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The Association of Histopathological and Clinical Chorioamnionitis with Neonatal Outcomes

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Abstract

Introduction: Chorioamnionitis (CA) is an infection or inflammation of the amniotic fluid, placenta, fetus, fetal membranes, or decidua, often resulting from a bacterial infection. CA occurs in 1-13% of term infants and 30-70% of preterm infants, causing significant neonatal morbidity and long-term complications. It can be diagnosed clinically or through histopathological examination.

Aim: To investigate the relationship between clinical and histopathological CA and their impact on neonatal and maternal outcomes.

Methods: This retrospective cohort study reviewed 393 mother-infant pairs at a NICU hospital from January 1, 2023, to December 31, 2023. Data included maternal and infant clinical characteristics, signs and symptoms of CA, and histopathological examination of placentas. Statistical analyses were conducted using SPSS.

Results: Among 393 pairs, 100 mothers had clinical CA. The mean rupture of membranes (ROM) was significantly longer in these mothers (32.75 hours) compared to those without clinical CA (6.74 hours). Clinical symptoms such as uterine tenderness, fever, tachycardia, and foul-smelling discharge were significant in mothers with clinical CA. Infants of these mothers had higher rates of respiratory distress (45%) and gastrointestinal symptoms (20%). NICU admissions and antibiotic use were more frequent in these infants.

Conclusion: Clinical CA in mothers is associated with poorer neonatal outcomes, including increased respiratory distress and gastrointestinal symptoms. Further research is needed to confirm these findings and explore preventive measures.

Keywords: Chorioamnionitis; Neonatal outcomes; Maternal morbidity; Histopathological examination; Retrospective cohort study

Introduction

Chorioamnionitis (CA), also known as intraamniotic infection, is infection or inflammation of the amniotic fluid, placenta, fetus, fetal membranes, or decidua [1]. It is often the result of a bacterial infection that begins in the vagina, anus, or rectum that then spreads up to the maternal uterus. It can also start in the uterus as the result of an amniotic sac rupture or tear [2]. CA occurs in approximately 1-13% of term infants and ranges from 30 to 70% in preterm infants [3]. It is associated with significant acute neonatal morbidity, including pneumonia, meningitis, sepsis, and death, alongside long-term infant complications such as bronchopulmonary dysplasia, cerebral palsy, and other adverse neurodevelopmental outcomes [4].

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There are two viewpoints for CA in the medical world: the clinical syndrome and histopathological definition. The Gibbs criteria for clinical CA is maternal fever plus two or more of the following: maternal tachycardia, fetal tachycardia, uterine tenderness, foul odor of the amniotic fluid, or maternal leukocytosis [5].

Histopathologically, CA is identified by examining the fetal membrane and chorionic plate [6]. Redline et al. [6] categorized acute inflammation of the placenta into two main types: maternal and fetal inflammatory responses. For the maternal inflammatory response, they used a staging system to describe how far the inflammation has spread within the placenta. In Stage 1, neutrophils, a type of white blood cell, are found in the chorion. Stage 2 involves neutrophils moving into the deeper connective tissue of the placenta or the amniotic sac. In Stage 3, the inflammation is severe enough to cause necrosis, or tissue death, in the amniotic sac. Staging and grading are also relevant for the fetal inflammatory response, but staging, which describes the location of the neutrophils, is often more important for assessing the severity of the inflammation than grading. Different systems have been developed and adjusted over time to evaluate these responses [6,7].

Yet, the incidence of histopathological CA is greater than that of clinically identified infection, indicating that clinical diagnostic techniques may be suboptimal [8]. Even though the clinical signs of CA are typically easy to identify and diagnose, histopathological CA is frequently underestimated or misdiagnosed due to negligent postpartum examination of the placenta and accessories [9].

This study aims to delineate the relationship between clinical and histopathological CA in relation to perinatal characteristics and comorbidities, and their effect on neonatal and maternal outcomes.

