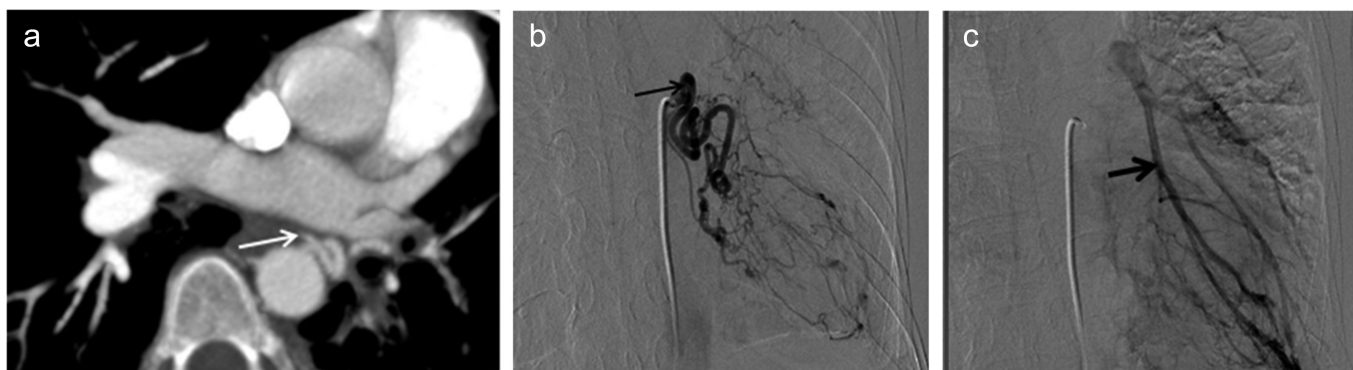


Figure 6. a-c. The discrepancy between DECTA and DSA. Axial thin MIP CTA image (a) shows a dilated, tortuous left bronchial artery (white arrow) in a non-shunting group patient with bronchiectasis. Corresponding DSA images (b, c) show selective left bronchial artery (thin black arrow) and opacification of the left inferior pulmonary artery (c, thick black arrow) suggestive of shunting (BPAS). DECTA, dual-energy computed tomography angiography; DSA, digital subtraction angiography; MIP, maximum intensity images; CTA, computed tomography angiography; DSA, digital subtraction angiography; BPAS, bronchial artery–pulmonary artery shunt.

The potential advantages of dual-energy CT over a single source include better visualization of vessels on angiograms, iodine mapping, and virtual non-contrast image generation.¹¹ Thus, identifying the site of SPS and abnormal vessels responsible for the same may be better depicted on CT angiograms generated through low kVp images of the DECT dataset. We, however, did not evaluate these advantages of DECTA in this study; rather as a practice use, a CT angiogram was generated from the low-energy dataset for interpretation. However, these signs of SPS can also be appreciated on conventional single-energy MDCT angiography, using a single-phase split-bolus contrast technique.¹⁴ It is important to identify whether shunting is occurring in the pulmonary artery or vein. Bronchial artery to pulmonary artery shunting is left-to-right shunting that leads to increased blood flow into the pulmonary arterial system which changes the blood flow dynamics and hence the oxygen saturation in the affected pulmonary artery. On the other hand, BPVS may increase the risk of non-target systemic artery embolization, but we did not encounter any major complications after BAE in our patients.

According to a previous study, BAE was able to achieve an immediate control rate of 97.8% and a long-term hemostasis rate of 85.4% in patients with SPS and hemoptysis.¹³ In our study, we found immediate control of hemoptysis in 99.4% of patients, but we did not analyze the long-term follow-up in these patients.

On CTA, just identifying the abnormal arteries is not enough, rather it is imperative to identify those arteries which are causing active bleeding in the patient. Signs of SPSs not only help in identifying these abnormal systemic vessels but also guide us during DSA to embolize these systemic vessels on a priority basis; this might reduce procedure time and radiation exposure.^{13,23-25}

Limitations of this study are as follows: (1) no correlation was done with bronchoscopy or with symptom-free survival in both the groups; (2) we did not assess interobserver agreement in analyzing the imaging findings; (3) this is a single-centered and retrospective study, and larger prospective and multicenter studies are needed to further validate these signs of shunting on CTA; (4) very small shunts are difficult to detect on DECTA; (5) it is difficult to discern

the presence of shunting in the pulmonary vein versus the pulmonary artery using DECTA in some cases.

In conclusion, our study postulated and validated the signs of SPSs on DECTA after comparison with DSA in patients with non-streaky hemoptysis. These signs may predict the active site of bleeding on DECTA and guide us during the BAE to target specific arteries that are causing shunting in a particular lobe or segment of the lung on a priority basis. A knowledge of these signs on DECTA not only provides targeted, faster, and accurate embolization but also limits the duration of BAE and subsequently reduces the radiation exposure to the patient as well as to the operator. However, DSA remains as a gold standard in detecting small SPSs and locating shunting to either pulmonary artery or vein in a few cases that could not be identified on DECTA.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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