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Original Research Article

Evaluation of clinicopathological profile of subjects with intrauterine fetal death

Sunil Kumar Pandey[1], Surabhi Porwal[2]*

[1]Department of Obstetrics and Gynecology, District Hospital, Satna, Madhya Pradesh, India
[2]Department Obstetrics and Gynecology, Gandhi Medical College, Bhopal, Madhya Pradesh, India

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***Correspondence:**
Dr. Surabhi Porwal,
E-mail: porwalsurabhi@gmail.com

ABSTRACT

Background: Intrauterine foetal death is an immense emotional burden for everyone concerned specially in the last trimester. Therefore, it is very important to find out, what has happened. Evaluation of clinical and pathological profile of women presenting with intrauterine foetal death has evoked great interest among the obstetricians.
Methods: The prospective observational study was conducted in the department of Obstetrics and Gynaecology N.S.C.B. Medical College Jabalpur during 1st June 2012 to 31st October 2013. A total of 155 intrauterine foetal death subjects admitted during this period were evaluated. Clinically and laboratory profile of subjects done. Histomorphology of placenta was performed in each case. Full HPR finding were then correlated with clinical and laboratory findings of subjects.
Results: Poor vascularity of villi and fever were significantly associated ($p<0.01$). Hypertension and Convulsion and fibrinoid necrosis, syncytial knot and placental infarcts were significantly associated ($p<0.001$) Premature placenta is associated with cytotrophoblastic layer ($p<0.01$). Conversely post mature placenta is associated with calcification and infarction. ($p<0.01$).
Conclusions: All placentae associated with foetal death have either gross or microscopic abnormalities. Present study is a step towards understanding and extrapolating the already known causes of intrauterine foetal death in the perspective of Jabalpur and its adjoining districts.

Keywords: Clinicopathological profile, Intrauterine foetal death, Placenta

INTRODUCTION

Intrauterine foetal death is a distressing situation for the obstetrician and a catastrophic loss to both the family and society. Intrapartum foetal death is usually the result of foetal distress and obstructed labour and reflects poor quality of clinical care. Majority of stillbirths (85-90%) in developed countries occur before the onset of labour, whereas this proportion is much lower and overall incidence of stillbirth is much higher in developing countries.[1] Only at later stages the important issue will be to understand and to know. To understand why their child was born dead and to know the risk for future pregnancies that most of them will experience. To know if there is anything they can do to avoid it.Globally, over 3 million babies are stillborn every year with the vast majority occurring in developing countries.[2] While less frequent in developed countries (<1% of births). Foetal death is related to maternal, placental or foetal complication.

Such a complication may be chronic (usual) or acute (rare) to produce placental insufficiency. Different placental pathology occurs in different gestational periods of pregnancy. Over 60% of foetal death (IUFD) are reported to have a placental cause of death.[3] Placenta is

unique and wonderful organ that arises de novo directly related to the growth and development of the foetus in the uterus. Being an organ of vital importance for the continuation of pregnancy and foetal nutrition, it has evoked great interest among the pathologists and the obstetricians as well, and much work has been done to understand the 'unique biological status, of this complex organ.[4]

METHODS

This prospective observational study was conducted in the department of Obstetrics and Gynaecology N.S.C.B. Medical College Jabalpur during 1st June 2012 to 31st October 2013. A total of 155 intrauterine foetal death subjects admitted during this period were evaluated. Written informed consent was obtained from all subjects. Data was collected including medical and obstetric history, maternal and foetal characteristics and pregnancy.

Laboratory profile of all subjects include routine investigations, sonography, maternal blood test include ABO and Rh grouping, VDRL, post prandial blood sugar and Hb AIC (Glycosylated Hb) for diabetes, routine urine examination, coagulation study to detect DIC, CBC as base line study. All subjects were thoroughly examined to exclude congenital anomaly or other foetal cause of death. Placental examination for villus immaturity and combine placental pathology was done. Statistical analysis was done by using t- test and chi-square test with the use of SPSS software.

