PART XVIII

WORKSHOP ON PREGNANCY AND THE KIDNEY

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THE KIDNEY AND PREGNANCY

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Drs Lamperi and Carozzi from Genoa [1] reported on immunological studies in 14 patients with normal pregnancy, 10 patients with pre-eclampsia, and 28 non-pregnant controls. They showed that normal pregnancy is associated with an overall decrease in cellular immunity, a decrease of the ratio helper/suppressor cells, and a fall in plasma Interleukin 2 and urinary Interleukin 1 activities. On the contrary, pre-eclampsia was associated with a decreased percentage of suppressor cells and an increased percentage of helper cells, leading to a higher helper/suppressor ratio. Spontaneous lymphocyte transformation index was increased in normal pregnancy and decreased in pre-eclampsia. The authors conclude that pre-eclampsia involves an abnormal immunological response of the mother to her fetus, and a deficient mother's immune tolerance as compared to normal pregnancy. Dr Lindheimer (Chicago) pointed out that it would be necessary to have the same tests before pregnancy in order to know whether this is a basic pattern of patients or a peculiar response to pregnancy. In answer to another question, Dr Carozzi said that none of those patients has yet completed another pregnancy.

Dr Herzog from Tel Hashomer [2] reported on renal function during pregnancy in five-sixths nephrectomized rats. The authors observed that pregnancy in those animals is associated with an increase in creatinine clearance (from 327μl/min to 525μl/min), in fluid intake, and in fractional excretion of sodium and potassium. Urinary protein excretion increases sharply during pregnancy, and returns to the control values within 24 hours after delivery. At this time, CCr remains higher than in non-pregnant animals, but FE_{Na} and FE_{TcH_2O} are significantly lower. The author's main conclusion is that a very reduced renal functional mass with an already high work load, is able to significantly increase in function during pregnancy. Dr Herzog had no explanation for the diminished FE_{Na} after pregnancy. Drs Lindheimer and Davison said that it could be related to the increased CCr, resulting in a rather identical elimination of the sodium load.

Dr Gokal from Manchester [3] presented the clinical features of six women

who had a nephrotic syndrome with high blood pressure and moderate renal failure in the late second or early third trimester of pregnancy. In all cases, blood pressure and renal functions had been normal until the onset of this syndrome. Fetal outcome was particularly poor, since two deaths occurred in utero and premature caesarean section was required in three other patients. In all cases, proteinuria disappeared and blood pressure and renal function returned to normal within six weeks post-partum. In answer to numerous questions Dr Gokal said that all patients were primiparas, and that none had distinct clinical features like migraine or asthma. None of them had yet completed a second pregnancy. Dr Gokal had no renal biopsies and only one placental histology for those patients. Several participants stated they had seen some such cases, and all agreed that the fetal prognosis is very poor. A semantic discussion on whether or not this could be named nephrotic syndrome did not reach a satisfying conclusion.

Dr Cosci and co-workers from Lodi [4] analysed the clinical features in 43 patients with pregnancy hypertension classified according to the ACOG criteria. They found that maternal and fetal prognosis were worse in group I (i.e. true pre-eclampsia) than in any other group; hypertension was more severe and less responsive to drug therapy, fetal growth retardation was common and perinatal mortality was high. On the contrary in group II (i.e. chronic hypertension), blood pressure responded well to drug therapy and complications were less frequent. Previous hypertension, as well as high blood pressure one year after delivery (overall incidence 30%), were considerably more frequent in group II than in group I patients. Finally, persistence of proteinuria in six per cent of patients suggested an underlying renal disease. Most participants agreed with Dr Cosci's data which are in keeping with most series in the literature. There remain, however, some difficulties in accurately classifying some patients. The limits of 'true' pre-eclampsia are not easy to draw, and the significance of groups III and IV remains obscure. Dr Lindheimer summarized his group's experience of renal biopsies in patients classified in group I, and he emphasized the pitfalls of this classification.

Dr Fiévet and co-workers from Amiens [5] studied the effect of evening primrose oil (a compound which is particularly rich in linoleic and gamma-linoleic acids) in 15 pregnant patients at high risk of pre-eclampsia. The study was blind, and olive oil served as placebo. The bleeding time increased in treated patients. No significant changes occurred in the various coagulation tests performed, nor in tests of platelet aggregation, except for spontaneous platelet disaggregation (after ADP induced aggregation) which was significantly increased in the treated group. The course of pregnancies was not strikingly different between groups, as regards hypertension, symptoms of pre-eclampsia and duration of pregnancy. The birthweights were, however, significantly higher in the treated group. The authors concluded that a supplemented intake of poly-unsaturated fatty acids may have a beneficial effect on fetal growth in high risk pregnancy. The discussion focused on the selection of patients. Dr Lindheimer argued that a selection of patients 'at risk' of pre-eclampsia is very empirical and leads to heterogeneous groups whose real risk is difficult to assess. This

problem is, however, difficult to solve, for in view of the frequency of spontaneous occurrence of pre-eclampsia and fetal growth retardation, such a study in non-selected primiparous women would require huge numbers of patients.

Papers presented

1 Lamperi S, **Carozzi S.** *Normal pregnancy and pre-eclampsia: immunological patterns*
2 **Herzog D,** Iaini A, Cohen D, Serban I, Gavendo S, Kapuler S, Eliahou HE. *Renal response to pregnancy in 5/6 nephrectomized chronic renal failure in rats*
3 Allen R, **Gokal R,** Donnai P, Mallick NP. *Early pre-eclampsia presenting as nephrotic syndrome*
4 **Cosci P,** Surian M, Malberti F, Corradi B, Colussi G, Minetti L. *Maternal and fetal prognosis and long-term follow-up in pregnancies with hypertension*
5 **Fievet P,** Fournier A, Tribout B, Castier B, Coevoet B. *Renin angiotensin aldosterone system and adrenergic system in pregnancy induced hypertension. Critical role of the stimulative conditions and of the lability of blood pressure in the assessment of their pathophysiological role*