Materials and Methods

This study is a retrospective cohort analysis involving 393 mother-infant pairs. In 2023 year, we had 948 deliveries. We reviewed clinical records and conducted histopathological examinations of placental tissues available. The study was conducted over a one-year period from January 1, 2023, to December 31, 2023, at a tertiary level Neonatal Intensive Care Unit (NICU) hospital. The study received approval from the hospital's Institutional Review Board (IRB).

Medical charts of all mothers who delivered during the study period were reviewed to identify those with a diagnosis or suspicion of clinical chorioamnionitis (CA). Collected maternal data included age, parity, gestational age, and pregnancy-related medical complications such as diabetes mellitus, hypertension, urinary tract infection, or other infections. Additional data comprised mode of delivery, prenatal laboratory results, antibiotic use and duration, duration of rupture of membranes, presence of meconium, and intrapartum antibiotic administration. Maternal signs and symptoms of CA documented from the charts included the highest recorded temperature prior to delivery, maternal tachycardia (heart rate > 100 bpm), fetal tachycardia (heart rate > 160 bpm), white blood cell count and differentials, uterine tenderness, and foul-smelling or purulent vaginal discharge. Placental histopathological examination reports, recorded in maternal charts, were reviewed and classified into 3 stages as described by Redline et al. [6] [10].

Corresponding infant charts were reviewed to collect data on gestational age, Apgar scores, birth weight, documented abnormal signs and symptoms (such as poor feeding, gastrointestinal symptoms, temperature instability, and respiratory distress symptoms), admission white blood cell count, band cell percentage, C-reactive protein levels, blood culture results, antibiotic administration and duration of therapy, NICU admission, and length of hospital stay.

Clinical chorioamnionitis was diagnosed based on maternal symptoms and laboratory findings, while histopathological chorioamnionitis was confirmed through microscopic examination of placental tissue. The inclusion criteria encompassed all maternal charts with a placental pathology report and mothers who administered antibiotics during labor for suspected infection. Exclusion criteria was the mother's placental histopathological report with still birth.

Statistical analyses were performed using the SPSS XX. Descriptive statistics using mean ± standard deviation and percentages. The t-test was used for continuous variables, while the Chi-square test or Fisher's exact test was applied for nominal data. Logistic regression analyses were conducted to determine the interaction effects between variables.

Results

393 mother-infant pairs were reviewed for this study. Out of these, 100 mothers were diagnosed with clinical chorioamnionitis, while 293 mothers were without clinical chorioamnionitis. Out of the 100 mothers diagnosed with clinical chorioamnionitis, 55 were found to have stage 2-3 histopathological chorioamnionitis upon microscopic examination, while 84 mothers out of 293 mothers without clinical chorioamnionitis had stage 2-3 histopathological chorioamnionitis. The analysis also focused on the data pertaining to mothers with stage 2-3 histopathological chorioamnionitis.

Table 1 presents maternal characteristics between those diagnosed with clinical chorioamnionitis and those without the condition, including various maternal demographics, medical histories, and pregnancy-related factors, highlighting differences and similarities between the two groups. The

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mean rupture of membranes in mothers with clinical chorioamnionitis was 32.75 hours (SD 117.69) compared to 6.74 hours (SD 8.99) in those without clinical chorioamnionitis (p=<0.001). Maternal clinical symptoms, such as uterine tenderness (35% vs 0%, p=<0.001), fever (89% vs 0.5%, p=<0.001), tachycardia (84% vs 0%, p=<0.001), and foul-smelling discharge (49% vs 0%, p=<0.001), were significant among those with a clinical diagnosis of chorioamnionitis.

Table 1: This Table 1 presents a comparison of maternal characteristics between those diagnosed with clinical chorioamnionitis and those without the condition. The data includes various maternal demographics, medical histories, and pregnancy-related factors, highlighting differences and similarities between the two groups.

