PLASMA TRANSFERRIN IN SINGAPORE WOMEN DURING PREGNANCY

By L. L. Chang, K. Chua, Y. C. Yong and R. Sivasamboo

SYNOPSIS

The plasma transferrin, measured by radial immunodiffusion, increases during pregnancy. This falls to below non-pregnant levels during the puerperium. Prophylactic iron therapy during pregnancy was able to maintain a satisfactory level of Hb, PCV and plasma iron but was unable to abolish the rise in plasma iron-binding capacity.

The total iron-binding capacity of serum (TIBC) has been observed to rise during the course of pregnancy (Fay et al, 1949; Morgan and Carter, 1960; Carr, 1971). This rise in TIBC, coupled with a lowered percentage saturation of transferrin and a fall in hemoglobin concentration, has given rise to the concept of the "physiological anemia of pregnancy." Many of the anemias in pregnancy, are however not physiological, but due to iron, folate or Vit B12 deficiencies. The administration of therapeutic doses of iron has been shown to bring the hemoglobin level back to non-pregnant levels but has not been able to prevent the rise in TIBC (Sturgeon, 1959; Morgan, 1961, Hancock et al, 1968). A similar increase in TIBC occurs during the use of oral contraceptives (Burton, 1967) and in both instances is probably due to endocrine factors.

This study was done to find out the changes in plasma transferrin, during normal pregnancy. Hb, PCV and plasma iron estimations were also carried out at the same time.

MATERIAL AND METHODS

The subjects were all Chinese women attending the antenatal clinic at Thomson Road General Hospital. The majority were primigravidae and all were supplied with supplemental iron and vitamins from their first antenatal visit.

Blood samples were taken by venepuncture in 26 non-pregnant women, 51 women at various stages in their pregnancy and 25 women at their first postnatal visit 6 weeks after delivery.

Plasma transferrin was measured by radial immunodiffusion. (Mancini et al, 1965). TIBC was calculated from the plasma transferrin concentration by multiplying with the factor 1.25 (Surgenor et al, 1949).

Hemoglobin was estimated by the cyanmethemoglobin method (Van Kampen et al, 1961). PCV readings were made using heparinised microhematocrit tubes (Strumia et al, 1954). Plasma iron was measured using bathophenanthroline sulphonate (Henry et al, 1958).

RESULTS

A summary of the results obtained in this study is shown in Table I.

The plasma transferrin level rises significantly from the non-pregnant level of 256.4 mg./100 ml. to 376.3 mg./100 ml. in the third trimester. (p<0.001). After delivery the plasma transferrin falls to a mean of 194.0 mg./100 ml. at the end of 6 weeks. The difference between this and the non-pregnant level is significant. (p<0.001).

Plasma iron levels fluctuate during pregnancy. The difference between means is not significant.

The percentage saturation of transferrin falls from 34.5% in the non-pregnant group to 26.8% in the third trimester. (p<0.05). It rises during the puerperium to 49.2% (p<0.001).

The Hb concentration in the third trimester is not significantly different from the non-pregnant levels of 14.9 gm./100 ml. During the puerperium the Hb level rose to 15.6 gm./100 ml. (p<0.01).

The PCV fell throughout pregnancy. The difference between the means at the third trimester and at the puerperium when compared to the non-pregnant group was significant. (p<0.01).

DISCUSSION

Under normal circumstances, all the iron in the plasma is carried bound to a β1 globulin, transferrin. The β globulin fraction of plasma proteins
TABLE 1
PLASMA TRANSFERRIN, TIBC, PLASMA IRON, % SATURATION OF TRANSFERRIN, Hb AND PCV IN PREGNANT AND NON-PREGNANT WOMEN IN SINGAPORE

