

## Chorioangioma of Placenta: A Rare Case of Near-Miss Mortality

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### About the Author



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### Introduction

Placental chorioangioma is the most common benign non-trophoblastic tumor of the placenta. It is derived from primitive chorionic mesenchyme and is typically

vascular accounting for approximately 1 % of all pregnancy [1]. Chorioangioma is the most common histological type of placental tumor. An increased incidence rate of chorioangioma is associated with maternal age, hypertension, diabetes, female sex, first delivery and multiple pregnancies. Placental chorioangioma is a relatively rare condition that can result in serious prenatal complications and adverse pregnancy outcome. It is usually symptomless and may be associated with serious maternal and fetal complications when it reaches the size of more than 5 cm, e.g., polyhydramnios, premature delivery, preeclampsia, fetal growth restriction, fetal distress, fetal anemia, cardiomegaly, non-immune hydrops, thrombocytopenia, consumptive coagulopathy, and infant fetal death [2]. Here, we report a rare case of placental chorangioma who presented with sudden maternal collapse.

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## Case Report

A 23-year female G<sub>3</sub> P<sub>2</sub> L<sub>1</sub> with 9 months amenorrhea came to our emergency with complaint of pain abdomen since 4 h. She had previous two uneventful vaginal deliveries. Her LMP was not known. She did not undergo any antenatal visit during this pregnancy. No investigations were available at the time of admission. On interrogation, no significant medical or surgical history was present. On examination, her general condition was fair, and she was conscious, cooperative and well oriented to time place and person. Mild pallor was present. She was hemodynamically stable with a pulse rate of 86 bpm, BP was 110/68 mmHg, afebrile, with 99 % SpO<sub>2</sub> with normal respiratory and cardiovascular function. On per abdomen examination, her fundal height was 34 weeks with cephalic presentation. Uterus was irritable, and FHR showed deceleration up to 80 bpm lasting for 20 s in post-contraction phase. On P/V-cervical dilation 6 cm with fully effaced and vertex (–2) station with bag of membrane present. As FHR was again dropping up to 60 bpm, ARM was done; liquor was meconium stained. Immediately after ARM, patient suddenly collapsed. Subsequently, her pulse was unrecordable. Patient became unconscious and unresponsive. Thinking it to be an embolic phenomenon patient was rushed to operation theater, resuscitation measures were started simultaneously in collaboration with anesthetist. Emergency LSCS was planned in view of fetal salvage. Patient was intubated, and inotrope noradrenaline infusion was started. Urgent arterial blood gas study revealed severe metabolic acidosis with pH 6.3. She was mechanically ventilated. Under general anesthesia, LSCS was commenced. On opening the abdomen, subcutaneous tissue was pale with no bleeding from any tissues. Uterus was also bluish and pale. A male baby of 1.4 kg with low Apgar score was delivered. Baby had no complications except hypoxia at birth. Subsequently, neonate was shifted to NICU. Uterus was flabby, so uterotonics were given to prevent postpartum hemorrhage. Actual assessment of hemostasis was not possible because patient was collapsed. Uterus was closed in single layer. As patient improved, her vitals were recordable, but oozing also started from several sites at uterus stitch line. Bilateral uterine artery ligation was done. Resuscitative measures were continued, and abdomen was closed. At the end of closure, her pulse rate was 110 bpm BP was 120/84 mmHg and SpO<sub>2</sub> 100 %. She was shifted to ICU on noradrenaline infusion. She was kept intubated in ICU for 24 h. She was transfused 5 packed cells, 4 units of fresh-frozen plasma, 2 units of random donor plasma, and 800 mg i.v iron was given. On

investigation, her hemoglobin was 8.2 gm%, platelet count was 1.1 lakh, FDP was positive, D-dimer 25,000, liver and renal function test and 2D-ECHO were normal. On examination, her placenta was abnormal with 3 nodular masses, the largest mass 10 × 7 × 2 cm another 6 × 4 × 2 cm and smallest 5 × 4 × 1.5 cm gray white to gray brown. The placenta was sent for histopathological examination. Postoperative period was uneventful except hematoma developed below rectus sheath and in subcutaneous tissue, which was managed by closed dressing.

## Histopathology Report

Cut sections from nodular masses show marked proliferation of capillaries and thin-walled vessels with area of hemorrhage and congestion suggestive of chorioangioma.

## Discussion

Benign placental vascular tumor has an incidence of approximately 1 % in all pregnancy. Vascular tumors were first described by Clarke in 1798. Since then, a number of related cases have been reported. These tumors have abnormal proliferation of vessels arising from the chorionic tissue. Three histological patterns of chorioangiomas seen are angiomatous, cellular and degenerate. Among these, tumor chorangiomas are usually asymptomatic (<5 cm). Larger tumor (>5 cm) acts as arteriovenous shunt and is associated with maternal and fetal complications such as polyhydramnios, preterm labor, growth retardation, hydrops and anemia. It can be diagnosed by ultrasound prenatally. Color Doppler may be used for differentiating degenerative fibroids, placental teratoma, placental hematoma from chorangiomas. Chorangiomas are found on the fetal surface of placenta around the umbilical cord insertion [3]. The pathophysiology for development of complications is uncertain. The most common explanation which relates pathophysiologic response is the transudation of fluid because of mechanical obstruction of blood flow and development of arteriovenous shunts which lead to fetal and maternal complications. The management of chorangiomas is conservative as they are mostly asymptomatic. Small tumors, if diagnosed on USG, are monitored at every 6- to 8-week interval while large vascular tumors are monitored with USG Doppler at 1–2 weeks. In severe cases, in utero interventions like ultrasound-guided laser therapy, endoscopic laser devascularization, alcoholic ablation, amnioreduction and indomethacin are done to

prevent perinatal mortality [4]. In our case, prenatal diagnosis of chorioangioma could not be done as patient was uninvestigated so the opportunity of antenatal diagnosis was lost. Our patient presented with IUGR with fetal distress. There was a rare coincidence of sudden maternal collapse. It has been not reported earlier as a complication of placental chorioangioma. As per clinical scenario and investigation, the probable cause of sudden collapse could be thromboembolic phenomena. As these tumors are of vascular origin, there is proliferation of fetal blood vessels, which are supported by scanty connective tissue and large arteriovenous shunt which could give origin to emboli directly in maternal circulation.

## Conclusion

Chorioangioma is a rare but challenging condition. Antenatal diagnosis of such cases is a must to prevent and manage complications associated with it. Hence antenatal booking of every woman in early pregnancy should be encouraged. To know the association of chorangioma with sudden maternal collapse, future case reports are needed.

## Compliance with Ethical Standards

**Conflict of interest** There is no conflict of interest.

**Human and Animal Rights and Informed Consent** This case report does not involve any research work involving human or animal. Informed consent for publication of this report has been obtained from the patients.

**Ethical Statement** Ethical clearance was obtained from the institutional ethical committee.

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