THE CAUSES OF ANTEPARTUM FETAL DEATH: A CLINICO-PATHOLOGICAL STUDY

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SUMMARY

Forty cases of antepartum fetal death occurring among 6668 deliveries at the Ist Department of Obstetrics and Gynecology of the University of Vienna between 1976 and 1979 were evaluated.

The fetal death rate was 6 per 1000 births. In 32 cases (80%) a diagnosis identifying the disorder, that initiated the cause of fetal death, could be established. The remaining 20% did not have a demonstrable diagnosis. The most frequent cause of death in this series was in 40% severe toxemia of pregnancy causing a chronic nutritive placental insufficiency. Histologically in such cases regressive changes of the placenta, such as microinfarcts, necrosis and deposition of intervillous fibrin were found. Rarer causes, also associated with intrauterine asphyxia were 2 cases each of placenta praevia (5%) and abruptio placentae (5%), one cord accident (2.5%) and one case of postmaturity (2.5%). The second most important etiological factor was in 12.5% the ascending intrauterine infection prior or after the premature rupture of the fetal membranes. Bacteriologically the most common organisms isolated were E. coli, enterococci and anaerobic bacteria. One fetal death was due to rhesus-incompatibility, and in one case both twins died in utero (2.5%). Our results suggest, that an early diagnosis and a successful treatment of placental insufficiency would permit a further reduction of the perinatal mortality rate. In conclusion the absolute necessity of the performance of all available diagnostic means in order to prevent recurrence of a stillbirth is emphasized.

INTRODUCTION

An antepartum fetal death (AFD) represents an enormous psychological burden for the patient concerned and implies severe possible complications, such as postpartum hemorrhage and the "dead-fetus-syndrome", a coagulopathy associated with hypofibrinogenemia ([5, 9, 15, 16, 26]).

According to the World Health Organisation (WHO) definition ([23, 26]) a fetal death is the cessation of fetal life prior to complete expulsion or extraction, irrespective of the duration of pregnancy. It is statistically expressed as a rate per 1000 births. Despite the improvement in data collection over the years, the reporting of fetal death remains one of the weakest links in the vital statistics system ([17, 25]). Therefore the present study was designed to investigate the causes of the abnormal gestational development.

MATERIAL AND METHODS

AFD was defined according to the WHO-recommendations as a stillbirth having a body-length (crown-heel) of 35 cm or more ([27]).

Detailed anamnestical and clinical information of the mother was collected in each case with special regard to previous abortions, premature births and perinatal or antenatal loss.

Serological, bacteriological, autoptical and histological examinations were performed in the mother, the fetus and the placenta. The evaluation of the findings was based on the correlation between the available clinical and pathological findings.

According to the WHO recommendations a "Certificate of Cause of Perinatal Death" was adopted for each stillbirth, differentiating between the main and other diseases or conditions in the fetus and the mother ([26]). A prime diagnosis was assigned to each case, intending to identify the disorder, that initiated the cause of death. When more than one factor was encountered, the more lethal was considered the primary cause.

RESULTS

Between 1976 and 1979 40 cases of AFD occurred among 6668 deliveries at the Ist Department of Obstetrics and Gynecology of the University of Vienna. This

Table 1. — *Anamnestical data of 39 mothers experiencing antepartum fetal death.*

Mean age	30 years
Parity	21 nulliparous, 19 parous
Previous abortion, premature birth or stillbirth	17 (43.6%)
Hypertension	5 (12.8%)
Recurrent urinary-tract infection	5 (12.8%)
Obesity	3 (7.7%)
Latent diabetes (pathological gtt)	4 (10.2%)
Hypothyroidism	1 (2.6%)
Excessive smoking	8 (20.5%)

is a fetal death rate of 6 per 1000 births. 31 patients were examined at our Department prior to the occurence of the AFD and 8 patients were admitted from other hospitals already with the diagnosis of an AFD.

22 fetuses were male and 18 female. The median birthweight was 1850 g, with a range from 900 g to 3500 g, the median length was 41 cm, the range 35 cm to 50 cm. The median duration of gestation was 33 weeks, with a range from 27 to 43 weeks. Nine infants with a gestational age of 37 completed weeks were classified as at term and 31 fetuses with a gestational age of less than 37 weeks were classified as preterm. Maceration was present in 33 babies.

Table 1 shows the anamnestical data of the mothers experiencing fetal death. The median age of the mothers was 30 years, the range 18 to 38 years. 20 mothers were nulliparous, 19 were parous. 17 patients (43.6%) had a history of one or several abortions, premature births or stillbirths. The anamnestical data of the mothers revealed, that 5 of them had a history of hypertension, 5 had recurrent urinary-tract-infections, 3 were obese and 5 had endocrinological disorders, such as latent diabetes (4 patients) and hypothyroidism (1 patient). 8 mothers were heavy smokers.

Table 2 shows the main causes of fetal death of the 40 cases.

In 32 cases (80%) a diagnosis, identifying the disorder, that initiated the cause of fetal death, could be established. The other 20% did not have a demonstrable diagnosis.

Severe toxemia of pregnancy and allied conditions such as hypertension, defined according to the recommendations of the Organisation Gestosis ([8]), were in 40% the most frequent cause of death in this series. This group includes 13 cases of severe toxemia and imminent eclampsia (EI) (32.5%) and 3 cases of convulsive eclampsia (EC) (7.5%).

