

**ROLE OF CT PULMONARY ANGIOGRAPHY IN DIAGNOSIS OF ACUTE  
PULMONARY THROMBOEMBOLISM****\*Dr. Tushar Kalekar**

Assistant Professor, Department of Radiology, BJMC, Pune, India.

**\*Corresponding Author: Dr. Tushar Kalekar**

Assistant Professor, Department of Radiology, BJMC, Pune, India.

Article Received on 21/11/2014

Article Revised on 11/12/2014

Article Accepted on 01/01/2015

**INTRODUCTION**

Since its introduction in 1992, computed tomography angiography (CTA) of the pulmonary arteries has become the main diagnostic test for the evaluation of pulmonary embolism (PE). With the advent of multi detector scanning, CTA has gained substantially in image acquisition speed and spatial resolution, which changed its diagnostic yield in many respects. Pulmonary embolism is the third most common acute cardiovascular disease after myocardial infarction and stroke and results in thousands of deaths each year because it often goes undetected.<sup>[1,2]</sup> Diagnostic tests for thromboembolic disease include (a) the D-dimer assay, which has a high sensitivity but poor specificity in this setting<sup>[3]</sup>, (b) ventilation-perfusion scintigraphy, which has a high sensitivity but very poor specificity<sup>[4]</sup> and (c) lower limb ultrasonography, which has a high specificity but low sensitivity.<sup>[5]</sup> Computed tomographic (CT) pulmonary angiography has been evaluated has demonstrated sensitivities of 53%–100% and specificities of 83%–100% in wide ranges.<sup>[6]</sup>

Pulmonary angiography, the diagnostic standard of reference for confirming or refuting a diagnosis of pulmonary embolism, remains underused.<sup>[7,8]</sup> For each lung the main, lobar, segmental and subsegmental arteries are examined for pulmonary embolism. Acute pulmonary embolism cause intraluminal filling defects that should have a sharp interface with the intravascular contrast material. The vessels are seen as either normal, containing acute pulmonary embolism, or indeterminate. The reason for indeterminacy is reported, along with the extent of normalcy. For example, vessels may appear normal to the level of the segmental arteries; however, the presence of pulmonary embolism in sub-segmental arteries may remain indeterminate depending on the quality of the study.

The diagnostic criteria for acute pulmonary embolism include the following: 1. Arterial occlusion with failure to enhance the entire lumen due to a large filling defect; the artery may be enlarged compared with adjacent patent vessels, 2. A partial filling defect surrounded by contrast material, producing the “polo mint” sign on images acquired perpendicular to the long axis of a vessel and the “railway track” sign on longitudinal images of the vessel, 3. A peripheral intraluminal filling defect that forms acute angles with the arterial wall.

Clinical comparison will be done with Wells criteria.<sup>[11]</sup> Criteria includes multiple parameters like deep vein thrombosis, tachycardia, haemoptysis, history of surgery and malignancy.

**AIMS AND OBJECTIVES**

- To diagnose pulmonary thromboembolism on CT pulmonary angiography.
- To compare the CT pulmonary angiography study results with
- Clinical status - Wells criteria.
- Biochemical marker – D-dimer status.

**MATERIALS AND METHODS**

After obtaining permission from hospital ethics committee, this prospective study was carried out at Sasson Hospital, Pune in the department of Radiodiagnosis from March 2012 to March 2014.

**4.1 Study population**

A total of 80 subjects were included irrespective of their age and sex with following inclusion and exclusion criteria.

**4.2 Inclusion criteria**

Patients referred for CT pulmonary angiography those who were clinically suspected to have acute pulmonary thromboembolism irrespective of biochemical (D-dimer) status.

**4.3 Exclusion criteria**

Patients with contrast allergy.  
Pregnancy

**4.4 Equipment used**

CT Machine: 64 slice siemens scanner.

#### 4.5 Method

Complete history was taken and thorough physical examination was done in all recruited patients. D-dimer values were noted. Other haematological tests were observed.

