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Intrauterine infection may be a major cause of stillbirth in Sweden

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Aim of the study. To investigate intrauterine infection as a cause for unexplained stillbirth.

Methods. Chorioamnionitis was studied in a material of stillbirths (117 subjects from the years 1985–1994) from a region in the south Sweden. Control material (126 alive and healthy newborns and with healthy mothers) was gathered from the same region.

Results. Chorioamnionitis was a common diagnosis both with stillbirths and ‘healthy’ deliveries (82 and 68%, respectively). Extension of the inflammation to decidua basalis was seven times more common among stillbirths than among controls (odds ratio 7.2, confidence interval 2.8–21.9). The most common bacteria found at cultures were Escherichia coli, Coagulase negative staphylococcus, Enterococcus faecalis and group B Streptococcus. The risk for stillbirth was doubled if both inflammation and bacteria were present (odds ratio 2.3, confidence interval 0.92–5.8). Meconium discharge was more common among stillbirths than controls (odds ratio 4.7, confidence interval 1.7–14). There were no differences in any respect regarding macerated and non-macerated stillbirths. Our findings are similar to the results from studies in developing countries except for the higher incidence of stillbirths in such countries.

Conclusions. Thus, a large part of otherwise unexplained stillbirths might be due to ascending infections.

Key words: chorioamnionitis; stillbirth

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In Sweden a stillbirth is defined as a fetus, who died in utero and is delivered after 28 completed gestational weeks. Known conditions causing stillbirth are placental insufficiency due to eclampsia or lupus anticoagulant syndrome in the mother, maternal diabetes mellitus, complex malformations, chromosomal anomalies, certain infections e.g. group B streptococci (GBS), Listeria and different viruses. However, in many stillbirths the cause remains unknown.

Chorioamnionitis was reported to be the cause of death in 7.9% of stillbirths in East Lausing, Michigan, USA (1) and in 2% in Helsinki, Finland (2). In Toronto, Canada, Quinn et al. (3) found 71% of chorioamnionitis among stillbirths with no other cause of death versus 22% among stillbirths with other proven causes of death.

The placenta and fetus can be infected by two major pathways: the hematogenous (transplacental) route and the ascending, amniotic (transcervical) route. Hematogenous infection produces an inflammatory response in the villi (villitis) with dominating lymphocytic infiltration. The etiologic agents are viruses, certain bacterial species (spirochetes, Listeria) and protozoa (Toxoplasma gono-
In ascending intrauterine infection (chorioamnionitis) bacteria from vagina spread through the uterine cervix between the chorion and amnion into the amniotic fluid, to the chorionic and/or to the basal plate of the placenta. Intrauterine infection is a unique situation, in which two individuals are exposed to infection at the same time. Morphologically, maternal as well as fetal responses may occur. Polymorphonuclear leucocytes (PMNs) in the free membranes, subchorionic fibrin, chorionic plate of the placenta and decidua basalis are maternal, while PMNs in the walls of arteries and veins inside the chorionic plate and the umbilical cord are of fetal origin.

More than 130 different bacterial species may be involved in intrauterine infections (4). Several studies have shown a relationship between bacterial isolation from free membranes (5), amniotic fluid (6) as well as placental villous tissue (3) and chorioamnionitis, preterm labor (5), perinatal morbidity and mortality (3). GBS, Fusobacteria, Escherichia coli (E. coli), Bacteroides and Ureaplasma usually cause inflammatory response in the membranes (5). Intrauterine bacterial invasion may occur without inflammatory response in the fetus or the placenta. In some cases of chorioamnionitis with PMNs infiltration no growth of bacteria is found. But mostly an inflammatory lesion in the chorioamniotic membranes seems to serve as a marker of microbial invasion (6). It was shown in experiments with rabbits that inoculation of E. coli induced inflammatory response in the endometrium, amnion and chorion (7), and positive cultures from the same localizations as well as pregnancy losses (7, 8). However, similar injections of sterile exogenous irritants such as meconium, gastric juice, acid, India ink, did not result in chorioamnionitis (9).

