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CASE REPORTS

ACUTE RESPIRATORY FAILURE IN AN ASTHMATIC PREGNANT PATIENT

Innayat Ullah, *Asma Khalid, **Imtiaz Ahmed, **Tayab Raza

CMH Abbottabad, *CMH Hyderabad, **CMH Okara

INTRODUCTION

Pregnancy affects virtually every organ and system. Many of these physiological changes appear to be adaptive and useful to mother in tolerating the stress of pregnancy, labour and delivery. Other changes require special considerations in caring of the patient. If a system is unable to keep balance between supply and demand that system fails. Acute respiratory failure remains an important cause of maternal and fetal morbidity and mortality.

Asthma is the most common respiratory problem during pregnancy [1]. Close monitoring and consistent control of asthma are crucial to maternal and fetal well-being. Assessment of the patient with an exacerbation includes the history, examination and an objective measure of lung function. Findings that predict hospitalization include diaphoresis, use of accessory muscles, assumption of up right posture, altered level of consciousness, pulse rate greater than 120 beats per minute, respiratory rate greater than 30 breaths per minute, pulses paradoxes greater than 18 mmHg and a peak expiratory flow rate less than 120 L/minute.

During acute attacks arterial blood gases typically reveal mild hypocapnia and moderate hypoxaemia. In pregnancy the baseline partial pressure of arterial carbon dioxide (PaCO2) is often already depressed and likely decreases further with an acute asthmatic attack. A partial pressure of arterial carbon dioxide (PaCO2) of 35 mmHg therefore may actually represent "pseudonormalization" caused by fatigue and possibly impending respiratory failure [2].

Correspondence: Lt Col Asma Khalid, Department of Obst/Gynae, Combined Military Hospital, Hyderabad.

Thromboembolism, amniotic fluid embolism and venous air embolism together account for approximately 20% of maternal deaths. Other causes of acute respiratory failure account for another 10-15% of maternal deaths. Generally acute respiratory failure from various causes accounts for 30-35% of maternal deaths. Maternal mortality may rise up to 70% in case of

Acute Respiratory Distress Syndrome (ARDS) [3-7]. Rare causes of acute respiratory failure in pregnancy include respiratory infections, Pneumonia, Beta-adrenergic tocolytic therapy associated with pulmonary edema, pneumomediastinum and pneumothorax. Generally the guide lines to intubate and to provide ventilatory support are same for both pregnant and non pregnant patients. However pregnant patients need additional care due to changes associated with pregnancy as has been mentioned subsequently.

Hyperemia associated with pregnancy can narrow the upper airway. Patients are at increased risk of upper airway trauma during intubation so small endotracheal tubes may be required. The decreased functional residual capacity may lower the oxygen reserve. Before any attempt at intubation, 100% oxygen should be administered, but hyperventilation must be avoided. Cricoid pressure can help to decrease gastric inflation and can prevent regurgitation. Hypotension not responding to modified supine position or fluid resuscitation will require vasopressors. Nutritional support is important for both maternal and fetal out come.

An early decision regarding ventilatory assistance and if pregnancy is near to its term, decision to perform natural or operative delivery may be a wise step for safety of mother and baby [8,9].

CASE REPORT

A 34 years old married woman was brought to emergency department on 14 Aug 2003 with complaints of severe difficulty in breathing, sweating, inability to lie in bed for last 02 hours. She had amenorrhoea for last 09 months and was a known case of Bronchial asthma for the last 15 years, for which she was taking regular treatment. She was 5th gravida, 4 Para, with no present history of labour pains, fever, leakage or bleeding per vaginum.

General and physical examination revealed sweating, eye brow lifting, nasal flaring, pursing of mouth, licking of lips, ineffective cough, active expiration, irritability, tracheal tug; Patient was restless all the time and was continuously requesting for help in breathing assistance. She rested in a forward sitting posture at one time but then changed posture sooner.

She frequently lifted arms, picked bed clothes, was very anxious due to air hunger. Her systemic examination revealed tachypnoea with 40 breaths per minute. Breathing was noisy with audible wheezing. Accessory muscles of respiration were in excessive use. Pulse was 120 beats per minute, regular, weak and fast. Blood Pressure (BP) was 90/70 mmHg. Sweating was excessive. She weighed 50 kg Her Last Menstrual Period (LMP) was 20th Nov 2002 and Expected Date of Delivery (EDD) was 27th Aug 2003. Ultrasound abdomen revealed single alive fetus with cephalic presentation. Weight of fetus was 2649±397 grams. Fetal heart rate was 90/min. No fetal movements detected in 20 minutes.

