A case of umbilical cord hemangioma

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Abstract: We report a rare case of an umbilical cord hemangioma. The umbilical cord consisted of Capillaries and cavernous blood vessels and the hemangioma stressed umbilical arteries and vein. At 40 weeks of gestation, it resulted in intrauterine death of the fetus. Microscopically, there were lots of vessels with various thicknesses in the umbilical cord. Autopsy shows evidences of smothering. These findings indicate there may be a causal connection between the umbilical cord hemangioma and fetal death because of the impaired umbilical circulation.

Key Words: umbilical cord hemangioma, umbilical blood vessel, fetal death.

As an important part of fetal appurtenance, umbilical cord is the channel for gas exchange, nutrient supplies and metabolites excretion between the mother and the fetus. The pathological change of the cords will have Serious impacts on not only the fetus, but also the mother. Among all kinds of the pathological changes of the umbilical cords, umbilical blood vessel malformation is the most common structure abnormality. Usually, there are two arteries and one vein in a umbilical cord. However, in some cases, there are some unexpected vascular patterns found in umbilical cords. Among them, fetal umbilical cord hemangioma is a rare kind of umbilical vessel abnormalities. It originates from the remnants of embryo hemangioblast and turn to vessel-like structure. These thin-walled capillary proliferation originate from umbilical arteries, umbilical vein or vitelline capillaries [1]. We report a case of umbilical cord hemangioma that led to fetal asphyxia.

CASE REPORT

A gravida with cessation of menstruation for 40+6 weeks was sent to hospital preparing for delivery on March 5th, 2013. The fetal heart rate was 140 bpm. Both of the maternal pelvis and fetal presentation were normal. There was no uterine contraction activity. On the vaginal examination, the gravida’s cervix canal was found to be midposition and 70% of it was effaced with cervix still being closed. B ultrasonic showed fetal presentation of LOA, biparietal diameter of 96 mm, femur length of 73 mm and cardiac rate of 142 bpm. The anterior placenta was 30 mm thick and was found to be Maturity Level II. Amniotic fluid index was 80 mm. Electrocardiogram showed sinus rhythm and normal electrocardiogra. Hospital diagnosis showed “G2P0, LOA, intrauterine pregnancy 40+6 weeks”. At 14 o’clock on next day, Oxytocin challenge was tested to be negative. At 23 o’clock, a nurse reported she couldn't feel the fetal heartbeat. Doctors rushed off to the gravida only to find the fetus had been dead intrauterine.

Autopsy findings

Autopsy was operated on next day. The stillborn weighted 4.03 kg and measured 55 cm in length. He had grown normal and was at medium nutrition level. Head circumference measured 32 cm with 1cm long hair and
chest circumference measured 33 cm. Rigor mortis could be found at large joints of limbs. Livor mortis was reddish and located on the back of the body where had not been pressed. When pressed with a finger, it would fade slightly. Palpebral conjunctiva and bulbar conjunctiva hyperaemia corneal were mild cloudy. The isocorias were dilated with diameters of 5 mm. It was cyanotic on lips, nails and toes. No obvious damages were found on face, neck, chest or abdomen. The spine was normal.

Visceral post-mortem examination: The subcutaneous hemorrhagic region measuring 8 cm × 7 cm was found on the top of the head. The anterior fontanel measured 3 cm × 3 cm and the posterior fontanel measured 0.8 cm × 0.8 cm. No skull fracture was seen. No hemorrhage was found in epidural space, subdural space or subarachnoid space. The brain weighs 520 g with vascular congestion. There was no damage, bleeding or space-occupying lesions in the section of the brain. Hemorrhagic spots could be found on the surface of the lung. Hydrostatic test of lung was negative. Visceral post-mortem examination: The subcutaneous hemorrhagic region measuring 8 cm × 7 cm was found on the top of the head. The anterior fontanel measured 3 cm × 3 cm and the posterior fontanel measured 0.8 cm × 0.8 cm. No skull fracture was seen. No hemorrhage was found in epidural space, subdural space or subarachnoid space. The brain weighs 520 g with vascular congestion. There was no damage, bleeding or space-occupying lesions in the section of the brain. Hemorrhagic spots could be found on the surface of the lung. Hydrostatic test of lung was negative. The pericardial was intact and 2.0 ml pale yellow liquid was found in pericardial cavity.