Intrauterine infection is regarded as an important cause of stillbirth in developing countries (4, 10, 11). The prevalence of acute chorioamnionitis tends to be higher in developing than in industrial countries (10). Salafia et al. (12) found a prevalence of 4% of acute chorioamnionitis in uncomplicated term pregnancies in Connecticut, USA and Payne et al. (13) reported a prevalence of 24% of chorioamnionitis in a British population. However, the intrauterine infection as a cause of stillbirth in well nourished communities has been questioned (14). The purpose of this study was to explore the incidence of intrauterine infection in stillbirths in a Swedish population.

Material and methods

The study is based on 117 stillbirths (gestational age 28 weeks to term), who were autopsied in the Department of Pathology of the University Hospital, Lund, Sweden. The study period was from August 1st, 1985 to December 31st, 1994. The material consisted of stillbirths due to placental insufficiency, known infections and due to unknown cause. Cases with multiple congenital malformations were excluded as well as cases in which bacteriological examination had not been performed because of lack of personal resources to perform the sterile procedures. No twins appeared in the material. At autopsy, material for bacteriological and morphological examination was taken from placenta and organs. All autopsies were performed by one pathologist (IH). All placental slides of stillbirths were reevaluated by another pathologist (ET) who also examined all the placentas from the controls. As controls we collected 126 placentas from normal singleton term deliveries from the Department of Obstetrics of the University Hospital, Lund, Sweden, between September 28th, 1995 and November 17th, 1995. The delivery was considered normal when a mother after vaginal delivery without complications went home with a healthy child. The macroscopical examination of the placentas was performed by three of the physicians (IH, EM, ET), and most specimens for histology and bacteriology were sampled by a pathologist (ET).

Culture sampling

Cultures were sampled from all the placentas and the external ears of the neonates and stillbirths. In stillbirths samples were taken from the throat, lung, heart blood, liver, brain and liquor. Stillbirths and placentas were put in sterile towels in the delivery room and brought to the pathology laboratory. The autopsy was performed within 24 hours after delivery. Placentas for control material were examined daily in the obstetric department. After the delivery, placentas as well as ear cultures were placed in the refrigerator (+4°C). All cultures were grown within 24 hours of the delivery. The culture samples from placenta and organs were obtained by disinfecting with 70% ethanol, incising through the skin or chorionic plate with the sterile scalpel blade and taking a piece of tissue with new sterile pincers and scalpels. The samples from placental tissue were taken with care to avoid contact with the decidua basalis. The heart blood was taken from the right ventricle and liquor was taken suboccpitally with sterile syringes. For sampling of brain tissue the large fontanelle was opened and tissue samples were taken with new sterile tools. Ear and throat cultures were placed in Stuart's transport medium. Liquor and blood were put in sterile tubes. Tissue samples of placenta and or-
gans were put into sterile boxes for subsequent bacterial culturing.

**Culture conditions**

Samples taken from the external ears and throats as well as from liquor and heart blood were inoculated on blood agar (5% horse erythrocytes) and hematin agar. Heart blood and liquor were also incubated in Tryptic soy broth (TSB) and incubated at 37°C for two days and then subcultured on the same media. Hematin agar and one blood agar plate were incubated in 5% CO₂ atmosphere, and one blood agar plate was incubated anaerobically for two days. Tissue samples from placenta, lung, liver and brain were homogenized before being inoculated in TSB for two days, after which they were subcultured as above. Isolated microorganisms were typed to the species level according to the ASM Manual (15).

**Chemicals**

Agar base was purchased from LabM, Salford, UK, and TSB from Difco Laboratories, Detroit, Michigan.

