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Role of Multi-detector Computed Tomography Pulmonary Angiography for patients with suspected Pulmonary Embolism

Nishita Pujary[1], Nitesh Karnire[2]

Abstract: ***Pulmonary embolisms has non specific clinical presentation, PE is often referred to as the "great masquerader" and remains a diagnostic challenge to both the clinicians and radiologists. In this study we researched how MDCT would help in early detection of pulmonary embolism in clinically suspected cases of pulmonary embolism. MDCTPA reveals significant additional diagnoses which ensure appropriate patient management without delay.***

1. Introduction

Pulmonary embolism is the partial or total occlusion of one or more central or peripheral pulmonary arteries by thrombi that originate typically in the large veins of the lower extremities or pelvis.

The first description of pulmonary embolism is attributed to Laennec in 1819. The first radiographic description of pulmonary embolism is that of Wharton and Pierson of a chest radiograph in 1922. Pulmonary angiography, which was introduced in the 1960s, had long been considered the gold standard for the diagnosis of pulmonary embolism .Pulmonary angiography is sometimes difficult to interpret, with disagreement more often about the absence of pulmonary embolism than about its presence . A pulmonary angiogram is an invasive procedure . At present with improved technology the complication rate has been low with a major non-fatal complication rate of 0.4% and a mortality rate of 0.1% . The role of pulmonary angiography in the diagnosis of pulmonary embolism nowadays is limited, because of the availability of alternative noninvasive strategies. As a consequence the practical experience with angiography in hospitals has declined. Nevertheless, in patients with inconclusive non-invasive test results pulmonary angiography remains an excellent diagnostic method.

In particular, MDCT equipment with 128 detector rows or more with the use of IV contrast can properly display pulmonary arteries down to the sub-segmental level, quickly providing images with voxel isotropy and maximizing the efficiency of the IV bolus of iodinated contrast medium. Despite the direct visualization of clot material, depiction of cardiac and pulmonary function in combination with the quantification of pulmonary obstruction helps to grade the severity of PE for further risk stratification.

2. Aims and Objectives

Broad Objective

To assess the clinical utility and the pattern of imaging findings on Multi-detector.

Computed Tomographic Pulmonary Angiography for patients with suspected pulmonar embolism.

Specific Objectives

1. Early diagnosis of Pulmonary emboli.
2. To determine the common clinical presentations of patients with suspected pulmonary embolism referred for MDCT-PA .
3. To determine the prevalence rate, age and gender distribution of pulmonary embolism on MDCT- PA
4. Correlation of the imaging findings with laboratory findings (D Dimer levels), lower limb Colour Doppler.
5. To determine other radiological findings on MDCT-PA in clinically suspected pulmonary embolism.
6. Post treatment follow up to see resolution of the disease.

Pathophysiology and Clinical Presentation

The effects of an embolus depend on the extent to which it obstructs the pulmonary circulation, the duration over which that obstruction accumulates, and the pre-existing state of the patient, which has been defined only imprecisely. Some mediators (for example, serotonin or thromboxane from activated platelets) can probably produce vasospasm in non-embolised segments of the lung. As a result, a degree of pulmonary hypertension may develop disproportionate to the amount of vasculature that is mechanically occluded. In general, a patient who has pre-existing cardiopulmonary disease or who is old, frail or debilitated will be more sensitive to the effects of pulmonary embolism than a patient who was well until the embolic event occurred. Most emboli are multiple. As both the extent and chronicity of obstruction vary so widely, pulmonary embolism can produce widely differing clinical pictures. Disregarding chronic thromboembolic pulmonary hypertension, it is convenient to classify pulmonary embolism into three main types.

Risk Factors for Pulmonary Embolism

Inherited Risk Factors

1) A number of hereditary abnormalities of the coagulation system are associated with an increased risk for venous thromboembolism. These genetic risk factors can be detected. approximately 50% of patients with a first episode of venous thromboembolism have 1. Antithrombin, protein C and protein S deficiencies. Patients with heterozygous deficiencies of one or more of these natural anticoagulants are at increased risk of developing thrombosis at a young age, which often occurs spontaneously. Moreover, thrombotic eventsin these patients have a tendency to recur.

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2) Prothrombin G20210A mutation. Patients with this mutation have an approximately 30% increase in plasma prothrombin levels, which therefore likely generates more thrombin. Carriers have a higher risk to develop venous thromboembolism,

Acquired Risk Factors
Most of the acquired risk factors are transient.

Increasing age and a history of previous venous thromboembolism are independent and persistent risk factors for life.

1. Estimated incidences of venous thromboembolism in the elderly are 10 times higher.
2. Malignancy (pancreatic, ovarian, gastrointestinal and pulmonary tumours) and major surgery (orthopedic and neurosurgery) (treatment within previous 6 months or palliative therapy)are probably the strongest risk factors for venous thromboembolism
3. Pregnancy and the post-partum period carry a 6 to 10-fold increased thromboembolic risk,
4. Oral contraceptives are known to increase the risk of venous thromboembolism and use of the second or third generation pills have odds ratios of 4 to 8 towards developing thrombosis as compared to non-users.
5. Venous stasis or injury. Immobilisation or other cause of venous stasis —for example, stroke, major trauma or surgery within 4 weeks.
6. Reduced cardiac output (congestive heart failure)
7. Obesity
8. Indwelling catheters and electrodes in great veins and right heart

Other signs include fever, tachycardia, decreased breath sounds, wheezing, pleural rub, rales, jugular venous distention and accentuated pulmonic component of second heart sound .

Clinical forms of Pulmonary Embolism

Acute minor pulmonary embolism
If an embolus obstructs less than 50% of the pulmonary circulation, it often produces no symptoms. For example, about 40% of patients with DVT who have no symptoms of pulmonary embolism have evidence of the condition on lung scans. If symptoms do develop the most common is dyspnea, possibly upon minor exertion. Sometimes, the first abnormality the patient notes results from pulmonary infarction, which occurs in obstruction of medium sized pulmonary artery branches. Sharp pleuritic pain develops, and there may be associated haemoptysis. Pulmonary infarction occurs in only about 10% of patients without pre-existing cardiopulmonary disease. If, however, there is already compromise of the oxygenation of the embolised area—either because the airways are abnormal or pulmonary venous outflow is impaired as a result of pre-existing left heart disease—then the incidence of infarction rises to 30%.

If there are any physical signs, they are those of pulmonary infarction. The patient is distressed with rapid and shallow breathing because of the pleuritic pain, but is not cyanosed because the disturbance of gas transfer is only slight. Signs of pulmonary infarction may be found in the lungs: a mixture of consolidation and effusion, possibly with a pleural rub. Fever is common and sometimes differentiation from infective

pleurisy is difficult. The fever and pain often produce a sinus tachycardia. Pulmonary artery mean pressure rarely exceeds 25mm Hg. As minor pulmonary embolism does not compromise the right ventricle, cardiac output is well maintained, hypotension does not occur, and the venous pressure and heart sounds are normal.

Acute Massive Pulmonary Embolism

When more than 50% of the pulmonary circulation is suddenly obstructed, the pathophysiology and clinical signs become dominated by the severe derangement of cardiac and pulmonary function. Obstruction of the pulmonary artery and mediator induced vasoconstriction cause a substantial increase in right ventricular afterload and, if the cardiac output is to be maintained, consequent elevation of pulmonary artery systolic pressure and an increase in right ventricular work. If this work cannot be sustained, acute right heart failure occurs. The thin walled right ventricle is designed to work against the normally low pulmonary vascular resistance; it performs poorly against a sudden obstruction. As a result, it dilates, and a moderate rise in the right ventricular and pulmonary artery systolic pressure occurs which rarely exceeds 55mm Hg because the ventricle, lacking time to develop compensatory hypertrophy, is unable to generate a higher pressure. The right ventricular end diastolic pressure and right atrial pressure rise to about 15–20mm Hg as the ventricle fails. Right ventricular dilatation leads to tricuspid regurgitation and may compromise the filling of the left ventricle. Cardiac output falls and the patient becomes hypotensive. This may occur so rapidly that syncope is either the presenting feature or easily induced by a relatively minor cardiovascular stress. If the degree of obstruction is sufficient, death occurs almost immediately. The fall in aortic pressure and the rise in right ventricular pressure may cause ischemia of the right ventricle through a critical reduction of right coronary perfusion. Electromechanical dissociation is the most frequent cause of final cardiac arrest.

Arterial hypoxaemia correlates roughly with the extent of embolism if there is no prior cardiopulmonary disease. Massive pulmonary embolism without hypoxaemia is so rare that if the arterial oxygen tension (PaO 2) is normal an alternative diagnosis should be considered. Hypoxaemia decreases tissue oxygen

Subacute Massive Pulmonary Embolism

This is caused by multiple small or moderately sized emboli that accumulate over several weeks. Because the obstruction occurs slowly, there is time for the right ventricle to adapt and for some hypertrophy to develop; consequently, the right ventricular systolic pressure is higher than in acute pulmonary embolism. The rises in the right ventricular end diastolic and right atrial pressures are of a lesser extent than in acute massive pulmonaryembolism since there is time for adaptation to occur and the degree of right ventricular failure is less for a given degree of pulmonary artery obstruction.

