Chorio-amnionitis in relation to mode of delivery at term

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Summary

The incidence of inflammatory changes on histological examination in the placenta, membranes and umbilical cord of 50 infants born by spontaneous vaginal delivery were compared with those of 50 infants born by elective caesarean section before the onset of labour at term. Inflammation was significantly more frequent after vaginal delivery (28%) than after caesarean section (6%). This suggests that intra-uterine bacterial colonisation is uncommon before the onset of labour and it is argued that chorio-amnionitis in the vaginally delivered placenta occurred during labour.

An acute inflammatory infiltration of the placenta, membranes and umbilical cord occurs when bacteria spread from the cervix and vagina into the uterine cavity. The reported incidence of such an inflammatory response, often referred to as chorio-amnionitis, varies widely in different studies and may depend on several factors, including the duration of membrane rupture, the gestational age at the onset of labour, and the amount of obstetric intervention during labour.

In addition to complicating labour, it has been suggested that chorio-amnionitis is a cause of labour owing to the stimulation of prostaglandin synthesis in the inflamed membranes. If chorio-amnionitis is either a cause or a complication of labour, then it should often occur in vaginal deliveries after the spontaneous onset of labour. In contrast, it should be uncommon with elective caesarean section.

To test the hypothesis that chorio-amnionitis is uncommon before the onset of labour, the incidence of acute inflammation in the placentas of infants delivered by caesarean section was compared with those of infants delivered by spontaneous vaginal delivery at term.

Material and methods

The placentas of 100 consecutive infants born at term (37 - 42 weeks) were studied. Fifty infants were born by normal vaginal delivery at term. The duration of membrane rupture was less than 24 hours in all infants born vaginally and none of the mothers showed clinical signs of membrane infection.

The placentas were examined within 24 hours of delivery. A light microscopy was used to detect the presence of an inflammatory infiltrate. The polymorphonuclear leucocytes were stained with haematoxylin and eosin.

The chi-square test was used in the analysis of the data.
Results

A maternal inflammatory response (membranitis or subchorionic intervillositis) was present in 14 vaginal deliveries (28%) and 3 caesarean sections (6%). This difference was significant ($X^2 = 7.09; P < 0.01$). Of the 50 placentas delivered vaginally, membranitis was present in 12 and intervillositis in 9, with 7 placentas having both membranitis and intervillositis. Three of the 50 placentas delivered by caesarean section had intervillositis, of which 2 also had a membranitis. In all cases with a fetal response, a maternal response was also present.

In contrast, a fetal inflammatory response (funicitis or chorionic vasculitis) was present in the placentas of 6 vaginal deliveries (12%) and 3 caesarean sections (6%). This difference was not significant ($X^2 = 0.49; P > 0.05$). In the vaginal deliveries, funicitis and chorionic vasculitis were present in 4 and 5 cases respectively. In the caesarean sections, funicitis was present in all 3 cases, of which 2 also had a chorionic vasculitis. The infants were all clinically well without signs of bacterial infection at delivery.

A maternal inflammatory response was therefore significantly more frequent after vaginal delivery than caesarean section, but there was no significant difference in the incidence of a fetal response.

Discussion

During pregnancy the intra-uterine cavity and its contents are usually sterile and the closed cervix is able to prevent an ascending spread of bacteria from the vagina. However, in some cases bacteria are able to penetrate the cervical barrier and migrate between the layers of the peripheral membranes to reach the placenta. This evokes an acute inflammatory response by the mother in the chorion, amnion and placenta. Later the bacteria may colonise the amniotic fluid and stimulate an inflammatory response by the fetus in the umbilical and chorionic vessels. Only occasionally is the fetus infected. Therefore the newborn infant is usually clinically well after delivery despite the presence of pus cells and bacteria in the gastric aspirate at delivery and an inflammatory infiltrate of the placenta and membranes on histological examination.

The ability of the cervix to prevent the ascending spread of bacteria before the onset of labour is supported by this study where an inflammatory response in the placenta, membranes and umbilical cord was uncommon in pregnancies terminated by elective caesarean section before the onset of labour. In the 3 exceptions, dilatation of the cervix with exposure of the membranes to the vaginal flora before the onset of contractions may have allowed colonisation of the uterine cavity.

In contrast, chorio-amnionitis was present in the placentas of 28% of the vaginal delivery patients confirming that infection of the placenta and membranes is commonly associated with the spontaneous onset of labour and vaginal delivery.

It is thought that bacterial invasion of the amnion, chorion and decidua stimulates the production of prostaglandins, which initiates myometrial contractions. It is suggested therefore that bacterial colonisation of the placenta and membranes, often before membrane rupture, is a common cause of the onset of labour, especially in preterm deliveries.

The findings of this study — that a maternal inflammatory response is unusual before, but common after, labour and vaginal delivery — support the hypothesis that bacterial colonisation of the intra-uterine cavity may play a role in the initiation or maintenance of labour at term. However, the infrequent finding of a fetal response suggests that the chorio-amnionitis is of short duration as it had not progressed to the stage of amniotic fluid colonisation with inflammation in the umbilical and chorionic vessels. Therefore the inflammatory changes noted probably reflect bacterial colonisation of the placenta and membranes, occurring with dilatation of the cervix and exposure or rupture of the membranes during the course of labour.

It is concluded that chorio-amnionitis is uncommon before the onset of labour at term. In contrast, it is significantly more frequent after spontaneous vaginal delivery, suggesting that bacterial colonisation of the placenta and membranes frequently takes place during labour.

REFERENCES