052

THE PLACENTA IN BILIARY ATRESIA: POINTERS TO AETIOLOGY?

Caroline Zabiejag Zwick, Etienne Nel, Pawel Schubert, Samuel Moore
University of Stellenbosch, Cape Town, South Africa

Aim: Investigation of placentas of infants developing surgical Biliary atresia (2001-2014), for possible aetiological factors.

Methods*:* Placentas of infants who developed surgical Biliary atresia (2001-2014), were identified from 5338 pathologically evaluated placentas of high risk pregnancies pathologically evaluated and compared with unaffected infants in the same study (Ethics number S12/02/061A) . Specific features of cytomegalovirus(CMV), inflammation, chorioamnionitis, funisitis, umbilical cord vasculitis or chronic villitis and other abnormal features were investigated.

Main Results: Eighteen of the 38 children(47%) identified with biliary atresia could be matched with their maternal placentas . Of these 18 one was normal. There were no signs of cytomegalovirus in the affected placentas although it was identified in 2 of the controls.

In 6(33.5%) signs of acute chorioamnionitis (acute inflammation with neutrophils)were noted. versus an overall 12% (647) incidence in unaffected patients. In 2 of the 6 placentas, vasculitis of the umbilical cord was observed, although only 1 had signs of necrotising funisitis (possible Candida infection).One further patient had a massive infarct suggestive of previous viral infection, with some associated decidual vasculopathy.

Other abnormalities noted included a small for gestational age placenta (1), features suggestive of cord accident (1), uteroplacental hypoperfusion (6), abruptio placenta (1), chorangiosis (3) oedematous villi with thrombotic vasculopathy (1) as well as maternal hypertension not associated with pregnancy (15/18). None of the placentas had signs of chronic villitis.

Conclusion: All but one of the matched placentas were abnormal, suggesting a possible causative link between antenatal pathology and biliary atresia. No prenatal signs of CMV infection were noted , suggesting postnatal infection. In 6 (33.5%), signs of acute inflammation was identified due to maternal infection. Foetal tissues were involved suggesting a possible crossover of inflammatory cells with possible aetiological implications