Published in final edited form as:

AJR Am J Roentgenol. 2013 April ; 200(4): 791–797. doi:10.2214/AJR.12.8624.

The Rate of Resolution of Clot Burden Measured by Pulmonary CT Angiography in Patients With Acute Pulmonary Embolism

Ayaz Aghayev¹, Alessandro Furlan¹, Amol Patil¹, Serter Gumus¹, Kyung Nyeo Jeon^{1,2}, Bumwoo Park¹, and Kyongtae T. Bae¹

¹Department of Radiology, University of Pittsburgh Medical Center, 3362 Fifth Ave, Pittsburgh, PA 15213.

²Department of Radiology, College of Medicine, Gyeongsang National University, Jinju, Korea.

Abstract

OBJECTIVE—The purpose of this article is to quantitatively assess the rate of resolution of clot burden detected on pulmonary CT angiography (CTA) in patients with acute pulmonary embolism (PE).

MATERIALS AND METHODS—We evaluated 111 consecutive patients (55 men and 56 women) in a retrospective cohort who were diagnosed with PE by pulmonary CTA and had at least one follow-up pulmonary CTA within 1 year. Two radiologists in consensus measured the volume of each clot using a semiautomated quantification program. Semiquantitative measures of clot burden were also computed. The resolution rates of the total clot volume, as well as clot volumes of the central (main and lobar) and peripheral vessels (segmental and subsegmental), were analyzed.

RESULTS—The mean ($\pm$ SD) clot volume per study was $3403.3 \pm 6505.6 \text{ mm}^3$ at baseline and $531.6 \pm 2383.5 \text{ mm}^3$ at the follow-up pulmonary CTA. Overall, 85 patients (77%) showed complete resolution at the follow-up pulmonary CTA. Complete resolution was seen in 17 of 30 patients (56.7%) at a follow-up interval of 1–14 days, in 24 of 31 patients (77.4%) at 29–90 days, and in 32 of 34 patients (94.1%) after 90 days. The total clot volume measurements summed for all patients decreased by 78% (central clot, 69.4%; peripheral clot, 86.0%) at 14 days, by 96.6% (central clot, 93.4%; peripheral clot, 100%) at 90 days, and by 97.7% (central clot, 95.9%; peripheral clot, 100%) after 90 days.

CONCLUSION—Clot burden resolved completely in 77% of patients during the follow-up period. Our analysis showed that clots resolved faster in the peripheral arteries than in the central pulmonary arteries.

Keywords

pulmonary CT angiography; pulmonary embolism; resolution

Pulmonary CT angiography (CTA) has been established as the first-line imaging modality for the diagnosis of acute pulmonary embolism (PE) in clinical practice [1]. In addition to its role as a superb diagnostic tool, pulmonary CTA contains a wealth of information, including characteristics of the clot that may be used as biomarkers to improve treatment and clinical management of patients diagnosed with PE.

Anticoagulation treatment is effective in preventing such complications as chronic thromboembolic pulmonary hypertension [2], thus reducing the recurrence rate of venous thromboembolism, including PE and deep vein thrombosis. However, prolonged anticoagulation treatment is associated with complications such as bleeding. There is increased interest in determining the duration of anticoagulant treatment according to individual risk for venous thromboembolism recurrence [3, 4], and clot burden has been indicated as a predictor of recurrent PE [3]. In this regard, clot burden measured on pulmonary CTA images may provide information that would help guide duration of anticoagulant therapy.

Clot burden can be assessed semiquantitatively, using various scoring systems, or fully quantitatively, via clot volume quantification. Qanadli et al. [5] and Mastora et al. [6] proposed semiquantitative scoring methods to assess the severity of PE. These scores, mainly reflecting the distribution of clots, were formulated by tabulating the presence and degree of arterial obstruction of clot in individual branches of pulmonary arteries. Recently, two studies proposed direct quantification of blood clot volume from pulmonary CTA images [7, 8]. With the use of advanced image-processing techniques, clots were segmented and volumetrically measured directly from pulmonary CTA images.

