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APPLICATION OF COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY TO EVALUATE CLINICALLY SUSPECTED CASES OF PULMONARY THROMBOEMBOLISM

Radiology

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ABSTRACT

Background: Pulmonary Thromboembolism (PTE) is a disease of high mortality and morbidity both in acute and chronic phases but frequently overlooked because the symptoms and signs are very nonspecific. Most patients present to the department of emergency medicine with acute onset chest pain, dyspnoea. After the introduction of Computed tomography pulmonary angiography (CTPA) as it is almost replaced Pulmonary artery catheterisation. CTPA is becoming the gold standard investigation used in diagnostic algorithms of PTE. **Objectives:** To evaluate the role of CT pulmonary angiography in diagnosing acute and chronic PTE and to correlate the findings of CTPA with clinical and laboratory findings. **Material And Methodology:** During the one and half year period, a total of 50 patients with clinical suspicion of PTE who were referred for CTPA were taken into the study .The various risk factors, lab parameters and multimodality imaging findings were analyzed and CTPA findings were categorized by PAOI, presence or absence of PHTN and RVD and correlated with lab parameter of D- Dimer, Troponin -I and other imaging modalities like X Ray, Ultrasound venous doppler and 2D echocardiography. **Results:** In this prospective observational study out of 50 patients,54% patients had evidence of PTE. The male to female ratio of the patients having PTE is 1.45:1. The most common symptoms evoking a suspicion of PTE is dyspnea followed by cough, palpitation and chest pain. The most common comorbidity associated are hypertension and diabetes mellitus.Out of 50 cases, 13 cases were having elevated D-dimer values with normal Troponin-I. But in those 13 cases, 10(76.9%) cases were shown positive for PTE on CTPA.14 patients(51.8%) found to have DVT on venous doppler study. Out of 27 patients of positive PTE 16 (59.2%) patients had abnormal x-ray at presentation and commonly multiple x-ray findings co-exists in single X- ray. The sensitivity of the D-Dimer and CTPA test to correctly identify those with the condition was 85.19% and specificity was 86.96%. The sensitivity of Serum Troponin-I to correctly identify those with the condition was 51.85% which indicates moderate sensitivity and specificity was 91.30% suggesting that serum troponin I is highly effective in ruling out the condition in negative cases **Conclusion:** As the first-line imaging technique, CT pulmonary angiography (CTPA) has superseded catheter pulmonary angiography and ventilation-perfusion scintigraphy to become the clinical "gold standard" for the detection of acute PTE. From this study, combination of CTPA with D-Dimer and Troponin I not only increases the accuracy in diagnosing PTE, but also stratification of patients with PTE.

KEYWORDS

Pulmonary thromboembolism, D-Dimer, Troponin -I, Computed Tomography pulmonary Angiography.

INTRODUCTION

Pulmonary Thromboembolism(PTE) is a disease of high mortality and morbidity both in acute and chronic phases following coronary artery disease and cerebrovascular disease. PTE is frequently overlooked because the symptoms and signs are very nonspecific and may be confused with a variety of other cardiopulmonary disorders that have similar presentations. Hence it is called "great masquerader".

Most patients present to the department of emergency medicine with acute onset chest pain mostly pleuritic in nature, tachypnoea with or without dyspnoea. But the classic clinical triad comprising sudden chest pain, dyspnoea and haemoptysis is present in a minority of cases only. Other symptoms and signs which are usually present in patients with PTE are cough, palpitations, tachycardia ,syncope ,fever with or without the signs and symptoms of Deep vein thrombosis (DVT), but may also presents with cardiovascular collapse or cardiac arrest in severe PTE especially who have high clot burden.

PTE is a multifactorial disease but can occur in patients without any identifiable predisposing factors. Hereditary risk factors like antithrombin deficiency, protein C and protein S deficiency, are strong risk factors for PTE, but account for only 1% of all cases of PTE. Acquired risk factors are prolonged surgical procedures like orthopedic surgery, onco-surgery and neurosurgery, previous history of venous thromboembolism, prolonged immobility, hospitalization, infection, pregnancy, Hormone Replacement Therapy including Oral contraceptives and cancer. Recently COVID-19 (Coronavirus -2019) became a rising cause of PTE.[1]

Since clinical diagnosis of PTE is very often inaccurate the combined use of various laboratory and imaging investigations like D-dimer, Duplex USG of lower limbs, echocardiography, lung scintigraphy /VQ Scan, computed tomography pulmonary angiography becomes mandatory. But the choice of these investigations depends primarily on the clinical conditions of patients; for example CTPA is not an investigation of choice in pregnancy. The approach to PE has changed considerably after the introduction of multi-detector Computed tomographic pulmonary angiography (CTPA) as it is almost replaced Pulmonary artery catheterisation. CTPA is becoming the gold standard investigation nowadays in patients withclinical suspicion of PTE and is an essential investigation used in diagnostic algorithms of PTE. CTPA has reached the sensitivity and specificity of 83% and 96% respectively as per PIOPED II trial and in combination with clinical and lab findings, the positive predictive value (PPV) becomes almost 96%.[2] CTPA is not limited to diagnosis of PTE but also can identify other pathologies of symptoms like chest pain, shortness of breath such as pneumonic consolidations, pericardial diseases, benign and malignant neoplastic nodules, musculoskeletal pathologies, vasculitis and even myocardial ischemia in some diagnostic protocols.

However, Even though it is easily available, there are some limitations like usage of iodinated contrast, ionizing radiation, contrast allergy and contrast induced nephropathy and relative contraindications in renal insufficiency and pregnancy . It is estimated that about 5 - 10% of patients with clinical suspicion of PTE have contraindications to undergo CTPA.[3]

Aims And Objectives

Aim: To assess the role of Computed Tomography Pulmonary Angiography (CTPA) in evaluation of clinically suspected cases of Pulmonary Thromboembolism (PTE).

Objectives:

1. To assess the role of CTPA in diagnosing both Acute as well as Chronic PTE with its complications.
2. To correlate the findings of CTPA with clinical history ,laboratory findings and other imaging modalities including Doppler ultrasonography, Chest X ray, 2D -echocardiography.

3. To identify the clinical mimics of PTE.

Materials And Methodology

Source Of Data: The subjects of this study included patients attending Kamineni Hospital, King Koti referred to our radiodiagnosis department with clinically suspected PTE for CTPA.

