A comparative study of arterial oxygen saturation in pregnant and non-pregnant women

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Abstract

Background: Pregnancy is a stressful condition with considerably altered physiological and metabolic functions. Several authors have reported that oxygen consumption increases by 30-40% during pregnancy. Maternal ventilation and blood gases undergo substantial changes in pregnancy. During pregnancy, the rib cage undergoes structural changes in response to progressive relaxation of the ligamentous attachments of rib increasing the subcostal angle from 68° to 103°. In pregnancy, the diaphragm is elevated by about 4 cm, and the lower ribcage circumference is increased by about 5 cm. Hence, the functional residual capacity (FRC) and residual volume are reduced in pregnancy. Decreased FRC with increased oxygen demand lowers oxygen reserve of the mother. The increased metabolic rate and low oxygen reservoir in the lung at end expiration make the pregnant women particularly susceptible to develop hypoxemia. The blood gas analyzers require blood sample from the patient, hence invasive and painful. Whereas, pulse oximetry is a non-invasive, inexpensive, bedside technique and an alternative for arterial blood gas measurement. There is conflicting evidence concerning arterial oxygen saturation during pregnancy, hence the present study was done.

Aims and Objectives: The aim of this study is to assess and compare the arterial oxygen saturation in pregnant and non-pregnant women.

Material and Methods: This study was done on 60 non-pregnant women and 60 pregnant women in each trimester of the age group 20-35 years. The arterial oxygen saturation was measured with pulse oximeter.

Result: The arterial oxygen saturation did not show any statistically significant difference in pregnant women in any trimester as compared to non-pregnant women.

Conclusion: The decreased FRC and increased oxygen demand does not affect the arterial oxygen saturation in pregnant women as progesterone mainly lowers the threshold of respiratory center and increases their sensitivity to carbon dioxide.

KeyWords: Oxygen Saturation; Pregnant Women; Pulse Oximeter

I. Introduction

Pregnancy is a stressful condition with considerably altered physiological and metabolic functions.[1] Several authors have reported that oxygen consumption increases by 30-40% during pregnancy.[2] The progressive rise is due primary to the metabolic needs of the fetus, uterus, and the placenta and secondarily to increased cardiac and respiratory work.[2] Maternal ventilation and blood gases

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undergo substantial changes in pregnancy.[3] During pregnancy, the rib cage undergoes structural changes in response to progressive relaxation of the ligamentous attachments of rib increasing the subcostal angle from 68° to 103°.[4] In pregnancy, the diaphragm is elevated by about 4 cm, and the lower rib cage circumference is increased by about 5 cm.[4] Hence, the functional residual capacity (FRC) and residual volume are reduced in pregnancy.[4]

Decreased FRC with increased oxygen demand lowers oxygen reserve of the mother.[5] The increased metabolic rate and low oxygen reservoir in the lung at end expiration make the pregnant women particularly susceptible to develop hypoxemia.[4] Nearly 70% of the healthy pregnant women are reported of having dyspnea or breathlessness during daily activity.[5] Pulse oximetry is a non-invasive method of measurement of arterial oxygen saturation for monitoring clinical patients.[6] Whereas, blood gas analyzers require blood sample from the patient, hence invasive and painful. Pulse oximetry is a non-invasive, inexpensive, bedside technique and an alternative for ABG measurement.[6] There is conflicting evidence concerning arterial oxygen saturation during pregnancy, hence the present study was done to assess the arterial oxygen saturation in pregnant women as compared to non-pregnant women using a pulse oximeter.

II. Material And Methods

This case control study was done in the outdoor patient Department of Obstetrics and Gynecology, Govt Maternity Hospital, Hyderabad. The study was done on 60 pregnant and 60 non-pregnant women of age group 20-35 years. Pregnant women were randomly selected from the females coming for the antenatal checkup. 60 pregnant females in each trimester were selected. The non-pregnant females were randomly selected from the relatives accompanying them.

III. Procedure

The arterial oxygen saturation percentage was measured using L and T planet 40 monitor with Nellcor pulse oximeter probe. The probe was connected to the index finger of the right hand in lying position and reading was taken.

Group 1: Cases (Pregnant Women)

Inclusion criteria
Healthy pregnant women, 20-35 years of age, without any complication were included in the study.

Exclusion criteria
The pregnant women with
- History of smoking or any addiction
- Anemia, asthma, pneumonia, or any other systemic illness
- Pregnancy-induced hypertension, diabetes, endocrinological pathology, and cardiovascular or respiratory illness
- Athlete.

The cases were divided into following three groups.
A1 - First trimester - 1-12 weeks of pregnancy
A2 - Second trimester - 13-28 weeks of pregnancy
A3 - Third trimester - 29-40 weeks of pregnancy.