**Morphologic examination**

Full autopsy with macroscopic examination of all the organ systems was performed. Maceration and meconium staining were noted. Samples from most organs for microscopical examination were taken. Five samples were taken from each placenta: umbilical cord next to placenta, a sample from the middle of the umbilical cord, a roll of the free membranes was performed from the border of rupture to the border of the placenta and two samples from each placenta, both including chorionic plate and decidua basalis. The tissue was fixed in 4% formaldehyde, and embedded in paraffin. Sections were performed and stained with hematoxylin and eosin. The degree of inflammation in the tissues was estimated as follows:

- Grade 0 = <5 PMNs/high power field
- Grade 1 = 5–19 PMNs/high power field
- Grade 2 = 20–49 PMNs/high power field
- Grade 3 = ≥50 PMNs/high power field
(high power field = ocular 10, objective 40)

The grade of chorioamnionitis was regarded as the most severe one in any of the localizations.

**Statistical analysis**

The study was analyzed as a case-control study, regarding the stillbirths (n = 117) as cases and the healthy newborns (n = 126) as controls. Odds ratio (OR) and 95% confidence intervals (CI) were calculated. A confidence interval that did not include the unity value of 1.0 corresponds to p < 0.05 and was regarded as statistically significant.

**Results**

**Morphology**

Table I illustrates the severity of inflammation and the distribution of PMNs. Prevalence of PMN increased the risk for a stillbirth. For PMNs with maternal origin (in chorion plate, subchorionic fibrin layer and decidua basalis) the risk was significantly elevated; PMNs in decidua basalis was seven times as common in stillbirths as compared to controls.

Vasculitis in the umbilical cord was found in 20% of the stillbirths and 17% of the controls. Pneumonia was found in two stillbirths. One case with villitis and cytomegalic cell changes was found among the stillbirths (cytomegalic virus infection was verified by immunohistochemistry).

**Table I. Morphological findings of polymorphonuclear leucocytes (PMNs) by localization and grade in stillbirths (S) and controls (C). Odds ratio (OR) for the risk for grade 1–3 as compared with grade 0 and 95% confidence intervals (CI). Chorionic plate and subchorionic fibrin missing in one stillbirth**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Free membranes</th>
<th>Umbilical cord</th>
<th>Chorionic plate</th>
<th>Subchorionic fibrin</th>
<th>Decidua basalis</th>
<th>PMNs in any localization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S</td>
<td>C</td>
<td>S</td>
<td>C</td>
<td>S</td>
<td>C</td>
</tr>
<tr>
<td>0</td>
<td>35</td>
<td>51</td>
<td>94</td>
<td>105</td>
<td>79</td>
<td>101</td>
</tr>
<tr>
<td>1</td>
<td>43</td>
<td>48</td>
<td>14</td>
<td>13</td>
<td>31</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>19</td>
<td>5</td>
<td>5</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>8</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>1–3</td>
<td>82</td>
<td>75</td>
<td>23</td>
<td>21</td>
<td>37</td>
<td>25</td>
</tr>
<tr>
<td>OR</td>
<td>1.6</td>
<td>1.2</td>
<td>1.9</td>
<td>1.2</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.90–2.8</td>
<td>0.80–1.5</td>
<td>1.01–3.6</td>
<td>0.71–2.1</td>
<td>2.8–21.9</td>
<td>1.1–4.1</td>
</tr>
</tbody>
</table>
Table II. Bacteria isolated from different localization in stillbirths (S) and controls (C)

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Localization</th>
<th>No. of still-births with growth in organ</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placenta</td>
<td>Ears</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Coag.neg.staphylococcus</td>
<td>6</td>
<td>18</td>
</tr>
<tr>
<td>Enterococcus fecalis</td>
<td>22</td>
<td>1</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Group B streptococcus</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Proteus mirabilis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Proteus vulgaris</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Klebsiella pneumoniae</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lactobacillus</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Enterobacter cloacae</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Alpha streptococcus</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Hemophilus</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Candida</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Streptococcus microaerofile</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Streptococcus milleri</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Corynebacterium</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Group B betastreptococcus</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Citrobacter</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Enterobacter aerogenes</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

No. of subjects with growth 48 34 38 89 79 17 17 10 17 9 32
Total no. of subjects examined 115 126 116 126 116 116 114 115 115 115 116

Bacteriology

Table II illustrates the different bacteriological species isolated in cultures from different localizations.

