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The changing pattern of fetal hydrops

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SUMMARY

*Fetal hydrops (*hydrops fetalis*) remains a significant cause of fetal and neonatal mortality. The decreased incidence of rhesus iso-immunisation due to prophylaxis with rhesus immune globulin (anti-D), improved antenatal ultrasound screening, and advances in neonatal intensive care have greatly altered the clinical outlook in this condition. A retrospective review of all 27 liveborn cases of hydrops in the Royal Maternity Hospital, Belfast in the period 1974 – 89 showed that in the last five years 40% of cases were non-immune in origin. The mortality rate fell from 100% in the first part of the study to 50% in the second.*

INTRODUCTION

There have been two major changes in the management of fetal hydrops in the last twenty years — the use of anti-D immunoprophylaxis which has greatly reduced the incidence of the disease and the development of perinatal intensive care. This has led to major changes in both the prenatal and postnatal management of the gravely ill fetus. We reviewed the records of all liveborn cases of fetal hydrops in the Royal Maternity Hospital, Belfast in the period 1974 – 89 to assess changes in aetiology, incidence, management, and prognosis.

METHODS

Fetal hydrops was defined as fluid accumulation in some or all serous cavities (peritoneal, pericardial, and pleural) with generalised skin and placental oedema. In the 15 year period 1974 – 89, there were 27 liveborn babies with hydrops among the 52,177 liveborn babies in the Royal Maternity Hospital, an incidence of 1 in 1932. We chose not to include stillbirths because in some cases a full assessment to ascertain cause had not been performed.

RESULTS

The specialised neonatal intensive care unit was opened in this hospital in 1978; we looked at babies managed before and after this date (Table). Of the 11 babies born in 1974 – 7 there were no survivors. In this early period, 10 were due to rhesus-D incompatibility, one to rhesus-E incompatibility, and none to other blood group incompatibilities or non-immune causes. The management of the cases of iso-immunisation, both pre- and post-natal, is outlined in the Table.

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TABLE

Clinical details and management of 27 cases of fetal hydrops

	1974–7	*1978–89*
Number of babies	11	16
Gestational age (weeks) (mean ± SD)	28·9 ± 3·2	30·9 ± 4·3
Birth weight (g) (mean ± SD)	1607 ± 741	1975 ± 802
Cause		
Rhesus-D	10 (91%)	10 (63%)
Rhesus non-D	1 (9%)	1 (6%)
Other iso-immunisation	0	1 (6%)
Non-immune	0	4 (25%)
Mortality — all causes (%)	11 (100%)	8 (50%)
Management of babies with iso-immunisation		
Number	11	12
Intra-uterine fetal transfusion	5 (45%)	5 (42%)
Exchange transfusion	10 (91%)	10 (83%)
Died in labour ward	1 (9%)	1 (8%)
Mechanical ventilation	0	11
Mortality (%)	11 (100%)	7 (58%)

In the later period, half of the babies with hydrops survived. Ten were due to rhesus-D incompatibility, 1 to combined rhesus — c and E antibodies, 1 to Kells antibody, and 4 to non-immune causes. The management of the four babies with non-immune hydrops included mechanical ventilation (in three), thoracocentesis (in two), paracentesis (in one), and treatment with digoxin and diuretics (in three). These cases were due to congenital cytomegalovirus infection, chronic feto-maternal transfusion, congenital heart disease (Ebstein's anomaly) and no cause was found after extensive investigation of the fourth leaving a diagnosis of idiopathic non-immune hydrops. Three survived, one with a mild residual hemiplegia.

DISCUSSION

In the past, the vast majority of cases of hydrops were due to iso-immunisation. Non-immune hydrops was first described in 1943 by Potter,[1] who defined it as hydrops due to any cause other than feto-maternal blood group incompatibility. Prior to 1970, Macafee[2] reported that 17% of cases of hydrops were non-immune in origin. In the last five years of our study, 40% of cases were non-immune, which has also been the experience of others.[3] Since the introduction of anti-D prophylaxis, an increasing number of cases of materno-fetal blood group incompatibility are due to groups other than rhesus-D. We have had cases of hydrops due to rhesus-E, rhesus-c, and Kells blood groups. O'Sullivan[4] found that the proportion of patients delivered in the Royal Maternity Hospital, Belfast with antibodies other than rhesus-D had risen from 11% in 1970 to 37% in 1980.

Hydrops has many causes, and new ones are reported every year. In a review by Machin[3] of 1414 cases published in the 1980's, 63% were due to five fetal disease processes: cardiovascular disorder, chromosome abnormalities, thoracic disorder, twin-twin transfusion, and anaemia. Formerly, the appearance of hydrops was considered to be a very poor prognostic factor in cases of iso-immunisation with death almost inevitable.[5] Recently, the prognosis has improved — we report a mortality rate of 50% in cases of rhesus iso-immunisation in the last eight years, and others have reported rates as low as 25%.[6] For non-immune hydrops our mortality rate was 25%, which compares favourably with reports of rates up to 80–95%.[7,8] The change in prognosis for the hydropic fetus is probably due to many factors, including delivery of the hydropic infant by elective caesarean section in a controlled setting with a neonatal team in attendance to perform intensive resuscitation.[9]

The prenatal diagnosis of hydrops is improving due to the use of high resolution ultrasound scanning. In the last eight years all cases of hydrops in pregnancies complicated by iso-immunisation were identified in the antenatal period so that management could be planned in advance. However, only 50% of our cases of non-immune hydrops were identified prior to birth. It has been suggested that certain maternal conditions can be used to predict the presence of non-immune hydrops. In one report, if maternal anaemia, hypertension or polyhydramnios had been used as indications for ultrasound examination, 80% of babies of greater than 28 weeks gestation with non-immune hydrops would have been detected.[8] If these criteria had been applied to our babies, all would have been identified prior to birth.

In the future, it is probable that the mortality rate in fetal hydrops will continue to fall due to better antenatal detection and more widespread use of cordocentesis for direct fetal intravascular transfusion in severe haemolytic disease.[10] For this to be achieved, the obstetrician must be alert to the possibility of this disease.

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