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Original Research Article

Clinical and subclinical chorioamnionitis in preterm births and late miscarriages: a prospective cohort study

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ABSTRACT

Background: Preterm birth is the leading cause of neonatal mortality worldwide and is associated with long-term neurodevelopmental morbidity. Nearly 40-50% of preterm births and up to two-thirds of late miscarriages are infection-related. Chorioamnionitis, a common intrauterine infection, significantly contributes to early-onset neonatal sepsis and maternal complications. Clinical chorioamnionitis is diagnosed using Gibb's criteria, whereas subclinical chorioamnionitis is confirmed only on histopathology and is more prevalent. This study aimed to determine the incidence of clinical and subclinical chorioamnionitis in spontaneous preterm deliveries, preterm premature rupture of membranes (PPROM) and late miscarriages.

Methods: This prospective cohort study was conducted over one year at a tertiary care center. One hundred antenatal women between 16 and 36+6 weeks with preterm labour, PPRM, or late miscarriage were enrolled. Clinical features, laboratory parameters like white blood cell count (WBC), C-reactive protein (CRP), high vaginal swab, urine culture, and neonatal outcomes were recorded. Placental tissues were examined for histopathology, and neonatal sepsis, if present, was documented.

Results: Fifty seven percent had PPRM, 42% spontaneous preterm labour, and 1% late miscarriage. Elevated WBC (>15,000) was seen in 38%, and 52% had CRP levels twice normal. Positive high vaginal swabs were noted in 39%. 10% had positive urine culture. Incidence of clinical, microbiological, and histopathological chorioamnionitis was 7%, 11%, and 34%, respectively. Four cases met criteria for triple I, and six neonates developed early-onset sepsis.

Conclusions: Clinical criteria showed high specificity but low sensitivity, missing 79.4% of histological cases. WBC and CRP had moderate predictive value. Early diagnosis remains crucial to balance prematurity risks against infection-related morbidity. Better diagnostic tools are needed to identify underlying intrauterine inflammation.

Keywords: Clinical chorioamnionitis, Early onset neonatal sepsis, Preterm births, Subclinical chorioamnionitis

INTRODUCTION

Preterm labour is defined as the onset of labour after viability and before 37 completed weeks of gestation. As per Federation of Obstetric and Gynecological Societies of India (FOGSI) guidelines, viability is considered as 24 weeks or a fetal weight of 500 gm. Globally, preterm birth affects 4-18% of pregnancies, with higher rates in developing countries. Prematurity remains a major cause of neonatal mortality and long-term morbidity.

Approximately 40-45% of preterm births are spontaneous, while 30-35% occur following preterm premature rupture of membranes (PPROM), defined as rupture of membranes before 37 weeks and prior to labour onset.

Late miscarriage, typically defined as pregnancy loss after 14 weeks, is increasingly linked to intrauterine infection, particularly beyond 16 weeks.¹ Its incidence between 16 and 24 weeks is around 1-2%. Causes include infections, chromosomal abnormalities, congenital anomalies,

cervical insufficiency, uterine anomalies, immunological factors, and maternal chronic illness. Nearly half of preterm births and late miscarriages are associated with infection.

Chorioamnionitis refers to inflammation and/or infection of intrauterine structures, including the chorion, amnion, placenta, and umbilical cord. Acute chorioamnionitis is characterised by neutrophilic infiltration of the chorion and amnion; involvement of the villous tree is termed acute villitis, and inflammation of the umbilical cord is called funisitis. It is seen in nearly one-third of preterm labour cases. The most common mechanism is ascending infection from the lower genital tract, though hematogenous spread (e.g., *Listeria monocytogenes*), iatrogenic introduction during procedures, and retrograde seeding may also occur. Frequently isolated organisms include *Ureaplasma urealyticum*, *Mycoplasma hominis*, *Group B streptococci*, *Escherichia coli*, anaerobic vaginal flora (e.g., *Bacteroides spp.*, *Gardnerella sp.*), *Pseudomonas*, *Peptostreptococcus spp.*, *Enterococci*, *Staphylococcus*, *Klebsiella*, *Fusobacterium spp.*, and others.²

Major risk factors include prolonged rupture of membranes (>12-18 hours), prolonged labour, nulliparity, repeated vaginal examinations, fetal scalp electrode use, GBS colonization, urinary tract infections, bacterial vaginosis, tobacco or alcohol use, and immunocompromised states. PPROM and preterm labour are both risk factors for and potential consequences of subclinical infection.

