A STUDY OF SERUM ENZYME—ALDOLASE, ACTIVITY DURING PREGNANCY

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Introduction.

A substantial role is played by the inmacellular concentration of hexosephosphate in the regulation of glycolysis during early embryogenesis. The level is maintained not only by the activity of enzyme of glycolytic sequence but also by those of hexose-monophosphates (HMP) shunt, as well as by glyconeogenesis (Yurovitskii and Milman 1968).

Aldolase (ALD) is an important enzyme in glycolytic pathway. It splits the hexose-phosphate into triose-phosphate and 2 m-olecules of ATP are generated at the stage for per molecule of glucose (Harper 1971). This aspect of carbohydrate metabolism is not much studied, especially in reference to its relation with feto-placental unit. In the pre-

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sent study serum ALD is studied in relation to feto-placental unit.

Material and Method

A total of 122 estimations were carried out on 83 women attending outpatient and inpatient of Obstetrics & Gynaecology section of J.N. Medical College Hospital. It consisted of 20 normal healthy nonpregnant women, 43 normal pregnant women and 20 women with pregnancy disorders.

Four c.c. of venous blood was taken, serum were separated with centrifuge and stored at—4°C until used. The estimation was carried out within 24-48 hours by method of Sibley and Lehninger (1949) with 0.05 M fructose-1-6 diphosphate as substrate and O.IM trishydrate buffer at pH 8.6. All the data were subjected to student 't' test and significance of result seen at P = 0.05 level.

Result

The normal range of serum ALD were 4.12 ± 0.388 unit (The normal range reported in literature 2-9.6 S.L. unit (1.2-7.2 IU) and 3.2 ± 0.4 IU by Sibley and Lehninger (1949) and Wilkinson (1962) respectively.

Tables I and II show the mean value of serum ALD activity in non-pregnant and during pregnancy in various trimesters.

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TABLE I

| Comparison of Mean | Values of Serum | -Aldolase in | Non-pregnant | and | Normal | Pregnancy in |
|----------------------|-----------------|---------------|--------------|-----|--------|--------------|
| TAXABLE IN THE OWNER | Differe | nt Trimesters | of Gestation | | | |

| | | and the second s | the second second s | a to i a day was a second as a to | a management a series | the second se |
|---|----------------------------|--|---------------------|-----------------------------------|-----------------------|---|
| | No. of Estima- tions | Mean Value | SD | t | df | Statistical Signi- ficance in Group |
| Non-pregnant | 20 | 4.12 | 0.388 | | - | - |
| Normal Pregnancy 1st Trimester | 4 | 6.065 | 0.796 | 7.0985 | 22 | Significantly high- er in pregnancy in c.f. of non-preg- nant |
| lInd Trimester | 22 | 7.77 | 0.596 | 4.778 | 24 | SH in IInd tri- mester c.f. of Ist trimester |
| IIIrd Frimester | 50 | 10.66 | 1.91 | 6.876 | 70 | SH in IIIrd tri- mester in c.f. of IInd trimester |

Note: SH = Significant Higher.

TABLE II

| iconging. | Inces | Serum | Aldolase Acti | vity at Various | Weeks of | Gestation | |
|---|-----------------------------|-------|---------------|-------------------------------------|----------|-----------|-----------------------------|
| Duration of Gesta- tion in weeks | No. Esti- ma- tion | Mean | SD | Compa- rison between weeks | t | df | Statistical Significance |
| 8 | 1 | 5.062 | - | - | - | | |
| 10 | 1 | 6.000 | | - | - | - | - |
| 12 | 1 | 6.156 | | - | - | - | - |
| 14 | 3 | 6.800 | ±.173 | - | - | - | |
| 16 | 2 | 7.150 | - | - | | | |
| 18 | 3 | 7.500 | ±.0 | - | | - | _ |
| 20 | 3 | 7.93 | ±.115 | | - | - | - |
| 22 | 2 | 7.7 | ±.36 | | | - | the same manual in |
| 24 | 2 | 7.73 | ±1.025 | - | | - | |
| 26 | 8 | 8.3 | ±.345 | | | - | - |
| 28 | 6 | 9.08 | ±.376 | 26-28 | 3.732 | 12 | SH in 28 weeks |
| 30 | 3 | 10 | 0 | 28-30 | 3.755 | 7 | SH in 30 weeks |
| 32 | 11 | 9.8 | ±0.678 | 30-32 | 0.476 | 12 | ISL in 32 weeks |
| 34 | 9 | 10.37 | ±.564 | 32-34 | 1.919 | 18 | SH in 34 weeks |
| 36 | 5 | 11.55 | ±.447 | 34-36 | 3.741 | 12 | SH in 36 weeks |
| 38 | 8 | 11.65 | ±.708 | 36-38 | 0.26 | 11 | ISH in 38 weeks |
| 40 | 8 | 12.06 | ±.76 | 38-40 | 1.044 | 14 | SH in 40 weeks |

