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Clinical significance of human chorionic somatomammotrophin (hCS) plasma levels in prolonged pregnancy

by
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INTRODUCTION

Since the discovery of hCS ([1]) and the establishment of acceptable and reproducible radio-immunological determination ([2,3]), a number of authors have carried out investigations on the efficacy of hCS as an index of placental function ([4,5,6]).

The absence of circadian rhythm ([5]), the significant correlation of foetal and placental weight ([7,8,9,10]), the brief plasma half-life ([11]) and the absence of wide fluctuations in the maternal blood in samples obtained at short intervals of time ([12]) have made of hCS an excellent parameter for foetoplacental monitoring in both physiological ([5,13,14,15,16,17]) and pathological pregnancy ([18,19,20,21,22,23,24,25]).

The object of our research was to study plasma levels of hCS protracted pregnancy. Under these conditions postmaturity lowers placental function and may lead to foetal involvement and death.

The purpose of this study was to see whether, by comparing the levels of hCS in protracted pregnancy with those of pregnancy at term, any variations might exist that would enable foetoplacental postmaturity to be diagnosed.

MATERIAL AND METHODS

We examined 76 pregnant women between the 38th and 43rd week. These patients were not in labour and those in the 41st week and beyond presented with clinical symptoms of protracted pregnancy.

A sample of blood was obtained from the cubital vein of all the patients at 8 a.m.; the heparinized blood was immediately centrifuged and the blood was deep-frozen at $-20°C$.

The hCS in each sample was analysed by a radio-immunological method, using CEA-IRE-SORIN kits.

The statistical analysis of the results was done by Student's 't' test.

RESULTS

At the 38th week (13 cases) the plasma values of hCS (mean $\pm$ standard error) were 7.73 ± 0.29 μg/ml, at the 39th week (15 cases), 8.36 ± 0.45 μg/ml and at the 40th week (10 cases), 9.08 ± 0.62 μg/ml, which is in agreement with what other authors have found ([18]).

At the 41th week (8 cases) the values for hCS were 6.29 ± 0.71 μg/ml, at the 42nd week (17 cases), 7.01 ± 0.52 μg/ml and at the 43rd week (13 cases), 6.68 ± 0.62 μg/ml.

The mean of the values for hCS in the 39th and 40th week (25 cases) was

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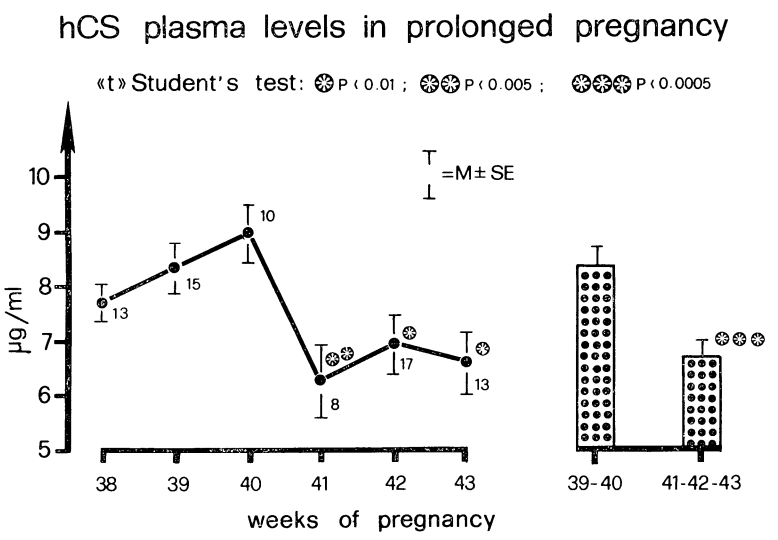

FIG. 1 - Plasma levels of hCS from 38th to 43rd week of pregnancy. Statistical analysis shows a significant difference between the levels for the 40th week and those for the 41st, 42nd and 43rd weeks. The difference between the means of the values of hCS at term (39-40) and those for values beyond term (41, 42, 43) are highly significant.

8.44 ± 0.33 µg/ml and that of the values in the 41st, 42nd, 43rd weeks (38 cases) was 6.75 ± 0.35 µg/ml (Fig. 1).

The mean weight of the neonates at term was 3440 ± 265 g, and that of the neonates beyond term was 3569 ± 315 g. The neonates, apart from clear signs of postmaturity, did not present at birth with any signs of acute illness.