Study Design: This is a prospective-observational study.

Duration Of Study: The study was done over a period of 18 months from September 2022 to March 2024 and data was collected during this period from all the patients who underwent CTPA in whom PTE was suspected.

Inclusion Criteria:

1. All patients of different age groups with suspected PTE clinically who were referred for CTPA to the radiodiagnosis department of Kamineni Hospital,King Koti were included in the study.

Exclusion Criteria:

1. Allergy to contrast agent,
2. Impaired renal function tests,
3. Pregnancy .

Sample Size - A total of 50 patients were included in the study who had clinical suspicion of PTE and were referred for CTPA to our radiodiagnosis department.

Study Data Collection Method:

This study was conducted as per the guidelines of the Indian Council of Medical Research with approval of our institutional Research Ethics Committee All selected subjects were briefed about the study. Individual's consent was taken to use their identified data for research purposes. Confidentiality was maintained. Relevant clinical history and laboratory data were entered into case sheet proforma. Also, case sheets of the subjects were referred for any other relevant details.

Statistical Analysis:

Data was entered in Microsoft Excel spreadsheet. The continuous variables such as age was expressed in terms of average ± standard deviation (SD).and categorical variables such as clinical signs, symptoms, risk factors etc were presented as percentages. Bar charts and pie charts were used to describe the data. Analysis was done in SPSS software version 17.0

RESULTS

During the one and half year duration, a total of 50 patients who were referred to our radiology department for Computed tomography pulmonary angiography (CTPA) with clinical suspicion of PTE were taken into the study after having met the prerequisites of the inclusion criteria of the study and consent were obtained. All the cases had been analyzed and the results were depicted in the form of tables and diagrams to carry out the objectives of my study.

Prevalence:

Out of 50 patients with clinical suspicion of PTE who underwent CTPA, 27(54%) patients had evidence of PTE and 23 (46%) patients did not have PTE.

Age Distribution:

In my study only one patient below 20 years of age, presented with clinical suspicion of CTPA diagnosed positive (100%). Most of the patients belong to the 61- 80 years group and the lowest belong in the ≤20 years age group. The mean age of the CTPA positive patients in the study was 52.8 years and the standard deviation was +/- 19.3 years. The youngest patient who participated in the study was aged 19 years and was subsequently diagnosed to be CTPA positive. The oldest patient was diagnosed CTPA positive aged 83. The oldest patient participated in the study was aged 92 years.

Gender Distribution:

In a total of 27 male patients who were referred for CTPA with clinical suspicion of PTE, 16 were positive (59.2%). In a total of 23 female patients who were referred for CTPA with clinical suspicion of PTE, 11 were positive (40.8%).In my study male patients are more who had clinically suspected PTE than females. The male to female ratio of the patients having PTE is 1.45:1.

Clinical Presentation:

Dyspnea is the most common symptom evoking clinical suspicion of PTE followed by cough, palpitation and chest pain. Out of 50 patients, 44 patients presented with dyspnea, 29 were with cough, 12 were with palpitations and 11 were with chest pain and leg swelling. Of the diagnosed cases of PTE, 85.2% of the patients presented with chief complaints of dyspnea. 44.4 % of the patients had cough as the major complaint. Palpitations (33.3%) is the 3rd most common presenting complaint. Cardiac arrest and syncope were exclusively seen in PTE positive patients though being a rare presentation and reflects the cardiovascular collapse in PTE pathology.

Risk Factors And Comorbidities:

In my study the most common comorbidity associated with clinically suspected PTE patients includes hypertension and diabetes mellitus. The risk factors of PTE such as recent history of surgery, tuberculosis, bronchial asthma and varicose veins are exclusively seen in the CTPA positive patients (100%) and absent in CTPA negative patients in my study.

Lab Investigations: The cut-off values for positive D-Dimer and positive Troponin-I were fixed to 0.5 micrograms per ml FEU and >0.4µg/L respectively. Out of 50 cases, 13 cases were having elevated D-dimer values with normal Troponin -I. But in those 13 cases, 10(76.9%) cases were shown positive for PTE on CTPA.

A total of 13 cases were having both elevated D-dimer values with elevated Troponin -I and all of them (100%) were positive for PTE on CTPA. In the 21 cases, in which both D- dimer and Troponin -I levels were less than the fixed cut-off, 18(85.7%) cases there was no evidence of pulmonary embolism on CT pulmonary angiography and 3(14.3%) cases had thrombus.

Table 1: Correlation of D-Dimer with CTPA in suspected cases of PTE

D-DIMER		CTPA		Total
		Negative	Positive	
Negative	Count	20	4	24
	%	40.0%	8.0%	48.0%
Positive	Count	3	23	26
	%	6.0%	46.0%	52.0%
Total	Count	23	27	50
	%	46.0%	54.0%	100.0%

The chi-square statistic is 25.897.The p-value is 0.00000238. The result is significant at $p < .05$. Kappa value is 0.719

This distribution indicates a strong agreement between the two tests, which is further supported by a Kappa value of 0.719, signifying substantial agreement beyond chance. The p- value of 0.001 indicates that this agreement is statistically significant, making it highly unlikely that the observed concordance is due to random variation. The statistical analysis of the D-Dimer and CTPA test results reveals robust diagnostic performance.

The sensitivity, or the ability to correctly identify those with the condition, is 85.19%, with a confidence interval (CI) ranging from 66.27% to 95.81%. Specificity, the ability to correctly identify those without the condition, stands at 86.96%, with a CI of 66.41% to 97.22%.

Table 2: Statistical Parameters Of D-dimer In Diagnosing PTE

Statistics	Value	95% CI
Sensitivity	85.19%	66.27% to 95.81%
Specificity	86.96%	66.41% to 97.22%
Positive Likelihood Ratio	6.53	2.25 to 18.98
Negative Likelihood Ratio	0.17	0.07 to 0.43
Positive Predictive Value	88.46%	72.51% to 95.70%
Negative Predictive Value	83.33%	66.62% to 92.61%
Accuracy	86.00%	73.26% to 94.18%

Overall, the accuracy of the test, which reflects the proportion of true results among the total cases examined, is 86.00%, with a CI from 73.26% to 94.18%. These metrics underscore the reliability and effectiveness of the D-Dimer test in conjunction with CTPA for diagnostic purposes.