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Women</th>
<th>Plasma Transferrin (mg./100 ml.)</th>
<th>TIBC (ug./100 ml.)</th>
<th>Mean ± 1 S.D. Plasma Iron (ug./100 ml.)</th>
<th>% Saturation of Transferrin</th>
<th>Hb (gm./100 ml.)</th>
<th>PCV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>26</td>
<td>256.4 ±39.8</td>
<td>311.7 ±80.7</td>
<td>108.6 ±35.3</td>
<td>34.5 ±11.4</td>
<td>14.9 ±8.6</td>
<td>42.9 ±1.95</td>
</tr>
<tr>
<td>Pregnant First trimester</td>
<td>9</td>
<td>215.7 ±48.3</td>
<td>269.7 ±60.3</td>
<td>133.4 ±26.4</td>
<td>51.3 ±13.7</td>
<td>15.2 ±3.2</td>
<td>40.2 ±2.2</td>
</tr>
<tr>
<td>Second trimester</td>
<td>8</td>
<td>258.1 ±30.1</td>
<td>322.6 ±37.7</td>
<td>115.8 ±28.1</td>
<td>36.8 ±11.0</td>
<td>15.8 ±2.8</td>
<td>36.4 ±2.8</td>
</tr>
<tr>
<td>Third trimester</td>
<td>34</td>
<td>376.3 ±84.3</td>
<td>470.4 ±105.4</td>
<td>121.7 ±39.4</td>
<td>26.8 ±5.8</td>
<td>14.4 ±3.0</td>
<td>40.6 ±3.0</td>
</tr>
<tr>
<td>Significance vs control</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
<td></td>
<td>Not Significant</td>
<td>p&lt;0.05</td>
<td>Not Significant</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Postnatal</td>
<td>25</td>
<td>194.0 ±24.4</td>
<td>242.6 ±30.5</td>
<td>116.9 ±33.7</td>
<td>49.2 ±15.2</td>
<td>15.6 ±1.3</td>
<td>41.2 ±2.2</td>
</tr>
<tr>
<td>Significance vs control</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
<td></td>
<td>Not Significant</td>
<td>p&lt;0.001</td>
<td>p&lt;0.05</td>
<td>p&lt;0.01</td>
</tr>
</tbody>
</table>

has been shown to rise conspicuously during pregnancy. This is in contrast to plasma albumin which falls during pregnancy (MacGillirray and Torey, 1957). Transferrin levels have been shown in this study to increase considerably during the third trimester of pregnancy. The magnitude of the rise is of the same order as that shown by Ventura and Klopper (1951), using TIBC as a measure of the amount of transferrin in the plasma. The rise in plasma transferrin occurs later in the course of pregnancy in this study compared to others (Morgan, 1961; Jacobi et al, 1969). It is likely that plasma transferrin rises earlier during pregnancy in those with concomitant iron deficiency (Hancock et al, 1968). The administration of supplemental iron in this series has probably contributed to the later rise in plasma transferrin.

The functional significance of the increase in carrier protein, transferrin, and therefore TIBC is uncertain. It may be a consequence of increased oestrogens during pregnancy stimulating the rise in serum-binding capacities, not only of iron but also of hormones like thyroxine (Dowling et al, 1960) and corticosteroids (Klopper and Billewicz, 1963). The use of oral contraceptives has been shown to result in similar increases in plasma transferrin (Jacobi et al, 1969) and TIBC (Burton, 1967). It has been suggested by Fletcher and Huehns (1968) that raised plasma transferrin levels may result in increased iron absorption. Laurell (1947) proposes that the rise in iron-binding capacity of plasma serves the useful purpose of enhancing mobilization of iron from maternal stores thus enabling easier transport of iron to the foetus. The fall in plasma transferrin seen in the puerperium could conversely reflect the conservation of iron now occurring in the maternal organism.

The changes in Hb and PCV during the course of pregnancy as seen in this study are similar to those reported previously in Singapore (Kwa and Ko, 1968). The fall in Hb and PCV during the second trimester is due to relative hemodilution because at this period the rise in plasma volume exceeds that of the red cell mass. The actual values obtained in this series are higher than those reported by Kwa and Ko (1968). The possible reasons for the difference is the improved nutritional status of women in Singapore, the greater awareness and hence more prevalent use of prophylactic iron therapy during pregnancy and the selectiveness of this study which was mainly on primigravidae.

REFERENCES