

PLACENTAL PATHOLOGY FINDINGS IN FETUSES AND NEONATES WITH CONGENITAL ZIKA VIRUS INFECTION AND MICROCEPHALY

David Alan Schwartz¹

¹Pathology, Medical College of Georgia, Augusta, USA

The causal relationship between Zika virus infection in pregnancy, vertical transmission of the virus, and the development of intrauterine microcephaly and other fetal malformations is now well-accepted. Attention is now becoming focused on the potential mechanism(s) for intrauterine Zika virus transmission from an infected mother to her fetus. Because the placenta is a vital component in understanding the fetal response to intrauterine Zika virus infection, it is critically important that the pathology features of this fetal organ in cases of congenital viral transmission be accurately characterized. This presentation describes the spectrum of pathological findings from placentas of fetuses and neonates with microcephaly and the Congenital Zika Syndrome (CZS). Unlike most other TORCH infections, placentas from fetuses with congenital Zika virus infection are concordant in not having viral-induced placental inflammation. There is an absence of microscopic inflammatory lesions including villitis, intervillitis, chorioamnionitis or funisitis as a result of Zika virus transplacental transmission. In addition, there is no necrosis, thrombosis, endothelial damage or other vascular pathology. These findings are in marked contradistinction to placentas from most other TORCH infections in which maternal- and fetal-derived inflammatory abnormalities are typically present. Immunohistochemical stains in Zika virus-infected placentas reveal proliferation and prominent hyperplasia of placental stromal macrophages, termed Hofbauer cells, in the chorionic villi of infected placentas. Immunohistochemical staining and RNA hybridization demonstrate persistence of Zika virus infection of Hofbauer cells in 2nd and 3rd trimester placentas. These findings indicate that Zika virus placental infection induces proliferation and prominent hyperplasia of Hofbauer cells in the chorionic villi, but does not elicit villous necrosis, vascular pathology, or a maternal or fetal lymphoplasmacellular or acute inflammatory cell reaction. The significance and potential implications of these findings will be discussed.