Research Article

Clinical evaluation and CTPA in acute pulmonary embolism

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Abstract

Introduction: Pulmonary embolism (PE) remains one of the leading causes of morbidity and mortality in the emergency and cardiovascular setting, especially when associated to hemodynamic instability. **Aim of the work**: to evaluate the accuracy of clinical dependent diagnosis by uses of CT angiography findings. **Patient and methods**: clinical evaluation for 70 patients who were recruited from ELMINIA university hospital with suspected PE and CTPA was done **Results**: there was significant difference between group I and group II regarding Systolic BP (p =0.0007) and Length of hospital stay while was no significant between groups regarding heart rate (p =0.122). **conclusion**: CTPA is gold stabderd in diagnosis of pulmonary embolism.

Key words: pulmonary embolism, CTPA, wells score, Revised Geneva

Introduction

Pulmonary embolism (PE) remains one of the leading causes of morbidity and mortality in the emergency and cardiovascular setting, especially when associated to hemodynamic instability. (Masotti et al., 2009).

Helical CT of the pulmonary arteries (CTPA) has become the first-line technique for the detection of emboli in the large and segmental vessels (CTPA) as it is deemed to offer an increasingly definitive detection of PE. However, increased CTPA examinations of suspected embolism associated with cost implications and increasing ionising radiation burden. (Ghaye and Dondelinger 2008)

A commonly utilised validated pre-test clinical probability (PTP) assessment tool is the Wells score . The simplified Wells score incorporates seven variables from the patient's history and initial clinical assess-ment from which a clinical probability of PE is determined as either low, moderate (intermediate) or high . Used in conjunction with a sensitive D-dimer assay PE can be safely excluded in patients with a negative D-dimer estimation and low PTP (Gibson et al., 2008)

Patients and Methods

This prospective study was carried out in the period from October 2014 to April 2017 on 70 patients who were recruited from Elminia university hospital and included patients presenting with signs or symptoms of suspected acute pulmonary embolism. (42 women and 28 men), aged from 19 to 80 years old (mean age 49±17)

The 70 patient divided into into 2 groups:

group (I): included 50 patients with proved pulmonary embolism (+ve PE)

group (II): included 20 patients (no PE) diagnosis excluded by CTPA.

We enrolled patients who completed laboratory records, clinical probability score, CTPA images, and echocardiography results and hospital length of stay.

Methods:

All patients were examined and subjected to the following:

I) Full history taking:

with special emphasis to: Age, sex, Special habits

Risk factors for venous thromboembolism as Surgery and related conditionsand Medical conditions or medications as Previous DVT or PE

Presenting symptoms including:
Dyspnea, Chest pain, Hemoptysis, Fainting, cough, leg pain or swelling.
Associated comorbidity as Ischemic heart disease, Hypertension, Diabetes mellitus.

II) Clinical examination:
General examination as Vital signs :blood pressure, heart rate ,respiratory rate
Local examination: (chest and heart) with emphasis on signs of pulmonary hypertension and signs of consolidations, Pleural effusion
Assessing clinical likelihood
Different probability scores for pulmonary embolism were calculated for each patient as wells score and revised Geneva score and PERC rule.

III) Laboratory investigations
1- D-Dimer assay
The D-dimer cut off value ⩾500 ng/ml was considered positive and results <500 ng/ml were considered negative.(Dale, 1994)
2- Renal function tests (urea and creatinine)
3- Complete blood picture mean platelet volume (MPV) and red cell distribution width (RDW) was measured using automated cell counter sysmex kx-21N (TAO Medical incorporation, Japan).
4- Arterial blood gases, including arterial partial pressure of oxygen (PaO2),partial pressure of carbon dioxide (Pa CO2) and arterial oxygen saturation(SaO2). Alveolar arterial gradient was calculated according to the formula A-a gradient= $PAO_2 – PaO_2$

IV) Imaging
1- CT Pulmonary Angiography (CTPA)
Multi-detector CT (MDCT) pulmonary angiography was performed at Radiology Department of Minia University Hospital for all patients of the study.
Pulmonary Artery Obstruction Index (PAOI) and RVD ratios were calculated in all patients without the knowledge of their clinical assessment or diagnostic exam results and The final PAOI for each patient was expressed as percent (Score/ 40× 100). (Qanadli et al., 2001)
2- Transthoracic echocardiography:
Transthoracic echocardiograms (TTE) was done to detect Signs of pulmonary hypertension

Results
This study was conducted on 70 patients who were recruited from from Elminia university hospital during the period from October 2014 to April 2017.
The seventy patients was divided into 2 groups according to CTPA
group I included 50 patients with proved pulmonary embolism (+ve PE) **group II** included 20 patients (no PE) diagnosis excluded by CTPA.

Table(1) showing comparison of clinical data between group I and group II

Clinical	group I N=50	group II (no PE) N=20	P
Systolic BP	110±20	120±15	0.0007
HR	105±15	100±10	0. 122
Length of hospital stay	11±3.5	8± 1.5	< 0.0001
wells score	6.5±2	2.5±1	< 0.0001
Revised geneva	11±7	5±2	<0.0001
sPAP	52±15	50±19	0.635
RV/LV	1±0.2	0.9±0.1	<0.0001

HR:heart rate Systolic BP: systolic blood pressure
sPAP**:** systolic pulmonary arterial pressure
RV/LV: The right ventricle to left ventricular diameter ratio

As shown in **table 1** The clinical characters of group I and group II were variable as Systolic BP was 110±20 in group I and 120±15 in group II and which was significantly different between groups (p =0.0007) while heart rate in 105±15 in group I and 100±10 in group II and which was not significant between groups (p =0.122) and Length of hospital stay was high significant between two group (p<0.0001).