RESULTS

This prospective observational study was conducted in the department of Obstetrics and Gynaecology N.S.C.B. Medical College Jabalpur during 1st June 2012 to 31st October 2013. A total of 155 intrauterine foetal death subjects admitted during this period were evaluated.

Clinically and laboratory profile of subjects done. Histomorphology of placenta was performed in each case. Full HPR finding were then correlated with clinical and laboratory findings of subjects.

Of the studied subject 76.77% of total subjects were from the age group 21-30 Years, the mean age of the studied subjects was 25 + 4.15 Years. Of the studied subjects 81.9% subjects were from rural area, 18.1% subjects were from urban area, 96.8% subjects belonged to low socioeconomic status, 3.2% subjects belonged to middle socioeconomic status, 32.9% subjects were illiterate, 28.4% subjects were educated up to primary,25.2% of subjects were educated up to middle,11% subjects were educated up to high school, 0.6% subjects were educated up to secondary and 1.9% subjects were graduates.

Of the studied subjects 94.84% of subjects were unbooked and 5.16% of subjects were booked. Of a total of 155 subjects 49.48% were primigravida and 55.32% subjects were multi gravida. Of the 155 subjects with IUD 92.26% subjects were delivered vaginally and 7.74% subjects by caesarean section.

Table 1: Frequency distribution of clinical presentation of intrauterine fetal death subjects by independent factors such as socio-demographic, booking, gravida and delivery status.

		Fever		Leaking		Bpv		Convulsion		Abdominal pain		Others	
		P	Ab	P	Ab	P	Ab	P	Ab	P	Ab	P	Ab
Locality	Rural	17	110	9	118	27	100	20	107	123	4	18	109
	Urban	4	24	2	26	3	25	3	25	28	0	1	27
SES	Low	20	130	11	139	30	120	23	127	146	4	19	131
	Middle	1	4	0	5	0	5	0	5	5	0	0	5
	Upper	0	0	0	0	0	0	0	0	0	0	0	0
Booking	Booked	2	6	1	7	1	7	0	8	7	1	1	7
	Unbooked	19	128	10	137	29	118	23	124	144	3	18	129
Literate	Illiterate	5	46	5	46	11	40	8	43	51	0	8	43
	Literate	16	88	6	98	19	85	15	89	100	4	11	93
Gravida Status	Primigravid	9	68	8	69	9	68	18	59	76	1	8	69
	Multigravid	12	66	3	75	21	57	5	73	75	3	11	67
Term/ Preterm	Preterm	17	104	6	115	26	95**	19	102**	117	4	10	111**
	Term	4	30	5	29	4	30	4	30	34	0	9	25

P: Present, Ab: Absent, *P<0.05; **P<0.01

Of the studied subjects 50.32% subjects foetuses delivered were males and 49.68% subjects were females. 47.74% subjects of 155 were term and 52.26% subjects were preterm. 80% subjects were low birth weight and 20% subjects were normal weight. Who were subjects from rural areas, low socioeconomic status, unbooked and were in preterm labour had increase frequency of clinical symptoms. Convulsions and bleeding per vaginal was more in primigravida subjects while only leaking in multi gravida. Significantly (P<0.01) less number of subjects with preterm delivery had no bleeding per vaginal. Similarly, convulsion was entry in significantly fewer subjects (Table 1). Of the 155 subjects 138 had adequate liquor, 14 subjects had oligohydramnios, 3 subjects polyhydramnios. 136 subjects had cephalic presentation, 12 breech and 8 with other presentation. Congenital anomaly present in 8 subjects. When BPV was a presenting complaint (n=26) 8 subjects had placenta in the lower segment whereas when BPV was absent then in only 2 subjects placenta in lower segment (P<0.0001) (Table 2). If fever (n=21) present then 8 subjects present with poor vascularity of villi and if fever (n=134) absent 17 subjects present with poor vascularity of villi (P<0.01). If convulsion (n=23) present 11 subjects present with syncytial knot, if convulsion (n=132) absent then 10 subjects present with syncytial knot (P<0.001). If convulsion (n=23) present then 21 subjects present with fibrinoid necrosis and if absent (n=132) then in 39 subjects fibrinoid necrosis present (P<0.001).