Images of pulmonary vasculature were obtained in axial planes by bolus tracking and maximum intensity projection. Reformatting in axial, coronal and sagittal planes was done using software provided.

#### 4.7 Technical parameters are as follows

Field of view - 300 mm, Length- 240 mm.  
Detector configuration (in mm) – 16 x 1.5 mm  
Pitch factor - 0.938

Gantry rotation time – 0.5 sec

Reconstructed slice thickness – 2.0 mm

Increment- 1.5 mm

Tube current (in eff. mAs) - 200, kV- 120

Contrast material: Volume – 2 ml/kg

- Concentration– 300 mg /ml

- Injection rate – 5 ml/s

#### OBSERVATIONS AND RESULTS

The present study was carried out at SGH Hospital, PUNE in the department of Radio diagnosis from May 2012 To May 2014. Total 80 patients of varied age groups with suspected acute pulmonary thrombo-embolism were scanned by multidetector spiral CT.

**Table 3: Age distribution.**

Age group	Number of patients	Percentage (%)
21 - 30	14	17.5
31 - 40	8	10.0
41 - 50	14	17.5
51 - 60	13	16.3
61 - 70	17	21.3
71 - 80	14	17.5
Total	80	100.0

From the table of age distribution it is seen that maximum numbers of patients were in age group of 61 to 70 years and followed by 71 to 80 years and 41 to 50

years. Males outnumbered females in most of the age group.

**Table 4: Gender distribution.**

Gender	Number of patients	Percentage (%)
Male	47	58.8
Female	33	41.3
Total	80	100.0

**Table 5: Pulmonary thrombo-embolism on CTPA.**

CT pulmonary angiography for pulmonary thromboembolism	Number of patients	Percentage (%)
Present	44	55.0
Absent	36	45.0
Total	80	100.0

On CTPA, 55% of patients were PE positive.

**Table 6: Lung vasculature involvement on CTPA**

Above table shows there is unilateral thromboembolism in 52.27% of patients and bilateral involvement in 47.73% of patients.

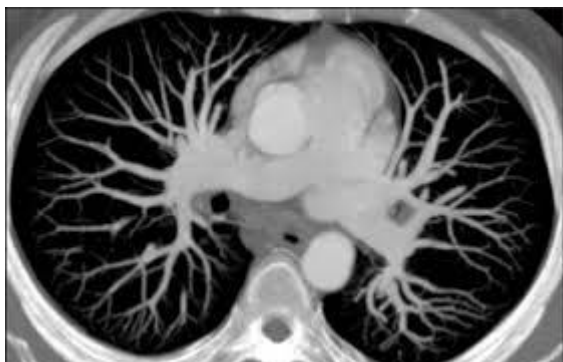
Most of the emboli were seen at segmental (53.75%) and lobar (30.0%) levels, followed by at subsegmental level (18.75%).

(Note: In some cases, there were multiple pulmonary artery segments involvement in single patient).

By using latex agglutination assay, 92.5% patients were positive for D-dimer and only 7.5% had showed negative results.

In present study pulmonary thrombo-embolism were seen in 44 (55%) patients. In 36 (45%) patients non-embolic pathologies were demonstrated including interstitial lung disease (n=7), pneumonia(n=6), congestive cardiac disease (n=4), COPD (n=4) and pleural effusion in 1 patient. Search of origin of emboli was made. Deep vein thrombosis was seen in 24 patients, 5 patients shown IVC thrombosis on colour doppler while 2 patients were of right ventricular thrombus on ECHO. In remaining 13 patients cause of pulmonary

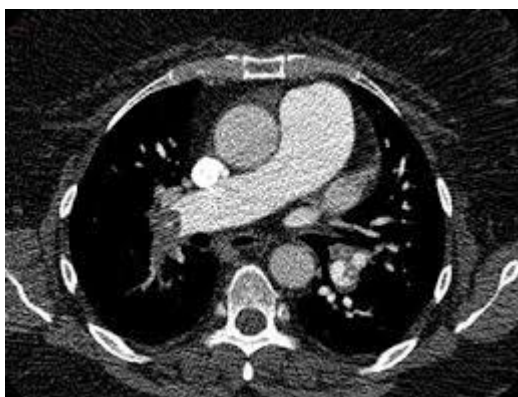
thrombo-embolism was inconclusive. After 1 month follow up, out of 44 patients diagnosed and treated for pulmonary thromboembolism, 5 patients died in 2 to 8 days period after diagnosis.