Information about the rate of clot resolution is important because it may facilitate objective diagnosis when patients with PE return with symptoms possibly due to recurrent or residual PE [9]. Previous studies have focused on the evolution of PE on CT using a qualitative approach (i.e., complete resolution vs persistent clot) [10–14]. According to these studies, complete resolution of PE occurs in 50–90% of patients, with a reported interval of 1–6 months. However, to our knowledge, no study has yet quantified the change in clot burden in follow-up CT studies. An approach based on a precise measurement of clot volume may offer an improved assessment of evolution of PE and efficiency of therapy. Thus, the purpose of this study was to quantitatively assess the resolution rate of clot burden on pulmonary CTA imaging in patients with acute PE.

Materials and Methods

This retrospective study was approved by the institutional review board and was HIPAA compliant. Patients' informed consent was waived. All pulmonary CTA images and reports were retrieved from the institutional PACS (iSite, Philips Healthcare) and were deidentified before analysis using an institutional honest broker system.

Study Population

A search through the institutional medical archive system using a 12-month interval (January through December 2007) was conducted. Subjects were initially identified via pulmonary CTA examination codes, and their radiology reports were retrieved and reviewed by two trained radiologists. There were 5425 pulmonary CTA studies performed at our institution for evaluation of PE during the study period. Pulmonary CTA studies reported as positive for PE and with subsequent follow-up pulmonary CTA studies were retrieved. Among 635 patients diagnosed with acute PE on baseline pulmonary CTA, 121 (19%) had at least one follow-up imaging study available; for patients with multiple follow-up studies available ($n = 26$), only the latest follow-up pulmonary CTA study was considered for the current study. Patients with new foci of PE at follow-up pulmonary CTA ($n = 10$) were excluded. New foci of PE were defined as clots identified at new locations different from those in the original pulmonary CTA study.

We analyzed 10 patients with new foci of PE to assess whether the clots were real new foci or distal propagations from the previous one. In the analysis of individual patients, three

patients had new foci of clot in the contralateral (right and left) pulmonary arteries. Three other patients had new clots in the central pulmonary arteries on the follow-up scan, whereas only segmental or lobar clots were present on the baseline pulmonary CTA. Another four patients presented with segmental and subsegmental clots at baseline but clots were seen in the segmental, lobar, or main arteries on the follow-up scan. Thus, we determined no apparent distal propagation of clots in the patients with new foci of PE.

Our final population included 111 patients (56 women and 55 men; mean [$\pm$ SD] age, 51.6 ± 17.8 years; range, 18–94 years). The mean interval between baseline and follow-up pulmonary CTA was 65 days (range, 1–312 days). All patients received anticoagulation therapy.

Pulmonary CTA Imaging Protocol

Baseline and follow-up pulmonary CTA imaging studies were performed on 64-MDCT ($n = 107$ patients) or 16-MDCT ($n = 4$ patients) scanners using a standard pulmonary CTA protocol for PE, with the following imaging parameters: detector width, 64×0.625 or 16×1.25 mm; section thickness, 1.25 mm; rotation time, 0.5 second; 120 kVp; and 380 mA. CT images were reconstructed with both standard soft-tissue and lung kernel algorithms. Contrast enhancement was achieved by injecting 125 mL of ioversol contrast medium (Optiray 350, Mallinckrodt Imaging), with a power injector at a rate of 4–5 mL/s. The scanning delay was determined by using a bolus-tracking method, in which a region of interest was placed on the main pulmonary artery or a 20-second delay after the start of the contrast medium injection.