N.B.: SH = Significantly higher ISL = Insignificant lower ISH = Insignificant higher

Significant increase in serum-ALD activity was observed during pregnancy and the activity increased progressively with advancement of pregnancy.

The cases of pregnancy disorders consisted of 8 cases toxaemia, 11 cases of high risk pregnancy and one case of IUD. The mean serum ALD-activity and detailed clinical data are shown in Tables III and IV respectively. There is significant increase in serum ALD activity both in toxaemia of pregnancy and high risk group. The activity in one case of IUD was almost that of non-pregnanct woman.

Discussion

The normal range of serum ALD activity in non-pregnant women reported in literature is 2.0 to 9.6 unit (1.2-7-2 IU), 3.2 ± 0.4 IU by Sibley and Lehninger (1949) and Wilkinson (1962) respectively. In present study of control group the serum ALD activity closely corresponded to them i.e. 4.12 ± 0.388 S.L. unit. The serum ALD activity reported by Sandhoo and Amma (1974) is lower than reported i.e. 1.14 ± 0.42 unit.

Serum ALD activity significantly increases during pregnancy. Increase aldolase activity was observed in pregnant rats uterus as compared with non-pregnant uterus by Lolli, et al (1959). They further observed that enzyme activity progressively increased during gestation and was maximum during labour and gradually decreased after delivery. The rise in aldolase activity may be as a result of increased glycolysis during embryogenesis, both through enzymes of glycolytic sequence and that of HMP shunt (Yurovitskii and Milman, 1968).

Increased aldolase activity was observed during labour and early puerperium by Lanza (1968). Onnis et al (1962) observed that aldolase is localized more abundantely in the epithelial tissue of female genital tract: i.e. wall of graffian follicle, glands of endometrium, cervical epithelium, basal vaginal epithelium, than in connective tissue. Similarly, more in placental amniotic epithelium than in extraplacental and less in connective tissue of placentae. Oestriol diproprionate injected into sexually immature rats increases uterine aldolase. Sandhoo and Amma (1974) did not find any change in serum aldolase activity in normal pregnancy but 75% increase in activity were observed in pathological pregnancy such as toxaemia (Shub and Smilgkalje, 1962;

| ************* | No. of Estima- | Mean Value | | No. of | 35 | | Statistical | | |
|---------------|-------------------|---------------|-------|-----------------|-------|--------|--|-------|----|
| | tion | value | SD | Esti- mation | Mean | SD | Compa- rison between | t | df |
| II | 22 | 7.77 | ±.596 | 4 | 23.75 | ±5.315 | Normal & 1 Essential Hyper- tension | 3.119 | 24 |
| HI 6 | 50 | 10.66 | ±1.91 | 10 | 20.12 | ±5.25 | Normal & Toxaemia | 9.732 | 58 |

TABLE III Comparison of S. Aldolase Value in Normal Premancy and Torgemin of Pres

