Al-Azhar Med. J. *Vol. 44(4), October, 2015, 373-384*
ORG. No 82291 ***DOI: 10.12816/0023135***

EFFECTIVENESS OF THROMBOLYTIC THERAPY FOR MASSIVE PULMONARY EMBOLISM

By

Mousa Elshamly*; Ahmad Shawky Sherif; Mohamed O. Nour*****

*Department of Chest Diseases, Faculty of Medicine, Al-Azhar University (Cairo)
**Department of Cardiology, Faculty of Medicine, Zagazig University
*** Department of Community & Occupational Medicine, Faculty of Medicine, Al-Azhar University (Damietta branch)

ABSTRACT

Background: Pulmonary embolism (PE) is a common disorder that is associated with significant morbidity and mortality. The primary cause of death in fatal PE is right-sided heart failure. Several randomized, controlled trials comparing thrombolytic therapy with heparin in patients with an acute PE have demonstrated more rapid clot resolution in those treated with thrombolysis.

Objective: Study the effectiveness and complications of thrombolytic therapy for massive pulmonary embolism.

Patients and Methods: Sixty patients with objectively confirmed massive pulmonary embolism were divided into two equal groups. First group with no contraindication for thrombolytic therapy was treated by (streptokinase or recombinant tissue plasminogen activator) and second group with absolute contraindication for thrombolytic therapy was treated by unfractionated heparin (UFH).

Results: CT pulmonary angiography showed that main pulmonary artery embolism was 25%, bilateral saddle shape pulmonary embolism 41.6%, left pulmonary artery embolism 15% and right pulmonary artery embolism 11.6% , and CT was not done in 4 patients because they were unstable. Mortality was high in patients with main pulmonary embolism 13.3%. Mortality was 20% in thrombolytic therapy treated group compared to 50% in heparin treated group and result was statistically significant . Moderate bleeding occurred into left knee in one patient of thrombolytic therapy treated group and mild Hematemesis occurred in one patient of heparin treated group but no mortality occurred due to bleeding.

Conclusion: Thrombolytic treatment reduced overall mortality of patients with acute massive PE, and was not associated with major bleeding.

INTRODUCTION

Pulmonary embolism (PE) is a common disorder that is associated with significant morbidity and mortality. The primary cause of death in fatal PE is right-sided heart failure. The most serious long-term complication of PE is pulmonary hypertension **(Almoosa, 2002).** In 2000, the European Society of Cardiology (ESC) developed a guideline to characterize PE by disease burden, classifying patients into ‘‘massive’’ and ‘‘nonmassive’ PE. The society de?ned massive PE as PE with shock or hypotension, (hypotension de?ned as a systolic blood pressure (SBP) of less than 90 mm Hg or a drop of 40 mm Hg for more than 15 minutes not caused by new-onset

arrhythmia, hypovolemia, or sepsis). They further subdivided the group that did not meet criteria for massive PE into submassive PE and nonmassive PE. Submassive PE was de?ned as acute PE with evidence of right ventricular (RV) strain without evidence of shock, whereas emboli with no shock or evidence of RV strain were considered nonmassive. The society hypothesized that hemodynamic consequences of PE are directly related to the size and number of PE **(Vyas and Donato, 2012).**

In 2008, the ESC reconvened and proposed an update and reclassi?cation of their guidelines. They proposed the terms ‘‘high risk,’’ ‘‘intermediate risk,’’ and ‘‘low risk’’ to replace massive, submassive, and nonmassive, and emphasized that the prognosis of PE depends on hemodynamic instability caused by recurrent embolization and deterioration of RV function in the first 24 to 48 hours rather than the amount of pulmonary artery obstruction **(Konstantinides et al., 2014).** Pulmonary embolism (PE) is a common disorder with significant morbidity and mortality. In the United States, PE occurs in approximately 600,000 patients and may be responsible for over 50,000 deaths annually **(Arcasoy & Kreit, 1999 and Goldhaber, 1998)**. Several randomized, controlled trials comparing thrombolytic therapy with heparin in patients with an acute PE have demonstrated more rapid clot resolution in those treated with thrombolysis **(Ly, 1978).**

The present study aimed to study the effectiveness and complications of thrombolytic therapy in massive acute pulmonary embolism.