Her vaginal examination revealed closed Os, with poor Bishop Score. Her history of past illness revealed history of frequent hospital admissions for management of asthma, pregnancies and deliveries.

She was seen by medical specialist, gynaecologist and anaesthetist and was diagnosed as "Full term pregnancy with acute on chronic severe persistent asthma and Acute respiratory failure". Along with necessary medical treatment and urgent investigations, she was planned for emergency C-Section and tubal ligation.

Patient was shifted to operation theatre in sitting position. She was not Nill per Oral (NPO) and took milk and food. Her O2 saturation was 88% with O2 being given by mask at 10 litres per minutes. She was unable to perform Sabrazze's breath holding test. Her peak expiratory flow rate was 60 L/minute.

Patient was given injection metochloperamide 10 mg I/V, lignocaine Hcl 100mg and Oxygenated at 10L/minute for 03 minutes. General anaesthesia was induced in modified supine position. Sallick's manoevure was performed by an assistant. Ventilation was controlled with Intermittent Positive Pressure Ventilation (IPPV).

I/V fluids were given after warming up to 38^{o}C. Intraoperatively Inj Hydrocortisone Sodium succinate 200 mg I/V, and Lignocaine 100 mg I/V were given in addition to already started Inj Aminophylline 250 mg I/V infusion in ITC. As her wheeze and bronchospasm was not relieved. She was given salbutamol (100 ugm) 03 puffs intra operatively during ventilation with anaesthesia circuit although adequate delivery of salbutamol was doubtful Inj Adrenaline 1:1000 ,0.5 ml subcutaneously was given and repeated which relieved bronchospasm and improved wheeze.

A full term male baby was delivered through a vertical incision. Apgar score of baby was 4/10 at 01 minute, 6/10 at 03 minutes and 8/10 at 05 minutes. Baby was seen by child specialist and shifted to nursery.

Patient after completion of operation was shifted to surgical ITC and ventilated with Adult star 2000 ventilator. Ventilators settings were as shown in (table-1). Sedation was provided with Inj Midazolam 17 ug/Kg/min and propofol 33 ug/Kg/min. Patient was successfully weaned off the ventilator and

was extubated after 48 hours ventilatory support on 16th Aug 2003 when her condition satisfied the intensivist as per criteria mentioned in (table-3).

Antibiotic cover, analgesia, and $Beta_2$ agonist, chest physiotherapy, incentive spirometry, nutritional support and nursing care continued in ITC post operatively. On 22nd Aug 2003, Patient's condition deteriorated again with weak pulse and low BP along with severe respiratory difficulty (Status asthmaticus).

Abdominal dressings were found wet. Wound examination revealed "Burst Abdomen". Patient was shifted to OT for closure of abdomen under general anaesthesia. Closure of the abdominal wound was carried out by gynaecologist and surgical specialist. Anaesthetic technique was similar to previous one. One unit of blood was transfused after grouping,cross matching and screening. Other treatment was continued.

On 26th Aug 2003, after 76 hours, patient was successfully weaned off the ventilator when she was haemodynamically stable and fulfilled criteria for weaning (table-3).

DISCUSSION

Our discussion will remain focused on following aspects of the case report: Pregnancy, Asthma, respiratory failure. Pregnancy, subsequent labour and delivery are accompanied by physiological changes in multiple organ systems [10]. Changes in the pulmonary system during pregnancy are manifested as alteration in the upper airway, minute ventilation, lung volumes and arterial oxygenation.

An increased circulatory level of progesterone is presumed to be the stimulus for increased minute ventilation. The resting PaCO2 decreases from 40 mmHg to near 30 mmHg during first trimester as a result of increased ventilation.

These changes can influence the selection of tracheal tube size, rate of induction and emergence from anaesthesia. The combination of increased minute ventilation and decreased functional residual capacity speeds the rate at which denitrogenation occurs in the term parturient as compared with the non-pregnant patient.

Induction of general anaesthesia in a parturient may be associated with a marked decrease in PaO2 if apnoea is prolonged as during intubation of the trachea. This tendency for a rapid decrease in arterial oxygenation reflects a decreased oxygen reserve secondary to a reduction in functional residual capacity. The near 20% increase in oxygen consumption present near term also contributes to a decreased oxygen reserve. These changes emphasize the potential value of Pre-oxygenation, before apnoea in a parturient. The parturients PaO2 often exceeds 100 mmHg breathing room air, reflecting the presence of chronic hyperventilation. But in order to maximize the fetal benefits of pre-oxygenation the maternal inhalation of oxygen may need to be continued for about 06 minutes at the time of induction of anaesthesia, since this is the estimated time required for maternal to fetal equilibration. When patient assumes the supine position, aortocaval compression, may further cause a decrease in PaO2 necessitating left uterine displacement [11].