	Clinical Chorioamnionitis	No Chorioamnionitis	P-Value
	N=100; Mean (SD); N (%)	N= 293; Mean (SD); N (%)	
Maternal Age	29.08 (6.94)	29.83 (6.29)	0.317
Gestational age	36.95 (3.41)	37.42 (3.47)	0.241
G	2.72 (1.63)	2.65 (1.56)	0.709
P	1.52 (1.57)	1.52 (1.57)	0.995
ROM	32.75 (117.69)	6.74 (8.99)	< 0.001
Mode of Delivery			
Vaginal	62 (62)	197 (67)	0.34
C-section	38 (38)	96 (33)	
Maternal Prenatal Issues			
HTN/PreE	46 (46)	125 (43)	0.596
DM	12 (12)	57 (19)	0.091
UTI	6 (6)	7 (2)	0.048
IUGR	8 (8)	20 (6)	0.611
GBS Positive			
Yes	31 (31)	59 (20)	0.069
No	54 (54)	191 (65)	
Unknown	15 (15)	43 (15)	
MSAF	16 (16)	37 (13)	0.394
Maternal Clinical Symptoms			
Uterine Tenderness	35 (35)	0 (0)	< 0.001
Foul Smelling Discharge	49 (49)	0 (0)	< 0.001
Maternal Tachycardia	84 (84)	0 (0)	< 0.001
Maternal Fever	89 (89)	1 (0.5)	< 0.001

Table 2 compares the histopathological characteristics of mothers diagnosed with clinical chorioamnionitis to those without the condition. This Table 2 includes detailed findings from placental tissue examinations, focusing on the presence and stages of histopathological chorioamnionitis (as per RED staging), providing insights into the relationship between clinical and histopathological diagnoses. Maternal placental grading and stage 2-3 and fetal placental grading stage 2-3 were significant.

Table 2: Table 2 compares the histopathological characteristics of mothers diagnosed with clinical chorioamnionitis to those without the condition. The data includes detailed findings from placental tissue examinations, focusing on the presence and stages of histopathological chorioamnionitis (as per MIR staging), and provides insights into the relationship between clinical and histopathological diagnoses.

Placental Histopathology	Maternal Diagnosis of CA	No Maternal Diagnosis of CA	P-value Only Qualitative
	N= 100; N (%)	N=293; N (%)	
Maternal Placenta Stages 2,3	55 (55)	84 (28)	< 0.001
Maternal Placenta Stages 0,1	45 (45)	207 (71)	
Fetal placenta Stage 2,3	28 (28)	28 (10)	< 0.001
Fetal placenta Stage 0,1	71 (71)	263 (90)	

Table 3 shows the characteristics of infants that demonstrate signs and symptoms. Among the infants, 282 out of 293 maternal-infant pairs were diagnosed without placental chorioamnionitis, while 99 out of 100 maternal-infant pairs were diagnosed with placental chorioamnionitis. Table 3 indicates that 45 infants had respiratory distress and 20 had gastrointestinal symptoms, both with a significance level of $p < 0.001$. NICU hospitalizations were more common in infants with maternal chorioamnionitis (N=60, $p < 0.001$). Antibiotic use was also higher in infants with maternal chorioamnionitis (N=98, $p < 0.001$). The need for oxygen use was not statistically significant in infants regardless of the maternal diagnosis of chorioamnionitis.

Table 4 presents maternal and infant characteristics with maternal placental histopathological diagnosis of staging 2 and 3, both with and without clinical chorioamnionitis. This Table 4 includes maternal demographics, medical histories, pregnancy-related factors, and detailed infant outcomes, providing a comprehensive overview of the impact of advanced stages of histopathological chorioamnionitis on both mothers and their infants. Maternal clinical symptoms such as uterine tenderness, fever, tachycardia, and foul-smelling discharge were significant among those with histopathological grade stage 2 and 3 and a clinical diagnosis of chorioamnionitis.