Chorioamnionitis significantly increases maternal and neonatal morbidity. Maternal complications include sepsis, dysfunctional labour, operative delivery, postpartum haemorrhage, and endometritis. Neonatal complications include respiratory distress syndrome, pneumonia, meningitis, early onset neonatal sepsis (EONS), intraventricular haemorrhage, bronchopulmonary dysplasia, periventricular leukomalacia, cerebral palsy, and fetal inflammatory response syndrome (FIRS).

EONS occurs within 48-72 hours of life and commonly results from ascending maternal infection. Pathogens include GBS, *E. coli*, *Coagulase-Negative Staphylococcus*, *Haemophilus influenzae*, and *Listeria monocytogenes*. Group B streptococci (GBS) or *Streptococcus agalactiae* is one of the major causes of EOS (early onset sepsis) in developed countries.³ Risk factors include maternal chorioamnionitis, GBS colonization, preterm birth, and prolonged rupture of membranes.

Timely diagnosis of chorioamnionitis is critical. Intrapartum antibiotic therapy can reduce neonatal sepsis by up to 80% and improve maternal outcomes.⁴ Clinical chorioamnionitis is traditionally diagnosed using Gibb's criteria: maternal fever ($\geq 38^{\circ}\text{C}$) plus at least two of the

following-maternal tachycardia ($>100/\text{minute}$), fetal tachycardia ($>160/\text{minute}$), leukocytosis ($>15,000/\text{mm}^3$), uterine tenderness, or foul-smelling amniotic fluid.⁵

Histologic (subclinical) chorioamnionitis is diagnosed postnatally by placental examination and may occur without clinical signs. It is characterized by maternal and/or fetal inflammatory response and is more common at lower gestational ages, with a reported incidence of up to 25-70%. Recognizing the spectrum of disease, experts have proposed the term "intraamniotic infection and inflammation" (triple I), categorizing cases as isolated maternal fever, suspected intraamniotic infection, or confirmed intraamniotic infection.⁶

METHODS

This prospective cohort study was conducted over a period of one year from September 2023 to September 2024 in the departments of high-risk obstetrics and perinatology/obstetrics and gynecology at Kimshealth, Trivandrum, a tertiary care referral centre. All antenatal women between 16 and 36+6/7 weeks of gestation admitted with preterm premature rupture of membranes (PPROM), spontaneous preterm delivery, or second-trimester miscarriage were eligible for inclusion. Women with iatrogenic preterm delivery for maternal or fetal indications, second-trimester medical termination of pregnancy, major congenital fetal anomalies, or those who did not provide informed consent were excluded. Fetal viability was considered at 23 weeks of gestation as per institutional protocol. The required sample size was calculated using the formula $n = Z^2PQ/d^2$, taking $Z=1.96$ for a 95% confidence interval, $P=52.9\%$ (prevalence of chorioamnionitis reported by Algerian et al), $q = 1 - P$, and allowable error (d) of 10%, yielding a minimum sample size of 96.⁷

After obtaining written informed consent, detailed history and clinical examination were performed. Patients were evaluated for features suggestive of chorioamnionitis, including maternal fever, uterine tenderness, purulent or foul-smelling cervical discharge, maternal tachycardia, and fetal tachycardia. At admission, a high vaginal swab, urine culture and sensitivity, total leukocyte count, and C-reactive protein were obtained. Ultrasound and cardiotocography findings were reviewed and documented in a structured proforma. Participants were followed longitudinally until delivery for the development of clinical chorioamnionitis, defined according to Gibb's criteria. After delivery, placental tissue and membrane samples were sent for culture, and placenta, membranes, and umbilical cord were examined for histopathological evidence of chorioamnionitis. Subclinical chorioamnionitis was defined as histological chorioamnionitis in the absence of clinical features. Neonates were monitored for early-onset neonatal sepsis, and in suspected cases, blood and urine cultures were recorded.

RESULTS

A total of 100 women fulfilling the inclusion criteria were enrolled in the study.

