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"UNDERSTANDING THE RISK FACTORS FOR PRETERM LABOUR WITH SPECIAL REFERENCE TO DENTAL HYGIENE"

Obstetrics & Gynaecology

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ABSTRACT

India is among the top ten countries with maximum preterm births, a rate of 21% and of the 3.6 million preterm births.

Aim & Objectives:

To study the risk factors for preterm labour with special reference to dental hygiene and their preventive aspects.
To study the risk factors for preterm labour.
To understand the correlation of risk factors for preterm labour
To study dental hygiene with respect to gingival index, plaque index and oral hygiene test.

Materials & Methods

- A prospective case control study of preterm delivery after 24 weeks and before 37 completed weeks with equal number of term vaginal delivery or LSCS after 37 completed weeks within 24 hours as control.
- Informed written consent was taken.
- Detailed obstetric history, medical and family history in both the cases and controls is noted.
- The risk factors of preterm labour like genitourinary infection, cervical incompetence, maternal conditions like anaemia, heart disease, PIH, hypothyroidism, recent history of febrile illness and history of contact within 24 hours, previous history of abortion and preterm delivery will be assessed in both cases and control group.
- Cervical length on USG at 18-20 weeks of gestation is noted in both case and control group.
- Dental examination is done in both case and control group to diagnose periodontal infection in dental OPD post-delivery by calibrated periodontal Williams probe.

Results & Conclusion

- Genitourinary infection, dental infection and PROM are risk factors for preterm labour.
- Short cervix is one of the risk factor for preterm birth
- Previous preterm delivery is one of treatable cause to avoid preterm labour in next pregnancy
- Fair plaque index suggest periodontal infection which is statistically significant in case group compared to control group.
- Oral Hygiene Index Simplified represents past and present condition of dental health and is reliable index for periodontal infection.

KEYWORDS

Preterm labour, Periodontal infection, Risk factors

INTRODUCTION

The World Health Organisation (WHO) defines preterm birth as any birth after 24 weeks and before 37 completed weeks of gestation or fewer than 259 days from first day of last menstrual period (LMP). Preterm labour is considered to be established if regular uterine contractions can be documented at least 4 in 20 minutes or 8 in 60 minutes with progressive change in the cervical score in the form of effacement of 80% or more and cervical dilatation >1cm. If uterine contractions are perceived in the absence of cervical change, the condition is called Threatened Preterm Labour. [1]

Preterm delivery is categorized as follows:

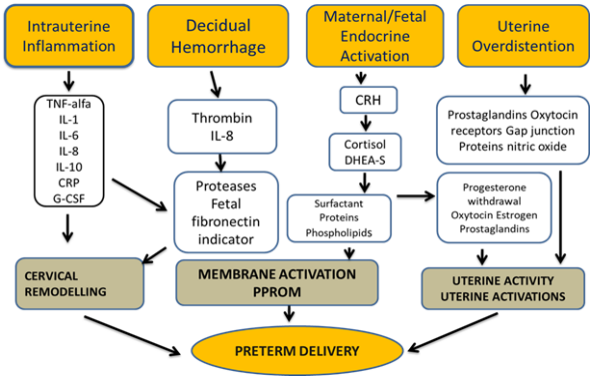
- Extremely preterm (24-28 weeks)
- Very preterm (28-34 weeks)
- Late preterm (34 to 37 weeks)

This is the most extensively used and accepted definition of preterm birth. [1]

According to World Health Organization (WHO) Globally 15 million babies are born prematurely every year and account for 40% of under-five deaths. More than one in ten babies are born preterm, affecting families all around the world, and over 1 million children die each year due to complications of preterm birth. [2] India is among the top ten countries with maximum preterm births, a rate of 21% and of the 3.6 million preterm births in India, 303,600 do not survive, in short, we have maximum deaths due to prematurity.[3] It is very imperative, hence in the Indian context to prevent preterm births and explore and implement preventive methodology. It is a major cause of perinatal morbidity and mortality. Predicting preterm delivery is a matter of considerable importance in Obstetrics.[4]

Various maternal demographic, behavioural, and clinical characteristics have been associated with preterm including maternal race/ethnicity, maternal age at either extreme, cigarette smoking, low pre-pregnancy weight, psychosocial stress, previous preterm, and maternal intrauterine infections.it predisposes the baby to long-term effects of visual and hearing impairment, chronic lung disease accelerated weight gain in adolescence, neurodevelopmental delay, psychiatric and behavioural problems. [5] An association of bacterial vaginosis (BV) and periodontitis with adverse pregnancy outcomes has been reported among women belonging to various ethnic groups and geographical locations. Despite substantial evidence suggesting the positive association of these infections with adverse pregnancy outcomes from elsewhere, routine screening of pregnant women for genital and periodontal infections is not a common practice in developing countries such as India. [6]

Etiopathogenesis Of Preterm Birth



We planned to do this study for early prediction of preterm labor and preventing preterm labor is utmost importance to decrease neonatal mortality. This study aims to know the highest prevalence of risk factors so that preventive measures can be taken accordingly. Periodontal infection can be one of commonest risk factor for preterm labour and this have not been screened previously, this study mainly focusses on dental examination and periodontal infection as a risk factor for preterm birth. If it is significant dental check-up may become part of routine antenatal check-up.

