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Happy new year 2023 to all medical fraternity of Indian & abroad.
As a chief editor of JIAOG, I am glad to inform you that Volume – 4 issue 2 of JIAOG is going to be released on 28th January 2023.
Since our first publication of JIAOG, we are highlighting various scientific evidence-based study for reducing MMR as well as family planning program.
In this issue we are mainly focusing on global decline of male infertility and a couple of scientific papers related to improved perinatal outcome in complicated pregnancies.
I am very much grateful to all contributors and our team for their active support and valuable suggestions to make this journal one of the best medical journals.
Lastly, we are trying to get index and striving to make this a PubMed journal in future.

With regards,

Dilip Kumar Dutta
Chief Editor
Journal of Indian Academy of Obstetrics and Gynaecology

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Special Issue from Chief Editor

GLOBAL DECLINE IN MALE INFERTILITY: THE ROLE OF ENVIRONMENT AND LIFESTYLE

Dilip Kumar Dutta

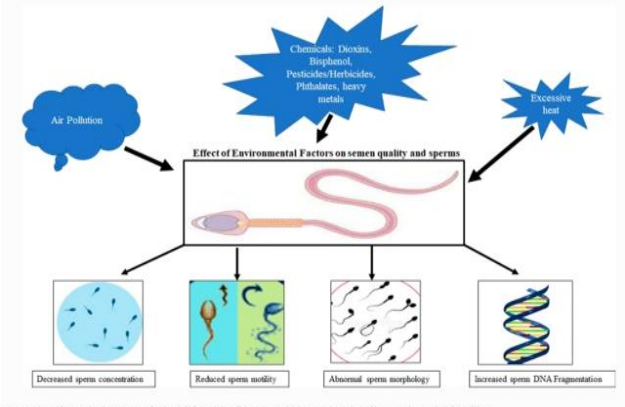
Recently it was observed that there is an increased tendency to decline life style of male partners globally, which is not only leading to socio-economic crisis but also to financial burden in the family too.

A. ENVIRONMENT AND MALE INFERTILITY

Word-wide rising trend in infertility – observed in the past few years, with male infertility arising as a major problem.
WHO, pubmed database, peer reviewed journals till June 2021 – focused on air pollution, chemicals, heat exposure and heavy metals - may cause male infertility.
Air pollution from motor vehicle exhaust, factories, oil refineries, ozone, nitrogen exude, sulfur dioxide, radiations, x-ray exposure →caused sperm dna fragmentation, morphological changes, and reduced sperm motility.
Harmful chemicals like pesticides, phthalates, heavy metals → maternal occupational exposure leads to low semen volume and total sperm count.
Dioxins → lipophilic chemicals, (TCDD), pops-> endocrine disruptions -> effects by binding to aryl hydrocarbon receptor (AHR)/ aryl hydrocarbon receptor nuclear translator (ARNT)-> damage testicular cell.
Bisphenol-> considered hazardous to human health -> leads to endocrine disruptions.
Heavy metals -> lead, cadmium, arsenic, mercury, barium -> reduce sperm viability and normal sperm morphology, dna damage etc.
Heat exposure -> normally scrotal temperature is 2-4˚C lower than the core body temperature. Any factors that cause rise of temperature (1-1.5˚c.) can result in oligospermia, teratozoospermia etc.

Hence environment increased sperm dna fragmentation which may cause male infertility and the same can be prevented and modified.

Fig. 1



Impact of environmental and lifestyle factors on semen quality and male fertility

B. **LIFESTYLE AND MALE INFERTILITY**

Smoking, alcohol, illicit drugs, obesity, psychological stress, diet, and caffeine intake are risk factors.
Testicular heat stress, intense cycling training, lack of sleep, use of mobile phone may also damage sperm.
Lavine group – studied 42935 men of 40 years, reported significant decline of 50 – 60 % in sperm count in North America, Europe, Australia and New Zealand.
Smoking → 7000 chemicals, nicotine carbon monoxide, cadmium and lead -> leucocytospermia – (ROS) reactive oxygen species -> impairing sperm function, quality, dna damage, aneuploidies, sperm apoptosis.
Alcohol →_HPG axis -> GNRH, FSH, LH, testosterone, as well as impair function leyden and sertoli cells -> semen morphology and motility.
Drugs ->__marijuana, cocaine, narcotics -> impaired HPG axis, testicular architecture and serum function.
Obesity ->_1.dna fragmentation 2. Abnormal morphology 3. Low mitochondrial membrane potential (MMP)
Psychological stress, advanced maternal age, Mediterranean diet - > enriched with omega -3 fatty acids, antioxidants, vitamins, low saturated and trans fatty acids is inversely related with low semen quality.
Western style diet -> high in red and processed meat, refined grains and high-energy drinks. Prudent diet -> while meat, fruit, vegetables and whole grains are good for infertility
Vegetable fruits, fish & poultry, cereals and low–fat dairy products improved sperm quality.

Further scope of research

Excessive eating of broiler chicken / egg with or without alcohol, smoking → estrogen level ↑ → imbalance between testosterone & estrogen → erectile dysfunction → reduced sex drive & sperm concentration.

Conclusion

Environmental & lifestyles factors cause decreased - sperm concentration and viability of normal morphological forms. Increased – sperm dna fragmentation index mitochondrial dysfunction. Hence early counseling and clinical intervention was found to be mandatory to prevent decline in male infertility.

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Original Article

COMPARISON OF EFFICACY OF DINOPROSTONE AND MISOPROSTOL IN INDUCTION OF LABOUR

Mobarok Ali[1], Pranoy Nath[2]✉, Manideepa Roy[3]

ABSTRACT

Introduction: Induction of labour denotes the artificial initiation of regular uterine contractions before spontaneous onset of labour with progressive dilatation and effacement of cervical and subsequent vaginal delivery of the baby. Labour induction is usually indicated when benefits of delivery to the mother or fetus outweigh the potential risks of continuing the pregnancy. Labour usually starts spontaneously in most of pregnant women at or near-term pregnancy and result in vaginal deliveries.

Aims and objectives: Aim of the study is to evaluate efficacy of misoprostol in comparison with dinoprostone for labour induction in term pregnant women.

Materials and methods: The study was carried out over 100 pregnant women admitted to the labour ward beyond 37 weeks of gestation, requiring induction of labour for various medical and obstetrical indications, in the department of Obstetrics and Gynaecology, Silchar medical college & hospital from 1st June 2021 to 1st June 2022. The participants were divided into two Groups-I and Group-II. Group-I, choosing the patients randomly. In Group-I, 50 patients received 25 µg Misoprostol per vaginally and in Group-II, 50 patients received Dinoprostone gel 0.5 mg endocervically.

Results: In Group-I, 25 cases (50%), Bishop score were ≥10/13 at 8 hrs. after induction of labour and in Group-II, 23 cases (46%), Bishop score were ≥10/13, (P= 0.3702). In Group-I, 43 cases had taken <6 hrs. interval for onset of labour and in Group-II, 24 cases had taken <6 hours interval to onset of labour. (P<0.0001).

Induction to delivery interval less in misoprostol group (mean ± S.D) 14.36±4.39 hours than dinoprostone group 16.68±4.43 hrs. (P=0.085). There was no significant difference in neonatal complication and NICU admission among both the study groups.

Conclusion: From the present study, it can be concluded that the tab. Misoprostol 25 µg pervaginal is more effective in comparison to Dinoprostone 0.5 mg endocervical for induction of labour with respect to the induction to onset of labour interval and induction to delivery interval.

Keywords: Misoprostol, Dinoprostone, Induction of labour.

INTRODUCTION

Induction of labour denotes the artificial initiation of regular uterine contractions before spontaneous onset of labour with progressive dilatation and effacement of cervical and subsequent vaginal delivery of the baby.[1] Labour usually starts spontaneously in most of pregnant women at or near-term pregnancy and results in vaginal deliveries. Labour induction is usually indicated when the benefits of delivery to the mother or fetus outweigh the potential risks of continuing the pregnancy[2]. Most common indications of labour are postdated pregnancy, pregnancy induced hypertension, draining pervaginum, intrauterine growth restriction, intrauterine fetal death. Labour induction may successfully end up in normal course of labour and vaginal delivery or it may end up in surgical intervention like caesarean section.[3] Induction of labour with prostaglandins offers the advantage of promoting cervical ripening while stimulating myometrial contractility.[4] Prostaglandins alter the extracellular ground substance of the cervix and also increase the activity of collagenase in the cervix which help to ripen the cervix. They also allow for increases in intracellular calcium levels, causing contraction of myometrial muscle.[5,6] Dinoprostone is a synthetic preparation of naturally occurring prostaglandin E_2. Dinoprostone gel is available in 2.5 ml syringe for an endocervical application of 0.5 mg of Dinoprostone.[7] Misoprostol, a synthetic prostaglandin E_1, initially was used for prophylaxis treatment in NSAID induced peptic ulcers. One of its "side effects" was the induction of uterine contraction during pregnancy and early pregnancy abortion. Thereafter, misoprostol was used for termination of first trimester pregnancy. It is stable at room temperature, low cost, and ease of oral sublingual and pervaginum administration. It is available as tablets of 25, 50, 100, 200 µg.

So, the present study is conducted with an aim of comparison of efficacy between the two drugs, i.e. vaginal misoprostol and endocervical dinoprostone administration in pregnant women with singleton pregnancy with vertex presentation at term pregnancy.

MATERIALS AND METHODS

This was a prospective analytic study carried out in the department of Obstetics and Gynaecology of Silchar Medical College & Hospital, Silchar, Assam, in the period of 1st June 2021 to 31st May 2022, over 100 pregnant women admitted to the labour ward beyond 37 weeks of gestation and requiring induction of labour for various medical and obstetrical indications. Induction of labour done with 25µg of misoprostol intravaginally and 0.5mg of dinoprostone gel endocervically with definite indication for vaginal delivery in primigravida at 37-42 weeks of gestation with vertex presentation with singleton pregnancy.

INCLUSION CRITERIA

Primigravida with 37 or more weeks of gestation with Singleton gestation with Cephalic presentation, having indication of vaginal delivery will be included in the study.

EXCLUSION CRITERIA

Previous uterine surgery, Multigravida, Multiple pregnancy, Placenta previa, Malpresentations, Abnormal fetal heart rate, Chorioamnionitis, Cephalopelvic disproportion,

STUDY APPROVAL

The study protocol was approved by the Ethical Committee of Srimanta Sankaradeva University of Health & Sciences, Assam. Patient or their family member was informed and written consent was taken for the same.

METHOD OF COLLECTION OF DATA

All the participants were selected from the patients admitted in the medical labour room for induction of labour. The participants and their family members were fully informed about the study and written consent was taken from the participants or their family members in the study. Participants were selected on the base of the inclusion and exclusion criteria. Detailed history, clinical examination and investigation

of all the participants were done.
The participants were divided into two groups, Group I & Group II selecting the patients randomly for Group I & Group II. Each group was having 50 participants.
Group I: Participants were received Tab. Misoprostol 25µgm per vaginally in posterior fornix and whenever needed more doses Tab. Misoprostol 25 µgm were given at 4 hours interval for maximum 5 doses.
Misoprostol doses were repeated till: 1) maximum 5 doses of misoprostol or 2) onset of adequate uterine contraction
Group II: Pregnant women were instilled intracervical Dinoprostone gel 0.5 mg and whenever more doses needed were given at 6 hours interval for maximum 3 doses.
Instillation of Dinoprostone was repeated till: 1) maximum 3 doses of dinoprostone or 2) onset of adequate uterine contraction

Monitoring of mother: Monitoring of the maternal vitals, were done.
Monitoring of the fetus: Fetal heart rate auscultation were done at 30 minutes interval in 1st stage and at 15 minutes interval in 2nd stage of labour.
After birth APGAR score were recorded at 1 minute and 5 minutes.
Monitoring of labour: P/V Examination was done as per protocol of partographic monitoring and was plotted in the partograph paper.
Particulars of delivery and Baby were recorded.
Mother and baby were observed for postnatal complications if any.

OPERATIONAL DEFINITIONS

Onset of labour: Defined as at least 3 regular uterine contractions in 10 minutes, each lasting for at least 40 seconds.
Successful induction: Vaginal delivery within 24 hours were taken as successful induction.
Failed induction: Adequate uterine contraction was not established after 6 hours of 5th dose of misoprostol and after 6 hours of 3rd doses of dinoprostone.
Uterine hypersystole: Each uterine contraction lasting for more than 2 minutes.
Uterine tachysystole: more than 5 uterine contractions in a 10 minutes interval.
Uterine hyperstimulation: Both uterine hypersystole and tachysystole cumulatively is known as uterine hyperstimulation associated with fetal distress.
Patients who achieved labour were examined and according to the presence or absence of membrane and meconium staining of liquor, the needful intervention was taken according to the institutional protocol as per the clinical assessment of the patients at that time which would include amniotomy or caesarean section. Successful inductions were considered for comparison of efficacy, NICU admissions were considered for comparison of fetal outcome. In all patients, the cervical status was assessed by using modified Bishop Score to induction.

STATISTICAL ANALYSIS

Statistical analysis was done by using descriptive and inferential statistics using Z-test for single proportion. Suitable software was used for descriptive statistics and others statistical analysis. Level of significance were at 5%.

RESULTS

Most of the participants of both the comparison Groups were between the maternal ages of 20 and 30 years. In misoprostol Group, 45 cases were in this age group and in dinoprostone Group, 44 cases were in this age group. There was not much significant difference in Mean age in both Groups (23.34±3.263 years vs. 23.33±3.462years) (Table 1). In misoprostol Group, 41 cases were >40 weeks of gestational age and in dinoprostone group, 39 cases were >40 weeks of gestational age. (p=0.6171) (Table 2). In both Groups, the indication of induction of labour in most of cases were decided for postdated pregnancy. In misoprostol group, 31 cases (62 %) were postdated pregnancy and in dinoprostone group, 26 cases (52%) were postdated pregnancy. (P=0.7741) (Table 3).
There was no significant difference in pre-induction Bishop Score of the cervical assessment of participants of both misoprostol and dinoprostone group. In misoprostol group, 30 cases (60%) the pre-induction Bishop score were 3/13, in dinoprostone group, 32 cases (64%) the

pre-induction Bishop score were 3/13. (P=0.7181) (Table 4).

The participants of misoprostol group took less time interval for onset of labour than dinoprostone group. In misoprostol group, 43 cases took <6 hrs. interval for onset of labour and in dinoprostone group 24 cases took <6 hours interval to onset of labour (P<0.0001) (Table 5).

Bishop Score of participants at 8 hours after induction of labour were more in misoprostol group than dinoprostone group. In misoprostol group, 25 cases (50%), Bishop score were ≥10/13 at 8 hrs. after induction of labour and in dinoprostone group, 23 cases (46%) Bishop score were ≥10/13 (P= 0.3702) (Table 6).

The participants of misoprostol group took less time for induction to delivery interval than dinoprostone group. The misoprostol group took (mean ± S.D) 14.36±4.39 hours and dinoprostone group took 16.68±4.43 hrs. (P=0.085) (Table 7).

Normal vaginal delivery achieved in 39 cases (78%**) of** dinoprostone group, and **in 37** cases (74%) of misoprostol group. Emergency LSCS intervention required for 11 cases (22%) of Dinoprostone and 13 cases (26%) of misoprostol (P=0.6396) (Table 8).

There was no significant difference in side effect of both the drugs. PPH occurred in 2 cases in misoprostol group and 3 cases in dinoprostone group. (p=0.5718) (Table 9).

There was no significant difference in APGAR score at 1minute and 5 minutes of the babies after birth in both the study groups. ($P_{1=}$ 0.8150 and $P_{5=}$ 0.9746) (Table 10).

There was no significant difference in neonatal complication and NICU admission among both the study groups. In misoprostol group, 7 babies admitted in NICU for meconium-stained liquor and in dinoprostone group, 4 babies were admitted in NICU for meconium-stained liquor. (P=0.6516) (Table 11).

Table 1: Comparison of Maternal Age.

Age (in years)	Misoprostol (N=50)		Dinoprostone (N=50)	
	No. of cases	Percentage	No. of cases	Percentage
14-19	4	8	5	10
20-25	35	70	33	66
26-30	10	20	11	22
31-36	1	2	1	2
Mean age± SD (yrs)	23.34±3.263		23.33=3.462	

Table 2: Comparison of Gestational Age.

