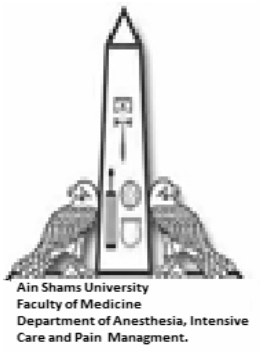

Intensive Care Management of Peripartum Pulmonary Embolism

an Essay

Submitted for partial fulfillment of master degree in general intensive care

By

Ahmed Mahmoud Ahmed Batah
(M.B., B.CH.)(Cairo University)

Under supervision of

Dr.Mohammed Abdelgaleel Sallam
Professor of Anaesthesia,Intensive Care and Pain Management
Faculty of Medicine-Ain Shams University

Dr.Ahmed Ali Mohamed Elshebiny
Assistant Professor of Anaesthesia,Intensive Care and Pain Management
Faculty of Medicine-Ain Shams University

Dr.Amin Mohammed Alansary
Lecturer in Anaesthesia,Intensive Care and Pain Management
Faculty of Medicine-Ain Shams University

Faculty of Medicine
Ain Shams University

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INTRODUCTION

Pregnancy and the puerperium (postpartum period) are well-established risk factors for venous thromboembolism (VTE).Pregnancy is associated with a number of physiological and anatomic changes that can increase the risk of VTE. Virchow described a triad of initiating factors for venous thrombosis, namely hypercoagulability, venous stasis and vascular damage, all of which occur during pregnancy **(Lussana et al., 2012).**

During pregnancy, Fibrin generation is increased, fibrinolytic activity is decreased, levels of coagulation factors II, VII, VIII, and X are all increased, free protein S levels are decreased, and acquired resistance to activated protein C is common, in addition to compression of inferior vena cava and pelvic veins by enlarging uterus**(Brenner.,2004).**

Pulmonary embolism (PE) is a leading cause of maternal mortality.It has been reported to complicate one in 1000–10,000 pregnancies in the prenatal period as The incidence of venous thromboembolism during normal pregnancy is six-fold higher than in the general female population of childbearing age **(Knight.,2008).**

The incidence of VTE, especially PE is believed to be much higher during the immediate postpartum period strongly associated with caesarean birth.And it was found that it is between 40% and 60% of all acute PE cases reported to occur postpartum; and an estimated 15-fold increased risk of PE postpartum, compared with antepartum **(Heit et al., 2005).**

Because the mortality rate for untreated pulmonary embolism in pregnancy approaches 15%–30%, a timely and accurate diagnosis is important as Delayed diagnosis, delayed or inadequate treatment, and inadequate thromboprophylaxis account for many of the deaths. A major challenge is that many symptoms of PE are similar to physical manifestations of pregnancy, for example swollen legs, dyspnoea and tachycardia **(Mallick , Petkova., 2006).**

In pregnant women with suspected is PE, the diagnosis is confirmed in approximately 4% of women as compared to 30% in non-pregnant patients, making accurate diagnostic tests imperative.So once PE suspected;Treatment with low-molecular-weight heparin or unfractionated heparin is recommended until the diagnosis is ruled out by objective testing, unless treatment is strongly contraindicated **(Ginsberg et al., 2001).**

AIM of THE ESSAY

The aim of this essay is to discuss the pathophysiology and ICU management of pulmonary embolism in pregnancy,labor and postpartum period.

CONTENTS

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علاج الرعاية المركزة للجلطة الرئوية في الفترة المحيطة بالولادة

رسالة

توطئة للحصول على درجة الماجستير في الرعاية المركزة العامة

مقدمة من

طبيب/أحمد محمود أحمد باطه

بكالوريوس الطب و الجراحة (جامعة القاهرة)

