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### SHORT REPORT

# Non-immunologic hydrops fetalis: study of 86 autopsies

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TROPICAL DOCTOR, 1996, 26, 78-79

#### INTRODUCTION

As a result of prophylaxis for Rh isoimmunization, non-immune causes are assuming an increasingly prominent role in the aetiology 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 nonimmunologic hydrops fetalis (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 haematoxylin-eosin and also by special techniques when

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required. To clarify infectious causes more recent methods such as nucleic acid hybridization (parvovirus 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 lesional 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 infections1. We classified these as intrauterine haematogenic infections (IUHI), 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)

Disease entity	No. of autopsy-cases
Syphilis Syphilis	31†
IUHI* of unknown actiology	30
Rubella	02
Human parvovirus P19	06 <sup>†</sup>
Toxoplasmosis	07
Genetic	08‡
Twinning	024
Total of NIHF	86

\*IUHI=Intrauterine haematogenic infection of unknown aetiology

<sup>†</sup>One case showed double infection by human parvovirus B19 and syphilis

Four of the cases showed association with IUHI

One of the cases showed association with IUHI

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In six autopsies, a genetic syndrome was suspected; in four of them infection with the same lesional 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<sup>2</sup> transplacental infections are mentioned only superficially. As in our series myocarditis and hepatitis were noted frequently in the IUHI group. Nakamura et al.<sup>3</sup> note the relationship between infection and tissue damage and the frequency of haemolyte anaemia in NIHF. Hutchinson et al.<sup>4</sup>, like ourselves, comment on the frequency of IUHI in their series of 61 cases. We agree with Jauniaux et al.<sup>5</sup> that more epidemiological data is required on this fetal disorder.

**ACKNOWLEDGEMENTS** 

This study was partially supported FAPERJ, Brazil.

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## 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 gentic exchanges in African trypanosomes. Thirteen candidatures were submitted to the jury of the Royal Academy of Medicine of 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 typanosomes while they develop 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 These discoveries are a major laws. 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 Trypanosomiasis 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 of 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).