CT imaging of pulmonary veins in patients with acute pulmonary embolism

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Learning objectives

- To describe non-enhancements of pulmonary veins detected in a group of 98 patients with acute pulmonary embolism.

- To learn changes in pulmonary veins in acute pulmonary embolism, its pathological explanation and its resolution in follow-up.

- To describe frequency and clinical significance of these non-enhancements of pulmonary veins.

Background

Pulmonary embolism is defined by the presence of a thrombus in the pulmonary arteries, seen in CT as intraluminal filling defects.

Thrombosis of pulmonary veins have been described in the early postoperative period following pulmonary lobectomy and lung transplantation, and as a rare complication of primary or secondary tumours of the lung, most commonly associated with bronchogenic carcinoma but also present in metastatic sarcoma, chondrosarcoma, and angiosarcoma [5, 6, 7]. It is important to realise that pulmonary embolism is not a cause of thrombosis of pulmonary veins although it can be visualized in CT a lack of enhancement of pulmonary veins in this setting but without an increase of the venous diameter.

Multiple factors can play a role in the physiopathology of thrombosis in pulmonary veins [5, 6, 7]: mechanical compression of the veins (like lung cancer or auricular myxoma) or increase of pressure in left atrium (like mitral insufficiency), both resulting in a difficulty of drainage and stasis, hypercoagulable state (like sickle cell disease),
and iatrogenic or immunologic damage to the endothelium that activates coagulation (like vein wall damage secondary to Swan-Ganz catheter, after surgery or interventional procedures as ablations, or after tumour invasion through the vein wall). None of these factors are present in pulmonary embolism, therefore it can be deduced that the lack of enhancement of pulmonary veins in pulmonary embolism is a pitfall.

**False filling defects within pulmonary veins** are one of the pitfalls in diagnosis of pulmonary embolism. According to literature [1, 2, 3, 4, 5], they are caused by flow artifacts due to poor mixture of unenhanced blood and contrast material or by early performance of CT after contrast material injection (time delay too short to enhance the veins).

There are some **strategies to recognise this pitfall** [1, 2, 3, 4, 5]. The knowledge of **vascular anatomy** is the most reliable method to distinguish arterial thrombus from pseudofilling venous defect. Pulmonary arteries run adjacent to the bronchi with the exception of the apical-posterior segment of the left upper lobe and the lingular arteries, which can course independently for a short distance before rejoining the bronchi. However, pulmonary veins are located within interlobular septa. It is useful to follow pulmonary arteries on contiguous sections to avoid misinterpretation of unenhanced pulmonary vein for an occluded pulmonary artery, and to follow pulmonary veins to confirm its drainage to the left atrium.

The **scan delay** is chosen depending on the objective of the CT. In pulmonary embolism, it is selected to study arterial pulmonary anatomy. This time is too short to enhance pulmonary veins resulting in complete pseudofilling defects or too short to obtain a correct mixture between opacified and unopacified blood resulting in partial pseudofilling defects (a dense peripheral rim of contrast medium surrounding a central hypoattenuated area that corresponds to venous return not opacified). The solution would be to acquire CT in a longer scan delay to evaluate better pulmonary veins and cardiac chambers, but with a suboptimal quality of arterial opacification to assess pulmonary arteries which are the main objective.

The **window setting** influences the visualization of vascular contrast. Dense undiluted contrast material can obscure emboli or pseudofilling defects in a standard mediastinal window widths (300-450) and level (30-50). Its modification towards wider widths (700) and high level (100) can be useful to observe clearly emboli or pseudofilling defects through contrast material and be able to differentiate between a sharply margmated embolus and an ill-defined artifact.
Other **artifacts related to the patient** have to be considered, as breathing and cardiac motions, which affect both enhancements of pulmonary arteries and veins.

However, there are few references about findings in pulmonary veins in acute pulmonary embolism. The **aim of this exhibit** is to review data of 98 patients diagnosed of pulmonary embolism to detect focal asymmetrical non-enhancements of pulmonary veins in acute setting.

**Imaging findings OR Procedure details**

98 patients studied with 64-slice multidetector row CT scanner and diagnosed of acute pulmonary embolism have been reviewed to search **focal asymmetrical non-enhancements within pulmonary veins** in acute setting.

In our study, we take into account those focal asymmetrical non-enhancements within pulmonary veins which drain the pulmonary arteries obstructed due to they can be explained by the physiologic mechanism proposed. But, we don't consider non-enhancements of pulmonary veins without any correlation with the location of thrombus within pulmonary arteries because they are pitfalls described previously in literature.

We have found that there are **two kinds** of focal asymmetrical non-enhancements: partial and complete (Figure 1). **Partial non-enhancements** are present in 17 of 98 patients (17.3%) and **complete non-enhancements** are present in 23 of 98 patients (23.5%). The rest of patients (58 of 98 patients, 59.2%) don't exhibit any imaging anomaly in pulmonary veins.

