

# PLACENTAL CHORIOANGIOMA: A CASE REPORT AND REVIEW

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## ABSTRACT

*Placental Chorioangioma occurs in 1% of pregnancies and are generally asymptomatic. However, they are known to cause a number of complications which are detrimental to the mother, fetus or the neonate. A typical case where acute polyhydramnios precipitated premature labour and delivery of the fetus as a result of a large placental chorioangioma is presented. A brief review of the other possible complications are also included.*

*Keywords: Placenta, Chorioangioma, Premature.*

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## INTRODUCTION

The only primary non trophoblastic tumours of the placenta are the relatively common haemangioma and the extremely rare teratoma. Haemangiomas or chorioangiomas occur in approximately 1% of all placentas<sup>(1)</sup>. They are usually single tumours but can be multiple. The highest recorded number was 25 discrete tumours, each with its own pedicle as described by Fisher (1940).

## CASE SUMMARY

A 24-year-old Chinese primigravida was seen by a private obstetrician since two and a half months amenorrhoea. An ultrasound scan done at her first antenatal visit and at subsequent regular intervals showed good growth.

The pregnancy was uneventful until 27 weeks of gestation when she developed acute hydramnios. The abdomen was tense and grossly distended. An ultrasound scan revealed that the fetus was in a flexed breech presentation. The fetal biometric parameters were equivalent to dates and no fetal abnormality was seen. The placenta was in the upper anterior segment and had an irregular growth on it. Blood flow pattern within this growth was consistent with that of a placental chorioangioma.

In an attempt to prevent premature labour, she was admitted on the same day for rest and treatment with diuretics (frusemide) and tocolytics (salbutamol). However, the next morning there was spontaneous rupture of the membranes and an in-utero transfer was made at our unit. The chorioangioma was again confirmed by ultrasound on arrival (Fig 1).

As her labour could not be inhibited, an emergency lower caesarean section was performed for premature breech. A baby girl 1095 g was delivered. Apgar was 3 and 7 at 1 and 5 minutes respectively.

The placenta measured 21 x 15 x 4 cm with an eccentrically placed umbilical cord. There was a nodular lesion just adjacent to the umbilical cord on the fetal aspect of the placenta measuring 12 x 8.5 x 4 cm (Fig 2)<sup>(2)</sup>. Cut section of the lesion showed a nodular area with haemorrhagic cystic changes containing mucoid substance and fleshy looking tissue. The rest of the placenta did not show any area of infarction. The umbilical cord had three vessels. On microscopy, the

chorioangioma was made of capillaries separated by oedematous stroma. Extramedullary haemopoiesis was seen (Fig 3 & 4)<sup>(3)</sup>.

Fig 1 - Ultrasound picture of the placental chorioangioma

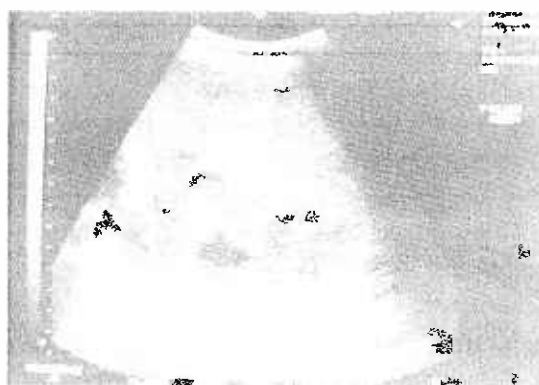


Fig 2 - Gross appearance upon delivery of the placenta - showing good correlation with ultrasound picture.



## Gross Pathology

Large haemangiomas as seen in the above case measuring more than 5 cm in diameter are rare and appear as bulging protuberances on the fetal surface. A minority occur on the maternal aspect where they may appear to replace the whole or part of a lobe. These large haemangiomas (as shown in the pictures) usually have a purplish red, glistening, encapsulated, smooth or bosselated outer surface which is sometimes deeply grooved by bands of fibrous tissue; they may be round, ovoid, or reniform and on section appear highly vascular.

