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RESEARCH ARTICLE

Prevalence of pulmonary embolism in patients with acute exacerbation of chronic obstructive pulmonary disease.

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Abstract

Background

Patients suffering from chronic obstructive pulmonary disease (COPD) are often admitted to hospital with an exacerbation of their disease, these episodes are most often attributed to an acute exacerbation of bronchial infection but the real cause is often unknown. COPD is considered a risk factor for pulmonary embolism (PE) and PE is part of the differential diagnosis of an acute exacerbation of COPD. Our aim was to detect the prevalence of pulmonary embolism in patients of COPD presenting with acute exacerbation of their disease.

Methods

Prospective randomized, clinical trial performed in 25 patients with COPD presenting with acute exacerbation of their disease Patients were categorized into two groups (Group A) patients without multislice CT pulmonary angiography findings of pulmonary embolism.(Group B) patients with multislice CT pulmonary angiography findings of pulmonary embolism. The patients of the two groups compared for duration of hospitalization, Use of mechanical ventilation and duration of mechanical ventilation.

Results

All studied patients were evaluated by SOFA score which ranged from 2 to 4 in all patients with a mean of 2.6 ± 0.6, while in patients with pulmonary embolism it ranged from 3 to 4 with a mean of 3.3±0.6. The duration of hospital stay in all studied patients ranged from 12 to 20 days with a mean of 9.4±3.5, while the 3 patients with pulmonary embolism stayed in hospital for 12 days each. Seventy eight percent of all studied patients (n=21) were mechanically ventilated while 22% (n=6) were not. Concerning patients with pulmonary embolism (n=3), two patients were mechanically ventilated while the other patient was not.

CONCLUSIONS

This study showed an 11% prevalence of PE in patients with COPD admitted for severe exacerbation of unknown origin and complicated by respiratory failure.

Introduction

Patients with COPD have airway obstruction and commonly exhibit pulmonary dynamic hyperinflation (1-3). Recent data (4) show that COPD itself is a powerful independent risk factor for cardiovascular morbidity and mortality (4).

COPD is a major health burden worldwide. It is the fourth-leading cause of mortality, accounting for > 3 million deaths annually; and by 2020, COPD will be the third-leading cause of death, trailing only ischemic heart disease and stroke (5). Most COPD-related deaths occur during periods of exacerbation (6,7). Previous studies(6) estimate that 50 to 70% of all COPD exacerbations are precipitated by an infectious process, while 10% are due to environmental pollution. Up to 30% of exacerbations are caused by an unknown etiology (6). Exacerbations of their disease that manifests itself with an increase in cough and/or sputum, increased dyspnea and sometimes chest pain(5)

These episodes are most often attributed to an acute exacerbation of bronchial infection but the real cause is often unknown. COPD is considered a risk factor for pulmonary embolism (PE) and PE is part of the differential diagnosis of an acute exacerbation of COPD. However, the symptoms of these two conditions overlap to a considerable extent and the investigation of PE is often ignored in these patients (8).Therefore the true prevalence of this situation is unknown.

A study (9) suggests that patients with COPD have approximately twice the risk of pulmonary embolism (PE) and other venous thromboembolic events (venous thromboembolic [VTE]) than those without COPD. Since thromboembolic events can lead to cough and dyspnea (just like infectious events), PE may be another common cause of COPD exacerbations. (10)

Rarely, pulmonary embolismpresents in such a dramatic fashion that the diagnosis is intuitivelyobvious and empiric treatment will be started, but the usual presentation is sufficiently nonspecific that further testing is necessary to establish or exclude the diagnosis (11)