The mean maternal age was 30.3±4.1 years (range: 21-45 years). The majority of women (43%) were between 30 and 34 years of age. Women aged 35 years and above constituted 15% of the study population, of whom 14% belonged to the advanced maternal age group (≥35 years) and 1% to the very advanced maternal age group (≥45 years) (Table 1).

Table 1: Table showing demographic data of the study participants.

Variables	Frequency	Percentage
Age (years)		
21-24	7	7.0
25-29	35	35.0
30-34	43	43.0
35-39	12	12.0
40-44	2	2.0
45-50	1	1.0
Mean±SD	30.3±4.1	
BMI		
<18.5	1	1.0
18.5-24.9	36	36.0
25.0-29.9	40	40.0
30.0-34.9	16	16.0
35.0-39.9	5	5.0
≥40.0	2	2.0
Obstetric score		
Primigravida	62	62.0
Multigravida	38	38.0
Gestational age at admission		
16 to 22+6 weeks	1	1.0
23 to 23+6 weeks	4	4.0
24 to 25+6 weeks	8	8.0
26 to 27+6 weeks	7	7.0
28 to 31+6 weeks	16	16.0
32 to 33+6 weeks	18	18.0
34 to 36+6 weeks	46	46.0

With respect to indication for admission, 57% of women were admitted with preterm premature rupture of membranes (PPROM), 42% with spontaneous preterm labour, and 1% with second-trimester miscarriage.

The mean gestational age at admission was 32±4.1 weeks, with a range from 21 weeks 3 days to 36 weeks 6 days. Nearly half of the women (46%) were admitted between 34 and 36+6 weeks of gestation (late preterm). Admissions between 32 and 33+6 (moderate preterm) weeks accounted for 18%, while 16% were between 28 and 31+6 weeks (very preterm). Extreme preterm admissions (<28 weeks) constituted 20% of the cohort, with only 1% admitted before 23 weeks of gestation.

As per guidelines and hospital policy, all these women were given conservative management, considering the gestational age was below 37 weeks. Antenatal corticosteroids (dexamethasone 6 mg intramuscularly, 4 doses 12 hours apart) were started for all pregnancies between 23 weeks and 34 weeks. Magnesium sulphate (24 hours) was started for fetal neuroprotection for pregnancies up to 30 weeks. Antibiotics were started for all women with PPROM (azithromycin + ampicillin regimen). The 42 women admitted with preterm labour progressed spontaneously and delivered within a maximum duration of 24 hours. 16 of them delivered vaginally, while 26 had a caesarean section. In PPROM, conservative management was abandoned when the woman progressed into labour spontaneously or when there were pathological cardiotocography results/severe oligamnios/features of clinical chorioamnionitis. Out of the 57 women admitted with PPROM, 30 underwent caesarean section and 27 delivered vaginally.

The mean interval between admission and delivery in days among study participants was 2.8 days±5.3. 74 % of them were delivered within a period of 24 hours. 11 women delivered between 24 and 48 hours, and 5 delivered between the 3rd and 5th day of admission. 3 women continued beyond 3 weeks. The maximum duration of conservative management was 34 days.

24% women had negative CRP value (<5). 9% had values above 40. The mean CRP value was 15.7±18.3. CRP values ranged from 0.6 to 83.0. 62% women had a total white blood cell count more than 15000. The mean count was 14122±4106.3 with a range of 5500 to 25300.

Table 2: Distribution of the sample according to high vaginal swab.

HVS	Count	Percent
Negative	61	61.0
E. coli	9	9.0
Candida	13	13.0
Enterococcus fecalis	8	8.0
GBS	5	5.0
Klebsiella	3	3.0
Acinetobacter baumannii	1	1.0

Table 2 shows the results of high vaginal swab cultures of these women. 61 % of these women had negative high vaginal swabs. Out of the 39% women with positive growth, 13 % had candida, 9% had *E. coli*, and 8% had *Enterococcus fecalis*. 5% women were GBS positive. Urine culture showed no growth in 86% women. 4% each had *Klebsiella* and *E. coli*.

Our study showed 7% incidence of clinical chorioamnionitis, 11% microbiological chorioamnionitis and 34% histopathological chorioamnionitis. All cases of clinical chorioamnionitis were confirmed by histopathology postnatally.