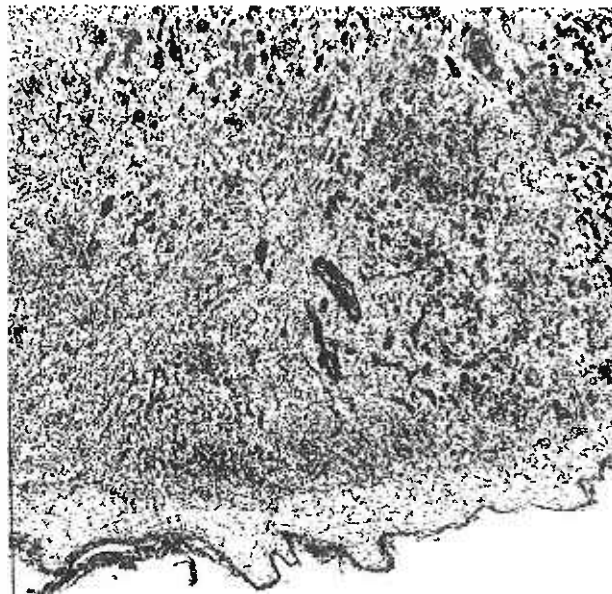
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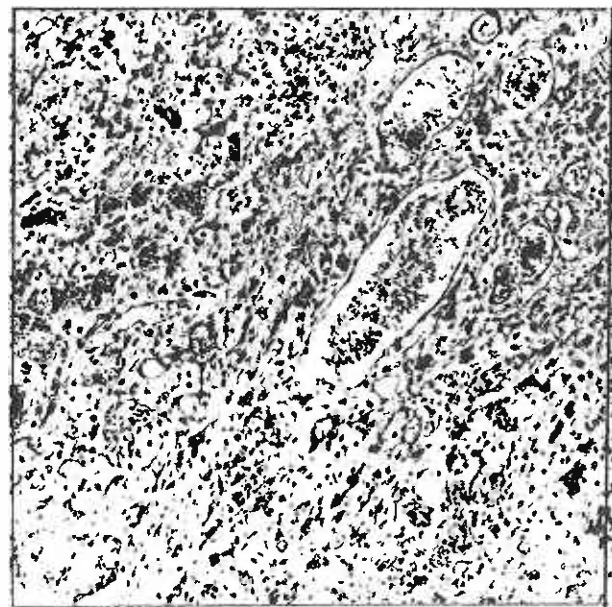
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Most placental haemangiomas are small and within the placental substance and hence are unlikely to be noticed unless the placenta is systematically sliced. These small intraplacental tumours are usually round and well demarcated from the surrounding normal villous tissue by an easily visible capsule; their cut surface is smooth and firm and may be yellow, brown, tan, red or white.

**Fig 3 - H&E x 20.** The tumour is covered by trophoblast on one surface and is made up of multiple, small vascular channels.



**Fig 4 - H&E x 100.** There is a network of capillaries of varying calibre.



#### Histology

Typical placental chorioangiomas have a microscopic appearance similar to that seen in haemangiomas elsewhere in the body, with numerous blood vessels set in loose scanty fibrous stroma. The vessels are usually small and of capillary size; marked dilatation which give rise to a cavernous appearance may occasionally be encountered. Sometimes, there is a predominance of the stromal component with only a few ill-formed vessels set in abundant loose, immature, cellular mesenchymal tissue. Degenerative changes, such as necrosis, calcification,

myxoid change, hyalinisation or even fat accumulation (Reddy et al, 1969), may complicate and confuse the histological picture.

#### Nature and Origin

The angiomatous nature of these tumours has been confirmed by electron microscopic and immunochemical malformation of the primitive angioblastic tissue of the placenta ie the placental mesenchyme. They arise in the early stages of placental development and therefore, have never been observed in placentas obtained from first trimester abortions.

#### DISCUSSION AND LITERATURE REVIEW

Most placental haemangiomas are of no clinical importance but those measuring more than 5 cm in diameter may be associated with a variety of complications which can affect the mother or the fetus.

#### Complications during pregnancy<sup>(4-7)</sup>

##### *Polyhydramnios*

Large chorioangiomas ie 5 cm and above are associated with polyhydramnios in about 30% of cases. The mechanism for the development of hydramnios is still unknown. It has been suggested that a tumour located near the cord insertion could cause mechanical obstruction of the umbilical vein leading to greater transudation of fluid. Increased transudation of fluid could also occur through the large vascular surface of the tumour on the fetal surface of the placenta. (Klabten 1929, Katz & Kaufman 1939). Maloney and Kelsey (1954) suggested that bypassing of fetal blood into the vascular tumour would stimulate an increased exudation of waste metabolites via the fetal kidneys resulting in increased fetal urine production and hydramnios. Benson and Joseph (1961) also proposed that chorioangioma might act as an arteriovenous shunt in the fetal circulation contributing to the development of hydramnios.