تحت إشراف

الدكتور/ محمد عبد الجليل سلام

أستاذ التخدير والرعاية المركزة وعلاج الألم

كلية الطب – جامعة عين شمس

الدكتور/ أحمد محمد علي الشبيني

أستاذ مساعد التخدير والرعاية المركزة وعلاج الألم

كلية الطب – جامعة عين شمس

الدكتور/ أمين محمد الأنصاري

مدرس التخدير والرعاية المركزة وعلاج الألم

كلية الطب – جامعة عين شمس

كلية الطب - جامعة عين شمس

٢٠١٦

المقدمة

إن الحمل والنفاس(فترة مابعد الولادة)هي عوامل خطر راسخة للجلطات الدموية الوريدية حيث يرتبط الحمل بعدد من التغيرات الفسيولوجية والتشريحية التي يمكن ان تزيد من خطر حدوث الجلطات الدموية الوريدية.وقد وصف فيركاو ثالوث العوامل المحفزة للتخثر الوريدي وهي فرط التجلط ,الركود الوريدي وتلف الأوعية الدموية ,والتي تحدث كلها اثناء الحمل.

وخلال فترة الحمل يزداد معدل انتاج الفيبرين مع انخفاض النشاط المكسر للفيبرين كما تزداد عوامل تخثرالدم الثاني والسابع والثامن والعاشر,و تنخفض معدلات بروتين اس الحر,كما انه من الشائع اكتساب مقاومة لبروتين سي النشط ,بالاضافة الي الضغط علي الوريد الأجوف السفلي وأوردة الحوض من خلال زيادة حجم الرحم.

ان الجلطة الرئوية هي السبب الرئيسي للوفاة عند الأمهات.وقد وجد انها تحدث كإحدي مضاعفات الحمل مرة من كل ١٠٠٠-١٠٠٠٠مرة من مرات الحمل في فترة ما قبل الولادة حيث ان معدل حدوث الجلطات الدموية الوريدية خلال فترة الحمل الطبيعي هوأعلي بستة اضعاف عما كان عليه جمهورالإناث العام في فترة الانجاب.

ويعتقد ان معدل حدوث الجلطات الدموية الوريدية خاصة الجلطة الرئوية أعلي بكثير في فترة مابعد الولادة مباشرة مرتبطا بشدة بالولادة القيصرية.وتبين أنه من بين ٤٠٪ -٦٠٪من الجلطات الرئويه تحدث في فترة مابعد الولادة ,ويقدر خطر الإصابة ب ١٥ضعف أكثر في فترة مابعد الولادة مقارنة بفترة ماقبل الولادة.

ولأن معدل الوفيات في الجلطة الرئوية غير المعالجة أثناء الحمل يقترب من ١٥٪-٣٠٪ فإنه من المهم جدا أن يكون التشخيص دقيقا وفي الوقت المناسب,حيث أن تأخر التشخيص وتأخر أو عدم كفاية العلاج أوالوقاية غير الكافية من الجلطة يتسبب في كثير من الوفيات.ويتمثل التحدي الأكبر في أن كثير من أعراض الجلطة الرئوية يتشابه مع المظاهرالجسمانية للحمل.وعلي سبيل المثال تورم الساقين ,وصعوبة التنفس, وزيادة ضربات القلب.

يتم تأكيد التشخيص في ٤٪ كحد أقصي في النساء الحوامل المشتبه بإصابتهن بالجلطة الرئوية مقارنة ب٣٠٪ في مثيلتهن من النساء غير الحوامل,مما يجعل الفحوصات التشخيصية الدقيقة للمرض ضرورية جدا.ولذلك فبمجرد الاشتباه في الإصابة بالجلطة الرئوية فإنه يوصي بالعلاج بالهيبارين منخفض الوزن الجزيئي أوالهيبارين غير المجزأ حتي يتم استبعاد التشخيص بالاختبارات المناسبة طالما أنه لا توجد موانع شديدة تحظراستخدام العلاج .

الهدف من الرسالة

الهدف من الرسالة هو مناقشة الفسيولوجيا المرضية وعلاج الرعاية المركزة للجلطة الرئوية في الحمل, والمخاض, وفترة مابعد الولادة.