Group 2: Control (Non-pregnant Women)

Inclusion criteria
Women of age 20-35 years in the follicular phase of menstrual cycle where the hormone variations are not influenced by progesterone were included in the study.
Exclusion criteria

- Women on oral contraceptive pills or any medication that can alter cardiovascular or endocrine function
- Lactating women
- Women within 2 years of post-partum
- Women with any systemic illness
- Athlete or women doing regular exercise
- Women with mehendi on hand or nail paint

All the ethical viewpoints were considered and a written informed consent was obtained from each subject after explaining the procedure and study in detail. The statistical analysis of the data was performed by unpaired $t$-test.[7]

IV. Results

The arterial oxygen saturation did not show any statistically significant difference in pregnant women in any trimester as compared to the non-pregnant women (Tables 1 and 2).

V. Discussion

In the present study, it was observed that the arterial oxygen saturation did not show any statistically significant difference in pregnant women in any trimester as compared to the non-pregnant women (Table 2).

<table>
<thead>
<tr>
<th>Groups</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 cases pregnant</td>
<td></td>
</tr>
<tr>
<td>First trimester A1</td>
<td>97.1±1.34</td>
</tr>
<tr>
<td>Second trimester A2</td>
<td>97.78±1.30</td>
</tr>
<tr>
<td>Third trimester A3</td>
<td>97.83±1.08</td>
</tr>
<tr>
<td>Group 2 (control) non pregnant</td>
<td></td>
</tr>
<tr>
<td>Non pregnant</td>
<td>90.13±1.05</td>
</tr>
</tbody>
</table>

SD: Standard deviation

Table 1: Mean of the arterial saturation of oxygen in percentage in both groups
Table 2: Comparison of the arterial saturation of oxygen (percentage) in pregnant and non-pregnant women

<table>
<thead>
<tr>
<th>Significance</th>
<th>Comparative groups</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 (first trimester) and Group 2 (non-pregnant)</td>
<td>0.053</td>
<td>Not significant</td>
</tr>
<tr>
<td>A2 (second trimester) and Group 2 (non-pregnant)</td>
<td>0.1073</td>
<td>Not significant</td>
</tr>
<tr>
<td>A3 (third trimester) and Group 3 (non-pregnant)</td>
<td>0.1247</td>
<td>Not significant</td>
</tr>
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</table>

This is in contrary to the observation of Sunyal et al.[8] They observed that the percentage saturation of oxygen increased gradually from the first to the third trimester of pregnancy. The value of percentage saturation of oxygen was statistically significantly increased in all the three trimesters of pregnancy as compared to the non-pregnant women.[8] Bourne et al. found that the pregnant women suffer from significant prolonged nocturnal hypoxemia (desaturation).[9] The increased oxygen demand and decreased FRC may lead to hypoxemia in pregnant women.[4] However, we observed no significant change in arterial oxygen saturation in pregnant women as compared to non-pregnant women. This may be due to the increased tidal volume as the apposition of diaphragm to the chest wall is increased in pregnancy.[4] Increased tidal volume increases the resting minute ventilation, and thereby the alveolar ventilation also increases.[4] Moreover, the dead space may be decreased in pregnancy because of increased cardiac output and better perfusion in the apices, so the ratio of dead space volume to the tidal volume becomes even more advantageous.[4] Lung compliance remains unaffected by pregnancy.[4]

There is evidence suggesting that progesterone is a respiratory stimulant.[10] The administration of exogenous progesterone in males caused increased minute ventilation and sensitivity to carbon dioxide.[10] Progesterone decreases the threshold and increases the sensitivity of the central ventilator chemoreflex response to carbon dioxide.[10] Moreover, progesterone gradually increases during pregnancy from 25 ng/ml at 6 weeks to 150 ng/ml at 37 weeks of gestation.[5] Moreover, increase in metabolic demands of the fetus, uterus, and the maternal organ causes increased oxygen consumption and production of carbon dioxide.[10] Progesterone also stimulates central neural sites in the medulla oblongata, thalamus, and hypothalamus which control ventilation. Progesterone directly increases the peripheral ventilator response of carotid body to hypoxia.[10] Thus, the mechanical effects of gravid uterus cause relatively little change in pulmonary mechanics. All the changes are mediated by the high progesterone level influencing respiratory center through lowering the threshold and increasing the sensitivity. However, the limitation of the study is that a detailed spirometry analysis of the subjects was not done.

VI. Conclusion

There is no significant change in arterial saturation of oxygen in pregnant women as compared to non-pregnant women. Thus, the increased oxygen demand and a decrease in FRC does
not affect the arterial oxygen saturation in physically fit pregnant women due to hormone-mediated changes but may be prone to hypoxemia in stress.

References