##### *Antepartum Haemorrhage*

Antepartum haemorrhage complicates 15 to 20% of cases. This can result either from a retroplacental haemorrhage from the tumour on the maternal aspect of the placenta or a rupture of the vascular pedicle of a pedunculated tumour, a catastrophe that may lead to fetal exsanguination.

##### *Premature Labour*

Premature labour is mainly precipitated by polyhydramnios or antepartum haemorrhage, as pregnancies without these complications usually proceed to term. It occurs in between 10 to 37% of cases ( Wallenburg, 1971)<sup>(8)</sup>.

Our patient presented as a case of acute polyhydramnios precipitating premature rupture of membranes followed by spontaneous onset of labour at 27 weeks of gestation.

##### *Complications during labour*

There have been exceptional cases in which a very large placental chorioangioma had obstructed vaginal delivery. (Emge, 1927). On rare occasions, the tumour became detached from the placenta during labour and was either passed separately per vaginum (Shaw-Dunn 1959, Bonneau et al 1968) or was retained in utero after the expulsion of the placenta with subsequent subinvolution and postpartum haemorrhage.

##### *Complications affecting the Fetus or Neonate*

Fetal complications include all the problems of a premature infant.

Fetal hypoxia and intrauterine growth retardation are thought to occur because of vascular shunting with the low pressure system of the angioma resulting in reduced perfusion of the chorionic villi affecting the exchange of nutrients and oxygen. Fetal compromise arising from pressure necrosis of the tumour on the normal placental tissue necessitating preterm delivery has also been described (Fox, 1967).

An increased cardiac output due to the chorioangioma act-

ing as an arteriovenous shunt may lead to cardiomegaly, cardiac failure and fetal death.

Fetal hydrops may result from cardiac failure or to hypoproteinuria caused by the transudation of protein from vessels of the tumour or to chronic fetomaternal bleeding.

Fetal anaemia could be caused by either sequestration of blood within the chorioangioma or a massive fetomaternal haemorrhage, or a microangiopathic haemolytic anaemia following destruction of fetal RBCs as they circulate through the chorioangioma.

Neonatal thrombocytopenia has also been described which has been attributed to platelet injury within the tumour vessels as well as a sign of DIVC triggered off by the release of a thromboplastic substance from the chorioangioma.

#### CONCLUSION

Keeping in mind all the possible complications, a routine antenatal screening ultrasound scan should include a screen of the placental composition and not just its location, to exclude the presence of a large chorioangioma. With prenatal diagnosis, more intensive monitoring of the pregnancy and perhaps means of decreasing the acute polyhydramnios may be attempted to decrease the high incidence of perinatal morbidity and mortality.

## BOOK REVIEW

# ACCESSORY CELL REGULATION OF B AND T CELLS

Immunological Reviews 1990 No. 117  
Munksgaard International Publishers Ltd

The Immunological Reviews series aims to publish comprehensive and analytical reviews within the fields of clinical and experimental immunology. The book fulfils this stated aim very well. The present volume deals with the various aspects of accessory cell function in humans and mice. The latest experimental findings are presented by various authors who are experts in their various fields. They are organised into nine different sections.

A reasonable background knowledge of immunology and experimental techniques is necessary as many of the technical terms and abbreviations are assumed to be understood by the reader eg TCR (T-cell Receptor), APC (Antigen Presenting Cell), LFA-3 (Leukocyte Function-associated Antigen-3), ICAM-1 (Intercellular Adhesion Molecule-1), Class II MHC restriction, stimulation of cells by LPS and PMA, Jurkat cells, etc. The main thrust of the series is aimed at the scientist/researcher who is working in this or a related field and hence the extensive technical and experimental details. The references given after each section are comprehensive and up-to-date.

The 9 sections, each a short review on its own, detail the roles of different accessory cells in the regulation of B and T

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cells. The accessory cells studied include macrophages, Langerhans cells, endothelial cells and follicular dendritic cells.

Experimental data presented show how accessory cells serve as antigen presenting cells that can convert antigen recognition into T-cell activation through engagement of T-cell surface molecules.

Regulation of B cells is reviewed in 2 of the sections. The question of stimulation versus tolerization was studied using a transgenic mouse model. Evidence was presented to suggest a major role for clonal paralysis in the induction and maintenance of tolerance.

In conclusion, the book is most suited for the serious scientist or researcher because of the large amount of experimental data in general. However this should not stop someone who just want to have an overall view of the subject but not to plough through large amount of unfamiliar data, from reading it. He can confine his reading to the well written and informative introductions and summaries of the sections. This can easily be achieved in a night's reading.

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