Statistical analysis of the results demonstrated a significant fall in the plasma values of hCS in the 41st ($p < 0.005$), 42nd ($p < 0.01$) and 3rd weeks ($p < 0.01$) as compared with the results for the 40th week.

The mean of values for the pregnancies at the 39th and 40th weeks was significantly higher than that for the values at the 41st, 42nd and 43rd weeks ($p < 0.0005$).

No significant difference was found between the weights and the Apgar scores of the infants at term as compared with those beyond term.

DISCUSSION

Research so far carried out indicates that hCS faithfully reflects the functional activity of the placenta. Experiments on monkeys ([26]) have proved that injury caused to the foetus is not accompanied by a decrease of hCS in the plasma; but this fenomenon is found when injury is caused to the placenta. This confirms that a phatological condition in the foetus cannot always be found when hCS is analysed; Saxena *et al.* ([6]) claim that a decrease of hCS levels to 50% indicates serious foetal involvement; on the contrary, Spencer *et al.* ([27]) attribute no clinical significance to this.

In protracted pregnancy the involutionary processes also affect the placenta, giving rise to disseminated thrombosis in the vessels; this leads to a diminution of secretive tissue with consequent lowering of the plasma levels of hCS.

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Genazzani *et al.* ([28]) and Varma *et al.* ([22]) found low levels of hCS in protracted pregnancy, while Seppala ([20]) and Spencer ([27]) found normal levels; Genazzani *et al.* ([23]) saw that the diminution was more significant when there was also some foetal involvement.

Our results show that secretion of hCS in protracted pregnancy shows a significant diminution; this agrees with what Genazzani and Varma have said.

This enables us to state that the determination of hCS is a means for the early diagnosis of placental postmaturity, even before the establishment of the complications that this syndrome produces upon the vitality of the foetus.

SUMMARY

We examined 76 pregnant women between the 38th and 43rd week of pregnancy. The patients beyond the 41st week presented with clinical symptoms of protracted pregnancy and the neonates had clear signs of postmaturity.

We carried out an analysis of hCS in the plasma and found a significant diminution in the levels of hCS in protracted pregnancy, as compared with the values for pregnancy at term.

Involutionary phenomena occurred in protracted pregnancy, affecting the placenta and diminishing the tissue that secretes hCS.

The determination of hCS in the plasma thus facilitates the early diagnosis of placental postmaturity, before this has an effected on the life of the foetus, with consequent foetal illness or death.

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BIBLIOGRAPHY

1. Josimovich J. B. and MacLaren J. A.: *Endocrinology, 71,* 209, 1972. - 2. Grumbach M. M. and Kaplan S. L.: *Trans. N. Y. Acad. Sci., 27,* 167, 1964. - 3. Kaplan S. L. and Grumbach M. M.: *Science, 47,* 751, 1962. - 4. Beck P. and Daughaday W. H.: *J. Clin. Invest., 46,* 103, 1967. - 5. Grumbach M. M., Kaplan S. L., Sciarra J. J. and Burr I. M.: *Ann. N. Y. Acad. Sci., 148,* 501, 1968. - 6. Saxena B. N., Emerson K. Jr., and Selenkow H. A.: *New Engl. J. Med., 281,* 225, 1969. - 7. Spellacy W. N., Carlson K. L. and Birk S. A.: *J. Obst. Gyn., 95,* 118, 1966. - 8. Sciarra J. J., Sherwood L. M., Varama A. A., and Lundeberg W. B.: *Am. J. Obst. Gyn., 101,* 413 1968. - 9. Cramer D. V., Beck P., Makowsky E. L.: *Am. J. Obst. Gyn., 109,* 649, 1971. - 10. Letchworth A. T., Boardman R. J., Bristow C.: *J. Obst. Gyn. Brit. Cwth., 78,* 542, 1971. - 11. Spellacy W. N., Carlson K. L., Birk S. A.: *Am. J. Obst. Gyn., 96,* 1164, 1966. - 12. Genazzani A. R., Nasi A., Medda F., Facchinetti F., De Murtas M., Parrini D., D'Antona N. and Fioretti P.: « Fluctations of hCS, hCG, Estriol, Estradiol, and Progesterone plasma levels in near term pregnancy » - « Poor Intrauterine Fetal Growth » - Parma 14-16 settembre, 1977 (in press). - 13. Samaan N., Yen S.S.C., Friesen H. and Pearson O. H.: *J. Clin. Endocr., 26,* 1302, 1966. - 14. Saxena B. N., Retetoff S., Emerson K. Jr., Slenkow H. A.: *Am. J. Obst. Gyn., 101,* 874, 1968. - 15. Samman N. A., Bradburt J. T., Goplerud C. P.: *Am. J. Obst. Gyn., 104,* 781, 1969. - 16. Spellacy W. N., Buhi W. C., Schram J. D.: *Obst. Gyn., 37,* 567, 1971. - 17. Crosignani P. G., Nencioni T., Brambati B.: *J. Obst. Gyn. Brit. Cwth., 79,* 122, 1971. - 18. Genazzani A. R., Cocola F., Neri P., and Fioretti P.: *Acta Endocr., 71,* suppl. 167, 1972. - 19. Genazzani A. R., Aubert M. L., Casoli M.: *Lancet, 2,* 1385, 1969. - 20. Seppala M., Rouslahti E.: *Acta Obst. Gyn. Scand., 49,* 143, 1970. - 21. Spellacy W. N., Teoh E. S., Buhi W. C.: *Am. J. Obst. Gyn., 109,* 588, 1971. - 22. Varma K., Driscoll S. G., Emerson K. Jr., Selenkow H. A.: *Obst. Gyn., 38,*