Table 3: Correlation Of Serum Troponin- I With CTPA .

Serum		CTPA		Total

Troponin I		Negative	Positive	
Negative	Count	21	13	34
	%	42.0%	26.0%	68.0%
Positive	Count	2	14	16
	%	4.0%	28.0%	32.0%
Total	Count	23	27	50
	%	46.0%	54.0%	100.0%

The chi-square statistic is 10.63.The p-value 0.00491728. The result is significant at p < .05. Kappa value is 0.417

The Kappa value of 0.417 suggests moderate agreement between Serum Troponin I levels and CTPA results beyond chance. The statistical significance of this agreement is confirmed by a p-value of 0.001, indicating that the observedrelationship is unlikely to be due to random variation. This data implies that while there is some concordance between Serum Troponin I and CTPA results, it is less pronounced compared to other diagnostic tests.

Table 4: Statistical Parameters Of Troponin I In Diagnosing PTE

Statistics	Value	95% CI
Sensitivity	51.85%	31.95% to 71.33%
Specificity	91.30%	71.96% to 98.93%
Positive Likelihood Ratio	5.96	1.51 to 23.54
Negative Likelihood Ratio	0.53	0.35 to 0.80
Positive Predictive Value	87.50%	63.94% to 96.51%
Negative Predictive Value	61.76%	51.71% to 70.91%
Accuracy	70.00%	55.39% to 82.14%

Ultrasound Venous Doppler:

Out of 27 patients with positive PTE, 14 patients(51.8%) found to have DVT on venous doppler study. In the 11 patients with negative PTE who underwent venous doppler study, only one patient (9.1%) was positive for DVT.

Chest Radiography:

Table 5: Frequency of Chest X-ray findings in clinically suspected PTE patients (N=50).

Chest X ray findings	CTPA Positive (n=27)	CTPA Negative (n=23)
Airspace Opacity	8(29.6%)	5(21.7%)
Reticular opacity	1(3.7%)	1(4.3%)
Pleural effusion	7(26.0%)	7(30.4%)
Cardiomegaly	8(29.6%)	11(47.8%)
Prominent hila	1(3.7%)	2(8.7%)
COPD	1(3.7%)	1(4.3%)
Focal bronchiectasis	0(0%)	1(4.3%)
Hydropneumothorax	1(3.7%)	0(0%)
Rib fractures	0(0%)	1(4.3%)
Nil findings	11(40.8%)	3(13.0%)

The chi-square statistic is 8.619797215711. The p-value is 0.473083723878. The result is not significant at p < .05.

Echocardiography:

Table 6: Comparison of 2D Echo with CTPA in diagnosing PHTN and their association with PAOI (N=27).

PAOI	2D Echo		CTPA	
	PHTN Present	PHTN Absent	PHTN Present	PHTN Absent
≤ 50%(n=23)	7	16	8	15
> 50%(n=4)	3	1	4	0
	The chi-square statistic is 2.902. The p-value 0.08846972. The result is not significant at p < .05.		The chi-square statistic is 5.87. The p- value 0.01540105. The result is significant at p < .05	

Table 7: Comparison of 2D Echo with CTPA in diagnosing RVD and their association with PAOI (N=27)

PAOI	2D Echo		CTPA	
	RVD Present	RVD Absent	RVD Present	RVD Absent
≤ 50%(n=23)	6	17	7	16
> 50%(n=4)	3	1	4	0
	The chi-square statistic is 3.668. The p- value 0.0554667. The result is not significant at p < .05.		The chi-square statistic is 6.83. The p- value 0.00896393. The result is significant at p < .05	

Correlation Of CTPA Findings With PAOI:

Table 8: Correlation of CTPA findings with PAOI of PTE positive patients (N=27).

Findings	PAOI	
	<50%(N=23)	>50%(N=4)
Infarct	5(21.7%)	2(50%)
Dilated MPA	6(26.0%)	3(75%)
Dilated RPA	7(30.4%)	4(100%)
Dilated LPA	9(39.1%)	4(100%)
IVS Bowing	7(30.4%)	4(100%)
RVD/LVD>1	7(30.4%)	4(100%)
IVC Reflux*	5(21.7%)	4(100%)

The chi-square statistic is 0.617172006737. The p-value is 0.996107279229. The result is not significant at p < .05.

*IVC reflux of contrast could not be assessed in one patient due to IVC thrombosis.

The features of RVS and PHTN are present in all patients with PAOI >50% and pulmonary infarct seen in 2 out of 4 (50%) patients with PAOI >50%. But the percentage of pulmonary infarct in the patient group with ≤50%PAOI is 21.7 % only.

Associated Findings:

Pleural effusion and cardiomegaly was the most common finding in the PTE positive patients each comprising 8 (29.6%) patients. Pulmonary infarcts seen in 6 (22.2%) patients and atelectactic bands seen in 4(14.8%) patients followed bymosaic attenuation(7.4%),concomitant post infective pneumonia(7.4%) .Other less common finding like COPD, Chronic bronchitis, aspiration pneumonia, pulmonary edema, hydropneumothorax and ILD seen in each 1 (3.7%) patients,

Differential Diagnosis:

Table 9: Frequency of differential diagnosis on CTPA in PTE negative patients(n=23).

Differential Diagnosis	No Of Cases (n=23)	%
Secondary Pulmonary Hypertension (2° To COPD/ ILD)	4	17.4
Idiopathic Pulmonary Hypertension	4	17.4
COPD	3	13
Post Infective Pneumonia	3	13
Normal	3	13
Interstitial Lung Disease	2	8.7
Pulmonary Edema	2	8.7
Tuberculosis	2	8.7
Lung Malignancy	1	4.3
Fibrosis	1	4.3
Heart Failure	1	4.3
Post Traumatic Sequelae	1	4.3
Aspiration Pneumonia	0	0

DISCUSSION

This study evaluated the diagnostic efficiency of computed tomography pulmonary angiography (CTPA) in the diagnosis of pulmonary thromboembolism(PTE). This was a prospective observational study conducted to compare the results of CTPA with clinical and laboratory results and assess the use of CT pulmonary angiography in the diagnosis of acute and chronic PTE. A total of fifty patients with clinical suspicion of PTE who were referred to our department of radiodiagnosis for CTPA over the course of a year and a half were included in the study after fulfilling the requirements for inclusion while providing their consent.