Table 2: Distribution of iufd between clinical finding and usg finding.

		Liquor			Presentation			Placental localization		Placental maturity			Congenital anomaly	
		Adequate	Oligo hydramnios	Poly hydramnios	Cephalic	Breech	Other (Transverse, Oblique)	Upper segment	Lower segment	Grad I	Grade II	Grade III	Present	Absent
Fever	P	20	1	0	18	3	0	19	2	0	17	4	0	21
	Ab	118	13	3	118	9	7	126	8	18	102	14	8	126
Leaking	P	8	3	0	10	1	0	11	0	1	9	1	0	11
	Ab	130	11	3	126	11	7	134	10	17	110	17	8	136
Bpv	P	29	1	0	28	1	1	22	8	6	22	2	0	30
	Ab	109	13	3	108	11	6	123	2***	12	97	16	8	117
Convulsion	P	23	0	0	21	1	1	22	1	0	21	2	0	23
	Ab	115	14	3	115	11	6	123	9	18	98	16	8	124
Abdominal pain	P	135	13	3	133	12	6	141	10	18	115	18	8	143
	Ab	3	1	0	3	0	1	4	0	0	4	0	0	4
Others	P	16	3	0	18	1	0	19	0	2	16	1	0	19
	Ab	122	11	3	118	11	7	126	10	16	103	17	8	128

P: Present, Ab: Absent, *P<0.05; **P<0.01; ***P<0.001

Table 3: Distribution of iufd between clinical finding and placental pathology.

		Syncytial Knot		Cytotrophoblastic layer		Fibrinoid Necrosis		Poor Vascularity Villi		Immaturity Villi		Infarcts	
		P	Ab	P	Ab	P	Ab	P	Ab	P	Ab	P	Ab
Fever	P	3	18	4	17	8	13	8	13	2	19	2	19
	Ab	18	116	45	89	52	82	17	117**	21	113	11	123
Leaking	P	0	11	4	7	5	6	2	9	2	9	1	10
	Ab	21	123	45	99	55	89	23	121	21	123	12	132
BPV	P	3	27	10	20	11	19	5	25	4	26	1	29
	Ab	18	107	39	86	49	76	20	105	19	106	12	113
Convulsion	P	11	12	4	19	21	2	1	22	2	21	4	19
	Ab	10	122***	45	87	39	93***	24	108	21	111	9	123
Abdominal pain	P	21	130	45	106	58	93	25	126	23	128	13	138
	Ab	0	4	4	0**	2	2	0	4	0	4	0	4
Others	P	0	19	2	17	4	15	2	17	4	15	1	18
	Ab	21	115	47	89*	56	80	23	113	19	117	12	124

P: Present, Ab: Absent, *P<0.05; **P<0.01; ***P<0.001

If abdominal pain absent (n=4) then in 4 subjects Cytotrophoblast layer present (p<0.01) (Table 3).In subject with polyhydramnios (n=3) poor vascularity villi present in 2 subjects (P<0.05) and similarly infract absent in 3 subjects (P<0.01). If placenta in lower segment (n=10) then fibrinoid necrosis absent in all subject (P<0.01).

Placental maturity grade-III (n=18) fibrinoid necrosis present in 11 subjects (P<0.05) (Table 4).If hypertension present (n=39) then syncytial knot present in 8 subjects (P<0.001), of these subject 30 subjects present with fibrinoid necrosis (P<0.001).

