

ERYTHROCYTE AND PLASMA CHOLINESTERASE ACTIVITY IN NORMAL PREGNANCY

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Abstract : Erythrocyte acetylcholinesterase and plasma cholinesterase was estimated in 50 normal pregnant women and 22 age matched normal non-pregnant women. Plasma cholinesterase was significantly decreased while erythrocyte cholinesterase was significantly increased during pregnancy. These changes may be related to altered haemodynamics and or other inter-related changes occurring in pregnancy.

Key words: pregnancy erythrocyte acetyl cholinesterase plasma cholinesterase

INTRODUCTION

Cholinesterases are an interesting group of enzymes which hydrolyse acetylcholine (Ach) and other choline esters. Augustinsson (1) classified them into two groups. Group I: Acetylcholinesterase (AchE) or true cholinesterase which is highly specific and hydrolyses only Ach. They are mainly found in the brain, motor end plates, muscles and erythrocytes. Group II: Pseudocholinesterase (PchE) occurring mainly in serum/plasma. These are eserine sensitive esterases and hydrolyse in addition to Ach other esters like Succinylcholine, Butyrylcholine, Procaine etc.

Both AchE and PchE concentrations can be influenced by a multiplicity of factors - physiological, nutritional and pathological. A decrease in the PchE activity in pregnancy is fairly well documented (2-6). PchE is formed by the liver and pregnancy is known to modify liver function, probably so, most of the research work in relation to ChE and pregnancy has been limited to the estimation of only PchE. Very little is known about the effect of PchE. Very little is known about the effect of pregnancy on erythrocyte AchE. The function of erythrocyte AchE is not clear. It is implicated as a protective agent against haemolysis (7) and shown to be high in young erythrocytes (8). Pregnancy alters haemodynamics also and therefore it was considered worthwhile to study the erythrocyte AchE levels in pregnancy, in comparison to that of PchE.

METHODS

Erythrocyte AchE and PchE activities were determined in 50 normal pregnant women who attended the antenatal clinic of St. John's Medical College Hospital, Lady Curzon & Bowring Hospital and a Rural Health Centre in Bangalore. Subjects were considered 'normal' if they were free of hypertension, oedema, unusual weight gain, significant albuminuria or any concurrent organic disease and if their haematocrit values were within normal limits.

10 ml venous blood samples were collected from the antecubital vein, using heparin as the anticoagulant. 5 ml was used for routine haematology and the other 5 ml for cholinesterase estimations. The latter was centrifuged for separation of plasma from red cells and were then stored at -5°C. Erythrocyte AchE and PchE levels were estimated within 24 hrs, by the method of Ellman et al (9) with minor modifications (10).

The activity was expressed in units: One unit of AchE activity being equivalent to one micromole of thiocholine liberated in 1 min from 1 g of haemoglobin; and one unit of PchE activity being equivalent to one micromole of thiocholine liberated in 1 min from 1 ml of plasma.

22 non-pregnant normal women of the same age served as the controls.

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degenerating trophoblasts, corpora lutea, atretic follicles and vaginal bleeding. The ovarian histology of the animals treated with 100 mg of chloroform extract showed degenerating corpus lutea and some atretic follicle. Uterus remained thick, long and empty in appearance with normal endometrium.

No toxic effect was observed either by naked eye appearance or by histological studies at these dose levels. After discontinuation of the extract treatment, six of the laparotomised animals in each group were mated, resulted in pregnancy and normal litter size indicating that the extracts action was reversible.

DISCUSSION

Hafez has described that in mouse, corpora lutea persist during the period of gestation and are the only source of progesterone(7). Histological observation of the ovaries of the animals treated with 100 mg/kg dose of both the extract indicated the possibility that resorption might be due to a change in progesterone level as shown by degenerated corpus luteum. It is well established that the inhibition of implantation in albino rats is due to

imbalance in the progesterone estrogen ratio. Further work is in progress to pin point the mode of action and to elucidate the mechanism of antifertility action of these extracts by studying antizygotic, blastocystotoxic, anti-implantation activity, by giving the extracts during different periods of gestation as described by Hafez(7). Research on Indian plants with antifertility activity has been exhaustively reviewed recently by Satyavati(8).

Further work is also in progress to screen the alcoholic and aqueous extracts of the plant for their antifertility activity.

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RESULTS

The erythrocyte AChE and PChE and levels in 50 pregnant and 22 non-pregnant normal women are shown in Table I. PChE activity was decreased in pregnancy, a significant and steady decline occurring from II trimester to III trimester. Erythrocyte AChE activity on the other hand was significantly increased during pregnancy. There was no significantly increased during pregnancy. There was no significant difference between the levels in II and III trimesters.

TABLE I : Plasma cholinesterase and Erythrocyte cholinesterase levels (I.U.) in pregnant and non-pregnant normal women.

	(n)	PchE Mean±S.E.	AchE Mean±S.E.
<i>Pregnant Women</i>			
— Second Trimester	19	7.2±0.2*	71±2*
— Third Trimester	29	5.9±0.3*+	74±3**
<i>Non-pregnant women</i>			
	22	9.7±0.02	64±2

*P < 0.001 as compared to control

+P < 0.05 as compared to second trimester

**P < 0.02 as compared to control

DISCUSSION

Results of the present study are in conformity with the earlier reports of a decrease in PChE in pregnancy except that the gestational period at which the concentration is lowest differ between investigators. We found a steady decline from II to III trimesters. (We could not estimate levels in I trimester as in India most people do not report to the hospital in the 1st trimester). Areekal and Srichairat (11) found lowest levels during first trimester, while Pritchard (4) found decreased PChE activity mainly in the III trimester. Others (2,6) report a decline in serum activity in the I trimester, the level remaining stable during II and III trimester. In contrast Howard et al (3) found a fall during the 1st trimester with a return to normal prepregnancy levels later in pregnancy.

Unlike PchE, erythrocyte AchE levels significantly increased during pregnancy, but there was no difference between the levels in II and III trimesters. Pritchard and Weisman (12) have reported a slight but persistent increase in erythrocyte AchE as pregnancy advanced, however, Areekal and Srichairat (11) did

not find any alteration in erythrocyte AchE in pregnant women.

The reduced PChE activity in pregnancy has been attributed to haemodilution, altered hepatic function and anticholinesterase activity of oestrogen (4, 6) Its clinical significance, however, is that it can modify the response of the body to certain drugs, eg : with succinylcholine, where a prolonged apnoea may occur in pregnant patients undergoing surgery (13). Drugs like quinidine are known to cause a further decrease in levels of PchE in pregnant women (14).

The significance of an increase in erythrocyte AchE in pregnancy is not clear. Young red cells contain more AchE activity than do older erythrocytes (8). In normal pregnancy there is an increase in plasma volume by about 25-35% - therefore in non-anaemic pregnant patients the rate of erythrocyte production may exceed erythrocyte destruction, thus a greater population of circulating red cells may have been released more recently (young cells) from the bone marrow in normal pregnant individuals (15, 16).

The AchE in erythrocytes is situated on the cell membrane, changes in its content therefore might indicate the effect of the modified physiological status produced by pregnancy on the metabolism of the red cell membrane - a biochemical alteration.

The pathophysiological implications of our findings, viz. a decrease in PChE with an increase in AchE in pregnancy cannot be easily explained particularly because the metabolic function of AchE in the red cells is still uncertain. Whatever be the mechanism and the pathophysiological implication, what is significant is that a change in the physiological status as occurs in pregnancy namely, altered hepatic function, altered hormonal status, altered haemodynamics and/or other multiple inter-related factors are able to modify red cell AchE and PChE.

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