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Case Report

Case Study of Psychological Stress Leading to Acute Respiratory Failure in a Primigravida of 38 Weeks of Pregnancy

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ABSTRACT

A 39-year-old pregnant woman with Primi-gravida was brought to the hospital two days prior to her scheduled caesarian section. About 13 years ago, the patient underwent balloon mitral valvuloplasty (BMV) for rheumatic heart disease. A year and a half ago, she had surgery for a uterine myoma under general anesthesia. We completed all of the patient's preoperative investigations. Every report fell within the typical range. The patient had acute dyspnea, shortness of breath, restlessness, productive cough, difficulty lying down in bed, and even trouble speaking during the preoperative block on the day of the procedure. Upon assessment, the patient's blood pressure was 150/90 mm/Hg, arterial oxygen saturation was 80%, respiratory rate was greater than 35 beats per minute, and pulse rate was greater than 110 beats per minute. Diffuse bilateral crepitations were detected by chest auscultation. Both hypoxemia and hypocarbia were detected in arterial blood gas. Since it was an elective caesarian section and the patient's condition needed to be improved before the procedure, the surgeon postponed it. Within 20 to 30 minutes, the patient lost consciousness, became completely cyanosed, and had foamy secretions coming out of their mouth and nose. Under general anesthesia, the internal medicine specialist recommended performing an urgent caesarian section. Without delay, the patient was moved to the operating room. The rapid sequence induction method was used to induce general anesthesia in addition to continuous suction at the nose and oral cavity. As soon as possible, the surgeon was asked to do a caesarian section. Following delivery, the infant's Apgar score was one. A pediatrician's resuscitation boosted the score to seven, at which point the baby was moved to the neonatal intensive care unit. Following the caesarian section, the patient was moved to the intensive care unit and, 48 hours later, was released from the hospital.

Keywords: *General anesthesia, Operation theatre, Endotracheal tube, Oxygen, Intravenous, Caesarian section, Apgar score, Pulse rate*

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INTRODUCTION

Feeling stressed is common during pregnancy. Because pregnancy is a time of many changes. High level of stress that continue for long time can cause health problems like high blood pressure, heart disease, gestational diabetes. There is risk of developing preeclampsia, chances of having premature baby or low birth weight baby. Some women may be worried about pregnancy loss, the health of their baby or about how they will cope with labor and birth or becoming a parent [1]. The effects of psychological stress demonstrate occurrence of hyperventilation resulting significant decrease in end-tidal carbon-dioxide (CO_2) and significantly increased heart rate [2]. Besides these, there is a reporting that a severely stressed patient can develop hypoxic hyperventilation and acute respiratory failure owing to massive pulmonary arteriovenous shunt [3]. Specific changes to maternal respiratory physiology in pregnancy i.e. an increase in tidal volume and minute ventilation acts as a contributing factor for raising the intrapulmonary shunt further. Therefore, severe stress in a term pregnant patient can lead to acute respiratory failure as a result of raised intra-pulmonary shunt.

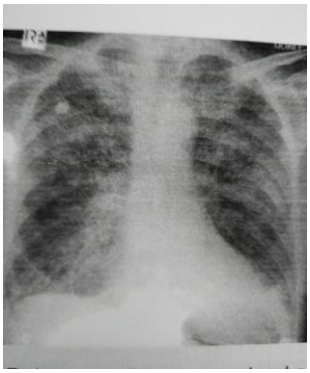

Figure – 1: X-Ray Findings of Acute Respiratory Failure in a Primigravida of 38 Weeks of Pregnancy

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CASE PRESENTATION

A 39-year-old female, possessing a body weight of 61 kilograms, was classified as a primigravida. She regularly attended the outpatient department (OPD) of a district hospital for her routine examinations. In her past medical history, she reported a diagnosis of rheumatic heart disease characterized by moderate mitral stenosis, which she had been enduring for a duration of three years, for which a balloon mitral valvuloplasty (BMV) was executed on 24th January 2007.