المحتويات

1

Anatomical and Physiological background of Pulmonary Circulation

Anatomical and Physiological background of Pulmonary Circulation

The pulmonary circulation differs dramatically from the systemic circulation in its biophysical and hemodynamic properties, structural organization, and physiology. While the systemic vasculature is designed to deliver nutrients and oxygen to the tissues, the pulmonary circulation is designed to maximize oxygen uptake in the blood that will ultimately nourish the rest of the body. In order to maximize gas exchange, the pulmonary circulation has an extremely high capacitance, receiving 100% of cardiac output during each cardiac cycle. The pulmonary circulation distributes this enormous volume of blood through a complex system of arteries to a capillary bed designed to optimize gas exchange, before circulating the blood volume back to the left heart for delivery to the systemic circulation. However, despite the expansive volume of blood present, the pulmonary circulation maintains a low pressure and a low resistance **(Yuan, Rubin., 2001).**

These homodynamic properties allow the right heart to effectively and efficiently pump this large volume of

blood through the pulmonary vascular circuit, despite its relatively thin walls, compared with the left ventricle**.** Additionally, the low pressure in the pulmonary vasculature protects against vascular leak across the pulmonary capillaries. This is particularly important due to the extremely fragile nature of the blood-gas barrier and the immense surface area of the pulmonary capillary network, which predispose the pulmonary circulation to devastating vascular leak, exemplified by acute respiratory distress syndrome (ARDS) **(Effros, Parker., 2009) (Matthay et al., 2012) (Lammers et al. 2012).**

In addition to the pulmonary circulation, the bronchial circulation also delivers blood to the lung. However, in contrast to the pulmonary circulation, the bronchial circulation is part of the systemic circulation, and thus, it is connected in parallel to the vasculature of the other organs and tissues of the body and is maintained at a high pressure and high resistance. Although its primary purpose is to deliver nourishment to the large conducting airways of the lung, it also plays a critical role in heat and water exchange with the environment as well as the recruitment of inflammatory cells to the airways **(Horvath, Wanner., 2010).**

Hemodynamics of the Pulmonary Circulation:

The pulmonary circulation is a high capacitance, low resistance, and low pressure system. The maintenance of these hemodynamic properties is absolutely critical to pulmonary vascular function as increases in pulmonary pressures can cause vascular remodeling, put additional stress on the right heart, and damage the delicate gas exchange barrier **(Naeije, Westerhof., 2011).**

Pulmonary Arterial Pressure and Pulmonary Vascular Resistance:

Although interindividual variation exists, the upper limit of normal mean pulmonary artery pressure (PAP) in disease free low landers is approximately 20 mmHg, compared with approximately 100 mmHg in the aorta**.** Elevated PAP is found in disease states, including PH, as well as numerous physiologic situations, including individuals living at altitude, divers, mountain climbers, and athletes **(Mandegar et al., 2004).**

PAP is calculated as the product of cardiac output **(CO)** and pulmonary vascular resistance **(PVR)**, which describes the resistance of the whole pulmonary circuit,

including the arteries, capillaries, and veins, as indicated in Eq. 1:

$$PAP = CO \times (PVR_{arteries} + PVR_{capillaries} + PVR_{veins}) \quad (1)$$

According to this equation, a dramatic increase in CO, such as during intense exercise, would be expected to cause an increase in PAP. However, contrary to this prediction, in healthy individuals, PAP does not dramatically increase in parallel with an increase in CO. This phenomenon is due to compensatory changes that occur to prevent large fluctuations in PAP. Specifically, in response to an increase in CO, an addition alreserve of unperfused pulmonary capillaries opens. This increases the cross sectional area of the pulmonary vascular bed, which decreases PVR and ultimately minimizes changes in PAP. The physiologic mechanisms of this response were investigated by Solbert Permutt, who proposed that the pulmonary vascular bed is composed of parallel vessels with a range of collapsing pressures, which he likens to a “vascular waterfall”**.** Subsequently, it was demonstrated that vascular wall distension also contributes to the maintenance of PVR in response to changes in CO **(Naeije, Westerhof., 2011)**

In order to fully appreciate how the physiologic properties of vascular wall distension and capillary