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Table 3: Characteristics of Infants Demonstrating Signs and Symptoms. This Table 3 presents the characteristics of infants who exhibited signs and symptoms. Among the study population, 282 infants out of 293 maternal-infant pairs were diagnosed without placental chorioamnionitis, while 99 out of 100 maternal-infant pairs were diagnosed with placental chorioamnionitis.

	Infants of Mothers with Clinical CA	Infants of the Mothers without Clinical Chorioamnionitis	P-value Only Qualitative
	N= 99 N (%); Mean (SD)	N= 282 N (%); Mean (SD)	
Gestational Age	36.95 (3.41)	37.43 (3.47)	0.241
Male	53 (53)	135 (46)	0.893
APGAR@ 1 min	8.43 (1.17)	7.35 (1.99)	< 0.001
APGAR@ 5 mins	8.95 (0.268)	8.49 (0.994)	< 0.001
Laboratory Parameters			
Newborn WBC	14.36 (5.18)	15.59 (5.16)	0.151
Positive Blood Culture	1 (1.0)	0 (0.0)	0.676
Infant Clinical symptoms			
Resp Symptoms	45 (45)	27 (10)	< 0.001
GI Symptoms			
Poor Feeding	15 (21)	11 (5.0)	< 0.001
Vomiting	6	4	
Hypoglycemia	22 (22)	39 (13)	0.038
Hyperbilirubinemia	16 (16)	20 (7.0)	0.006
Cardiac Symptoms			
Tachycardia	0 (3.0)	1 (0.35)	0.01
Bradycardia	3	0	
Fever	1 (1.0)	1 (0.35)	0.195
NICU Hospitalization	62 (62)	48 (16)	< 0.001
Antibiotics	100 (100)	31/62 (50)	< 0.001
O_2 Support			
Non-invasive	32 (36)	24/62 (38)	0.235
Invasive	4	0	
Histopathological Findings			
MIR Stage 0 (normal)	34 (43)	169 (58)	< 0.001
MIR stage 1 (mild)	11 (11)	39 (13)	< 0.001
MIR Stage 2 (moderate)	52 (52)	76 (26)	< 0.001
MIR Stage 3 (severe)	3 (3)	8 (3)	< 0.001
FIR Stage 0 (normal)	60 (60)	234 (80)	< 0.001
FIR Stage 1 (mild)	12 (12)	31 (10)	< 0.001
FIR Stage 2 (moderate)	9 (9)	16 (5)	< 0.001
FIR Stage 3 (severe)	19 (19)	16 (5)	< 0.001

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Table 4: Maternal and Infant Characteristics with Maternal Placental Histopathological Diagnosis of Staging 2 and 3. This Table 4 presents the characteristics of mothers and their infants who were diagnosed with stage 2 and 3 histopathological chorioamnionitis. It includes maternal demographics, medical histories, pregnancy-related factors, and detailed infant outcomes, providing a comprehensive overview of the impact of advanced stages of histopathological chorioamnionitis on both mothers and their infants.