It has been found a **relationship between arterial clot burden and the intensity of non-enhancements** in pulmonary veins (Figures 2, 3). In this way, 19 of 23 complete non-enhancements (82.6%) have occurred in massive pulmonary embolisms, understanding as massive the affection of trunk and main pulmonary arteries. On the contrary, 11 of 17 partial non-enhancements (64.7%) have occurred in lobar or segmental pulmonary embolisms.
In our series, the location of non-enhancements is showed in figures 4, 5. Right inferior pulmonary vein (RIPV) is the location more frequent, being detected in 31 of 40 cases (77.5%): 15 cases only in RIPV, 10 cases in association with left inferior pulmonary vein (LIPV), 4 cases in association with right superior pulmonary vein (RSPV), 1 case in association with left superior pulmonary vein (LSPV) and 1 case in association with LIPV and RSPV. Left inferior pulmonary vein (LIPV) is the second location more frequent, being detected in 18 of 40 cases (45%). They are less frequently found in right superior pulmonary vein (10 cases) and left superior pulmonary vein (3 cases).

There are some examples in figures 6 - 13.

The existence of these venous non-enhancements in pulmonary embolism can be explained by the knowledge of pulmonary circulation. The presence of thrombus in a pulmonary artery causes an increase of pulmonary resistance that results in changes in vascular flux making it slower, therefore there are a delayed arrival of contrast material to pulmonary veins not being enhanced in the moment of CT acquisition. Although the imaging can mimic a pulmonary vein thrombosis, it has to be considered a physiologic consequence secondary to hemodynamic flux slowness.

Based on this pathologic theory, it can be easily understood that inferior pulmonary veins are the main location of non-enhancements: perfusion is greater in inferior lobes, then arterial thrombus migrates more frequently to inferior lobes and consequently venous non-enhancements secondary to flux changes are more frequent in inferior pulmonary veins. Also, it can explain the relationship between arterial clot burden and venous non-enhancement: a greater thrombus implies slower flux, therefore complete non-enhancements predominate in massive pulmonary embolisms.

These focal asymmetrical non-enhancements resolve in follow-up because hemodynamic flux returns to normal due to resolution of pulmonary arteries occlusion with correct management and treatment.

Images for this section:
Fig. 1: Frequency of venous non-enhancements
Fig. 2: Relationship between arterial clot burden and intensity of non-enhancements
**FOCAL ASYMMETRICAL NON-ENHANCEMENTS IN PULMONARY VEINS**

Relationship between arterial clot burden and the existence of partial or complete non-enhancements

- 23 complete non-enhancements
- 19 in massive EP (82.6%)
- 4 in lobar/segmental EP (17.4%)
- 17 partial non-enhancements
- 11 in lobar/segmental EP (64.7%)
- 6 in massive EP (35.3%)

**Fig. 3:** Relationship between arterial clot burden and intensity of non-enhancements
FOCAL ASYMMETRICAL NON-ENHANCEMENTS IN PULMONARY VEINS

LOCATION

In absolute terms, this is the distribution:

- Right inferior pulmonary vein (RIPV) 31 cases (77.5%)
- Left inferior pulmonary vein (LIPV) 18 cases (45%)
- Right superior pulmonary vein (RSPV) 10 cases (25%)
- Left superior pulmonary vein (LSPV) 3 cases (7.5%)

Fig. 4: Location of venous non-enhancements
FOCAL ASYMMETRICAL NON-ENHANCEMENTS IN PULMONARY VEINS

LOCATION

The distribution in cases is:

- RIPV: 15 cases
- LIPV: 2 cases
- RIPV + LIPV: 10 cases
- RIPV + LIPV + RSPV: 1 case
- RSPV + RIPV: 4 cases
- LSPV + LIPV: 2 cases
- RSPV + LIPV: 3 cases
- LSPV + RIPV: 1 case
- RSPV: 2 cases

**Fig. 5:** Location of venous non-enhancements
Fig. 6
Fig. 8
Fig. 9
Fig. 10
Fig. 11
Fig. 12
Fig. 13
Conclusion

Focal asymmetrical non-enhancements of pulmonary veins are found in draining pulmonary veins of obstructed pulmonary arteries and resolved in follow-up parallel to arterial thrombus resolution. They are probably caused by delayed arrival of contrast material to pulmonary veins due to slow contrast flux through obstructed pulmonary arteries. This pathologic theory can be proved by the fact that inferior pulmonary veins are the main location and the relationship between arterial clot burden and intensity of venous non-enhancements.

These findings can mimic pulmonary vein thrombosis but they should be considered physiologic consequences to avoid their misinterpretation as a pathologic condition.

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References