Imaging Analysis

For each patient, deidentified images from baseline and follow-up pulmonary CTA examinations were transferred to a PC workstation equipped with an in-house–developed DICOM reader. The imaging studies were analyzed by two radiologists (each with 4 years of experience in chest and body CT) in consensus, and any disagreement was resolved by the senior investigator (with 15 years of experience in interpretation of pulmonary CTA images). Readers assessed PE on pulmonary CTA images by detecting the presence of an endoluminal central filling defect partially or completely occluding pulmonary arteries [15]. A standard mediastinal window was used, with readers allowed to adjust the window and level settings for optimal vessel visualization according to the windowing scheme published in a previous study [16]. For each pulmonary CTA imaging study, the number, location, and degree of arterial obstruction (1, < 25%; 2, 25–49%; 3, 50–74%; 4, 75–99%; and 5, 100%) of each clot was recorded; on the basis of these data, the Qanadli score (i.e., pulmonary arterial obstruction index) and Mastora score were calculated as described elsewhere [5, 6]. Moreover, readers used a semiautomated segmentation program to segment each clot from pulmonary CTA images and measure clot volume (cubic millimeters). Accuracy and reproducibility of the clot volume measurement have been previously reported [8].

Statistical Analysis

Measures of clot burden included clot volume and semiquantitative scores (Qanadli and Mastora index). Changes in clot burden between the baseline and the follow-up pulmonary CTA were expressed as a percentage. The interval between baseline and follow-up pulmonary CTA imaging studies was further divided into four groups: 1–14, 15–28, 29–90, and more than 90 days. Changes in clot volume and Qanadli and Mastora index were calculated for each interval subgroup. Significance of the difference in clot burden between baseline and follow-up pulmonary CTA was assessed using paired Student *t* test. A *p* value of less than 0.05 was considered to indicate a statistically significance difference (SPSS statistics software version 19, IBM). The percentage change in clot volume was calculated

as follows: $[(\text{baseline volume} - \text{follow-up volume}) / \text{baseline volume}] \times 100$. Then that percentage was used to divide the patients into three groups of clot resolution: minimal (0–50%), significant (51–99%), and complete (100%).

Finally, patients were divided in two subgroups according to the most proximal extension of the clot on the baseline pulmonary CTA. Changes in clot volume were calculated for patients with PE extending into the central pulmonary arteries (i.e., pulmonary artery trunk, main right and left pulmonary arteries, right and left interlobar pulmonary arteries, and lobar arteries) and for patients with PE limited to the peripheral pulmonary arteries (i.e., segmental and subsegmental pulmonary arteries).

Results

Patients' demographic characteristics and comorbidities are summarized in Table 1. Quantitative measurements of clot burden at baseline and follow-up pulmonary CTA are shown in Table 2. The mean interval time between the baseline and follow-up pulmonary CTA examinations was 68 days (range, 1–312 days). The mean clot volume per study was $3403.3 \pm 6505.6 \text{ mm}^3$ (range, 6.8–31,683.4 mm^3) at baseline and $531.6 \pm 2383.5 \text{ mm}^3$ (range, 0–17,027.1 mm^3) at follow-up pulmonary CTA ($p < 0.001$). The mean percentage change in clot volume was 91%. The mean Qanadli score changed from 6 ± 6.3 at baseline to 1.4 ± 3.6 at follow-up ($p < 0.001$), representing a mean percentage change of 76%. The mean Mastora score changed from 13.8 ± 18.9 at baseline to 1.7 ± 5.9 at follow-up ($p < 0.001$), representing a mean percentage change of 87%. The mean percentage changes in clot volume, as well as Qanadli and Mastora indexes, over time are plotted in Figures 1A and 1B. The mean percentage clot volume reduction from baseline to follow-up was 90.1% within 28 days and 97.7% when the imaging follow-up study was acquired more than 90 days after baseline. The mean percentage decline in Qanadli and Mastora score from the baseline to the follow-up CTA was 74.6% and 76.5%, respectively, within 28 days, and 97.6% and 97.9%, respectively, when the imaging follow-up study was acquired more than 90 days after baseline.