**FIG. 1** Normal appearance of pulmonary angiography.



**FIG.2** Filling defects in bilateral distal main pulmonary representing thrombus.



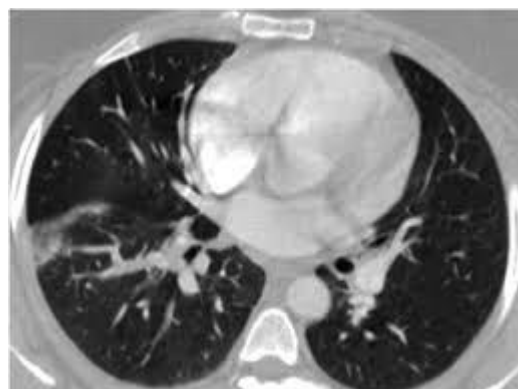
**Fig. 3** Filling defects in right distal main pulmonary representing thrombus.



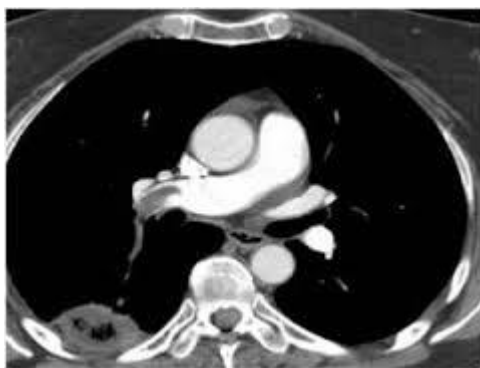
**Fig 4.** Sagittal image showing thrombus in left lower lobe segmental artery.



**Fig. 5** Coronal image showing thrombus in right distal main pulmonary artery and upper lobar artery.



**Fig 6.** Subpleural ground glass densities in right lower lobe superior segment due to pulmonary infarct.



**Fig 7 showing thrombus in right descending pulmonary trunk with subpleural cavitary infarct in lower lobe.**

### DISCUSSION

This study of “Role of CT pulmonary angiography in diagnosis of clinically suspected acute pulmonary thromboembolism its comparison with clinical (Wells criteria) and biochemical (D-dimer) status” was done on 80 cases between time period March 2012 to March 2014.

The present study included 80 patients ranging from 20 to 80 years of age. 58.8% of the patients were males in the present study whereas only 41.3% of the patients were females. Quinn D et al<sup>[12]</sup> performed on 103 patients ranging from 16 to 87 years of age. 37% of the patients were males in the present study where as 63% of the patients were females.

The present study included 80 patients. 44 (55%) patients had PE detected on CTPA. 21 (47.72%) had bilateral PE and 23 had unilateral involvement. Out of which 55 patients (17%) had PE detected on CT. 39 (71%) had bilateral PE and 16 (29%) had unilateral involvement.

#### 6.4 Distribution of thrombo-embolic materials

In present study, 44 out of 80 (55%) patients show pulmonary thromboembolism on CTPA. Out of 44 patients, single location thromboembolic material was found in 15 (34%) patients, double location in 14 (31.8%) patients and triple location in 12 (27.27%) patients while all segments were involved in 3 patients. Centrally (main and lobar branch) localized filling defects were visualized in 33(75%) patients. Segmental filling defects corresponding to thrombo-embolic material were demonstrated in 43 (97.72%) patients. In 15 (34.09%) patients, subsegmental filling defects were shown.