Gestational age (in weeks)	Misoprostol		Dinoprostone		P value
	No. of cases	Percentage	No. of cases	Percentage	
37-40	9	18	11	22	0.6171
>40	41	82	39	78	
Total	50	100	50	100	

Table 3: Comparison of Indications for Induction of Labour.

Indications for Induction	Misoprostol (N=50)		Dinoprostone (N=50)		P value
	No. of cases	Percentage	No. of cases	Percentage	
Postdated	31	62	26	52	0.7741
PIH	8	16	11	22	
Draining PV	9	18	11	22	
APE	2	4	2	4	
Total	50	100	50	100	

Table 4: Comparison of Pre-induction Bishop Score.

Initial Bishop Score	Misoprostol group		Dinoprostone gel group		P value
	No. of cases	%	No. of cases	%	
1	1	2	2	4	0.7181
2	8	16	10	20	
3	30	60	32	64	
4	9	18	5	10	
5	2	4	1	2	
Mean ± SD	3.06±0.77		2.86±0.73		

Table 5: Comparison of Induction to Onset of Labour Interval.

Time in IOL (In hrs.)	Group I No. (%)	Group II No. (%)	Total No. (%)	P value
≤6	43 (86%)	24 (48%)	67 (67%)	<0.001
>6	7 (14%)	26 (52%)	33 (33%)	
Total	50(100%)	50 (100%)	100 (100%)	

Table 6: Comparison of Bishop Score after 8 hours.

After 8hrs Bishop Score	Misoprostol group		Dinoprostone gel group		P value
	No. of cases	%	No. of cases	%	
<4	0	0	0	0	0.3702
4-5	4	8	10	20	
6-7	9	18	8	16	
8-9	12	24	9	18	
≥10	25	50	23	46	
Mean ± SD	8.82±1.97		8.3±237		

Table 7: Comparison of induction to delivery interval.

Interval (hours)	Misoprostol (N=50)		Dinoprostone (N=50)		P value
	No. of cases	Percentage	No. of cases	Percentage	
7-10	10	20	3	6	0.0851
11-14	23	46	18	36	
15-18	9	18	19	38	
19-22	5	10	6	12	
23-26	3	6	4	8	
Mean ± S.D	14.36±4.39		16.68±4.43		

Table 8: Comparison of Mode of delivery.

Mode of delivery	Misoprostol (N=50)		Dinoprostone (N=50)		P value
	No. of cases	Percentage	No. of cases	Percentage	
Vaginal	37	74	39	78	0.6396
Caesarean section	13	26	11	22	
Total	50	100	50	100	

Table 9: Comparison according to Maternal Complication

Side effects	Misoprostol		Dinoprostone		P value
	No. of cases	%	No. of cases	%	
Vomiting	0	0	1	2	0.5718
Diarrhoea	3	6	1	2	
Shivering	1	2	0	0	
Pyrexia	1	2	0	0	
Hypersti mulation	1	2	0	0	
Tachysyst ole	1	2	0	0	
PPH	2	4	3	6	

Table 10: Comparison of APGAR score in study groups

Neonatal outcome		Misoprostol		Dinoprostone		P value
		No. of cases	%	No. of cases	%	
APGAR at 1 minute	≤7	17	34	19	38	0.8150
	>7	33	66	31	62	
APGAR at 5 minutes	≤8	23	46	22	44	0.9746
	>8	27	52	28	56	

Table 11: Comparison of indication for NICU Admission.

Indication for NICU Admission	Misoprostol		Dinoprostone		P value
	No. of cases	%	No. of cases	%	
Birth asphyxia	1	2%	1	2%	0.6516
LBW	3	6%	3	6%	
Meconium stained	7	14%	4	8%	
RDS	0	0	1	2%	
Total	11	22%	9	18%	

DISCUSSION

The present study was an analysis of the maternal and fetal outcome in 100 cases of induction of labour with 25 µg vaginal misoprostol and 0.5 mg endocervical dinosprostone gel in primi gravida at 37-42 weeks of gestation with singleton gestation with vertex presentation with indication of labour with postdated pregnancy, draining PV, pregnancy induced hypertension and antepartum eclampsia in the Silchar Medical College & Hospital, Silchar, Assam.

The induction of labour with the Prostaglandins dramatically decreased major difficulties of labour induction to clinical practice, especially their local use for cervical ripening and labour induction without majors' complication of mother and baby.

There was no statistically significant difference in baseline characteristics of age, height and weight in both groups. All the women were primigavida. Most of them were with the maternal age group 20 to 30 years in both the comparison groups. There was no significant difference in both the groups in respect of maternal age. Most of the women were in the period of gestation of more than 40 weeks in the both study groups. There was no significant difference regarding period of gestation of pregnancy in both the groups. Both groups were also in sync with the study of Olav Lapaire et al., (2007)[8] in terms of demographic and obstetric data such as maternal age, gravidity, parity and period of gestation. In the present study, postdatism was indication for induction of labour, 62% and 52% in group I and group II respectively followed by PIH in 16% cases in group I and 22% cases in group II. There was no significant difference in both groups regarding indication of induction of labour (P=0.7741). Greagsons et al.,[9] in their study also showed that 95% patients in misoprostol group and 94% in dinoprostone group were induced for postdatism. Similarly, C. N. Sheela et al.,[10] demonstrated that postdatism (36% and 32% respectively) and PIH (22% and 26% respectively) were most common indications in both groups. Dr. Ankita Mishra, et al.,[11] in their study reported that most common indication was post-dated pregnancies followed by pre-eclampsia, oligohydramnios, Rh-Negative pregnancy, IUGR, GDM in both the groups.

The pre-induction bishop score in 30 cases in the misoprostol group (60%) were 3/13, in dinoprostone group, 32 cases (64%) were 3/13. The mean pre-induction bishop score was 3.06±0.77 in the misoprostol group and 2.86±0.73 in the dinoprostone group. There was no significant difference in pre-induction bishop score of the cervical assessment of participants in both groups (P=0.7181). In the study of Olav Lapaire et al., (2007)[12] the bishop scores collected prior to induction of labour were not statistically different in both groups (P=0.33). Dr. Shefali Bansal (2016)[13] study had also the initial bishop Score in the range of 1 to 4 with mean induction bishop scores of 3.25 ± 0.44 and 3.14±0.68 for dioprostone gel and misoprostol group respectively.

The Bishop Score at 8 hours after induction of labour were more in misoprostol group than dinoprostone group. In misoprostol group, in 25 cases (50%), bishop score is ≥10/13 at 8 hrs. after induction of labour and in dinoprostone group, in 23 cases (46%), bishop score is ≥10/13, (P= 0.3702). B. H. Radhika, et al, (2013) reported that the mean Bishop score at the end of 8 hours and 16 hours of cervical ripening was almost similar in both the groups.

In this study, most of the participants took less time interval to onset of labour in misoprostol group than dinoprostone group. In misoprostol group, 43 cases take <6 hours interval to onset of labour and in dinoprostone group, 24 cases take <6 hours interval to onset of labour. There was statistically significant difference regarding the

onset of labour after induction of labour (P<0.0001). In the study of Swaran Gupta (2015)[14] also reported that in misoprostol group, majority of patients (90%) had gone into labour within six hours, whereas in dinoprostone 52% had gone into labour within 6 hours, the difference of time taken in the two groups was statistically significant (p<0.001)

The mean time had taken for induction to delivery interval was less in the misoprostol group (14.36±4.39 hrs) than in the dinoprostone group (16.68±4.43 hours) (P=0.0851). Gemund et al., (2004)[15] have reported longer induction delivery intervals in the misoprostol group than with dinoprostone (25 vs. 19 h, P = 0.008). Evangelos G et al., (2004)[16] in their study conducted on 163 eligible clients reported that the induction delivery interval was significantly lower in the misoprostol group than in the dinoprostone group (11.9 hrs vs. 15.5 hrs, p < 0.001). In the study of Murthy Bhaskar Krishnamurthy (2006)[17] induction delivery interval was shorter in the misoprostol group. Smiti Nanda et al., (2007) reported the mean induction delivery interval regardless of the route was shorter in the misoprostol group 13.30±8.74 (3–40.15) hours as compared with dinoprostone group, 18.53±11.33 (2–48.07) hours (P =0.011). Dr. Afia Ansar et al., (2014)[18] have reported the induction to delivery interval was 13.03+3.52 hours in misoprostol group while it was 14.12+3.31 hours in dinoprostone group. Swaran Gupta (2015)[14] reported that the mean induction delivery interval was 11.23 hours in misoprostol group and 18.5 hours in dinoprostone group, the difference of induction delivery interval was statistically significant (p=0.02). Ramya D, Jaju PB (2017)[19] reported that the mean induction delivery interval in dinoprostone is 10.29±7.19 hours. The mean induction delivery interval in misoprostol was 7.64±5.75 hours, (P value=0.014). Dr. Ankita Mishra, et al., (2020)[11] in their study found the mean induction delivery interval, mean±S.D was 11.8±2.03 hours in the misoprostol group and 15.54±2.63 hours in the dinoprostone group.

In our study 39 cases (78%) of the dinoprostone group proceeded for normal delivery and 11 cases (22%) required emergency LSCS intervention. In misoprostol group, 37 cases (74%) proceeded for normal delivery and 13 cases (26%) required emergency LSCS intervention. (P=0.6396). Olav Lapaire et al., (2007)[12] in their study found as a total of 78% (40/51) in the misoprostol group delivered by vaginal delivery as compared to 64% (30/47) in the dinoprostone group (P=0.123). Dr. Afia Ansar et al., (2014)[18] reported that out of 63 patients in the misoprostol group, 43 (67.1%) women had Normal vaginal delivery (NVD) while 26 (63.4%) patients out of 41 in dinoprostone group had NVD. Ankita Mishra et al., (2020)[11] found 42(84%) participants in misoprostol group and 40(80%) participants in dinoprostone group normal vaginal delivery occurred and 16% participants in misoprostol and 20 % participants in dinoprostone group underwent caesarean section.

In our study, there was no significant difference found in the side effect of both the drugs like maternal nausea, vomiting, pyrexia and hyprstimulation. In 2 cases of misoprostol group, PPH occurred and in 3 cases of Dinoprostone, PPH occurred (P=0.5718). Smiti Nanda et al., (2007)[8] found that the maternal side-effects like nausea, vomiting, diarrhoea, shivering and pyrexia were infrequent and the incidence was almost similar in both groups (P ¼ 0.75, RR ¼ 0.81, 95% CI 0.40–1.64). Swaran Gupta (2015)[14] reported that rate of tachysystole (>6 contraction/10 minutes) was higher in misoprostol group (18%) as compared to dinoprostone group (6%).

In our study, there was no significant difference found regarding at 1- and 5-minute APGAR score of the babies after birth in both study groups. ($P_{1=}$ 0.8150 and $P_{5=}$ 0.8150). Smiti Nanda et al., (2007)[8] also showed that there was no significant difference in the APGAR score at 1 and 5 min in the two groups. Olav Lapaire et al., (2007)[12] reported that APGAR scores (<7) were lower at five minutes observed in the dinoprostone group versus the misoprostol group (P<0.05). In misoprostol group, there were 7 babies admitted in NICU for meconium-stained liquor whereas in dinoprostone group 4 babies were admitted in NICU for meconium-stained liquor. In our study, there was no significant difference in both groups in neonatal complication (P=0.6516). The study by Olav Lapaire et al., (2007)[12] found that in dinoprostone group (n=12) more neonates were admitted to

the NICU, compared to the misoprostol group (n=6, P=0.068).

CONCLUSION

From the present study it can be concluded that the tab. Misoprostol 25 µg pervaginal is more effective in comparison to Dinoprostone 0.5 mg endocervical for induction of labour with respect to the induction to onset of labour interval and induction to delivery interval. With respect to maternal outcome and the neonatal outcome no significant statistical difference was noted in either of the groups. The timely monitoring of fetal heart rate and labour progress reduced complication of induction of labour with prostaglandin. However, the above conclusion was made with the study of small group of participants, the study in a larger group of participants may give a better evaluated conclusion.

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REFERENCES

1. *Memon A, Sikandar R. Misoprostol for induction of labour: the Hyderabad experience. J Liaqat Uni Med Health Sci 2007; 6: 56-9. [Journal]*
2. *Beischer, N.A. (1997). Maternal well-being during pregnancy. In: Beischer NA, Mackay EV, Colditz PB, eds. Obstetrics and the Newborn. An Illustrated Textbook. 3rd ed. Philadelphia: Saunders, 449.*
3. *Vahratian A, Zhang J, Troendle JF, Sciscione AC,Hoffman MK. Labor progression and risk of cesarean delivery in electively induced nulliparas. Obstet Gynecol. 2005;105(4):698-704.*
4. *Ramos LS, Peterson S, Delke I, et al. Labor induction with prostaglandin E1 misoprostol compared with Dinoprostone vaginal insert. A randomized trial. Obstet & Gynecol. 1998;91:401-5.*
5. *Witter, F.R. (2000). Prostaglandin E2 preparations for pre- induction cervical ripening. Clin Obstet Gynecol, 43; 469-74.*
6. *Arias, F. (2000). Pharmacology of oxytocin and prostaglandins. Clin Obstet Gynecol, 43; 455-68.*
7. *F.Gary Cunningham, Kenneth J.Leveno, Steven L.Bloom, John C., Rouse, Spong, Williams Obstetrics,2010;23:502*
8. *Nanda S, Singhal SR, Papneja A. Induction of labour with intravaginal misoprostol and prostaglandin E2 gel: a comparative study. Tropical doctor. 2007 Jan 1;37(1):21-4.*
9. *Gregson, S., Waterstone, M., Norman, I., & Murrells, T. (2005). A randomised controlled trial comparing low dose vaginal misoprostol and dinoprostone vaginal gel for inducing labour at term. BJOG: An International Journal of Obstetrics & Gynaecology, 112(4), 438-444.*
10. *Sheela, C. N., Mhaskar, A., & George, S. (2007). Comparison of vaginal misoprostol and oral misoprostol with intracervical dinoprostone gel for labor induction at term. J Obstet Gynaecol India, 57, 327-30.*
11. *Mishra A, Rohatgi R. To Compare the Safety and Efficacy of 25mcg Intravaginal Misoprostol And 0.5 mg Intracervical Dinoprostone Gel For Induction of Labour*
12. *Lapaire O, Zanetti-Dällenbach R, Weber P, Hösli I, Holzgreve W, Surbek D. Labor induction in preeclampsia: is misoprostol more effective than dinoprostone.*
13. *Bansal S. Comparison of Intravaginal Misoprostol Tablet (Prostaglandin E1) and Intracervical Dinoprostone (Prostaglandin E2) Gel in Induction of Labour.*
14. *Gupta S, Hak J, Kumar D. Comparative Study of Efficacy of Misoprostol Vs Dinoprostone Gel For Induction of Labour. JK Science. 2015 Jul 1;17(3).*
15. *Van Gemund N, Scherjon S, LeCessie S, Van Leeuwen JH, Van Roosmalen J, Kanhai HH. A randomized trial comprising low dose vaginal misoprostol and dinoprostone for labor induction. Br J Obstet Gynecol 2004;111:42–9*

16. Evangelos G Papanikolaou, Nikos Plachouras, Aikaterini Drougia, Styliani Andronikou, Christina Vlachou, theodoros Stefos, Evangelos Paraskevaidis,1 And Konstantinos Zikopoulos. Comparison of misoprostol and dinoprostone for elective induction of labour in nulliparous women at full term: a randomized prospective study. reprod biol endocrinol. 2004; 2: 70

17. Murthy, B.K., Arkalgud, M.S. (2006). Misoprostol alone versus a combination of cerviprime and oxytocin for induction of labour. J Obstet Gynecol India, 56(5); 413-6.

18. Afia Ansar Shafiq, Samia Shuja, Faiqa Imran, Nasreen Kishwar, Nagina Liaquat. Comparison of Misoprostol with Dinoprostone for Induction of Labor in Postdated Pregnancy. Journal of Surgery Pakistan (International) 19 (1) January - March 2014

19. Jaju PB, Ramya D. Comparative Study of Intra-Vaginal Misoprostol with Intra-Cervical Dinoprostone Gel for Induction of Labour.