Clinical probability score wells score, Revised geneva was in group I 6.5±2, 11±7 and in group II was 2.5±1, 5±2 there was high significant difference between group I and group II (p=<0.0001 and p=<0.0001 respectively).

Echocardiography finding of sPAP in groups there was no significant difference between group s (p=0.635). Regarding RV/LV ratio measured at CTPA was 1±0.2 in group I and 0.9±0.1 in group II. there was high significant different (p=<0.0001)

Table (2): Comparison of platelet indices between group I (+ve) and group II (no PE) pulmonary embolism .

Laboratory data	group I (PE)	group II (no PE)	P
	N=50	N=20	
Platelet ×103 µL	283260 ± 88063	253300 ± 94035	0.227
MPV fL	8.8±1	7.5±1.2	0.0002**
PDW fL	17±1	16±1	0.0001**

MPV: mean platelet volume PDW: platelet distribution width

Table 2 showed that MPV of group I patients was 8.8 ±1 fL and group II was MPV 7.5 ± 1.2 fL and There were statistically significant differences between groups with P = 0.0002 and PDW values was 17 ±1 fL in group I and group II 16±1 fL which was significant differences between groups P = 0.0001 While no significant difference between both groups as regards to platelets (0.227).

Table (3): Comparison of ABG between group I (+ve) pulmonary embolism and group II (no PE) pulmonary embolism

ABG	group I (+ve) N=50	group II(no PE) N=20	P
PaO_2	64±14	68±16	0.346
$PaCO_2$	32±8	42±11	< 0.0001**
SaO_2	89±11	89±9	0.814
A-aO_2	45±18	29±15	0.0008**

PaO_2= partial pressure of oxygen, $PaCO_2$ = arterial partial pressure of carbondioxide
SaO_2=arterial oxygen saturation, A-aO_2= Alveolar–arterial oxygen gradient

Table 3 showed that in groupI hypocapnea is a feature however groupII has normal $PaCO_2$ value there was significant difference between two groups P= < 0.0001 and $(A–a)O_2$ was more wide in group I than group II which was statistically significant P =0.0008** While PaO_2 and SaO_2 was no significant difference between two groups

Discussion

In the present study, the most common clinical findings were hypotension and tachycardia with significant difference regarding to systolic BP and Length of hospital stay between the PE (+) and PE (–) groups and this results was supported by Turan et al., 2017, Although tachycardia is a common finding in patients with pulmonary embolism, The reason of the lack of significant difference in pulse rate was interpreted as the presence of an alternative cause of tachycardia such as COPD and this agree with Yetgin et al., 2014 who found no significant difference regarding to heart . also there was no difference between the groups regarding sPAP and it was elevated in both group due to presence of underlying cause of increase sPAP in group II as COPD and ILD and obstructive sleep apnea.

In the current study MPV was significantly higher in patients with acute pulmonary embolism than in negative cases of

pulmonary embolism this agree with Varol et al., 2011 who determined the increased MPV levels in patients with acute PE compared with controls While Kostrubiec et al., 2010 found that MPV levels were similar in control subjects and PE patients.

Vagdatli et al., 2010 reported that both the PDW and MPV are increased in diseases associated with platelet activation and they underlined PDW as a more specific marker of platelet activation than MPV because PDW was not affected during simple platelet swelling. Also in the present study PDW was higher in patient with pulmonary embolism and it is supported by Günay et al., 2014 who found that PDW levels were significantly higher in PE patients when compared with healthy controls . Gulcan et al., 2012 said that increase in MPV is closely correlated with thrombosis and to reflect thrombosis burden in different condition and increase in MPV has been identified as an independent risk factor of recurrent vascular events

Regarding ABG results in our study the mean $(A–a)O_2$ gradient was high in both groups but it is more higher in PE group and there is statistically significant difference between the PE and non-PE groups . However, there was no difference between the two groups in either the mean PaO_2 or the SaO_2 as PE patient were hypocapnic and has wide $(A-a)O_2$ gradient and this is agreed by Rodger et al., 2000 but different studies show limited role of ABG analysis in diagnostic utility in suspected PE. as McFarlane and Imperiale, 1994 tried to improve the sensitivity of the $(A–a)O_2$ gradient in excluding PE by combining it with the absence of a prior history of thromboembolic disease (i.e., a normal $[A–a]O_2$ and no prior thromboembolic disease excludes PE) but neither McFarlane and Imperiale's nor Cvitanic and Marino's rules as reported were 100% sensitive

Stein et al.,1996 reported that a normal $(A–a)O_2$ gradient in the absence of prior thromboembolic disease had a sensitivity of only 89% and that a normal $(A–a)O_2$ gradient and a $PaCO_2$. 35 mm Hg had a sensitivity of only 92% in excluding PE

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