487, 1971. - 23. Genazzani A. R., Cocola F., Nasi A., Neri P. and Fioretti P.: *J. Nucl. Biol. Med., 18,* 260, 1974. - 24. Garoff M. D., Seppala M.: *Am. J. Obst. Gyn., 121,* 257, 1975. - 25. Gartside M. W. and Tindall V. R.: *Brit. J. Obst. Gyn., 82,* 303, 1975. - 26. Belanger C., Shome B., Friesen H., Myers R. E.: *J. Clin. Invest., 50,* 2660, 1971. - 27. Spencer T. S.: *J. Obst. Gyn. Brit. Cwth, 78,* 232, 1971. - 28. Genazzani A. R., Cocola F., Casoli M.: *J. Obst. Gyn. Brit. Cwth, 78,* 577, 1971.

Pathogenesis of polycystic disease of the ovary

by

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INTRODUCTION

Polycystic disease of the ovary constitutes an extensive chapter in gynaecological pathology, comprising a set of pictures, different on the clinical, biological and anatomo-pathological planes, but which physiologically have in common: deficient ovulation, constant luteinic insufficiency and frequently sterility.

Ovarian polycystic disease is also called ovarian dystrophy.

Within ovarian dystrophy two forms are classically distinguished: macropolycystic dystrophy, commonly called oophoritis or « sclerocystic ovariopathy » and micropolycystic dystrophy.

Macropolycystic ovarian dystrophy is secondary to numerous dysfunctional conditions in which recurrent anovular cycles are found, either with a central origin or with a local cause. The ovaries are increased in volume, have a lumpy, asymmetrical surface, of variable volume at different periods of the cycle, painful either spontaneously or on palpation, irregular in shape and dimensions, present scars of the corpus luteum, with a thin capsule of variable thickness, and histologically follicles are found in various stages of development, some follicles being atresic (Fig. 1).

Included among the micropolycystic dystrophies, on the other hand, are ovaries upon whose surface no apparent cyst follicles can be seen; these only become evident when a section is made. These have a smooth surface, a mother-of-pearl colour and hard consistency, with an inspissated and sclerotic, sometimes leathery, cortex. They are indolent and painless, of variable dimensions but bilaterally constant in each individual case; their volume may be normal, but is more often 2 to 5 times greater than normal (Fig. 2); they are never less than normal in volume, and show hypertrophy of the stroma and hyperplasia of the internal theca of the follicles, with diffuse interstitial thecomatosis.

The standard description given to these ovaries is that of Stein-Leventhal's syndrome (abbreviated S-L in this paper).

We shall not consider macropolycystic dystrophy in this study, since its pathogenesis is already well known.

This is not so in the case of micropolycystic dystrophy, still today defined as of obscure ([6, 40]) or enigmatic ([39]) pathogenesis. We shall consider the most recent views and our own.

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