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Original Article

A PROSPECTIVE OBSERVATIONAL STUDY OF VITAMIN D3 LEVEL IN REPRODUCTIVE AGE GROUP WOMEN WITH LEIOMYOMA UTERI

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ABSTRACT

Uterine leiomyomas are common benign tumours of the uterus whose pathogenesis remains poorly understood.In India, the incidence is high and it inflicts a heavy burden on women`s health and healthcare system by being a common aetiology for menorrhagia and dysmenorrhea in women of reproductive age group.Incidence is between 5.4 to 77%.Vitamin D deficiency is a proven risk factor in the pathogenesis of uterine fibroid in many studies conducted in different parts of the world but not many studies have been conducted on Indian women.

Methods: A total of 200 women of age group 18 to 45 years attending District Hospital,Howrah,West Bengal, were included in the study.Out of which,100 had leiomyoma and rest healthy women without leiomyoma serves as controls.Routine ultrasound examination and serum hormone analysis of Vitamin D3 were done.Serum FSH(Follicle Stimulating Hormone), LH (Luteinizing Hormone) were done on day 2 of menstruation.Statistical analysis of data was performed using SPSS Version 22 software.

Results: The mean serum concentration of vitamin D3 was significantly lower in women with uterine fibroids compared to controls ($p<0.003$).On further analysis, 68.4% of cases were found to be severely deficient (vitamin D3 <9 ng/ml) as compared to 27.12% of controls ($p<0.0002$).Besides that only 3.67% of cases had sufficient vitamin D level as compared to 22.16% of controls ($P<0.0002$).The Odds Ratio (OR) of occurrence of fibroid with serum Vitamin D3 level of < 12 ng/dl compared to that level > 12 ng/dl was 5.38 ($p<0.0001$).

Conclusion: Serum Vitamin D3 level inversely correlated with the burden of leiomyoma and possibly its deficiency is a causative factor for the occurrence of uterine fibroid in the reproductive age group women.

Keywords: Risk Factor, Uterine fibroid/ leiomyoma, Vitamin D deficiency.

BACKGROUND

Uterine leiomyomas are common benign tumours of the uterus whose pathogenesis remains poorly understood. In India, the incidence is high and it inflicts a heavy burden on women`s health and healthcare system by being a common an etiology for menorrhagia

and dysmenorrhea in women of reproductive age group. Incidence is between 5.4 to 77%[1].

Multifactorial etiopathology with hormonal factors, African-American race, nulliparity, obesity, and a positive history of fibroids are the risk factors for high rate of leiomyoma. They may be asymptomatic or can cause abnormal bleeding, pelvic pressure symptoms, infertility and growth or regress throughout the life[2].

Vitamin D3 deficiency has been suggested to be a risk factor in many chronic conditions like cardiovascular disease, autoimmune disease, and also in several types of cancers[3].Several examples of consistent in vitro and in vivo experimental evidence support in Europe and Africa support its implications in the pathogenesis of uterine fibroid. Three studies showed that both myometrial and leiomyoma cells are highly sensitive to the regulatory effect of 1,25-dihydroxyvitamin D3[4].

The biological effects of vitamin D3 is essentially through its activation of VDR (Vitamin D receptor) cellular receptor, which in turn alters transcription rate of target genes responsible for various biological responses. This includes reduction in cell proliferation and regulation of biological processes including angiogenesis, extracellular matrix production and immune response. In an vivo model of leiomyomas in rats, Halder et al demonstrated that 1, 25 – dihydroxy vitamin D3 causes a dramatic reduction in the dimension of the lesions[5,6].

Hypovitaminosis including vitamin D deficiency is very common in Indian women. To shed more light on the possible role of vitamin D in the development of this pathology, we took up this study to assess serum levels of vitamin D3 in reproductive age group women with and without fibroids.

METHODOLOGY

This study was designed as a cross-sectional observational study after approval of the Institutional ethics committee of District Hospital Howrah, West Bengal, India. (Ref no: HDH/How/IEC/Non-spon/592/06-2019) The study population included women between 18 to 45years of age visiting Obs & Gynae dept. Of District Hospital Howrah from 1st Feb 2019 to 31st Jan 2020.Women with at least one uterine fibroid of >1.5cm^3 in volume or larger in TVS along with serum FSH and LH level <10 mIU/ml measured on day 2 of their menstrual cycle were eligible as cases. Control subjects were recruited from women of similar age group as cases with normal uterus on ultrasound examination. Exclusion criteria were women with history of pregnancy or miscarriage within last 6 months, and women currently on hormonal therapy or vitamin supplements, patients with chronic diseases like hypertension, diabetes, autoimmune disorders, coronary, hepatic, or renal diseases were also excluded from both the groups. A written informed consent was obtained from all patients.

After a brief history and physical examination, all recruited patients underwent TVS (transvaginal sonography) using 6 MHz transvaginal probe. Parameters like uterine size (in three perpendicular planes), number of fibroid lesions, volume of all fibroid lesions (by Prolate Ellipse Formula = a x b x c x 0.523 where a is height, b is width, and c is depth) were noted down. Patints in whom TVS was not sufficient to evaluate fibroid lesion in their entire entity, especially in large sized fibroid, Transabdominal sonography (TAS) was performed. Blood samples were collected from all patients to measure serum FSH and serum vitamin D 25 (OH) D_3 level. Both these quantitative parameters were measured by automated chemiluminescent immunoassay (CLIA) technology.

Data analysis was performed using Statistics Package for Social Sciences version 22.0 (SPSS, Chicago, Illinois). Statistically significant differences were determined using Fisher`s exact test, $X^2$ test, unpaired Wilcoxon test, or Student`s t test, as appropriate. A stepwise forward logistic regression model was used to adjust for variables known to be associated with leiomyomas (body mass index (BMI), black ethnicity, parity). A P value <= 0.5 was considered statistically significant.

The sample size was calculated based on an expected concentration of 25-hydroxyvitamin D_3 in controls of 20.5 +/- 11.3 ng/ml from a previous study, and it was hypothesized that a decrease of 20% of this value in women with uterine fibroid would be clinically significant. Considering alpha error of 5% and power of study 80%, a calculated sample would have been a minimum of 170 patients with 85 patients in each group. However, we included all eligible 100 patients visited to our hospital during the study period along with 100 matching controls.

RESULTS

Baseline parameters of both case and control groups were comparable except a statistically significant higher BMI was noticed in women with fibroid (Table 1). Most of the patients were multiparous in both the groups. Menorrhagia (50%) was the most common presenting complaint followed by pain in abdomen (27.45%) and dysmenorrhea (14.22%) in women with fibroid.

Table 1: Baseline parameters of cases with fibroid and control

Parameters	Cases of fibroid (n = 100)	Controls (n = 100)	p value
Age (years)	40.79 +/- 3.21	42.18 +/- 4.37	0.649
BMI (kg/m2)	28.12 +/- 2.56	27.87 +/- 2.23	0.016
Parity (%)			
P_0	5.78%	5.78%	0.96
P_1	18.44%	14.78%	0.88
P_2	38.89%	42.06%	0.67
>= P_3	34.74%	37.11%	0.78
Age at menarche(years)	13.40 +/- 1.04	13.56 +/- 1.04	0.158
Day 2 serum FSH (IU/ml)	6.48+/- 2.20	6.88+/- 1.03	0.543
Day 2 serum LH (IU/ml)	7.45+/- 3.20	7.89 +/- 1.21	0.512
Demographic distribution			
Urban (%)	70.23%	66.12%	0.756
Rural (%)	29.77%	33.88%	0.675

Serum levels of 25-hydroxyvitamin D_3 were significantly lower in women with fibroids than in controls (Table 2). Severe deficiency of vitamin D_3 were significantly seen in women with fibroids than in controls (Table 2). Severe deficiency of vitamin D_3 (< 10 ng/dl) was noticed in 66% of women with fibroids and 28% in controls. Moreover, only 3.66% of women with fibroids had sufficient (>30 ng/dl) serum levels of vitamin D_3 as compared to 24% in controls. The odds ratio (OR) of occurrence of fibroid with serum vitamin D_3 level of <10 ng/dl compared to that of serum vitamin D_3 level of > 10 ng/dl was 3.56 (95% C.I: 2.11 – 8.94) (p = 0.0001)

Table 2: Serum vitamin D_3 level in cases with fibroid and control

Parameters	Cases of fibroid (n = 100)	Controls (n = 100)	p value
Serum level (ng/dl)	10.27 +/- 5.14	24.45+/- 15.12	<0.0002
Serum vitamin D level categories n (%)			
Severe Deficiency (<10)	50	32	<0.0002
Deficiency (10-20)	30	24	0.67
Insufficient (20-30)	12	24	0.002
Sufficient (>30)	8	20	0.001

Further analysis of cases in terms of fibroid number and size was performed in relation to serum vitamin D_3 level to explore possible association. In women with number of fibroids more than two, the serum vitamin D_3 level was lower in comparison with women with fibroids less than two. However, the result was not statistically significant (8.36 +/- 6.45 ng/dl vs 11.24 +/- 7.22 ng/dl; p = 0.36). We failed to find out any correlation between volume of the largest fibroid and serum vitamin D_3 level.

Table 3 Serum vitamin D_3 and number of fibroids

No. of Fibroids	No. of patients	Level of serum vitamin D (ng/ml) (mean +/- SD)	p value
< 2	70	12.25+/- 6.45	0.29
>= 2	30	7.56+/- 4.77	0.11

DISCUSSION

Our study demonstrated significantly lower serum vitamin D_3 level in women with fibroid as

compared to control population ($p<0.0002$). Furthermore, the relative odd of the presence of fibroid in a woman with vitamin D_3 level <10ng/dl was 5.34 (95% confidence interval (CI) 3.45-8.67) ($p=0.0001$). This finding suggests a possible inverse correlation between serum vitamin D_3 and uterine fibroid in the present study population which corroborates the results of the studies conducted on different populations outside India.

A cross sectional study conducted by Halder et al included 104 women with fibroid and 50 controls without the disease. They similarly reported lower mean serum vitamin D_3 concentration among cases with fibroid. A retrospective analysis of the data by Baird and colleagues reported that women with sufficient vitamin D_3 (>20 ng/dl) had an estimated 32% lower odds of fibroids compared with those with vitamin D insufficiency (adjusted odds ratio 0.68, 95% confidence interval 0.48-0.96)[7,8,9]. So, all these studies conducted in different geographical locations confirm our study hypothesis. However, in contrast to most of these studies, serum vitamin D_3 level is significantly lower with fibroid in our study[10,11].

On categorical analysis of vitamin D levels, we found that 66% of women with fibroid are associated with severe deficiency (<10 ng/dl) as compared to 28% in controls. A similar kind of analysis was performed by Paffoni et al in their study which revealed that 15% of women with fibroid had severe deficiency as compared to 7% in controls and sufficient vitamin D level was found in 37% of cases as compared to 45% in controls. So, the correlation appears to be quite relevant in our study as compared to the study by Paffoni et al.as a greater number of women with fibroid in our study have severe vitamin D_3 deficiency compared to controls[12,13].

The benefit of measuring 25-hydroxyvitamin D_3, for monitoring serum vitamin D level, is that it represents the total body vitamin D from dietary intake, sunlight exposure and peripheral conversion of vitamin D. On the other hand, it has a shorter half-life of 15days.Therefore, its level as a causative factor in the development of fibroid can be erroneous. However, studies found that baseline serum vitamin D_3 level remains stable over long period of time (i.e person with a particular serum level tends to remain constant during multiple years of follow up)[14]

The biggest problem in drawing inferences from a cross-sectional designed studies are confounders. We tried to address some of the risk factors associated with the development of fibroid like age, parity, and BMI by carefully choosing controls. The possibility of reverse causation is also an issue to be dealt with.

Another pertinent question, which needs to be answered, is whether vitamin D deficiency is implicated in the development or growth of the fibroid. Our data did not reveal any significant correlation between the fibroid number and size with serum vitamin D_3 level, which indirectly suggested its role in both development and growth of fibroid. In contrast, Paffoni reported that the vitamin D deficiency correlated more with the number than the size of the fibroid, suggesting its implication more with the development than the growth of fibroid[15,16].

The most suitable approach to fulfill the aim of demonstrating a causal relationship would be a prospective long-lasting cohort study with serial monitoring of vitamin D status and regular follow up of patients as to see how many of them actually develop uterine fibroid. This type of exhaustive analytical study may take many years and difficult to achieve[17,18].

Halder et al reported reduction in size of uterine fibroid in the Eker rat model after vitamin D_3 supplementation. Some in vitro studies have also found a dose-dependent inhibitory effect of vitamin D on human fibroid cell growth. Most of the study demonstrated the effect of 1, 25-dihydroxyvitamin D_3 on apoptosis, modulation of several cell growth genes, protein synthesis, and cell proliferation. These functions are the base of anti-tumor effects of 1,25-dihydroxyvitamin D_3 on leiomyoma[19].

One of the plausible explanations towards fibroid development is altered extracellular matrix production due to aberrant response to tissue repair. Vitamin D might suppress this

abnormal response by regulating the extracellular matrix production. In addition, studies have speculated that it inhibits catechol-O-methyl transferase an enzyme supposed to be overexpressed in human uterine fibroid leading to suppression of growth of fibroid cells[20].

In India dietary deficiency appears to be the primary a etiology of vitamin D deficiency in females. Moreover, the incidence of uterine fibroid in reproductive age group is around 37% as reported by a study in South India. This number in all probabilities might br higher as many more are undetected. So, implication of vitamin D deficiency in uterine fibroid will bring in a whole new therapeutic aspect and it will impart a huge impact in the outcome[21,22].

CONCLUSION

Our study showed a definite indirect association of vitamin D deficiency and uterine fibroid in this part of India. To start supplements at the correct time and spreading awareness among patients should also be given due importance. However, further studies are warranted in this regard.

LIMITATION

This study has some limitations that should be addressed in future studies. Data regarding the extent of sun exposure, measurement of dietary intake of calcium and vitamin D, skin measurement of dietary intake of calcium and vitamin D, skin colour, use of sunscreens and other likely factors that are associated with vitamin D deficiency are not included in this study. Further researches are needed to evaluate these causes of vitamin D deficiency as well before assessing its relation with uterine fibroids in women of reproductive age group.

Compliance with Ethical standards

Conflict of interest Dr Annesha Dutta, Dr Murari Mohan Koley, Dr Preeti Gautam, declare that they have no conflict of interest.

Informed Consent in Studies with Human Subjects All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008. Informed consent was obtained from all patients for being included in the study.

REFERENCES

1.Myers ER, Barber MD, Gustilo-Ashby T, Couchman G, Matchar DB, McCrory DC. Management of uterine leiomyomata: what do we really know? Obstet Gynecol. 2002;100:8 –17.

2. Laughlin SK, Schroeder JC, Baird DD. New directions in the epi- demiology of uterine fibroids. Semin Reprod Med. 2010;28:204– 217.

3. Baird DD, Dunson DB, Hill MC, Cousins D, Schectman JM. High cumulative incidence of uterine leiomyoma in black and white wom- en: ultrasound evidence. Am J Obstet Gynecol. 2003;188:100 –107.

4. Somigliana E, Vercellini P, Daguati R, Pasin R, De Giorgi O, Cro- signani PG. Fibroids and female reproduction: a critical analysis of the evidence. Hum Reprod Update. 2007;13:465–476.

5. LukJ, TorrealdayS, NealPerryG, PalL.Relevance of vitamin D in reproduction. Hum Reprod. 2012;27:3015–3027.

6. Sabry M, Al-Hendy A. Innovative oral treatments of uterine leio- myoma. Obstet Gynecol Int. 2012:943635.

7. Bläuer M, Rovio PH, Ylikomi T, Heinonen PK. Vitamin D inhibits myometrial and leiomyoma cell proliferation in vitro. Fertil Steril. 2009;91:1919 –1925.

8. Sharan C, Halder SK, Thota C, Jaleel T, Nair S, Al-Hendy A. Vi- tamin D inhibits proliferation of human uterine leiomyoma cells via catechol-O-methyltransferase. Fertil Steril. 2011;95:247–253.

9. Halder SK, Goodwin JS, Al-Hendy A. 1,25-Dihydroxyvitamin D3 reduces TGF-beta3-induced fibrosis-related gene expression in hu- man uterine leiomyoma cells. J Clin Endocrinol Metab. 2011;96: E754 –E762.

10. Halder SK, Sharan C, Al-Hendy A. 1,25-Dihydroxyvitamin D3 treatment shrinks uterine leiomyoma tumors in the Eker rat model. Biol Reprod. 2012;86:116.