In 60 (51%) cases bacteria were isolated in placenta and/or organs in stillbirths; in 37 (31%) cases one and in 23 (20%) cases multiple bacterial strains were isolated. The most frequent microorganisms isolated in stillbirths were *E. coli* and *Enterococcus fecalis*. Enterococcus fecalis was found in the placenta in 22 (19%) stillbirths, in one (0.8%) control (OR = 30; 95% CI: 4.1–600). The same bacterial species as in placental cultures were isolated in the organs. Coag.neg. staphylococci, Lactobacilli, Alph streptococci and *E. coli* predominate in controls, being more frequent in cultures from external ears than from placenta.

Comparison between the morphological and microbiological findings

Table III illustrates the most common bacteria isolated in placenta from stillbirths and controls. Most placentas with growth of these bacteria demonstrated chorioamnionitis. The frequency and severity of inflammatory response is presented.

Table IV illustrates associations between prevalence of chorioamnionitis and microbiological findings of the placentas. The risk for stillbirth was
Table IV. Comparison between occurrence of isolated bacteria and chorioamnionitis in stillbirths (S) and controls (C). Odds ratio (OR) and 95% confidence intervals for prevalence of bacteria and/or inflammation

<table>
<thead>
<tr>
<th>Inflammation</th>
<th>Bacteria isolated in placenta</th>
<th>Bacteria</th>
<th>S</th>
<th>C</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>Present</td>
<td>42</td>
<td>26</td>
<td></td>
<td>2.3</td>
<td>0.92–5.8</td>
</tr>
<tr>
<td>Present</td>
<td>Absent</td>
<td>53</td>
<td>60</td>
<td>1.3</td>
<td>0.54–3.0</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>Present</td>
<td>6</td>
<td>8</td>
<td>1.1</td>
<td>0.25–4.2</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>Absent</td>
<td>14</td>
<td>20</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of subjects</td>
<td></td>
<td>115</td>
<td>126</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Comparison between macerated and non macerated stillbirths

Maceration did not increase the risk for chorioamnionitis (Tables V and VI).

Meconium stained membranes

Prevalence of meconium discoloration of the free membranes increased the risk for a stillbirth in subjects with chorioamnionitis and bacteria in the placenta (Table VII). Out of 96 stillbirths with chorioamnionitis 25 had had meconium discharge but only six out of 86 controls resulting in a statistically significant OR = 4.7 (90% CI: 1.7–14).

Gestational age

No statistical significant difference in chorioamnionitis pattern was proven. Infarcts were more frequent among preterm (birth before 37 weeks of gestation) than term stillbirths (Table VIII).

Chorioamnionitis and placental infarcts

Deciduitis was more frequent together with infarcts than in placenta without infarcts (N=18; 12/31 vs 6/86 – p<0.0001) (Table VIII). Funisitis was found in 2/18 (11.2%) placenta with infarcts and in 21/99 (21.2%) without infarcts (p=0.50). The infarcted areas were occupying more than one third of the placental volume (Table VIII).