In present study of 80 patients by using latex agglutination assay and pulmonary arteriogram as a diagnostic criteria d-dimer show 97.73% of sensitivity, 13.89% of specificity and 83.33% of negative predictive value.

### CONCLUSIONS

- Multidetector-row spiral CT technology has overcome past limitations of CT and is a preferred modality for imaging patients with suspected acute PE.
- In our study, out of total sample of 80 patients, 44 (55%) patients were showed pulmonary thromboembolism on CTPA.
- Bilateral distribution of emboli was seen in 21(47.73%) patients.
- Thrombo-embolic material was observed in more than one region. Most of the emboli were seen at segmental (53.75%) and lobar (30.0%) levels, followed by at subsegmental level (18.75%). MDCT made the detection of subsegmental emboli which is difficult in single detector scanner.
- Using the Wells score, out of 44, 1 patient with low risk of PE went to be diagnosed with PE on CTPA. 24 patients with moderate risk and 19 patients with high risk on Wells score went to be diagnosed with PE on CTPA. There is significant association between CT pulmonary angiography diagnosis and Wells grade (P value <0.05).
- Out of 44 patients, 43 patients with positive d-dimer value went to diagnosed PE on CTPA. 1 patient with negative d-dimer value went to diagnosed PE on CTPA. There is no significant association between CT pulmonary angiography diagnosis and D-dimer status (P value >0.05). D-dimer had very low specificity also.
- In 36 of 80 patients (45%), non-embolic pathologies were demonstrated including evidence of interstitial lung disease (n = 7), pneumonia (n = 6), congestive cardiac failure (n = 4), COPD (n = 4) and malignancy (n = 3).

### REFERENCES

1. Anderson FA Jr, Wheeler B, Goldberg RJ. A population-based perspective of the hospital incidence and case fatality rates of deep vein thrombosis and pulmonary embolism: the Worcester DVT study. *Arch Intern Med*, 1991; 151: 933–938.
2. Giuntini C, Ricco GD, Marini C. Pulmonary embolism: epidemiology. *Chest*, 1995; 107(suppl): 3S–9S.
3. Brown MD, Rowe BH, Reeves MJ, Birmingham JM, Goldhaber SZ. The accuracy of the enzyme linked immunosorbent assay D-dimer test in the diagnosis of pulmonary embolism: a meta-analysis. *Ann Emerg Med*, 2002; 40: 133–134.
4. PIOPED investigators - Value of ventilation/perfusion scan in acute pulmonary embolism: results of the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED). *JAMA*, 1990; 263: 2753–2759.
5. Borris LC, Christiansen HM, Lassen MR, Olsen AD, Schutt P. Comparison of real-time B-mode ultrasonography and bilateral ascending phlebography for detection of postoperative deep vein thrombosis following elective hip surgery: the