PATIENTS AND METHODS

After approval of local ethical committee, 60 patients with objectively confirmed symptomatic acute massive pulmonary embolism admitted to intensive care unit of King Saud Hospital, Saudi Arabia from January 2008 to June 2015 were divided into two equal groups: first group (14 males and 16 females with age ranged from 29 to 65 years old) with no contraindication for thrombolytic therapy was treated by (streptokinase or recombinant tissue plasminogen activator) and second group (10 males and 20 females with age ranged from 30 to 70 years old) with absolute contraindication for thrombolytic therapy was treated by unfractionated heparin (UFH).

Any patient presented by symptoms and sign suggestive of pulmonary embolism such as chest pain, dyspnea, tachypnea, hemoptysis, diaphoresis, syncope or cardiac arrest were exposed to:

1- Full history taken and physical examination.
2- Chest X ray.
3- Arterial blood gases analysis.
4- Electrocardiography (ECG).
5- Echocardiography.
6- Computerized tomography pulmonary angiography.
7- Complete blood picture.
8- Kidneys and liver functions.
9- Serum electrolytes.

10- Duplex both lower limbs.

Fibrinolytic dosing regimens in PE (Brady, 2011):

1- Streptokinase, 250,000 units over 30 min, then 100,000 units/h over 24 hours.
2- Alteplase, 15 mg IV bolus followed by 85 mg IV infusion over 2 hours.

Heparin:

If IV UFH is chosen, an initial bolus of 80 U/kg or 5000 U followed by an infusion of 18 U/kg/h or 1300 U/h should be given, with the goal of rapidly achieving and maintaining the activated partial thromboplastin time(a PTT) at levels that correspond to therapeutic heparin levels **(Guyatt et al., 2012).**

Exclusion criteria for thrombolytic therapy (Vyas and Donato, 2012).

History of intracranial hemorrhage.
Known intracranial neoplasm.
Ischemic stroke in last 3 months.
Aortic dissection .
History of head or facial trauma within 3 months.
Active bleeding/bleeding diathesis.

Criteria of right (RV) ventricular dysfunction by echocardiography

The main criteria, used in the above mentioned studies to define RV dysfunction, were qualitative criteria including mild, moderate, or severe RV hypokinesis and quantitative criteria including RV dilatation with a RV/LV end-diastolic diameter > 1, a RV end-diastolic diameter > 30 mm, and paradoxical septal systolic motion, pulmonary hypertension with a pulmonary artery systolic pressure > 30 mm Hg and a tricuspid regurgitant velocity > 2.8 m/s, and absence of RV wall hypertrophy (free wall thickness > 7 mm) (**Sekhri et al., 2012).**

Statistical Analysis of data

Statistical analysis was carried out using the SPSS computer package version 17.0 (SPSS Inc., Chicago, IL, USA). The collected data were statistically managed as follows:

- For descriptive statistics: The mean ± SD were used for quantitative variables, while the number and percentage were used for qualitative variables.
- For analytic statistics: Chi-square test was used to assess the differences in frequency of qualitative variables, while Fischer's exact test (FET) was applied if sample size is small.
- In order to assess the differences in means of quantitative variables between both groups, independent samples t-test was applied.

The statistical methods were verified, assuming a significant level was $P < 0.05$.

RESULTS

There was no statistically significant difference between the two studied groups as regard age and predisposing factors (Table 1).

Table (1): Comparison between thrombolytic therapy- treated group and heparin- treated group according to demographic data and risk factors.

