Journal of Biology, Agriculture and Healthcare
ISSN 2224-3208 (Paper) ISSN 2225-093X (Online)
Vol.4, No.3, 2014

Morphopathology of human placenta in diabetic pregnancy

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Abstract

Normal foetal growth and survival depends on proper development and function of the placenta . The diabetic pregnancy is characterized by numerous disturbances in the fetal growth and development. The current study was undertaken to determine the effects of gestational diabetes on the morphology and histology of the placenta to confirm the magnitude of damage caused by diabetes to human placenta. Twenty five placentas of full term pregnancy were collected from Al-Hilla Teaching Hospital. In diabetic pregnancy, placental weight is higher in comparison to normal pregnancy, large size placenta, central thickness more than normal group, there is change of shape i.e. two lobes in one placenta. All other placenta were singly lobed ,two marginal attachment of umbilical cord ,The terminal villi in placentas of diabetes controlled on insulin showed the increased number of syncytial knots.the stroma of the villi demonstrated villous edema, fibrinoid necrosis, foetal capillary proliferation.

Keywords: *gestational diabetes, Morphometric , Placenta, terminal villi*

1. Introdution

The placenta is a maternal-fetal organ that separates the maternal and fetal circulations plays a central metabolic role in pregnancy.The placenta of diabetic women has attracted much interest, primarily because it is thought that placental damage may be partially responsible for the high incidence of fetal complications in pregnancies complicated by Diabetes mellitus (1) The diabetic pregnancy is characterized by numerous disturbances in the fetal growth and development. Fetal macrosomia, congenital malformation and intrauterine growth retardation are commonly seen in poorly controlled diabetes (2).Gestational diabetes increases the risk for fetal macrosomia and stillbirths along with increased frequency of maternal hypertension and caesarean delivery .These complications are attributed to the abnormalities of placenta (3). There is controversy regarding the risk of adverse outcomes associated with types of maternal glucose intolerance that are less severe than overt diabetes. (4) The effect of diabetic pregnancy facilitated anabolism reduced and hyper accelerated starvation. The need for insulin treatment in gestational diabetes (GDM) is associated with raised circulating pro insulin levels, implying1 that greater B-cell dysfunction leads to worse glucose intolerance, Woman with a previous history of GDM who become glucose-tolerance postpartum show continuing B-cell dysfunction, characterized by impaired insulin release in response to oral glucose, and impaired lypolysis despite normal insulin sensitivity. These points to a decreased B-cell function in GDM women, which make them susceptible to the future development of type 2 diabetes. Foetal hyper-insulinemia is the cause of macrosomia. Even mild disturbances of maternal carbohydrate metabolism can lead to fetal hyperinsulinemia. Placental examination is of critical value as it can be used in gathering Knowledge about management conducted during pregnancy, identification of etiologies and pathological process contributing to the adverse outcome of pregnancy and improving management in subsequent pregnancies (5). Maternal diabetes is associated with increased perinatal morbidity and mortality (increased incidence of congenital anomalies, macrosomia and intra-uterine foetal death) (6). The current study is undertaken to determine the effects of gestational diabetes on the morphology and histology of the placenta to confirm the magnitude of damage caused by diabetes to human placenta.

1.1 Materials and Methods

The sample is randomly taken inclusion and exclusion criteria was applied and consent was taken from the pregnant mothers .The placenta of full term pregnancy were collected from Labour Room and Gynaecology Operation theatre of Hilla Teaching Hospital (Babylon Governorate) .A total of 25 cases were studied.. Placenta with cord and membrane were collected immediately after delivery. Any abnormality of placenta along with the umbilical cord identified by corresponding code numbers were preserved in 10% formalin . The weight of placenta, newborn was noted in the labor room, the site of attachment of umbilical cord, vascular pattern of the chorionic blood vessels of placenta, the eccentricity index(EI) ,(CI)and (CCI) were calculated

according to Pathak 2010(7). For light microscopy, two centimeter of tissue was taken from the centre of each placenta and fixed in 10% formalin for one week .The tissue was dehydrated and followed by embedding in paraffin and 7 micron serial sections were generated with the help of rotator microtome. The tissue sections were stained with hematoxylene and eosin(8).