If Hypertension absent (n=116) then only 6 subjects present with infract (P<0.01).If urine albumin 3+(n=6) then syncytial knot present in 3 subjects (P<0.01) and fibrinoid necrosis present in 6 subjects (P<0.01) (Table 5). If infarctions absent (n=149) in gross finding then fibrinoid necrosis present in 55 subjects (P<0.01) and microscopic infarct present in 8 subjects (P<0.01). If calcification absent (n=137) then 48 subjects present with cytotrophoblastic layer (P<0.01), Whereas infarct present in 8 subjects (P<0.01) (Table 6).

DISCUSSION

Determining the cause of foetal death aids the psychological adaptation to a significant loss, help to assuage the guilt that is part of grieving, makes counselling regarding recurrence more accurate, and many prompt therapy or intervention to prevent a similar out comes in the next pregnancy. The data of placental finding are documented by the macroscopic evaluation and microscopic survey of the placental tissue of each stillbirth case.

The study evaluates many different foetal, maternal, and placental parameters which have an effect on the outcome of gestations. In the present study majority of subjects (81.9%) were from rural area, 96.8% of subjects belonged to low socioeconomic status, 32.9% were illiterate. Srushti R. Kantha et al, in their study at Karnataka, India found that maximum number of subjects were found in age group of 21-30 years (67.5%), most subjects were unbooked (86%) and were from rural area and 83.5% subjects were from low socioeconomic status.[5] Our observation on demographic variables of the studied subjects shows that majority of them belonged to low socioeconomic class possibly due to the fact that the Medical College caters to a large rural and tribal population. In present study, majority of studied subjects (94.84%) were unbooked. Srushti et al, found 86% subjects of unbooked.[5] Most of the subjects in our study were uneducated and were unaware of the importance of receiving antenatal checkup.

Table 4: Distribution of IUFD between placental pathology and USG finding.

		Syncytial Knot		Cytotropho-blastic layer		Fibrinoid Necrosis		Poor Vascularity Villi		Immaturity Villi		Infarcts	
		P	Ab	P	Ab	P	Ab	P	Ab	P	Ab	P	Ab
Liquor	Adequate	19	119	45	93	55	83	22	116	20	118	8	130
	Oligo hydra minos	2	12	3	11	5	9	1	13	3	11	5	9
	Poly hydra-minos	0	3	1	2	0	3	2	1*	0	3	0	3**
Presentation	Cephalic	18	118	41	95	55	81	21	115	19	117	10	126
	Breech	1	11	7	5	3	9	3	9	3	9	2	10
	Other (Transverse, Oblique)	2	5	1	6	2	5	1	6	1	6	1	6
Placental Localization	Upper segment	21	124	45	100	60	85	23	122	21	124	13	132
	Lower segment	0	10	4	6	0	10**	2	8	2	8	0	10
Placental Maturity	Grad I	1	17	11	7	3	15	3	15	2	16	0	18
	Grade II	16	103	37	82	46	73	17	102	16	103	9	110
	Grade III	4	14	1	17	11	7*	5	13	5	13	4	14
Congenital Anomaly	Present	0	8	3	5	1	7	0	8	1	7	0	8
	Absent	21	126	46	101	59	88	25	122	22	125	13	134

P:Present,Ab:Absent,*P<0.05;**P<0.01

Table 5: Distribution of IUFD between placental pathology and hypertension, pallor, oedema, urine albumin.

		Syncytial Knot		Cytotrophoblastic layer		Fibrinoid Necrosis		Poor Vascularity Villi		Immaturity Villi		Infarcts	
		P	Ab	P	Ab	P	Ab	P	Ab	P	Ab	P	Ab
Hypertension	0	8	108	40	76	30	86	20	96	21	95	6	110**
	1	13	26***	9	30	30	9***	5	34	2	37*	7	32
Pallor	1	8	104	33	79	37	75	19	93	12	100	6	106
	2	11	19	11	19	16	14	2	28	8	22	6	24
	3	2	9***	5	6	6	5	3	8	3	8*	1	10*
Oedema	1	5	17	9	13	16	6	3	19	2	20	4	18
	2	4	6	2	8	9	1	2	8	0	10	3	7
	3	2	1	1	2	2	1	2	1	0	3	1	2
Urine albumin	1	1	13	9	5	8	6	1	13	1	13	2	12
	2	10	7	3	14	16	1	0	17	1	16	1	16
	3	3	3**	1	5**	6	0**	1	5	0	6	4	2**

P: Present, Ab: Absent, *P<0.05; **P<0.01; ***P<0.001

Table 6: Distribution of IUFD subjects according to gross and microscopic placental pathology.