11. SabryM,HalderSK,AllahAS,RoshdyE,RajaratnamV, Al-Hendy A. Serum vitamin D3 level inversely correlates with uterine fibroid volume in different ethnic groups: a cross-sectional observational study.

Int J Womens Health. 2013;5:93–100.
12.Khyade RL. A study of menstrual disturbances in cases of fibroid uterus.Int J Reprod Contracept Obstet Gynecol. 2017;6:2494-7.
13.Paffoni A, Somigliana E, Vigano` P, et al. Vitamin D status in women with uterine leiomyomas. J Clin Endocrinol Metab.2013;98(8);E1374-8.
Lippman SA, Warner M, Samuels S, et al. Uterine fibroids and gynecologic pain symptoms in a population- based study. Fertil Steril 2003; 80: 1488-94.
14.Flake GP, Andersen J, Dixon D. Etiology and pathogenesis of uterine leiomyomas: a review. Environ Health Perspect 2003; 111: 1037-54.
15. Kjerulff K, Langenberg P, Seidman J, Stolley P, Guzinski G. Uterine leiomyomas: racial differences in severity, symptoms and age at diagnosis. J Reprod Med 1996; 41: 483-90.
16. Munusamy MM, Sheela WG, Lakshmi VP.Clinical presentation and prevalence of uterine fibroids: a 3 year study in 3-decade rural South Indian women. Int J Reprod Contracept Obstet Gynecol. 2017;6:5596-601.
17. Pérez-López FR, Ornat L, Ceausu I, et al. EMAS position statement: management of uterine fibroids. Maturitas 2014; 79; 106-16.
18. Parker WH. Etiology, symptomatology and diagnosis of uterine myomas. Fertil Steril 2007; 87: 725-36.
19.Olive DL, Lindheim SR, Pritts EA. Non-surgical management of leiomyoma: impact on fertility. Curr Opin Obstet Gynecol 2004; 16: 239-43.
20.Sharan C, Halder SK, Thota C, et al. Vitamin D inhibits proliferation of human uterine leiomyoma cells via catechol-omethyl-transferase. Fertil Steril. 2011;95:247-53.
21.Ylikomi T, Laaksi I, Lou YR, et al. Anti-proliferative action of vitamin D. Vitam Horm. 2002;64:357-406.
22.Gupta A. Vitamin D deficiency in India: prevalence, causalities and interventions. Nutrients. 2014;6(2): 729-75.

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Original Article

IMPACT OF COVID 19 PANDEMIC ON MATERNAL HEALTH CARE SERVICES

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ABSTRACT

BACKGROUND: COVID-19 pandemic suddenly attracted focus of entire health care system which leads to disrupted routine maternal health services. Furthermore, lockdown and curfew made scarcity of vehicle for transportation; there was also fear of attending hospital facilities or health stuffs. There are multiple studies indicates drastic decrease at hospital admission and institutional delivery rate and increased maternal mortality and miscarriage. This retrospective study is to find out vaginal delivery, caesarean section, maternal mortality and admission rate changes in pre and post COVID lockdown period in India.

METHODS: *Study Design:* This is a retrospective, observational study of women admitted at Maternity ward of Department of Obstetrics and Gynaecology, COM &JNM Hospital, Kalyani, West Bengal from January 2019 to December 2021 were included in the study. The data was taken and compiled from the admission register and was analysed in terms of vaginal delivery, caesarean section, maternal mortality etc.
Study area: Maternity ward of Obstetrics & Gynaecology Department of, College of Medicine & JNM Hospital, Kalyani.

Study population: All women admitted in the Maternity ward of Obstetrics & Gynaecology, College of Medicine & JNM Hospital, Kalyani.

RESULTS: We can clearly see decline in admission rate after COVID-19 pandemic with reduced vaginal and caesarean section rate. Delivery to admission rate increased during lockdown and post lockdown period indicating people seeking medical attention only when it is unavoidable. We also observed very high maternal mortality rate in lockdown and post lockdown period compared to pre-pandemic time.

CONCLUSION: This study identifies a significant decline in hospital admission, vaginal delivery and caesarean section rate after India was hit by COVID-19. Though this was mediated by multiple factors there is no doubt that mothers experienced either delay or denial of maternal health services during this pandemic.

KEY WORDS: Pandemic, Health care services Surgical termination of pregnancy, Dilatation and evacuation, Contraception.

INTRODUCTION

COVID 19 was first documented in Wuhan, China at the end of 2019, which spread rapidly across the globe. World Health Organization declared COVID-19 as pandemic on 11th March' 2020 and India declared nationwide lockdown on 26th March' 2022.[1] This COVID-19 pandemic suddenly attracted entire focus of health care system which leads to disrupted routine medical services including ante-natal check-up.

Emerging evidence suggests that rates of decreased hospital admission and institutional delivery rate and increased maternal mortality, miscarriage, stillbirth and preterm birth might have changed substantially during the pandemic.[2, 3] Reductions in health-care-seeking behaviour, as well as reduced provision of maternity services, has been suggested as a possible cause. Furthermore, lockdown and curfew made scarcity of vehicle for transportation; there was also fear of attending hospital facilities or health stuffs.

This retrospective study is to find out vaginal delivery, caesarean section, maternal mortality and admission rate changes in pre and post COVID lockdown period in India.

MATERIALS AND METHODS

Study Type: Retrospective Observational Study
Study design: Longitudinal
Study area: Maternity ward of Obstetrics & Gynaecology Department of, College of Medicine & JNM Hospital, Kalyani.
Study population: All pregnant women admitted in the Maternity ward of Obstetrics & Gynaecology, College of Medicine & JNM Hospital, Kalyani.
Study duration: This study will be conducted for 4 months after getting approval

Preparation	1 month
Data Collection	2 month
Analysis	1 month

Inclusion Criteria:

I. Only pregnant women were included.
II. Patient who was admitted in our institute.

Exclusion Criteria:

I. Women admitted for gynaecology problems.

Intervention: Not Done
Methods: Admission Data was collected from Emergency and Maternity Ward admission register, further patient details were collected from Operation Theatre, Labour room and Discharge register. We collected data from March'2019 to February'2022 and divided this entire 3-year period into 3 parts; From March'19 to Feb'20 is Pre-lockdown period, March'20 to Feb'21 to Lockdown period and March'21 to Feb'22 as post-lockdown period. Data was put on Microsoft Excel 2020 spread sheet and appropriate statistical method was used for analysis.
Data Analysis: For statistical analysis data were entered into a Microsoft excel spread sheet. A chi-squared test ($\chi 2$ test) was used to examine the differences between categorical variables in the same population when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. P-value ≤ 0.05 was considered for statistically significant. If the calculated p-value is below the threshold then the null hypothesis is rejected in favour of the alternative hypothesis.

RESULTS

We found total 32,192 patients admitted in our study period, among them 28,694 patients matched our inclusion criteria. Total 11286 patients were admitted in pre-pandemic 1 year period, after Government of India declared strict lockdown, we observed sharp decline in admission number to 9168 (- 18.76%), which further declined to 8240 in post-pandemic one year (- 26.98%).

During lockdown except month of June we have seen reduction in patient admission in every month, with July (- 30.39%) and August (- 32.77%) month had highest amount of patient admission reduction compared to previous year (Table 1). Only June month had increased rate of admission with 3.92% rise.

	Pre-lockdown	Lockdown		Post- lockdown	
	No	No	% Reduction	No	% Reduction
March	925	751	-18.81	587	-36.54
April	874	748	-14.41	590	-32.49
May	825	663	-19.63	508	-38.42
June	637	662	3.92	528	-17.11
July	997	694	-30.39	649	-34.90
August	1083	728	-32.77	818	-24.46
September	1050	810	-22.85	811	-22.76
October	1075	971	-9.67	850	-20.93
November	1036	936	-9.65	797	-23.06
December	1072	881	-17.81	773	-27.89
January	967	712	-26.37	672	-30.50
February	745	612	-17.85	657	-11.81
Total	11286	9168	-18.76	8240	-26.98

Table 1: Distribution of patient admission according to month during Pre-lockdown, Lockdown, and Post-lockdown period

In post-lockdown period, admission rate was further reduced compared to pre-lockdown time. We found March and May month had respectively 36.54% and 38.42% reduction in patient admission (Fig 1).

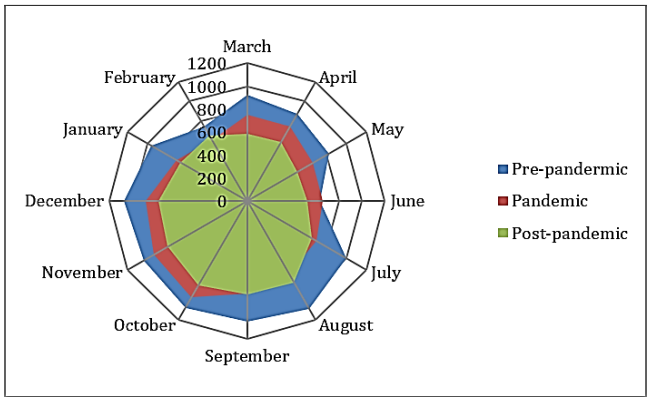

Fig 1: Rader Diagram showing gradual reduction in patient admission according to month

We have observed admission for pregnancy related complications other than delivery requirement declined remarkably. Whereas before lockdown 67% of total antenatal admission was for delivery, we found sharp rise of that to 75% and 78% in lockdown and post-lockdown respectively. This was also statistically significant in both cases with p value < 0.00001 (Table 2).

	Total Admission	Admission for Delivery	
Pre-lockdown	11286	7693	χ^2= 25.3 P < 0.0001
Lockdown	9168	6953	

Pre-lockdown	11286	7693	χ^2= 35.8 P < 0.00001
Post-lockdown	8240	6417	

Table 2: Comparison between Number of total admission and admission for delivery in Pre-lockdown period with Lockdown and Post-lockdown period

We noticed both during lockdown and post-lockdown period with reduction in total delivery number, there is also decline in C-section rate. From 52% during pre-lockdown time, C-section rate reduced to 45% during lockdown period and 44% during post-lockdown time.

	VD	C-Section	
Pre-lockdown	3692 (48%)	3981 (52%)	χ^2= 75.6 P < 0.0001
Lockdown	3846 (55%)	3107 (45%)	

Pre-lockdown	3692 (48%)	3981 (52%)	χ^2= 92.0 P < 0.00001
Post-lockdown	3608 (56%)	2809 (44%)	

Table 3: Comparison between Vaginal Delivery and C-section in Pre-lockdown period with Lockdown and Post-lockdown period

Compared to pre-lockdown period both in case of lockdown (p < 0.00001) and post-lockdown (p <

0.00001) period C-section rate reduced significantly in our study (Table 3).

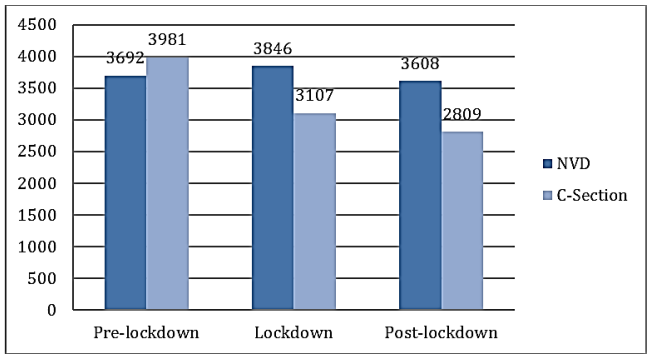

Fig 2: Bar Diagram showing type of delivery during Pre-lockdown, lockdown, and post-lockdown period

During lockdown and post-lockdown period maternal death increased abruptly. Rise of about 84% (n=24) observed in maternal death during lockdown period. Even in post-lockdown period 61% (n=21) higher maternal death was seen compared to pre-lockdown period. We found that rise in maternal death was statistically significant in both lockdown and post-lockdown period with p value 0.014 and 0.021 respectively (Table 3).

	Total Admission	Maternal Death	
Pre-lockdown	11286	13	χ^2= 5.99 P = 0.014
Lockdown	9168	24	

Pre-lockdown	11286	13	χ^2= 5.32 P = 0.021
Post-lockdown	8240	21	

Table 3: Comparison between Number of total admission and Maternal Death in Pre-lockdown period with Lockdown and Post-lockdown period

Similar to maternal death, still birth was also high during lockdown (n=257) and post-lockdown (n=231) period. Pre-lockdown period our Institutional stillbirth rate was 24 per 1000 total births that increased to 37 per 1000 during lockdown period and 36 per 1000 total births during post-lockdown period. Comparing to pre-lockdown period, rise in still birth rate both in lockdown (p <0.00001) and post-lockdown (p <0.00004) period was statistically significant.

	Total Delivery	Still Birth	
Pre-lockdown	7673	184	X^2= 19.78 P < 0.00001
Lockdown	6953	257	

Pre-lockdown	7673	184	X^2= 16.62 P < 0.00004
Post-lockdown	6417	231	

Table 4: Comparison between Number of total delivery and still birth in Pre-lockdown period with Lockdown and Post-lockdown period

DISCUSSION

We have done a retrospective study by collecting data from March'2019 to February'2022 for a period of 3 years and compiled it to understand the impact of COVID 19 pandemic on maternal health care services.

We have found a clear decline in admission rate during lockdown and also post-lockdown period by 18.76% and 26.98% respectively. Most common reason maybe was anxiety with fear of acquiring infection in the hospital. Similar to our findings *Manzoni E et al* [4] reported 35.4% reduction in admission rate. *Kumari V et al* [5] also revealed a 43·2% reduction in hospitalization in their institute.

There was drastic reduction in admission for pregnancy related complications other than delivery. Whereas during pre-lockdown period, among all admitted pregnant women only 67% were for delivery, which increased to 75% during lockdown period and 78% during post-lockdown period subsequently. Similar to our study *Manzoni E et al* [4] found frequency of admission for

elective caesarean section or labour induction increased from 47.5 % in 2019 to 53.6 % in 2020.

We found 4.17% increase in vaginal delivery in lockdown period (n=3846) compared to pre-lockdown (n=3692) period. We found significant increase in vaginal delivery during lockdown and post-lockdown period (p<0.0001), this may be due to delayed referral or unavailability for transport or ambulance services. *Eleje GU et al* [6] There was a significant decline in C-section rates from 46.8% in the pre-COVID-19 period to 40.0% during the COVID-19 period (p = 0.027). On the contrary, *Padhye R et al* [7] found Caesarian section deliveries in both government (32.8%, n = 19) and private health facilities (86.7%, n = 13) was high.

During lockdown period and even in post-lockdown period, there was shift of focus from routine ante-natal services and early picking up of high-risk cases by health care professionals. There was also negative sentiment for attending hospital facility unless extremely necessary takes its toll on overall maternal and foetal wellbeing. We have seen an overwhelming rise of 84% maternal death during lockdown period compared to pre-lockdown 1 year calendar year (p=0.014). Even during post-lockdown 1 year period maternal death was high, that also came statistically significant (p = 0.021). *Chmielewska B et al* [8] identified significant increases in stillbirth (OR 1·28 [95% CI 1·07-1·54]) and maternal death (OR 1·37 [95% CI 1·22-1·53]). Similar to maternal death we also found our institutional still birth rate rises from 24/1000 total birth to 37/1000 (p <0.00001) and 36/1000 (p < 0.00004) total births during lockdown and post-lockdown period respectively, both came statistically significant in our study. *Gurung, R et al* [9] also found that their institutional stillbirth rate increased from 14 per 1000 total births before lockdown to 21 per 1000 total births during lockdown (p=0·0002). Contrary to this *Kugelman N et al* [10] reported that though obstetric emergency events increased, the rates of neonatal and maternal morbidity did not change. For this, though we had seen somewhat increase in vaginal delivery, sharp rise in maternal and neonatal mortality indicates grossly neglected routine health care services.

CONCLUSION

In our retrospective study, we have observed a clear decline in overall admission rate during lockdown and also post-lockdown period by 18.76% and 26.98% respectively. There was drastic reduction in admission for pregnancy related complications other than delivery. Admission for delivery increased from 67% during pre-lockdown period to 75% during lockdown period and 78% during post-lockdown period subsequently. We have seen an overwhelming rise of 84% maternal death during lockdown period compared to pre-lockdown 1 year calendar year (p=0.014). Even during post-lockdown 1 year period maternal death was high, that also came statistically significant (p = 0.021). Similar to maternal death we also found our institutional still birth rate rises from 24/1000 total birth to 37/1000 (p <0.00001) and 36/1000 (p < 0.00004) total births during lockdown and post-lockdown period respectively.

