ARDS WITH SEPTIC SHOCK

Essay

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List of Abbreviations

AECC ..... American-European Consensus Committee

ALI ..... Acute lung injury

APRV ..... Airway pressure release ventilation

ARDS ..... Acute respiratory distress syndrome

ARF ..... Acute renal failure

AVP ..... Arginine vasopressin.

BAL ..... Bronchoalveolar lavage

BNP ..... Brain Natriuretic Peptide

BOOP ..... Bronchitis Oblitrans with Organizing Pneumonia

BUN ..... Blood Urea Nitrogen.

BVV ..... Biological Variable Ventilation

$C^{-}vO2$ ..... Mixed venous oxygen content.

CGMP ..... Cyclic guanosine monophosphate

C-GMP ..... Cyclic guanosine monophosphate

COP ..... Cryptogenic organizing pneumonia

CPAP ..... Continous positive airway pressure

CT ..... Computed tomography

CVP ..... Central venous pressure

DAD ..... Diffuse alveolar damage

DIC ..... Disseminated Intravascular Coagulation.

DVT ..... Deep Venous Thrombosis

EVLW ..... Extravascular lung water

FDPs ..... Fibrin degradation products

FFAS ..... free fatty acids

FiO2 ..... Fraction of inspired Oxygen Concentration

GRV ..... Gastric Residual Volume

HFOV ..... High-frequency oscillation ventilation

List of Abbreviations

IRDS ..... Infant Respiratory Distress Syndrome

I: E ratio ..... Inspiration: expiration ratio

IAEP ..... Idiopathic Acute Esinophilic Pneumonia

ICAM-1 ..... Intercellular adhesion molecule-1

ICU ..... Intensive care unit

IL ..... Interleukins

LPS ..... Lipopolysaccharide.

LIS ..... Lung Injury Score

MODs ..... Multi-organ Dysfunction Syndrome

MAS ..... Macrophage activation syndrome

MIF ..... Macrophage migration inhibitory factor

NAC ..... N-acetylcysteine

NO ..... Nitric oxide

PICCO ..... Pulse induced Contour Cardiac Output

PaCO2 ..... Partial pressure of Carbon Dioxide in arterial blood

PAF ..... Platelet activating factor

PaO2 ..... Partial pressure of Oxygen in arterial blood

PAOP ..... Pulmonary artery occlusion pressure

pap ..... pulmonary artery pressure

PCWP ..... Pulmonary capillary wedge pressure

PEEP ..... Positive end-expiratory pressure

PGI2 ..... Prostacyclin

Plt ..... Platelet

PLV ..... Partial liquid ventilation

PMNs ..... Polmorph nuclear granulocytes

Pplat ..... Plateau pressure

RNS ..... Reactive Nitrogen Species

List of Abbreviations

ROS..... Reactive Oxygen Species

RSBI..... Rapid Shallow Breathing Index

RT ..... Respiratory therapist

SaO2..... Haemoglobin Oxygen Saturation in arterial blood

SH..... Thiol group

SIRS ..... Systemic Inflammatory Response Syndrome

SpO2 ..... Arterial O2 saturation

SBT..... Spontaneous Breathing Trial

TLRs ..... Toll-like receptors.

TNF-α ..... Tumor necrosis factor α

Vt ..... Tidal volume

V/Q ratio ..... Ventilation /perfusion ratio

VALI..... Ventilator associated lung injury

WBC ..... White blood cell count.

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Introduction

New Berlin definition for acute respiratory distress syndrome (ARDS) has categorized ARDS based on degree of hypoxemia: mild (200 mm Hg < Pao2/Fio2 ≤ 300 mm Hg), moderate (100 mm Hg <Pao2 / Fio2 ≤ 200 mm Hg), severe (Pao2 / Fio2 ≤ 100 mm Hg). in the presence of bilateral alveolar infiltrates on chest x-ray left after exclusion ventricular failure ***(Fanelli et al., 2013)***.

Sepsis, Mechanical ventilation, shock, pneumonia, aspiration, trauma (especially pulmonary contusion), major surgery, massive blood transfusion, may all trigger ARDS ***(Rubenfeld et al., 2005)***

ARDS is associated with diffuse damage to the alveoli and lung capillary endothelium. The early phase is described as being exudative, whereas the late phase is fibroproliferative in character. Injury to the endothelium results in increased capillary permeability and influx of protein rich fluid into the alveolar space. Injury to the alveolar lining cells also promotes pulmonary edema formation ***(Irwin and Rippe, 2003)***

ARDS is usually treated with mechanical ventilation in the Intensive Care Unit. ***(Marino, 2007)***

AIM OF THE WORK

This study aims to discuss ARDS with septic shock, explaining diagnosis, pathogenesis, management and prognosis.