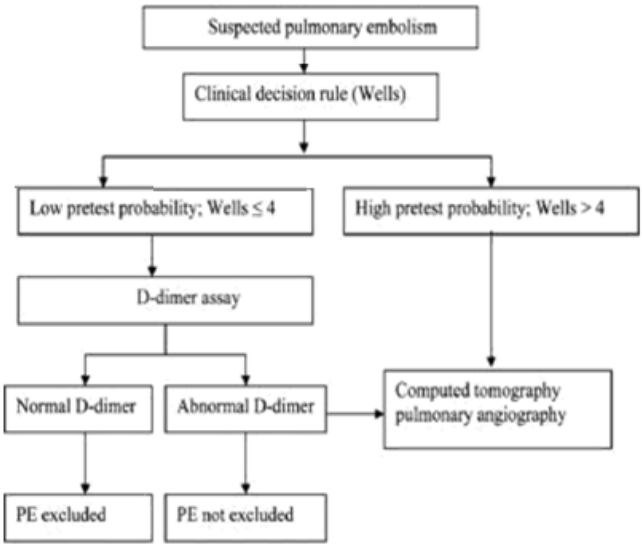
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The main symptoms are increasing dyspnoea and falling exercise tolerance.

There is often an associated dry cough. The breathlessness is usually out of proportion to all other findings, and there may be central cyanosis. The blood pressure and pulse rate are usually normal because the cardiac output is well maintained. Commonly, the venous pressure is raised and a third heart sound is audible at the lower sternum which may be accentuated by inspiration. The pulmonary component of the second heart sound is sometimes loud. There may also be intermittent symptoms and signs of pulmonary infarction that occurred during the build up of the obstruction. In advanced cases, cardiac output falls and frank right heart failure develops. A further pulmonary embolus may change the picture to that resembling acute massive pulmonary embolism.

Prognosis

90% of patients with acute pulmonary embolism will reach hospital to allow a diagnosis to be made, and a classification of massive, sub-massive and non-massive pulmonary embolism has been proposed . Massive pulmonary embolism, consisting of patients with hemodynamic instability, is relatively rare, occuring in less than 5% of patients. These patients require aggressive therapy to prevent death. Fibrinolytic therapy is the first line of treatment for this critically ill patient population. In the group of hemodynamic stable patients with pulmonary embolism, standard treatment consists of (low molecular weight) heparin followed by a course of vitamin K antagonists.



3. Materials And Methods

Design and Methodology

Study Area

The study was conducted at the Radiology department in D.Y. Patil Hospital situated in Nerul, Navi Mumbai equipped with a 128 slice MDCT scanner (GE Optima CT 660)

Study Population

This included 73 consecutive patients referred by clinicians with a clinical suspicion of PE who presented to the Radiology departments for MDCTPA and had no history of contrast allergy.

Study design

It was a prospective cross-sectional study that was conducted from October 2012 to September 2014. Upon obtaining an informed consent the patients' clinical summary was obtained from the request form and filled into the data collection form. Further scrutiny of the patients' files was done manually for relevant information. The MDCT-PA findings were then reviewed on a workstation.

Materials

The study was conducted at the Radiology department in D.Y. Patil Hospital situated in Nerul, Navi Mumbai equipped with a 128 slice MDCT scanner (GE Optima CT 660).

Methodology

The patient was recruited into the study upon informed consent. Diagnosis of PE was based on the direct visualization of intra luminal clots either as complete filling defects, mural defects or partial filling defects. The right ventricular enlargement was looked for which is a marker of pulmonary hypertension attributable to PE. Other investigations like D dimer levels and USG for deep venous thrombosis was evaluated and compared.

Inclusion criteria

1. This included 73consecutive patients with clinical suspicion of pulmonary embolism referred by clinicians
2. No known history of allergy to Iodinated contrast media.
3. Cases of all age groups.

Exclusion criteria

1. Non-consenting patients.
2. Patients with drug allergy
3. Patients with high serum creatinine levels (renal failure)
4. Pregnant patients.
5. Known case of chronic pulmonary embolus.

Diagnostic Criteria of Acute PE:

1. Complete arterial occlusion with failure to opacify vessel lumen (vessel cut-off sign).
2. Central filling defect surrounded by contrast.
3. Peripheral intraluminal filling defect that makes an acute angle with the arterial wall.

Diagnostic criteria of Chronic PE:

1. Complete occlusion of vessel that is smaller than others of same order of branching
2. Peripheral filling defect that makes obtuse angles with the vessel wall.

4. Results

During the 2 year study period, a total of 73 consecutive patients with a clinical suspicion of PE and referred for MDCTPA were identified and recruited into the study after having met the inclusion criteria. A review of these cases is done and the results, presented in form of tables and graphs to fulfill the objectives of the study.

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Table 6: Prevalence of Pulmonary Embolism (N=73)

Pulmonary Embolism	*Frequency*	*Percentage*
Present	43	58.9
Absent	30	41.1
Total	73	100.0

Graph 1: Prevalence of Pulmonary Embolism (N=73)

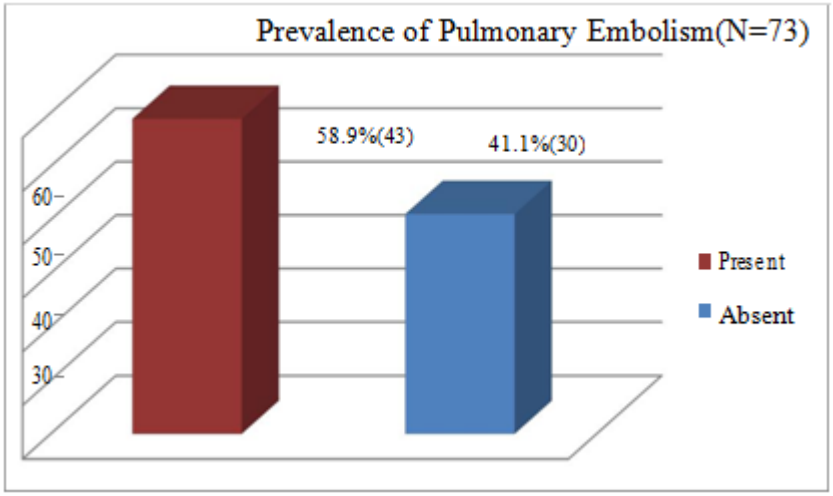


Table 6 and graph 1 shows the prevalence of pulmonary embolism in the 73 patients ,who presented with symptoms suspected of pulmonary embolism, and underwent MDCT pulmonary angiography .Out of 73 patients , 43(58.9%) patients had evidence of pulmonary embolism.

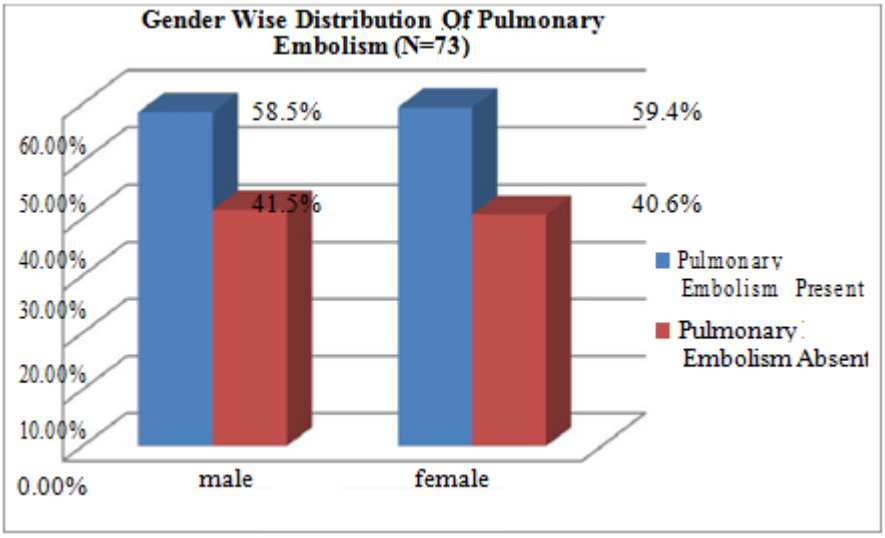
Table 7: Gender Distribution of Patients with Suspected Pulmonary Embolism (N=73)

Gender	Pulmonary Embolism		
	Present	Absent	Total
Male	24	17	41
	58.50%	41.50%	100.00%
Female	19	13	32
	59.40%	40.60%	100.00%
Total	43	30	73
	58.90%	41.10%	100.00%

Table 7 shows gender wise distribution of pulmonary embolism.

In a total of 41 males who presented with clinical suspicion on pulmonary embolism 24 were positive(58.5%)
In a total of 32 females who presented with clinical suspicion on pulmonary embolism 19 were positive59.4%)
P value= 0.942 sows no significance of gender criteria for pulmonary embolism.