	Maternal Diagnosis of Clinical CA Along with MIR (Stage 2,3)	No Maternal Diagnosis of Clinical CA but has MIR (Stage 2,3)	P-value Only Qualitative
	N= 55; N (%)	N= 84; N (%)	
Mode of Delivery			
Vaginal	41 (75))	62 (74)	0.923
C-section	14 (25)	22 (26)	
Gestational Age	37.1 (3.19)	38.3 (1.72)	0.004
Maternal Age	27.96 (7.21)	28.74 (6.49)	0.511
Birth Weight	2733 (853)	3127 (508)	< 0.001
APGAR @ 1 min	7.22 (2.05)	8.35 (1.28)	< 0.001
APGAR @ 5 min	8.49 (0.96)	8.94 (0.28)	< 0.001
ROM	14.93 (15.47)	11.80 (9.96)	0.566
Maternal Prenatal Issues			
Hypertension/PreE	28 (51)	44 (52)	0.862
Diabetes	5 (9.0)	12 (14)	0.435
UTI	1 (2.0)	2 (2.0)	1
IUGR	1 (2.0)	0 (0)	0.396
HIV	2 (4.0)	2 (2.0)	0.648
HSV	1 (2.0)	2 (2.0)	1
Syphilis	0 (0)	3 (4.0)	0.277
GBS Status			
Positive	17 (31)	15 (18)	0.208
Unknown	6 (11)	11 (13)	
MSAF	5 (9.0)	3 (4.0)	0.264
Maternal Clinical Symptoms			
Uterine tenderness	17 (31)	0 (0)	< 0.001
Foul smelling Discharge	27 (49)	0 (0)	< 0.001
Maternal Tachycardia	45 (82)	0 (0)	< 0.001
Maternal Fever	53 (96)	1 (2.0)	< 0.001
Infant Clinical Symptoms			
Resp Symptoms	26 (47)	7 (8.0)	< 0.001
GI Symptoms	10 (18)	2 (2.0)	0.004
Hypoglycemia	10 (18)	6 (7.0)	0.059
Hyperbilirubinemia	9 (16)	5 (6.0)	0.08
SGA	11 (20)	1 (2.0)	<0.001
Cardiac Symptoms	0 (0)	0 (0)	--
Fever	0 (0)	0 (0)	--
Blood Culture Done	55 (100)	10 (12)	< 0.001
NICU Hospitalization	30 (55)	11 (13)	< 0.001
NICU Stay Duration	14.9	11.8	0.556
Antibiotics	55 (100)	6 (7)	< 0.001
O_2 Support	18 (33)	4 (5)	0.89

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Infant clinical symptoms such as being small for gestational age (SGA) and respiratory symptoms were significant among those with maternal histopathological grade stage 2 and 3 and clinical chorioamnionitis. Specifically, respiratory symptoms were seen in 26 infants with maternal placental staging of grade 2 & 3 and clinical chorioamnionitis, compared to 7 infants with maternal placental staging of grade 2 & 3 without clinical chorioamnionitis, with a significance level of $p < 0.001$.

Table 5 demonstrates that advanced stages (Stages 2 and 3) of fetal inflammatory response syndrome (FIR) significantly impact both maternal and neonatal outcomes. Infants at FIR stages 2 and 3 born to mothers diagnosed with clinical chorioamnionitis show considerable clinical implications compared to those without this diagnosis. Maternal age (27.75 vs. 28.82 years, $p = 0.561$) and mode of delivery (vaginal: 57% vs. cesarean section: 79%, $p = 0.860$) did not significantly differ between groups. However, infants in the clinical chorioamnionitis group exhibited lower 1-minute Apgar scores (7.04 vs. 8.36, $p = 0.008$) and a lower incidence of meconium-stained amniotic fluid (0% vs. 14%, $p = 0.038$). Maternal clinical symptoms such as uterine tenderness (39% vs. 0%, $p < 0.001$), foul-smelling discharge (46% vs. 0%, $p < 0.001$), tachycardia (75% vs. 0%, $p < 0.001$), and fever (89% vs. 0%, $p < 0.001$) were markedly elevated in infants born to mothers with clinical chorioamnionitis compared to those born to mothers without it. Furthermore, infants born to mothers with clinical chorioamnionitis were more likely to present with respiratory symptoms (50% vs. 4%, $p < 0.001$) and required NICU hospitalization (61% vs. 11%, $p < 0.001$).

Table 5: Maternal and Infant Characteristics With Fetal Placental Histopathological Diagnosis of Staging 2 and 3. This Table 5 presents the characteristics of mothers and their infants who were diagnosed with stage 2 and 3 Fetal histopathological chorioamnionitis. It includes maternal demographics, medical histories, pregnancy-related factors, and detailed infant outcomes, providing a comprehensive overview of the impact of advanced stages of histopathological chorioamnionitis on both mothers and their infants.