AIM

To study the risk factors for preterm labour with special reference to dental hygiene and their preventive aspects.

OBJECTIVES

To study the risk factors for preterm labour.
To understand the correlation of risk factors for preterm labour
To study dental hygiene with respect to gingival index, plaque index and oral hygiene test.

MATERIALAND METHODS

Study Design:
Type Of Study: Case control study
Study Centre:
Patients with preterm and full-term labour getting admitted in labour room of MGM Medical College and hospital, Aurangabad
Study Period: October2017 to October 2019.
Sample size: 120 cases and control each

Inclusion Criteria

- Case: Spontaneous established Preterm vaginal or LSCS delivery before 37 completed weeks.
- Control: Age matched full term vaginal or LSCS within 24 hours

Exclusion Criteria

- Multiple pregnancy

METHODS

- A case control study of preterm delivery after 24 weeks and before 37 completed weeks with equal number of term vaginal delivery or LSCS after 37 completed weeks within 24 hours as control.
- Informed written consent was taken from all the enrolled patients.
- Detailed obstetric history, medical and family history in both the cases and controls is noted.
- The risk factors of preterm labour like genitourinary infection, cervical incompetence, maternal conditions like anaemia, heart disease, PIH, hypothyroidism, recent history of febrile illness and history of contact within 24 hours, previous history of abortion and preterm delivery will be assessed in both cases and control group.

- Cervical length on USG at 18-20 weeks of gestation is noted in both case and control group.
- Dental examination is done in both case and control group to diagnose periodontal infection by gingival index, plaque index and simplified oral hygiene index in dental OPD post-delivery by calibrated periodontal Williams probe.
- Gingival index (GI) was developed solely for purpose of assessing the severity of gingivitis in selected index teeth.

16- Maxillary right first molar
12- Maxillary left lateral incisor
24- Maxillary left first premolar
36- Mandibular left first molar
32- Mandibular left first incisor
44- Mandibular right first premolar

Each of four surface gingival units is assessed according to criteria

0 – Absence of Inflammation
1 – Mild inflammation
2 – Moderate inflammation
3 – Severe inflammation

GI PER TOOTH = distal facial + facial margin+ mesial facial+ lingual surface
4
GI PER PERSON = GI OF 16+12+24+36+32+44
6

Interpretation of gingival index score

GI SCORE	CONDITION
0.1 – 1	Mild gingivitis
1.1 – 2	Moderate gingivitis
2.1 – 3	Severe gingivitis

- Plaque index (PI)assess the thickness of plaque at the gingival area.

In our study same selected tooth were examined for the plaque index
Each of four areas are scored according to the criteria
0 – No plaque
1 – A film of plaque adherent to free gingival margin only after using probe
2 – Moderate accumulation of plaque seen on naked eye
3 – Abundance of soft matter within gingival pocket

PI PER TOOTH = distal facial + facial margin+ mesial facial+ lingual
4
PI PER PERSON = PI OF 16+12+24+36+32+44
6

Interpretation of plaque index score

PI SCORE	CONDITION
0	Excellent
0.1-0.9	Good
1.0 -1.9	Fair
2 – 3	Poor

- Oral hygiene test simplified (OHI- S) is sum of debris index and calculus index simplified and selected tooth and their specific lingual or buccal surfaces are examined.
-

Debris index = 16 buccal +11 labial +26 buccal +36 lingual
calculus index +31 labial +46 lingual
6

Oral hygiene test simplified = Debris index + Calculus index

Each tooth are scored according to criteria

SCORE	DEBRIS INDEX	CALCULUS INDEX
0	No debris or stain	No calculus
1	Soft debris <1/3rd of tooth	Supra gingival calculus <1/3rd of tooth
2	Soft debris > 1/3rd of tooth	Supra gingival calculus 1/3rd -2/3rd of tooth
3	Soft debris >2/3rd of exposed tooth	Supra gingival calculus >2/3rd of tooth

Interpretation of OHI- S score

SCORE	CONDITION
0 -1.2	Good
1.3 – 3	Fair
3.1 - 6	Poor

- All the index were scored in all case and control group and statistical analysis is done using SPSS software and the test of significance applied is the Chi-square test. Values below 0.05 were considered to be statistically significant.

RESULTS AND OBSERVATIONS

Table 01: Demographic Comparison Of Case And Control Group

Age Group	Cases (N = 120)		Controls (N = 120)		χ2-Value	p - value
	Number	Percentage (%)	Number	Percentage (%)		
< 19 years	21	17.5	16	13.33	6.029	0.1101 NS
20 – 29 years	91	75.83	101	84.17		
30 – 34 years	07	5.83	01	0.83		
> 34 years	01	0.83	02	1.67		
RELIGION						
Hindu	92	76.67	101	84.17	2.143	0.1432 NS
Muslim	28	23.33	19	15.83		
GRAVIDA STATUS						

Primigravida	59	49.17	51	42.50	1.097	0.578 NS
Multigravida	51	42.50	57	47.50		
Grand Multigravida	10	8.33	12	10.00		
Total	120	100	120	100		

There is no statistical significance of demographic factors in case and control group.