Graph 2: Genderwise Distribution Of Pulmonary Embolism(N=73)



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Graph 3: Genderwise Distribution of Pulmonary EMBOLISM(N=73)

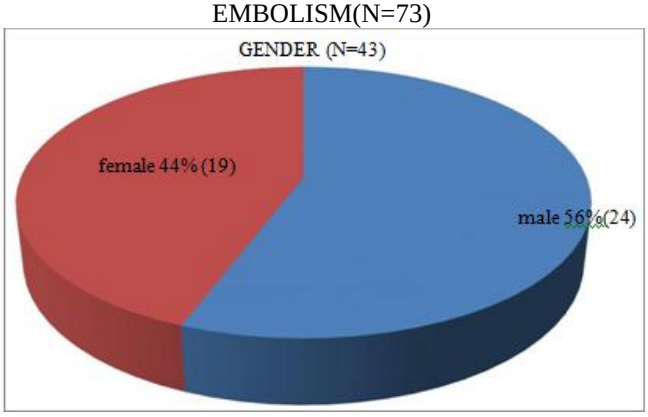


Table 7, Graph 2 and 3 presents the gender of patients recruited into the study. Total male patients in the study were 41 and female patients were 32. More male patients had clinically suspected PE as compared to the females. Males represented 41(58.9%) of all participants while the females were 32 (41.1%) The male to female ratio of patients with clinically suspected PE was 1.28.

In pulmonary embolism positive cases 24 (56%) were males and 19 (44%) were females. P value of 0.942 sows no significance of gender criteria for pulmonary embolism.

Table 8: Age Wise Distribution Of Pulmonary Embolism(N=73)

Age Groups in Years	Pulmonary Embolism		Total
	Present	Absent	
<30	3	8	11
	27.30%	72.70%	100.00%
31-40	16	5	21
	76.20%	23.80%	100.00%
41-50	9	10	19
	47.40%	52.60%	100.00%
51-60	4	4	8
	50.00%	50.00%	100.00%
>61	11	3	14
	78.60%	21.40%	100.00%
Total	43	30	73
	58.90%	41.10%	100.00%

(P value = 0.03. Statistically Significant)

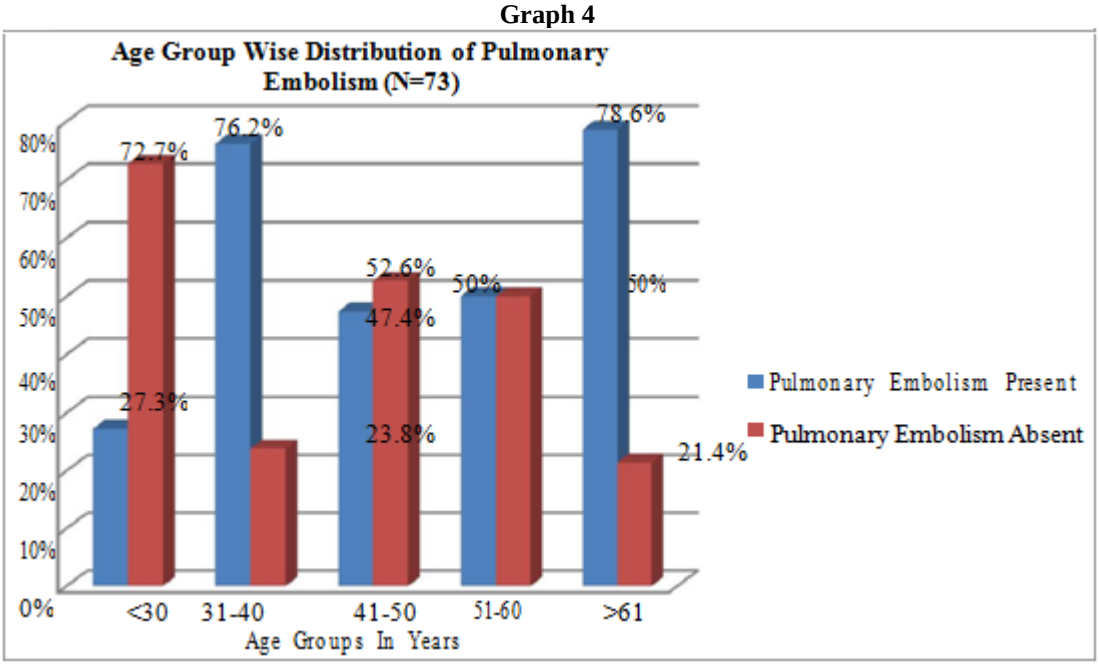
Below 30 years of age, 3 patients (27.3%) who presented with clinical suspicion of pulmonary embolism, were positive and 8 (72.7%) were negative. In 31-40 years range 16 (76.2%) patients who presented with clinical suspicion of pulmonary embolism were positive and 5(23.8%) were negative.

In 41-50 years range, 9 (47.4%) patients who presented with clinical suspicion of pulmonary embolism were positive and 10 (52.6%) were negative.

In 51-60 years range 4 (50%) patients who presented with clinical suspicion of pulmonary embolism were positive and 4(50%) were negative.

Above 61 years 11 (78.6%) patients were positive and 3(21.4%) patients were negative

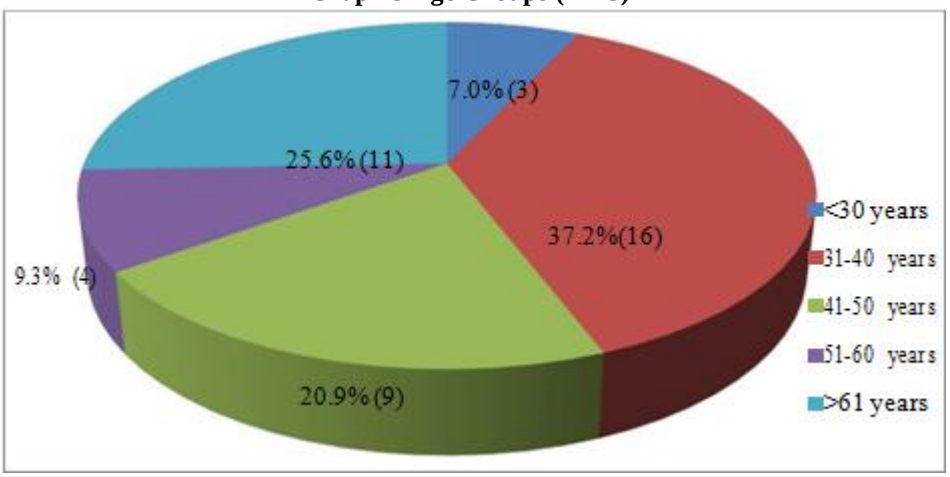
Graph 4



The above graph shows the age group wise distribution of pulmonary embolism

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Graph 5 Age Groups (n=43)



Graph 4 and 5 show the age wise distribution of pulmonary in this study.

Majority of the patients were in the 31-40 years age range and lowest were in below 30 range. The mean age was of the study participants was 45.4 years and the median age was 44.5 years (interquartile range (36-70). The youngest patient was aged 20 years and the oldest was 80 years old.

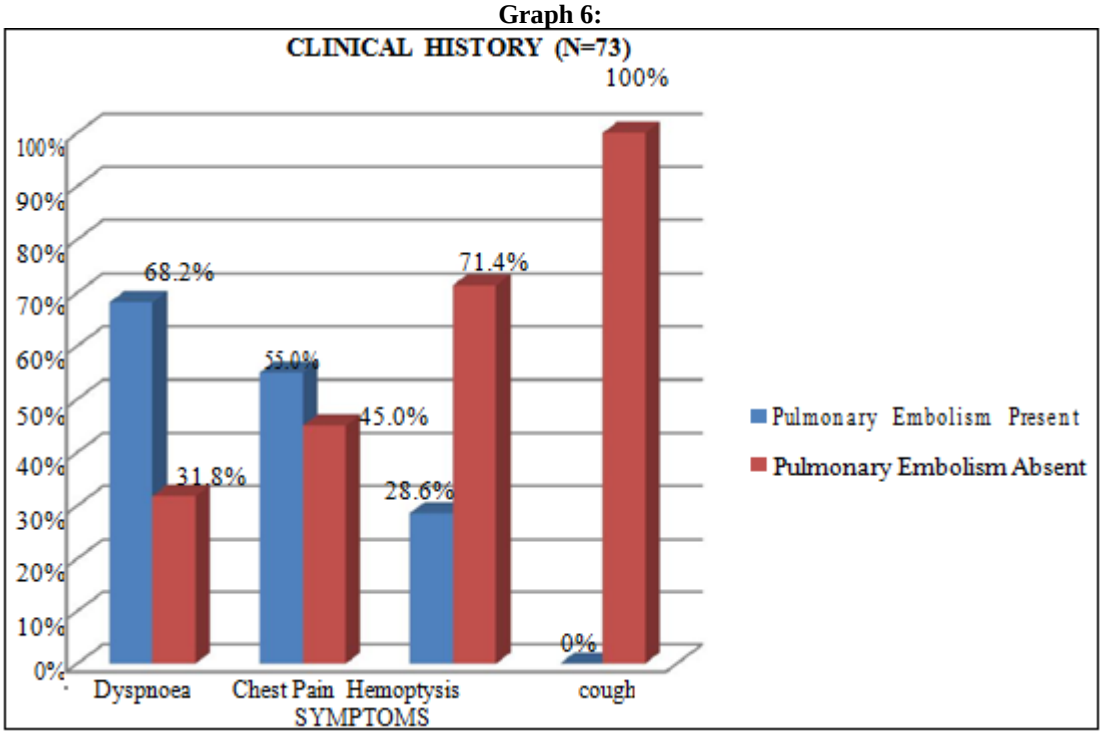
Table 9: Clinical History

Symptoms	Pulmonary Embolism		Total
	Present	Absent	
Dyspnoea	30	14	44
	68.20%	31.80%	100.00%
Chest Pain	11	9	20
	55.00%	45.00%	100.00%
Hemoptysis	2	5	7
	28.60%	71.40%	100.00%
cough	0	2	2
	0.00%	100.00%	100.00%
Total	43	30	73
	58.90%	41.10%	100.00%

The most common symptoms evoking a suspicion of pulmonary embolism is dyspnea, followed by chest pain, hemoptysis and cough.

