

PATHOLOGY OF THE UMBILICAL CORD IN HYPERTENSIVE DISORDERS OF PREGNANCY

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SUMMARY

This paper studies the pathology of the umbilical cord in hypertensive disorders of pregnancy in 60 toxæmic patients. Cord haemorrhages were found in 28 patients and could be correlated with the degree of hypertension, the placental pathology and the foetal outcome. Of the 6 patients with a hypertrophic media in the cord vessels, on histopathology, only 2 patients had a normal foetal outcome.

Introduction

It is a cliché of obstetrical writing that the umbilical cord is the 'lifeline of the fetus'. Despite the obvious truth of this statement it is extraordinary how little attention has been paid to lesions of the cord. Studies of the umbilical cord are extremely rarely done, and it is sufficient to comment that the last detailed study of cord pathology to be published in an English obstetrical journal appeared over 50 years ago.

The current study seeks to study umbilical cord pathology in cases of toxemia of pregnancy.

Material and Methods

The study was conducted on 60 women who presented with toxemia of pregnancy. The criteria for diagnosis of pre-eclampsia were elevated blood pressure in excess of 130/90 mm Hg recorded for

the first time after the 28th week of pregnancy with oedema and/or albuminuria.

Their delivery record was carefully documented along with the outcome of pregnancy.

The placentae with the umbilical cord were collected soon after delivery and evaluated by both gross and microscopic examination. In addition to the 60 patients, 20 placentae with umbilical cords collected after delivery from 20 normal patients with normal full-term spontaneous vaginal deliveries were evaluated as a control group. The placentae with umbilical cord were examined macroscopically and then fixed on 10% formalin for 3 weeks. 8 whole thickness tissue blocks were taken from each placenta from both macroscopically normal and abnormal areas. The cord cut-section was examined at a point about 2 to 4 cm above its insertion into the fetal surface and cut sections taken from this point, and from the middle of the cord and other macroscopically abnormal areas.

The tissues were prepared by the procedure of making paraffin blocks. Sec-

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tions were cut at 5-7 micron thickness and stained with haematoxylin and eosin.

The placentae were graded according to the severity of placental affection based on the microscopic findings by the following scoring system:

Score 0: Normal placenta: no fibrin or infarcts

Score 1: Fibrin deposition +

Score 2: Both infarcts and fibrin deposition.

The foetal outcome was classified into 3 groups:

Group 1: Normal full term infants.

Group 2: Infants which were growth retarded; with low Apgar score or preterm.

Group 3: Stillbirths.

Results

On analysing the incidence of cord haemorrhage in this study, 46.6% of the patients showed some evidence of cord haemorrhage (Table I). Also, the rate was significantly higher in the group of cases with severe hypertension as against those with mild hypertension (Table I).

TABLE I

Cord Haemorrhage (Macroscopic)

Total cases: 60

Evidence of cord haemorrhage: 28

Control group: 20—No evidence of cord haemorrhage

Cord Haemorrhage and Hypertension Correlate

Blood pressure	No. of cases	Cases with cord haemorrhage
Mild (less than 160/110 mm Hg)	33	13 (30.4%)
Severe (160/110 mm Hg and above)	27	15 (55.6%)

Table II shows that the haemorrhage occurred more frequently in those cases

which had a compromised fetal outcome i.e. 54.5% and 50% in Groups 2 and 3 respectively as compared to 36.7% in Group 1.

TABLE II

Cord Haemorrhage (Macroscopic) and Foetal Outcome Correlate

Foetal outcome	No. of cases	Cases with cord haemorrhage
Group 1	30	11 (36.7%)
Group 2	22	12 (54.5%)
Group 3	8	4 (50.0%)

Where Group 1 = Normal fullterm infants.

Group 2 = Infants which were preterm; growth retarded or with low Apgar score

Group 3 = Stillbirths.

The incidence of cord haemorrhage was higher in those cases with an abnormal placental pathology (Table III). However, 40% of those patients with no evidence of placental pathology also showed some evidence of cord haemorrhage.

TABLE III

Correlation of Cord Haemorrhage (Macroscopic) with Placental Pathology

Placental Pathology score	No. of cases	Cases with cord haemorrhage
0	30	12 (40%)
1	4	2 (50%)
2	26	14 (53.8%)

The umbilical cord sections on being microscopically examined revealed the pathology in the cord vessels as shown in Table IV. Of the 6 cases, 4 (66.7%) had a severe placental pathology. Also, only 2 cases with the finding of a hypertrophic media had a normal fetal outcome.

TABLE IV
Histological Finding/Placental Pathology/Foetal Outcome Correlate

Histological finding	Placental Pathology score			Foetal outcome group		
	0	1	2	1	2	3
A. Hypertrophic media with haemorrhages in the media with obliteration of the vessel lumen	—	—	3	1	2	—
B. Hypertrophic media with haemorrhage in the media but no obliteration of the vessel lumen	1	1	1	1	1	1
Total	1	1	4	2	3	1

Discussion

One of the few detailed studies of cord pathology published in an English journal (Browne, 1925) appeared over 50 years ago. The aetiology of cord haematomata is as obscure as over 50 years ago. The bleeding may occur from a torn umbilical vein or rarely an artery. However, in the majority of cases no obvious large vessel lesion can be found and it is suggested that the haemorrhage at the fetal end of the cord may be from persistent omphalomesenteric capillaries which may have persisted at the extreme fetal end of the cord³ and therefore this explanation is not feasible for the placentae studied in this group.

It is possible that the blood may accumulate as a result of diapedesis of erythrocytes through weakened vessel walls,¹ or due to 'toxic' damage to the cord vessels, mucoid or fatty change in the umbilical vessels, a deficiency of Whar-

ton's jelly, or a haemorrhagic diathesis in the infant (Bret and Bardiaux).

The perinatal mortality rate in infants whose cord contains a haematoma is in the region of 40 to 50% (Fox, 1978) but it is far from clear whether, in such cases, the haematoma is responsible for fetal demise or whether both the haematoma and fetal death are mutually dependant upon some common causal factor.

Acknowledgement

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References

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