Tablet Pentid 400 mg was prescribed to her bi-daily as part of her medication regimen. On 18th March 2019, she underwent a laparoscopic myomectomy for the removal of a large fibroid uterus, conducted under general anesthesia. At 38 weeks of gestation, she was admitted to the hospital two days prior to the scheduled elective cesarean section (CS). All standard investigations were performed, and all results were within normal limits, with the exception of mild mitral stenosis (mean gradient 4.8 mm/hg) (MVA 1.9 cm^2) and trace tricuspid and mitral regurgitation observed in the echocardiogram. The ejection fraction was recorded at 67%. A pre-anesthetic evaluation was conducted. A consultation with a cardiologist was sought to assess the patient's status (document-1). The patient was maintained NPO (nothing per os) for a period of six hours. A dose of 7.5 mg of tablet diazepam was administered at bedtime the night prior to the surgical procedure. The administration of tablet omeprazole - 40 mg every twelve hours before meals and tablet Pentid 400 mg every twelve hours was continued as previously prescribed. On the day of the operation, an intravenous line was established using an 18G cannula two hours before the patient was transported to the operating theatre (OT). Additionally, Foley catheterization was performed. A high-risk bond was obtained from her husband, taking into account the patient's historical background of rheumatic heart disease.

The patient was promptly transferred to the pre-operative block in accordance with the established schedule. The on-call Anesthesiologist observed that the patient exhibited signs of anxiety, restlessness, and an inability to recline on the trolley. She presented with tachypnea, experiencing dyspnea, a productive cough, and was exceedingly restless, rendering her unable to communicate with the physician. Symptoms of confusion, somnolence, agitation, and diaphoresis were evident. Upon examination, the respiratory rate (RR) exceeded 35 breaths per minute, the pulse rate (PR) was greater than 110 beats per minute, blood pressure (BP) measured at 150/90 mm of Hg, and the saturation of arterial oxygen (SaO2) was recorded at 80% via pulse oximetry. Following the application of oxygen (O2) through a mask at a flow rate of 5-6 liters per minute, there was no improvement in SaO2; in fact, the saturation continued to decline. Arterial blood gas (ABG) analysis revealed a partial pressure of arterial oxygen (PaO2) of 50 mm Hg, partial pressure of arterial carbon dioxide (PaCO2) of 32 mm Hg, bicarbonate (HCO3) level of 23 mEq/L, base deficit of 1 mEq/L, SaO2 of 80%, and a pH level of 7.30. The chest examination indicated the presence of diffuse bilateral crepitations. Subsequently, the surgeon elected to postpone the operation until the patient's condition exhibited improvement, given that it was an elective procedure. Within 20 to 30 minutes within the preoperative block, the patient's condition deteriorated, with frothy secretions emerging from the mouth and nose. The patient became completely cyanotic and abruptly lost consciousness. The consultant in internal medicine assessed the patient and recommended the immediate performance of a cesarean section under general anesthesia (GA). At that juncture, the patient was transported to the operating theater. General anesthesia was administered. Upon monitoring in the operating theater, the patient's BP was noted to be 150/90 mm Hg, despite her cyanotic and unconscious state. Through the process of intermittent suctioning via the mouth and nose, and utilizing rapid sequence induction, the patient was intubated with an endotracheal tube (ETT) number 7, with the cuff inflated and secured at a right angle to the mouth following confirmation of equal chest inflation bilaterally. The induction of anesthesia was achieved via intravenous (i.v.) administration of Propofol at a dosage of 2 mg/kg body weight and suxamethonium at a dosage of 1.5 mg/kg body weight. Anesthesia was sustained using 100% O2 and Isoflurane at a concentration of 0.7-0.9% through a semi-circular closed system connected to the anesthesia machine. Morphine at a dosage of 0.1 mg/kg was administered intravenously. The patient's respiration was managed utilizing intermittent positive pressure ventilation (IPPV). Muscle relaxation was facilitated with vecuronium at a loading dose of 0.8 mg/kg body weight, with subsequent doses of one-fourth of the initial dosage administered as necessary. Furosemide at a dosage of 20 mg was administered intravenously on multiple occasions to mitigate pulmonary edema; totaling 60 mg. Morphine was also re-administered once. Throughout the entirety of the operative procedure, intermittent suctioning through the ETT was conducted to ensure the unobstructed flow of O2 and anesthetic gases. During the operation, efforts were made to maintain blood pressure at a normal level through the regulation of Isoflurane concentration. Fluid administration was meticulously managed to avoid both overload and hypotension in the patient. The total volume of fluid administered amounted to 1500 ml, comprised of a variety of Ringer's lactate and Normal saline. The total blood loss incurred during the operation was approximately 400 ml. The duration of the operation was approximately one hour, with total urinary output recorded at 300 ml.