Incidence And Prevalence Of Pulmonary Thromboembolism:

The annual incidence of PTE ranges from 39 to 115 per 100,000 people; theincidence of deep vein thrombosis (DVT) is 53 to 162 per 100,000 people.[4] Acute PTE is the 3rd most common type of cardiovascular disease, following stroke and coronary artery disease(CAD).[5] Acute PTE is more common in men than in women.[6] The high accuracy and ease of use of CT scans led to a five-fold increase in CTPA examinations within six years, but also decreased PTE prevalence.[7] In the present study, 27 patients (54%) had evidence of PTE, while 23 patients (46%) did not have any PTE.

These results were in line with those of Attia NM et al, who found that 50% of patients tested positive for PE in CTPA.[8] On the other hand, Ooi MWX et al. found that 38% of COVID-19 patients who underwent CTPA study had PTE.[9] Nduta et al. found similar outcomes, with positive results in 27% of patients.[10] Burkill GJ et al. found that CTPA tested positive for PTE in 28 out of 101 patients (28%).[11] Qanadli SD et al. performed CTPA on 158 patients, of whom 54 (34%) were found to have PTE.[12]

Age And Sex Distribution:

Men and women have similar overall age-adjusted incidence of PTE, but women have higher relative rates of PTE in early and mid-adulthood (ages 20–40), while men have higher rates of PE after age 60.[13] Pathophysiological mechanisms that have traditionally been used to explain sex-based differences surrounding PTE include differences in the prevalence of vasculopathies, changes in sex hormones, physiological changes during pregnancy and postmenopause, and use of oral contraceptive pills (OCPs) or hormone replacement therapy (HRT).[14] Like HRT, pregnancy can cause a prothrombotic state that can result in venous thrombosis and subsequent PTE.[15] In the current study, only one patient was under the age of 20. The patients were divided into two age groups: those with 61–80 years old and those with ≤20 years old .

The CTPA positive patients in this study had a mean age of 52.8 ± 19.3 years. The average age was found to be 61 years in another study by Rodrigues B et al.[16] Ahmad DS et al. reported an average age of 53 years, and Kang DK et al. reported an average age of 55 years for acute PE cases.[17,18] The male to female ratio of PTE patients in this study is 1.45:1. These results were consistent with studies by Nduta et al. that found an average age of 52.6 years. These were in line with the male predominance results of Nduta et al., Rodrigues et al, and Ahmad DS et al. [10,16,17]

Clinical Presentation:

The presenting symptoms and signs of patients with PTE are usually non- specific, and the clinical presentation varies widely, contingent on the duration,degree of pulmonary vascular bed obstruction, patient's age and other factors.[19] In terms of diagnosis and treatment planning, it is critical to distinguish between patients who are hemodynamically stable and those who are unstable. Patients classified as unstable may have hypotensive shock, which is defined as a systolic blood pressure of less than 90 mmHg or a pressure drop of more than 40 mmHg for a minimum of 15 minutes without the presence of hypovolemia, sepsis, or new onset arrhythmia.[20] The patient's cardiopulmonary reserve, which can be influenced by underlying cardiac or pulmonary diseases, also influences the patient's clinical presentation and outcome .[21]

The presenting clinical symptoms of dyspnea, especially sudden onset, chest pain, or syncope, either alone or in combination, are what typically lead to a suspicion of PTE in the vast majority of patients. Classical presentations of PTE include acute onset dyspnea, chest pain, cough with or without hemoptysis which reflects pulmonary infarct in PTE patients. On the one end of the PTE spectrum, a patient with non-massive PTE/ low risk may be normotensive and have no or mild complaints whereas on the other end, the patient may experience severe dyspnea, tachypnea and chest pain during mild exertion or even at rest. It is crucial to take syncope into account as a distinct, albeit rare, manifestation of PE that also suggests a decreased hemodynamic reserve. Syncope affects approximately 8–19% of PTE patients. Rapidly onset isolated dyspnea is frequently linked to a PTE which involves large central pulmonary arteries. Thus has more noticeable associated hemodynamic instability and increased risk of pulmonary infarction. It can also be connected to angina-like chest pain that could be indicative of right ventricular ischemia .[22] As a result, diagnosing PE in patients who already have heart or lung disease may be particularly challenging.

The most frequent symptoms in the current study that raised the possibility of PTE were dyspnea, which was followed by cough, palpitations, and chest discomfort. Dyspnea was the primary complaint mentioned by 85.2% of the individuals with PTE diagnosis . Cough was the main complaint in 44.4% of the patients. The third most frequent presenting ailment was palpitations (33.3%). Despite being an uncommon presentation, cardiac arrest and syncope were only seen in PTE positive individuals, which is indicative of the circulatory collapse in the PTE pathogenesis.

These clinical symptoms aligned with the results of Nduta et al., who found that cough and dyspnea were the most prevalent clinical findings.[10] However, their investigation did not identify a statistically significant correlation between clinical symptoms and PE status. Therefore, not every instance of dyspnea is the same as apulmonary embolism. Additionally, according to Rodrigues B et al., there is no discernible correlation between the QS score and the clinical presentation upon admission (chest discomfort, cough, dyspnea).[16]

Comorbidity And Risk Factors:

Patients with PE often have significant comorbidities, such as chronic cardiorespiratory disorders or neoplasms, which might contribute to the development of this condition and can lead to a poorer short-term prognosis. Comorbidities such as chronic renal disease, cancer, diabetes, obesity, and hypertension some of which are recognized risk factors for PE—have grown dramatically over time.[23] Comorbidities have been reported to be increasing over time.

Venous thromboembolism (VTE) is thought to be the consequence of a combination of the risk factors specific to each patient and the environment in which the event takes place. Risk factors connected with the patient are often long-term, whereas the conditions are often temporary. Age, a personal history of VTE, an active cancer or other debilitating illness like heart or lung failure, congenital or acquired coagulation disorders, hormone replacement therapy, and oral contraception are all considered risk factors for patients.[23] The most common comorbidity linked to patients with clinically suspected PTE was diabetes mellitus and hypertension. Only the PTE positive patients (100%) in this research had the risk factors for PTE, such as varicose veins, a recent history of surgery, TB, and bronchial asthma.Diabetes was shown by Keller K et al. as an independent predictor of in-hospital mortality in PTE patients.[24]

Comparison Of CTPA And Lab Investigations (D-dimer And Troponin -I):

However, there is limited information available about other prognostic markers and their clinical implications.[25,26] Troponin levels are most helpful when used in conjunction with clinical prognostic scores and evidence of right ventricular strain on echocardiography or computed tomography.[27] Of the 50 patients, 13 had normal Troponin I but increased D- dimer readings. Of those 13, 10 (76.9%) had positive PTE results on CT pulmonary angiography. In the current investigation, there was a statistically significant correlation between CTPA and D-dimer and troponin-I.

