Original Research Article

Placental changes in patients of asthma and tuberculosis with relation to fetal outcome

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Abstract

Background: The potential benefits of placental examination include clarification of pathological features, improved management of subsequent pregnancies. **Aim:** To study the placental changes in patients of asthma and tuberculosis with relation to fetal outcome. **Material and Methods:** Among the 200 placentae from patients having medical disorders, 13 were from asthma patients and three were from tuberculosis patients. The cases of uncomplicated pregnancies not having any medical disorder in pregnancy were considered as control group. Patients with clinical diagnosis of Asthma (those using low or high dose glucocorticoids regularly and those not using glucocorticoids regularly for relief of symptoms) and tuberculosis were considered as cases. **Results:** The caseous lesions were seen in 66.66% of TB cases which were not seen in any of control group patients. Increased syncytial knots, fibrinoid degeneration and hypervascular villi were seen in 33.33% of TB cases Microscopic pathologies like fibrinoid degeneration, stromal fibrosis, hypovascularisation of villi were seen significantly in cases of asthma using glucocorticoids regularly for relief of their symptoms as compared to cases who use steroids occasionally for relief of their symptoms and control group. There was one case (33.33%) of perinatal death in tuberculosis patient. No perinatal death was seen in asthmatic cases. **Conclusion:** Exacerbations of asthma specifically increases the risk of adverse perinatal outcomes. The differential diagnosis of acute villitis and intervillitis in the placenta should include tuberculosis. The placental examination improves the management of subsequent pregnancies.

Key Word: Placenta examination, tuberculosis, asthma, fetal outcome

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INTRODUCTION

Placenta is essentially a fetal organ, which functions to support growth of the fetus, interact with two individuals, the mother and the developing fetus. Placenta is also a potent endocrine, immunologic and metabolic organ.[1] Placental findings in perinatal tuberculosis include chronic necrotizing granulomas in the decidua or endometrium, if the uterus is also examined. If sufficiently active and productive of mycobacteria, they are a potential source of fetal infection in the birth canal.[2] The placenta is a key regulator of fetal maturation and its function may be compromised by maternal asthma.[3]The potential benefits of placental examination include clarification of pathological features, improved management of subsequent pregnancies by diagnosing pathological conditions that may have risks of recurrence or may even be preventable or treatable.[4] The present study was conducted to study the placental changes in patients of asthma and tuberculosis with relation to fetal outcome.

MATERIAL AND METHODS

In this prospective study, a total number of 400 placentae were studied. Out of these 400 placentae, 200 placentae from normal term pregnancy were taken as control group and 200 placentae were from patients having medical disorder during or associated with pregnancy that were randomly selected.Among the 200 placentae from patients having medical disorders, 13 were from asthma patients and three were from tuberculosis patients. These 16 placentae were studied for morphometric changes and it's relation with fetal outcome. Detailed obstetric and

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medical history was recorded and clinical examination of the included cases were done. Necessary haematological and biochemical investigations were carried out.

Inclusion criteria

These were the cases of uncomplicated pregnancies not having any medical disorder in pregnancy**.** Patients with clinical diagnosis of Asthma (those using low or high dose glucocorticoids regularly and those not using glucocorticoids regularly for relief of symptoms). Patients showing acid fast bacilli in sputum or pleural fluid.

Exclusion criteriafor cases and control

Cases having multiple pregnancies. Cases having medical disorders ofpregnancy other than asthma and tuberculosis

Examination of placenta

The placentae were washed thoroughly with water, membranes trimmed and cord cut 4 cm from insertion. Gross examination of the placenta included dimensions and weight. Umbilical cord was examined for length, knot and type of insertion. Membranes were examined for type of insertion and colour. Fetal surface was examined for any abnormality. Maternal surface was examined for completeness, infarction, calcification and succenturiate cotyledons.Then the placentae were fixed in 10% formalin and sent for histopathological examination. After performing gross examination of placenta by pathologist, sections from placentae were taken. Blocks from placentae included central portion and the portion where pathology was suspected. Blocks were processed routinely. Paraffin embedded blocks were trimmed and cut into 3 to 5 micron thickness. Staining procedures were performed according to the technique described by Bancroft JD.[5] Microscopic examination of the placentae included villous pattern abnormalities, thickening of basement membrane, excess of syncytial knots, fibrinoid degeneration, villous vascularity, stromal fibrosis, congestion of capillaries, infarction and calcification. In cases of Tuberculosis, microbiological examination of placental abscesses was done with Ziehl and Neelsen staining for Acid fast bacilli. Examination of newborn included sex, weight, Apgar score at 1 min and congenital anomalies. Fetal outcome was compared with the placental pathology. Correlation was made regarding mode of delivery with placental pathology.