Occasionally, the condition presents atypically so that even experienced clinicians overlook its presence. The clinical presentations can be classified intothree large groups. The first and most common presentation is dyspnea with or without pleuritic chest pain and hemoptysis. The second presentation is hemodynamic instability and syncope, which is usually associated with massive embolism; the third and least common presentation mimics indolent pneumonia or heart failure, especially in the elderly (12)
Owing to multiple perfusion and ventilation abnormalities frequently observed in COPD lungs (even in the absence of VTE), noninvasive diagnosis of PE using imaging modalities was a significant challenge until quite recently. With the advent of contrast-enhanced (multidetector) CT, it is now possible to reliably diagnose PE in COPD subjects with minimal discomfort or risk to the patients.
Computed tomography (CT) is increasingly being used as the mainthoracic imaging technique in suspected pulmonary embolism (13-15)
First-generation single-detector-row helical CT scanner shave 90 percent specificity but only a 70 percent sensitivityfor pulmonary embolism (16-19)
The advent of multidetector-row CT scanners has improved the visualization of the segmental and subsegmental pulmonary arteries (20-22)The criteria of acute pulmonary embolism are tomographic equivalent of the classic angiographic signs of pulmonary embolism(partial or complete filling defects and railway track signs). (23)

Patients and methods

This prospective randomized study was conducted after written informed consent from every patient and approval of local ethical committee was also obtained.
This study was conducted in the Department of Intensive Care Unit at Qena University Hospital. The study was carried out on 25 patients suffering from acute exacerbation of chronic obstructive pulmonary disease (COPD) with respiratory failure.
All patients included in this study were selected to fulfill the following criteria:
Adults , history of COPD, and patients with X-ray findings showing signs of COPD hyperinflated lungs with flattened diaphragm, hyperlucent lungs and central pulmonary artery enlargement (24)
We excluded Patients with known history of deep vein thrombosis, Patients with known hypersensitivity to radiographic contrasts and pregnant women.

Patients presenting with acute exacerbation of COPD were followed for matching with inclusion criteria, for those who matched the following data were collected:
Patient Characteristics:, age and sex, indication for mechanical ventilation for ventilated patients, smoking history and its severity. Smoker patients were categorized according to smoking severity into mild moderate and severe smokers and history of previous DVT
Clinical data:

- Vitalsigns: Blood Pressure (mm Hg), Heart rate (beat /minute), Respiratory rate (breath/minute) and Temperature (^{o}C).
- Thorough physical examination.
- ECG on admission.
- Continuous pulse oximetry for arterial oxygen saturation.

Scoring systems:
a. SOFA Score: Assessment of disease severity using the sequential organ failure assessment (SOFA) system. (Table 1)
b. APACHE score: Assessment of disease severity using acute physiological and chronic health Evaluation (APACHE) on admission.(Table 2).APACHE score is a sum of three parameters: acute physiological score(APS),age points and points for the presence of chronic health condition(CHP).(Table 2)

RESULTS

The study was conducted on 25 patients Between April 2012 and December 2013 admitted to Intensive care Unit in Qena university hospital with acute exacerbation (AE) of their COPD which was diagnosed after suggestive history. All patients presented with dyspnea grade (IV),tachypnea and excessive sweating. Increased sputum secretion was reported by 20 patients. Five patients presented with disturbed consciousness level most probably due to hypercapnia. Severe productive cough was encountered in all patients but haemoptysis was present in only 2 patients who showed presence of pulmonary embolism (PE) thereafter.

Table (3) shows the relation between occurrence of pulmonary embolism, age and gender. For all of the 25 patients the mean age was 53.8 ± 11.5 years (range: 24-68), while for patients with pulmonary embolism the mean age was 46.3±19.8 years (range: 25 - 64 years)].Considering gender, 82% percent of the studied patients (n=22) were males while 18 % were females. The 3 positive patients for pulmonary embolism were all males.