Table 3: Comparison of latency period and occurrence of clinical or subclinical chorioamnionitis.

Latency period	Any of clinical or sub clinical chorioamnionitis				χ^2	P value
	No		Yes			
	N	%	N	%		
1	54	73.0	20	27.0	8.76*	0.013
2	7	63.6	4	36.4		
>3 days	5	33.3	10	66.7		

*Significant at 0.05 level.

Table 4: Microbiological profile of placental (maternal and fetal) and neonatal cultures.

Organisms	Placental culture (maternal side) N (%)	Placental culture (fetal side) N (%)	Neonatal blood culture N (%)
Negative	90 (90.0)	96 (96.0)	93 (93.9)
<i>Escherichia coli</i>	5 (5.0)	2 (2.0)	2 (2.0)
<i>Klebsiella</i>	1 (1.0)	0 (0.0)	2 (2.0)
<i>Group B Streptococcus (GBS)</i>	1 (1.0)	2 (2.0)	1 (1.0)
<i>Enterococcus faecalis</i>	1 (1.0)	0 (0.0)	0 (0.0)
<i>Proteus mirabilis</i>	1 (1.0)	0 (0.0)	0 (0.0)
<i>Citrobacter koseri</i>	1 (1.0)	0 (0.0)	0 (0.0)
<i>Staphylococcus epidermidis</i>	0 (0.0)	0 (0.0)	1 (1.0)

Table 3 shows the association between latency period and occurrence of clinical or subclinical chorioamnionitis. The association was found to be statistically significant. Hence, prolonged conservative management is associated with increased incidence of chorioamnionitis.

Table 4 shows that 10% women had positive culture results from the maternal side of the placenta. The most common microorganism isolated was *Escherichia coli* (5%). 4% women had positive culture results from the fetal side of the placenta. The organisms isolated were *Escherichia coli* and group B *Streptococcus*. Out of the 11 cases of microbiological chorioamnionitis, 4 cases showed growth similar to the high vaginal swab.

but stopped as cultures were sterile. The microorganisms isolated from these cases were *Escherichia coli*, *Klebsiella*, GBS and *Staphylococcus epidermidis*. There were 7 cases of neonatal mortality.

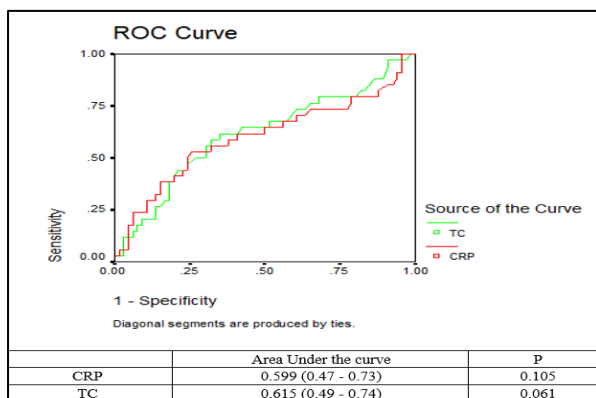
On analysing the ability of CRP to predict subclinical chorioamnionitis, it had a sensitivity of 52.9%, specificity of 74.2%, positive predictive value of 25.8%, negative predictive value of 75.4%, and an accuracy of 67% (Figure 1). Total count had a sensitivity of 58.8%, specificity of 68.2%, positive predictive value of 48.8%, negative predictive value of 76.3%, and an accuracy of 65%. So both tests had only moderate predictive value in detecting subclinical chorioamnionitis.

The sensitivity of Gibb's criteria to predict subclinical chorioamnionitis is only 20.59%, while specificity is 100%. It has a positive predictive value of 100%, a negative predictive value of 70.97%, and an overall accuracy of 73%.

DISCUSSION

Chorioamnionitis remains a significant cause of maternal and neonatal morbidity. Previous studies report a varying incidence of 4-25% for clinical chorioamnionitis and 25-70% for subclinical (histological) chorioamnionitis in women with preterm labour and PPRM.