The correlation between D-dimer test results and Troponin I levels with CPTA was statistically significant in the current study (p=0.001). Previous studies by Perrier A et al and Crawford F et al reported the significant increase in D-dimer levels in patients with acute PTE.[28] On the other hand, a study by Burkill GJ et al on the use of D-dimer assay in patients undergoing CTPA found that the assay's negativepredictive value was 94%. (p = 0.001).The accuracy rate in identifying those with the condition was 85.19%, while the accuracy rate in identifying those without it was 86.96%. These parameters highlight the D- Dimer test's dependability and efficacy when used in combination with CPTA for diagnostic purposes. In respect to CPTA findings, troponin I is more useful for verifying rather than ruling out the condition due to its high PPV, moderate sensitivity, and NPV.

Specificity And Sensitivity Of D-dimer In CTPA-Positive Group:

As a byproduct of cross-linked fibrin degradation, D-dimer levels rise in acute thromboembolic events.[29] D-dimer concentrations can be used to diagnose or rule out PTE, but their specificity is low because they can also be elevated in other clinical conditions such as postoperative states, infections, cancer, and old age, which are all associated with additional fibrin formation.[30] In the current study, the sensitivity to correctly identify those with the condition was 85.19%, and the specificity to correctly identify those without the condition was 86.96%. PTE was diagnosed with a sensitivity of 70.3% and specificity of 70.1% in patients hospitalized to the intensive care unit (ICU) by Hajsadeghi S et al.[31]

Sensitivity And Specificity Of Troponin-I In CTPA- Positive Group:

The most precise and sensitive biochemical indicators of myocardial damage are cardiac troponins. Along with myocardial infarction, other conditions that may cause elevated troponin levels include acute pericarditis, myocarditis, acute PTE, severe cardiac failure, sepsis, and acute renal failure.[32] When pulmonary artery blockage occurs,

increasing pulmonary vascular resistance raises the right ventricle's mechanical load in acute PTE. An abrupt dilation of the right ventricle may result from that. Elevations in troponin levels especially Troponin -I and severe myocardial ischemia may result from the right ventricle's dilation and hypokinesia.[33]

Other biomarkers that have been considered include cardiac troponin-T and pro- BNP. Pro-Brain Natriuretic Peptide (Pro-BNP), which is primarily secreted from ventricles that are under stress from hemodynamic status instability and congestive heart failure, is one of several biomarkers that have recently been considered for risk stratification and predilection of mortality of PTE cases.[34] An accurate and sensitive marker for assessing ventricular function is pro-BNP.[35] In addition, Kucher et al. demonstrated that pro-BNP levels > 90 pg/ml could predict risk of mortality and the need for mechanical ventilation and cardiopulmonary resuscitation in patients with PTE.[36] Lega et al. conducted a meta analysis ,linked elevated Pro-BNP and troponin levels to a worse prognosis for PTE patients.[37] Elevated Troponin I and T correlatedwith increased mortality rate owing to the right ventricular dysfunction.[38]

The positive likelihood ratio (PLR) is 5.96, indicating that patients with a positive Serum Troponin I result are nearly six times more likely to have a positive CPTA result compared to those with a negative Serum Troponin I result. The negative likelihood ratio (NLR) is 0.53, implying that a negative Serum Troponin I result does not strongly rule out the condition.

The positive predictive value (PPV) is 87.50%, meaning that 87.5% of those with a positive Serum Troponin I result actually have the PTE, while the negative predictive value (NPV) is 61.76%, indicating that about 61.8% of those with a negative result are truly absence of PTE . The overall accuracy of Serum Troponin I in this context is 70.00%, with a CI from 55.39% to 82.14%, reflecting a moderate level of correctness in identifying both true positive and true negative cases. In the present study, sensitivity was 51.85% and specificity was 91.30%.In contrast, elevated cTnI levels were detected in 11.6% of patients without PTE and 50.8% of patients with PTE (P<0.001) in a study by Kilinc G etal.[39] The test's sensitivity and specificity for the diagnosis of PTE were 50.7% and 88.3%, respectively.

Venous Doppler Studies:

While venous doppler ultrasonography is currently the first-line modality in diagnosing for DVT due to significant improvement in ultrasound image quality and updation of new sonographic software , X-ray / catheter contrast venography has played a significant role in the diagnosis of DVT in the past.[40] CT venography is the preferred next modality of choice in case of patients with poor body habitus causing poor ultrasonic penetration, equivocal cases and in the cases of DVT in deep pelvic veins.

In the current study, of the 27 patients with positive PTE, 14 patients (51.8%) had DVT on venous doppler study. Just one patient (or 9.1%) tested positive for DVT out of the 11 individuals with negative PTE who had venous doppler studies. In contrast to Ismail et al,[41] in Kano, who reported positive results in 55.8% of cases, Agunloye et al [42]. a study on suspected cases of DVT, from Ibadan of South-Western Nigeria and another study from Jos, North Central Nigeria, by Salaam et al. [43]found that only 46.6% and 56.3% respectively were positive after Doppler sonography who had clinical signs of DVT . However these results are somewhat higher than the present study observation of positive DVT in only 43% who had PTE.