1.1.1 Results

The results of gross observation(table-1) revealed that magistral pattern was more common in the GDM controlled on insulin. While the dispersal vascular pattern of the chorionic blood vessels of the placenta was more frequent in the normal placenta. Also the mean number of cotyledons was 19.16 in GDM group .Site of umbilical cord insertion was centric (0 vs 2) eccentric (6 vs 6) ,marginal (0 vs 2) . The mean central thickness of placenta was 2.15 in GDM group compared with 3.50 in normal placenta group .The mean number of chorionic blood vessels of the placenta was more frequent in the normal group than GDM controlled on insulin group. The data in table (2) exhibit the umbilical coiling index /perinatal outcome ,. The umbilical cord without coiling was seen in 5(20%).the hypocoiled was in 3(12%) , the normocoiled was in 13(52%), and the hypercoiled 4(16%.).Also the anticlockwise coiling was more common. The pregnancy outcome revealed that the birth weight was $\leq$ 2.5 kg(n=2) within the hypocoiled ,while the apgar score was < 7 (n=2) within the hypercoiled.The morphometric measurements exhibited in table 3 shows that the mean birth weight of new born babies was 3.76 kg in the GDM controlled on insulin group and 2.80 kg in the normal group .Babies of mothers with GDM were mostly foetal macrosomia and Apgar score was below 7 in two newborn babies .Apgar score of the control group was within normal limits. The mean placental weight higher in the GDM controlled on insulin than in normal, while mean foetoplacental weight ratio were higher in the GDM controlled on insuliun . In GDM the microscopic picture of the villus structure of placenta was dysmature and persistant of cytotrophoblast cells which usually disappeared in full-term placentas of normal pregnancy. The tendency of syncytiotrophoblast toward formation of cluster (syncytial knots) was more than those in normal placentas with edema (Fig.1).Furthermore, the size of these Knots was increased also. The villi showed features of fibrinoid necrosis and hyalinized area (Fig 2)Fetal capillaries have usually increased in most villi but decreased in GDM(Fig.3). The terminal villi exhibit degenerative changes (Fig.4).

1.1.2 Discussion

The delineation of placental tissues in maternal diabetes has been also made unduly complex by other associated complications. In the present study, complicating factors were excluded, and the degree of control of diabetes was considered to be excellent. In our study on gross observation, there was no major difference in the placenta of diabetic group and normal were noticed .The weight of placenta and fetus in pregnancies complicated with diabetes was more as compared to the normal pregnancy and linear relationship was also maintained between fetal and placental weight at term. These observations are not agree with study in which the mean weights were similar in diabetic and normal pregnancy (9, 10). In our study have shown that many placentas are essentially normal, although in more than half, the placenta was larger, heavier and thicker than normal placenta of the same gestational age. Placenta weight reflects placental development and functions is correlated with maternal age, gestational age, history of maternal diabetes, birth weight, parity, route of delivery, infant's gender Apgar score and foetal distress.(11).In another study, it was observed that the placental weight and neonatal weight were increased, provided the diabetes was not complicated with vascular disease (12).

In the present study the dispersal vascular pattern of chorionic blood vessels of placenta was more frequently observed in the normal pregnancy while magistral pattern was more common in the placentas of GDM controlled on insulin. Usually, the dispersal vascular pattern the chorionic blood vessels of placenta were more frequent than the magistral pattern. Also it has been noticed that the site of attachment of umbilical cord eccentric or central in most of the placentas except in two cases in which there was marginal insertion of placenta in GDM controlled on insulin.

Research showed that there is a probable relationship between low and high umbilical vascular coiling with adverse perinatal outcome; therefore, it may be used to find the fetuses that are at risk. In the present study, neonatal weight of less than 2500 g associated with hypo coiled, and an Apgar score of less than 7 in 5 minute, associated with hyper coiled cord. In another study (13) non-coiling and hypercoiling were significantly more frequent with diabetic mother than with normal pregnancy. (14)The hypocoiled cords were related with obesity gestational diabetes and pre-eclampsia. In another study (15) on umbilical cords with abnormal coiling, they mentioned that fetal death, fetal intolerance to labor, were associated with abnormal coiling.

In the present study, the number of cotyledons in normal group ranged from 20-25.in diabetic group was decreased. This may explained by an altered distribution of fetal blood in complicated placenta resulting in

different modes of arrangement of intra cotyledonary vessels of complicated pregnancy. The placental weight reflects placental development and functions and is correlated with maternal age, gestational (38-39 weeks)age and parity (16). The birth weight gain in diabetic's placentas may be attributed to macrosomia and compensatory hyperplasia. Macrosomia affects the fetus and fetal part of placenta i.e. chorionic plate. .The macrosomia may be attributed to fetal hyperinsulinemia in response to hyperglycemia in fetuses of diabetic mothers(17). With direct light microscopic there were fibrin thrombi, villous edema, hyperplasia and thickening of basement membrane in placenta of poorly controlled diabetic mothers along with this various changes in structure of syncytio-trophoblast were mention by AL-Okail (18) and co-workers.Increashyalinized villous space is seen comparatively more than the normal placenta. The presence of hyalinized villi is in agreement with Majumdar et al 2005(19).