The numbers of patients with different degrees of clot resolution imaged at different follow-up time intervals are shown in Table 3. Overall, 85 patients (77%) showed a complete resolution on follow-up pulmonary CTA. Complete resolution was seen in 17 of 30 patients (56.7%) at 14 days after baseline pulmonary CTA, in 12 of 16 patients (75%) at 15–28 days, and in 24 of 31 patients (77.4%) at 29–90 days. Within the first 3 months, 53 of 77 (68.8%) patients showed complete clot resolution; finally, 32 of 34 patients (94.1%) showed complete clot resolution after 90 days (Figs. 2 and 3).

At baseline pulmonary CTA, the most proximal involved pulmonary artery was the central artery in 58 patients (mediastinal, $n = 25$; lobar, $n = 33$) and the peripheral artery in 53 patients (segmental, $n = 32$; subsegmental, $n = 21$). The numbers of patients with different degrees of clot resolution at different follow-up time intervals are shown in Table 3 for clots involving the central and peripheral pulmonary arteries. The percentage of patients with complete resolution was 42.9% (6/14) for central and 68.8% (11/16) for peripheral clots in 14 days, 66.7% (6/9) for central and 85.7% (6/7) for peripheral clots at 15–28 days, 56.3% (9/16) for central and 100% (15/15) for peripheral clots at 29–90 days, and 89.5% (17/19) for central and 100% (15/15) for peripheral clots at more than 90 days. The mean percentage volume changes of the central and peripheral clots at the same intervals are shown in Figures 4A and 4B. The mean percentage volume change was 69.4% for central and 86.0% for peripheral clots at 14 days, 88.0% for central and 92.8% for peripheral clots at 15–28 days, 93.4% for central and 100% for peripheral clots at 29–90 days, and 95.9% for central and 100% for peripheral clots at more than 90 days.

Discussion

The quantitative volumetric and semiquantitative analysis performed in our study revealed that clot burden in patients with acute PE progressively decreases at pulmonary CTA imaging follow-up. The mean percentage decrease in clot volume was 91%, whereas the mean percentage decrease in Qanadli and Mastora scores was 76% and 87%, respectively. Among the 111 patients with acute PE included in our final population, 19 (17%) showed a significant decrease in clot burden, whereas 85 (77%) showed completely resolved PE at follow-up pulmonary CTA. Several studies published variable degrees of complete PE resolution by comparing the initial and follow-up pulmonary CTA. Although some studies [11, 13, 15] reported relatively higher proportions of complete resolutions (90%, 91%, and 85%), other studies [10, 12] published lower degrees (48% and 32%). However, these data are difficult to compare between them or with our study, because time intervals for follow-up pulmonary CTA were not either specified or standardized.

In our study, follow-up pulmonary CTA studies at 15–28 days after baseline showed that clot volume was reduced by 90.1%; two of 16 patients (12.5%) had a significant reduction in clot volume, with a complete resolution in 12 patients (75%). A complete resolution was seen in 17 of 30 patients (56.7%) at 14 days after the baseline pulmonary CTA, 24 of 31 patients (77.4%) at 29–90 days, and 32 of 34 patients (94.1%) after 90 days. This suggests that a repeated pulmonary CTA scan 1 month after the initiation of therapy may be useful to assess a substantial reduction of clot burden and the efficacy of therapy. On the other hand, the majority of conventional pulmonary angiography literature [17–22] reported that a complete resolution was seen in fewer than 50% of patients within 30 days. Only one study [23] published a considerably faster resolution, a complete resolution in 85% of patients at 20 days of follow-up angiograms. The rate of clot resolution in this article appears comparable to our study results, despite differences in imaging modality.