During lockdown period and even in post-lockdown period, there was shift of focus from routine ante-natal services and early picking up of high-risk cases by health care professionals. There was also negative sentiment for attending hospital facility unless extremely necessary takes its toll on overall maternal and fatal wellbeing.

LIMITATIONS

In spite of every sincere effort my study has lacunae. The notable short comings of this study are:

1. The study has been done in a single centre.
2. As lockdown was withdrawn in phased manner, defining exact lockdown period could not be done.

The study was carried out in a tertiary care hospital, so hospital bias cannot be ruled out.

REFERENCES

1. *Ghosh, P., Ghosh, R., & Chakraborty, B. (2020). COVID-19 in India: Statewise Analysis and Prediction. JMIR public health and surveillance, 6(3), e20341. https://doi.org/10.2196/20341*
2. *Padhye R, Purushotham A, Paul M, et al. Accessing Maternal Health Care in the Midst of the COVID-19 Pandemic: A Study in Two Districts of Assam, India. Front Glob Womens Health.*

2022;3:750520. Published 2022 Mar 31. doi:10.3389/fgwh.2022.750520

3. *Calvert C, John J, Nzvere FP, et al. Maternal mortality in the covid-19 pandemic: findings from a rapid systematic review. Glob Health Action. 2021;14(sup1):1974677. doi:10.1080/16549716.2021.1974677*

4. *Dell'Utri, C., Manzoni, E., Cipriani, S., Spizzico, C., Dell'Acqua, A., Barbara, G., Parazzini, F., & Kustermann, A. (2020). Effects of SARS Cov-2 epidemic on the obstetrical and gynecological emergency service accesses. What happened and what shall we expect now?. European journal of obstetrics, gynecology, and reproductive biology, 254, 64–68. https://doi.org/10.1016/j.ejogrb.2020.09.006*

5. *Kumari, V., Mehta, K., & Choudhary, R. (2020). COVID-19 outbreak and decreased hospitalisation of pregnant women in labour. The Lancet. Global health, 8(9), e1116–e1117. https://doi.org/10.1016/S2214-109X(20)30319-3*

6. *Eleje GU, Ugwu EO, Enebe JT, et al. Cesarean section rate and outcomes during and before the first wave of COVID-19 pandemic. SAGE Open Med. 2022;10:20503121221085453. Published 2022 Mar 23. doi:10.1177/20503121221085453*

7. *Padhye R, Purushotham A, Paul M, et al. Accessing Maternal Health Care in the Midst of the COVID-19 Pandemic: A Study in Two Districts of Assam, India. Front Glob Womens Health. 2022;3:750520. Published 2022 Mar 31. doi:10.3389/fgwh.2022.750520*

8. *Chmielewska B, Barratt I, Townsend R, et al. Effects of the COVID-19 pandemic on maternal and perinatal outcomes: a systematic review and meta-analysis [published correction appears in Lancet Glob Health. 2021 Jun;9(6):e758]. Lancet Glob Health. 2021;9(6):e759-e772. doi:10.1016/S2214-109X(21)00079-6*

9. *Kc, A., Gurung, R., Kinney, M. V., Sunny, A. K., Moinuddin, M., Basnet, O., Paudel, P., Bhattarai, P., Subedi, K., Shrestha, M. P., Lawn, J. E., & Målqvist, M. (2020). Effect of the COVID-19 pandemic response on intrapartum care, stillbirth, and neonatal mortality outcomes in Nepal: a prospective observational study. The Lancet. Global health, 8(10), e1273–e1281. https://doi.org/10.1016/S2214-109X(20)30345-4*

Kugelman N, Lavie O, Assaf W, et al. Changes in the obstetrical emergency department profile during the COVID-19 pandemic. J Matern Fetal Neonatal Med. 2022;35(21):4116-4122. doi:10.1080/14767058.2020.1847072

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CLOMIPHENE CITRATE VERSUS LETROZOLE FOR OVULATION INDUCTION IN INTRAUTERINE INSEMINATION CYCLES: AN OBSERVATIONAL COMPARATIVE STUDY COMPARATIVE STUDY.

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ABSTRACT

BACKGROUND & AIM: Ovulation induction is widely practised in conjunction with intrauterine insemination (IUI). The present study is aimed to compare commonly used clomiphene citrate and letrozole for ovulation induction in IUI cycles.

MATERIALS & METHODS: In this observational study 120 infertile couples considered for ovulation induction and IUI were allocated to either clomiphene citrate group or letrozole group. After day2 baseline transvaginal sonography (TVS) tab clomiphene citrate 50mg or tab letrozole 2.5mg administered in respective groups twice daily from day2-6 of period and followed up by TVS adding injection human menopausal gonadotropin (HMG) and tab estradiol valerate accordingly till dominant follicle(s) reached >17mm when trigger for ovulation given and IUI performed 36hours later. Those who conceived were followed till ultrasound confirmation of cardiac activity. Statistical analysis was done using SPS Software 22.

RESULTS: Endometrial thickness on both day9 and the day of ovulation trigger was more favourable in letrozole group (pvalue<0.001). Size of dominant follicle on day9 and day11 were comparable in both the groups, however, on the day of ovulation trigger was statistically more in clomiphene citrate group (pvalue 0.0202). Mean number of dominant follicles was higher in clomiphene citrate group (pvalue<0.0074). Pregnancy up to cardiac activity was similar in both the groups (pvalue 0.836). HMG and estrogen were required more in clomiphene citrate group (pvalue 0.0016).

CONCLUSION: Letrozole can be used as first line treatment for ovulation induction in any infertile women undergoing IUI reducingthe need for gonadotropins and estrogen.

KEY WORDS: Letrozole, Clomiphene Citrate, Ovulation Induction, Intrauterine Insemination.

INTRODUCTION

The term "intrauterine insemination" (IUI) refers to the introduction of sperm into the uterine cavity. The history of IUI dates back to the 18th century. Dr John Hunter, a German physician is

regarded to have performed the procedure for the first time around 1790 which was later published by his executor Sir Everard Home after his death in the year 1799.[1] It has now become a common procedure in infertility practice& involves sorting & washing sperm & transferring it into the uterus around the time of ovulation. Controlled ovarian stimulation is commonly used in conjunction with IUI which usually results in better pregnancy rate than IUI done in natural cycles. Clomiphene citrate is a selective estrogen receptor modulator (SERM)that has been widely used for ovulation induction since its approval for clinical use. In spite of having both estrogen agonistic & antagonistic activity its action is primarily anti-estrogenic except in conditions where endogenous estrogen levels is extremely low.[2]The desirable central anti-estrogenic activity is the basis behind its mechanism of action but the peripheral anti-estrogenic activity as suggested by various studies is related to its associated untoward effects on the endocervix, endometrium, ovary, ovum & the embryo.[2,3,4,5]Estrogen receptor depletion might explain the poor pregnancy rate & increased early pregnancy loss in women receiving clomiphene citrate for ovulation induction.[6] It is also frequently associated with multi-follicular development increasing the overall risk of multiple pregnancy to approximately 7-10%.[2]

Letrozole, a third-generation aromatase inhibitor, has been successfully used for ovulation induction. It decreases estrogen production directly but does not deplete estrogen receptor in target tissues thus having no persistent anti-estrogenic effects. [2,5] Unlike clomiphene citrate it no undesirable effects on the endometrium or the endocervix& the cycles are mono-ovulatory.[2] Therefore, the study was conducted to compare the effects of clomiphene citrate and letrozole on ovulation induction in IUI cycles.

MATERIALS & METHODS

This was an observational study conducted in our infertility clinics from 1st July 2019 to 31st December 2020. Prior institutional ethical committee clearance was taken All infertile couples who attended the infertility clinics underwent basic infertility evaluation. Those who were considered for ovulation induction and IUI were counselled and referred to the assisted reproductive unit of our department. After taking proper consent couples recruited in the study were allocated to either clomiphene citrate group or letrozole group. 120 such couples completed the study (Unperformed) were included for comparison with 60 in each group.

Artificial insemination is considered for treating many couples presenting with unexplained infertility, various types of sexual dysfunction, cervical factor infertility &mild to moderate male subfertility owing to the fact that in IUI gamete density is increased at fertilization site. Couples with severe male infertility (sperm concentration<10million/ml), women> 35 years of age, women with bilateral blocked fallopian tubes, ovarian cyst or reaction to drugs were excluded from the study.

All women underwent baseline day 2 transvaginal sonography (TVS) for antral follicle count (AFC), endometrial thickness (ET) and to rule out any other pathology. Tablet clomiphene citrate 50mg or tablet letrozole 2.5mg administered orally in respective groups twice daily from day2 to day6 of menses. Women of both groups were followed up by TVS on day9, day11 of the cycle and on the day of ovulation trigger. On Day9womenwith more than three follicles of >10mm diameter and on the day of ovulation trigger those with >14 follicles of>11mm diameter or >11 follicles of >10mm diameter were excluded from study. On Day9 if the follicular size was less than 12mm and ET relatively thin (less than 5mm) injection human menopausal gonadotropin (HMG) 75IU intramuscular and tablet estradiol valerate 2mg twice daily orally was supplemented. When mature leading follicle(s) reached >17 mm in diameter, ovulation was triggered with10000 IU of human chorionic gonadotropin (HCG) intramuscularly or injection leuprolide 0.5mg subcutaneously for patients with polycystic ovarian syndrome & IUI was performed 36 hours later. Tablet dydrogesterone 10 mg twice daily orally was started from day16 onwards for 15 days for luteal phase support in all women. Two

weeks after IUI in women with amenorrhoea urine pregnancy test was done and those positive were followed up for 6 weeks till TVS confirmation of cardiac activity. We have performed IUI for maximum three times for each couple. Data collected for each woman was the mean of the outcome of total number of ovulation induction cycles she received.

RESULTS

The results of the present study have been tabulated in Table 1.

Table 1. Comparison of demographic variables causes of infertility

	Clomiphene citrate group		Letrozole group			
	Mean	SD	Mean	SD	CI	p value
Age	28.2273	3.5848	28.4286	3.5426	-1.0872 to 1.4898	0.7576
Body Mass Index	24.33	1.545	24.59	1.089	-0.2232 to 0.7432	0.28
Male factor infertility	14		12			0.825
Anovulatory infertility	19		23			0.5661
Unexplained infertility	27		25			0.854

TABLE 2 PRE-INDUCTION AFC AND RESULTS OF OVULATION INDUCTION

	Clomiphene citrate group	Letrozole group				
	Mean	SD	Mean	SD	CI	p value
ET on Day 9 (in mm)	4.3000	1.0801	5.4714	.2992	0.8849 to 1.4579	<0.0001
ET on day of trigger (in mm)	7.9636	1.8613	11.5643	.3633	-4.0855 to -3.1159	<0.0001
Size of dominant follicle on day 9 or day 11(in cm)	13.7818	1.5380	14.0750	1.3000	0.5148 to 0.5148	1.00
Size of dominant follicle on day of trigger (in cm)	18.47	1.26	18.03	0.712	0.0700 to 0.8100	0.0202
Mean number of dominant follicles	1.3	0.4754	1.1	0.3247	-0.3454 to -0.0546	<0.0074
Post wash sperm count (in million)	30.9091	2.9906	31.5000	2.9627	-0.4853 to 1.6671	0.2791
Post wash sperm progressive motility (in %)	64.1818	3.97	64.14	3.65	64.1429	0.954
Estrogen and HMG requirement (no. of patients)	11		28		-0.3454 to -0.0546	0.0016
Pregnancy upto cardiac activity (no. of patients)	15		17			0.836

DISCUSSION

Both clomiphene citrate and letrozole group participants in our study had comparable baseline characteristics with regards to age, body mass index and cause of infertility (p value non-significant 0.7576, 0.28, 0.825, 0.566, 0.854). Day 2 AFC in both the groups was similar with non-significant p value of 0.1695. Post wash sperm count and progressive motility was also similar in both the groups (p value 0.2791 & 0.954 respectively).

In the present study endometrial thickness on day 9 was statistically higher in letrozole group than in clomiphene citrate group (5.4mm vs. 4.3mm respectively, p value<0.0001). Similarly on the day of ovulation trigger endometrial thickness was higher in letrozole group than in clomiphene citrate group (11.56mm vs. 7.96mm respectively, pvalue<0.0001). Sohrabvand et al. in their study also found that the mean endometrial thickness on the day of HCG administration was significantly higher in letrozole group (0.82±0.13 vs. 0.55±0.28 cm respectively, p value 0.0009) corresponding to the findings of our study.[7] Atay et al. also reported significantly higher endometrial thickness in the letrozole group than in clomiphene citrate group (p value 0.0001).[8]

Mean size of dominant follicle on the day of ovulation trigger in the present study was statistically more in clomiphene citrate group than in letrozole group (18.47±1.26 vs18.03±0.712respectively, pvalue0.02). This may be due to the fact that in clomiphene citrate group sometimes two dominant follicles emerged with a variation from 17mm to22mm. Pregnancy rate, however, was similar in both the groups (pvalue0.836). The peripheral anti-estrogenic action of clomiphene citrate on endometrium and cervical mucus might partly explain the discrepancy in ovulation rate and pregnancy rates. [2,5] Pregnancy rate per cycle was also reported to be similar in both letrozole & clomiphene citrate group in a study by Bayar et. al. (p value 0.9).[9] However a statistically significant higher clinical pregnancy rate was noted in letrozole group in a randomized control trial by Ibrahim et al. (23.07 vs 10.68%, p value

<0.001).[6] Also in the study by Atay et al. pregnancy rate was statistically higher in letrozole group as compared to clomiphene citrate group (p value 0.037).[8] Unlike clomiphene citrate, letrozole is devoid of any anti-estrogenic peripheral action.[5]

Clomiphene citrate acts through the depletion of central estrogen receptors, which in turn reduces the negative feedback mechanism of estrogenon hypothalamus and pituitary.[2] The effect is an increase in gonadotropin secretion leading to multiple follicular development. On the other hand, letrozole causes a decrease in peripheral oestrogen production, which is responsible for the increased gonadotropin secretion in the early part of the cycle; however, due to its short half-life, its effect wears off in the late follicular phase, estrogen production by the growing follicles restores the normal negative feedback mechanism on the gonatotropin secretion ultimately resulting in mono-follicular development in the late follicular phase of the cycle. In our study mean number of dominant follicles was statistically higher in clomiphene citrate group (p value <0.0074). Similarly in a study by Akbary-Asbagh et al.the mean number of dominant follicles was 1.8 in the clomiphene citrate ± HMG group as compared to 1.4 in the letrozole ± HMG group.[10]In another study by Jee et al.the mean number of dominant follicles in the letrozole + HMG group was 3.2 ± 1.7 and in the clomiphene citrate + HMG group was 5.6 ± 2.4, which was statistically significant with a pvalue of <0.0001.[11]In our study the requirement of HMG and estrogen was statistically less in letrozole group (p value 0.0016).

CONCLUSION

Letrozole can be used as first line treatment for ovulation induction in any infertile women irrespective of the indication which would reduce the need for supplemental gonadotropins and estrogen in women undergoing IUI.