Chest X-Ray Findings:

The most frequent x-ray findings in the current investigation were cardiomegaly and airspace opacity, with pleural effusion, which was seen in 26% of the patients ,coming in second. Our results were in line with those of Elliot et al., who reported that the most frequent abnormality on chest radiography linked to acute pulmonary embolism was cardiomegaly.[44] Greenspan et al. observed that while conventional chest X-ray findings were abnormal in the majority of PTE cases, they were normal in 40% of cases.[45]

Correlation Of CTPA Findings With Paoi Of CTPA Positive Patients:

Various studies evaluating pulmonary embolism by CTPA have used various pulmonary vascular bed obstruction scores like miller index. modified miller index, Qanadli index, mastora score etc. Qanadli index was used to quantify the degree of pulmonary arterial obstruction in my study as it is not time consuming with high reproducibility. Various studies use various PAOI cut off values of Qanadli index to categorize the PTE into severe or non severe.Based on a receiver operating characteristic (ROC) curve that corresponds to 45% PAOI, Rodrigues B et al. created a cut off value of 18 for Qanadli score.[16] Attia NM et al. built a ROC curve to determine the cut off value of 43% for PAOI.[8]

Correlation Of CTPA Findings With 2D Echocardiography Of CTPA Positive Patients:

The diagnostic capability of 2-D echocardiography was analyzed in assessing RVD and PHTN in patients with positive PTE considering the CTPA as gold standard along with correlation of Pulmonary Artery Obstruction Index (PAOI). It was observed that in the PAOI≤ 50% group the proportion of PTE patients with PHTN diagnosed by 2D echocardiography was 30.4% whereas in the PAOI >50%group it was 75%, 68.8%, 81.3% and 75% respectively. The proportion of RVD in positive PTE patients with PAOI≤ 50% group and PAOI >50%group were 26% and 75% .However, on statistical analysis it was found that statistically not significant. In the study by Rodrigues et al[16],in the CTPA positive patients with >18 Qanadli score had higher incidence of PHTN on echocardiography on admission with no statistical significance.This was similar to this study. Qanadli SD et al 27 and Miller GA et al[46] also reported similar observations. In PTE patients, obstruction of the pulmonary arterial tree with thromboemboli results in increased pulmonary arterial bed resistance, in turn causing secondary pulmonary hypertension.

Thus 2-D echo is the most useful imaging modality in day to day clinical practice to assess RVD and PHTN as it is non-invasive and relatively low cost. Hence, RVD assessed on 2D echocardiography has been considered as one of the strongest predictors of early mortality in PTE.

Correlation Of CTPA Findings For PHTN And RVD With PAOI:

It was noted that in PAOI ≤50% group ;the signs of PHTN SUCH AS the proportion of dilated main pulmonary artery (PA/Ao >1), enlarged right pulmonary artery and enlarged left pulmonary artery were 26%,30.4% and 39.1% .Whereas in the PAOI >50% group it was 75%,100% and 100% respectively. This indicates the frequency developing PHTN secondary to PTE is significantly high in the PAOI> 50% group. The proportion of features of RVD like enlarged right ventricle (RV/LV>1),IVS bowing and IVC reflux in the PAOI ≤50% group were 30.4%,30.4% and 21.7 % respectively. In the PAOI >50% group it was 100% in all parameters. Pulmonary infarct incidence also reached up to 50% in the current study with the patients of PAOI >50% .

These above observations suggest that incidence of PHTN and RVD is significantly increased as the PAOI values increases and are found to be statistically significant. A study done by Rodrigues B et al[16] showed that incidence of RVD is significantly higher in the QS> 18 group in CTPA; whereas PHTN incidence was almost similar in both QS> 18 and QS< 18 groups.This current study's findings are similar to the study conducted by Attia NM et al [8], where incidence of RVD and PHTN was significantly higher with increasing PAOI.

In the current study ,the most common associated finding in PTE positive patients are pleural effusion and cardiomegaly seen in 29.6% each followed by pulmonary infarct in 22.2% . Apart from the PTE the close clinical mimics which are diagnosed in the CTPA are idiopathic PHTN and secondary PHTN as they share common clinical presentation and can be ideally diagnosed by CTPA.

CONCLUSION

As the first-line imaging technique, CT pulmonary angiography (CTPA) has superseded catheter pulmonary angiography and ventilation-perfusion scintigraphy to become the clinical "gold standard" for the detection of acute pulmonary embolism (PE). 54% patients had evidence of PTE with majority of the patients belonging to 61–80-year age group and higher male preponderance. The most common symptoms evoking a suspicion of PTE were dyspnea followed by cough, palpitation and chest pain.

The most common comorbidity associated with clinically suspected PTE patients included hypertension and diabetes mellitus. We observed that pleural effusion and cardiomegaly was the most common finding in the PTE positive patients. Chest Xraywas normal in 40.8% CTPA Positive cases

The sensitivity of the D-Dimer and CTPA test to correctly identify those with the condition was 85.19% and specificity was 86.96%. The sensitivity of Serum Troponin I to correctly identify those with the condition was 51.85% which indicates moderate sensitivity and specificity was 91.30% suggesting that serum troponin I is highly effective in ruling out the condition in negative cases.

PE, or pulmonary embolism, is a potentially fatal condition that has to be properly diagnosed and treated. Because computed tomography pulmonary angiography (CTPA) is exceptionally efficient at identifying and ruling out acute PE, it is the diagnostic imaging method of choice for individuals who may be experiencing acute PE. Dual-energy or subtraction procedures are examples of new CT innovations that may further enhance prognosis and diagnosis for better patient care.