In our study, clinical chorioamnionitis was identified in only 7% of women, whereas microbiological and histopathological evidence was present in 11% and 34%, respectively. Thus, histological chorioamnionitis occurred much more frequently than clinical chorioamnionitis. This

**Figure 1: ROC Curve for CRP and TC in detecting subclinical chorioamnionitis.**

Six babies had confirmed early-onset neonatal sepsis. 34 babies had suspected sepsis and were started on antibiotics

distinction is important because the terminology and biological understanding of chorioamnionitis have evolved with current diagnostic frameworks recognizing a spectrum including microbial invasion of amniotic cavity (MIAC), sterile intraamniotic inflammation, intraamniotic infection, maternal signs or symptoms of systemic inflammation without evidence of intraamniotic inflammation or infection and histologic chorioamnionitis.⁸ Our findings, although based on placental histopathology rather than amniotic-fluid analysis, are consistent with this broader concept: a clinically silent inflammatory process may be present even when overt maternal signs are absent.

Clinical chorioamnionitis developed in 7% of women in our study. All had maternal pyrexia and tachycardia, and one referred patient with prolonged conservative management fulfilled all of the Gibb's criteria. Five of the seven women had elevated leukocyte counts and CRP levels, and four had fetal tachycardia.

The incidence of histological chorioamnionitis in our cohort was 34%, comparable to the 35.06% reported by Wu et al, but lower than the 68% incidence reported by Pelaez et al.^{9,10} The comparatively lower incidence in our study may reflect improved antenatal care, strict aseptic practices, adherence to protocols, and close fetomaternal monitoring.

All women with clinical chorioamnionitis had histopathological confirmation, whereas other studies, such as that by Palmsten et al, reported discordance between clinical and histological findings.¹¹

In our study, Gibb's criteria demonstrated high specificity (100%) but low sensitivity (20.59%) for predicting histological chorioamnionitis, with nearly 79% of histological cases missed clinically, consistent with findings from Algerian et al.⁷ These findings suggest that the absence of conventional clinical features cannot reliably exclude underlying inflammation in women delivering preterm. Guidelines continue to recommend clinical criteria for the diagnosis of suspected intraamniotic infection in practice, while acknowledging that definitive confirmation may depend on amniotic-fluid testing or postpartum placental pathology.⁸

In our cohort, C-reactive protein (CRP) had a sensitivity of 52.9% and specificity of 74.2%, while total leukocyte count had a sensitivity of 58.8% and specificity of 68.2%. These findings are broadly consistent with published evidence showing that maternal leukocytosis and serum CRP have insufficient accuracy to reliably diagnose subclinical intrauterine infection. A meta-analysis by Sabogal et al reported that leukocyte counts above 15,000/mm³ had only 51% sensitivity and 65% specificity for detecting subclinical chorioamnionitis, while maternal serum CRP had pooled sensitivity and specificity of 68.7% and 77.1%, respectively.¹² Thus, our findings reinforce that these tests should be regarded as supportive

inflammatory markers rather than definitive diagnostic tests. Normal WBC and CRP reports should not be interpreted in isolation as evidence against intrauterine inflammation.

The poor performance of clinical criteria and laboratory markers in diagnosing chorioamnionitis may contribute to both delayed recognition of intrauterine infection as well as potentially unnecessary antibiotic exposure. A recent expert review by Jung et al suggests that definitive assessment of intra-amniotic inflammation and infection should rely on amniocentesis and sending the fluid for analysis of inflammatory markers such as interleukin-6 and matrix metalloproteinase-8, alongside amniotic fluid leukocyte count, glucose concentration, Gram staining, culture, and molecular techniques for pathogen detection.⁸

High vaginal swab cultures were positive in 39% of women, with *Candida* species, *Escherichia coli* and *Enterococcus faecalis* among the more frequent isolates. Sharma et al reported a culture positivity rate of 37.7% in women with PPROM, with *E. coli* being the predominant organism.¹³ In contrast, Ambalpadhy et al documented a much higher positivity rate (85.09%), predominantly monomicrobial infections, with *Enterococcus faecalis*, *Staphylococcus aureus*, and *E. coli* as common isolates.¹⁴ Variations in culture positivity across studies may reflect differences in patient demographics, socioeconomic conditions, hygiene practices, and healthcare access. Urine cultures were sterile in 86% of our cohort; 4% each had *Klebsiella* and *E. coli* growth. Similar findings were reported by Tanshen et al, who observed no bacteriuria in 84% of women with PPROM.¹⁵