The proportion of patients presenting with difficulty in breathing was significantly higher than that presenting with chest pain, cough or hemoptysis. However there was no significant association between the clinical history and the status of the CTPA (p-value 0.065). In a total of 73 patients,44 patients presented with dyspnea,20 with chest pain ,7 with hemoptysis and 2 with cough.

Graph 6:

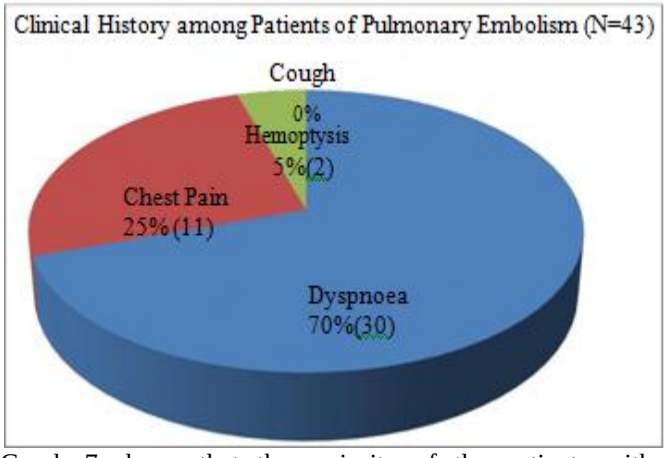


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Table 9,Graph 6 and Graph 7 show the distribution of the symptoms in patients with clinical suspicion of pulmonary embolism.

Out of 44 patients presenting with dyspnea 30(68.2%) patients had pulmonary embolism and 14 (31.8 %)patients did not have pulmonary embolism. Out of 20 patients presenting with chest pain 11(55%) patients had pulmonary embolism and 9 (45 %)patients did not have pulmonary embolism. Out of 7 patients presenting with hemoptysis2 (28.6%) patients had pulmonary embolism and 5(71.4 %)patients did not have pulmonary embolism. Out of 2 patients presenting with cough, both did not have pulmonary embolism.

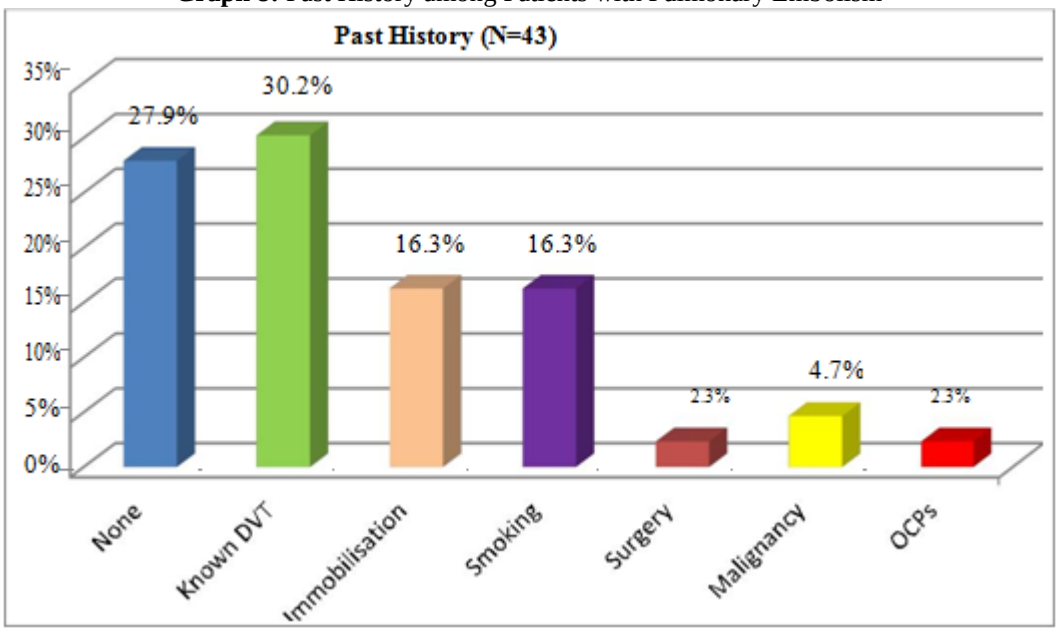
Graph 7



Graph 7 shows that the majority of the patients with pulmonary embolism presented with dyspnea (70%), followed by chest pain (25%),hemoptysis 5%.

No patient with pulmonary embolism had cough as a primary symptom.

Graph 8: Past History among Patients with Pulmonary Embolism

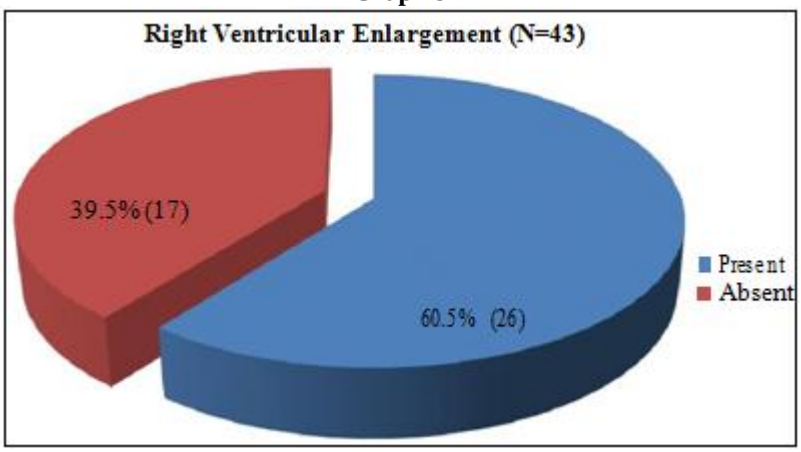


Graph 8 shows the significant past history in patients with pulmonary embolism.

Majority of the patients with pulmonary embolism had a history of DVT 30.2%. 16.3% had history of immobilization .16.3% patients were known smokers . 4.7 % patients had history of malignancy .2.3 % patients had history of surgery. 2.3 % patients had a history of intake of OCPs. 27.9 % of the patients had no significant past history.

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Graph 9



Graph 9 shows right ventricular enlargement in patients with pulmonary embolism.

Out of 43 pulmonary embolism patients 26(60.5%) patients had right ventricular enlargement with impending pulmonary hypertension. 17 (39.5%) patients did not have right ventricular enlargement. This parameter is important as it is an indicator of right ventricular strain due to pulmonary hypertension attributable to PE.

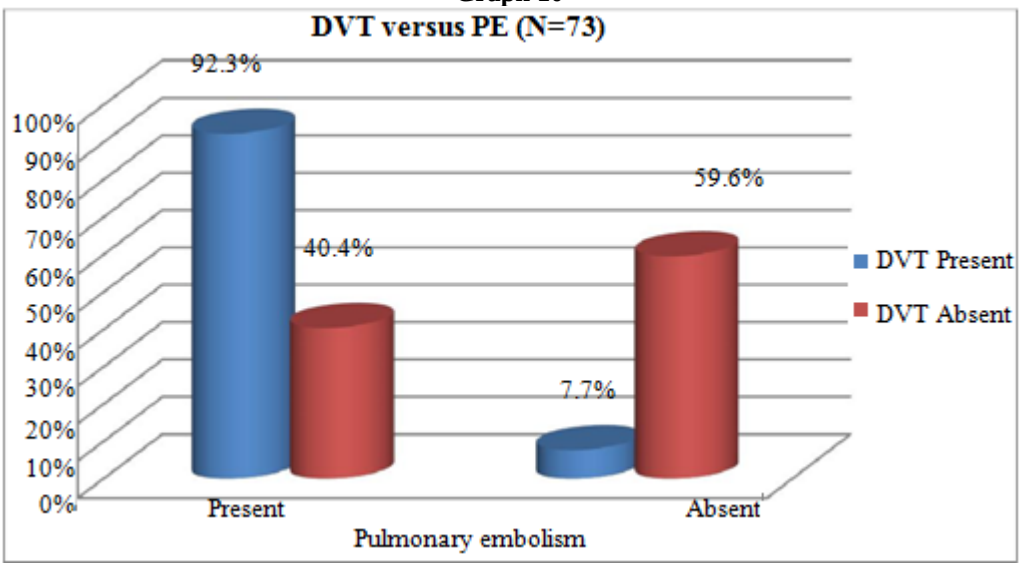
Table 10

DVT	Pulmonary Embolism		Total
	Present	Absent	
Present	24	2	26
	92.30%	7.70%	100.00%
Absent	19	28	47
	40.40%	59.60%	100.00%
Total	43	30	73
	58.90%	41.10%	100.00%

(P value= <0.001 statistically significant) Table 10 shows deep venous thrombosis in patients with pulmonary embolism.