Table 02: Distribution Of Patients According To Gestational Age

Gestational Age		Cases (N = 120)		Control (N = 120)	
		Number	Percentage (%)	Number	Percentage (%)
Group I	Extreme Preterm birth (24 -28 Weeks)	04	3.33	00	00
Group II	Very Preterm birth (28- 34weeks)	56	46.66	00	00
Group III	Late Preterm birth (34-37weeks)	60	50.00	00	00
Group IV	Full Term (38-40 weeks)	00	0.00	120	100
Total		120	100	120	100

Table 03: Association Of Various Infections In Case And Control Group

Maternal Infections		Cases (N = 120)		Control (N = 120)		χ2- Value / *Fisher Exact Test	p-value
		Number	Percentage (%)	Number	Percentage (%)		
Genitourinary infection	Yes	46	38.33	14	11.67	22.755	0.000 HS
	No	74	61.67	106	88.33		
Febrile illness in last 24 hours	Yes	06	5.00	02	1.67	2.162	0.281 NS
	No	114	95.00	118	98.33		
Dental infection	Yes	102	85.00	38	31.67	56.23	0.000 HS
	No	18	15.00	82	68.33		
PROM	Yes	33	27.50	10	8.33	15.660	0.000 HS
	No	87	72.50	110	91.67		

All infections are risk factors for preterm labour.

Genitourinary infection, dental infection and PROM are statistically significant risk factors for preterm labour.

Table 04: Association Of Medical Diseases In Case And Control Group

Maternal Effects		Cases (N = 120)		Control (N = 120)		χ2- Value / *Fisher Exact Test	p-value
		Number	Percentage (%)	Number	Percentage (%)		
Anemia	Yes	28	23.33	17	14.17	3.309	0.0688 NS
	No	92	76.67	103	85.83		
Heart Disease	Yes	01	0.83	00	00	1.004*	1.000 NS
	No	119	99.17	120	100		
PIH	Yes	06	5.00	02	1.67	2.162*	0.281 NS
	No	114	95.00	118	98.33		
GDM	Yes	00	00	00	00	-	-
	No	120	100	120	100		
Hypothyroidism	Yes	04	3.33	05	4.17	0.116*	1.000 NS
	No	116	96.67	115	95.83		

There is no statistical significant association of medical disease in case and control group in my study.

Table 05: Effect Of Short Cervix (< 2.5 Cm) In Case And Control Group.

Short cervix is one of the risk factor for preterm birth which is statistically significant between case and control group.

Short cervix	Cases (N = 120)		Control (N = 120)		χ2-Value	p-value
	Number	Percentage (%)	Number	Percentage (%)	6.142	0.013 S
Yes	12	10.00	03	2.50		
No	108	90.00	117	97.50		
Total	120	100	120	100		

Table 06: Correlation Of Past Obstetric History In Case And Control Group.

Table06-A

Past Obstetric History		Cases (N = 120)		Control (N = 120)		χ2-Value	p-value
		Number	Percentage (%)	Number	Percentage (%)		
Previous Abortion	Yes	19	15.83	17	14.17	0.131	0.857 NS
	No	101	84.17	103	85.83		

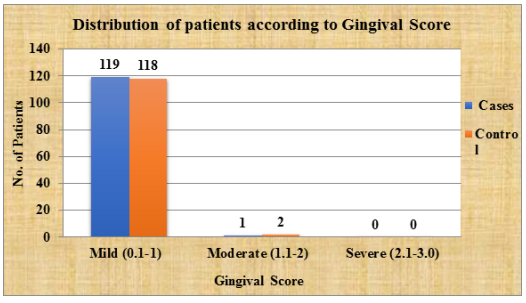
Table 06-B

Past Obstetric History		Cases (N = 120)		Control (N = 120)		χ2-Value	p-value
		Number	Percentage (%)	Number	Percentage (%)		
Previous Preterm Delivery	Yes	03	2.50	15	12.50	9.382	0.006 HS
	No	117	97.50	105	87.50		

There is no statistical significance of previous abortion history between case and control group.