The rate of complete clot resolution measured on lung perfusion scans was reported to be highly variable. One study [24] showed complete resolution in 7% of patients within 2 weeks, whereas another study found complete resolution in 43% of patients during the same follow-up period [24, 25]. Two lung perfusion scan studies at 3 months of follow-up showed complete clot resolution in 34% [26] and 57% [25] of patients, respectively. In a recent study, complete clot resolution was seen in 72% of patients at a 9-month follow-up lung perfusion scan [14]. Complete resolution on a 1-year follow-up lung perfusion scan was reported in 65–85% of patients [27–29]. This wide range of discrepancies in the rate of complete resolution between the published lung perfusion scan and pulmonary CTA studies including ours is not surprising, given the large interobserver difference in the interpretation of the presence of residual clot in lung perfusion scan [30].

The relative reduction in semiquantitative scores (Qanadli and Mastora scores) at follow-up pulmonary CTA showed a trend similar to that of the clot volume changes. However, the overall mean percentage change in clot volume of 91% was closer to the mean percentage changes in the Mastora score of 87% than to the Qanadli score of 76%. Moreover, the mean percentage change in clot volume of 78.2% within the first 2 weeks of follow-up was much closer to the mean percentage changes in the Mastora score of 75.2% than the Qanadli score of 42.5% during the same period. This discrepancy between Mastora and Qanadli scores is expected because the Mastora scoring system is designed to improve the Qanadli scoring system by considering finer degrees of clot burden in each scored pulmonary artery [5, 6].

The resolution rate of the peripheral clots was consistently lower than that for the central clots when the rate was evaluated in terms of both the numbers of patients showing complete resolution and the clot volume percentage changes. Although peripheral clots resolved

completely at 1-month follow-up CT, trace of central clots persisted (the mean percentage reduction of central clot volume was 93.4% at 29–90 days and 95.9% after 3 months). To the best of our knowledge, there is only one study [11] reporting the rate of resolution of PE according to the order of vessels. In that study, the authors concluded that PEs resolved faster in the main and lobar pulmonary arteries than in the segmental branches. This appears to contradict our results that peripheral clots resolved faster than clots in the main and lobar arteries. We postulate that this seemingly contradictory result is from the two main differences between their study and ours. First, our study cohort includes a larger number of isolated segmental and subsegmental clots than the other study. Peripheral clots without larger central clots would resolve faster because their resolution is not hindered by upstream central clots obstructing the downstream distal blood flow. An underrepresentation of peripheral clots without central clots in the study cohort may result in faster resolution of central than peripheral clots, as reported in the previous study [11]. For example, subsegmental clots were not included in that study. Second, that study was primarily based on qualitative evaluation without the use of quantitative clot measurement information. Quantitative analysis of the changes in the clot volume may offer more precise information regarding the resolution of clots associated with the order of pulmonary arteries.

Our study has several limitations. First, the study was retrospective; therefore, we could not control the follow-up periods of pulmonary CTA. Although we think that the given distribution of follow-up periods was reasonably broad for the evaluation of the trend of the clot resolution rate, a rigorously designed prospective study, with appropriate institutional review board approval and including a low-dose protocol, is required to confirm and generalize our findings. It would be ideal to follow the same subjects with multiple follow-up pulmonary CTA studies to reduce subject variability for the accurate measurement of clot resolution. Second, because of the retrospective design of the study and because current clinical guidelines do not necessarily recommend follow-up pulmonary CTA for patients with PE, only 19% of patients had follow-up scans. These patients might have specific clinical indications or symptoms requiring follow-up pulmonary CTA compared with the majority of PE patients, who did not have follow-up pulmonary CTA. However, this comparative clinical information was not available to investigate in our study. Third, because of the retrospective design of the study, we had limited clinical information for the patients. It would be interesting to investigate the effect of clinical data and treatment regimens on clot resolution. Patients with ineffective anticoagulation therapy would delay clot resolution and even recurrent PE. We also did not specifically evaluate clinical indications for the follow-up pulmonary CTA. Fourth, we only evaluated the volume and not other morphologic characteristics of clots. For example, in theory, not only the volume but also the surface area of clot may play an important role in clot resolution rate, provided that clots with higher surface areas would be exposed to higher thrombolytic effect of heparin and anticoagulant circulating in the blood. Similarly, no attempt was made to divide and analyze clots at different parts of the lung, such as the upper, middle, and lower lungs.