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| TABLE IV | |
|----------|--|
| | |

| Serum Aldolase | Activity and | Clinical I | Findings in | Pregnancy | Disordered | Group |
|----------------|--------------|------------|-------------|-----------|------------|-------|
|----------------|--------------|------------|-------------|-----------|------------|-------|

| Type of | No. of | Age | Parity | Trimester of Pregnancy | | | Preg- | |
|----------------------------------|----------|--------|----------|------------------------|-----------|---------|------------------|--------------------------|
| Disorder | cases | (Yrs.) | | I | П | III | nancy outcome | Remarks |
| Postmaturity | 2 | 18 | P0+0 | - | | 18.3 | ND | |
| | | 30 | P3+0 | | - | 18.0 | ND | |
| Hydramnios | 1 | 20 | P1+0 | - | 10 | 14.0 | ND | |
| Bad obstetric | | | | | | | | |
| History (O.H.) | | | | | | | | |
| Previous 2 Still birth (S.B.) | 1 | 20 | P2+0 | | | 12.4 | NND | FH 28 Wks |
| and the second second | | | | | | 16.0 | (3rd | FH 32 |
| I manufacture a | | | | | | | day) | |
| Unexplained (S.B.) | 1 | 20 | P0+0 | | | 17.0 | Fresh | FH 32 Wks. |
| | No. | | - Cilmer | | | 20.0 | Still | FH 36 Wks |
| Intrauterine | | | | | | | birth | |
| retarded growth | 1 | 20 | P1+0 | | 11 - 11 - | 24.0 | | 32/28 FH |
| | | | | | | | | 30/28 FH |
| mail - window ?! | | | | | | 26.0 | N.D. | 36/34 FH I.V. Glucose |
| in ' cetterite | | | | | | | | Complamina |
| Habitual | | | | | | | | given |
| abortion | 3 | 33 | P4+3 | | 1.2 | 13.44 | N.D. | Prolutron |
| | pand one | 24 | P3+5 | | 1 - 01 | 16.00 | N.D. | depot |
| | | 18 | P0+3 | 5-0 | 6-0 | | N.R. | given |
| Threatened | | | | | 7.05 | | | |
| abortion | 2 | 30 | P2+0 | | 12.45 | and all | N.D. | |
| Technication | | 18 | P0+0 | 14.7 | - 11 | | | |
| Intrauterine death (IUD) | 1 | 28 | P2+0 | | | 3.44 | IUD | FH 20 Wks. |
| (102) | | 20 | F2+0 | | amonto; | 3.94 | TOD | FH 20 WKS. |
| Mean Value S.D. | | | | 9.8 | 5 9.46 | 14.45 | | |

N.B.: ND = Normal delivery FH = Fundal height

NND = Neonatal death

(Camurri et al, 1962) and in initial stages of infective hepatitis (Hornik and Kowalezyk, 1962). In present study group of pathological pregnancy (Table III & IV) increased serum aldolase activity was observed, mean values were 9.85, 9.46 \pm

Crisp et al, 1959), cardiac patients 2.11, 14.45 \pm 3.5 in first, second and third trimesters respectively (Table IV) except in 1 case of IUD where serum aldolase activity was 3.44 S.L. unit, equal to that of non-pregnant level. This observation further potentiates that during pregnancy more aldolase is required for glycolysis from active placental tissue which is absent in IUD.

Crisp et al (1959) (Hernandez and Aleantara and Perez-Sandoval; 1966) Furjaro et al (1968), Halbrecht et al (1968) and Didenko (1969) have related the serum aldolase activity in toxaemia to degree of liver involvement. The increased serum aldolase activity in pathological pregnancy may be as a result of added placental tissue damage due to premature ageing of placenta.

Summary

A total of 83 patients were studied including 20 normal non-pregnant, 43 normal pregnant and 20 of pregnancy disorder.

The serum aldolase (SADL) activity in non-pregnant women was in the range of 4.12 ± 0.388 unit. The SADL activity was found to be increased significantly during pregnancy and linear rise was observed with advancement of pregnancy. In group of pregnancy disorder the activity was further increased significantly. The increase of SADL activity was possibly due to increase in glycolysis for embryogenesis both through glycolytic pathway and HMP shunt.

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