Microbiological chorioamnionitis, based on placental culture, was observed in 11% of cases, with *E. coli* being the most common isolate, similar to findings by Berezowsky et al.¹⁶ Out of these, four demonstrated an organism similar to that identified on the high vaginal swab. This provides some support for an ascending pathway of infection, although concordance between vaginal and placental cultures was limited and a causal relationship cannot be established from these data alone. Most isolates were sensitive to penicillins, cephalosporins, amikacin, and meropenem; however, one case involved multidrug-resistant *E. coli* associated with prior repeated antibiotic exposure requiring antibiotic escalation to tigecycline. This case was complicated by early-onset neonatal sepsis due to the same resistant organism, highlighting the implications of antibiotic stewardship and the potential emergence of resistant strains in high-risk pregnancies. Unlike Western countries, as the predominant vaginal pathogen is not GBS, the choice of empiric therapy or prophylaxis in PPROM becomes challenging, ampicillin may be inadequate for gram negative pathogens.

The mean interval between admission and delivery was 2.8 days, with three women undergoing prolonged conservative management beyond three weeks; the

maximum latency was 34 days. The incidence of chorioamnionitis was significantly higher among women with prolonged conservative management. Of the 15 women managed for more than three days, 10 (66.7%) developed subclinical chorioamnionitis. These findings highlight the delicate balance between prolonging pregnancy to reduce prematurity-related morbidity and avoiding the risks of ascending infection. Chorioamnionitis is inversely proportional to gestational age in previous studies, possibly because longer latency is often attempted in extreme preterm gestations. In our cohort, clinical chorioamnionitis was more frequent in the extreme preterm group, and in some cases, infection may have preceded and triggered preterm labour.

Neonatal outcomes reflected the burden of prematurity and infection. Thirty-four neonates had suspected early-onset sepsis, and six had culture-proven sepsis, due to *E. coli*, *Klebsiella*, and group B *Streptococcus*. Four of these neonates were extreme preterm. Although three of the six had maternal clinical, microbiological, and histological chorioamnionitis, the association between chorioamnionitis and early-onset neonatal sepsis was not statistically significant in our study, possibly due to limited sample size. There were seven neonatal deaths, all in the extreme preterm group, with four associated with early-onset sepsis. These findings show that though gestational age remains the most important determinant of neonatal survival, intrauterine infection may increase morbidity.

Chorioamnionitis cannot be effectively treated with antibiotics alone and ultimately requires delivery. Management decisions must therefore balance the risks of prematurity against the dangers of ongoing intrauterine infection. Soraisham et al demonstrated that, after adjusting for gestational age, chorioamnionitis in preterm infants was strongly associated with early-onset sepsis and severe intraventricular hemorrhage.¹⁷ A study by Zaharie et al on association between chorioamnionitis and neonatal morbidity in preterm births between 23 and 28 weeks showed that histological chorioamnionitis was independently associated with severe respiratory distress syndrome.¹⁸ The fetal inflammatory response, mediated by elevated cytokines, may result in white matter injury, periventricular leukomalacia, and cerebral palsy. Importantly, this inflammatory cascade may begin before clinical signs become apparent, showing the need for close monitoring and timely intervention in PPRM and preterm labour.

Our study has several strengths. It prospectively evaluated clinical, microbiological and histopathological components of chorioamnionitis within the same cohort and examined the performance of commonly used maternal inflammatory markers and clinical criteria against histopathological findings. At the same time, several limitations should be acknowledged including the relatively small sample size and the absence of amniotic fluid studies. Polymerase chain reaction techniques for detecting atypical or fastidious organisms were not done,

which may have led to underestimation of microbiological infection. Larger studies incorporating molecular diagnostic tools are required to better understand the burden of subclinical infection and for deciding time of delivery in preterm pregnancies.

CONCLUSION

Histopathological chorioamnionitis was nearly five times more frequent than clinical chorioamnionitis showing a significant burden of clinically silent intrauterine inflammation. Clinical criteria and common inflammatory markers could not reliably exclude subclinical disease. These findings highlight the limitations of conventional clinical and laboratory markers and support the need for more sensitive, preferably minimally invasive, diagnostic strategies to identify intrauterine inflammation and guide timely management of preterm pregnancies.

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