RESULTS

Out of 200 cases, 3 were suffering from Tuberculosis and 13 were suffering from Asthma. Out of the 13 cases of asthma, 6 (46.15%) were booked and remaining 7 (53.85%) cases were unbooked. Whereas, in cases of tuberculosis all 3(100%) cases were unbooked. Among control group 42% cases were unbooked. Most of the cases i.e., 8 (61.53%) were in the age group of 16-20 yrs followed by 4 (30.76%) cases in the age group of 21-25 yrs age group. Two (66.66%) cases from tuberculosis were in the age group of 21-25 yrs and one (33.33%) case was from 26-30 yrs age group. Four cases from asthma were primigravida and other were multigravida. Among tuberculosis cases, one was primigravida. One case from asthma had preterm delivery, whereas, 33.34% of cases having Tuberculosis had preterm deliveries. Maximum placental diameter of $\leq$15 cms was observed in Asthma (61.53%). All cases of tuberculosis had maximum placental diameter of $\leq$15 cms.In control group, 48% of patients had max placental diameter between 16-20 cms.

Table 1: Placental changes in Tuberculosis

Macroscopic changes	Control group (n=200)		Tuberculosis (n=3)	
	No.	%	No.	%
Infarction	22	11	0	*0
Calcification	40	20	2	66.66
Microscopic changes				
Syncytial knots	22	11	1	33.33
Cytotrophoblostic Proliferation	6	3	0	*0
Fibrinoid Degeneration	20	10	1	33.33
BM Thickening	12	6	0	*0
Stromal Fibrosis	16	8	1	33.33
Villous edema	0	0	0	0
Hypervascular villi	20	10	1	33.33
Hypovascular Villi	14	7	0	*0
Villous immaturity	28	14	0	0
Infarction	34	17	0	*0
Calcification	36	18	1	33.33
Endarteritis Obliterans	0	0	0	0
Caseous lesions	0	0	2	*66.66
AFB in placental lesions	0	0	2	*66.66

(* indicates significant p value < 0.05)

None of the Tuberculosis patients had infarcted areas on macroscopic examination while 66.66% of patients had calcified areas as compared to 11% and 20% in control group. Increased syncytial knots, fibrinoid degeneration, stromal fibrosis and hypervascular villi were seen in 33.33% of cases as compared to controls. Caseous lesions were seen in 66.66% of cases which were not seen in any of control group patients. Acid Fast Bacilli were recovered from 2 cases by Ziehl Neelsen Staining (Table 1).

Table 2: Placental changes in Asthma

Macroscopic changes	Control group (n=200)		Asthma(n=13) Not using steroids (n=9)		Asthma(n=13) Using steroids (n=4)		Total (n=13)	
	No.	%	No.	%	No.	%	No.	%
Infarction	22	11	1	11.11	2	50	3	23.07
Calcification	40	20	1	11.11	3	*75	4	30.76
Microscopic changes								
Syncytial knots	22	11	1	11.11	0	0	1	7.69
Cytotrophoblastic Proliferation	6	3	0	0	0	0	0	0
Fibrinoid Degeneration	20	10	1	11.11	3	*75	4	30.76
BM Thickening	12	6	2	22.22	2	50	4	30.76
Stromal Fibrosis	16	8	2	22.22	3	*75	5	*38.46
Villous edema	0	0	0	0	0	0	0	0
Hypervascular villi	20	10	3	33.33	0	0	3	23.07
Hypovascular Villi	14	7	1	11.11	3	*75	4	30.76
Villous immaturity	28	14	0	0	0	0	0	0
Infarction	34	17	0	0	2	50	2	15.38
Calcification	36	18	0	0	3	*75	3	23.07
EndarteritisObliterans	0	0	0	0	2	*50	2	15.38

Microscopic pathologies like fibrinoid degeneration, stromal fibrosis, hypovascularisation of villi and endarteritis obliterans, infarctions and calcifications were seen significantly in cases of asthma using glucocorticoids regularly for relief of their symptoms as compared to cases who use steroids occasionally for relief of their symptoms and control group. In control group, 32% had low birth weight. Among asthma group 4 (30.76%) cases had birth weight <2500 gms. All cases of Tuberculosis had birth weight <2500 gms. The mean birth weight in control group was 2619 gm. It was decreased in cases of asthma and tuberculosis which was 2769.23 gm and 2400 gm respectively. The feto-placental ratio in control group was 5.86 whereas it was increased in asthma and tuberculosis cases to 6.85 and 7.34 respectively.