Chapter 1

DEFINITION OF ARDS AND CLASSIFICATION

The first published scientific description, Laennec described the gross pathology of the heart and lungs and described idiopathic anasarca of the lungs; pulmonary edema without heart failure in "A Treatise on Diseases of the Chest" ***(Laennec, 2000).***

In the 1950s, pulmonary edema had become a medical subject heading by the National Library of Medicine; however, no distinction was made at that time between cardiac and noncardiac causes. They stated that what clearly moved ARDS from a nearly universally fatal form of "double pneumonia" was the development of methods of establishing secure airway access using tubes that could be attached to mechanical ventilators to deliver adequate pulmonary distending pressures ***(Ibsen, 2004)***.

These techniques extended the lives of these patients from a few hours to many days or even weeks; long enough to recover in some cases. As this kind of patient began to populate the

established intensive care units, their condition rapidly became recognized as one of the most challenging acute clinical processes to treat. Since acute, diffuse, and dense bilateral infiltrates were almost never observed except in patients requiring prolonged mechanical ventilation, many attributed the cause of such infiltrates to the ventilator, hence the term "respirator lung" ***(Ware and Matthay, 2000)***.

The term ARDS was first used in *1967*, it is not a new disease. Well-documented descriptions of hypoxemic respiratory failure after severe trauma were reported during World War II, and there were even earlier reports of pulmonary edema associated with severe infection ***(Swan et al., 2000)***.

In *1970s*, ARDS became increasingly recognized, but hydrostatic causes (e.g. volume overload) were difficult to rule out. The potential for confusion was so great that measurements of pulmonary artery wedge pressure became a very common means of diagnosis. In fact, entry criteria for one of the earliest randomized trials of corticosteroids required a measured pulmonary wedge pressure < 19 mmHg ***(Bernard et al., 2007)***.

This once nearly routine measurement is now much less frequent with the realization that exceeding an arbitrary

Pulmonary Artery Occlusion Pressures (PAOP) does not exclude the diagnosis of ALI, and that there are usually other clinical data and historical clues that allow a fairly secure diagnosis of ALI apart from volume overload to be made. Even when the PAOP is less than 18 mmHg, one cannot be certain that edema is the result of altered permeability. Reduced colloid oncotic pressure as observed in hypoalbuminemic states promotes edema in the absence of permeability changes ***(Mangialardi et al., 2000)***.

Also notable in this 1967 report, ARDS was noted as "acute" respiratory distress syndrome. However, in 1971, Petty and Ashbaugh used the term "adult" respiratory distress syndrome in another publication, probably to address the perception of ARDS as an adult version of the previously described Infant Respiratory Distress Syndrome (IRDS) ***(Ware and Matthay, 2000)***

In ***1979, National heart, Lung and Blood Institute*** revised the criteria and defined them more strictly as to select patients to be enrolled in a collaborative study. Two operative definitions to select patients with ARDS were designed: the first to enhance specificity (not including temporary conditions) - the fast entry criteria - included a PaO_2 lower than 50 mmHg for more than two hours with a FiO_2 equal to 1 and a PEEP level equal to or higher than 5 cm H_2O; the second, not to limit

sensibility (including very severe cases) - the slow entry criteria - included a PaO_2 lower than 50 mmHg for more than twelve hours with a FiO_2 equal to 0.6, a PEEP level equal to or higher than 5 cm H_2O and a shunt fraction higher than 30% after 48 hours of maximal medical therapy ***(Bethesda, 1979).***

Non-cardiogenic origin of the pulmonary oedema was introduced, as a necessary characteristic in the definition of ARDS. ARDS is defined as a clinical picture satisfying all the following criteria: 1) PaO_2 less than 75 mmHg with FiO_2 of 0.5 or greater; 2) new diffuse bilateral infiltrates on chest roentgenography; 3) pulmonary artery wedge pressure less than 18 mmHg ***(Pepe et al., 1982).***

These criteria were revised again in ***1983*** by ***Bell et al***. and ***Fein et al***. who enrolled patients in their studies on ARDS according to the following: 1) diffuse radiographic infiltrates; 2) hypoxemia requiring a FiO_2 equal or higher than 0.5 to maintain a partial arterial oxygen pressure greater than 50 mmHg and 3) a pulmonary artery wedge pressure lower than 15 mmHg ***(Bell et al., 1983; Fein et al., 1983).***

Fowler et al., 1985 introduced, among the criteria, the need of a total static pulmonary compliance value equal to or lower than 50 ml/cm H_2O together with a pulmonary capillary

wedge pressure lower than or equal to 12 mmHg and an arterial to alveolar PO_2 ratio lower than or equal to 0.2***(Fowler et al., 1985)***.

Up till 1988 no major revision were introduced and the criteria published in literature to define ARDS all moved around a combination of the aspects reported above. In ***1988*** a new approach to the definition of ARDS was designed by ***Murray et al.*** who developed a **"lung injury score" (LIS)** to quantify: the presence, severity and evolution of acute and chronic damage involving lung parenchyma. Different components were taken into account and different values were attributed to these components according to the degree of abnormality of them (Table (1)). Three levels in severity of lung injury were defined: absence of lung injury (LIS=0), mild to moderate lung injury (LIS=0.1-2.5), severe lung injury (LIS > 2.5) ***(Murray et al., 1988)***.