Groups / Parameters	Thrombolytic therapy treated group (n = 30)	Heparin therapy treated group (n = 30)	Total (n = 60)	P-value
Age/years				
Mean ± SD	49.6 ± 11.2	54.8 ± 10.5		0.069 [1]
Min – Max	29 – 65	30 – 70		
Gender				
Male	14 (46.7%)	10 (33.3%)	24(40.0%)	0.192 [2]
Female	16 (53.3%)	20 (66.7%)	36(60.0%)	
Risk factors				
Vasculitis	1 (3.3%)	1 (3.3%)	2 (3.3%)	
DVT	4 (13.3%)	3 (10.0%)	7 (11.6%)	
Post operative	4 (13.3%)	4 (13.3%)	8 (13.3%)	
Bone fracture	8 (26.7%)	2 (6.7%)	10(16.6%)	
Bedridden	7 (23.3%)	11 (36.7%)	18(30.0%)	0.118 [3]
Right atrial thrombus	1 (3.3%)	0 (0.0%)	1 (1.6%)	
Malignancy	1 (3.3%)	7 (23.3%)	8 (13.3%)	
Postpartum	3 (10.0%)	0 (0.0%)	3 (5.0%)	
Pregnant	0 (0.0%)	1 (3.3%)	1 (1.6%)	
Obese	1 (3.3%)	1 (3.3%)	2 (3.3%)	

[1.] Independent Samples t-test.
[2.] Fisher's Exact test.
[3.] Chi- Square test.

The main clinical presentation was acute dyspnea 96.6%, palpitation 88.3%, chest pain 71.6%, hypoxemia 65% and cardiac arrest 3.3% , but there was no statistically significant difference between both groups (Table 2).

Table (2): Clinical presentation of both groups.

Groups / Parameters	Thrombolytic therapy treated group n = 30	Heparin therapy treated group n = 30	Total n = 60	P-value [1]
Palpitation (heart rate more 100 beats/min)	26 (86.7%)	27 (90.0%)	53 (88.3%)	1.000
Acute dyspnea	28 (93.3%)	30 (100.0%)	58 (96.6%)	0.492
SPO2 less than 90%	19(63.3%)	20(66.6%)	39 (65.0%)	1.000
Chest pain	21 (70.0%)	22 (73.3%)	43 (71.6%)	1.000
Cardiac arrest Cardiopulmonary resuscitation	2 (6.7%)	0 (0.0%)	2 (3.3%)	0.492

[1] : Fisher's Exact test.
SPO2 = arterial oxygen saturation.

The main ECG findings were sinus tachycardia in 88.3%, S1Q3T3 pattern in 15%, incomplete right bundle branch block in 5%, complete right bundle branch block in 3.3% and a systole in 3.3%. The difference between both groups was statistically non significant (Table3).

Table (3): Electrocardiographic finding (ECG) in both groups.

Groups / Parameters	Thrombolytic therapy treated group (n = 30)	Heparin therapy treated group (n = 30)	Total (n = 60)	P-value [1]
Sinus tachycardia (> 100 beats/min)	21 (70.0%)	23 (76.6%)	44 (73.3%)	1.000
Incomplete right bundle branch block	1 (3.3%)	2 (6.7%)	3 (5.0%)	1.000
Complete right bundle branch block	1 (3.3%)	1 (3.3%)	2 (3.3%)	1.000
S1Q3T3 pattern	5 (16.7%)	4 (13.3%)	9 (15%)	1.000
A systole	2 (6.7%)	0 (0.0%)	2 (3.3%)	0.492

[1]: Fisher's Exact test.

Normal chest X ray was found in 23.3%, right mid-zone segmental atelectasis in 15%, left mid-zone segmental atelectasis in 15%, prominent central pulmonary artery in 21,6%, right side pleural effusion in 11.6%, left side pleural effusion in 8.3%, right lower lobe consolidation in 3.3%, and left lower lobe consolidation in 1.6%. The difference between both groups was statistically non significant (Table 4).

Table (4): Chest x ray findings in both groups.