REFERENCES

1. Sakr Y, Giovini M, Leone M, Pizzilli G, Kortgen A, Bauer M, Tonetti T, Duclos G, Zieleśkiewicz L, Buschbeck S, Ranieri VM, Antonucci E. The clinical spectrum of pulmonary thromboembolism in patients with coronavirus disease-2019 (COVID-19) pneumonia: A European case series. J Crit Care. 2021 Feb;61:39-44. doi: 10.1016/ j.jcrc. 2020.09.021. Epub 2020 Sep 25. PMID: 33075608; PMCID: PMC75 18181.
2. Stein PD, Fowler SE, Goodman LR, Gottschalk A, Hales CA, Hull RD, Leeper KV Jr, Popovich J Jr, Quinn DA, Sos TA, Sostman HD, Tapson VF, Wakefield TW, Weg JG, Woodard PK; PIOPED II Investigators. Multidetector computed tomography for acute pulmonary embolism. N Engl J Med. 2006 Jun 1;354(22):2317-27.doi:10.1056/ NEJMoa052367. PMID: 16738268.
3. Medson K, Vargas-Paris R, Nordgren-Rogberg A, Sigbergsdottir A, Nyrén S, Lindholm P. Primary diagnosis of pulmonary embolism with unenhanced MRI for patients not eligible for CTPA: Clinical outcome. Eur J Radiol Open. 2019 Oct 3;6:315-319. doi: 10.1016/j.ejro.2019.08.004. PMID: 31692624; PMCID: PMC6804887.
4. Wendelboe AM, Raskob GE. Global Burden of Thrombosis: Epidemiologic Aspects. Circ Res. 2016 Apr 29;118(9):1340-7.
5. Raskob GE, Angchaisuksiri P, Blanco AN, Buller H, Gallus A, Hunt BJ, Hylek EM, Kakkar A, Konstantinides SV, McCumber M, Ozaki Y, Wendelboe A, Weitz JI., ISTH Steering Committee for World Thrombosis Day. Thrombosis: a major contributor to global disease burden. Arterioscler Thromb Vasc Biol. 2014 Nov;34(11):2363-71.
6. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003 Jul 28;163(14):1711-7.
7. Raja AS, Ip IK, Prevedello LM, Sodickson AD, Farkas C, Zane RD, Hanson R,Goldhaber SZ, Gill RR, Khorasani R. Effect of computerized clinical decision support on the use and yield of CT pulmonary angiography in the emergency department. Radiology. 2012 Feb;262(2):468-74. doi: 10.1148/radiol.11110951. Epub 2011 Dec 20. PMID: 22187633; PMCID: PMC3267081.
8. Attia NM, Seifeldein GS, Hasan AA, Hasan A. Evaluation of acute pulmonary embolism by sixty-four slice multidetector CT angiography: Correlation between obstruction index, right ventricular dysfunction and clinical presentation. The Egyptian Journal of Radiology and Nuclear Medicine. 2015. 46, 25–32.
9. Ooi MWX, Rajai A, Patel R, Gerova N, Godhamgaonkar V, Liong SY. Pulmonary thromboembolic disease in COVID-19 patients on CT pulmonary angiography - Prevalence, pattern of disease and relationship to D-dimer. Eur J Radiol. 2020 Nov;132:109336. doi: 10.1016/j.ejrad.2020.109336. Epub 2020 Oct 6. PMID:33069 986; PMCID: PMC7537636.
10. Nduta DW. The pattern of findings on multi-detector computed tomographic pulmonary angiography for suspected pulmonary embolism in Nairobi. Thesis submitted at University of Nairobi. 2013.
11. Burkill GJ, Bell JR, Chinn RJ, Healy JC, Costello C, Acton L, Padley SP. The use of a D-dimer assay in patients undergoing CT pulmonary angiography for suspected pulmonary embolus. Journal of Clinical radiology. 2002;57(1):41-6.
12. Qanadli SD,El Hajjam M, Vieillard-Baron A, Joseph T,Mesurolle B, Oliva VL,Barre O,Bruckert F, Dubourg O, Lacombe P. New CT index to quantify arterial obstruction in pulmonary embolisam: comparison with angiographic index and echocardiography. American
13. Jarman AF, Mumma BE, Singh KS, Nowadly CD, Maughan BC. Crucial considerations: Sex differences in the epidemiology, diagnosis, treatment, and outcomes of acute pulmonary embolism in non-pregnant adult patients. J Am Coll Emerg Physicians Open. 2021 Jan 27;2(1):e12378.
14. Anderson R.D., Pepine C.J. Gender differences in the treatment for acute myocardial infarction: Biasor biology?Circulation.2007;115:823–826.doi:10.1161/ CIRCULATIONAHA.106.685859.
15. Gialeraki A., Valsami S., Pittaras T., Panayiotakopoulos G., Politou M. Oral Contraceptives and HRTRiskofThrombosis. Clin.Appl.Thromb.Hemost.2018;24:217–225. doi: 10.1177/1076029616683802.
16. Rodrigues B, Correia H, Figueiredo A, Delgado A, Moreira D, dos Santos LF, Correia E, Pipa J, Beirão I, Santos O. Clot burden score in the evaluation of 85 right ventricular dysfunction in acute pulmonary embolism: quantifying the cause and clarifying the consequences. Revista Portuguesa de Cardiologia (English Edition). 2012 Nov 30;31(11):687-95.
17. Ahmad DS, Esmadi M, Todd A, Kavanagh K, Ahsan H. Incidental vascular findings on CT pulmonary angiography (CTPA). The Egyptian Journal of Radiology and Nuclear Medicine. 2013;44(2):175-82.
18. Kang DK, Thilo C, Schoepf UJ, Barraza JM, Nance JW, Bastarrika G, Abro JA, Ravenel JG, Costello P, Goldhaber SZ. CT signs of right ventricular dysfunction: prognostic role in acute pulmonary embolism. JACC: Cardiovascular Imaging. 2011;4(8):841-9
19. Goldhaber S. Deep venous thrombosis and pulmonary thromboembolism. In: Fauci A, Barunwald E, Kasper DL, Hauser SL, editors. Harrison's principles of internal medicine. New York, Chicago, San Francisco: Mac Graw Hill Medical; 2008.
20. Agnelli G, Becattini C. Acute pulmonary embolism. N Engl J Med 2010 Jul 15;363(3):266–74.
21. Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, Hull RD, Leeper KV Jr, Sostman HD, Tapson VF, Buckley JD, Gottschalk A, Goodman LR, Wakefied TW, Woodard PK. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007 Oct;120(10):871-9. doi: 10.1016/ j.amjmed. 2007. 03.024. PMID: 17904458;PMCID: PMC2071924.
22. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, Gibbs JS, Huisman MV, Humbert M, Kucher N, Lang I, Lankeit M, Lekakis J, Maack C, Mayer E, Meneveau N, Perrier A, Pruszczyk P, Rasmussen LH, Schindler TH, Svitil P, Vonk Noordegraaf A, Zamorano JL, Zompatori M; Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014 Nov 14;35(43):3033-69, 3069a-3069k. doi: 10.1093/ eurheartj/ehu283. Epub 2014 Aug