Discussion

There are few studies on the subject. Olding’s (Uppsala, Sweden, 1966, 16) and Quinn et al. (To-
Toronto, Canada, 1987, 3) studies in addition to studies performed in Zimbabwe (Moyo et al. 1996, 11) and Mozambique (Folgosa et al. 1997, 10) are most appropriate for comparison with our study. One study from Ireland briefly denied the role of intrauterine infection in stillbirth only on the basis of histological examination of the lungs (14). Other studies, dealing with morphology and bacteriology of the placenta, extended their investigations to prematurity (1, 5, 6, 17) or investigated only the prevalence of intrauterine infection in uncomplicated term pregnancies (12, 18, 19). Because of great variations in bacteriological techniques and different numbers of samples examined from placental tissue (from one to eight) as well as different evaluation of the inflammation by the pathologists a comparison is difficult. Bacteria were isolated in placenta in 41% of stillbirths and 26% of controls in our study. Figures from similar studies are usually lower (25% Olding; 26% Quinn et al.; 33% Moyo et al.; 28% Folgosa et al., regarding stillbirths; 7% Moyo et al., 28% Folgosa et al., 44%* Pankuch et al. regarding normal term deliveries).

Chorioamnionitis was registered in 82% of stillbirths in this study. Olding has found 71%, Quinn et al. 71%, Moyo et al. 79%, Folgosa et al. 96% in stillbirths with unexplained cause of death. The prevalence of chorioamnionitis in normal term pregnancies in our study was 68%. Varying prevalences were found in previous studies (Pankuch et al. 25%, Salafia et al. 54%, Dong et al. 85%, Moyo et al. 30%, Folgosa et al. 67%).

In our study the bacteriological findings explained the morphological chorioamnionitis in 50% of stillbirths and in 37% of controls. Interestingly, the predominating bacteria were the same as found in similar studies from developing countries: E. fecalis, E. coli, Coag neg Staphylococci, GBS and Staphylococcus aureus. More than one bacterial strain were isolated in 20%. Since GBS and E. coli and/or E. fecalis often were isolated together and from the placenta as well as from the internal organs, contamination is unlikely. There are several reasons why no bacteria were isolated in some subjects with chorioamnionitis. We did not include special growth media for fastidious agents like Mycoplasma species and Chlamydia trachomatis. Bacterial culture of the placental tissue is not the optimal site for recovering of the bacteria. Cultures from the space in between the fetal membranes seem to be more adequate (5). A slight inflammatory infiltration of the free membranes might be caused by the normal vaginal flora without subsequent spread to the placenta. The bacteria might have been killed by the inflammatory response. There may be a patchy distribution of bacteria.

In eight stillbirths and ten controls bacteria were isolated unassociated with chorioamnionitis. Some of the bacterial species are probably contaminants such as Lactobacillus species, alpha streptococci, Streptococcus milleri and Coag. neg. staphylococci. In other cases pathogens such as Proteus species, E.coli and GBS might have caused the death before the inflammatory response was evoked and perhaps due to toxic agents.

The high frequency of chorioamnionitis and isolated bacteria in controls is remarkable. Bacterial invasion and chorioamnionitis during the normal term delivery might be a physiological phenomenon, playing a role in the regulation of parturition.
being the cause of or the result of labor (20). The bacteria might be introduced into the uterus with seminal fluid during coitus (4). Repeated vaginal examinations may influence the vaginal flora and introduce bacteria in the uterus. Mucous plug in the cervix of pregnant women is a barrier for bacteria; therefore losing it (e.g. multiparous women) might permit the bacteria to spread freely (4).

Whether or not a microorganism will cause disease depends on its virulence and on the defence abilities of the host. That's why highly pathogenic bacteria do not cause disease in some individuals meanwhile in others bacteria of low virulence may be harmful. In our study chorioamnionitis was a common finding both in stillbirths and controls, however, with some morphological differences: inflammation in stillbirths was more frequent and more severe and an inflammatory reaction in the decidua basalis was more frequent compared to controls. The spread of infection to the decidua basalis may induce decidual necrosis due to bacterial toxins or cytokines from inflammatory cells (21). Consequent coagulation disturbances, ablatio and infarcts may occur (4). Placentas with infarcts had an increased frequency of deciduitis, which indicates that an inflammatory process in decidua basalis may induce decidual necrosis due to bacterial toxins or cytokines from inflammatory cells (21). Consequent coagulation disturbances, ablatio and infarcts may occur (4). Placentas with infarcts had an increased frequency of deciduitis, which indicates that an inflammatory process in decidua basalis may trigger the development of infarcts, placental insufficiency and stillbirth. It is worth noting that in no case was the inflammation with PMNs restricted to the decidua basalis and the PMN infiltration was differentiated from the regularly appearing lymphocytes of the decidua.