Conclusion: Clinicaly the adverse effect of diabetes on the outcome of pregnancy are well established but we have seen their gross morphological impacts on placenta. Significant changes in gross morphology have been observed in diabetes. Diabetic placenta showed increased in weight, central thickness diameter. One out of 25 placenta there is change in shape of placenta with tow lobes in one placenta in diabetic group .Site of insertion of umbilical cored is important but also the distribution of umbilical vessels in the placenta is equally important. Both dispersal and magistral type of distribution of umbilical cord blood vessels are found in GDM as compare to normal pregnancy.Microscopic examination exhibited, lesions like syncytia knots , fibrinoid necrosis, villous edema, villous fibrosis and capillary proliferation.

Acknowledgements

Thanks to all the clinicians and patients for their cooperation in the study.

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Table (1): Gross observation of placenta in diabetes (insulin) disease.

Observation		Diabetes (insulin)	Control	p-value (P≤0.05)
Mean of placenta weight (gm)		590	580	0.804
Vascular pattern (No.)	**Megistral**	8	0	0.020
	Dispersal	2	6	0.157
Mean of cotyledons per placenta		19.40	20.50	0.125
Site of umbilical cord attachment (No.)	**Centric**	2	0	0.564
	Eccentric	6	6	1.000
	Marginal	2	0	0.564
Mean of central thickness of placenta area (mm)		13.20	16.00	0.339
Number of blood vessels		4.80	5.16	0.496

Table (2): Umbilical cord index and neonatal/perinatal outcome (diabetes (insulin)).

Outcome	Hypocoiled (n=6)		Normal coiled (n=0)		Hypercoiled (n=4)	
	No.	%	No.	%	No.	%
Apgar scor (< 7)	0	0	0	0	2	50
Birth weight (≤2.5 Kg)	2	33.33	0	0	0	0
Gestation age (< 37 wks)	0	0	0	0	0	0

Table (3): Placenta morphometic study (diabetes (insulin)).

Observation	Diabetes (insulin))	Control	(P≤0.05)
Mean birth weight of babies (Kg)	3.47	3.76	0.33
Mean placental weight in gram	590	580	0.80
Mean foeto-placental weight ratio	5.81	6.42	0.01