29. Erratum in: Eur Heart J. 2015 Oct 14;36(39):2666. doi: 10.1093/eurheartj/ehv131. Erratum in: Eur Heart J. 2015 Oct 14;36(39):2642. doi: 10.1093/eurheartj/ehu479. PMID: 25173341.
23. De Miguel-Diez J., Albadalejo-Vicente R., Lopez de Andres A., Hernández-Barrera V., Jiménez D., Monreal M., et al. Changing trends in hospital admissions for pulmonary embolism in Spain from 2001 to 2018. J Clin Med. 2020;9:3221.
24. Keller K, Hobohm L, Ebner M, Kresoja KP, Münzel T, Konstantinides SV, Lankeit M. Trends in thrombolytic treatment and outcomes of acute pulmonary embolism in Germany. Eur Heart J. 2020 Jan 21;41(4):522-529.
25. Ghanima W, Abdelnoor M, Mowinckel MC, Sandset PM. The performance of STA-Liatest D- dimer assay in out-patients with suspected pulmonary embolism. Br J Haematol 2006;132(2):210–5.
26. Galle C, Papazyan JP, Miron MJ, Slosman D, Bounameaux H, Perrier A. Prediction of pulmonary embolism extent by clinical findings, D-dimer level and deep vein thrombosis shown by ultrasound. Thromb Haemost 2001;86(5):1156–60.
27. Jiménez D, Uresandi F, Otero R, Lobo JL, Monreal M, Martí D, Zamora J, Muriel A, Aujesky D, Yusen RD. Troponin-based risk stratification of patients with acute non massive pulmonary embolism:systematicreviewandmetaanalysis.Chest.2 0 0 9 Oct;136(4):974-982.doi:10.1378/chest.09-0608. Epub 2009 May 22. PMID: 19465511.
28. Crawford F, Andras A, Welch K, Sheares K, Keeling D, Chappell FM. D-dimer test for excluding the diagnosis of pulmonary embolism. Cochrane Database Syst Rev. 2016:CD010864.
29. Adam SS, Key NS, Greenberg CS. D-dimer antigen: current concepts and future prospects. Blood. 2009;113(13):2878–2887.
30. Lippi G, Cervellin G, Franchini M, Favaloro EJ. Biochemical markers for the diagnosis of venousthromboembolism:thepast,presentandfuture. JThromb Thrombolysis. 2010; 30(4):459–471.
31. Hajsadeghi S, Kerman SR, Khojandi M, Vaferi H, Ramezani R, Jourshari NM, Mousavi SA, Pouraliakbar H. Accuracy of D-dimer:fibrinogen ratio to diagnose pulmonary thromboembolism in patients admitted to intensive care units. Cardiovasc J Afr. 2012 Sep;23(8):446-56. doi: 10.5830/CVJA-2012-041. PMID: 23044500; PMCID: PMC372 1944.
32. Roongsritong C, Warraich I, Bradley C.Common causes of troponin elevations in the absence of acute myocardial infarction: incidence and clinical significance. Chest 2004;125:1877-84
33. Sheehan P, Vasikaran SD. The evolving clinical role of cardiac troponins and new acute myocardial infarction guidelines: Implications for the clinical laboratory. Clin Biochemist Rev 2001;23:52-65
34. Daniels LB, Maisel AS. Natriuretic peptides. Journal of the American college of cardiology. 2007;50(25):2357–68.
35. Hutchinson KR, Guggilam A, Cismowski MJ, Galantowicz ML, West TA, Stewart JA Jr, Zhang X, Lord KC, Lucchesi PA. Temporal pattern of left ventricular structural and functional remodeling following reversal of volume overload heart failure. J Appl Physiol (1985). 2011 Dec;111(6):1778-88.
36. Kucher N, Printzen G, Goldhaber SZ. Prognostic role of brain natriuretic peptide in acute pulmonary embolism. Circulation. 2003;107(20):2545–7.
37. Lega J-C, Lacasse Y, Lakhal L, Provencher S. Natriuretic peptides and troponins in pulmonary embolism: a meta-analysis. Thorax. 2009;64(10):869–75.
38. Lankeit M, Jiménez D, Kostrubiec M, Dellas C, Hasenfuss G, Pruszczyk P, Konstantinides S. Predictive value of the high-sensitivity troponin T assay and the simplified Pulmonary Embolism Severity Index in hemodynamically stable patients with acute pulmonary embolism: a prospective validation study. Circulation. 2011 Dec13;124(24):2716-24. doi: 10.1161/CIRCULATIONAHA.111.051177. Epub 2011 Nov 14.PMID: 22082681.
39. Kilinc G, Dogan OT, Berk S, Epozturk K, Ozsahin SL, Akkurt I. Significance of serum cardiac troponin I levels in pulmonary embolism. J Thorac Dis. 2012 Dec;4(6):588-93. doi: 10.3978/j.issn.2072-1439.2012.10.13. PMID: 23205283; PMCID: PMC3506790.
40. Khaladkar SM, Thakkar DK, Shinde K, Thakkar DK, Shrotri H, Kulkarni VM. Deep vein thrombosis of the lower limbs: A retrospective analysis of Doppler ultrasound findings. Med J DY Patil Univ. 2014;7:612–9.
41. Ismail A, Tabari AM, Isyaku K. Doppler sonographic evaluation of venogenic extremity swellings: Analysis of 170 patients from Kano, Nigeria. Niger J Clin Pract. 2017;20:930–5.
42. Agunloye MA, Akinmoladun JA, Ogbole GI, Akingbola T, Adeyinka AO. The role of Doppler ultrasound in clinically suspected deep vein thrombosis of the lower limb in black African population. West Afr J Ultrasound. 2010;11:110.
43. Salaam AJ, Danjem SM, Ibinaiye PO, Ama JT, Ekedigwe JE, Igoh EO, Ukaonu GO, Salaam AA. Lower limbs Doppler vascular findings in Jos university teaching hospital.
44. Elliott CG, Goldhaber SZ, Visani L, DeRosa M. Chest radiographs in acute pulmonary embolism. Results from the International Cooperative Pulmonary Embolism Registry. Chest. 2000 Jul;118(1):33-8.
45. Greenspan RH, Ravin CE, Polansky SM, McLoud TC. Accuracy of the chest radiograph in diagnosis of pulmonary embolism. Invest Radiol. 1982 Nov-Dec;17(6):539-43. doi: 10.1097/00004424-198211000-00003. PMID: 7152856.
46. Miller GA, Sutton GC, Kerr IH, Gibson RV, Honey M. Comparison of streptokinase and heparin in treatment of isolated acute massive pulmonary embolism. Br Med J. 1971 Jun 19;2(5763):681-4. doi: 10.1136/bmj.2.5763.681. PMID: 5556052; PMCID: PMC179 6248.