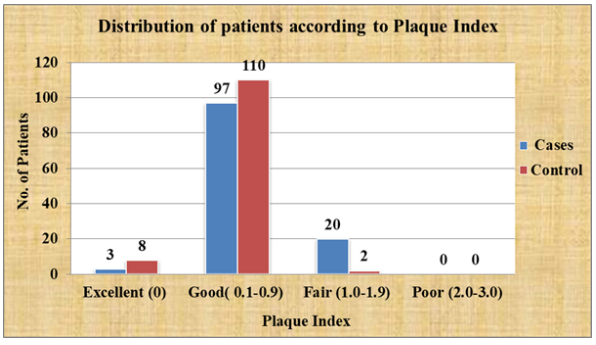
Previous preterm delivery is one of treatable cause to avoid preterm labour in next pregnancy

Table 07: Gingival Score In Case And Control Group



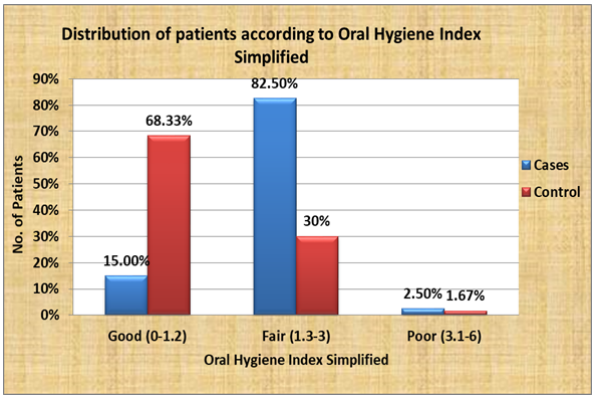
Mild form of gingival inflammation is common in pregnancy and there is no statistical significant association of gingival score between case and control group

Table 08: Plaque Index In Case And Control Group



Fair plaque index suggest periodontal infection which is statistically significant in case group compared to control group

Table 09: Oral Hygiene Index Simplified In Case And Control Group



Oral Hygiene Index Simplified represents past and present condition of dental health and is reliable index for periodontal infection.

It is compromised in 82.5% of cases which is statistically significant.

Table 10: Association Of Gestational Age With Neonatal Outcome.

Gestational age	Neonatal outcome					
	Mother		NICU		FSB	
	Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
Extreme preterm birth (<28 weeks)	00	0.00	02	1.67	02	1.67
Very Preterm birth (28- 34weeks)	21	17.5	34	28.33	01	0.83
Late Preterm birth (34-37weeks)	48	40.0	12	10.0	00	0.00
Full Term (38-40 weeks)	117	97.5	03	2.50	00	0.00

Though there is no difference in number of cases according to gestational age in group II and group III, there is remarkable difference in perinatal morbidity.

DISCUSSION

According to World Health Organization among Indian women the Preterm birth rate is 15%. [7] Preterm birth is a prevalent obstetric complication associated with significant neonatal mortality and morbidity worldwide. Addressing the burden of Preterm birth in developing countries is of public health importance due to its high (9 to 16%) prevalence.The present study was undertaken to elucidate the infectious and non-infectious risk factors for Preterm birth among Indian pregnant women seeking routine antenatal care at a tertiary care hospital. [8]

In our study, incidence of preterm birth is 17.5% which is slightly more in teenage pregnancy (<19 years) this result is similar to retrospective case control study in Thailand to determine association between teenage pregnancy and perinatal outcome. Though our findings showed a marginal protective association between maternal age > 34 years, this result is similar with other studies. [9].

In our study, 23.3% Muslims delivered preterm proving Muslims have more incidence of preterm birth which is usually multifactorial e.g more teenage pregnancy, multigravida etc which is similar to study by Rashed shah conducted in Bangladesh 2014 stating 95% of preterm birth were Muslims [10] In the present study, 49.17% were primigravida and 42.5% were multigravida in the case group. In the control group, there were 42.5% and 47.5% of Primigravida and multigravida respectively stating that Primigravida are at more risk for preterm birth. On evaluating the previous pregnancy outcome studies 19 of 33 women in the cases and 10 of 48 women in the control group respectively had previous abortions or a preterm birth or both (p value of 0.024). [37]

In our study, 50% and 0% of patients in cases and control group had delivered at a gestational age between 34-37 weeks. In addition, among the cases there were 1.67% women who had delivered extreme preterm (<28 weeks) and very preterm birth at 28 to 34 weeks accounted for more than 48% of all preterm births. Consistent with previous studies, [11] our study also found that the preterm birth rate was highest at 34 to 37 weeks' gestation in the population. The reason for the increase in preterm births at 34 to 37 weeks is not well understood. Several theories have been raised. [12] First, the increased use of reproductive technologies can result in an increase in multifetal pregnancies; advances in perinatal management, foetal health monitoring, medical intervention during pregnancy, and neonatal intensive care could also be contributing factors. [13] As a result, fetuses considered to be at risk for stillbirth may be identified earlier, resulting in more deliveries at 34 to 37 weeks of gestation. If we could support these pregnancies to continue an additional 1–2 weeks, that could lead to a substantial decrease in the preterm birth toll and burden of disease due to preterm. A small of number of behavioural (e,g. smoking cessation), clinical (e,g. progesterone supplementation) and health system interventions (e,g. reducing non-medically indicated labor induction or caesarean delivery) have been shown to reduce the preterm birth rate. [14]