Table 3: Comparison of fetal outcome in Asthma and tuberculosis cases

Fetal outcome	Low Apgar		Perinatal death		Uneventful	
	No.	%	No.	%	No.	%
Control group (n=200)	24	12	28	14	158	79
Tuberculosis(n=3)	1	33.33	1	33.33	2	66.66
Asthma(n=13)	1	7.69	0	*0	12	92.3

There was one case (33.33%) of perinatal death in tuberculosis patient. Low apgar score was seen in one case each from tuberculosis and asthma group. No perinatal death was seen in Asthmatic cases as compared to 14% in the control group.

DISCUSSION

Placenta is the only vital organ in perinatal life, which can be examined without hazards either to the mother or to the baby. The placenta is the paradox, as it is one of the most readily available organs for examination, yet one of the least studied.

Placental changes in tuberculosis

In our study, caseous lesions were seen in 66.66% of cases which were not seen in any of control group patients. Increased syncytial knots, fibrinoid degeneration, stromal fibrosis and hyper vascular villi were seen in 33.33% of cases as compared to controls.

Cantwell MF *et al* in their case report of 2 cases of congenital tuberculosis found the placenta showing Acid-Fast Bacilli and inflammatory cells in an intervillous thrombus.[2] Abramowsky CR*et al* described 2 cases of placental involvement with MTb in which an acute abscess-like inflammatory response with Myeloperoxidase and CD68-positive neutrophils and histiocytes causing acute villitis and intervillitis, with abundant acid-fast mycobacteria. They suggest that the differential diagnosis of acute villitis and intervillitis in the placenta should include tuberculosis aside from the more common bacterial infections such as listeriosis.[6]

Wong *et al* in a case report of atypical presentation of congenital tuberculosis in preterm infant have stated that placenta may develop caseous lesions in maternal tuberculosis and if these lesions burst into amniotic cavity may lead to congenital tuberculosis by aspiration of these contents.[7]

Placental changes in asthma

The effect of asthma on placental function has been reported by several authors. Stereological assessment of placental morphology shows that fetoplacental growth is compromised in pregnancies complicated by asthma.[8] Placentae collected after delivery from women with asthma show significant reduction in Corticotrophin Releasing Hormone (CRH)-induced vasodilatation in moderate and severe asthmatics. A systematic review and meta-analysis indicates that there are significantly increased moderate risks of poor perinatal outcomes in pregnant women with asthma compared to pregnant women without asthma.[9] This review also highlighted that some adverse perinatal outcomes (particularly preterm labour and delivery) may be improved by active asthma management, suggesting that prevention of exacerbations, maternal hypoxia or inflammation may be useful in improving pregnancy outcomes. Mayhew TM *et al* conducted the study to test for volumetric differences in placental composition between non-asthmatic pregnancies and those associated with maternal asthma grouped according to asthma severity and glucocorticoid (GC) treatment. They found that compared to non-asthmatic controls, asthmatics had reduced absolute volumes of fetal capillaries which were most marked in those with moderate/severe asthma and those using low and high doses of inhaled glucocorticoids.[3] Changes in the total length and mean cross-sectional area of capillaries and peripheral villi were also observed. Lengths were greater in mild asthmatics and lowest in those with high glucocorticoids usage. Calibre areas were lower in mild asthmatics and villous calibres in the high glucocorticoid group were greater than those in asthmatics not taking glucocorticoids. Those making greatest use of inhaled glucocorticoids also had villi which were hypovascularized in terms of capillary: villus length ratios. In conclusion, exacerbations of asthma are an important component that specifically increases the risk of adverse perinatal outcomes. The differential diagnosis of acute villitis and intervillitis in the placenta should include tuberculosis. The potential benefits of placental examination include clarification of pathological features and improved management of subsequent pregnancies.

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