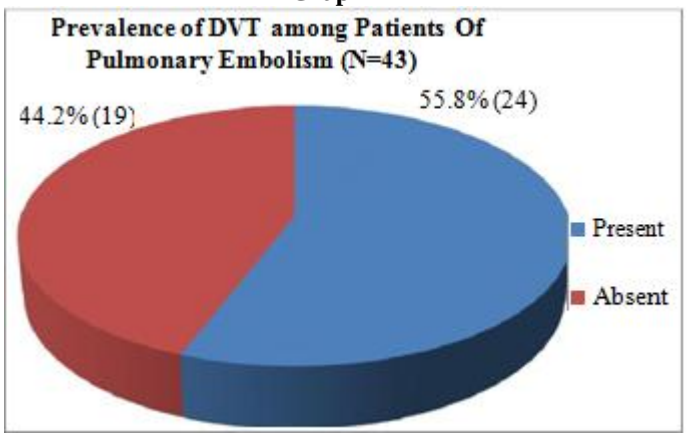
Out of 43 patients with pulmonary embolism 24 (92.3%)patients had DVT on USG and 19 (40.4%) did not have Deep venous thrombosis on USG .Out of 30 patients not having pulmonary embolism 2(7.7%) patients had deep venous thrombosis and 28 patients (59.6%) did not have deep venous thrombus. Majority of the patients diagnosed with pulmonary embolism had deep venous thrombosis on USG.

Graph 10



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Graph 11



Graph 10 and 11 show the prevalence of deep venous thrombosis among patients diagnosed with pulmonary embolism on MDCT PA 24 (55.8%)of the patients diagnosed with pulmonary embolism on MDCT PA had deep venous thrombosis on USG and 19(44.2%) patients did not have deep venous thrombosis.

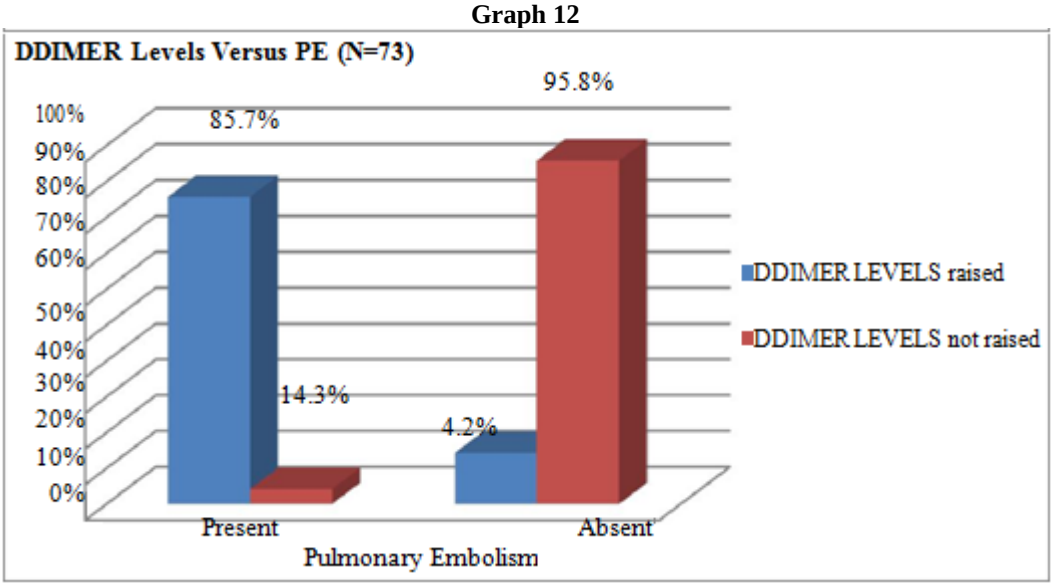
Table 11: DDIMER Levels and Pulmonary Embolism

DDIMER LEVELS	Pulmonary Embolism		Total
	Present	Absent	
raised	42	7	49
	85.70%	14.30%	100.00%
Not Raised	1	23	24
	4.20%	95.80%	100.00%
Total	43	30	73
	58.90%	41.10%	100.00%

Table 11 shows the D dimer levels in all patients recruited in the study. Out of 73 patients 49 patients had raised D dimer levels and 24 had normal D dimer levels. 42 patients with raised d dimer levels (85.7%) had pulmonary embolism and

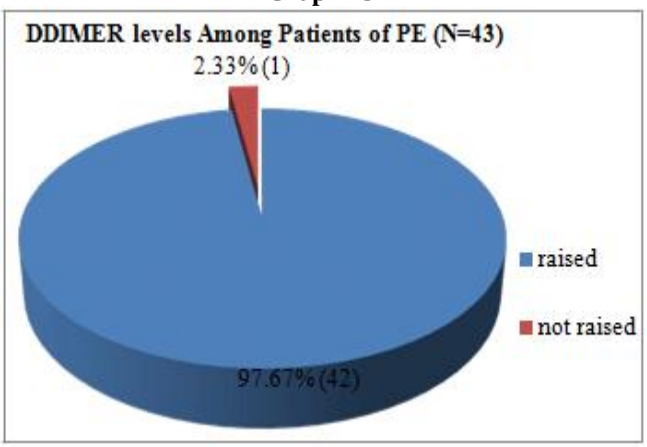
7(14.3%) patients did not have pulmonary embolism. The commonest cause of raised D dimer in these 7 patients was DVT. A significant P value<0.001 shows that d dimer is raised in almost all cases of pulmonary embolism.

Graph 12



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Graph 13



Graph 12 and 13 show the D dimer levels and its significance in patients with pulmonary embolism.

Table 12: Anatomical Distribution of the Location of Pulmonary Embolism on MDCT-PA (n=43)

Location of Pulmonary	Frequency	Percentage
EMBOLISM		
Right lung	32	24.43%
Left lung	24	18.32%
Main Pulmonary Artery	1	0.76%
Right main pulmonary artery	20	15.27%
Left main pulmonary artery	13	9.92%
Lobar	14	10.69%
Segmental	20	15.27%
Sub Segmental	7	5.34%
Total	131	100%

Graph 14: Anatomical Distribution of the Location of Pulmonary Embolism on MDCT-PA (n=30)

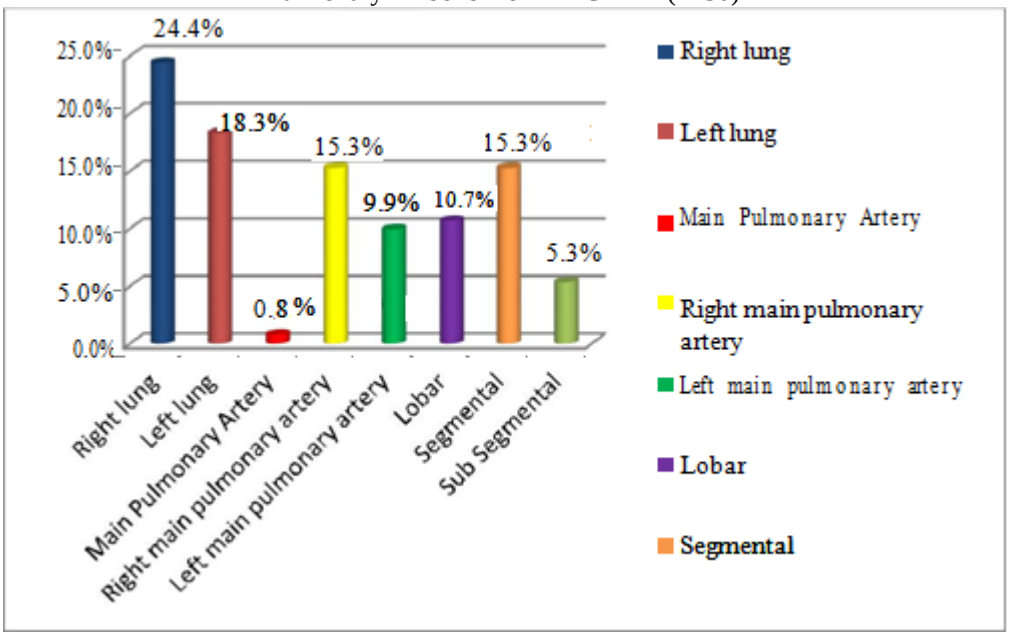


Table 13

Subgroup	Frequency	Percentage
MPA	1	1.33%
Right main pulmonary artery	20	26.67%
Right- lobar arteries	9	12%
Right- segmental arteries	10	13.33%
Right sub- segmental arteries	5	6.67%
Left main pulmonary artery	13	17.33%
Left -Lobar arteries	5	6.67%
Left- segmental arteries	10	13.33%
Left -sub segmental arteries	2	2.67%
Total	75	100%