		Syncytial Knot		Cytotrophoblasticlayer		Fibrinoid Necrosis		Poor Vascularity Villi		Immaturity Villi		Infarcts	
		P	Ab	P	Ab	P	Ab	P	Ab	P	Ab	P	Ab
CordInsertion	Ab	21	134	49	106	60	95	25	130	23	132	13	142
Sing UA	Ab	21	134	49	106	60	95	25	130	23	132	13	142
Infarction	P	2	4	2	4	5	1	0	6	1	5	5	1
	Ab	19	130	47	102	55	94**	25	124	22	127	8	141**
Calcification	P	6	12	1	17	9	9	5	13	2	16	5	13
	Ab	15	122**	48	89**	51	86	20	117	21	116	8	129**
Retro Pal Clot	P	0	15	5	10	7	8	1	14	2	13	0	15
	Ab	21	119	44	96	53	87	24	116	21	119	13	127

P: Present, Ab: Absent, *P<0.05; **P<0.01; ***P<0.001;

The overall mortality rate in unbooked case were 3 to 4 time higher that booked subjects. In this study majority of subjects (49.68%) is primigravida and 26.45% patient is primipara. Srushti et al, found in their study that (52.5%) subjects were primigravida.[5] B. Mishra et al, found that intrauterine death rate highest in primigravida.[6]

Our observation was that majority of primigravida were unaware of importance of regular antenatal visit. In the present study majority 92.26% were delivered vaginally and 7.74% delivered by Caesarean section for indications such as obstructed labour, transverse lie, previous section with cephalopelvic disproportion. Ifnan F et al, at multan found in their study that mode of delivery was vaginal in 87.4% and caesarean section in 12.6% subjects.[7]

In the present study found that 52.26% subjects delivered preterm. Srushti R, et al, found in their study that maximum subjects 76.5% of preterm birth. In the present study found that 80% subjects were low birth weight and small for gestational age.[9] Pillared RA et al, found that increase risk of intrauterine foetal death in small for gestational age foetus compared to none small gestational age foetus.[9] In the present study subjects from rural area, low socioeconomic status and in preterm labour had increased frequency of clinical symptom such as fever, leaking, bleeding per vaginum, convulsion with other complications like hand prolapse, cord prolapse, obstructed labour, rupture uterus. Significantly (P<0.01) less number of patient with preterm delivery had no bleeding per vaginal. Similarly convulsion was significantly less in these subjects, One explaination could be that besides iatrogenic preterm delivery, pathology which leads to convulsions eg eclampsia in itself can cause preterm delivery. William H et al found that foetal anomaly, toxaemia of pregnancy and maternal acute infection have been recognized cause of intrauterine foetal death.[10] In our observation clinical symptoms related to intrauterine foetal death was more in low socioeconomic group because they not only had improper antenatal check up but also presented late in labour.

Al mulhim AA et al, in their study on perinatal outcome in subjects of preeclampsia found that perinatal outcome with preeclampsia shows stillbirth 2.34% and early neonatal death 1.02% which comprise an overall motility of 33.6/1000.[8] In our study 17.4% subjects of intrauterine foetal death because of convulsion. In the present study

138 subjects had adequate liquor, 14 oligohydramnios, 3 subject polyhydramnios. Schneider H. et al, found that in oligohydramnios foetal complication are asphyxia, intrauterine false posture and under developed lung which lead to intrauterine foetal death.[9]

