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The usefulness of limited placental sampling in stillbirths

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Abstract

144 placentas were sampled from all cases of stillbirth weighing 500g and above seen over a period of thirteen months in the UKM Unit of the Maternity Hospital, Kuala Lumpur. Sampling was limited to 1-3 blocks per placenta for histological study. Placental abnormalities were found in 121 (85%) placentas, 78 of which had definite lesions known to contribute to foetal death while the remainder showed lesions suggestive of an underlying disease. This study supports the usefulness of limited sampling of the placenta in the face of unavailability of complete placental examination and autopsy for assessment of the cause of stillbirth.

Key words: Stillbirth, placenta, histology

INTRODUCTION

Placental examination is an integral part of the perinatal autopsy. However, in Asia, cultural and religious beliefs may prevent examination of the placenta as it may be taken home by families for burial. This has proven to be a major obstacle in our efforts to establish clinicopathological causes of stillbirth in Malaysia, where the perinatal mortality rate is 25.1%' and permission for autopsy of the stillborn or the placenta is often not forthcoming. To overcome this we explored the usefulness of limited sampling of the placenta, confined to 1 to 3 blocks taken for histology.

MATERIALS AND METHODS

This study was conducted over a period of 13 months in the Universiti Kebangsaan Malaysia (UKM) Unit of the Maternity Hospital, Kuala Lumpur. Cases were included if the stillborn weighed 500g and above and if there was adequate clinical history. Samples of the placenta were taken by two assigned obstetricians. Full thickness blocks were taken from the centre and the edge of the placentas and in some cases, enface blocks were taken also.[2]. Each block measured approximately 50 mm x 60 mm. Histological sections were prepared from these blocks and examined using accepted nomenclature.[3,4,5] Histological findings were correlated with the clinical'information and the laboratory investigations. Lesions that were categorised as definite in this study were specific lesions that are known to have contributed to foetal death.[6] These included infarction, acute atherosis with infarction, retroplacental haematoma, chorioamnionitis, villitis, teratoma and arteriovenous malformation. Probable lesions were those that may or may not have contributed to the stillbirth such as acute atherosis without infarction, meconium-laden macrophages, foetal artery thrombosis and a single umbilical artery. Lesions that suggested the presence of an underlying disease were grouped under possible lesions, which are lesions showing Tenney-Parker changes, the presence of nucleated red blood cells. intervillous thrombosis, perivillous fibrin deposition, intervillous neutrophils, villous oedema, trophoblastic inclusions and chorangiosis.

RESULTS

Placenta samples from 144 stillbirths were obtained. All were suitable for histological studies. Histological evaluation revealed abnormalities in 121 cases (Table 1). The most frequent placental abnormalities were lesions indicative of uteroplacental vascular insufficiency, which included retroplacental haematomata, infarction, acute atherosis and Tenney-Parker changes; acute chorioamnionitis and villous oedema being other frequent findings.

Placental findings was supportive of impressions from clinical examination where available. Examples included the finding of

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TABLE 1: Placental abnormalities in stillbirth

Histological Changes	Definite	#Probable	#Possible
Uteroplacental vascular insufficiency			
– Infarction	*26		
– Acute atherosis	5	4	
– Tenney-Parker changes			7
– Retroplacental haematoma	*35		
Hypoxia changes			
– nucleated foetal red blood cells			5
– meconium-laden macrophages		4	
Other vascular pathology			
– Intervillous thrombosis			6
– Foetal artery thrombosis		5	
– Perivillous fibrin			8
Infection			
– Amniotic fluid infection	20		3
– Villitis	1		
Other lesions			
– Villous oedema			10
– Trophoblastic inclusions			3
– Chorangiosis			1
– Teratoma	1		
– Arteriovenous malformation	1		
– Single umbilical artery		1	
TOTAL	**89**	**14**	**43**

* Eleven cases had common histological changes.
# Fourteen cases from these two groups had overlapping histological changes.

