

Immunohistochemical study of placental endothelium in physiologic and gestosis-complicated pregnancies

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Summary: In this study we examined the placentae of gestosis patients and controls, with immunoistochemical method and HLA-DR monoclonal antibody, in order to show the role of placental endothelium in gestosis pathology onset.

Our results show a marked expression of class II histocompatibility antigens in gestosis placentae with respect to controls. We suppose, in gestosis patients, a role for a particular, genetically determined HLA haplotype which increases disease receptivity.

Key words: E.P.H. gestosis; Placental endothelium.

INTRODUCTION

Although several Authors have faced this complex problem, the etiology of EPH gestosis is still unclear. In an attempt to identify the "primum movens" responsible for the onset of this syndrome, we performed an immunohistochemical study on the placental endothelium in physiological and in gestosis complicated pregnancies.

The placental endothelium plays a very important role in pregnancy, such as that

of maternal-fetal barrier. An alteration of this barrier, caused by some risk conditions (post-prandial hypercholesterolemia, shear stress, smoke, advanced glycosilated end-products)⁽⁵⁾ or of particular genetic predisposition^(4, 6, 7), may origin a maternal immunitary reaction of rejective type.

MATERIALS AND METHODS

We examined 30 placentae: 20 from patients with EPH gestosis and 10 controls.

The placentae were divided into 4 quadrants, and four samples were taken from each quadrant: 2 from the basal deciduous zone and 2 from a more central zone.

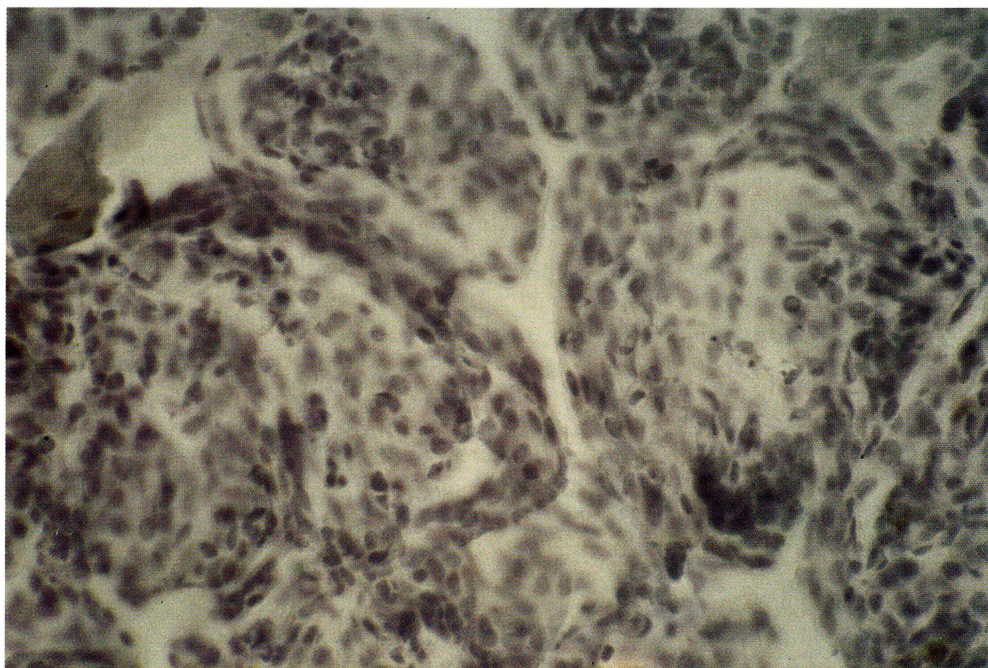
All samples were submitted to immuno-histochemical examination (Avidin-Biotin Complex, ABC, method) and tested with Mon Ac HLA-DR (1:100 dilution).

HLA-DR antibody recognizes the products of DX and DQ subregions of the human class-II major histocompatibility complex (MHC). Therefore it identifies several elements involved in

