Pulmonary Embolism During Pregnancy in relation to Anesthesia and ICU

An Essay

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وأنزل الله
عليك الكتاب
والحكمة وعَلَّمَكَ
مَا لَمْ تَكُن
تَعْلَمُ وكَانَ
فَضْلُ اللَّهِ
عَلَيْكَ عَظِيماً
صدق الله العظيم

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LIST OF ABBREVIATIONS

PE= pulmonary embolism.

DVT= deep vein thrombosis.

VTE= venous thrombo embolism.

LMWH= low molecular weight heparin.

VE= minute ventilation.

RQ= respiratory quotient.

TV= tidal volume.

CSF= cerebro spinal fluid.

PIOPED=prospective investigations of pulmonary embolism disease.

SC= subcutaneous.

FDA= food and drug administration.

GI= gastro intestinal.

ED=emergency department.

APSAC= anisoylated purified streptokinase activator complex.

AMI=acute myocardial infarction.

BP= blood pressure.

rt-PA=recombinant tissue plasminogen activator.

tPA= tissue plasminogen activator.

qd= once daily.

PO= per orum.

IVC filter=inferior vena cava filter.

MPEs= massive pulmonary embolism.

TEE= trans esophageal echo.

PEA= pulseless electrical activity.

MAPPET=management strategy and prognosis of pulmonary embolism registry.

AFE=amniotic fluid embolism.

MPV=maternal pulmonary vasculature.

DIC= disseminated intravascular coagulopathy.

VQ= ventilation perfusion.

CPAP= continuous positive airway pressure.

PEEP= positive end-expiratory pressure.

FES=Fat embolism syndrome.

ESR= erythrocyte sedimentation rate.

CPR=cardiopulmonary resucsitation.

Aptt=activated partial thrombo plastin time.

IVC=inferior vena cava.

Et Co2=end tidal carbon dioxide.

ARDS=acute respiratory distress syndrome.

ml=millilitre.

g/dl=gram/decilitre.

L=litre.

m osm/kgm=osmolar/kg

min=minute.

mmhg=millimetre mercury.

ECG= electrocardiogram.

Paco2=partial pressure of carbon dioxide in the blood.

kpa=kilopascal.

mEq/l=milliequivalents per litre.

IV=intravenous.

RBC'S=red blood cells

RES=reticular activating system.

PVR=pulmonary vascular resistance.

RAP=right atrial pressure.

FDP=fibrin degradation products.

CNS=central nervous system.

INR=International Normalized Ratio.

Pao2= partial pressure of oxygen in the blood.

LV=left ventricle.

RV=right ventricle.

mg=milligram.

RPF=renal plasma flow.

GFR=glomerular filtration rate.

cm=centimetre.

ICH=intra cerebral hemorrhage.

MPV=maternal pulmonary vasculature.

Introduction

Pulmonary embolism is one of the predominant causes of maternal deaths. Embolic diseases including veonous thrombo embolism and amniotic fluid embolism are the commonest cause of acute hemodynamic and respiratory collapse during pregnancy. The clinical picture of embolism can range from being asymptomatic to sometimes a life-threatening emergency **(Yap et al., 2002).**

Venous thrombo embolism is approximately five times more common in pregnant women compared to non pregnant women of similar age. Pregnancy is a state characterized by Virchow's triad (hypercoagulability, venous stasis and turbulence, endothelial injury and dysfunction) **(Marik, 2008).** The most constant predisposing factor is increased venous stasis. The physiologic changes of pregnancy result in increased venous distensibility and capacitance, which are evident in the first trimester. Other factors that contribute to venous thromboembolism are caesarian delivery, advanced maternal age, prolonged bed rest, hemorrhage, sepsis, multiparity and obesity **(Liu et al., 2009).**

The most important risk factor for a woman experiencing pregnancy-related venous thrombo embolism is prior personal history of venous thrombo embolism, which increases the risk of venous thrombo embolism three fold to five fold. The next most common risk

factor is thrombophilia, which is present in 20-50% of women with venous thrombo embolism in pregnancy **(James et al., 2005).**