Asthma is a reversible obstructive lung disease with an estimated frequency during pregnancy of 0.4 to 1.3%. Weinstein et al have evaluated the effects of pregnancy on the clinical disease activity of asthmatic patients [12]. Approximately half demonstrated no change in their asthma, one fourth improved and one fourth experienced a worsening of their disease. Pregnant asthmatics should be identified as high risk. Maternal complications are increased including complicated labor and toxemia. Perinatal mortality is doubled in pregnant asthmatics and frequency of growth retardation is increased. Management of asthma during pregnancy is similar to non-pregnant asthmatic patients. In addition fetal

surveillance, serial ultrasonography, and non stress tests should be performed. Xanthine bronchodilators and corticosteroids administration are not harmful to the fetus and should be used when indicated [13].Respiratory failure in pregnancy is characterized by severe maternal and fetal hypoxemia and/or hypercarbia, acid base disturbances hemodynamic consequences and impaired nutrition to the fetus [4].The causes of respiratory failure include some conditions that are unique to pregnancy and others that are not (table-2). Most causes of respiratory failure in pregnancy are characterized by problems associated with hypoxemia [14].Causes of hypercarbic venlitatory failure include severe asthma and extra pulmonary factors (table-2).Acute severe asthma in a pregnant patient requires rapid and intensive management including early hospital admission and aggressive management of ventilation in intensive care setting [15].

A serious problem may occur if the decision to delay mechanical ventilation is made without considering the normal range for arterial blood gases in pregnancy. A PaCO2 of 38-40 mmHg which is normal for the non-pregnant state may signal the need for urgent intervention in a pregnant woman. A decision to provide supportive ventilation must take into account coexisting hypoxemia, tachypnoea, metabolic acidosis, and maternal fatigue.

Hypoxia and hypercarbia are relatively late signs of respiratory failure. Decision to ventilate the patient may be taken on clinical grounds and hemodynamic instability. This becomes even more dependent on clinical judgement when facilities of Arterial Blood Gases (ABGs) do not exist as in our case for example. Thus therapy for respiratory failure includes oxygenation, tracheal intubations and ventilation, haemodynamic and nutritional support. Pharmacologic therapy consists of direct airway delivery of aerosolized $Beta_2$ agonists, corticosteroids, and cromolyn sodium. This route minimizes systemic absorption and reduces their impact on uterus and fetus [16]. As in CPR, these drugs should not be withheld because of concerns about fetal effects. The well being of mother comes first and her welfare is inextricably linked to that of the passenger. Ventilator settings are similar to those for non-pregnant patients. The aim is to keep F102 below 50%, use of Positive End Expiratory Pressure (PEEP) if PaO2 falls below 60-65 mmHg and maintain PaCo2 at 30-32 mmHg before delivery and 35-40 mmHg after delivery. Criteria to wean off the

Table-1: Ventilator's settings.

Cycling	Volume Cycled
Mode	Assist/Control
Tidal Volume (10ml/kg)	500 ml
Respiratory rate	14/min
I.E. Ratio	1:2.5
PEEP	Zero
Peak flow rate	50 L/min
Sensitivity	0.5 cm H2O
FiO2	0.5
Ventilator used	Adult Star 2000.

Table-2: Causes of respiratory failure in pregnancy.

Asthma
Aspiration of gastric contents
ARDS
Beta - adrenergic tocolysis
Pulmonary embolism (thrombus, amniotic fluid, air)
Pneumothorax
Pneumomediastinum
Extra-pulmonary causes of upper airway obstruction
CNS disorders
Respiratory muscles weakness

Table-3: Guidelines for assessing withdrawal of mechanical ventilation.

Patient awake, alert, co-operative.
PaO2 > 60 mmHg with an FiO2 < .5
PEEP ≤ 5 cm H2O.
PaCO2 and PH acceptable.
Spontaneous tidal volume > 5 ml/Kg.
Vital capacity > 10 ml/Kg.
Minute Ventilation (MV) < 10 L/min.
Maximum Voluntary Ventilation double of MV.
Maximum Negative Inspiratory Pressure (MIP) > -25cm H_2O.
Respiratory rate < 30 breaths/min.
Static compliance > 30 ml/cm H2O.
Rapid shallow Breathing Index (RSBI) < 100.

ventilatory support are similar to non-pregnant asthmatic patients (table-3).

Follow up of the Case

Patient had an incisional hernia after operation for burst abdomen. She underwent mesh repair operation for inciscional hernia. She had uneventful recovery during this operation. She is at present asymptomatic and being examined fortnightly in out patients department.

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