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THE HISTOPATHOLOGICAL CHANGES OF PLACENTA IN GROWTH RESTRICTED FOETUSES

ABSTRACT

INTRODUCTION

Foetal growth restriction is defined as a failure of the foetus to gain appropriate weight for a given gestational age. The weight of these foetuses at birth will be lesser than 10^{th} percentile for their gestational age. Foetal growth restriction(FGR) is one of the leading causes of perinatal morbidity and mortality. The placenta is the mediating organ between maternal and foetal circulations. Any imbalance in this system may result infoetal growth restriction. The causes of FGR can be grouped under maternal, placental and foetal factors.

Key words: foetal growth restriction, oligohydramnios, pregnancy induced pregnancy, umbilical cord abnormalities, maternal vascular malperfusion, foetal vascular malperfusion.

AIMS AND OBJECTIVES

- To describe the gross and histopathological changes in the placentas of growth restricted foetuses.
- To correlate the histopathological findings of placentas with the clinical and radiological presentations.

- To elucidate the importance of histopathological examination of the placentas of growth restricted foetuses for the effective clinical management of the pregnancies.

MATERIALS AND MATERIALS

All placentas of growth restricted foetuses received in the Department of Pathology, PSG IMS&R during the period between January 2015 and April 2017 were studied. A proformacontainingclinical details of the cases were sent to the histopathology laboratory along with the specimens. The other clinical details were also be got through hospital information system.

The specimens were grossed, processed, sectioned, stained and studied under microscope.

RESULTS AND ANALYSIS

Our study included 50 placentas of growth restricted foetuses. Of our study group, the placental findings in 48 cases, maternal co-morbidities in 35 cases and foetal cause (twin pregnancy) in 1 case were noted.

The predominant maternal co-morbidities associated with foetal growth restriction were oligohydramnios and pregnancy induced hypertension (PIH) that were seen in 17 (34%) and 14 cases (28%) respectively. About one-third of the cases showed combined clinical features of oligohydramnios with PIH, PIH with autoimmune disorders, PIH with GDM, abruption with PIH, etc.

Of the study group, umbilical cord abnormalities were found in 26 cases (52%). The most common placental changes identified were maternal vascular malperfusion in 32 cases (64%) and foetal vascular malperfusion in 10 cases (20%). Combined lesions of foetal vascular malperfusion with maternal vascular malperfusion (7 cases) and foetal vascular malperfusion with umbilical cord abnormalities (5 cases) were noted. There were villous capillary lesions- chorangiosis and chorangiomatosis (9 cases), villitis of unknown etiology (8 cases) and distal villous immaturity (6 cases). The other less common placental lesions identified were massive perivillous fibrin deposition (4 cases), acute chorioamnionitis (3 cases) and abruption (2 cases)

DISCUSSION

Maternal co-morbidities such asoligohydramnios and pregnancy induced hypertension occupied the predominant maternal causes. These causes may produce growth restriction in foetuses due to insufficient uteroplacental circulation.

We observed that hypercoiling of the cord followed by velamentous insertion were the two common umbilical cord abnormalities in our study population. Any gross abnormality of the cord may cause mechanical compression resulting in reduced blood flow to the foetal vessels.

Maternal vascular under/malperfusion (MVM) and foetal vascular malperfusion (FVM) were the most commonly found histopathological placental causes of FGR. Increased syncytial knots, increased perivillous fibrin

deposition and distal villous hypoplasia were the common pathological findings among the cases of MVM. The more specific predictors such as decidual vasculopathy that included acute atherosis or fibrinoid necrosis of the vessel wall were also seen in about one-third of cases. In FVM cases, the histopathological findings such as vascular ectasia, avascular villi, villous stromal karyorrhexis, nucleated RBCs and thrombi in foetal vessels reflect the foetal blood flow obstruction and the resulting placental hypoxia.

Although adverse perinatal outcome is distressing and not infrequent, examination of the placenta form a minor component of specimens received in the histopathology laboratory. Despite understanding the importance of placental examination, inhibition still exists in the minds of general pathologists to report placentas in routine practice. The complex and heterogenous lesions of placentas make it more difficult to handle for a general pathologist. Rapid progress in the understanding and characterization of placental lesions in the recent past solve this problem to a great extent.

SUMMARY AND CONCLUSION

- The study was done on placental examinations of 50 growth restricted foetuses.
- It provided distinctive gross and histological abnormalities in the placentas of growth restricted foetuses.

- Placental lesions with significant risk of recurrence in the subsequent pregnancy were studied that included DVI, VUE, MPVFD and histological chorioamnionitis.

Although, a number of causes produce foetal growth restriction individually, the severity depends on the cumulative effects of all the lesions. Hence, a meticulous examination should be done on all placentas submitted for histopathological analysis. Proper correlation with clinical details is also essential for a better understanding of the underlying pathology. It cannot be overemphasized that the effects of any form of therapy essentially depend on the accurate histopathological diagnosis.