Clinical examination:
All of the 25 patients were thoroughly clinically examined at admission. History taking showed that all of the patients were previously admitted to hospital by similar conditions, eighteen patients gave a history of increased sputum secretions within one week prior to admission. All of the patients were mild to heavy smokers. Two patients were admitted with altered consciousness level yet arousable. Increased somnolence was stated by 14 patients being noticed within 2 days prior to admission.
Chest examination revealed severe wheezes in 23 patients. Two patients presented with silent chest, one of whom proved to be positive for PE thereafter. Diminished air entry was noticed in all patients. Signs of right heart failure were seen in 8 patients including: congested neck veins, abdominal ascites and bilateral lower limb edema.
Past history taking revealed some patients to have chronic health diseases including: hypertension (n=12), diabetes mellitus (n=8), ischemic heart disease (n=12), chronic liver disease (n=1).
As regards symptoms of the 3 patients with PE on admission, dyspnea, cough and wheezes were present in all of them. Hemoptysis was present in one patient, congested neck veins was present in 2 patients, while fever was absent in the 3 patients. One of the patients gave a history of previous PE a year ago while the other 2 patients had no history of previous PE. The 3 patients were hypertensive by history while only one patient was diabetic.
As shown in table (3) the mean values for heart rate(HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate (RR) and temperature on admission in all patients were 104.2±10.8 beat/min, 123.7±28.3 mmHg, 74.6±12.6 mmHg, 32.7±3.3 breath/min and 37.5±0.5 °C ,respectively, while the mean values for pulmonary embolism patients were 116.7±15.3 beat/min,93.3±5.8 mmHg, 61.7±2.9 mmHg, 36.7±3.1 breath/min and 37.2±0.3 °C, respectively.

Arterial blood gasometric study;

Table (4): The average values for arterial blood gases parameters at admission for all patients (n=25) were as follows: pH: 7.20 ± 0.10 (range: 7.13-7.32), PCO2: 89.5 ± 20.8 mmHg (range: 65-140.5), PO2: 80.7 ± 7.3 mmHg

(range 66-92) , O2 sat: 90.7 ± 3.8 % (range 84-96) and $HCO3^-$: 31.4 ± 3.1 mEq/L (range 25-37). The mean values of arterial blood gases for patients with pulmonary embolism were as follows: pH : 7.25±0.15 (range 7.18-7.32) , P_aCO2: 69.4±4.1 mmHg (range 65-73) , P_aO2:73.0±7.5 mmHg (range 66-81) , O2 sat 89.0±4.4 % (range 84-92) and $HCO3^-$:28.4±3.5 mEq/L (range 25-32). This shows that patients with pulmonary embolism were more hypoxic but less hypercapnic, less hypercarbic . The 3 patients with PE were all mechanically ventilated.

Investigations:

Table (4) shows CBC values on patients' admission. Hemoglobin level in all studied patients ranged from 11 g/dl to 12.2 g/dl with a mean of 16.4±19.2 g/dl. In patients with pulmonary embolism it ranged from 11 to 13.5 g/dl with a mean of 12.2±1.3 g/dl.Red blood cells (RBCs) ranged from (4.1 - 4.8)× $10^6/mm^3$ with a mean of $4.1±0.4×10^9$ cell/mm^3 in all patients, while it was $4.5±0.4×10^9$ cell/mm³ in patients with pulmonary embolism..White blood cells (WBCs) ranged from 6000 to 18,900 cell/mm³ in all patients with a mean of 10,900±2.7 cell/mm³. In patients with pulmonary embolism it ranged from 6000 to 8000 cell/mm³ with a mean of 7.100±1.000 cell/mm³.Platelets ranged from 296,000 to 391,000/mm³ in all patients with a mean of 303,000±45.600/mm³. In patients with pulmonary embolism it ranged from 296,000 to 378,000/mm³ with a mean of 332,300±41,800/mm³. Concerning their coagulation profile (Table4) the mean prothrombin time (PT) in all patients ranged from 12 to 14 seconds with a mean of 12.4±0.7 seconds while the mean PT in patients with pulmonary embolism was 12.1±0.1 seconds (range 12-12.2) The mean partial thromboplastin time (PTT) in all patients was 32.4±2.6 sec while in patients with pulmonary embolism it was 30.0±1.0 sec. INR in all patients ranged from 1.0 to 1.4 with a mean of 1.2±0.1 while in patients with pulmonary embolism the mean was 1.1±0.2 (range 1.0-1.3).