Five to ten minutes after the anesthetic was administered, the baby was delivered. The infant had complete cyanosis. At birth, the Apgar score (AS) was 1. Following resuscitation, AS increased to 5 after one minute and to 7 after five. Following that, the infant was moved to the neonatal intensive care unit (NICU). The patient had tachycardia, foamy discharges that persisted through the ETT, blood pressure of 110/70 mm Hg, and a SaO2 level of greater than 90% at the completion of the procedure. With the aid of vecuronium infusion, the patient was not turned around and was sent to the intensive care unit (ICU), where they were kept on a ventilator in PPV mode.

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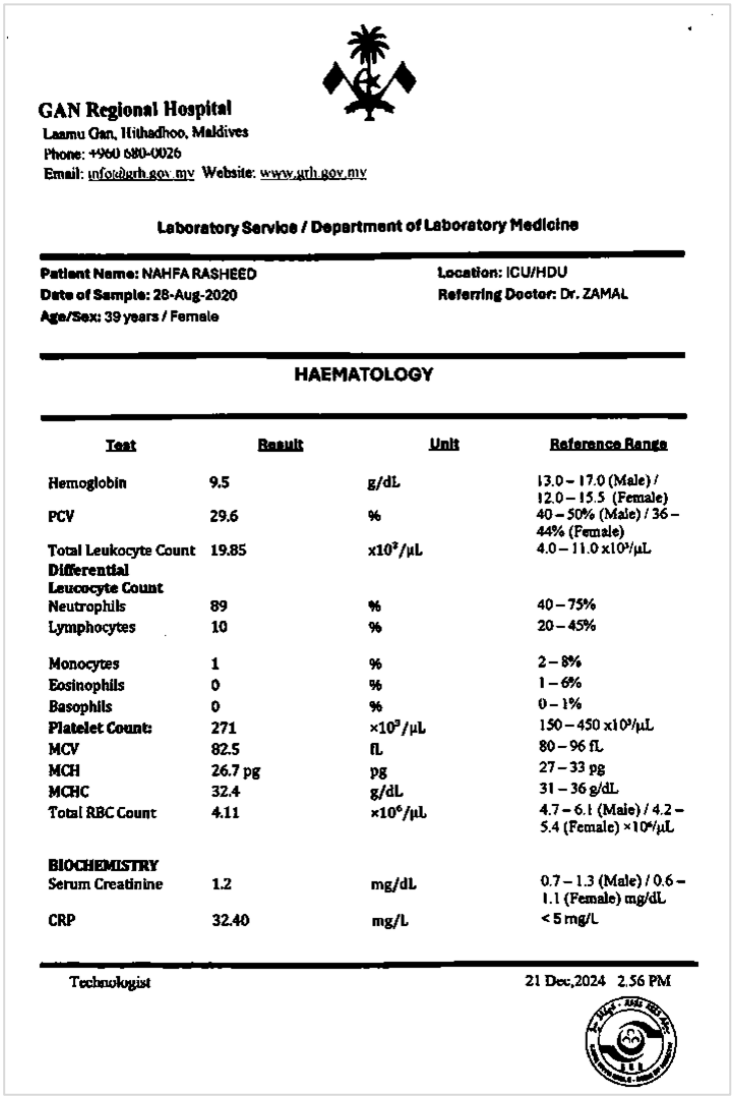
SaO2, ECG, and blood pressure were tracked. ETT intermittent suction was maintained. A chart of intake and outflow was also kept up to date. Every inquiry that was required was completed (document-2). ECG and chest x-rays were also performed. The ECG revealed sinus tachycardia and bilateral diffuse congestion on the chest X-ray. Weaning off of ventilation was done after 24 hours, when the patient's condition stabilized, and extubation, or the removal of the ETT, was carried out after 36 hours. After 48 hours, the patient was released from the intensive care unit. Thankfully, the infant was also released from the intensive care unit the same day.