In conclusion, during the follow-up period of our retrospective study, the majority of patients (77%) showed complete resolution of clot burden. Within the first 3 months, 68.8% of patients (53/77) showed complete clot resolution, whereas after 3 months, 94.1% of patients (32/34) showed complete clot resolution. Our volumetric analysis showed that clots resolved faster in the peripheral than in the central pulmonary arteries.

Acknowledgments

This study was supported by the National Institutes of Health (grant HL095115).

References

1. Remy-Jardin M, Pistolesi M, Goodman LR, et al. Management of suspected acute pulmonary embolism in the era of CT angiography: a statement from the Fleischner Society. *Radiology*. 2007; 245:315–329. [PubMed: 17848685]
2. Pengo V, Antonio WA, Lensing MD, et al. Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism. *N Engl J Med*. 2004; 350:2257–2264. [PubMed: 15163775]
3. Agnelli G, Becattini C. Treatment of DVT: how long is enough and how do you predict recurrence. *J Thromb Thrombolysis*. 2008; 25:37–44. [PubMed: 17906973]
4. Kahn SR, Lim W, Dunn AS, et al. Prevention of VTE in nonsurgical patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed—American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012; 141(suppl 2):e195S–e226S. [PubMed: 22315261]
5. Qanadli SD, El Hajjam M, Vieillard-Baron A, et al. New CT index to quantify arterial obstruction in pulmonary embolism: comparison with angiographic index and echocardiography. *AJR*. 2001; 176:1415–1420. [PubMed: 11373204]
6. Mastora I, Remy-Jardin M, Masson P, et al. Severity of acute pulmonary embolism: evaluation of a new spiral CT angiographic score in correlation with echocardiographic data. *Eur Radiol*. 2003; 13:29–35. [PubMed: 12541107]
7. Nakada K, Okada T, Osada H, Honda N. Relation between pulmonary embolus volume quantified by multidetector computed tomography and clinical status and outcome for patients with acute pulmonary embolism. *Jpn J Radiol*. 2010; 28:34–42. [PubMed: 20112091]
8. Furlan A, Patil A, Park B, Chang CC, Roberts MS, Bae KT. Accuracy and reproducibility of blood clot burden quantification with pulmonary CT angiography. *AJR*. 2011; 196:516–523. [PubMed: 21343492]
9. Nijkeuter M, Hovens MM, Davidson BL, Huisman MV. Resolution of thromboemboli in patients with acute pulmonary embolism: a systematic review. *Chest*. 2006; 129:192–197. [PubMed: 16424432]
10. Remy-Jardin M, Louvegny S, Remy J, et al. Acute central thromboembolic disease: posttherapeutic follow-up with spiral CT angiography. *Radiology*. 1997; 203:173–180. [PubMed: 9122389]
11. Stein PD, Yaekoub AY, Matta F, et al. Resolution of pulmonary embolism on CT pulmonary angiography. *AJR*. 2010; 194:1263–1268. [PubMed: 20410413]
12. Van Rossum AB, Pattynama PM, Tjin ATE, Kieft GJ. Spiral CT appearance of resolving clots at 6 week follow-up after acute pulmonary embolism. *J Comput Assist Tomogr*. 1998; 22:413–417. [PubMed: 9606382]
13. López-Beret P, Pinto JM, Romero A, Orgaz A, Fontcuberta J, Oblas M. Systematic study of occult pulmonary thromboembolism in patients with deep venous thrombosis. *J Vasc Surg*. 2001; 33:515–521. [PubMed: 11241121]
14. Cosmi B, Nijkeuter M, Valentino M, Huisman MV, Barozzi L, Palareti G. Residual emboli on lung perfusion scan or multidetector computed tomography after a first episode of acute pulmonary embolism. *Intern Emerg Med*. 2011; 6:521–528. [PubMed: 21461909]
15. Wittram C, Maher MM, Halpern EF, Shepard JA. Attenuation of acute and chronic pulmonary emboli. *Radiology*. 2005; 235:1050–1054. [PubMed: 15833986]
16. Bae KT, Mody GN, Balfe DM, et al. CT depiction of pulmonary emboli: display window settings. *Radiology*. 2005; 236:677–684. [PubMed: 15972332]
17. Simon, M.; Sasahara, AA. Observations on the angiographic changes in pulmonary thromboembolism. In: Sasahara, AA.; Stein, M., editors. *Pulmonary embolic disease*. Grune & Stratton; New York, NY: 1965. p. 214–224.
18. Sautter RD, Fletcher FW, Emanuel DA, Lawton BR, Olsen TG. Complete resolution of massive pulmonary thromboembolism. *JAMA*. 1964; 189:948–949. [PubMed: 14174336]
19. Dalen JE, Banas JS Jr, Brooks HL, Evans GL, Paraskos JA, Dexter L. Resolution rate of acute pulmonary embolism in man. *N Engl J Med*. 1969; 280:1194–1199. [PubMed: 5767460]