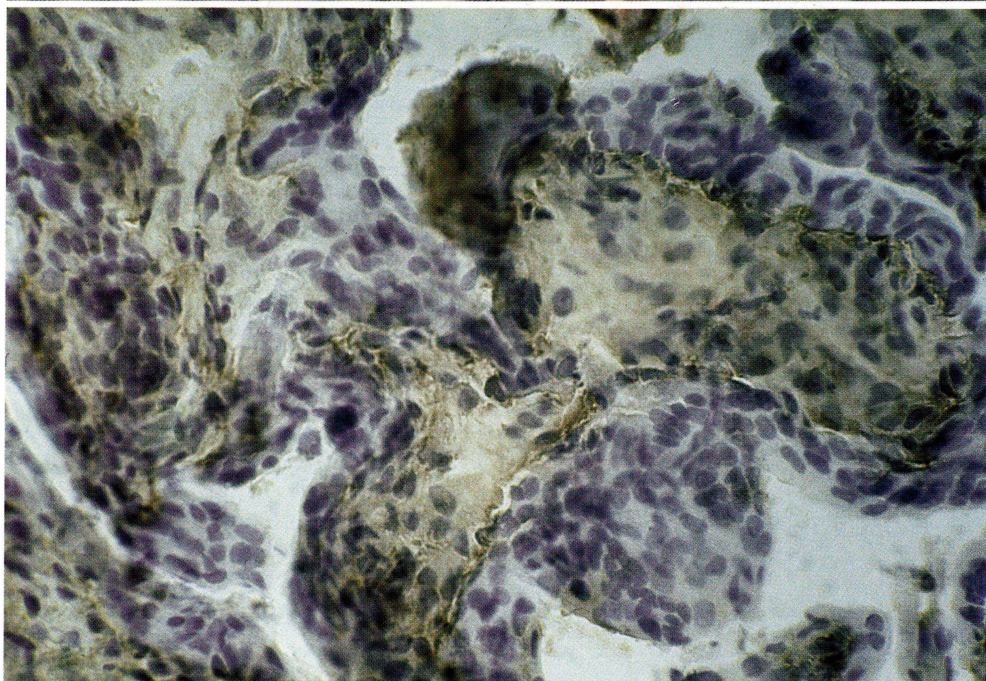
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Fig. 1. — Placenta from physiological pregnancy. We observe both syncytiotrophoblast and stromal cells negative to HLA-DR monoclonal antibody determination (1:100 dil, 425 $\times$).
 Fig. 2. — Gestosis placenta. Positivity to monoclonal antibody determination of internal stromal cells, and negativity of external syncytiotrophoblast cells.

activating and maintaining immunitary response (monocytes-macrophages, activated T lymphocytes, endothelial cells, interdigital reticular elements).

RESULTS

All the cases we examined evidenced a marked positivity of HLA-DR histocompatibility antigen in gestotic placentae with respect to controls (Fig. 1 and 2).

DISCUSSION

The activating mechanism for such an evident immunologic reaction in gestosis is not yet known. Several hypotheses may be advanced:

1) gestosis, as well as other diseases (ankylosing spondylitis, insuline-dependent type I diabetes), may correlate to particular, genetically determined HLA haplotypes which arouse disease receptivity^(9, 10, 11);

2) different, even transitory, stimuli may induce a different genic regulation, capable of modifying functional response at endothelial cells DNA level;

3) a reduced antigenic disparity between parents (as regards MHC) may be the cause of the difficulties in activating an effectual protective reaction^(4, 9).

CONCLUSIONS

HLA typing in gestotic patients will show whether HLA combinations play a role in disease etiopathogenesis.

REFERENCES

- 1) Beer A.E., Billingham R.E.: *J. Reprod. Fert.*, 1974, 21 (suppl.), 59.
- 2) Bolis P.F. *Ginecologia e Ostetricia*, 1986, 1, 56.
- 3) Bolis F., Di Pietro G. *Ginecologia e Ostetricia*, 1986, 2, 43.
- 4) de Luca Brunori I., Filippeschi M., Mariotti B., Pecori F., Tarani A., Urbano M., Fossella P. Atti VI Congr. Naz. OIGIG, Milan, October 14-16, 1993, p. 328-330, CIC Ediz. Intern.
- 5) Gimbrone M.A. Jr.: In: "Vascular endothelium in hemostasis and thrombosis". Churchill-Livingstone, Edinburgh, 1986, p. 1-13.
- 6) Pober J.S., Collins T., Gimbrone M.A. Jr., et al. *Transplantation*, 1986, 41, 141.
- 7) Pober J.S., Gimbrone M.A. Jr. *Proc. Natl. Acad. Sci USA*, 1982, 79, 6641.
- 8) Power D.A., Catto G.R.D., Mason R.J., McLeod A.M., Stewart G.M., Stewart K.N., Shewan W.G. *Lancet*, 1983, 2, 701.
- 9) Redman C.W., Bodmer J.G. et al. *Lancet*, ii, 1978, 397.
- 10) Simon P., Fauchet R., Monot M., et al. *Kidney Int.*, 1980, 17, 705.
- 11) Simon P., Fauchet R., Pilorge R., et al. *Kidney Int.*, 1986, 34 (Suppl. 25), S125.

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