Sputum cultures were done for all the patients using sputum specimens obtained at their admission. Eighteen patients were positive for presence of bacterial infection of which 12 patients had gram negative organisms including pseudomonas (n=7) and klebsiella (n=5) and 6 patients had gram positive organisms mainly staphylococcus aureus (n=5) and streptococcus haemolyticus (n=1). The rest of the patients (n=7) including those with pulmonary embolism had sterile sputum cultures. So 6 patients might have presented with acute exacerbation due to causes other than bacterial infection or PE, these might include viral infection, pollutants exposure or allergens (25-33)

Scoring systems:

1-APACHE II score: The mean APACHE II score for the whole studied patients ranged from 3 to 24. While the APACHE II score in patients with pulmonary embolism ranged from 12 to 24 with a mean of 19.3±6.4.figure (1)

2-SOFA score: All studied patients were evaluated by SOFA score which ranged from 2 to 4 in all patients with a mean of 2.6 ± 0.6, while in patients with pulmonary embolism it ranged from 3 to 4 with a mean of 3.3±0.6. Figure (2).None of the studied patients had renal impairment, none of them had elevated liver bilirubin but all of them were relatively hypoxic.

Length of hospital stay:

As shown in Table (5) the duration of hospital stay in all studied patients ranged from 12 to 20 days with a mean of 9.4±3.5 , while the 3 patients with pulmonary embolism stayed in hospital for 12 days each . All of the studied patients survived till hospital discharge.

Relation between occurrence of pulmonary embolism and level of smoking: The 3 patients with pulmonary embolism were all heavy smokers.

Relation between occurrence of pulmonary embolism and need for mechanical ventilation: Table (6) Seventy eight percent of all studied patients (n=20) were mechanically ventilated while 22% (n=5) were not. Concerning patients with pulmonary embolism (n=3), two patients were mechanically ventilated while the other patient was not.

Relation between occurrence of pulmonary embolism and presence of ECG changes: Table (6) one patient had supraventricular tachycardia with no history of previous similar attacks, 2 patients showed atrial fibrillation with rapid ventricular response, 10 patients had inverted T waves in at least two of the anterolateral leads and 2 patients had inverted T waves in the inferior leads. Left bundle branch block was present in 1 patient. In patients with pulmonary embolism (n=3), 1 patient had ECG changes in the form of S1 Q3 T3, 2 patients had no ECG changes.

Radiography: Plain X ray chest done for all the patients just after admission revealed the following: bilateral hyperinflated lungs (n=25), increased bronchovascular markings (n=12), unilateral pleural effusion (n=3), enlarged aortic knuckle (n=10), pulmonary hilum congestion (n=4), patches of consolidation (n=6) and lung infiltrates (n=1) .

All the patients were subjected to CTPA after stabilization of general condition. Only 3 patients had pulmonary artery emboli located as follows: one patient had pulmonary emboli in the segmental and subsegmental arteries in the right lower lobe, the other patient had a pulmonary embolus present in the segmental artery of the laterobasal segment of the right lower lobe and the third patient had pulmonary emboli in the subsegmental arteries of the laterobasal segment of the right lung.

Table (7) shows incidence of pulmonary embolism detected by multislice CT pulmonary angiography (CTPA) on the studied patients. Eighty nine percent (n=22) of the twenty five patients were negative regarding presence of pulmonary embolism. Whereas 3 patients (11.1%) were positive for pulmonary embolism. Diagnosis was established by CTPA which showed pulmonary arteries filling defects in the 3 cases.

Table 1

The Sequential Organ Failure Assessment (SOFA) score					
SOFA Score					
	0	**1**	**2**	**3**	**4**
Respiration Pa_{O2} /FIO 2 (torr)	>400	≤400	≤300	≤200 With respiratory support	≤100 With respiratory support
Coagulation Platelets (×10 3 /mm 3)	>150	≤150	≤100	≤50	≤20
Liver Bilirubin (mg/dL) (µmol/L)	<1.2 <20	1.2-1.9 20-32	2.0-5.9 33-101	6.0-11.9 102-204	>12.0 >204
Cardiovascular Hypotension	No hypotension	MAP <70 mm Hg	Dopamine≤5 or dobutamine (any dose)	Dopamine >5 or epi ≤0.1 or norepi = 0.1	Dopamine >15 or epi>0.1 or norepi>0.1
Central Nervous System Glasgow Coma Score	15	13-14	10-12	6-9	<6
Renal Creatinine (mg/dL) (mumol/L) or urine output	<1.2 <110	1.2-1.9 110-170	2.0-3.4 171-299	3.5-4.9 300-440 or <500 mL/day	>5.0 >440 or <200 mL/day