Groups / Parameters	Thrombolytic therapy treated group (n = 30)	Heparin therapy treated group (n = 30)	Total (n = 60)	P-value[1]
Normal chest x ray	8 (26.7%)	6 (20.0)	14 (23.3%)	0.788
Right mid-zone segmental atelectasis	5 (16.7%)	4 (13.3%)	9 (15.0%)	
Left mid-zone segmental atelectasis	4 (13.3%)	5 (16.7%)	9 (15.0%)	
Prominent central pulmonary artery	7 (23.3%)	6 (20.0%)	13 (21.6%)	
Right side Pleural effusion	3 (10.0%)	4 (13.3%)	7 (11.6%)	
Left side pleural effusion	3 (10.0%)	2 (6.7%)	5 (8.3%)	
Right lower lobe consolidation	0 (0.0%)	2 (6.7%)	2 (3.3%)	
Left lower lobe consolidation	0 (0.0%)	1 (3.3%)	1 (1.6%)	

[1]: Chi- Square test.

Echocardiography showed right ventricular dilatation in 55% and right ventricular dilatation with hypokinesia and tricuspid regurge in 45%, mortality was significantly high in patients with right ventricular dilation and tricuspid regurge. There was no statistically differences between both groups (Table 5).

Table (5): Echocardiography findings in both groups.

Groups / Parameters	Thrombolytic therapy treated group (n = 30)	Heparin therapy treated group (n = 30)	Total (n = 60)	P-value [1]
Right ventricular dilatation	19 (63.3%)	14 (46.7%)	33(55.0%)	
Died	2 (6.7%)	2 (6.7%)	4 (6.6%)	0.100
Survived	17 (56.7%)	12 (40.0%)	29 (48.3%)	
Right ventricular dilatation with hypokinesia & Tricuspid regurge	11 (36.7%)	16 (53.3%)	27(45.0%)	0.040 *
Died	4 (13.3%)	13 (43.3%)	17(28.3%)	
Survived	7 (23.3%)	3 (10.0%)	10 (16.6%)	

[1]: Fisher's Exact test. *: Significant.

CT pulmonary angiography showed that main pulmonary artery embolism was 25%, bilateral saddle shape pulmonary embolism was 41.6%, left pulmonary artery embolism 15%, right pulmonary artery11.7%, and CT was not done in 4 patients (6.6%) because they were unstable. Bilateral saddle shape pulmonary embolism was more common than other types and result was statistically significant (Table 6).

Table (6): Computed tomography (CT) pulmonary angiography findings in both groups.

Groups / Parameters	Thrombolytic therapy treated group (n = 30)	Heparin therapy treated group (n = 30)	Total (n = 60)	P value
Main pulmonary artery embolism	5 (16.7%)	10 (33.3%)	15 (25.0%)	
Died	2 (6.7%%)	6 (20.0%)	8 (13.3%)	0.608
Survived	3 (10.0%%)	4 (13.3.0%)	7 (11.7%)	
Bilateral saddle shape pulmonary embolism	17 (56.7%)	8 (26.7%)	25 (41.6%)	
Died	2 (6.7%)	5 (16.7%)	7 (11.7%)	0.017 *
Survived	15 (50.0%)	3 (10.0%)	18 (30.0%)	
Left pulmonary artery embolism	4 (13.3%)	5 (16.7%)	9 (15.0%)	
Died	0 (0.0%)	1 (3.3%)	1 (1.6%)	1.000
Survived	4 (13.3%)	4 (13.3%)	8 (13.3%)	
Right pulmonary artery embolism	2 (6.7%)	5 (16.7%)	7 (11.7%)	
Died	0 (0.0%)	1 (3.3%)	1 (1.6%)	1.000
Survived	2 (6.7%)	4 (13.4%)	6 (10.0%)	
CT not done	2 (6.7%)	2 (6.7%)	4 (6.66%)	
Died	2 (6.7%)	2 (6.7%)	4(6.6%)	1.00
Survived	0 (0.0%)	0 (0.0%)	0 (0.0%)	

: Fisher's Exact test. *: Significant.

Mortality was 20% in thrombolytic treated group compared to 50% in heparin treated group and result was statistically significant (Table 7).

Table (7): Comparison between both groups regarding mortality.

Groups / Mortality rate	Thrombolytic therapy treated group n = 30 (%)	Heparin therapy treated group n = 30 (%)	P-value [1]
Died	6 (20.0)	15 (50.0)	0.029 *
Survived	24 (80.0)	15 (50.0)	

[1]: Fisher's Exact test. *: Significant.