In the present study, 38.33% and 11.67% of women in the case group and control group were had genitourinary tract infection during preterm labour respectively and was highly significant statistically. Gandhimadhi and Mythili in their study observed that 11% and 11.8% in the case and control groups had genitourinary tract infection. [15] In Jacob and Nath study, 6.47% and 4.7% in case and control groups had genital tract infection. Alves and Ribeiro in their study observed that 36.8% and 35% of women in group 1 and group 2 respectively had urinary tract infection. In all of the above studies, genitourinary tract infection was associated with preterm labour and low birth weight, though not statistically significant. [16]

Some studies have suggested an association between Genitourinary infection and Preterm birth due to released proteolytic enzymes and elevated pH, which can increase the risk of Genitourinary infection by 10-fold. [17] Similar results were reported by Discacciati et al and Verstraelen et al. [18,19] However, several tested bacterial species were found to be associated with Preterm birth namely *T. vaginalis*, *M. hominis*, and coryneform bacteria. Furthermore, similar results were previously reported. [20] In control group streptococci were suspected of being associated with Preterm birth; however, this was not in the cases in other study. [21]

Moreover, the infection of the genitourinary system is the most prevalent bacterial infections occurred during pregnancy. Similar to our results, Schieve *et al* has considered urinary system infection as a risk factor for premature birth. [22] Infection may raise release of inflammatory chemokine's and cytokines such as interleukins and tumour necrosis factors. Microbial Endotoxins and pro inflammatory

cytokines stimulate the production of prostaglandins (other inflammatory mediators) and matrix-degrading enzymes that finally result in stimulation of uterine contractions, preterm rupture of the membrane, and preterm birth. [23]

We also evaluate whether the preterm birth-Cardiovascular disease association persisted even in pregnancies not complicated by hypertensive disorders of pregnancy. Two studies have previously investigated the risk of CVD associated with preterm delivery in a pregnancy not complicated by preeclampsia. [24] Our results similarly show that preterm delivery remains associated with CVD even in pregnancies not complicated by hypertensive disease of pregnancy (HDP), suggesting that women with preterm pregnancies alone may benefit from additional prevention and screening along with women who experience both preterm and HDP. This is important as the majority in cases and control group were 99.17% and 100% respectively of preterm pregnancies in our study were not complicated by heart disease. [25]

In addition to the development of CVD risk factors emerging after a preterm birth, we also hypothesize that preterm birth and CVD are linked through subclinical shared risk factors that predate both preterm birth and CVD. The causes of preterm delivery generally depend on whether the premature delivery was spontaneous or medically indicated. [26] Spontaneous preterm deliveries typically result from intrauterine infection or inflammation, utero placental ischemia or haemorrhage, uterine over distension, stress or vascular disease, while medically indicated preterm deliveries are often caused by preeclampsia, intrauterine growth restriction, or other maternal factors including obesity and chronic hypertension. [27] Intrauterine infection, which triggers the release of inflammatory chemokines and cytokines, has been shown to cause approximately 30% of all preterm deliveries. [28] Inflammatory processes also contribute to the development of atherosclerosis, plaque rupture, and, ultimately, CVD. Inflammation, along with pre-pregnancy subclinical vascular disease and obesity, may underlie both preterm delivery and CVD. In support of this hypothesis, high C-reactive protein (CRP) levels in pregnancy, a marker of inflammation, are associated with spontaneous preterm delivery,and CRP is also a strong predictor of CVD risk. [29]

Hypertension increases resistance of uterine vessels and reduce uteroplacental fluid, which in turn causes intrauterine growth restriction. Moreover, the high rate of disorders like placenta abruption and pre-eclampsia and intrauterine growth restriction among women with hypertension may results in surgical operations and preterm birth. [30] Although the difference was not statistically significant; Renzo *et al* reported the likelihood of preterm birth to be 2.6 times greater among women with chronic hypertension. Various factors including fetal abnormalities, hypertension, pre-eclampsia, blood transfusion between twins, and chronic leakage of ammonite in ruptured areas of the membrane may lead to Oligohydramnios. [31]

Whereas, poor glycaemic control was associated with both spontan eous and induced preterm delivery. Our study did not allow the identification of the mechanisms of this association. However, it has been shown that hyperglycaemia directly induces endothelial dysfunction and increased oxidative stress leading to blunted nitric oxide–dependent vasodilatation. [32] During pregnancy, decreased synthesis of nitric oxide in the uterus is associated with initiation of labour in animals, and nitric oxide has been shown to be a uterine relaxant. To our knowledge, the effect of hyperglycaemia on nitric oxide synthesis and/or activity in the myometrium has not been studied. These observations suggest that strict glycaemic control might reduce the rate of preterm delivery and deserve further research. [33]

The Pedersen's hypothesis, formulated more than 50 years ago, suggested that fetal overgrowth was related to increased transplacental transfer of maternal glucose, stimulating the release of insulin by the fetal pancreatic beta cells. Because insulin is a major fetal growth factor, subsequent macrosomia occurs. [34] Infants from mothers with GDM at very preterm birth had a lower rate of intrauterine growth restriction.