Table 2

	Score								
	+4	**+3**	**+2**	**+1**	**0**	**-1**	**-2**	**-3**	**-4**
Rectal temperature	≥41°	39-40.9		38.5-38.9	36-38.4	34-35.9	32-33.9	30-31.9	≤29.9

Arterial mean blood pressure	≥160	130-159	110-129		70-109		50-69		≤49
Heart rate	≥180	140-179	110-139		70-109		55-69	40-54	≤39
Respiratory rate	≥50	35-49		25-34	12-24	10-11	6-9		≤5
Oxygenation	≥500	350-499		200-349	<200 >70	61-70		55-60	<55
pH	≥7.7	7.6-7.69		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	<7,15
Serum sodium	≥180	160-179	155-159	150-154	130-149		120-129	111-119	≤110
Serum potassium	≥7	6-6.9		5,5-5,59	3.5-5.	3-3.4	2.5-2.9		≤2,5
Creatinine	≥3.5	2-3.4	1.5-1.9		0.6-1.4		<0,6		
Hematocrit	≥60		50-59.9	46-49,9	30-45.9		20-29,9		<20
White blood cells X 1000	≥40		20-39.9	15-19,9	3-14.9		1-2.9		<1
Glasgow coma scale	15 minus Glasgow coma scale								

Table (3): AGE and gender and clinical data of the studied patients

	All Patients	With pulmonary embolism "n=3"
Age **Rang** **Mean ±S.D.**	25 - 68 53.8 ± 11.5	**25-64** **46.3 ±19.8**
gender **Male** **Female**	22 (81.5%) 5 (18.5%)	**3 (100.0%)** **0 (0.0%)**
HR **Range** **Mean ±S.D.**	100 – 130 104.2 ± 10.8	**100-130** **116.7±15.3**
SBP **Range** **Mean ±S.D.**	90 – 190 123.7 ± 28.3	**90-100** **93.3±5.8**

DBP **Range** **Mean ±S.D.**	60 – 100 74.6 ± 12.6	**60-65** **61.7±2.9**
R.R. **Range** **Mean ±S.D.**	34 -40 32.7 ± 3.3	**34-40** **36.7±3.1**
Temp. **Range** **Mean ±S.D.**	**37 - 38.5** **37.5 ± 0.5**	**37-37.6** **37.2±0.3**

Table (4): arterial blood gasometric data, Complete blood count and Coagulation profile on admission.

	All Patients	With pulmonary embolism
pH **Range** **Mean ±S.D.**	7.13 - 7.32 7.20 ± 0.1	**7.13-7.30** **7.20 ±0.1**
P_aCO_2 (mm Hg) **Range** **Mean ±S.D.**	65 - 140.5 89.5 ± 20.9	**65-73** **69.4 ±4.1**
HCO_3^- (mEq/l) **Range** **Mean ±S.D.**	25 – 37 31.4 ± 3.1	**25-32** **28.4 ±3.5**
O_2 sat.(%) **Rang** **Mean ±S.D.**	84 -96 90.7 ± 3.8	**84-92** **89.0 ±4.4**
P_aO_2 **Range** **Mean ±S.D.**	66 – 92 80.7 ± 7.3	**66-81** **73.0 ±7.5**
Hb **Range** **Mean ±S.D.**	11 - 12.2 16.4 ± 19.2	**11-13.5** **12.2±1.3**
RBCs **Range** **Mean ±S.D.**	4.1 - 4.8 4.1 ± 0.4	**4.1-4.8** **4.5±0.4**
HCT **Range** **Mean ±S.D.**	4 – 43 36.3 ± 7.2	**4-37** **25.5±18.6**
Platlets **Range** **Mean ±S.D.**	296 – 391 303.0 ± 45.6	**296-378** **332.3±41.8**