Moderate bleeding occurred into left knee joint in one patient of thrombolytic therapy treated group and mild Hematemesis in one patient of heparin treated group but no mortality occurred due to bleeding, the difference was statistically non significant (Table 8).

Table (8): Comparison between both groups regarding complications.

Groups / Parameters	Thrombolytic therapy treated group no. = 30	Heparin therapy treated group no. = 30	P-value [1]
Bleeding into left knee joint	1 (3.3%)	0 (0.0%)	0.368
Hematemesis	0 (0.0%)	1 (3.3%)	
No complication	29 (96.7%)	29 (96.7%)	

[1]: Chi- Square test.

CT pulmonary angiography findings of pulmonary embolism:

Figure (1): CT pulmonary angiography showed large filling defect(pulmonary embolism) in the left pulmonary artery (arrow) in 42 years old male patient with fracture tibia presenting by sudden loss of consciousness and cardiogenic shock .

Figure (2): Bilateral pulmonary embolism (arrows) in male patient 38 years old with traumatic fracture pelvic presenting by shortness of breath.

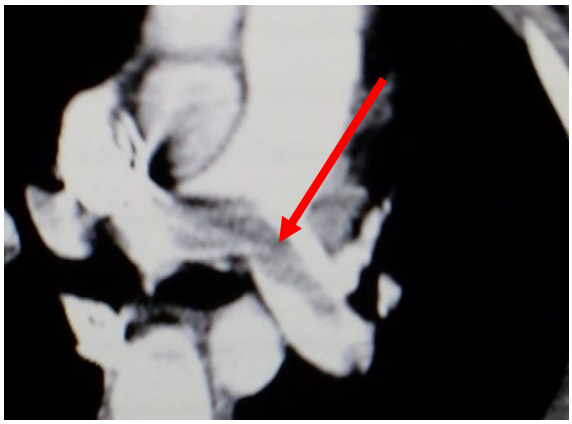

Figure (3): Saddle shape pulmonary embolism (arrow) in post partum female patient 28 years old presenting by chest pain and shortness of breath.

DISCUSSION

Ten percent of all of the diagnosed cases of PE meet the de?nitions of high risk or massive PE. Short-term mortality associated with massive/high-risk PE in untreated patients is as high as 60% **(Wood, 2002).** Specific symptoms and signs are not helpful diagnostically because their frequency is similar among patients with and without PE. In the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II), the following frequencies of symptoms and signs were noted among patients with PE who did not have preexisting cardio-pulmonary disease. The most common symptoms were dyspnea at rest or with exertion (73 %), pleuritic chest pain (44%), cough (34%), orthopnea (28%), calf or thigh swelling (41%), and wheezing (21%) **(Stein et al., 2007).**

In this study, the main clinical presentations were dyspnea (96.6%), palpitation (88.3%), chest pain (71.6%), hypoxemia (65%) and cardiac arrest (3.3%). The most common ECG finding was sinus tachycardia (73.3%), S1Q3T3 pattern (15%), incomplete right bundle branch block (5%), complete right bundle branch block (3.3%) and a systole (3.3%). In a study conducted by **Geibel et al., (2005),** they found that right ventricular strain was diagnosed in the presence of one or more of the following ECG findings: complete or incomplete right ventricular branch block, S1Q3T3, and negative T wave in V1-V4. In a study conducted by **Rodger et al., (2000**) they found that the typical S1Q3T3 sign is usually not seen on most ECG's in patients with PE. Characteristically, most of the patients had a tachycardia, signs of right axis deviation with an incomplete right bundle branch block pattern.