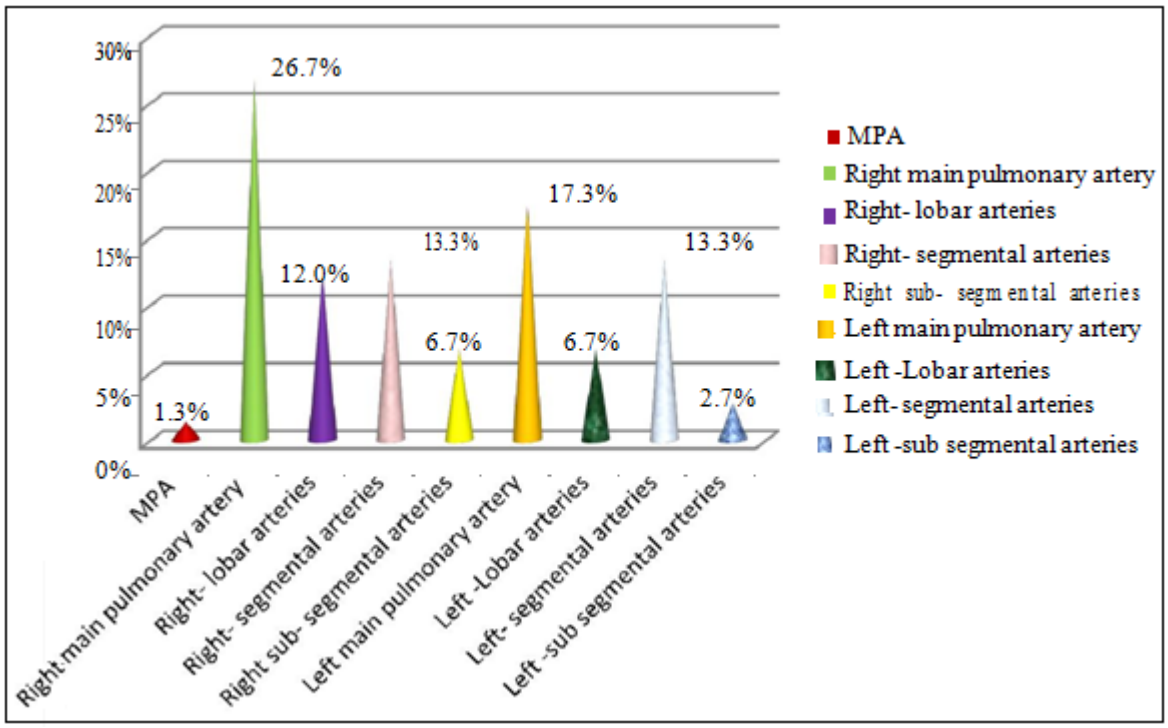
Table 12,13 and Graph 9 show the distribution of the pulmonary embolus located in the pulmonary artery and its branches (lobar, segmental and sub segmental) in the patients with pulmonary embolism seen in MDCT PA.

Majority of the patients had embolus in the right main pulmonary artery(26.6%). 32(24.4%) patients had embolus in the right side and 24 (18.3%)patients had on the left side. 20 (26.6%) patients had embolus in the right main pulmonary artery and 13 (17.33%)patients had in the left main pulmonary artery. 9 (12%)patients had embolus in the right lobar artery and 5(6.67%) patients in the left lobar artery. 10(13.3%) patients had embolus in the right segmental artery and 10(13.3%) patients had embolus in the left segmental artery. 5(6.67%)patients had embolus in the right sub segmental artery and 2(2.67%) patients had embolus in the left sub segmental artery.

Graph 15: Anatomical Distribution of the Location of Pulmonary Embolism on MDCT-PA (n=43)

Subgroup

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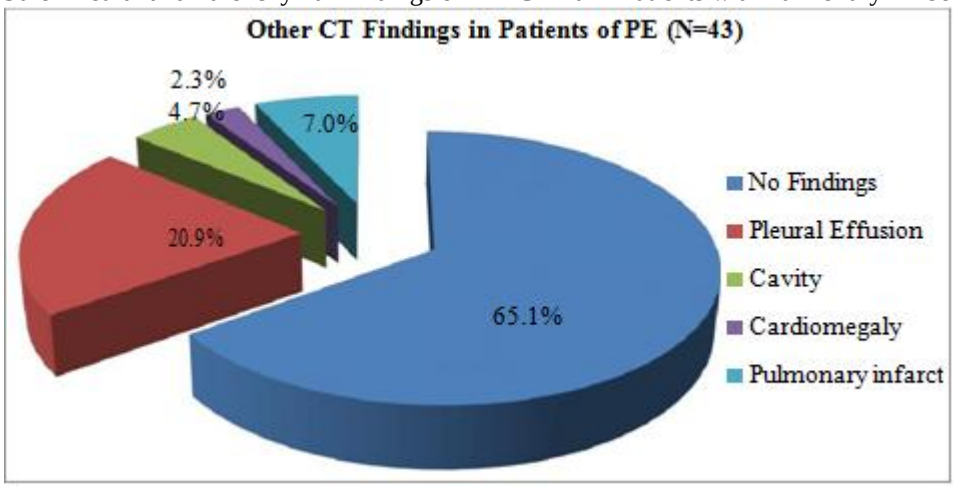


Graph 15 shows the anatomical distribution of the location of pulmonary embolism on MDCT PA

Table 14: Other Pleural and Parenchymal Findings on MDCT-
PA (n=73)

Other CT Findings	Frequency	Percent
No Findings	28	65.1
Pleural Effusion	9	20.9
Cavity	2	4.7
Cardiomegaly	1	2.3
Pulmonary infarct	3	7
Total	43	100

Graph 16: Other Pleural and Parenchymal Findings on MDCT-Pa in Patients with Pulmonary Embolism(N=43)



As shown in table 8 and graph 16, pleural and parenchymal abnormalities were commonly reported in 40 patients out of 73.

These findings were reported in 15 patients with PE and 25 patients without PE.

Pleural effusion was the commonest finding comprising 9 patients (23.3%) with PE and 10patients without PE.

This was followed by pulmonary infarct comprising 3 patients (7%) with PE. Among the above findings only the presence of infarct (wedge shape opacity) was significantly associated with PE (p value 0.020). 2 (4.7%) patients with pulmonary embolism had cavity seen in MDCT PA. 1(2.3%) patient with pulmonary embolism had cardiomegaly.

28 (65.1%) patients with pulmonary embolism did not have any additional CT findings on MDCT PA. The sensitivity of MDCT PA for detection of pulmonary embolism is 97.6%

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and specificity is 93.8%. The sensitivity of D Dimer for detection of pulmonary embolism is 100% and specificity is 75%

5. Discussion

The aim of this study is to assess the clinical utility and the pattern of imaging findings on Multi-detector Computed Tomographic Pulmonary Angiography for patients with suspected pulmonary embolism and its utility for early detection of acute pulmonary embolism.

Over the past decade, Multidetector-row computed tomography pulmonary angiography (CTPA) has become the primary tool for the diagnosis of Pulmonary embolism as it is non-invasive, takes minutes to perform, and has very high sensitivity and specificity.

6. Prevalence

In this study, out of 73 patients who presented with symptoms suspected of pulmonary embolism, and underwent MDCT pulmonary angiography, 43(58.9%) patients had evidence of pulmonary embolism.

In a study done by K Hogg et al [26] on diagnosis of pulmonary embolism with CT pulmonary angiography in which 13 were diagnostic and 11 follow up studies .The prevalence of pulmonary embolism was 19–79%

Van Belle A et al [27] did a prospective cohort study to assess the clinical effectiveness of a simplified algorithm using a dichotomized clinical decision rule, D-dimer testing, and computed tomography (CT) in patients with suspected pulmonary embolism. The prevalence of pulmonary embolism was (20.4%) in 674 patients.

In a study done by. MosI Cet al [47] to determine the safety of a simple diagnostic strategy using the Wells clinical decision rule (CDR), quantitative D-dimer testing and computed tomography pulmonary angiography (CTPA) overall prevalence of PE was 33%

Gender: In our study more male patients had clinically suspected PE as compared to the females, males represented 41(58.9%) of all participants (73)while the females were 32 (41.1%) The male to female ratio of patients with clinically suspected PE was 1.28. 24 (56%) males and 19 (44%) females were diagnosed cases of pulmonary embolism on MDCT PA .P value of 0.942 showed no significance of gender criteria for pulmonary embolism.

Tambe J et al [42]did a study on 37 patients (32.4%) who had CT angiograms that were positive for PE, of which majority of the patients were females(7 were males and rest females).

In a study done by Stein PD [38]et all the rate of diagnosis of PE, not adjusted for age, was higher in women(60 PE/100,000 women) than in men (42 PE/100,000 men)

However there was no significance of gender criteria for pulmonary embolism.

Age: In my study, majority of the patients were in the 31-40 years age range and lowest were in below 30 range.

The mean age of the study participants was 45.4years and the median age was 44.5 years. The youngest patient was aged 20 years and the oldest was 80 years old. Winer-Muram et al [19] did a study to determine diagnostic accuracy of four-channel multi-detector row computed tomography (CT) in emergency room and inpatient populations suspected of having acute pulmonary embolism (PE) in 93 patients with median age, 56 years. Sensitivity, specificity, and accuracy of CT were 100%, 89%, and 91%, respectively

Tambe J et al [38] did a study on Acute pulmonary embolism in the era of multi-detector CT in a total of 37 patients with pulmonary embolism, the mean age of these patients was 47.6±10.5 years (age range from 33 to 65 years).