Amniotic fluid embolism is potentially catastrophic complication, with a mortality rate of 10-80%. This usually occurs with labor and delivery but can be associated with uterine manipulation, uterine trauma, and the early postpartum period. Amniotic fluid containing particulate cellular elements enters the vascular circulation through endocervical veins or uterine tears, obstructs the pulmonary vessels and causes vascular spasms resulting in pulmonary hypertension. Acute left ventricular failure might occur, probably due to humoral events mediated by cytokines **(Clark et al., 1995).**

The pathophysiology of amniotic fluid embolism is poorly understood. Based on the original description, it was theorized that amniotic fluid and fetal cells enter the maternal circulation, possibly triggering an anaphylactic reaction to fetal-antigens **(Knight et al., 2010).**

Treatment of pulmonary embolism during pregnancy can be divided into supportive and specific therapy. Supportive measures in patients who develop pulmonary embolism are aimed at improving and preserving adequate oxygenation and circulation. Specific measures aim to limit the damage of the material embolized and prevent the expansion or further embolization of other thrombi **(Farrell, 2001).**

Aim of the work

The main aim of this essay is to discuss predisposing factors to pulmonary embolism during pregnancy,diagnosis and management of pulmonary embolism intraoperatively and the ICU.

Physiologic changes during pregnancy

It is necessary to start with review of some physiologic changes that occur during pregnancy. Understanding these changes enables us to know how pregnancy affects organ system, many of these changes are useful to the mother in tolerating the stresses of pregnancy and delivery **(Bernhard and John, 2003)**.

A- Hematology

1- Blood volume:

One of the earliest and most fundamental changes in pregnancy is an increase in blood volume. This is detectable by the sixth week, greatest during the second trimester and reaches a plateau at 34-36 weeks. At term, the blood volume is around 48% greater than non pregnant values. Greater increase may be seen in multiparous women. Oestrogen stimulates uterine growth and the low resistance circuit created contributes to a fall in mean arterial pressure. This leads to activation of the renin-angiotensin axis and ultimately to renal retention of water and sodium by aldosterone. Oestrogen may also directly increase plasma renin activity. Progesterone is a vasodilator and contributes to this process. It enhances aldosterone production but inhibits the effects of aldosterone on the renal tubules. Potassium secretion by the renal tubules is reduced and the secretion of atrial natriuretic peptide is increased **(Bernhard and John, 2003)**.

Part of the increase in blood volume occurs through the increase in plasma volume. Plasma volume then remains stable for the rest of the pregnancy. There is also an increase in total red cell mass. The increase in the number of red cells appears to start slightly later than the changes in plasma volume towards the end of the first trimester and shows a steady rise until term. Because of plasma volume and red cell mass change at different rates, haemoglobin concentration and hematocrit also change with gestation. This dilutional effect is sometimes referred to as the physiological anemia of pregnancy. In general a haemoglobin concentration of at least 11.5g/dl is regarded as normal in pregnancy. Values less than this cannot be assumed to be due to physiological change **(Chesnutt, 2004).**

The increased blood volume of pregnancy enhances circulation to the uterus, meets the excretory demands of the kidneys and compensates for blood loss at delivery. The latter is in the range of 500 ml for a spontaneous vaginal delivery though instrumental vaginal delivery is associated with higher blood loss. The typical blood loss at Cesarean section is around 1000ml **(Bernhard and John, 2003)** .

2- White Blood Cells:

There is relative leukocytosis in pregnancy with the white cell count rising up to 12×10^9 /L at term. During labor, counts as high as 25×10^9 - 30×10^9 /L have been noted. Most of the increase is represented by granulocytes with lymphocytes and monocytes remaining unchanged. Granulocyte increase is driven by cortisol and oestrogen **(Thornburg et al., 2000).**