In our study ,chest X rays was normal in 23.3%, right mid zone segmental atelectasis in 15%, left mid zone segmental atelectasis in 15%, prominent central pulmonary artery in 21.6%, right side pleural effusion in 11.6%, left side pleural effusion in 8.3%, right lower lobe consolidation in 3.3% and left lower lobe consolidation in 1.6%, this is not specific finding with no significant difference between both groups and this has been stated to be the case in the study of **Piazza and Goldhaber (2006).** The characteristic features usually quoted include focal oligaemia (Westermark sign); a peripheral

wedge-shaped opacity and an enlarged descending right pulmonary artery (Hampton's hump) are usually rare and not readily seen **(Piazza and Goldhaber, 2006) .**

In our study, echocardiography showed right ventricular dilation in 55% and right ventricular dilatation with hypokinesia and tricuspid regurge in 45%. Mortality was significantly high in patients with right ventricular dilatation with hypokinesia and tricuspid regurge which indicate more right ventricular dysfunction. In a study conducted by **Ronny et al. (2012)**, they found that the patients with massive pulmonary embolism have right ventricular enlargement, right ventricular dysfunction and more than 90% have right ventricular hypokinesi **.**

In our study, CT pulmonary angiography showed bilateral saddle shape pulmonary embolism in 41.6% with 11.7% mortality. Main pulmonary artery embolism was present in 25% with 13.3% mortality. Left pulmonary artery embolism was in 15% with 1.6% mortality. Right pulmonary artery embolism was in 11.7% with 1.6% mortality and CT pulmonary angiography was not done in 4 patients due to hemodynamic instability and the four patients died.

In this study, mortality was significantly low 20% in thrombolytic therapy- treated group compared to (50%) in heparin- treated group. There were two patients presenting by history of sudden loss of conscious and on arrival to the hospital they were in cardiac arrest, first case male patient 42 years old with history of fracture left tibia, gypsum splint was done and patient discharged from the hospital after 10 days cardiopulmonary resuscitation (CPR) for 45 minutes and streptokinase was given, patient recover and shifted to intensive care unit ,he developed sign of brain hypoxia ,but after 10 days became fully conscious and discharged in very good condition. Second patient 58 years female with history of left knee replacement and she was discharged from the hospital after one week , on arrival she was in shock and developed cardiac arrest CPR done and she revived after few minutes. Streptokinase and hemodynamic support was given . In both patients diagnosis of pulmonary embolism was confirmed by CT pulmonary angiography.

In our study, bleeding complication occurred only in one patient of thrombolytic- therapy treated group in form of left knee joint bleeding which responded to palliative management, and mild hematemesis occurred in one patient of heparin- treated group. Differences in bleeding between patients who received heparin alone and patients who received heparin plus thrombolytic therapy were not significant (**Fasullo et al., 2010).**

Patients treated with a fibrinolytic agent have faster restoration of lung perfusion at 24 hour (30% to 35% reduction in total perfusion defect), while patients treated with heparin have no substantial improvement in pulmonary blood flow **(Michael et al., 2011).** In an overview of 5 randomized controlled trials that included patients with massive PE, thrombolytic therapy has been shown to reduce the risk of death or recurrent PE by 55% **(Wan et al., 2004)** . Only one trial of 8 patients with cardiogenic shock due to acute PE showed that the 4 patients treated with

streptokinase survived,while the 4 patients treated with heparin alone died **(Jerjes et al., 1995).** All case fatality attributable to pulmonary embolism are lower in unstable patients with pulmonary embolism who received thrombolytic therapy than in those who did not receive thrombolytic therapy **(Stien and Matta, 2012)**.

Agnelli et al. (2002) found a lower death rate and recurrence of pulmonary embolism with thrombolytic therapy than with heparin therapy alone. Thrombolytic therapy has been shown to reduce the risk of death or recurrent PE by 55% **(Wan et al., 2004)**. Thrombolytic therapy causes rapid lysis of clot and more rapid improvement in RV hemodynamics. Thrombolytic therapy is recommended as standard, first-line treatment in patients with massive PE, unless contraindicated **(Kearon et al., 2008)** .

The bleeding risk of thrombolytics has to be wisely evaluated against the compulsion of the indication and risk of death rather than just excluding thrombolytics in the presence of a major contraindication **(Kearon et al ., 2008).**

CONCLUSION

Thrombolytic treatment reduced overall mortality of patients with acute massive PE and not associated with major bleeding.

REFERENCES

1. **Agnelli G, Becattini C and Kirschstein T. (2002):** Thrombolysis vs heparin in the treatment of pulmonary embolism: a clinical outcome–based meta-analysis. *Arch Intern Med*, 162: 2537–2541.