In a study done by Stein PD et al [38]The incidence of PE and DVT increases exponentially with age. There is no cut-off age at which there is no risk of venous thromboembolism (VTE). Even children may suffer a PE or DVT. Age, therefore, does not exclude the diagnosis, but it is uncommon in infants and children.

Clinical History: The most common symptoms evoking a suspicion of pulmonary embolism is dyspnea, followed by chest pain, hemoptysis and cough.

In our study, the proportion of patients presenting with difficulty in breathing was significantly higher than that presenting with chest pain, cough or hemoptysis However there was no significant association between the clinical history and the status of the CTPA (p-value 0.065). In a total of 73 patients, 44 patients presented with yspnea, 20 with chest pain ,7 with hemoptysis and 2 with cough. In this study majority of the patients with pulmonary embolism presented with dyspnea (70%), followed by chest pain (25%), hemoptysis 5
Guo Z et al[48] did a study to explore the incidence and clinical features of PE with normal DD concentrations. The frequency of normal DD concentrations in patients with PE was 4%.). Fever, tachycardia, and tachypnea occurred less frequently in the group ($P < 0.05$) and time between onset of symptoms and DD testing was longer ($P = 0.001$). The diagnosis of PE was delayed in 22 of the 29 cases. Nineteen and seven cases with normal DD concentrations were classified according to pretest scores as intermediate and low risk, respectively.

7. Significant Past History/Risk Factors among Patients with

Pulmonary Embolism

In this study majority of the patients with pulmonary embolism had a history of DVT 30.2%. 16.3% had history of immobilization .16.3% patients were known smokers . 4.7 % patients had history of malignancy .2.3 % patients had history of surgery. 2.3 % patients had a history of intake of OCPs. 27.9 % of the patients had no significant past history.

In a study done by Lee EYet al[39] to determine the risk

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factors for pulmonary embolism (PE) among older children and young adults who underwent pulmonary CT angiography (CTA) for evaluation of clinically suspected PE. Out of 116 patients Sixteen (14%) patients were found to have PE on pulmonary CTA. Three risk factors--immobilization ($p < 0.001$), history of prior PE or DVT ($p = 0.001$), and cardiac disease ($p = 0.004$)--were found to be significant independent risk factors for the presence of PE detected by pulmonary CTA. When two or more risk factors were used as the clinical threshold, the sensitivity for positive PE was 75% (12/16 patients) and the specificity was 99% (99/100 patients).

Thus risk factors especially old history of DVT has a significant role in causing pulmonary embolism.

8. Right Ventricular Enlargement in Pulm Embolism

In this study Out of 43 pulmonary embolism patients 26(60.5%) patients had right ventricular enlargement. 17 (39.5%) patients did not have right ventricular enlargement. This parameter is important as it is an indcator of right ventricular strain due to pulmonary hypertension attributable to PE.

Kumamaru KK et al did a study to retrospectively evaluate prognostic accuracy of subjective assessment of right ventricle (RV) enlargement on CT pulmonary angiography (CTPA) images in comparison with objective measures of RV enlargement in patients with acute pulmonary embolism (PE) for 200patients. The specificity for subjective RV enlargement (55.4-67.7%) was significantly higher than objective measures (45.8-53.1%).

Cecilia Becattini[39] defined right ventricle dysfunction at MDCT as the right-to-left ventricle dimension ratio. Which proved that right to left ventricle dimension ratio ≥ 0.9 at MDCT had 92% sensitivity for right .In patients with acute pulmonary embolism MDCT can be used as a single procedure for diagnosis and risk stratification. Patients without right ventricle dysfunction at MDCT have a very low risk of in-hospital adverse outcome and could therefore candidate for home treatment

9. DVT in Patients With Pulmonary Embolism

In this study ,Out of 43 patients with pulmonary embolism 24 (92.3%)patients had DVT on USG and 19 (40.4%) did not .Out of 30 patients not having pulmonary embolism 2(7.7%) patients had deep venous thrombosis and 28 patients (59.6%) did not have deep venous thrombus .Majority of the patients diagnosed with pulmonary embolism had deep venous thrombosis on USG. 24 (55.8%) of the patients diagnosed with pulmonary embolism on MDCT PA had deep venous thrombosis on USG and 19(44.2%) patients did not have deep venous thrombosis.

Silverstein MD et al [8.5] did a study to estimate the incidence of deep vein thrombosis and pulmonary embolism in 2218 patients and showed that the overall average age- and sex-adjusted annual incidence of venous thromboembolism was 117 per 100000 (deep vein thrombosis, 48 per 100000; pulmonary embolism, 69 per 100000), with higher age-adjusted rates among males than females (130 vs 110 per 100000, respectively). The incidence of venous thromboembolism rose markedly with increasing age for both sexes, with pulmonary embolism accounting for most of the increase. The incidence of pulmonary embolism was approximately 45% lower during the last 15 years of the study for both sexes and all age strata, while the incidence of deep vein thrombosis remained constant for males across all age strata, decreased for females younger than 55 years, and increased for women older than 60 years.

Nazaroğlu Het al[31] did a study to investigate whether CT venography (CTV) performed after CT pulmonary angiography (CTPA) using MDCT provides additional findings in the diagnosis of thromboembolic disease on 360 patients. Acute PE and acute DVT were observed in 25.2% and 18.0%, respectively. The percentage of subsegmental emboli among patients with acute PE was 15.6%. The percentage of patients with thromboembolic disease was 29.1%. Of patients who were diagnosed as having thromboembolic disease, 13.5% (12 of 89 patients) had DVT only. Of all patients, 3.9% (12 of 306) had only isolated DVT. The number of patients with subsegmental PE who had DVT was two (0.7% all patients).

Ravenel J G et al[36] did a study on 181 patients A total of 41 patients (22.7%) were diagnosed with venous thromboembolism, 29 (70.7%) with PE, 8 (19.5%) with PE and DVT, and 4 (9.8%) with DVT. Seventeen deaths occurred within 30 days of CTA/CTV, of which none was felt to be related to PE/DVT. Of the 140 studies, 4 were determined to have venous thromboembolism (3 PEs and 1 DVT) within 30 days of the initial study (NPV = 97.1%).

Goodman LR et al [5]did CT venography following CT angiography (CTA) to detect pulmonary embolus. DVT was detected in 105 of 737 patients (14.2%). There was agreement for the presence of DVT in at least one leg (same leg) or for the absence of DVT in both legs in 133 of the 150 study patients (89%). The kappa statistic showed substantial agreement between the consensus interpretations and the test interpretations (kappa = 0.75; 95% CI = 0.64-0.86) per patient.

Sohns C et al 31did a study in 200 patients of Multidetector-row spiral CT in pulmonary embolism with emphasis on incidental findings. PE was detected in 60 of the 200 patients with a high clinical probability of having PE (30%). Thirty-four patients had a positive CT scan result for venous thrombosis (17%). Twenty-four of the 60 patients had proximal deep venous thrombosis (40%), and 2 patients had arm venous thrombosis (3%). Thirty-four of the 60 patients had PE without venous thrombosis (57%). Eight of the 200 patients had deep venous thrombosis without suspicion of PE (4%). The distribution of the proximal thrombi showed 15 in a central artery (25%), 13 in a main pulmonary artery (22%), and 32 in a lobar segmental artery (53%).

10. DDIMER and Pulmonary Embolism

In this study .Out of 73 patients 49 patients had raised D

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dimer levels and 24 had normal D dimer levels. 42 patients with raised d dimer levels (85.7%) had pulmonary embolism and 7(14.3%) patients did not have pulmonary embolism. The commonest cause of raised D dimer in these 7 patients was DVT. A significant P value<0.001 shows that d dimer is raised in almost all cases of pulmonary embolism.

The sensitivity of D Dimer for detection of pulmonary embolism was 100% and specificity was 75%.Hence a normal D-dimer level rules out pulmonary embolism.

Van Belle A et al[27] did a prospective cohort study to assess the clinical effectiveness of a simplified algorithm using a dichotomized clinical decision rule, D-dimer testing, and computed tomography (CT) in patients with suspected pulmonary embolism . Patients were categorized as "pulmonary embolism unlikely" or "pulmonary embolism likely" using a dichotomized version of the Wells clinical decision rule. Patients classified as unlikely had D-dimer testing, and pulmonary embolism was considered excluded if the D-dimer test result was normal. Pulmonary embolism was considered a possible cause of death in 7 patients after a negative CT scan (0.5% [95% CI, 0.2%-1.0%]). The algorithm was completed and allowed a management decision in 97.9% of patients. They concluded that a diagnostic management strategy using a simple clinical decision rule, D-dimer testing, and CT is effective in the evaluation and management of patients with clinically suspected pulmonary embolism. Its use is associated with low risk for subsequent fatal and nonfatal VTE.

