

**A study of the correlation between massiveness of the pulmonary embolism
assessed by pulmonary embolism index in multi-detector computed
tomography and clinical and laboratory predictors**

Thesis

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List of Abbreviations and Acronyms

2D	two-dimensional
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ABG	Arterial Blood Gases
AFE	Amniotic Fluid Embolism
AIDS	Acquired Immune Deficiency Syndrome
Anti-Xa	Anti-factor X activity
aPTT	activated Partial Thromboplastin Time
ARDS	Acute Respiratory Distress Syndrome
ASD	Atrial Septal Defect
BNP	brain natriuretic peptide
Bp	Blood Pressure
CHF	Congestive Heart Failure
CI	Confidence Interval
COPD	Chronic Obstructive Pulmonary Disease
CT	Computed Tomography
CTEPH	Chronic Thromboembolic Pulmonary Hypertension
cTnI	Cardiac troponin I
cTnT	Cardiac troponin T
CUS	Compression Venous Ultrasonography
CVS	Cerebro-vascular stroke
DSPA	Digital Subtraction Pulmonary Angiogram
DVT	Deep Vein Thrombosis
ECG	Electrocardiogram
ED	Emergency Department
ELISA	Enzyme-linked Immunosorbent Assay
HIT	Heparin-induced Thrombocytopenia
ICOPER	International Cooperative Pulmonary Embolism Registry
ICU	Intensive Care Unit
IMA	ischemia-modified albumin
INR	International Normalized Ratio
IV	Intravenous
IVC	Inferior Vena Cava
LA	Left Atrium
LMWH	Low Molecular Weight Heparin
LPA	Left Pulmonary Artery
LV	Left Ventricle
MAPPET	Management Strategy and Prognosis of Pulmonary Embolism Trial

MDCT	Multidetector Computed Tomography
MDCTA	Multidetector Computed Tomographic Angiography
MDCTPA	Multidetector Computed Tomographic pulmonary Angiography
MI	Myocardial infarction
MRV	Magnetic resonance venography
NAIDs	Non steroidal anti-inflammatory drugs
No	Number
NPV	Negative Predictive Value
NT-proBNP	N-terminal pro brain natriuretic peptide
OR	Odds Ratio
PA	pulmonary Angiography
PaCO₂	Partial arterial pressure of carbon dioxide
PaO₂	Partial arterial pressure of Oxygen
PAOI	Pulmonary Artery Obstruction Index
PCTA	Pulmonary Computed Tomographic Angiography
PDE	Paradoxical Embolism
PE	Pulmonary Embolism
PEI	Pulmonary Embolism Index
PFO	Patent Foramen Ovale
PIOPED	The Prospective Investigation of Pulmonary Embolism Diagnosis
PMRA	Pulmonary Magnetic Resonance Angiography
PPV	Positive Predictive Value
PT	Prothrombin Time
RA	Right Atrium
RHC	Right Heart Catheterization
RPA	Right Pulmonary Artery
r-PA	reteplase
RTA	Road traffic accident
rtPA	recombinant tissue plasminogen activator
RV	Right Ventricle
RVD	Right Ventricular Dysfunction
S1Q3T3	S wave in lead V1, Q wave in lead V3, and T wave inversion in lead V3
SaO₂	arterial oxygen saturation
SBP	Systolic Blood Pressure
SC	Subcutaneous

SD	Standard Deviation
SDCT	Single-detector computed tomography

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	TB	Tuberculosis	
	t-PA	alteplase	
	U	Unit	
	UK	United Kingdom	
	USA	United States of America	
	V/Q	Ventilation–Perfusion Scintigraphy	
	VKA	Vitamin K Antagonist	
	VTE	Venous Thromboembolism	
	WBC	White Blood Cell	

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Introduction

Rapid and accurate diagnosis of pulmonary embolism will improve survival and quality of life because treatment decreases mortality and the likelihood that thromboembolic pulmonary hypertension or the post-thrombotic syndrome will develop ^[1].

Due to its mostly unspecific clinical presentation, pulmonary embolism (PE) is often referred to as the great masquerader and remains a diagnostic challenge ^[2].

However, conventional X-rays are of limited value in this respect. Furthermore, V/Q scintigraphy has a poor spatial resolution and provides only indirect evidence for PE ^[3].