In the present study of the 155 subjects 8 (5.16%) subjects presented with congenital anomaly. Zanconoto G et al, found that in Verona in evaluation of 59 subjects of intrauterine foetal death 13.5% subjects of intrauterine foetal death because of congenital foetal anomaly.[10]

In the present study when bleeding per vaginum was a presenting complaint (n = 26) 8 subjects had placenta in lower segment. However, when bleeding per vagina was absent only 2 subjects had placenta in lower segment (P<0.001). In the present study, when subjects presented with fever (n = 21), then 8 subjects presented with poor vascularity of villi and when fever was absent (n = 134), 17 subjects presented with poor vascularity of villi (p<0.01). This may imply that in subjects with fever, the fever rather than the placental pathology could have lead to the untimely foetal demise. This could have been preventable cause of IUD.

When convulsion was present (n = 23), 11 subjects presented with syncytial knot, when absent (n = 132), then 10 subjects presented with syncytial knot (p<0.001). Similarly when convulsion were present, 21 subjects had fibrinoid necrosis of placenta and when absent, then 39 subjects had fibrinoid necrosis present (p<0.001). This establishes a positive correlation between fibrinoid necrosis, placental knots and convulsions.

When abdominal pain is absent (n = 4), all 4 subjects had cytotrophoblast layer (p<0.01). Fox H noted in their study that in preeclampsia decreased perfusion of placental circulation leads to prominent focal syncytial necrosis. Similarly when calcification absent (n = 137) then 48 subjects presented with cytotrophoblastic layer (p<0.01) whereas infarct present in 8 subjects (p<0.01).[11]

In the present study in subject with polyhydramnios (n = 3) poor vascularity of villi is present in 2 subjects (p<0.05) but infract was absent in all 3 subjects (p<0.01). When placenta is in lower segment (n = 10) then fibrinoid necrosis absent in all subjects (p<0.01) but if placental maturity grade-III (n = 18) fibrinoid necrosis is present in 11 subjects (p<0.05).

When hypertension was present (n = 39) then syncytial knot present in 8 subjects (p<0.001) and 30 subjects presented with fibrinoid necrosis (p<0.001). If hypertension absent (n = 116) then only 6 subjects present with infract (p<0.01). This means that infarction is a significant association of hypertension.

In the present study, If urine albumin is 3+ (n = 6) then syncytial knot present in 3 subjects (p<0.01) and fibrinoid necrosis present in 6 subjects (p<0.01), as was there association with convulsion. Aep heazell. SJ moll, CJP jones et al, in their study found that increased syncytial knot in subjects of preeclampsia and intrauterine growth retardation is because of hypoxia and oxidative stress present in these conditions.[12] Udaina et al, found a direct correlation between the degree of pregnancy induced hypertension and amount of infarction of placenta.[13] In the present study if infarction absent (n = 149) in gross finding then fibrinoid necrosis present in 55 subjects (p<0.01) and microscopic infract present in 8 subjects (p<0.01).

Sodhi S. et al, in their study on placental pathology in preeclampsia- eclampsia syndrome found that gross abnormalities noted in subjects of preeclampsia were placental infarct, retroplacental clot and calcification. The finding were correlated well with severity of maternal disease.[14] Moscuzza F et al, in their study found that placental lesions are associated with impaired pregnancy and neonatal outcome. These observations correlate with our observations as well admissions.[15]

CONCLUSION

Present study is a step towards understanding and extrapolating the already known causes of intra uterine foetal death in the perspective of Jabalpur and its adjoining districts. Since a number of placental pathology adversely affect the perinatal outcome, an earnest effort can be made to diagnose them by prenatal ultrasound examination. Though it is less sensitive in detecting infections, it is highly specific in detecting retro-placental clot and placenta in lower segment. Since Jabalpur is endemic for many febrile illnesses, an early diagnosis and treatment can avoid many IUFDs. Similarly, vigilant follow up and careful management of subjects with pre-eclampsia and postmaturity can avoid many untoward accidents.

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