REFFRENCES

1. *Gary N. Clarke, A.R.T. and history, 1678–1978.* Human Reproduction. *Jul 2006; 21:1645-50.*
2. *Fritz MA, Speroff L. Clinical Gynaecologic Endocrinology & Infertility. 8th ed. Philadelphia: Lippincott Williams & Wilkins;2011. 1296-1309p*
3. *Nakamura Y, Ono M, Yoshida Y, Sugino N, Ueda K, Kato H. Effects of Clomiphene citrate on the endometrial thickness and echogenic pattern of the endometrium. FertilSteril. 1997;67:256–60.*
4. *Nelson LM, Hershlag A, Kurl RS, Hall JL, Stillman RJ. Clomiphene citrate directly impairs endometrial receptivity in the mouse. FertilSteril. 1990;53:727–31.*
5. *Fisher SA, Reid RL, Van Vugt DA, Casper RF. A randomized double-blind comparison of the effects of Clomiphene citrate and the aromatase inhibitor letrozole on ovulatory function in normal women. FertilSteril. 2002;78:280–5.*
6. *Ibrahim MI, Moustafa RA, Abdel-Azeem AA. Letrozole versus Clomiphene citrate for superovulation in Egyptian women with unexplained infertility: a randomized controlled trial. Arch Gynecol Obstet. 2012;286:1581–7.*
7. *Sohrabvand F, Ansari SH, Bagheri M. Efficacy of combined metformin–letrozole in comparison with metformin–clomiphene citrate in clomiphene-resistant infertile women with polycystic ovarian disease. Hum Reprod. 2006;21:1432–1435.*
8. *Atay V, Cam C, Muhcu M, Cam M, Karateke A. Comparison of letrozole and clomiphene citrate in women with polycystic ovaries undergoing ovarian stimulation. J Int Med Res. 2006;34:73–76.*
9. *Bayar U, Tanriverdi HA, Barut A, Ayoğlu F, Ozcan O, Kaya E. Letrozole vs. clomiphene citrate in patients with ovulatory infertility. FertilSteril. 2006;85:1045–1048.*
10. *Akbary-Asbagh F, Heidar Z, Frozan-Fard F, Nouri K, Azmodeh O, Ghasemynejad A et al. Evaluation of letrozole therapeutic effect in infertile women. ActaMedicaIranica 2007;45:199-203 .*
11. *Jee BC, Ku SY, Suh CS, Kim KC, Lee WD, Kim SH. Use of letrozole versus clomiphene citrate combined with gonadotropins in intrauterine insemination cycle: A pilot study. FertilSteril 2006;85:1774-7.*

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Original Article

FETAL KIDNEY LENGTH, TRANSVERSE DIAMETER OF FETAL HEART, CONVENTIONAL USG PARAMETER FOR ESTIMATION OF GESTATIONAL AGE IN GROWTH RESTRICTED FETUSES IN THIRD TRIMESTER PREGNANCY.

Subrat Panda[1]✉, Ananya Das[2]

ABSTRACT

INTRODUCTION: An accurate determination of the gestational age and the expected date of delivery is fundamental to the successful management of pregnancies specially in fetal growth restriction. In Fetal Growth Restricted (FGR) fetuses, there is a huge discrepancy in gestational age by last menstrual period and per abdominal obstetric findings, which gets even worse when the lady is not sure of her last menstrual period. Currently there is no available third trimester ultrasound parameter which can predict gestational age accurately and one such parameter which remains unaltered even by Fetal Growth Restricted Fetuses (FGR) should solve the therapeutic dilemma of the obstetrician and hence nullify the probability of iatrogenic prematurity. There are studies on fetal kidney length and transverse diameter of the heart alone can be used to predict the gestational age in third trimester with normal pregnancy. But for the FGR fetuses, we did not come across any such parameter to estimate the gestational age at third trimester. The present study was done to determine whether fetal kidney length and transverse diameter of the heart can be used to estimate the gestational age in case of growth restricted fetuses. In third trimester of pregnancy.

METHODOLOGY: It is an observational cohort study carried out in the department of Obstetrics & Gynaecology, NEIGRIHMS in collaboration with the department of Radiology from January 2016 to January 2018. A total number of 70 women with singleton pregnancy with spontaneous conception and excellent dates and diagnosed as small for gestational age clinically and ultrasonologically were included for the study. In our present study we included cases of FGR in third trimester having excellent dates and subjected them for conventional biometry for dating and at the same time measured fetal kidney length and fetal transverse diameter of heart for dating of pregnancy. The obtained data were recorded in Microsoft Excel sheet and analysed to obtain PEARSON Correlation coefficient and prediction of p value by using SPS SOFTWARE 21 VERSION.

OBSERVATION: In our study fetal kidney length was found to be the superior parameter to estimate the gestational age in growth retarted fetuses (r^2-0785 and p value<0.0001). Next parameter was transverse diameter of fetal heart which can accurately estimate the gestational age in growth retatded fetuses (r^2-0.4686and p value<0.0001). Amongst the conventional parameters femur length was most

accurate (r^2-0.3080, p value <0.0001). Other conventional diameters like BPD, HC, AC and combined parameter did not correlate with the gestational age calculated from LMP or CRL in first trimester ultrasound.

CONCLUSION: Fetal kidney length can be used as a reliable parameter to estimate the gestational age in growth retarded pregnancies where all the conventional ultrasonological parameter fails to estimate the gestational age accurately.

Key words- Fetal growth restriction; conventional USG parameter; Fetal kidney length; Transverse diameter of fetal heart.

KEY WORDS: Letrozole, Clomiphene Citrate, Ovulation Induction, Intrauterine Insemination.

INTRODUCTION

An accurate determination of the gestational age and the expected date of delivery is fundamental to the successful management of pregnancies especially the fetal growth restricted babies. Proper assignment of the expected date of delivery is necessary in order to obtain and appropriately interpret laboratory tests and to plan and execute therapeutic maneuvers. There is increase in the number of high-risk pregnancies coming to the hospitals for the first time in third trimester. This poses a challenge to the obstetrician as timely decision to terminate the pregnancy is of utmost importance. This in turn necessitates the confirmation of gestational age precisely. Because without confirmation of the gestational age, termination of pregnancy might lead to iatrogenic prematurity. This may not be difficult in those who are sure of their gestational age or who had a first trimester or early second trimester scan. However, the situation is different in India where most of the pregnant women come to hospital for the first time in third trimester for institutional delivery. This also includes quite a number of fetal growth restricted fetuses (FGR). There is a huge discrepancy in gestational age by last menstrual period and per abdominal obstetric findings, which might be even worse when the lady is not sure of her last menstrual period. Because FGR needs to be terminated in appropriate time, gestational age has to be confirmed by the early ultrasound findings which might not available putting the obstetrician in therapeutic dilemma.

Currently there is no available third trimester ultrasound parameter which can predict gestational age accurately. One such parameter which remains unaltered even by Fetal Growth Restricted Fetuses (FGR) will solve the therapeutic dilemma of the obstetrician and nullify the probability of iatrogenic prematurity. Nirmala Shivalingaiah et al. conducted a study on "Fetal Kidney length as a parameter for determination of gestational age in pregnancy" and observed that the mean deviation from the gestational age at all the weeks is least for Kidney Length which correlated well with the assigned gestational age and found almost same as all the ultrasound biometric parameters put together [1]N. Hephzibah Kirubamani, M.R. Meenatshi et al found that transverse diameter of heart is more accurate for measuring the gestational age in third trimester in normal growing fetuses where LMP is not known[3]. But these studies are for normal growing fetuses. The present study is conducted whether fetal kidney length and transverse diameter of the heart are used to measure the gestational age in case of growth restricted fetuses in third trimester.

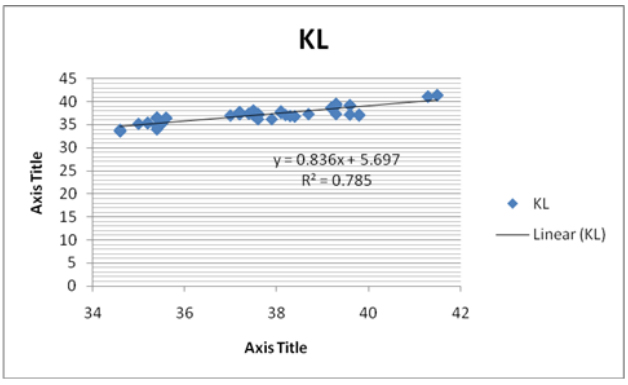
METHODOLOGY

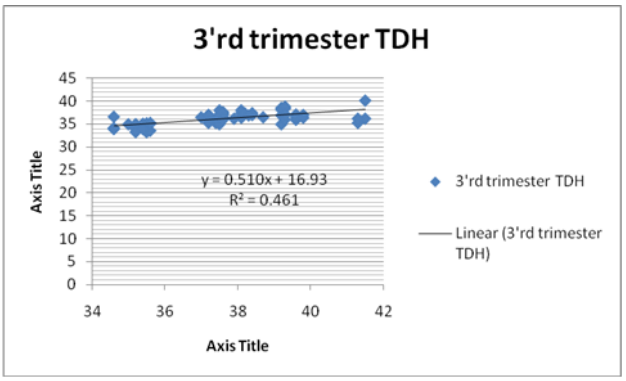
It is an observational study carried out in the department of Obstetrics & Gynaecology, NEIGRIHMS from January 2016 to January 2018. A total number of 70 women attending to our hospital for safe confinement and institutional delivery were enrolled for the study. Women with singleton pregnancy with spontaneous conception and with accurate dating scan and

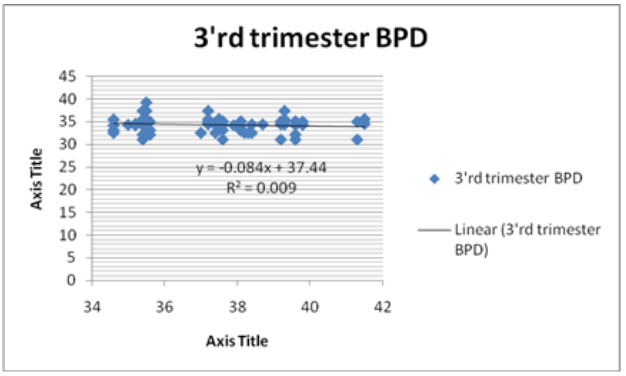
sure LMP and diagnosed as small for gestational age clinically and ultrasonologically were included for the study. Unknown last menstrual period, irregular menses, multiple gestation and fetal anomaly were excluded from the study group. Dating of pregnancy was done with women with adequate clinical information having known. L.M. P.; 28-30 days cycle; no recent use of Oral Contraceptives Pills; uterine size in agreement with the dates and the ultrasound examination between 16 to 24 weeks indicating that the fetal measurements are in agreement with the gestational age. b) Patients with inadequate or incomplete clinical information but with two ultrasound examination between 16-24 weeks showing linear fetal growth and similar USG EDD. Here we considered the dating of pregnancy with CRL length in first trimester. We suspected FGR if symphysiofundal height is less than 4cm from the gestational period, abdominal girth lesser than expected and serial growth curve by palpation lags and liquor is diminished clinically. After clinical suspicion we considered Ultrasound for biometry and biophysical profile. In our present study we included cases of FGR with excellent dates and subjected them to conventional biometry for dating and at the same time measured fetal kidney length and fetal transverse diameter of heart for dating of pregnancy in the third trimester. Transverse diameter of heart was taken in mm at closed atrioventricular valve junction, outer to outer points in four chamber view of heart. (This is normally done in our center) Three measurements of transverse diameter was done and the mean value was taken finally. Kidney length measurements were obtained in sagittal plane, when full length of kidney with renal pelvis is visualized. Maximum length of anyone single kidney is measured from upper pole to lower pole at least thrice and mean of the measurement is taken. Normal renal length measurements in the fetus increase with gestational age. If the difference in gestational age calculated from Last menstrual Period and first trimester crown rump length measurement is less than 7days gestational age is finally calculated based on LMP. If the difference is more than 7days than we calculated gestational age based on CRL length. It was a consecutive sampling method and 76 cases of of FGR was found in 2years the obtained data were recorded in Microsoft Excel sheet and analyzed to obtain PEARSON Correlation coefficient and prediction of p value by using SPS SOFTWARE 21 VERSION.

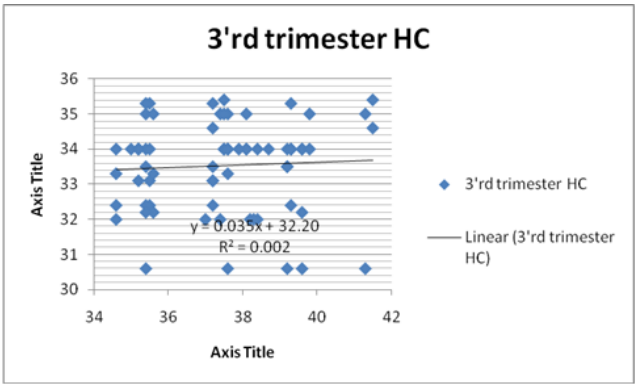
OBSERVATION

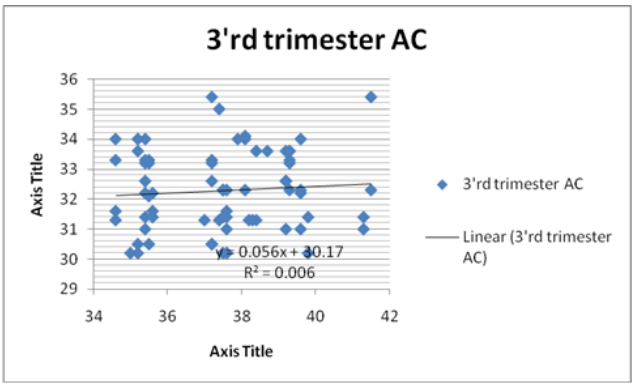
Parameter	Slope	Y intercept	R Square	95% confidence interval	P Value
Kidney Length	0.8369 ± 0.05299	5.698 ± 1.988	0.7858	0.7310 to 0.9427	< 0.0001(S)
Transverse Diameter of Heart	0.5186 ± 0.06698	16.65 ± 2.514	0.4686	0.3848 to 0.6524	< 0.0001(S)
BPD	-0.08408 ± 0.1032	37.44 ± 3.872	0.009663	-0.2902 to 0.1221	0.4182
HC	0.03534 ± 0.08314	32.21 ± 3.119	0.002649	-0.1307 to 0.2014	0.6722
FL	0.4131 ± 0.07510	18.44 ± 2.817	0.3080	0.2631 to 0.5631	< 0.0001(S)
AC	0.05597 ± 0.08729	30.18 ± 3.274	0.006011	-0.1184 to 0.2303	0.5235
Combined Gestational Age	0.1051 ± 0.06166	29.57 ± 2.313	0.04097	-0.01806 to 0.2282	0.0929

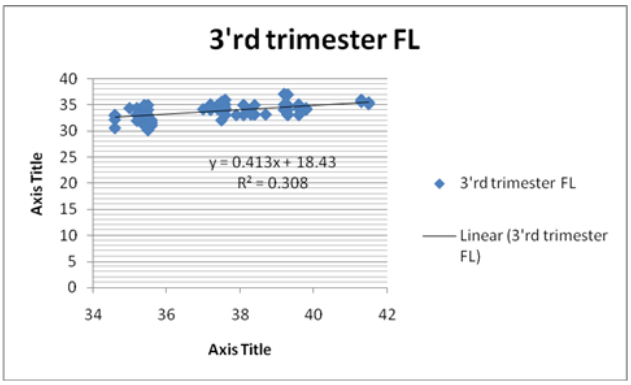


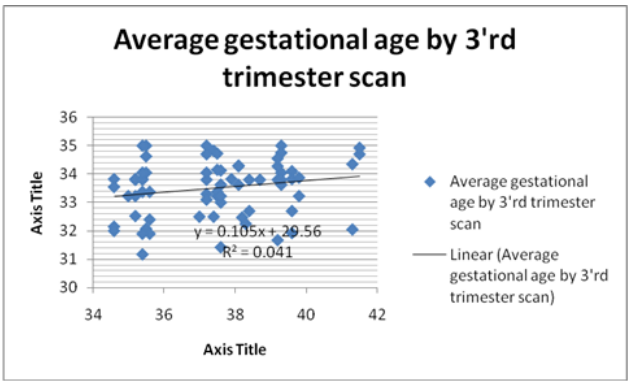












In our study fetal kidney length was found to be superior parameter to estimate the gestational age in growth retarted fetuses (r^2-0785 and p value<0.0001). Next parameter was transverse diameter of fetal heart which can accurately estimate the gestational age in growth retarded fetuses (r^2-0.4686 and p value<0.0001). Among the conventional parameters, femur length was most accurate (r^2-0.3080, p value <0.0001). Other conventional parameters like BPD, HC, AC, FL and combined parameter did not correlate to the gestational age calculated from LMP or first trimester CRL

DISCUSSION

In Fetal Growth Restricted Fetuses (FGR), there is a huge discrepancy in gestational age by last menstrual period and per abdominal obstetric findings or even worse when the lady is not sure of her last menstrual period. Because FGR needs to be terminated in appropriate time, gestational age has to be confirmed by the early ultrasound findings which is not available most of the time and leaves the obstetrician in the therapeutic dilemma. Usually conventional ultrasonological parameter like BPD, HC, AC, FL do not correspond to gestational age.(4) Nirmala Shivalingaiah et al. conducted a study on "Fetal Kidney length as a parameter for determination of gestational age in pregnancy" and observed that the mean deviation from the gestational age at all the weeks is least for Kidney Length which correlated well with the assigned gestational age and found almost same as all the ultrasound biometric parameters put together [1].Indu Kaul et tal in their study found that fetal kidney length is the most accurate single parameter for estimating gestational age than other biometric indices[2].N Hephzibah Kirubumani et al found in their study that fetal heart diameter at 32-36 weeks of gestation was equally effective to estimate the gestational age in third trimester compared to conventional USG parameter[3]. But these studies are confined to normal growing fetuses. In our study we used fetal kidney length and transverse diameter of heart to measure the gestational age in Fetal Growth Restriction. In our study, kidney length was found to be superior parameter to estimate the gestational age in FGR (r^2-0785 and p value<0.0001). Transverse diameter of fetal heart can also accurately estimate the gestational age in FGR (r^2-0.4686and p value<0.0001). Among the conventional parameters, femur length was most accurate (r^2-0.3080, p value <0.0001). According to Witzani L, Brugger PC, Hörmann M, et al, Fetal kidney growth is constant, increases ≈1.7 mm fortnightly throughout pregnancy and unchanged by growth disorders [5]. Study by

Konje et al also found that growth restriction predominantly affects the antero posterior diameter and transverse diameter of fetal kidney but length remains unchanged [6]. Brennan S, Watson D, Rudd D, Schneider M, Kandasamy Y have done systematic analysis in FGR fetuses and found kidney length remained similar to appropriately grown fetuses whereas AP and TS dimensions were significantly decreased [7]. Hill et al in their study showed inconsistent effect of growth restriction and heart circumference [8]. In our study we had better correlation of fetal kidney length and gestational age than other ultrasonological parameters and that is also not affected by fetal growth restriction.