retroplacental haematomata in 26 cases of clinical abruption, uterovascular insufficiency in maternal hypertension and acute chorioamnionitis in maternal vaginal infections in some cases. Villous oedema were seen in hydropic foetuses, twin-to-twin transfusion, storage disease and pre-eclampsia. Nucleated red blood cells were present in mature villi, dysmature villi, atherosis with retroplacental haemorrhage and foetal artery thrombosis and these associations are suggestive of hypoxia. Trophoblastic inclusions were seen in one case each of Trisomy, multiple congenital anomalies and congenital teratoma. The clinical abnormalities detected in the mothers and foetuses are listed in Table 2.

DISCUSSION

In limited sampling, findings may not be as representative as compared to extensive sampling? In abnormal findings, the extent of the lesion cannot be assessed because such lesions as infarction, perivillous fibrin deposition, Tenney-Parker changes and villitis, when focal, are of questionable significance. It has been claimed that infarcts involving less than 5% of the parenchyma or a perivillous fibrin deposition in less than 30% of its villi are of no clinical significance.[3] Although the extent of the infarcts in our 26 placentas could not be assessed, there were definite underlying lesions leading to the infarction, which is comparable to a study by Hovatta et *al.*[6] Most villitis are regarded as being the result of undiagnosed infection[8] which is true in our series as only one placenta which showed plasma cell villitis had a positive serology for syphilis. The incidence of acute chorioamnionitis as seen by several authors

TABLE 2: Clinical maternal and foetal abnormalities

Abnormalities	Number
Clinical Maternal abnormalies	
Hypertension	34
Infection	20
Anaemia	15
Antepartum hemorrhage	12
Diabetes mellitus	11
Multifoetal gestation	7
Post-date	7
Heart disease	2
Thyroid disorders	2
Others	12*
Foetal abnormalities	
Multiple abnormalities	13
Hydrop fetalis	3
Hydrocephalus	3
Anencephaly	2
Congenital syphilis syndrome	2
Twin-to-twin transfusion	1
Edward's trisomy	1
Imperforate anus	1
Storage disease	1
Congenital toxoplasmosis	1

* Cord accidents, birth trauma, uterine rupture, motor vehicle accident

varied widely and our incidence is similar to a study by Bernirschke and Clifford.[9] Microscopical retroplacental haematomas seen in 18% of our series is higher than other studies[6,8] and six of these cases were pre-eclamptic.[3] Acute atherosis which is one of the vascular abnormalities seen in pre-eclampsia[10] was present in the placentas of 5 of our pre-eclamptic mothers.

There were other placental changes observed which could be of significance. The presence of nucleated red cells in immature stillbirth is an expected normal finding. However, when present in the third trimester placenta, it is abnormal and could be related to hypoxia.[11,12,13] The presence of meconium-laden macrophages in the membrane is an indication that meconium had been discharged more than 3 hours[14] previously and suggested possible foetal asphyxia. Trophoblastic inclusions although now known to be present in normal placentas, are suggestive of chromosomal abnormalities[3] as seen in 3 of our cases. Chorangiosis is more common in perinatal death and chronic cord problems and may be the sequelae of malperfusion of the maternal vascular bed.[15,16] Our single case of chorangiosis was from a mother with pre-eclampsia with microscopical evidence of a retroplacental haematoma.

There were setbacks in some aspects of this study. Most of the placental tissue cultures for cytogenetic studies failed to grow. Some of the histological findings could not be correlated with clinical findings as the mothers presented with foetal death in utero without prior antenatal visits. The aim of this study was to see whether limited sampling was feasible and attempts at correlating it with other laboratory investigations was not systematically performed. Nevertheless, placental examination which is often neglected, is shown in our study to be useful and essential because autopsy examination is often not consented to. Based on the limited samples obtained, uteroplacental vascular insufficiency was the most frequent suggested cause of stillbirth, a finding similar to that of Rayburn et *al.*[7] We feel that histological examination of these limited samples can provide helpful information in counseling the parents and in planning future childbearing.

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