- Venous Thrombosis Group. *Thromb Haemost*, 1989; 61: 363–365.
6. Rathbun SW, Raskob GE, Whitsett TL. Sensitivity and specificity of helical computed tomography in the diagnosis of pulmonary embolism: a systematic review. *Ann Intern Med*, 2000; 132: 227–232.
  7. Schluger N, Henschke C, King T. Diagnosis of pulmonary embolism at a large teaching hospital. *J Thorac Imaging*, 1994; 9: 180–184.
  8. Henschke CI, Mateescu I, Yankelevitz DF. Changing practice patterns in the workup of pulmonary embolism. *Chest*, 1995; 107: 940–945.
  9. Stein PD, Goldhaber SZ, Henry JW. Arterial blood gas analysis in the assessment of suspected acute pulmonary embolism. *Chest*. 1996; 109: 78–81.
  10. Stein PD, Goldhaber SZ, Henry JW. Alveolar-arterial oxygen gradient in the assessment of acute pulmonary embolism. *Chest*. 1995; 107: 139–143.
  11. Wells PS, Ginsberg JS, Anderson DR. Use of a clinical model for safe management of patients with suspected pulmonary embolism. *Ann Intern Med*, 1998; 129: 997–1005.
  12. Quinn DA, Fogel RB, Smith CD, Laposata M, Thompson BT, Johnson SM. D-dimers in the diagnosis of pulmonary embolism. *Am J Respir Crit Care Med*, 1999; 159: 1445–1449.
  13. Mettler FA, Guiberteau MJ. *Essentials of nuclear medicine imaging*. Elsevier 5<sup>th</sup> ed, 2006; p159–202.
  14. Hartmann IJC, Prokop M. Pulmonary embolism: is multislice CT the method of choice? *Eur J Nucl Med Mol Imaging*, 2005; 32: 103–107.
  15. Dalen JE, Alpert JS. Natural history of pulmonary embolism. *Prog Cardiovasc Dis.*, 1975; 17: 257–270.
  16. Pabinger I, Grafenhofer H. Thrombosis during pregnancy: risk factors, diagnosis and treatment. *Pathophysiol Haemost Thromb*, 2002; 32: 322–324.
  17. Ridge CA, McDermott S, Freyne BJ. Pulmonary Embolism in Pregnancy: Comparison of Pulmonary CT Angiography and Lung Scintigraphy. *Am J Roentgenol*, 2009; 193: 1223–1227.
  18. Silverstein MD, Heit JA, Mohr DN. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. *Arch Intern Med*, 1998; 158: 585–593.
  19. White RH. The epidemiology of venous thromboembolism. *Circulation*, 2003; 107: I-4–I-8.
  20. Moser K, LeMoine J. Is embolic risk conditioned by location of deep venous thrombosis? *Ann Intern Med*, 1981; 94: 439–444.
  21. Stern JB, Abehsera M, Grenet D, Friard S, Couderc LJ, Scherrer A, et al. Detection of pelvic vein thrombosis by magnetic resonance angiography in patients with acute pulmonary embolism and normal lower limb compression ultrasonography. *Chest*, 2002; 122: 115–121.
  22. Pengo V, Lensing AW, Prins MH. Thromboembolic Pulmonary Hypertension Study Group. Incidence of chronic pulmonary hypertension after pulmonary embolism. *N Engl J Med*, 2004; 350: 2257–2264.
  23. Bagot CN, Arya R. Virchow and his triad: a question of attribution. *Br. J. Haematol*. 2008; 143: 180–190.
  24. Lehmann R, Suess C, Leus M, Edelgard Lindhoff, et al. Incidence, clinical characteristics and long-term prognosis of travel-associated pulmonary embolism. *Eur Heart J*, 2009; 30: 233–241.
  25. Torbicki A, Perrier A, Konstantinides S. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). *Eur Heart J*, 2008; 29: 2276–2315.
  26. Eriksson L, Wollmer P, Olsson CG, U Albrechtsson, H Larusdottir; R Nilsson, et al. Diagnosis of pulmonary embolism based upon alveolar dead space analysis. *Chest*, 1989; 96: 357–362.
  27. Price DG. Pulmonary embolism: Prophylaxis diagnosis and treatment. *Anaesthesia*, 1976; 31: 925–932.
  28. Reid JH, Coche EE, Inoue T. International Atomic Energy Agency (IAEA) Consultants' Group. Is the lung scan alive and well? Facts and controversies in defining the role of lung scintigraphy for the diagnosis of pulmonary embolism in the era of MDCT. *Eur J Nucl Med Mol Imaging*, 2009; 36: 505–521.
  29. Miniati M, Bottai M, Monti S. Comparison of 3 clinical models for predicting the probability of pulmonary embolism. *Medicine*, 2005; 84: 107–114.
  30. Wells PS, Anderson DR, Rodger M, Stiell I, Dreyer JF, Barnes D, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. *Ann Intern Med.*, 2001 Jul 17; 135(2): 98-107.