2. **Almoosa K. (2002):** Is thrombolytic therapy effective for pulmonary embolism? Am Fam Physician , 65(6):1097–1102.

3. **Arcasoy SM and Kreit JW. (1999):** Thrombolytic therapy of pulmonary embolism: a comprehensive review of current evidence. Chest, 115: 1695–707.

4. **Brady WJ. (2011):** Fibrinolytic therapy in pulmonary embolism. Emergencias , 23:319–323.

5. **Fasullo S, Scalzo S, Maringhini G, Ganci F, Cnnizzaro S, Terrazzino G and Dipasquale P. (2011):** Six-month echocardiographic study in patients with submassive pulmonary embolism and right ventricle dysfunction: comparison of thrombolysis with heparin.Am J Med Sci, 341 (1):33-39.

6. **Geibel A, Zehender M, Kasper W, Olschewski M, klima C and Konstantinides S. (2005):** Prognostic value of the ECG on admission in patients with acute major pulmonary embolism. Eur Respir J, 25(5):843–848.

7. **Goldhaber SZ. (1998):** Pulmonary embolism. N Engl J Med , 339:93– 104.

8. **Guyatt GH, Akl EA, Crowther M, Gutterman DD and Schuünemann HJ. (2012):** Executive Summary: Antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest , 141 (2 Suppl):7S–47S.

9. **Jerjes-Sanchez C, Ramirez-Rivera A, de Lourdes Garcia M, Arriaga-Nava R, Valencia S, Rosado-Buzzo A, Pierzo JA, and Rosas E. (1995):** Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomized controlled trial. J Thromb Thrombolysis, 2(3):227–229.

10. **Kearon C, Kahn SR, Agnelli G, Agnelli G, Goldhaber S, Raskob GE and Comerota AJ. (2008):** American College of Chest Physicians. Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th edition). Chest, 133:454S-545S.

11. **Konstantinides S, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, Huisman M, Humbert M, Kucher N, Lang I and Lankeit M. (2014):** ESC Guidelines on the

diagnosis and management of acute pulmonary embolism: The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). European Heart Journal , 35:3033–3080.

12. **Ly B, Arnesen H, Eie H and Hol R. (1978):** A controlled clinical trial of streptokinase and heparin in the treatment of major pulmonary embolism. Acta Med Scand , 203(6):465–470.

13. **Michael R, McMurtry MS, Stephen L, Arche SL, Mary C, Neil G, Samuel Z, Goldhaber J, Stephen J, Jeffrey A, Andrew D, Michaels P and James W. (2011):** Management of Massive and Submassive Pulmonary Embolism, Iliofemoral Deep Vein Thrombosis, and Chronic Thromboembolic Pulmonary Hypertension: a scientific statement from the American Heart Association. Circulation , 123(16):1788–1830.

14. **Piazza G and Goldhaber SZ. (2006):** Acute Pulmonary Embolism Part I: Epidemiology and Diagnosis, Circulation ; 114(2):e28–32.

15. **Rodger M, Makropoulos D, Turek M, Quevillon J, Raymond F, Rasuli P and Wells PS. (2000)**: Diagnostic value of the electrocardiogram in suspected pulmonary embolism. Am J Cardiol., 86(7):807–809.

16. **Ronny C, Pablo L, Victor N and Brooks M (2012):** Echocardiographic findings in pulmonary embolism: An important guide for the management of the patientWorld Journal of Cardiovascular Diseases, 2: 161-164.

17. **Sekhri V, Mehta N, Rawat N, Lehrman SG and Aronow WS. (2012):** Management of massive and nonmassive pulmonary embolism. Arch Med Sci, 8(6):957–969.

18. **Stein PD, Beemath A and Matta F. (2007):** Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med, 120(10):871-879.

19. **Stein PD and Matta F. (2012):** Thrombolytic Therapy in Unstable Patients with Acute Pulmonary Embolism: Saves Lives but Underused. Am J Med, 125(5):465–470.