5 patients (12.5%) developed an amniotic fluid infection syndrome prior or after premature rupture of the membranes in the 29th to 31st week of gestation and amnionitis was thought to be responsible for the stillbirth. 3 of the mothers had a cerclage operation for an incompetent cervix. In 4 cases the AFD was due to severe bleedings caused by placenta praevia in 2 cases (5%) and by abruptio placentae in 2 more cases (5%). The diagnosis of abruptio placentae was confirmed on pathological examinations of the placenta by the finding of extensive retroplacental blood clots or hemorrhages. The diagnosis of a congenital anomaly was established

Table 2. — *Main causes of antepartum fetal death (n=40).*

Main cause of AFD	n=40	Percent
Unknown	8	20 %
Toxemia	13	32.5%
Eclampsia	3	7.5%
Amnionitis	5	12.5%
Placenta praevia	2	5 %
Abruptio placentae	2	5 %
Congenital anomaly	2	5 %
Twins	2	5 %
Rh-incompatibility	1	2.5%
Cord-accident	1	2.5%
Postmaturity	1	2.5%

twice. Both malformations were in themselves incompatible with life. In 1 case Rhesus-incompatibility was found. Both infants of a twin-pregnancy died in utero. A cord accident was thought to be the cause for the stillbirth in 1 case and in another postmaturity, defined as pregnancy exceeding the 42nd week of gestation, was made responsible for the AFD.

DISCUSSION

Reviewing the literature the causes of AFD are found to be unknown even in large epidemiological statistics up to 50% of cases ([1, 2, 5, 9, 15, 16, 19, 23, 25]). According to Stander fetal death accounts for one half of the total perinatal mortality ([26]). The reduction of this large potential would improve the perinatal mortality statistics considerably ([25]). Thus the identification of the causes of AFD could be an important step to its reduction ([2, 5, 16, 19]).

In the present study careful evaluation of the clinical data and thoroughly performed laboratory examinations made it possible to identify the cause of fetal death in 80%. However in 20% no etiological factor could be demonstrated, whereas frequently more than one cause for the death was present.

Toxemia of pregnancy and allied disorders represented the numerically most important clinical cause of AFD. Histological examinations of the placenta revealed regressive changes such as microinfarcts, necrosis and deposition of intervillous fibrin, which are due to the decreased utero-placental blood flow ([12, 14]). Arteriolar spasms, which are initially present in toxemia, cause a hypocirculation leading to a chronic nutritive placental insufficiency ([14]). In severe cases AFD occurs with a high incidence between the 30th and 36th week of gestation and thus determines perinatal-mortality-rate ([2, 19]). Thus placental insufficiency, representing the patho-morphological substrate of toxemia, must be considered as the most important etiological factor of AFD. This clearly points out the importance of an early recognition of this disorder. An early diagnosis and successful treatment would permit a further reduction of the perinatal-mortality-rate ([25]).

Further causes associated with intrauterine asphyxia, due to acute, respiratory placental insufficiency ([14]) were the cases of placenta praevia, abruptio placentae, the cord accident and the case of postmaturity. Abruptio placentae is frequently associated with toxemia and hypertension ([15]) and also in both of our cases hypertension was found as associate finding.

The second most important etiological factor was the ascending intrauterine infection prior and after the premature rupture of the fetal membranes. An incompetent cervix and a cerclage operation are further predisposing factors for this disorder. The most common organisms isolated from amniotic fluid samples in this study were E. coli, Enterococci and anaerobic bacteria ([4]). Also Naye and Peters ([21]) found congenital pneumonia originating from amniotic fluid infections as a common cause of premature labor and perinatal death ([20]). The authors state, that a few of these infections may have a hematogenous origin, but most appear to be initiated by bacteria, which have ascended through the cervix. The septicemia that often follows the congenital pneumonia appears to kill the fetuses.

No specific infections, such as syphilis, toxoplasmosis, cytomegalosis, listeriosis or rubella could be proved in this series.

Of the two fetal malformations one was an anencephaly, the other a sympodia (sirenomelia) – a multiple malformation with combined legs. Erythroblastosis, due to Rhesus-isoimmunization caused one death in utero in the 29th week of gestation. An attempt of a transabdominal transfusion had failed, because of an unfavourable anterior wall localisation of the

hydropic placenta. This mother previously had given birth to a hydropic infant, who died perinatally. The AFD of both twins in utero was suggested to be due to relative placental insufficiency. The cord accident was a tightly coiled umbilical cord twice around the neck of the fetus, probably resulting in a subacute respiratory-placental-insufficiency. One fetus was delivered dead after more than 42 weeks of gestation. The rate of stillbirth is known to increase considerably in postmaturity pregnancies ([15]).

Whereas in the older literature up to 20% of the cases of AFD were due to abnormal labor and obstetrical complications sub partu ([16]), in this study no case of fetal death had to be assigned to an obstetrical failure.

Our results are in good agreement with the recently published data of the Vienna Perinatal Study 1978 ([1]) and also with the Munich Perinatal Study ([25]). The first showed a rate of AFD of 8 per 1000 births, in the Munich series a rate of 6.6 was found ([1, 24]). Concerning the etiology also other authors state, that placental-insufficiency must be considered as main cause for an AFD ([2, 5, 15, 22, 23]).

We emphasize, that despite the employment of modern diagnostic means, such as biochemical monitoring (HCG, AFP, HPL, E3, uric acid [[10, 11]]), placental perfusion measurement ([6]), ultrasound biometry ([24]) and ante-partum cardiotocographic monitoring ([3]), we sometimes still do not succeed in the prevention of an AFD. Frequently in these fatal cases immaturity of the fetus is limiting an active obstetrical management.

The therapeutical possibilities in the treatment of placental-insufficiency are still modest ([13]). Yet several promising approaches, such as continuous administration of β-2-mimetics ([12]), adbominal decompression ([18]) and transcutaneous electric dorsal stimulation ([7, 13]) could reduce the rate of AFD considerably.

In conclusion we like to point out the absolute necessity of the performance of all available diagnostic means in order to prevent recurrence of a stillbirth.

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