DISCUSSION

Respiratory failure affects up to 1 in 500 pregnancies, more commonly in the postpartum period [4]. The causes are pregnancy specific conditions like preeclampsia, amniotic fluid embolism and peripartum cardiomyopathy. Pregnancy by increasing the severity of other conditions like asthma, thromboembolism, viral pneumonitis and gastric acid aspiration may lead to respiratory failure. Acute respiratory failure requiring mechanical ventilation is a rare complication of pregnancy affecting 0.1 to 0.2 percent of pregnancies [5]. In the case presentation, the patient developed respiratory symptoms when the patient was transferred from the ward to the preoperative block. The ABG report showed hypoxemia and hypocarbia. It indicated that the respiratory failure developed was type-1 respiratory failure.



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Laboratory Service / Department of Laboratory Medicine

Patient Name: NAHFA RASHEED
Location: ICU/HDU
Date of Sample: 28-Aug-2020
Referring Doctor: Dr. ZAMAL
Age/Sex: 39 years / Female

HAEMATOLOGY

Test	Result	Unit	Reference Range
Hemoglobin	9.5	g/dL	13.0 – 17.0 (Male) / 12.0 – 15.5 (Female)
PCV	29.6	%	40 – 50% (Male) / 36 – 44% (Female)
Total Leukocyte Count	19.85	$x10^3/\mu L$	4.0 – 11.0 $x10^3/\mu L$
Differential Leucocyte Count			
Neutrophils	89	%	40 – 75%
Lymphocytes	10	%	20 – 45%
Monocytes	1	%	2 – 8%
Eosinophils	0	%	1 – 6%
Basophils	0	%	0 – 1%
Platelet Count:	271	$x10^3/\mu L$	150 – 450 $x10^3/\mu L$
MCV	82.5	fL	80 – 96 fL
MCH	26.7 pg	pg	27 – 33 pg
MCHC	32.4	g/dL	31 – 36 g/dL
Total RBC Count	4.11	$x10^6/\mu L$	4.7 – 6.1 (Male) / 4.2 – 5.4 (Female) $x10^6/\mu L$
BIOCHEMISTRY			
Serum Creatinine	1.2	mg/dL	0.7 – 1.3 (Male) / 0.6 – 1.1 (Female) mg/dL
CRP	32.40	mg/L	< 5 mg/L

Technologist
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Figure – 2: Laboratory Findings of Acute Respiratory Failure in a Primigravida of 38 Weeks of Pregnancy

The patient was 39 years. It was her 1st pregnancy. As she was elderly Primi, therefore the baby was valuable for the family. On the other hand, the patient was of having rheumatic heart disease for that she used to take tablet Pentid 400mg twice daily for about 13 years. In the ward, the patient was accompanied by her husband and other relatives too. She was having at all no problems after taking admission into the ward for two days. She was prepared for elective CS. When she was shifted to pre-operative block for the sake of CS, she became emotionally upset probably due to stress of surgery or about

