PLACENTAL HYPERTROPHY

(A Case Report)

by

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The morphological changes of placenta still pose a real challenge to the clinician and pathologist, and all attempts at intelligent interpretation in relation to the clinical syndromes and foetal outcome have so far resulted in confusion. The quaintly archaic terminology invading the vocabulary of placental pathology, the constant flush of histological changes in the placenta, the quantitative rather than the qualitative nature in the pathological significance: all have contributed to this perplexity facing the student of placental pathology (Fox 1975). Pooling of experience in relation to placental abnormality and clinical manifestations may help in negotiating the present state of impasse to some extent. Hence this unique case of placental hypertrophy associated with Pre-eclampsia and foetal abnormality is reported.

CASE REPORT

Mrs. S., a fifth gravida was admitted to RLSS Hospital, Marand, Iran on 21-11-76 complaining of oedema all over the body since one week, following 8 months' amenorrhoea.

She was married since 7 years. The past obstetrical history is given in Table I.

General examination revealed a fairly nourished woman with oedema and puffy face.

TABLE I
Obstetric History

S. No.	Year	Duration	Place of birth	Outcome
1.	1971	Full-term	Hospital	Male. Died in 7th month
2.	1971	8 Months	Home	Male. Died on 2nd day
3.	1973	Full-term	Hospital	Male. SB.
4.	1975	8 Months	Home	Male. SB.

Pulse rate was 76 per minute and the blood pressure was 150/100. Abdomen showed pitting oedema. The uterus was 36 weeks' size. Foetal parts could not be made out. Foetal heart sounds were heard.

Routine Investigations: Blood Grouping and Rh Typing: 0 +. Haemoglobin: 12 G/dl. Urine. Albubin + sugar: nil. Microscopy: nil abnormal. VDRL: Negative.

A provisional diagnosis of twins with pre-eclampsia was made and the patient was treated conservatively with sedation, diuretics and rest in bed.

A plain X-Ray Abdomen was taken on 23-11-76, which revealed a single baby presenting as breech.

Since she did not respond to the sedative line of treatment, she was put on hypotensives and on alternate days on diuretics from 24-11-76. On this regime the blood pressure came down to 130/90, and the albuminuria disappeared. The oedema persisted.

On 28-11-76 at about 3.50 p.m. she started labour pains and vaginal bleeding. On examination the BP was 140/100. Uterus was acting. FHS were heard. On vaginal examination the dilatation of cervix was 3 cm and the membranes were present. Because of the APH, the mem-

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Accepted for publication on 11-5-1978.

branes were ruptured and clear liquor was let out. Sedation was given. Seven hours later at 10.45 p.m. a still-born male baby was delivered as assisted breech.

The baby showed microcephaly and ascites and weighed 2.9 Kg. Ten minutes later a huge placenta was delivered by Brandt-Andrew's method. There was moderate PPH which was controlled by giving syntocinon drip.

Description of placenta. The placenta was completely circummarginate in type. Cord was centrally inserted. Umbilical vessels were normal. The extrachorialis portion appeared polypoidal along the circumference (Figs. 1 and 2). The weight of placenta was 2 Kg.

Microscopical examination of the placenta:

1. Section from the normal looking area near insertion of cord showed extensive fibrosis with hyalinization. There was scanty round cell infiltration. The blood vessels showed focal areas of intimal proliferation and obliteration of lumen.

2. Section from polypoidal area looked normal with only scanty fibrosis. No vascular changes or cellular infiltrate were seen.

3. Sections were stained for spirochaete and were negative. Postpartum period: The blood pressure came down to 120/80 by 30-11-76. Oedema also cleared. The patient was discharged home on 5-12-76.

Discussion

Placental hypertrophy has been recorded in cases of diabetes, pre-eclampsia, Rh Incompatibility and syphilis (Fox 1975). In the case reported pre-eclampsia might have been the cause of the hypertrophy. The histological alterations in the central area causing a functional deficiency might have induced the hypertrophy which manifested as polypoidal extensions along the periphery of the placenta.

The embarrassed foetal circulation with resultant anoxia in early life though not sufficient to compromise the life of the foetus, might have been critical enough to cause the foetal abnormality. The hypertrophy was able to sustain the malformed foetus which at 34 weeks weighed 2.9 Kg. The present case is unique in the sense that such a heavy placenta has not been reported so far.

Summary

Unique case of placental hypertrophy is reported. This appears to be the heaviest placenta so far reported.

Acknowledgement

I wish to thank Prof. Haneez, the Pathologist of RLSS Hospital, Tabriz, Iran for the HPE Report.

References

1. Fox, H.: J. Obst. & Gynaec. India. **25**: 441, 1975.

See Figs. on Art Paper V