Its ambiguity led to an array of murky descriptions, such as “intermediate probability,” “indeterminate probability,” “moderate probability,” “low end of moderate probability,” or “moderately high probability.” ^[4, 5]

Selective pulmonary angiography, which was then the “gold standard,” is uncomfortable for the patient. Passage of a catheter through the right ventricle in patients prone to arrhythmia and can provoke non-sustained ventricular tachycardia. After positioning and securing the catheter, the patients with dyspnea are asked to hold their breath for about 30 seconds. Each lung requires at least two separate angiographic views and, therefore, at least two injections ^[5].

Multi-detector computed tomography (MDCT) is today a widely available technique. It has been shown to be a validated non-invasive tool for the diagnosis of acute central pulmonary embolism (acute PE), and, from recent reports, also for the diagnosis of peripheral pulmonary embolism ^[6, 7, 8, 9].

Therefore, the major breakthrough of MDCT over single detector CT (SDCT) has been its ability to evaluate the sub-segmental arteries, giving rise to expectation of a greater diagnostic capability of MDCT in diagnosing peripheral thrombi ^[8, 9, 10].

The interesting issue is however that it is possible to visualize segmental and subsegmental arteries with high reproducibility for the diagnosis ^[1, 5, 11].

MDCTA allows the evaluation of the degree of pulmonary arterial obstruction i.e. quantification of pulmonary embolus (PE) with computed tomographic (CT) pulmonary angiography by using a standardized index of **Qanadli et al.** The PE index was defined as:

PE index =N×D

Where N is the value of the proximal clot site, equal to the number of segmental branches arising distally, and D is the degree of the obstruction [9].

But the correlation between the massiveness of the pulmonary embolism assessed by imaging modalities and clinical outcome still remains under investigated. In a more recent study, standardized indices for the quantification of PE have been suggested as a valuable predictor of patient outcome, and according to their study, the CT PE index has been shown to be an important predictive factor of patient death, since patients with pulmonary vascular obstruction of more than 60% tend to have a poor clinical outcome [12, 13].

Aim of the work

The objective of this study was to determine whether quantification of PE with MDCT pulmonary angiography by using a standardized index is a predictor of patient outcome, and can express the severity of PE as

determined by blood oxygenation, biochemical and cardiological investigational findings. So could therefore be of value for rapid risk stratification of PE.

Review

Background

Pulmonary embolism (PE) is a common and potentially lethal condition that can cause death in all age groups. A good clinician should consider the diagnosis if any suspicion of pulmonary embolism exists, because prompt diagnosis and treatment can dramatically reduce the morbidity and

mortality of the disease. Unfortunately, the diagnosis is often missed, because pulmonary embolism frequently causes only vague and nonspecific symptoms. ^[14]

The variability of presentation sets the patient and clinician up for potentially missing the diagnosis. The challenge is that the "classic" presentation with abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia is rarely the case. ^[15]

Studies of patients who died unexpectedly of pulmonary embolism revealed that they complained of nagging symptoms for weeks before death related to pulmonary embolism. Forty percent of these patients had been seen by a physician in the weeks prior to their death. ^[16, 17]

Pathophysiology:

Pulmonary emboli usually arise from the thrombi originating in the deep venous system of the lower extremities; in cases of pulmonary embolism, it will usually propagate proximally to the popliteal vessels, and from that area embolize. ^[17]

However, rarely they may originate in the pelvic, renal, or upper extremity veins or the right heart chambers. After traveling to the lung, large thrombi can lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause hemodynamic compromise. Smaller thrombi typically travel more distally, occluding smaller vessels in the lung periphery **Fig (1)**. These are more likely to produce pleuritic chest pain by initiating an inflammatory response adjacent to the parietal pleura. Most pulmonary emboli are multiple, and the lower lobes are involved more commonly than the upper lobes. ^[12, 13, 14, 15]

Respiratory consequences:

Acute respiratory consequences of pulmonary embolism include increased alveolar dead space, pneumoconstriction, hypoxemia, and hyperventilation. Later, 2 additional consequences may occur: regional loss of surfactant and pulmonary infarction. Arterial hypoxemia is a frequent but not universal finding in patients with acute PE. ^[17]

The mechanisms of hypoxemia include ventilation-perfusion mismatch, intrapulmonary shunts, reduced cardiac output, and intracardiac shunt via a patent foramen ovale. Pulmonary infarction is an uncommon consequence because of the bronchial arterial collateral circulation. ^[18]

Hemodynamic consequences:

Pulmonary embolism reduces the cross-sectional area of the pulmonary vascular bed, resulting in an increment in pulmonary vascular resistance, which, in turn, increases the right ventricular afterload.

If the afterload is increased severely, right ventricular failure may occur. ^[18, 19]

Fig (1): The pathophysiology of pulmonary embolism ^[17]