The ponderal index in children from mothers with GDM was slightly higher than children of mothers without GDM. This can be explained by a small number of groups and short duration prenatal exposure of maternal hyperglycaemia in the case of early preterm birth. Children from mothers with pre-pregnancy diabetes have a risk throughout the pregnancy, but the children from mothers with GDM have especially risk only at the end of pregnancy, when the regulation of maternal metabolism could exceed its ability to synthesis of insulin. [35]

The mechanism that hypothyroidism can increase the risk of premature birth may be affected by different paths. One possible explanation is that inflammatory process with a change in the regulation of cytokine networks in the uterus and omission of the pair-control inflammatory processes can be linked with premature birth. Another suggestion is that thyroid hormones may influence foetal development directly through action on maternal and fetal metabolism. [36]

Cervical incompetence (CI) between case and control group was statistically significant. CI represents cervical failure that results in mid trimester pre-/peri-viable pregnancy loss or Spontaneous preterm birth. Its presumed cause is “weak” cervical tissue, intrinsic or acquired. Painless cervical dilation after the first trimester with subsequent expulsion of the pregnancy in the second trimester, typically before 24 weeks of gestation, without contractions or labor and in the absence of other clear pathology. [38] Some women who are ultimately diagnosed with CI initially present with pelvic pressure, cramping, and/or vaginal discharge whereas others have a “late presentation” characterized by advanced dilation and shortening in addition to spotting, prolapsed or ruptured membranes, and/or irregular, infrequent contractions that seem inconsistent with the cervical findings. [39]

Preterm birth requires cervical ripening, rupture of membranes, and uterine contractions. The assumed sequence, given appropriately timed activation and interaction of these pathways, is cervical remodelling/ripening → decidual activation → uterine contractions. In contrast, spontaneous preterm birth (sPTB) is thought to originate mostly from factors (e.g. bleeding, uterine overdistension, infection) that cause preterm premature rupture of membranes (PPROM; decidual activation) or preterm labor (PTL; uterine contractions), which then secondarily activate cervical remodelling/ripening. In other words, the presumed sequence is decidual activation or uterine contractions → cervical remodelling/ripening. Primary cervical dysfunction leading directly to sPTB is considered only a minor contributor. [40]

Whereas, in our study febrile illness is statistically not significant. Infection is a leading cause of preterm birth. A focus of many studies over the past decade has been to characterize microorganisms present in the uterine cavity and document any association with negative pregnancy outcome. A range of techniques have been used to achieve this, including microbiological culture and targeted polymerase chain reaction assays, and more recently, microbiome-level analyses involving either conserved, phylogenetically informative genes such as the bacterial 16S rRNA gene or whole shotgun metagenomics sequencing. [41] These studies have contributed vast amounts of data toward characterization of the uterine microbiome, specifically that present in the amniotic fluid, fetal membranes, and placenta. However, an overwhelming emphasis has been placed on the bacterial microbiome, with far less data produced on the viral and fungal/yeast microbiomes. With numerous studies now referring to Preterm birth as a polymicrobial condition, there is the need to investigate the role of viruses and fungi/yeasts in more detail and in particular, look for associations between colonization with these microorganisms and bacteria in the same samples. [42]

Although the major pathway by which microorganisms are believed to colonize the uterine cavity is vertical ascension from the vagina, numerous studies are now emerging suggesting haematogenous transfer of oral microbiota to the uterine cavity. [43] Evidence of this has been produced in mouse models and although DNA-based evidence in humans appears convincing in some aspects, use of methodologies that only detect viable cells as opposed to lysed cells and extracellular DNA are needed to clarify this. Such techniques as RNA analyses and viability polymerase chain reaction are likely to play key roles in the clinical translation of future microbiome-based data, particularly in confined environments such as the uterus, as detection of viable cells plays a key role in diagnosis and treatment of infection. [44]

In our study observed that 15.8% of patients in the case group had previous abortion and 14.7% of the patients in the control group had previous abortion which was not statistically significant. Moreover, previous preterm delivery in both case and control were statistically

significantly associated with the incidence of preterm birth.

12.5% in our study had previous preterm delivery but with regular antenatal care and treatment they delivered full term hence previous preterm delivery is treatable cause of preterm labour. Many other studies in the literature had considered prior abortions and previous preterm births as separate parameters. Gandhimadhi and Mythili in their study observed that 22.9% and 3.9% in the cases and controls respectively had previous abortions.[45] Among the 22.9% of patients in cases, 20.2% and 2.7% had spontaneous and induced abortions respectively. All the 3.9% of patients with a prior history of abortion in the control group had an induced abortion. Alves and Ribeiro also observed that 15.1% and 17.5% of women in case and control groups respectively had previous spontaneous abortions. [46] Incidence of spontaneous abortions in the case group was significant in all the above-mentioned studies.