WBCs **Range** **Mean ±S.D.**	6 - 18.9 10.5 ± 2.9	**6-8** **7.1±1.0**
PT **Range** **Mean ±S.D.**	12 – 14 12.4 ± 0.7	**12-12.2** **12.1±0.1**
PTT **Range** **Mean ±S.D.**	29 – 38 32.4 ± 2.6	**29-31** **30.0±1.0**
INR **Range** **Mean ±S.D.**	**1 - 1.4** **1.2 ± 0.1**	**1.0-1.3** **1.1±0.2**

PT = prothrorombin time.
PTT = partial thromboplastin time.
INR = international normalization ratio.

Table (5): Duration of stay in hospital for the studied patients groups.

	All Patients	**With pulmonary embolism**
Duration of stay in hospital Range Mean ±S.D.	 12 – 20 days 9.4 ± 3.5	 12-12 days 12.0±0.0

Table (6): Incidence of mechanical ventilation and ECG changes in the studied groups.

	Frequency		**With pulmonary embolism**	
	No.	**%**	**No.**	**%**
Positive	20	77.8	2	66.7
Negative	5	22.2	1	33.3
Yes	16	63	1	33.3
No	9	37	2	66.7

Table (7): Incidence of pulmonary embolism detected by multislice CT pulmonary angiography chest on the studied patients.

