

UPDATE ON DIAGNOSIS AND MANAGEMENT OF PULMONARY EMBOLISM

Essay
Submitted by

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List of Abbreviation

ABG	: Arterial Blood Gas
ACCP	: American College of Chest Physicians
ACEP Physicians	: American College of Emergency Physicians
APC	: Activated Protein C
APS	: Antiphospholipid Syndrome
APTT	: Activated Partial Thromboplastin Time
AST	: Aspartate Aminotransferase
AT	: Antithrombin
ATS	: American Thoracic Society
BMI	: Body Mass Index
BNP	: Brain Natriuretic Peptide
BTS	: British Thoracic Society
CDT	: Catheter-Directed Thrombolysis
CLD	: Chronic Liver Disease
COPD	: Chronic Obstructive Pulmonary Disease
CPTP	: Clinical Pretest Probability
Crcl	: Creatinine Clearance
CTEPH Hypertension	: Chronic Thromboembolic Pulmonary Hypertension

CT-PA Angiography	: Computed Tomography- Pulmonary
CUS	: Compression Ultrasonography
CVC _s	: Central Venous Catheters
DVT	: Deep Venous Thrombosis
ECG	: Electrocardiogram
ELISA	: Enzyme-Linked Immunosorbent Assay
ESC	: European Society Of Cardiology
ESR	: Erythrocyte Sedimentation Rate
ETCO ₂	: End-Tidal CO ₂ Pressure
GCS	: Graduated Compression Stockings
H-FABP	: Heart Type Fatty Acid Binding Protein
HIT	: Heparin-Induced Thrombocytopenia
HITT Thrombosis	: Heparin-Induced Thrombocytopenia with
HRT	: Hormone Replacement Therapy
Icus	: Intensive Care Units
INR	: International Normalized Ratio
IPC	: Intermittent Pneumatic Compression
IV	: Intravenous
IVC	: Inferior Vena Cava
IXa	: Active Factor 9

LDH	: Lactate Dehydrogenase
LMWH	: Low Molecular Weight Heparin
MAP	: Mean Arterial Pressure
MDCT	: Multidetector Computed Tomography
MI	: Myocardial Infarction
MRA	: Magnetic Resonance Angiography
MVS	: Micro Vesicles
NPV	: Negative Predictive Value
NT-proBNP	: N-Terminal Pro-Brain Natriuretic Peptide
PCO ₂	: Partial CO ₂ Pressure in Blood
PE	: Pulmonary Embolism
PESI	: Pulmonary Embolism Severity Index
PIOPED	: Prospective Investigation of Pulmonary Embolism Diagnosis
PO ₂	: Partial O ₂ Pressure in Blood
PS	: Protein S
PSGL-1	: P-Selectin Glycoprotein Ligand-1
RHD	: Right Heart Dysfunction
RNA	: Ribonucleic Acid
RT	: Renal Transplantation
RV	: Right Ventricle
SBP	: Systolic Blood Pressure

SC	: Subcutaneous
SK	: Streptokinase
SPECT Tomography	: Single Photon Emission Computed
SPESI Index	: Simplified Pulmonary Embolism Severity
Tc	: Technetium
TEE	: Transesophageal Echocardiography
TF	: Tissue Factor
TPA	: Tissue Plasminogen Activator
UFH	: Unfractionated Heparin
UK	: Urokinase
V/Q	: Ventilation-Perfusion Ratio
V _a	: Active Factor 5
V _E	: Minute Ventilatory Gas Volume
VFP	: Venous Foot Pumps
VIII a	: Active Factor 8
VTE	: Venous Thromboembolism
VWF	: Von Willebrand Factor
Xa	: Active Factor 10

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Aim of the Work

The aim of the work is to highlight recent updates in pulmonary embolism in the ICU as regards; risk factors, diagnosis, management, and outcome.

Introduction

Pulmonary embolism is a relatively common cardiovascular emergency and the third most common cause of death from cardiovascular disease after heart attack and stroke. ***(Goldhaber SZ, et al; 2012)***

Pulmonary embolism (PE) and deep vein thrombosis are two elements of the same pathophysiologic process referred to as venous thromboembolism. ***(Key NS, et al; 2010)***

A pulmonary embolism most commonly results from deep vein thrombosis (a blood clot in the deep veins of the legs or pelvis) that breaks off and migrates to the lung. ***(Banovac F, et al; 2010)***

The causes of venous thrombosis can be divided into two groups: hereditary and acquired causes. ***(Lijfering WM, et al; 2010)***

PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. However, early diagnosis is fundamental, since immediate treatment is highly effective. **(Roy PM, et al; 2006)**

Depending on the clinical presentation, initial therapy is primarily aimed either at life-saving restoration of flow through occluded pulmonary arteries or at the prevention of potentially fatal early recurrences. Both initial treatment and the long-term anticoagulation that is required for secondary prevention must be justified in each patient by the results of an appropriately validated diagnostic strategy. **(Roy PM, et al; 2006)**

Chapter I

Pathophysiology of venous thromboembolism

Virchow's triad

A major theory delineating the pathogenesis of venous thromboembolism (VTE), often called Virchow's triad proposes that VTE occurs as a result of:

- Alterations in blood flow (stasis).
- Alterations in the constituents of the blood (inherited or acquired hypercoagulable states).
- Vascular endothelial injury. (*Bagot CN, et al; 2008*)

Risk factors for venous thromboembolism

VTE is currently regarded as the result of the interaction between patient-related and setting-related risk factors.

Patient-related predisposing factors are usually permanent whereas setting-related predisposing factors are more often temporary.

Patient-related predisposing factors include age, history of previous VTE, active cancer, neurological disease with extremity paresis, medical disorders causing prolonged bed rest, such as heart or acute respiratory failure, and congenital or acquired thrombophilia, hormone replacement therapy and oral contraceptive therapy. (*Alikhan R, et al; 2004*)

The causes of venous thrombosis can be divided into two groups: inherited and acquired causes. (*Table 1.1*) (*Lijfering WM, et al; 2010*)

Inherited Thrombophilia

Inherited thrombophilia is a genetic tendency to venous thromboembolism. (*Dahlbäck B; 2008*)