20. McDonald IG, Hirsh J, Hale GS. Early rate of resolution of major pulmonary embolism: a study of angiographic and haemodynamic changes occurring in the first 24 to 48 hours. *Br Heart J.* 1971; 33:432–437. [PubMed: 5557454]
21. Hirsh J, McDonald IG, Hale GA, O'Sullivan EF, Jelinek VM. Comparison of the effects of streptokinase and heparin on the early rate of resolution of major pulmonary embolism. *Can Med Assoc J.* 1971; 104:488–491. [PubMed: 5549989]
22. Dalla-Volta S, Palla A, Santolicandro A, et al. PAIMS 2: alteplase combined with heparin versus heparin in the treatment of acute pulmonary embolism—Plasminogen Activator Italian Multicenter Study 2. *J Am Coll Cardiol.* 1992; 20:520–526. [PubMed: 1512328]
23. Fred HL, Axelrad MA, Lewis JM, Alexander JK. Rapid resolution of pulmonary thromboemboli in man: an angiographic study. *JAMA.* 1966; 196:1137–1139. [PubMed: 5952515]
24. Menéndez R, Nauffal D, Cremades MJ. Prognostic factors in restoration of pulmonary flow after submassive pulmonary embolism: a multiple regression analysis. *Eur Respir J.* 1998; 11:560–564. [PubMed: 9596102]
25. Begic A, Jogi J, Hadziredzepovic A, Kucukalic-Selimovic E, Begovic-Hadzimiratovic S, Bajc M. Tomographic ventilation/perfusion lung scintigraphy in the monitoring of the effect of treatment in pulmonary embolism: serial follow-up over a 6-month period. *Nucl Med Commun.* 2011; 32:508–514. [PubMed: 21403584]
26. Wartski M, Collignon MA. Incomplete recovery of lung perfusion after 3 months in patients with acute pulmonary embolism treated with antithrombotic agents: THESEE Study Group—Tinzaparin ou Heparin Standard: Evaluation dans l'Embolie Pulmonaire Study. *J Nucl Med.* 2000; 41:1043–1048. [PubMed: 10855632]
27. The urokinase pulmonary embolism trial: a national cooperative study. *Circulation.* 1973; 47(suppl 2):II1–II108. [No authors listed]. [PubMed: 4266893]
28. Miniati M, Monti S, Bottai M, et al. Survival and restoration of pulmonary perfusion in a long-term follow-up of patients after acute pulmonary embolism. *Medicine (Baltimore).* 2006; 85:253–262. [PubMed: 16974210]
29. Paraskos JA, Adelstein SJ, Smith RE, et al. Late prognosis of acute pulmonary embolism. *N Engl J Med.* 1973; 289:55–58. [PubMed: 4710405]
30. Donadini MP, Dentali F, Cosmi B, et al. Presence of residual thromboemboli at least six months after a first episode of symptomatic pulmonary embolism: do perfusion scintigraphy and angio-computed tomography agree? *Thromb Haemost.* 2009; 102:1287–1289. [PubMed: 19967166]

Fig. 1.