20. **Vyas PA and Donato AA. (2012):** Thrombolysis in acute pulmonary thrombo-embolism. Southern Medical Journal, 105(10):560–570.

21. **Wan S, Quinlan DJ, Agnelli G and Eikelboom JW. (2004):** Thrombolysis compared with heparin for the initial treatment of pulmonary embolism: a meta-analysis of the randomized controlled trials. Circulation , 110(6):744–749.

22. **Wood KE. (2002):** Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest, 121: 877-905.

[illegible]

موسي الشاملى* - أحمد شوقى - محمد أسامة نور*****

*** قسم الأمراض الصدرية بكلية طب الأزهر بنين (القاهرة)**
**** قسم أمراض القلب بكلية الطب جامعة الزقازيق**
***** قسم طب المجتمع وطب الصناعات كلية الطب جامعة الأزهر (دمياط)**

خلفية البحث : يعتبر إنسداد الشريان الرئوى الشديد من الإضطرابات الشائعة المصاحبة للكثير من حالات الإعتلال و الوفيات وقد وجد أن السبب الرئيسى للوفيات فى حالة إنسداد الشريان الرئوى القاتل هو فشل الجانب الأيمن من القلب . وفى العديد من الدراسات العشوائية التى قارنت بين إستخدام علاج التخثر التحللى والهيبارين فى علاج إنسداد الشريان الرئوى الشديد وجد زيادة سرعة تحلل التخثر فى المرضى الذين استخدموا علاج التخثر التحللى .

الهدف من الدراسة : دراسة فاعلية ومضاعفات علاج التخثر التحللى لإنسداد الشريان الرئوى الشديد.

المرضى وطرق البحث: تمت الدراسة على ستين مريضا (24 رجل و36 إمرأة) من المرضى الذين اصيبوا بانسداد الشريان الرئوى الشديد . تم تقسيم المرضى الى مجموعتين متساويتين: المجموعة الأولى تم إعطاؤها علاج التخثر التحللى ، وتم إعطاء عقار الهيبارين للمجموعة الثانية (المجموعة الأولى ليس لديهم موانع لإستخدام علاج التخثر التحللى والمجموعة الثانية لديهم موانع قاطعة لإستخدام علاج التخثر التحللى).

النتائج : وجد بالتصوير المقطعى للأوعية الرئوية أن إنسداد الشريان الرئوى الرئيسى كان يمثل 25% من عدد المرضى و إنسداد الشريان الرئوى بالجانبين على شكل سرج كان يمثل 41% من عدد المرضى وأن إنسداد الشريان الرئوى الأيسر كان يمثل 15 % من عدد المرضى وأن إنسداد الشريان الرئوى الأيمن كان يمثل 11.7% من عدد المرضى ولم يتم عمل تصوير مقطعى للأوعية الرئوية لعدد 4 من المرضى وذلك لان الحالة الصحية لهم كانت غير مستقرة .و كانت نسبة الوفيات فى المرضى المصابين بإنسداد الشريان الرئوى الرئيسى أعلى وكانت تمثل 13.3% من العدد الكلى للمرضى .و كانت نسبة الوفيات فى المرضى الذين تم إعطاؤهم عقار علاج التخثر التحللى20 % وكانت نسبة الوفيات فى المرضى الذين تم إعطاؤهم عقار الهيبارين 50% وكان الفرق ذو دلالة احصائية .وكانت مضاعفات العلاج فى المرضى الذين تم إعطاؤهم عقار علاج التخثر التحللى مساو للمضاعفات فى المرضى الذين تم إعطاؤهم عقار الهيبارين ولم تكن لها فروق إحصائية .وقد حدث نزيف متوسط فى مفصل الركبة اليسري فى مريض واحد من مرضى المجموعة الاولى وحدث قئ دموي بسيط فى مريض واحد من مرضى المجموعة الثانية ولكن لم تحدث وفيات نتيجة النزيف فى كلتا المجموعتين

الخلاصة
علاج التخثر التحللى لمرضى إنسداد الشريان الرئوى الشديد يقلل عدد الوفيات ولا يصاحب ذلك حدوث نزيف شديد.