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Placental Chorangiosis – A report of two cases with unusual associations and review of literature

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Abstract

Chorangiosis is a placental change that is associated with prolonged hypoperfusion or tissue hypoxemia. We report 2 cases of chorangiosis, one associated with maternal pleural tuberculosis and the other with a single umbilical artery.

Introduction

Chorangiosis (villous vascular proliferation) is a vascular lesion involving the terminal chorionic villi. It is commonly associated with various feto-maternal and placental conditions. We present two cases, one associated with maternal tuberculosis and the other with a single umbilical artery.

Case No. 1

A 26 year old primigravida diagnosed with pleural tuberculosis on antituberculous medications, was admitted to the hospital at 35 weeks gestation with history of mild fetal distress. She delivered a preterm, healthy baby and bits of placenta was sent for routine examination and culture.

The placental bits weighed 25 g and was grossly unremarkable. On microscopy, the villi showed more than 10 vascular channels per villous, increased syncitial knots and areas of calcification. No granulomas were seen and AFB culture was negative (Fig 1).

Case No. 2

The second case was a 26 years female primigravida with Rh negative blood group (husband Rh positive) who underwent an emergency cesarean section at 39 weeks for fetal distress. A healthy baby was delivered. Grossly, the placenta was nodular and fibrotic.

The placenta weighed 650 gms and measured 21x13x2cms. There was an eccentrically attached umbilical cord measuring 15cms in length. The fetal surface of placenta showed grey white areas. The cut section of umbilical cord showed a single artery and vein. Microscopically, the placenta showed enlarged villi with numerous vascular channels per villi, increased syncitial knots and areas of calcification (Fig 1).
Discussion

In normal placenta, chorionic villi rarely contain more than 5 vascular channels. The diagnostic criteria for chorangiosis was described by Altshuler in 1984, as the presence of a minimum of 10 terminal villi, containing more than 10 capillaries per villus in 10 medium power fields in at least 3 or more random, noninfarcted placental areas. The severity of chorangiosis can be assessed by determining the number of vessels within each villus and the placental area throughout which the vasculature is seen.

The differential diagnoses for chorangiosis include congestion, tissue ischemia and chorangiomatosis. In placental congestion, the vasculature is numerically normal. In tissue ischemia, there is associated shrinkage of the villi. In chorangiomatosis, the vessels have a thick wall containing actin-positive smooth muscle cells. Etiological factors associated with chorangiosis may be maternal, placental or fetal conditions. The maternal conditions include pre-eclampsia, eclampsia, diabetes mellitus, jaundice, syphilis, drug ingestion, urinary tract infection and women living in high altitudes. The placental associations are umbilical cord anomalies, single umbilical artery, abruptio placente, placenta previa, amnion nodosum and villitis due to rubella, cytomegalovirus, syphilis and Bartonella infection. The fetal factors are the presence of major congenital anomalies and an Apgar score of less than 5.

The pathogenesis of chorangiosis is thought to be hypoxic stimulus which causes excessive villous capillary and connective tissue proliferation probably due to the induction of growth factors. This condition is usually associated with increased neonatal morbidity and mortality.

Till date, to the best of our knowledge, no cases associated with maternal tuberculosis have been reported in the literature. The exact incidence of chorangiosis with single umbilical artery has not
been documented and seems to be a rare observation. There was no adverse neonatal outcomes in both these cases.

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