The association between induced abortions and the length of gestation of a subsequent pregnancy was also statistically not significant. As with miscarriages the risk of having a preterm baby is increased with the history of such abortions. In Greece induced abortions form a specific social, demographic and medical problem. It has been estimated that every year twice as many abortions are performed than babies are born. [47] In the literature there is no clear consensus as to whether abortions do or do not affect the length of gestation of a subsequent pregnancy; the published results varied considerably between countries. A number of studies have indicated a substantially greater incidence of preterm births in subsequent pregnancies when mothers had had induced abortions and/or miscarriages. [48] Other investigations have found different associations in subsequent studies of the same population. Schoenbaum reported that the risk increased when the mother had had one spontaneous abortion while he found no increase in the risk after one induced abortion. Another large group of investigators had reported that there was no association between either factor. [49]

In our study found that gingival score was not associated with cases than controls in mild condition (99.17% vs 98.33%). Alves and Ribeiro in their study found that the incidence of periodontitis was more associated with cases than controls (84.2% vs 37.5%) and this was statistically significant with a p value 0.00114. [50]The frequent gingival inflammation of women presenting periodontal diseases especially the pregnancy associated gingivitis, facilitates bacteraemia process. The proposed link to preterm labor involves the descent of microorganisms from the oral cavity and subsequent colonization of the fetal membranes and endometrium. Once bacteria colonize these areas, they release lipopolysaccharides (endotoxins), and trigger systemic inflammation. Inflammatory mediators, such as interleukin-1, interleukin-6, tumor necrosis factor (TNF), and prostaglandin E2 (PGE2), are released and induce uterine contractions, mediate cervical thinning and dilation, and incite premature labor. [51]

Mild gingival inflammation is commonly seen in pregnancy due to increased circulating levels of progesterone in turn cause dilation of gingival capillaries, permeability, and gingival exudates that may explain the redness and increased bleeding tendency during pregnancy. And the physiological female sex hormones during pregnancy also influence the gingival tissues. [58] This is in contradictory with Zadeh-Modarres et al, indicating that the control group had a better periodontal condition than the case group. [55]

In the present study, the plaque indices for the case and control groups were 80.83% and 91.67 respectively. Comparing the two groups, the mean plaque index of the case group was significantly more than the control group. Mannem and Chava in their study observed that the mean plaque index for the case and control groups were 1.21±0.56 and 0.63±0.31 respectively. [52] The mean plaque index is higher in case group and this was also statistically significant (p value < 0.0001). It can be hypothesized that cytokines produced in periodontal tissues promote inflammation in maternal-fetal unit. Clinically, high-gingival crevicular fluid levels of PGE-2, IL-1β, or IL-6 have been associated with their elevated levels in amniotic fluid. The inflammatory response appears to be the privileged pathway of the pathogenic periodontal disease influence on pregnancy, as suggested for other major systemic diseases, including cardiovascular diseases or diabetes. [53] In the last two decades, many studies have examined the relationship between periodontitis and Preterm birth. Periodontitis may be a risk factor for preterm birth due to the presence in the bloodstream of bacteria and proinflammatory cytokines during infection that can affect distant organs. This could be because; during pregnancy, the oral microflora uses the progesterone and oestrogen hormones as vitamin k growth factors and they form the plaque on the gingival and tooth surfaces. [54]

The oral hygiene index of fair (1.3-3) in cases was 82.5%, and 30% among control group showed statistically highly significant (<0.001) difference as compared to cases group. This is in agreement with Zadeh-Modarres *et al*. indicating that the control group had a good periodontal condition than the case group. [55]The increased circulating levels of progesterone in turn cause dilation of gingival capillaries, permeability, and gingival exudates that may explain the redness and increased bleeding tendency during pregnancy. [56]

Importantly, in the present study these finding support the hypotheses of Offenbacher et al., [10] that gram-negative anaerobic periopathogens, their associated endotoxins, and pro- inflammatory mediators can have possible adverse effects on the developing fetus. Moreover, periodontal infections may lead to excessive production of the pro-inflammatory cytokines and prostaglandins, all of which are established biochemical mediators of parturition. However; the observation of the elevated IL-6 levels in the present study was a consistent and reproducible finding in those subjects who experienced a preterm pregnancy. [57]

This could be due to one of the two mechanisms. First, women with periodontal disease may experience more frequent and severe bacteraemia than periodontal healthy women. As a result, the uterine cavity may become exposed to or colonized by periodontal bacteria or their by-products (e.g., lipopolysaccharides). Once they reach the maternal-fetal unit, oral bacteria may elicit an inflammatory cascade that leads to preterm labour. A second putative mechanism does not require oral bacteria to colonize the uterine cavity. Rather, cytokines generated within the diseased periodontal tissue may enter the systemic circulation and precipitate a similar cascade, again leading to spontaneous preterm labour and birth. [59]