LIMITATION OF STUDY

The weakness of this study is that we did not get sufficient literature about kidney length and transverse diameter of the heart in FGR fetuses. Also, our sample size is only 70 as we get FGR pregnancies mostly unbooked in 3rd trimester with no first or second trimester ultrasound.

CONCLUSION- Most of the conventional ultrasonological parameter fails to estimate the gestational age in 3rd trimester for unbooked pregnant women with no previous early trimester USG. Fetal kidney length, femur length and transverse diameter of the heart can be used as a parameter to estimate the gestational age in growth restricted fetuses. Among these, fetal kidney length was the most accurate and reliable parameter to estimate the gestational age in FGR pregnancies.

Comment (usually conclusion is written in general,not according to study)

REFERENCES

1. *Nirmala Shivalingaiah , Sowmya K, Ananya R , Kanmani TR , Marimuthu P. Fetal kidney length as a parameter for determination of gestational age in pregnancy Int J Reprod Contracept Obstet Gynecol. 2014 Jun;3(2):424-427.*

2. *Kaul Indu,, Menia Vinay, K Anand Amandeep, Gupta Renu. Role of Fetal Kidney Length in Estimation of Gestational Age. JK SCIENCE, Vol. 14 No. 2, April - June 2012, 232-234.*

3. *Kirubamani,N N Hephzibah,m.r.Meenatshi,and B.Aupamma. "New Gestational Age Parameter-Transverse Diameter of Heart-TDH". Indian Journal of Science and Technology 6.7(2013):4990-4993.*

4. *Dacaj R, Izetbegovic S, Stojkanovic G, Gjocaj C. Hepato - Cephalic Index as a Predictor of Intrauterine Growth Restriction. Acta Inform Med. 2016 Feb;24(1):12-5. doi: 10.5455/aim.2016.24.12-15. Epub 2016 Feb 2. PMID: 27046941; PMCID: PMC4789733.*

5. *Witzani L, Brugger PC, Hörmann M, Kasprian G, Csapone-Balassy C, Prayer D. Normal renal development investigated with fetal MRI. Eur J Radiol. 2006; 5,7:294–302.*

6. *Konje JC, Okaro CI, Bell SC, de Chazal R, Taylor DJ. A cross-sectional study of changes in fetal renal size with gestation in appropriate- and small-for-gestational-age fetuses. Ultrasound Obstet Gynecol. 1997 Jul; 10(1):22-26.*

7. *Brennan S, Watson D, Rudd D, Schneider M, Kandasamy Y. Evaluation of fetal kidney growth using ultrasound: A systematic review. Eur J Radiol. 2017 Nov; 96:55-64.*

Hill LM, Guzick D, Peterson C, DiNofrio D, Maloney J, Nedzeksy P. Fetal heart circumference as a predictor of menstrual age in fetuses affected by disturbances in growth. Am J Obstet Gynecol. 1993 Aug; 169(2 Pt 1):347-51.

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Case Report

A CASE OF SCAR ECTOPIC PREGNANCY IN A TERTIARY CARE INSTITUTION

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ABSTRACT

Cesarean scar ectopic pregnancy has a high rate of morbidity with nonspecific signs and symptoms making identification difficult. As cesarean delivery numbers rise, a subsequent increase in scar ectopic pregnancies can be anticipated. The ability to accurately diagnose and treat this morbid condition is vital to the practice of any Obstetrician & G2naecologist.

KEY WORDS: Scar ectopic, abnormal implantation, uterine rupture, previous

INTRODUCTION

Caesarean scar ectopic pregnancy (CSP) is defined as a condition where the implantation occurs on muscle or fibrous tissue of previous caesarean scar. The incidence of CSP continues to rise with increasing caesarean section rates [1.] Incidence is 1 in 800 to 1 in 2500 of all ectopic pregnancies. It comprises of 6.1% of all ectopic pregnancy with a recurrence rate of approximately 5%. Mortality rate is about 1 in 500 cases.[2] Thus, there is an increased need of clinical suspicion for early diagnosis for prevention of catastrophic events.

CASE

A 24 years old housewife, resident of Kankinara, West Bengal, third gravida, with previous two LSCS, having a non-consanguineous marriage, belonging to Middle socio-economic class, attended ER on 31st August, 2022 with the chief complaint of bleeding per vagina for three days. She gave history of three months of amenorrhea. Bleeding was moderate in amount. It was not associated with pain in abdomen. There was no history of abortifacient intake.

Patient attended menarche at 14 years of age. She used to have regular menstrual cycle where bleeding lasting for 4-5 days every 28 days with mildly painful and moderate flow.

Her last menstrual period (LMP) was on 22nd May, 2022 and expected date of delivery was on 26th February, 2023. Her estimated gestational age by LMP was 14 weeks 2 days and by USG 13 weeks 2 days. She gave history of infrequent use of condom as contraception. Patient was married for 6 years. She was third gravida with two living issues delivered at term by **LSCS** with indication of Foetal Distress and Previous LSCS with Scar tenderness respectively. Last child birth was 2 years back.

She did not give history of Bilateral Tubal Ligation or any other surgery performed previously. There was no history of blood transfusion or drug allergies.

There was no significant medical history of diabetes mellitus, hypertension, bronchial asthma, thyroid disorders, tuberculosis, heart conditions in patient or in her Family.

GENERAL EXAMINATION

Patient was examined after obtaining proper informed consent. She was conscious, alert, cooperative, moderately built and well nourished. She had mild pallor but no icterus/ edema/ clubbing / cyanosis. She was afebrile with oral temperature 97F, Pulse rate 87 bpm, regular, normal volume, no radio-radial or radio-femoral delay. Blood pressure 110/70 mmHg in supine position.

SYSTEMIC EXAMINATION:

No abnormalities detected in Respiratory, Cardiovascular or central nervous system.

OBSTETRICAL EXAMINATION:

Abdomen was soft, non-tender.

On per vaginal examination, uterus was found to be of 12 weeks size approximately, anteverted, bilateral fornices were free, no cervical motion tenderness noted, cervical Os admitted tip of finger, mild bleeding was found to be present.

Patient came with USG Report dated 23rd August, 2022 suggestive of **missed abortion** and another USG dated 27th August, 2022 having **bulky uterus with huge amount of echogenic material seen in Endometrial cavity suggestive of incomplete abortion.**

In ER urine pregnancy test was done which was found to be positive. Patient was given Tb. misoprostol (200 mcg) Sublingual and per vaginal stat. Her USG (lower Abdomen and pelvis) was repeated in the institute on 01st September, 2022 in view of retained product of conception. USG Suggested **heterogeneous echogenic material in endometrial cavity 4.8*2.7cm. Uterus found to be bulky. Right and Left ovary normal, adnexa pouch of Douglas free.** She was planned for *Dilatation and Evacuation* in OT on very next day with 1 unit blood reserved. Serial dilatation had to be done and product of conception was removed. Post D&E, patient's vitals were stable. Before discharge, after evacuation, USG was repeated and report showed presence of Heterogeneous mass even after evacuation suggestive of incomplete procedure.

Keeping in mind the history of previous two LSCS, the Radiologist of the institution was consulted and in the presence of Obstetrician, USG was repeated which denoted presence of **5*4 cm heterogeneous mass, at the scar line, invading bladder anteriorly, having vascular supply thus taking colour on Doppler study.**

This radiologically confirmed the diagnosis of **SCAR ECTOPIC PREGNANCY** and immediately EXPLORATORY LAPAROTOMY was planned on that day itself. Two-unit Blood was arranged and two-unit blood was kept in reserve. High Risk party counselling was done. Patient was prepared for OT.

Exploratory Laparotomy was done with B/L Tubal ligation and Excision of Ectopic Product of Conception Under spinal anesthesia. Abdomen was opened layer by layer after vertical skin incision and peritoneum was reached. Uterus identified. Ectopic pregnancy noted in anterior uterine wall. A **gestational sac measuring 7*7cm** noted. Dense adhesions were noted with uterus and bladder which were carefully dissected by sharp dissection. Excision of ectopic product was done and homeostasis secured. Uterus closed in layers. Product of conception was sent for histopathological examination. peritoneal washing done and abdomen closed in layers. Meanwhile one unit blood was transfused to the patient. Operation went uneventful.

Post OT reports were as follows:

CBC		
Heamoglobin	13.9	gm/dl
WBC	7,600	mm3
Neutrophils	73	%
Lymphocyes	24	%
Monocytes	02	%
Basophils	01	%
Eosinophils	00	%
Platelets	2.0	L

Liver Function Test (LFT)		
SGPT	40	IU/L
SGOT	38	IU/L
ALP	140	IU/L
Bilirubin Total	0.7	mg/dl
Direct	0.4	mg/dl
Indirect	0.3	mg/dl

Renal Function Test (RFT)		
Blood urea	13	mg/dl
Creatinine	0.3	mg/dl

Serum Electrolytes		
Na+	138	Mmol/L
K++	4.0	Mmol/L

Table 1: post operation Blood reports of the patient

Patient was stable after OT. She was discharged after a few days of observation with advice of regular follow up.

DISCUSSION

As it is a rare diagnosis, most of the evidence for management comes from case reports and small case series. Recent research supports any method that removes the pregnancy and scar to reduce morbidity and improve fertility[3]. Surgical treatment or combined systemic and intra gestational Methotrexate both are successful in the management of cesarean delivery scar pregnancy. In scar ectopic pregnancy, gestational sac is not uterine cavity and chorionic villi implants on scar. Hence, trophoblastic tissue is unreachable to curette. So, dilatation and curettage have a doubtful role. Seow et al in their series of 12 cases of cesarean section ectopic pregnancy concluded that TVS or TAS guided methotrexate injection emerged as treatment of choice to terminate CS ectopic pregnancy. Regression of scar ectopic mass occurred between 2 months to 1 year. However, some of the researchers reported higher failure rates with methotraxate[4]. Although expectant management has been attempted in some cases, currently available data supports termination of such a pregnancy once the correct diagnosis has been made. A cesarean scar ectopic pregnancy complicates 1 in 2226 pregnancies [4]. As subsequent pregnancies may be complicated by uterine rupture, the uterine scar should be evaluated before as well as during these pregnancies. Cesarean scar ectopic pregnancies can have disastrous outcomes, including uterine rupture, massive hemorrhage and maternal death [5].

CONCLUSION

Cesarean Scar Ectopic pregnancy is rare type of ectopic pregnancy. This condition can be catastrophic if not manage on time, leading to significant morbidity and mortality. Early diagnosis by trans vaginal ultrasonography (TVS) and a high degree of clinical suspicion for probability of such scar ectopic pregnancy in previous uterine surgery patients may help in initiation and success of conservative treatment, prevention of complications and preservation of fertility.

REFERENCES

1. 1. *Roche C, McDonnell R, Tucker P, Jones K, Millward K, McElhinney B, Mehrotra C, Maurois P. Caesarean scar ectopic pregnancy: Evolution from medical to surgical management. Aust N Z J Obstetric Gynaecology. 2020 Dec;60(6):852-857. doi: 10.1111/ajo.13241. Epub 2020 Aug 20. PMID: 32820539.*

2. *Indian journal of Obstetrics and Gynaecology Research (Jan – March 2023)*

3. *Cesarean scar ectopic pregnancy: current management strategies. Glenn TL, Bembry J, Findley AD, Yaklic JL, Bhagavath B, Gagneux P, Lindheim SR. Obstet Gynecol Surv. 2018;73:293–302.*

4. *KM Seow LW Huang YH Lin MYS Lin YL Tsai JL Hwang Caesarean scar pregnancy: issues in management Ultrasound Obstet Gynecol200423324753*

5. *A Ash A Smith D Maxwell Caesarean scar pregnancy JOG: Intl J Obstet Gynaecol2007114253263*

Figure 1: USG plate suggestive of SCAR ECTOPIC PREGNANCY invading bladder anteriorly.

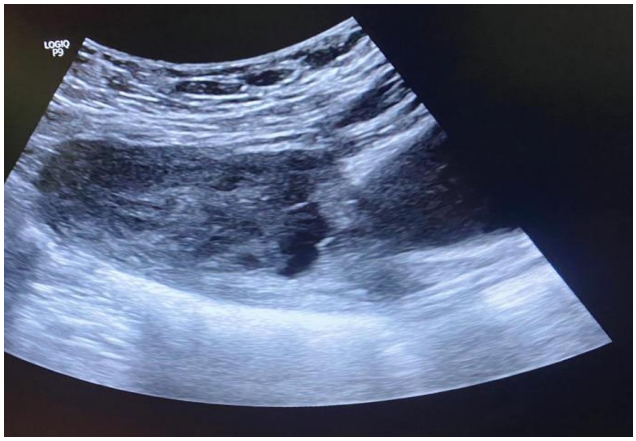


Figure 2: intraoperative finding showing Gestational sac at scar line

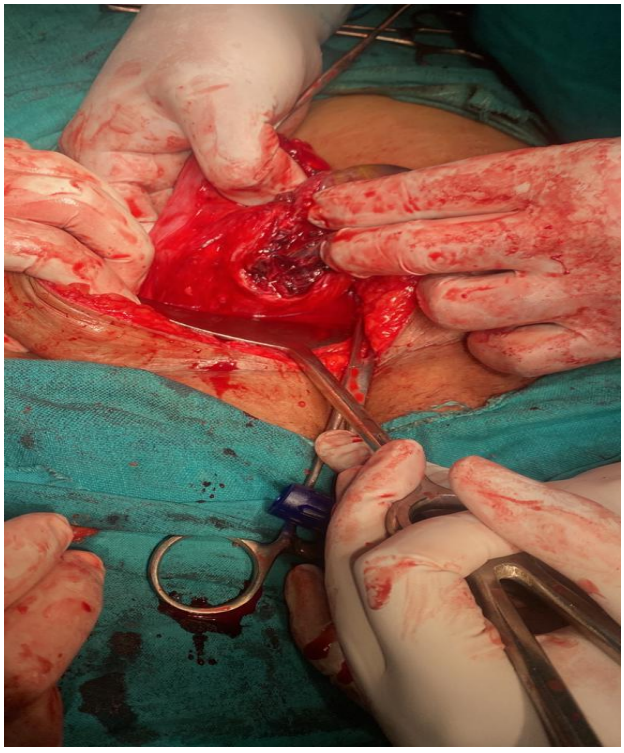
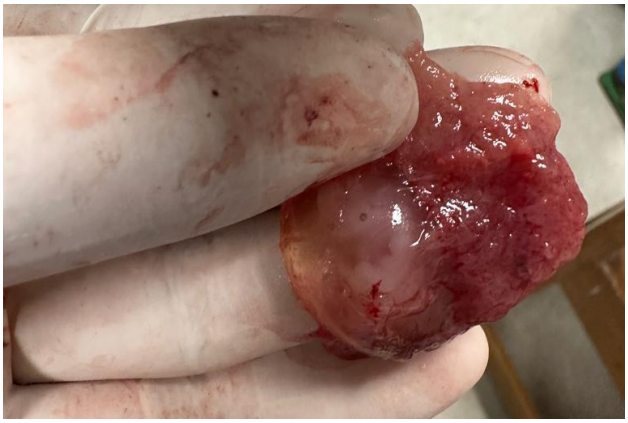


Figure 4: Embryo with gestational sac at scar line



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Case Report

PLACENTA ACCRETA AND ITS SURGICAL MANAGEMENT: A CASE REPORT

Siddhartha Dewasi[1]✉, Prithu Bandyopadhyay [2], Arijit Debnath[3], Suvasmita Saha [4], Ranita Roy Chowdhury [5]

INTRODUCTION

Placenta accreta is defined as abnormal trophoblast invasion of part or all of the placenta into the myometrium of the uterine wall[1]. Placenta accreta remains a major cause of severe and life-threatening post-partum haemorrhage, which often requires peripartum hysterectomy[2].