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the outcome of the child. The patient's psychological stress along with mild form of rheumatic heart disease made her condition worst. The patient developed respiratory distress. It resulted increase the work of accessory respiratory muscles leading to fatigue and ultimately unconsciousness ensued. SaO_2 was not at all improved after giving oxygen via mask, even fell downwards indicating that the hypoxic respiratory failure developed was not due to ventilation-perfusion mismatch, but due to clinically significant pulmonary shunt. Reported that in ventilation perfusion mismatch increases in FIO_2 result in increase in PaO_2. On the other hand, shunts are associated with a widened alveolar-arterial oxygen tension [P(A-a) O_2] gradient and the resultant hypoxemia is resistant to correction with supplemental oxygen alone when the shunt fraction of the cardiac output is >30% [6]. Therefore, in our case report, the acute respiratory failure is due to large pulmonary shunt that is probably greater than thirty percent of cardiac output of the patient. Evidences of rheumatic heart disease of the patient and her psychological stress with presented clinical findings strongly indicate that the acute pulmonary oedema was precipitated with the result of stress cardiomyopathy. Specific changes to maternal respiratory physiology in pregnancy i.e. an increase in tidal volume and minute ventilation accentuate the maternal symptoms. Stress cardiomyopathy is a condition precipitated by severe physical or emotional stress which was first identified in the 1990s [7]. After an emotional trauma, normal plasma level of epinephrine, norepinephrine and dopamine raises to 10-30 times, that remains high for several days. This raising of circulating catecholamine is thought to 'stun' the heart and prevent it from pumping properly [8]. So, stress cardiomyopathy is also named as 'myocardial stunting'. The result is a dangerous reduction in blood flow, chest pain, breathlessness, arrhythmia and sometimes congestive heart failure or shock. In our case presentation, the patient was fully stable psychologically and hemodynamically for two days in the ward after taking admission into the hospital. The period she was shifted to the preoperative block, she developed all the symptoms abruptly as described which mimic the clinical features of acute pulmonary edema. So, stress cardiomyopathy was the prime cause behind the development of acute pulmonary oedema. Increased circulating neurohormones caused peripheral vasoconstriction i. e. increased afterload. The patient had elevated BP i.e. 150/90 mm Hg in the preoperative block resulting from sympathetic activation. Splanchnic vasoconstriction led to the redistribution of blood contributing to increased preload and pulmonary volume overload. Another important point is that in distinction to an acute myocardial infarction, stress cardiomyopathy per se is generally transient without long term damage to the heart [9]. In our case, the patient was kept in ICU under ventilator only for 36 hours after which the patient was extubated i.e. ETT was removed and after 48 hours, she was discharged from the hospital. In ICU, The ECG reporting was within normal limit except tachycardia and Troponin-I was also negative. Therefore, the presented case is due to stress cardiomyopathy. The lungs in acute pulmonary oedema are like an overflowing bathtub. The line of management is to simultaneously stop the inflow by turning off the tap (preload reduction) and increase out flow by unclogging the drain (afterload reduction).In the management of pulmonary oedema in this particular case, during operation under GA and afterwards in ICU, by controlling the i.v. administration of fluid according to central venous pressure and judicial use of morphine in incremental doses and of furosemide i.v. repeatedly both preload & afterload were tried to lower. On the other hand, SaO_2 was maintained >90% with IPPV and intermittent suction through ETT. Finally, at ICU while the patient clinically improved, the ABG report was normal, the patient's hemodynamic condition was stable and the patient was conscious and oriented, ETT was removed. The respiratory failure in this case was not due to acute respiratory distress syndrome (ARDS). According to the Berlin definition, ARDS is characterized by lung injury of acute onset within 1 week of an apparent clinical insult and with progression of respiratory symptoms [10]. Clinically this criterion was not present in the case. As ARDS is associated with extensive alveolar damage, so there is slow resolution. This case showed prompt resolution due to raised endothelial permeability due to raised preload as well as stress cardiomyopathy without causing alveolar damage for which the patient was discharged from ICU only after 48 hours. Therefore, the acute respiratory failure developed in this presentation is due to acute pulmonary oedema resulting from stress cardiomyopathy.

It is not a case of hyperventilation syndrome (HVS) as the symptoms developed were acute & sudden onset and also distressful leading to type-1 respiratory failure. When a patient has repeated episodes of hyperventilation symptoms, then it might be diagnosed with HVS in which symptoms usually last 20-30 minutes, feeling anxious, nervous or tense, frequent sighing or yawning, feeling air hunger. Many women have problems with hyperventilation during pregnancy, but it usually goes away on its own after delivery [11].