	No.	**%**
Positive	3	11.1
Negative	22	88.9
Total	25	100.0

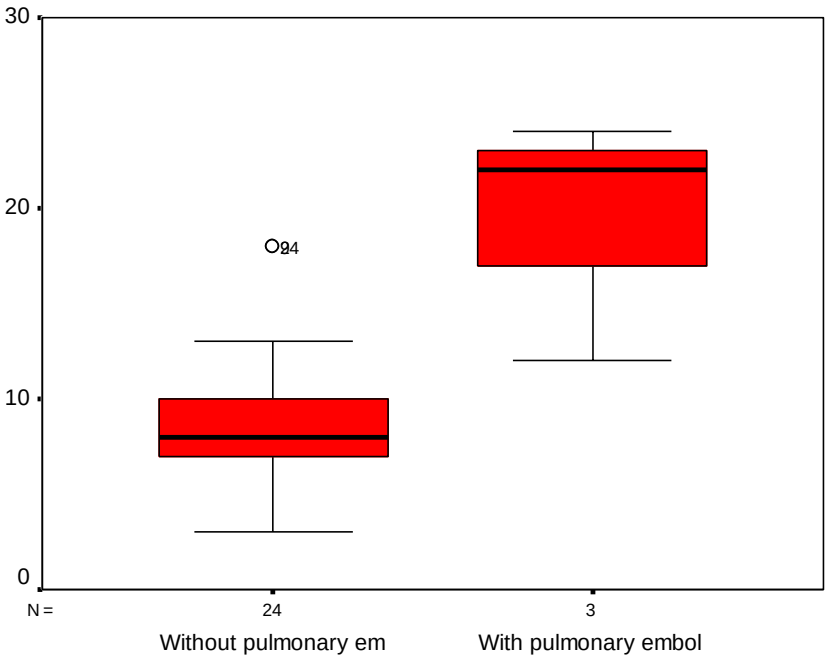

Figure (1): Box plot for APACHE in relation to pulmonary embolism

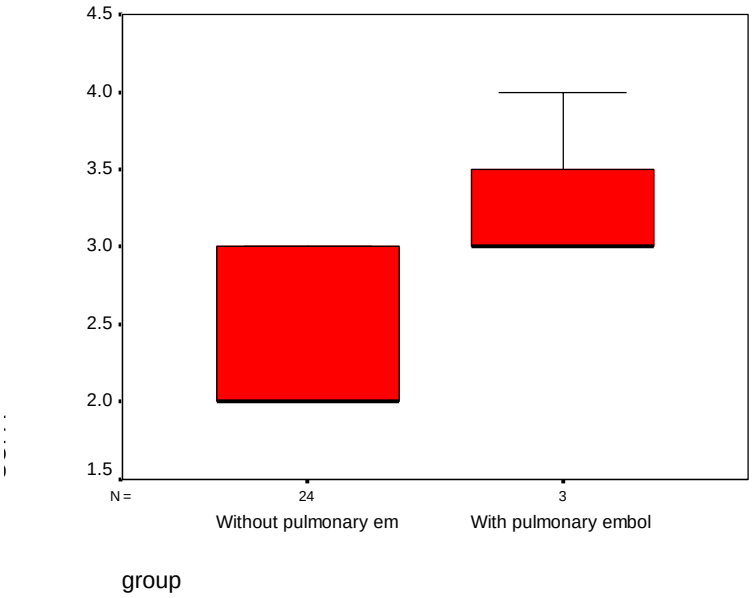

Figure (2): Box plot for SOFA in relation to pulmonary embolism

DISSCUSSION

The most important finding of our study was the relatively high prevalence of PE among patients who required hospitalization for acute exacerbations of COPD. Overall, one of four COPD patients who were hospitalized and investigated for VTE had objective evidence for PE requiring anticoagulant therapy. While striking, these data should be interpreted cautiously owing to the heterogeneity in the design, the setting, and enrollment criteria among the included studies. As regards gender and age 82% of the studied patients were males(n=22) while 18% were females(n=5). The 3 patients who were found to be positive for PE were all males. The mean age of the whole studied group was (53.8±11.5) years while the mean age for patients with pulmonary embolism (PE) was (46.3±19.8) years. Our results agree with Palla et al (34).who showed that age and gender may only partially influence the possibility of raising the suspicion of PE.

The clinical diagnosis of acute PE is unreliable because symptoms, signs and data are non specific. Moreover, the clinical diagnosis of acute PE in patients with COPD is often difficult because there is no characteristic sign of PE. The presentation of acute PE may be similar to that of AECOPD whatever the cause.

In the present study the whole study group was thoroughly clinically examined on admission to emergency department where it was found that 92.6% of patients (n=25) presented with severe chest wheezes while 2 patients presented with silent chest of whom one patient was proved to be positive for the presence of PE thereafter. Lung crepitations whether coarse (n=10) or fine (n=6) were encountered by chest examination. All of the patients were at dyspnea (IV) and had diminished intensity of their breath sounds while 66% of them (n=18) stated increased sputum volume within one week prior to admission and 74% of them stated increased cough frequency(n=20). Our results almost agree with the results of a study made by Rutschmann et al(35)were 100% of the studied patients had severe wheezes on admission, 49% of them had lung rales, 51% had increased sputum volume prior to admission and 64% had increased cough frequency. Our results almost agree with the results of a study made by Rutschmann et al (35) where 93% of patients with AECOPD who were proved thereafter to have PE presented with cough, 100% of them were dyspnic on admission. In the present study COPD patients with PE showed lower levels of PaCO2 (mean = 69.4±4.1) than those without PE (mean =89.5±20.9). This may be due to tachypnea accompanying PE as a reflex to increased dead space ventilation. These results agree with Isabelle et al (36) who found that a decrease of PaCO2 of at least 5 mm Hg from baseline was the only ABG abnormality associated with PE. In contrast to our results Shaker (37) reported higher level of PaCO2 in AECOPD presenting with PE referring that to increased dead space and respiratory muscle fatigue in these patients. Patients with PE were more hypoxemic(mean PaO2=73±7.5 mmHg) than the whole studied group (mean PaO2=80.7±7.3 mmHg) which may be due to increased dead space due to PE in addition to COPD which affects gas exchange. Our results agree with Jerald et al (38) who reported PE and COPD to cause great decrease in PaO2.However stein et al (39) who studied the alveolar arterial oxygen gradient in assessment of acute pulmonary embolism in a study conducted upon 779 patients found that PaO2 might be normal in a significant number of patients of PE and that about 25% of patients with PE have a room air Pao2 greater than 80 mmHg.