According to the WHO systematic analysis published in THE LANCET in 2014, post-partum haemorrhage, which accounts for 27.1% of all maternal deaths worldwide, is the leading cause of maternal deaths[3], and abnormally invasive placenta is the second most common cause of PPH (24.8%, with the leading cause being uterine atony, 38.3%)[4].

NOVELTY OF THE CASE

Though this case is not some kind of new or unknown case, but it comes under obstetric emergency and managing it was indeed very difficult. It was one of the most challenging cases I have faced in my PG life. I chose this case because I want to learn about and improve my management skills in such obstetric emergencies

CASE

Fulkumari Devi, 24 years old, 2nd gravida (G_2P_1) mother from Hazinagar, attended ER on 3/12/22 at 8 am with chief complaint of bleeding per vagina since early morning with lower abdominal pain in her 37th week of pregnancy. The bleeding was bright red colour and moderate in amount.

She was married for last 5 years and had a child delivered by LSCS 2 years ago. This time she had spontaneous conception pregnancy confirmed by
UPT at 8th week. It was a booked case of JNM with regular antenatal visit. Her LMP was 17/3/2022, and EDD was 24/12/22. Upon current admission, estimated gestational age by LMP was found to be 37 weeks, and calculating from 1st trimester USG, the gestational age was 38 weeks 1 day.

Her first trimester went uneventful. There was no history of excessive nausea or vomiting, nor any spotting per vagina.

During second trimester, she felt quickening at 5th month of gestational age. In this trimester also there was no history of bleeding P/V. Anomaly scan was not done. She took Inj. TT at 16th and 20th week of gestational age.

On her third trimester, she was diagnosed as a case of placenta previa at 30th week of gestational age. She was advised to take complete bed rest. During this period, she was admitted twice at 30th week and 35th week with complain of mild per vaginal bleeding and was discharged within 7days after conservative management.

Her menstruation began at the age of 14years. She used to have regular menstrual cycle where bleeding lasting for 4-5days every 28 days, moderate flow with dysmenorrhea.

Nothing significant was found in her medical and family history. Her surgical history was also not significant except 1 LSCS 2 years ago.

On general physical examination, patient was alert, conscious and cooperative. She was moderately built and nourished, her height was 156cm and weight was measured 68kg. On examination, she had mild pallor. No icterus, cyanosis, clubbing or bilateral pitting pedal edema was seen. Neck veins and glands were not enlarged. Her breasts were normal and thyroid was not enlarged.
Pulse rate was found to be 82 beats per minute, with regular rhythm and normal volume. Her blood pressure was 128/82 mm of Hg, oxygen saturation 99%, respiratory rate 14/min and temperature was 98.8^{o}F.

On neurological examination, no focal neurological deficit was found. On cardiovascular examination, S1 and S2 was audible, and no murmur was heard on auscultation. On auscultation of the lungs, vesicular breath sounds were heard on bilateral lung fields. Examination of GI system was within normal limit.

Coming to the obstetric examination, on inspection, her abdomen was ovoid in shape, linea nigra and striae gravidarum was present. A lower abdominal scar mark of previous LSCS was also observed. There was no venous prominence.

On palpation, her uterus was term size, non-tender on palpation, fetal movement present and FHR was found to be 136 beats per minute. Her liquor was adequate. During P/V examination, per speculum examination showed os 1 cm dilated with mild bleeding red in colour coming out from os. Digital per vaginal examination was not done, because PV examination is preferably not to be done in a known case of placenta previa, except inside an OT set-up.

DIAGNOSIS

Keeping in mind the history of previous LSCS 2 years back and reddish per vaginal bleeding, the radiologist of the institution was consulted in the presence of the obstetrician. USG was done which denoted that placenta anterior, low-lying, completely covering the internal os, implying placenta previa grade-III.

This radiologically confirmed the diagnosis of post-CS pregnancy with central placenta previa. Immediately Em LUCS was planned after USG.

Colour doppler was not advised along with USG, so placenta accreta cannot be diagnosed preoperatively. The diagnosis of placenta accreta was made based on OT findings, and later on confirmed by histopathological examination of the resected uterus following hysterectomy.

INVESTIGATIONS

Hb = 8.6; PCV-23.8%; RBS-108mg/dl

RFT: Urea-24mg/dl; Creatinine-0.5 mg/dl; Uric acid-34 mg/dl

LFT: Total Bilirubin-0.5 mg/dl, Direct Bilirubin-0.2 mg/dl, Indirect bilirubin-0.3 mg/dl.
SGOT- 19 mg/dl; SGPT- 18 mg/dl; GGT- 20
Total Protein- 7 g/dl, Albumin-5 g/dl,
Globulin- 1.2 g/dl

SEROLOGY:
HIV-N/R; HbsAg-Negative; VDRL-N/R

COAGULATION PROFILE:
BT -1'40"; CT- 3'15"; Platelet-2.25 lakh/mm^3
PT-11Sec; APTT-26sec; INR-1.2

USG (3/12/22):

Single live fetus of 38 weeks 4 days in cephalic presentation, longitudinal lie.
Placenta placed anteriorly, low lying, completely covering internal os, implying placenta previa grade-III of grade 2 maturity.
FHB- 155/min
Fetal weight-3500gm
Liquor- Adequate, AFI-12cm

OT PROCEDURE

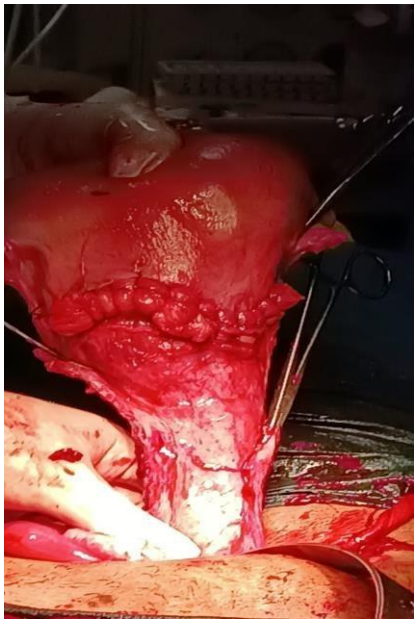
Preparation:

2-unit PRBC was arranged and 2-unit blood requisition was done. High risk party counselling was done.

Total abdominal hysterectomy consent was taken.

Procedure:

Em LSCS was done with bilateral internal iliac artery ligation followed by total obstetric hysterectomy under spinal anaesthesia.



STEPS OF OPERATION:

Spinal Anaesthesia given

⬇

ASS and ASD given

⬇

Patient placed in Supine Position

⬇

Lower transverse skin incision was made over skin

⬇

Abdomen opened in layers

⬇

Lower transverse incision was made over uterus

⬇

Baby delivered and cried at birth

⬇

Placenta removed manually

⬇

Some bits of placenta adhered with lower segment of uterus after manual removal of placenta.

⬇

Profuse bleeding from lower segment (approx. 1.5 lit)

⬇

uterus closed in two layers quickly

⬇

Bilateral internal iliac artery ligation done

⬇

Devascularization of uterus done and local haemostatic sutures given

⬇

Compression sutures given

⬇

Haemostasis could not be achieved

⬇

Total abdominal hysterectomy done

⬇

Vault closed

⬇

Haemostasis secured

⬇

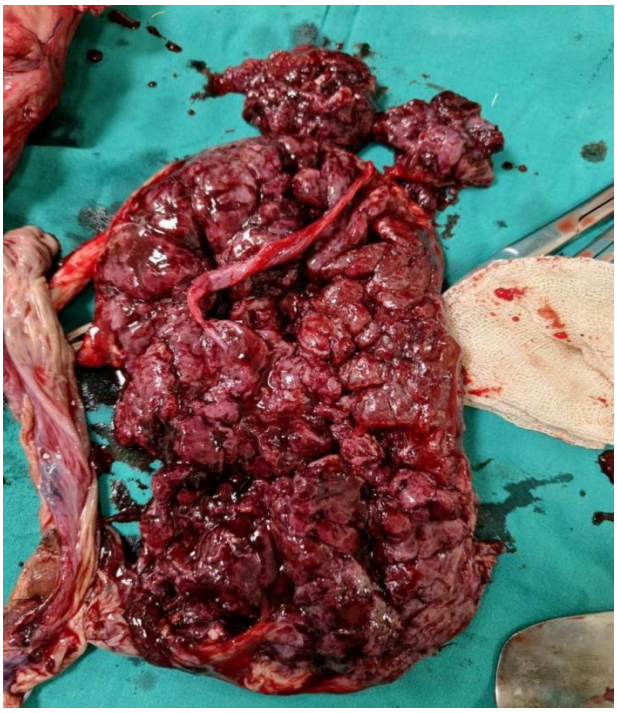
Peritoneal washing done and peritoneal suction drain given and placed in POD.

⬇

Specimen of uterus and placenta sent for HPE

⬇

Intra operatively 2unit of PRBC was transfused.



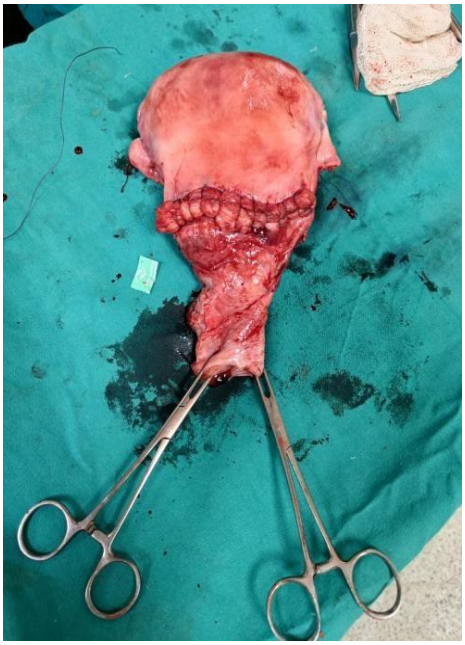
OT FINDINGS

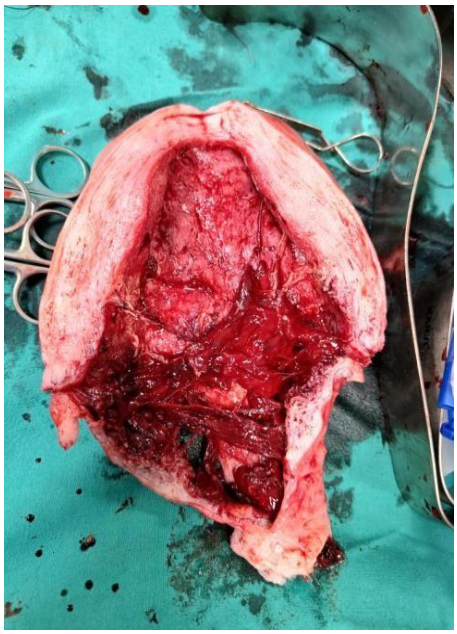
There was no adhesion seen between uterus and other adjacent structures. Bilateral ovaries were found to be normal.

There were engorged blood vessels near the lower uterine portion, signifying neo-vascularisation. Profuse bleeding from lower uterine segment was seen. Also, there were some

retained bits of placenta adhered to lower uterine segment.

Intraoperative blood loss was almost 1.2 litres.





POST-OPERATIVE FOLLOW-UP

IMMEDIATE POST-OPERATIVE EVENTS

- No per vaginal bleeding was noted after operation.
- Packed Red Blood Cells (PRBCs), Fresh Frozen Plasma (FFPs) and Platelets were transfused in 1:1:1 ratio.
- Vitals were stable, urine output was adequate and clear, and there were no signs of abdominal distension.

POST-OPERATIVE FOLLOW UP

- During post-operative period, initially she was advised NPM (nothing per mouth) and IV fluid (IVF RL: DNS 1:1 @ 6 hourly), with proper antibiotics (Ceftriaxone + Metronidazole) and other conservative therapies (PPI, anti-emetic, analgesic etc.)
- Her post-operative follow-up remained uneventful. Liquid diet was allowed 24hrs after the surgery, with IPS being clearly audible. Gradually IV fluid was omitted and patient was shifted to oral drugs after omitting injectables. After 48 hours of the surgery, patient was able to tolerate normal diet and passed urine and stool normally.
- On investigations, her haemoglobin level was found to be 10.2 g/dl, total count 10,400/mm^3 and platelet count was 2.26 lakh/mm^3 after transfusion of PRBC, FFP and platelet.
- PV examinations were performed on a regular basis to rule out any per vaginal bleeding that might arise suspicion of any retained products of conceptus. No such PV bleeding was found.
- Regular dressing was done and the wound remained healthy. The patient was discharged on day 14 and she was advised to maintain outdoor-basis follow-up, once per week for first 6 weeks.
- HISTOPATHOLOGY REPORT of the sent specimen was obtained from her during her first post-op OPD visit that showed evidence of placental invasion inside myometrium, signifying placenta accrete.

COUNSELLING

Patient was counselled about the hysterectomy and its consequences i.e., surgical menopause, and explained properly about the symptoms of surgical menopause such as hot flushes, dryness of vagina, mood swings etc.

DISCUSSION

Placenta accreta characterised by abnormally

implanted, invasive or adhered placenta. The major clinical problem is placental failure to separate normally from the myometrium after fetal delivery because of absence of decidua basalis.

If not managed on time, it can cause significant morbidity and mortality [4]. Preoperative assessment ideally begins once recognised antenatally. A major decision concerns the timing and ideal facility for delivery. After fetal delivery the extent of placental invasion is assess without attempts of manual placental removal.

It is evident that chance of bleeding can be reduced by bilateral internal iliac artery ligation before attempting hysterectomy [5].

REFERENCES

1. Usta IM, Hobeika EM, Musa AA, Gabriel GE, Nassar AH. Placenta previaaccreta: Risk factors and Complications. Am J Obstet Gynecol 2005; 193: 1045-9.

2. Shellhaas CS, Gilbert S, Landon MB, Varner MW, Leveno KJ, Hauth JC et al. The frequency and complication rates of hysterectomy accompanying caesarean delivery. Obstet Gynecol 2009; 114: 224-9.

3. Lale S, Doris C, Alison G, Ozge T, Ann-Beth M, Jane D et al. Global Causes of Maternal Death: A Who Systematic Analysis. The Lancet 2014; Vol. 2 Issue 6; E323-E333.

4. Edwards HM. Aetiology and Treatment of Severe Postpartum Haemorrhage. Dan Med J. 2018 Mar; 65(3): B5444. PMID: 29510809.

5. ***Ayman EN, Yossef H, Abdel Q et al. Therapeutic Effect of Internal Iliac Artery Ligation and Uterine Artery Ligation Techniques for Bleeding Control in Placenta Accreta Spectrum: A Meta-Analysis of 795 Patients. Front Surg. 2022; 9: 983297.***

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1. Frederick J, Fletcher H, Simeon D, Mullings A, Hardie M. Intramyometrial vasopressin as a haemostatic agent during myomectomy. *Br J Obstet Gynaecol* 1994; 101(5):435-7.

BOOK

2. Shaw RW, Soutter WP, Stanton SL (eds) *Gynaecology*, 3rd edn. Philadelphia: Churchill Livingstone, 2003.

CHAPTER IN A BOOK

3. Menefeee SA, Wall LL. Incontinence, prolapse, and disorders of the pelvic floor. In: Berek JS (eds) *Novak's Gynecology*, 13th edn. Philadelphia: Lippincott

Williams & Wilkins, 2002; p 645-710.

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