Von Lode P [28] did a study on Sensitive and quantitative, 10-min immune fluorometric assay for D-dimer in whole blood. The limits of detection (background + 3SD) and quantification (CV <15%) were 0.05 and 0.2 mg/L D-Dimer, respectively, and the assay was linear up to 400 mg/L. D-dimer (r=0.190, n=149) were carried out using citrated plasma. Diagnostic sensitivity, specificity, and negative (NPV) and positive (PPV) predictive values were 98.7%, 64.4%, 99.1% and 55.1%, and 92.2%, 81.0%, 95.9% and 68.3%, respectively. The high sensitivity and NPV suggest that the rapid immune fluorometric assay could be valuable for rapid exclusion of VTE in outpatients. With appropriate cut-offs, the assay could potentially be used as a stand-alone test or combined with clinical probability assessment, but further studies are required.

Mos I C et al [47] did a study in 516 consecutive patients with clinically suspected acute recurrent PE without using anticoagulants. An unlikely clinical probability (Wells rule 4 points or less) was found in 182 of 516 patients (35%), and the combination of an unlikely CDR-score and normal D-dimer result excluded PE in 88 of 516 patients (17%), without recurrent venous thromboembolism (VTE) during 3month follow-up (0%; 95% CI 0.0-3.4%). CTPA was performed in all other patients and confirmed recurrent PE in 172 patients (overall prevalence of PE 33%) and excluded PE in the remaining 253 patients (49%). A diagnostic algorithm consisting of a clinical decision rule, D-dimer test and CTPA is effective in the management of patients with clinically suspected acute recurrent PE.

Guo Z et al [48] did a study to explore the incidence and clinical features of PE with normal DD concentrations. The frequency of normal DD concentrations in patients with PE was 4%. Previous episode(s) of PE were more common in patients with normal DD concentrations than in those with abnormal DD concentrations (P = 0.001). Fever, tachycardia, and tachypnea occurred less frequently in the former group (P < 0.05) and time between onset of symptoms and DD testing was longer (P = 0.001). The diagnosis of PE was delayed in 22 of the 29 cases. Nineteen and seven cases with normal DD concentrations were classified according to pretest scores as intermediate and low risk, respectively.

PE with normal DD concentrations is uncommon. Although most diagnoses of PE are ruled out by normal DD values, a small number of cases with PE are missed. A combination of pretest probability score and normal DD concentration increases the probability of making the correct diagnosis, but cannot completely exclude patients with suspected PE. When the clinical manifestations cannot be otherwise explained, clinicians should be alert to the possibility of PE with normal DD concentrations in patients with previous episode(s) of PE or a long interval between onset of symptoms and DD testing.

11. Anatomc Distribution of Thrombus on MDCT PA

In this study, majority of the patients had embolus in the right main pulmonary artery(26.6%). 32(24.4%) patients had embolus in the right side and 24 (18.3%) patients had on the left side.20 (26.6%) patients had embolus in the right main pulmonary artery and 13 (17.33%)patients had in the left main pulmonary artery.9 (12%)patients had embolus in the right lobar artery and 5(6.67%) patients in the left lobar artery.10(13.3%) patients had embolus in the right segmental artery and 10(13.3%) patients had embolus in

the left segmental artery.5(6.67%)patients had embolus in the right sub segmental artery and 2(2.67%) patients had embolus in the left sub segmental artery.

In a study done by Lee E Y et al[39] on 116 patients Sixteen (14%) patients were found to have PE on pulmonary CTA. The level of involvement of PE was segmental in 16 of 31 PEs (52%), lobar in eight (26%), subsegmental in five (16%), and main or central in two (6%).

Kritsaneepaiboon et al [32] did a study on 84childrenAll pulmonary CTA studies were technically successful in visualizing arteries to the level of segmental pulmonary arteries, but the evaluation of subsegmental pulmonary arteries was limited in 78 (80%) examinations. Thirteen (15.5%) of 84 children were found to have PE on pulmonary CTA. PE was localized in the lobar pulmonary artery in 12 (39%), the segmental pulmonary artery in 11 (35%), the subsegmental pulmonary artery in five (16%), and the main or central pulmonary artery in three (10%) patients. PE was distributed in the right lower lobe in 12 (37%), the left lower lobe in eight (24%), the right upper lobe in five (15%), the right middle lobe in four (12%), and the left upper lobe in four (12%) patients

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Smita Patel et al [14] did a study 60 patients suspected of having acute pulmonary embolism To compare the frequency of well-visualized pulmonary arteries according to anatomic level by using different collimation with single– and multi–detector row computed tomography (CT) Three thoracic radiologists independently reviewed examination findings to determine if each main, lobar, segmental, and subsegmental artery was well visualized for presence of pulmonary embolism. Reader 1 scored 95% (114 of 120), 96% (115 of 120), and 99% (119 of 120) of lobar arteries (*P*> .05); 76% (304 of 400), 86% (346 of 400), and 91% (363 of 400) of segmental arteries (*P*< .001); and 37% (300 of 800), 56% (448 of 800), and 76% (608 of 800) of subsegmental arteries as well visualized (*P*< .001) using techniques 1, 2, and 3, respectively. Reader 2 scored 97% (116 of 120), 95% (114 of 120), and 99% (119 of 120) of lobar arteries (*P*> .05); 77% (308 of 400), 87% (349 of 400), and 93% (371 of 400) of segmental arteries (*P*< .001); and 39% (310 of 800), 53% (422 of 800), and 78% (621 of 800) of subsegmental arteries (*P*< .001) as well visualized using techniques 1, 2, and 3, respectively. Reader 3 scored 86% (103 of 120), 82% (98 of 120), and 91% (109 of 120) of lobar arteries (*P*> .05); 63% (252 of 400), 70% (280 of 400), and 85% (339 of 400) of segmental arteries (*P*< .001); and 39% (310 of 800), 56% (451 of 800), and 71% (572 of 800) of subsegmental arteries (*P*< .001) as well visualized using techniques 1, 2, and 3, respectively. Sixteen patients had pulmonary mbolism. Interobserver agreement for detection of pulmonary embolism was significantly better for semental and subsegmental arteries for all readers with technique

3 (segmental, κ = 0.79–0.80; subsegmental, κ = 0.71–0.76) than that with technique 1(segmental, κ = 0.47–0.75; subsegmental, κ = 0.28–0.54).Multi–detector row CT at 1.25-mm collimation significantly improves visualization of segmental and subsegmental arteries and interobserver agreement in detection of pulmonary embolism

Winer-Muram HT et al [19]did a study to determine diagnostic accuracy of four-channel multi-detector row computed tomography (CT) in emergency room and inpatient populations suspected of having acute pulmonary embolism (PE) who prospectively underwent both CT and pulmonary arteriography (PA) in 93 patients. At PA, 18 patients (19%) had PE at 50 vessel levels (five main and/or inter lobar, 24 segmental, and 21 subsegmental), 17 (94%) of which had PE at multiple sites. At CT, 26 patients (28%) had PE at 71 vessel levels (24 main and/or inter lobar, 33 segmental, and 14 subsegmental). Twenty patients (77%) had PE at multiple sites. Review of eight false-positive CT studies showed an appearance highly suggestive of acute PE in three patients, chronic PE in one, and no PE in three; one study was inconclusive. CT better demonstrated large-level vessel involvement (P < .01), while PA better demonstrated small-level vessel involvement (P < .01).

Ghaye B et al [12] did a study to analyze the influence of multi-detector row spiral computed tomography (CT) on identification of peripheral pulmonary arteries. and concluded that Multi-detector row CT with reconstructed scans of 1.25-mm-thick sections enables accurate analysis of peripheral pulmonary arteries down to the fifth order on spiral CT angiograms.

Sohns C et al[31]did a study in 200 patients. PE was detected in 60 of the 200 patients with a high clinical probability of having PE (30%). Thirty-four patients had a positive CT scan result for venous thrombosis (17%). Twenty-four of the 60 patients had proximal deep venous thrombosis (40%), and 2 patients had arm venous thrombosis (3%). Thirty-four of the 60 patients had PE without venous thrombosis (57%). Eight of the 200 patients had deep venous thrombosis without suspicion of PE (4%). The distribution of the proximal thrombi showed 15 in a central artery (25%), 13 in a main pulmonary artery (22%), and 32 in a lobar segmental artery (53%).

12. Conclusion and Summary

MDCT-PA was found to be readily available even off routine hours, fast, relatively affordable and minimally invasive imaging modality in clinically suspected PE in patients without a contraindication to CTPA.

CTPA reveals significant additional diagnoses which ensure appropriate patient management is instituted without delay.

Parenchymal abnormalities and pleural effusion are present in the majority of patients undergoing CTPA for the clinical suspicion of PE, irrespective of the presence or absence of PE.

Other than wedge-shaped opacities, parenchymal and pleural abnormalities on CTPA do not correlate with the presence of PE in this study.

Evidence of right venticular strain on CTPA as evidenced by right ventricular to left ventricular diameter ratio >1 is significantly associated with pulmonary embolism.

Not all dyspnea is due to pulmonary embolism.

The referral algorithm is suboptimal as evidenced by the lack of a standardized clinical referral criteria and investigations as preliminary work-up to MDCTPA in suspected PE hence a large number of patients may be getting unnecessary CTPA in the local setup.

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