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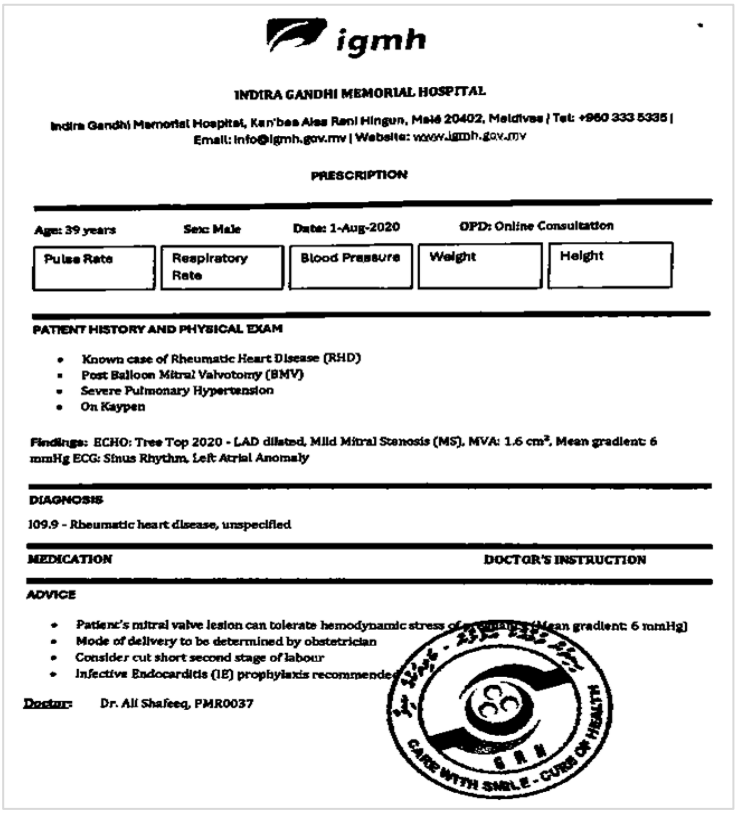

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PRESCRIPTION

Age: 39 years Sex: Male Date: 1-Aug-2020 OPD: Online Consultation

Pulse Rate	Respiratory Rate	Blood Pressure	Weight	Height

PATIENT HISTORY AND PHYSICAL EXAM

- Known case of Rheumatic Heart Disease (RHD)
- Post Balloon Mitral Valvotomy (BMV)
- Severe Pulmonary Hypertension
- On Kaypen

Findings: ECHO: Tree Top 2020 - LAD dilated, Mild Mitral Stenosis (MS), MVA: 1.6 cm², Mean gradient: 6 mmHg ECG: Sinus Rhythm, Left Atrial Anomaly

DIAGNOSIS

I09.9 - Rheumatic heart disease, unspecified

MEDICATION DOCTOR'S INSTRUCTION

ADVICE

- Patient's mitral valve lesion can tolerate hemodynamic stress of pregnancy (Mean gradient: 6 mmHg)
- Mode of delivery to be determined by obstetrician
- Consider cut short second stage of labour
- Infective Endocarditis (IE) prophylaxis recommended

Doctor: Dr. Ali Shafeeq, PMR0037

CARE WITH SMILE - CURE OR HEALTH

Figure – 3: Laboratory Findings of Acute Respiratory Failure in a Primigravida of 38 Weeks of Pregnancy

CONCLUSION

This instance demonstrates the significant risk that intense mental stress poses, especially for pregnant women who are at term and have rheumatic heart disease. It can cause acute pulmonary edema brought on by stress cardiomyopathy, as our case presentation explains. Acute pulmonary edema is diagnosed clinically. However, the condition's genesis is not always evident. The absence of a precise etiological diagnosis should not be an excuse for delaying the start of a particular treatment. Otherwise, the outcome for the mother or the fetus would be impacted. Because it contributes to the development of additional intrapulmonary shunting from atelectasis, the management's vision is that the decision to deliver the fetus immediately should be made. Treatment options include immediate CS under GA, cautious titration of afterload reduction, and prevention of volume overload, which involves reducing preload, in order to maintain normal blood pressure during operation. Thus, it may be concluded that early diagnosis and a multidisciplinary approach to management are essential for the best outcomes for both the mother and the fetus.

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