Changes in measures of clot burden over time.

A and **B**, Graphs show mean percentage change in total clot volume (**A**), calculated as $[(\text{baseline volume} - \text{follow-up volume}) / \text{baseline volume}] \times 100$, and semiquantitative Qanadli [5] and Mastora [6] scores (**B**).

Fig. 2.

47-year-old woman with acute pulmonary embolism and deep venous thrombosis.

A, Baseline pulmonary CT angiography (CTA) shows thrombus (*arrow*) within segmental branches of left lower lobes. Total clot volume is 712.64 mm³.

B, Follow-up pulmonary CTA obtained 73 days after baseline shows residual clot (*arrow*) in same location. Total clot volume is 320.25 mm³.

Fig. 3.

56-year-old woman with malignancy who presented with acute pulmonary embolism.

A, Baseline pulmonary CT angiography (CTA) shows clots in main and right pulmonary arteries and left pulmonary arteries (*arrows*). Total clot volume is 717.48 mm³.

B, Follow-up pulmonary CTA obtained 105 days after baseline pulmonary CTA shows complete resolution.

Fig. 4.

Changes in clot volume over time by location.

A and **B**, Graphs show changes in clot volumes in central (**A**) and peripheral (**B**) pulmonary arteries.

TABLE 1

Demographic Characteristics and Comorbid Conditions of 111 Patients

Variable	Value
Age (y), mean $\pm$ SD	51.6 $\pm$ 17.8
Male sex	55 (49.5)
Ischemic heart disease	17 (15.3)
Congestive heart failure	15 (13.5)
Cerebrovascular disease	8 (7.2)
Chronic lung disease	36 (32.4)
Chronic kidney disease	11 (9.9)
Cancer	24 (21.6)

Note—Except for age, data are no. (%) of patients.

TABLE 2

Clot Burden at Baseline and Follow-Up Pulmonary CT Angiography (CTA)

Variable	Baseline Pulmonary CTA	Follow-Up Pulmonary CTA
Blood clot volume (mm ³)	3403.3 ± 6505.6 (6.8–31,683.4)	531.6 ± 2383.5 (0–17,027.1)
No. of individual clots	2.2 ± 1.8 (1–11)	0.5 ± 1.2 (0–6)
Qanadli score	6 ± 6.3 (0–25)	1.4 ± 3.6 (0–20)
Mastora score	13.8 ± 18.9 (0–84)	1.7 ± 5.9 (0–43)

Note—Data are mean ± SD (range).

TABLE 3

Degree of Clot Resolution by Follow-Up Interval and Clot Location

Degree of Clot Resolution ^a	Follow-Up Interval, Clot Location ^b									
	1–14 Days			15–28 Days			29–90 Days			> 90 Days
	Total	Central	Peripheral	Total	Central	Peripheral	Total	Central	Peripheral	Total
Minimal	4	3	1	2	1	1	0	0	0	1
Significant	9	5	4	2	2	0	7	7	0	1
Complete	17	6	11	12	6	6	24	9	15	32
Total	30	14	16	16	9	7	31	16	15	34
										19
										15

Note—Data are no. of patients.

^aMinimal clot resolution is defined as 0–50% resolution, significant clot resolution is defined as 51–99% resolution, and complete clot resolution is defined as 100% resolution.

^bCentral refers to pulmonary artery trunk, main right and left pulmonary arteries, right and left interlobar pulmonary arteries, and lobar arteries. Peripheral refers to the segmental and subsegmental pulmonary arteries.