There is highly association between the periodontal diseases and the preterm birth explained by the direct and/or indirect effect of periodontopathogens on the developing fetus. In addition, the biologic mechanism initiating by the Gram-negative bacterial endotoxins present in the periodontal diseases. These gram-negative endotoxins can stimulate the production of the pro-inflammatory cytokines and prostaglandin. [60] Some cytokines such as IL-1 ß, IL-6, and TNF-α, as well as prostaglandins in appropriate quantities, are able to stimulate labour. However, the presence of Gram negative bacteria and the elevated IL-6 levels in the preterm birth group compared to the normal pregnant in the present study support this biologic mechanism. Moreover, bacterial identification offers an inexpensive and fast lab technique for predicting preterm birth. [61]

In the present study, the birth weight distribution 25.83% and 15.83 in the case and control groups respectively with 2-2.5 kg. Menon R. in their study observed that the mean birth weight was 2.01±0.36kg and 2.87±0.32kg in the case and control groups respectively. [62] Dasanayake in their study observed that the mean birth weight was 1999.7±303.0 g and 2785.6±321.6g in the case and control groups respectively. Comparing cases and controls, i.e. among women with either preterm labour, periodontitis was more significantly associated with cases and this was statistically significant. [63]

In our study, 28.3% were admitted in NICU in very preterm birth (28-34 weeks) compared to only 10% required NICU care in late preterm birth (34-37 weeks) which is similar to study by nandini kupuswamy in Tamil Nadu

One theory linking periodontitis to pregnancy outcomes posits that oral bacteria seed the placenta, membranes, or amniotic fluid through blood-borne routes, eliciting an inflammatory cascade that precipitates preterm labour. [64] We did not assess bacteraemia, but recent reports cast doubt on this theory. For example, although one report showed that periodontal disease was more prevalent in mothers who delivered preterm than in those who delivered full term, periodontal pathogens were detected in placentas of only 2 of 59 mothers who delivered preterm and of only 3 of 44 mothers delivering full term. [65] Another study failed to detect periodontal bacteria in the amniotic fluid of women with periodontitis who delivered preterm, even though these microorganisms were frequently found in dental plaque. Moreover, the

presence of *Fusobacterium nucleatum* in dental plaque and vaginal-swab samples was not associated with the presence of the bacteria in amniotic fluid. [66]

CONCLUSIONS

The etiology of preterm birth and low birth weight are multifactorial. Periodontitis is one of the risk factors for preterm labour and low birth weight. In the present study, periodontal factors like Plaque Index (PI), Gingival Index, oral hygiene index were analysed.

We observed the following conditions in patients who delivered prematurely

- Plaque index was significantly more in case group as compared to control group.
- Oral hygiene index was also significantly more in case group.
- Evidence of periodontitis was seen among more number of cases and was significantly associated with preterm labour. There was a significant association between moderate periodontitis and preterm labour.

Periodontitis is not a significant independent risk factor but with higher prevalence rate and obstetric factors contributes a major risk for preterm and/or low birth weight babies. It would be more appropriate to carry out future longitudinal studies to clarify the issue.

On the basis of these findings, periodontal infections in pregnant women can be viewed as a potential obstetric risk factor. The fact being periodontal infections are both preventable and readily treated, this study findings provide opportunities for intervention strategies to reduce the incidence of preterm low birth weight

Based on the results obtained in this study, it can be concluded that:

- Genitourinary infection is significant risk factor of preterm birth.
- Though the obstetric maternal risk factors were not significant, cervical incompetence is major contributing factor for preterm birth.
- The prevalence of periodontal disease among the preterm birth pregnant patient's samples in Aurangabad is high.
- There was a correlation between maternal periodontal disease and preterm birth among Aurangabad people, suggesting that periodontitis may be regarded as a true risk factor for the preterm birth.
- Dental health proves to be significant risk factor for preterm labour, routine dental check-up and screening of periodontal infection can be part of routine antenatal care to diagnose periodontal infection in early trimester so that timely interventions can be done to prevent its complications.

SUMMARY

- Incidence of preterm birth is slightly more in teenage pregnancy (<19 years) and age > 34 years gives marginal protection to preterm birth.
- Preterm delivery is more common in Muslims and Primigravida
- Preterm labour have multiple risk factors, genitourinary infection is more significant in case group and associated with preterm birth.
- There is no correlation between anaemia, PIH, hypothyroidism and heart disease and spontaneous preterm labour in my study.
- Cervical incompetence is the major contributing risk factor for preterm labour stating the importance of digital examination of cervix and cervical length on USG in 2nd trimester.
- Previous preterm delivery is treatable cause of preterm labour and there was no association between preterm labour and previous abortion in my study.
- Premature rupture of membrane (PROM) affects the perinatal outcome and is the one of major contributing factor to preterm labour.
- Plaque index measure the plaque on gingival surface of tooth and vertical deep pockets and fair plaque index suggest periodontal infection which is significant in case group and is detectable and curable cause of preterm labour.
- Oral hygiene test simplified is the sum of debris index and calculus index which measures the debris and calculus indicating dental health and it is reliable index for periodontal infection leading to majority of patients to preterm labour.
- Neonatal outcome and requirement of NICU care is significantly affected whether patient is very preterm <34 weeks or late preterm 34-37 weeks.

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