Endotoxins of Gram negative bacteria such as E. coli, Proteus and Pseudomonas may cause vaso-dilatation, intravascular coagulation, endothelial damage and capillary exudation both in placenta and fetus. We found growth of E. coli in placenta and/or organs in 19 cases (16%) and of Proteus in two cases (1.7%) among the stillbirths.

Vasculitis of the umbilical cord and chorionic vessels is a proof of a fetal response to infection. The frequency of funisitis was equal in stillbirths (20%) and in controls (17%) in this study. The results are variable in other studies (6%* Olding, 7.9%* Rayburn et al., 21%* Folgosa et al. in stillbirths; 0% Salafia et al., 5%* Folgosa et al., 35.8%* Dong et al. in uncomplicated term cases; 39%* Romero et al. in preterm deliveries with intact membranes) probably due to different sampling and analyzing techniques. Vasculitis of the umbilical cord occurred with the same frequency in macerated and nonmacerated children, which means that infection started before the death. Vasospasm or thrombosis of the umbilical vessels secondary to the vasculitis might cause stillbirth.

Hypoxia of the baby caused by the complications of the chorioamnionitis (infarcts, ablatio, vasculitis) may evoke meconium discharge. Naeye has found that 64% of cases with meconium in the amniotic fluid were associated with acute chorioamnionitis (4). At worst meconium is aspirated by the child and meconium aspiration syndrome is associated with high perinatal mortality (22). Altshuler and Hyde (23) exposed segments of the umbilical vein to meconium in vitro, and vaso-constriction occurred immediately. This may explain why the meconium exposed baby suffers from cerebral hypoperfusion (23). Meconium inhibits the intracellular killing of bacteria and phagocytosis (24) and may therefore lead to progression of infection. Intrauterine death might occur due to anoxia. The macroscopic discoloration from meconium might even have gone away at the time of examination. Meconium is phagocytized by amniotic epithelial cells and macrophages in the chorion. Only macroscopic evaluation of meconium discharge was reported in our study.

Before the 30th week of gestation the important consequence of intrauterine infection is villous edema, which, when occupying more than half of the placenta, leads to compression of blood vessels and makes fetuses hypoxic (4). In our material we found no placenta with villous edema to any important extent.

The bacterial growth in the blood and inner organs of the stillbirths means that bacteriemia and sepsis developed, which might be another cause of death. Intrauterine pneumonia was proven only in two stillbirths.

Which findings are necessary for the conclusion that acute chorioamnionitis is the cause of stillbirth? A thorough investigation including a careful postmortem with microscopy should be performed in each stillbirth excluding any other obvious cause of death.

Due to facts stated above an otherwise unexplained stillbirth with bacteria isolated in inner organs with histological chorioamnionitis reasonably has been caused by infection. This corresponds to 28/116 (24%) stillbirths in our material.

Stillbirths with growth of pathogenic bacteria in inner organs without morphological chorioamnionitis may reasonably also have died from infection. Four subjects in our material belonged to this group corresponding to further 3% of unexplained stillbirths.

Otherwise unexplained stillbirths with microscopic chorioamnionitis beyond the membranes (i.e. deciduitis, chorionitis, subchorionitis, funisitis) also without isolated bacteria might have been caused by infection. In our material these cases were 50/117 (43%).

* Figures were counted by the authors of this article.
Thus, a large part of otherwise unexplained stillbirths might be due to ascending infections also in a high socio-economic population.

References


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