Pulmonary embolism may be an important cause of acute exacerbation in COPD patients especially those with acute respiratory failure. Yet pulmonary embolism as a whole and especially small ones can easily be misdiagnosed and are still underestimated as a cause of COPD worsening and exacerbation. This may be attributed to the majority of AECOPD patients being admitted due to chest infection. Also the diagnosis of pulmonary embolism in COPD patients is still difficult and so the prevalence of PE in COPD is rarely studied. In recent years, technical advances in CT have prompted enormous interest in the use of this technique for the diagnosis of PE. Spiral CT angiography enables the direct visualization of PE within the pulmonary arteries as low attenuation filling defects within the vessel, partly or completely surrounded by opacified blood, or as a complete filling defect which leaves the distal vessel totally unopacified.

In the present study PE was detected in 11% (n=3) of the whole studied patients with AECOPD and respiratory failure (n=25) using multislice CT detector with angiography (CTPA).One patient had pulmonary emboli in the segmental and subsegmental arteries in the right lower lobe, the other patient had a pulmonary embolus present in the segmental artery of the laterobasal segment of the right lower lobe and the third patient had pulmonary emboli in the subsegmental arteries of the laterobasal segment of the right lung. Erelel et al (40) studied 56 AECOPD patients and found 5 patients (8.9%) of them to have PE which was diagnosed by ventilation perfusion scanning. Mispelaere et al (41) reported 10 patients to have PE out of 50 AECOPD studied patients (20%) and in the prospective part of the study they reported PE in 29% of their patients. This high results was attributed to the fact that their study was

made on non infective exacerbated COPD. Tillie leblond et al (36) showed the frequency of PE to be 25% among a series of 197 consecutive patients of COPD referred for severe exacerbation of unknown origin. They excluded patients with increased sputum volume and/or increased sputum purulence, fever, history of cold and sore throat. We included these patients and this may partially explain the difference in PE prevalence in our study and their one. Again 29% of the patients of COPD studied byLeblond had cancer while none of the patients in our study had cancer.

On the other side others reported much lower prevalence of PE in patients with AECOPD. Rutschmann et al (35) reported only 3 cases (4%) of PE in a prospective study including 123 patients with moderate to severe COPD presenting with worsening of their dyspnea. They attributed their results being much lower than others to many factors; first, the imaging method used for diagnosing pulmonary embolism is crucial in a population of patients with chronic lung diseases. Ventilation–perfusion scintigraphy was used in previous studies.(40,41) Although scintigraphy is an acceptable tool for the diagnosis of pulmonary embolism in patients without underlying lung diseases, the interpretation of lung scintigraphy is difficult in patients with COPD, which might have led to an overestimation of the true prevalence of pulmonary embolism in that population.(42) In contrast, the performance of computed tomography scan is not altered in patients with COPD.(42,43) Secondly, most studies exploring the relationship between COPD exacerbation and deep-vein thrombosis or pulmonary embolism were retrospective or were based on autopsy findings(44-46). Thirdly and most importantly, we studied an unselected population of patients admitted to the emergency room of two general teaching hospitals with an acute exacerbation of their COPD. Some studies included only the most severely ill patients admitted to intensive care units(47).Others excluded patients with normal D-dimer values.(41)
Our study used multislice CT detector as a main diagnostic tool for the presence of PE. Yet we studied also unselected population admitted to emergency room with AECOPD without concerning its severity. This study showed a 11% prevalence of PE in patients with COPD admitted for severe exacerbation of unknown origin and complicated by respiratory failure, presence of PE was accompanied by high need of mechanical ventilation, presence of PE did not affect the length of hospital stay among patients with AECOPD and respiratory failure, presence of PE did not affect the mortality rate and chest infection is still the leading cause of exacerbation in patients with COPD. Thus, clinicians should consider PE in the diagnostic workup of COPD exacerbations, especially in patients where the underlying etiology is not apparent and in whom there is a history of malignancy or other additional risk factors that may increase the clinical likelihood of PE.

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