Non-immunologic hydrops fetalis: study of 86 autopsies

Aparecida Gomes Pinto Garcia MD
Claudia Schwartiz Pegado MD
Hilda Irec de Bem Ramos MD
Regina Lucia Souza Marques MD
Rita de Cassia Nasser Cubel MD
Jussara Pereira do Nascimento MD

INTRODUCTION
As a result of prophylaxis for Rh isoinmunization, non-immune causes are assuming an increasingly prominent role in the etiology of hydrops fetalis (HF).

We report our experience of non-immunologic hydrops fetalis (NIHF) based on 86 autopsies done in the Instituto Fernandes Figueira (Rio de Janeiro, Brazil). Of 3111 paediatric autopsies performed during 1954-1992, 86 cases of non-immunologic hydrops fetals (NIHF) were reviewed. Cases were identified as HF when generalized oedema and cavity effusions were present. Family history, complications of pregnancy and delivery, blood typing of both mother and infant, Coomb's test, serological examination for syphilis, toxoplasmosis and other laboratory tests were recorded. Postmortem roentgenograms and chromosomal analysis were also occasionally made. Placentas were available for pathological examination in all cases. During the same period 12 cases of immunologic hydrops (Rh immunization) also were autopsied.

METHODS
Histological preparations were stained by haemotoxylin-eosin and also by special techniques when required. To clarify infectious causes more recent methods such as nucleic acid hybridization (parovirus B19) were applied. The pathological conditions associated to our cases of NIHF are listed in Table 1.

RESULTS
Intrauterine infections were diagnosed in 80 cases. Syphilis was also diagnosed in 31 cases by the identification of its peculiar lesonal complex allied to the identification of Treponema pallidum (Levaditi).

30 autopsies disclosed a complex of inflammatory-degenerative lesions, in different combinations, involving placenta and fetal visceral tissues. This constellation of lesions was similar to the one described in congenital rubella and later observed in other viral infections. We classified these as intrauterine haematogenic infections (IUIH), cause unknown. We presume that these lesions occurred during fetal development, as associated malformations were rarely present. Human parvovirus B19 DNA was detected on one fetus by in situ and in five others by dot blot hybridization. Congenital rubella, as diagnosed by laboratory, clinical and pathological data, was present in two cases. Cytomegalovirus, the most frequent viral infection in our autopsy series, was not observed in our patients, neither was herpes simplex.

Toxoplasmosis was observed in seven cases; the diagnosis was confirmed on maternal serology, a complex of lesion and the presence of the protozoon in histological sections.

Table 1. Non-immunologic hydrops fetalis (NIHF)-associated pathology (1954 to 1992)

<table>
<thead>
<tr>
<th>Disease entity</th>
<th>No. of autopsy-cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syphilis</td>
<td>31</td>
</tr>
<tr>
<td>IUHI* of unknown etiology</td>
<td>30</td>
</tr>
<tr>
<td>Rubella</td>
<td>02</td>
</tr>
<tr>
<td>Human parvovirus P19</td>
<td>06</td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>07</td>
</tr>
<tr>
<td>Genetic</td>
<td>08</td>
</tr>
<tr>
<td>Twinning</td>
<td>02</td>
</tr>
<tr>
<td>Total of NIHF</td>
<td>86</td>
</tr>
</tbody>
</table>

*IUHI = Intrauterine haematogenic infection of unknown etiology

1Case showed double infection by human parvovirus B19 and syphilis
2Four of the cases showed association with IUHI
3One of the cases showed association with IUHI
In six autopsies, a genetic syndrome was suspected; in four of them infection with the same lesion complex as in IUHI was present. The involvement of only one twin was noted in two cases; in one twin a complex of lesions compatible with IUHI was also found.

CONCLUSION
In a review of several series of NIHF by Machin\(^2\) transplacental infections are mentioned only superficially. As in our series myocarditis and hepatitis were noted frequently in the IUHI group. Nakamura et al.\(^3\) note the relationship between infection and tissue damage and the frequency of haemolytic anaemia in NIHF. Hutchinson et al.\(^4\), like ourselves, comment on the frequency of IUHI in their series of 61 cases. We agree with Jauliaux et al.\(^5\) that more epidemiological data is required on this fetal disorder.

REFERENCES

The Dr Albert Dubois Prize of Tropical Pathology

The third Dr Albert Dubois Prize (1990-1994) has been awarded to Dr Wendy Gibson, Bristol University, for her work on genic exchanges in African trypanosomes. Thirteen candidatures were submitted to the jury of the Royal Academy of Medicine in Belgium.

African trypanosomes are the causative agents of sleeping sickness in humans and of ‘nagana’, an animal disease which hinders cattle breeding in Africa. Reproduction of these trypanosomes proceeds by simple asexual bipartition. Epidemiological and molecular studies by Dr Gibson have demonstrated genetic exchanges in trypanosomes while they develop in their insect vector, the tsetse fly. By crossing trypanosomes bearing different molecular markers, Dr Gibson demonstrated the several genes are transmitted according to Mendel's laws. These discoveries are a major contribution to the field of tropical pathology, providing new insight into the mechanisms of strain variability in these parasites.

Dr Wendy Gibson PhD of British nationality, devoted her career to the study of African trypanosomes, first at the Kenya Trypanosomiases Research Institute of Nairobi, thereafter in the laboratory of Professor Piet Borst in Amsterdam and, since 1986, at the Department of Pathology of the Faculty of Veterinary Medicine of Bristol University.

The Dr Albert Dubois Prize of Tropical Pathology and the medal of the Royal Academy of Medicine in Belgium was presented to Dr Gibson on 10 February 1996 at the Palais des Académies during the official presentation of the Prizes of the Royal Academy.

The fourth quinquennial period of the Dr Albert-Dubois Prize of Tropical Pathology is now open, covering the years 1995-1999. Applications for the Prize, amounting to 5000 Belgian francs, should be submitted to the Royal Academy of Medicine in Belgium before 31 May 1999. Complementary information may be obtained from the Secretariat of the Academy (Académie royale de Médecine de Belgique, 1 rue Ducale, 1000 Bruxelles).