The heart weighs 30 g with scattered hemorrhagic spots on the surface, especially at right edge of the heart and the atrioventricular junction. There was a coloboma measured 0.4 cm × 0.3 cm on the interatrial septum. Each of the cardiac valves was normal. The liver, gallbladder, kidneys, esophagus and stomach were normal, too. Placenta measured 20 cm × 18 cm × 2 cm with part of umbilical cord measured 44 cm attached. Both the fetal surface and the maternal surface were smooth. The other part of the umbilical cord, 20 cm long, was attached to the fetal navel. A dark red-black hemorrhagic region surveying 8 cm could be found, which was 10 cm far from the navel (Fig. 1A).

Microscopy: In subarachnoid space, vessels was extended with hyperemia and leakage hemorrhage was found, and pericapillary space became broader. Sheet hemorrhage could be found in the epicardium with focal myocardial gap widened and interstitial vascular hyperemia. Angiotelectasis and hyperaemia also could be found in lungs, hepatic sinusoid, spleen, renal interstitial, thymus and thyroid interstitium. In Wharton’s jelly, there were lots of hemorrhagic vessels with various thickness (Fig. 1B). The vessels, highly dilated, were connected in dendritic with endothelial cells in the lining of the blood vessels and few connective tissues in the interstitium. Venous congestion and arterial sheet hemorrhage were found in umbilical cord (Fig. 1C). The umbilical artery was squashed nearly occlusive (Fig. 1D). Angiotelectasis and hyperaemia also be found in placental interstitium. Immunohistochemical staining of coagulation factor (F)VIII and CD34 was performed, FVIII-positive cells were observed in the area of dense vessels and CD34 labelling cells were found to be negative (Figure 1. E-F).

**DISCUSSION**

Both of benign and malignant tumours are extremely rare lesions of the umbilical cord in clinical cases. Among them, angiomas, angiofibromas and angioavcarnoma are benign tumours [2]. Different from the malignant tumours, the benign ones have a special impact on fetus by vascular compression. Umbilical hemangioma are mostly located in distal third of the cord and mainly affect umbilical vessels and circulation as in our case [3]. They are often not of uniform size, vary in colour and surrounded by Wharton’s jelly. Coagulation factor (F)VIII and CD34 are often used as markers of vascular endothelial cells.

In the present case, immunohistochemical results shows positive of coagulation factor (F)VIII, indicating it is the vascular tissue.

Both clinical course and the result of autopsy support a sudden fetal death due to asphyxia. This is easily explained hemodynamically. The vessels have thinner and severely deformed blood-vessel walls. As time goes on, umbilical facial blood flow increases gradually. When the fetus is coming out from the uterus, due to the strong intermittent pressure in utero, capillaries of the umbilical hemangioma expand and abnormal vessels may burst easily without protection of amniotic fluid, finally leading to intrauterine asphyxia even fetal death [4]. There are also other reports point out umbilical cord torsion is another potential mechanism. What’s more, coupled with the proliferation of the hemangioma, the umbilical vessels could be more and more narrow and limited [5].

The poor blood circulation caused by umbilical hemangioma could also lead to other problems. There are references pointing out that umbilical thrombus could cause non-immune hydrops, polyhydramnios, fetal disseminated intravascular coagulation and fetal hydrops [6-9]. It is possible that these changes might lead to further reduction in umbilical circulation which has already been impaired by tumor compression. Many researchers speculate this additional reduction in umbilical blood flow would result in fetal demise. Some researches illustrate the umbilical hemangioma may be caused by trisomy 18 [10-11].

The morbidity and mortality rate of umbilical cord hemangiomas has been reported to be 35% [12], but till now we are still lack of sufficient evidence to specify how umbilical cord hemangiomas leads to series of severe consequences. In order to decrease the risk of umbilical cord hemangiomas, comprehensive and detailed evaluation of clinical events and stillborn fetus should be operated the sooner be better. Routine ultrasound, as the most popular detecting artifice for prenatal diagnose and regular reexamination, should be emphasized [13]. It is reported that amniotic fluid oc-fetoprotein concentration may also be a valuable reference on umbilical cord hemangiomas [5].
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References