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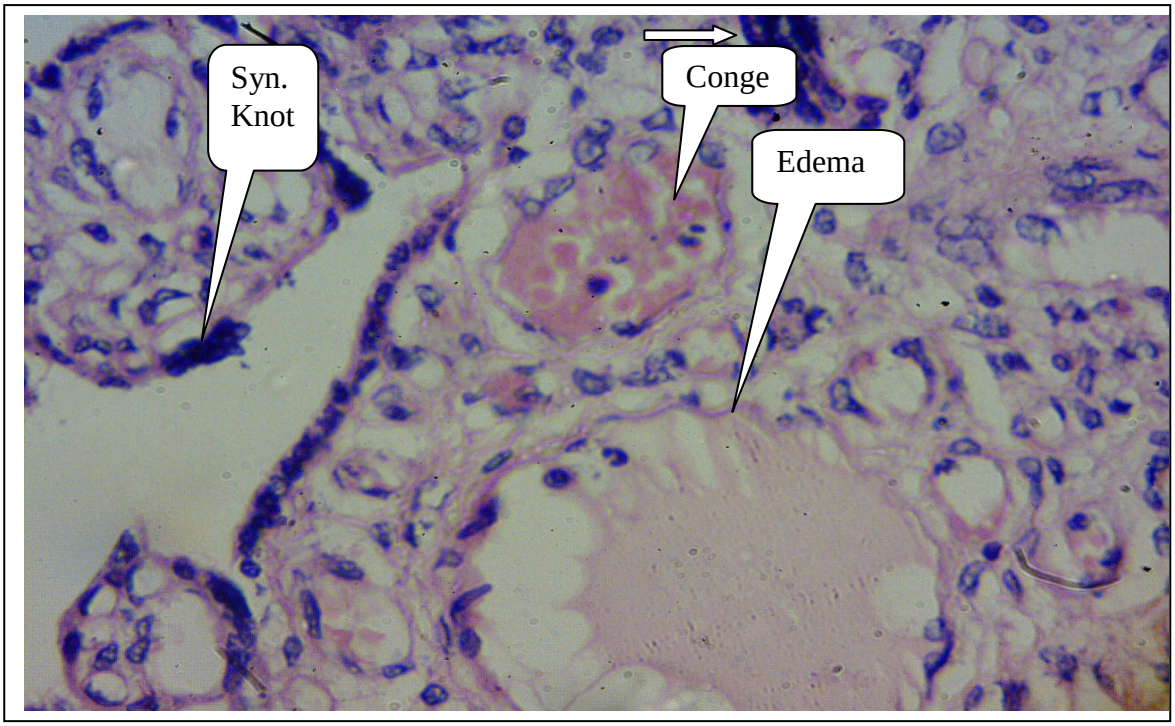
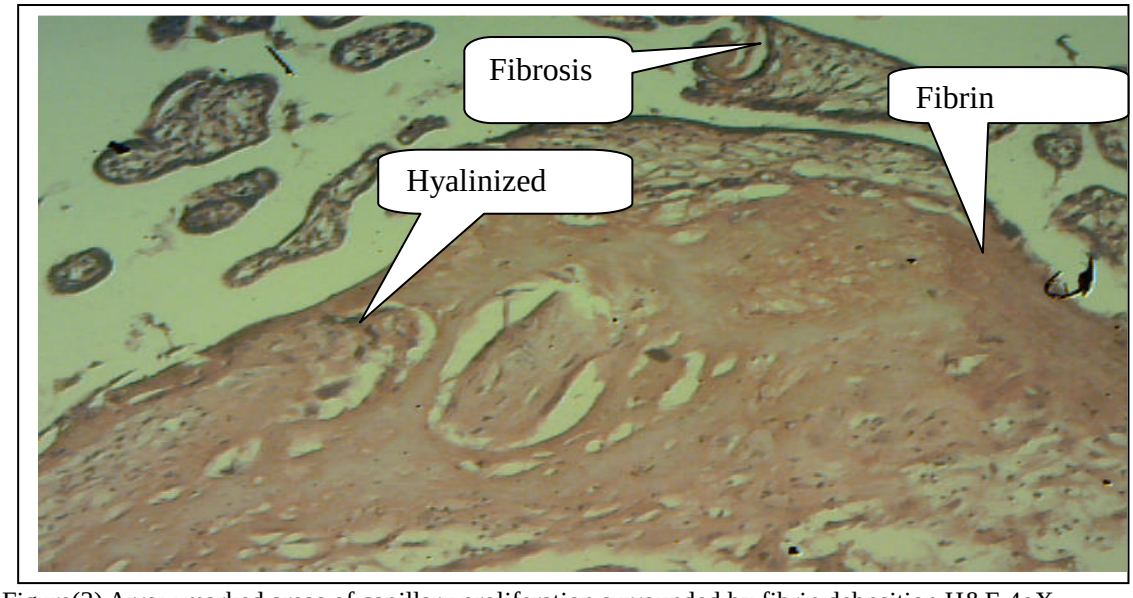
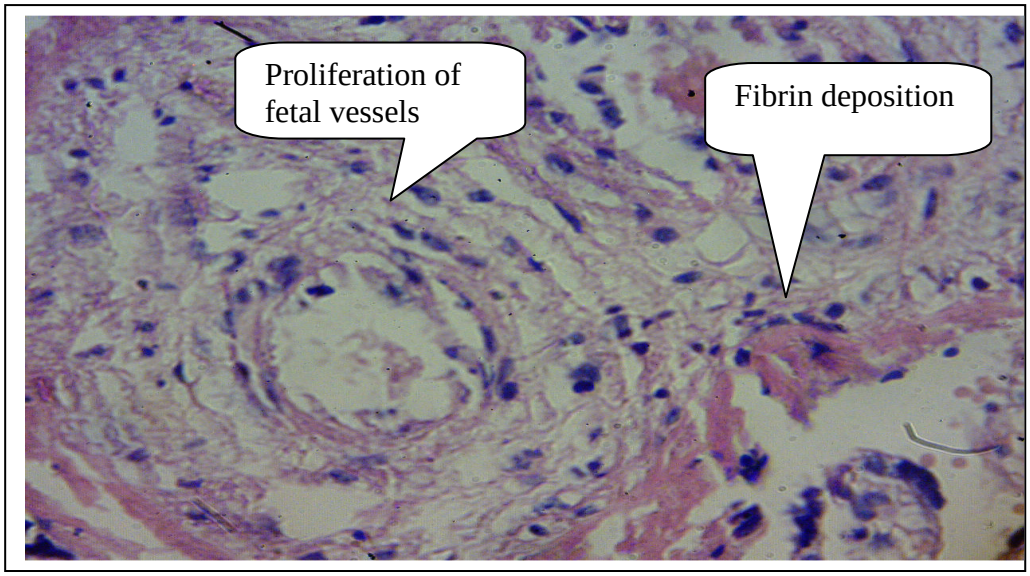
Figure(1) Chorionic villi in placenta from GDM showing edema congestion and syncytial knot.H&E 40X



Figurre(2) placenta of GDM . The core of villi showed an increased fibrin deposition , fibrosis and a thick vascular membrane .H&E 40X



Figure(3) Arrow marked areas of capillary proliferation surrounded by fibrin debpsition.H&E 4oX



Figure(4)Chorionic villi placenta of GDM showing the terminal villi with degenerative changes and fibrin deposition.H&E 40X