	Maternal Diagnosis of Clinical CA along with FIR (Stage 2,3)	No Maternal Diagnosis of Clinical CA but has FIR (stage 2,3)	P-value Only Qualitative
	N= 28; N (%)	N= 28; N (%)	
Mode of Delivery			
Vaginal	16 (57)	22 (79)	0.86
C-section	12 (43)	6 (21)	
Gestational Age	37.5 (3.81)	38.4 (1.48)	0.217
Maternal Age	27.75 (6.86)	28.82 (6.85)	0.561
Birth Weight	2920 (997)	3098 (459)	0.395
APGAR @ 1 min	7.04 (2.20)	8.36 (1.22)	0.008
APGAR @ 5 min	8.25 (1.32)	8.93 (0.38)	0.12
ROM	88.36 (214)	10.86 (9.77)	0.061
Maternal Prenatal Issues			
Hypertension/PreE	11 (39)	11 (93)	0.912
Diabetes	2 (7.0)	6 (21)	0.127
UTI	0 (0)	1 (4.0)	0.313
IUGR	0 (0)	0 (0)	--
HIV	0 (0)	0 (0)	--
HSV	2 (7.0)	0 (0)	0.15
Syphilis	0 (0)	0 (0)	--
GBS Status			
Pos	6 (21)	7 (25)	0.775
Unknown	1 (4.0)	2 (7.0)	
MSAF	0 (0)	4 (14)	0.038
Maternal Clinical Symptoms			
Uterine tenderness	11 (39)	0 (0)	< 0.001
Foul-Smelling Discharge	13 (46)	0 (0)	< 0.001
Maternal Tachy	21 (75)	0 (0)	< 0.001

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Maternal fever	25 (89)	0 (0)	< 0.001
Infant Clinical Symptoms			
SEX, Male	15 (54)	20 (71)	0.168
Resp Symptoms	14 (50)	1 (4.0)	< 0.001
GI Symptoms	4 (14)	0 (0)	0.116
Hypoglycemia	4 (14)	1 (4.0)	0.16
Hyper Bilirubin	5 (18)	1 (4.0)	0.084
SGA	2 (7.0)	0 (0)	0.15
Cardiac Symptoms	0 (0)	0 (0)	--
Fever	0 (0)	0 (0)	--
Blood culture done	28 (100)	1 (4.0)	< 0.001
NICU Hospitalization	17 (61)	3 (11)	< 0.001
NICU Stay duration	15.88 (17.33)	8 (-)	0.666
Antibiotics	28 (100)	1 (4.0)	0.841
O2 Support	11 (39)	1 (4.0)	0.8

Discussion

Chorioamnionitis (CA) is a common condition encountered during pregnancy, often associated with preterm prolonged rupture of membranes (PPROM) or prolonged rupture of membranes (PROM) in term infants. CA can be diagnosed through maternal clinical symptoms or histopathological examination of the placenta. In our study, we identified 100 mothers whose placentas were examined for clinical chorioamnionitis.

This study demonstrates a strong association between clinical CA and factors such as prolonged rupture of membranes (PROM), maternal urinary tract infection, and maternal symptoms including uterine tenderness, fever, tachycardia, and foul-smelling discharge. However, Group B Streptococcus (GBS) status was not associated with clinical CA in our study, a finding consistent with other studies that showed that GBS status is not linked to CA after adjusting for confounders [3].

Infection is known to cause PROM, with previous studies indicating that 30% of premature PROM cases have positive fetal blood cultures for microorganisms. Gupta et al. highlighted the role of cytokine-mediated stimulation of prostaglandins and proteases in intrauterine inflammation leading to PROM. Our study supported these findings, showing a mean ROM of 32.75 hours in mothers with clinical CA versus 6.74 hours in those without. However, when adjusted for stage 2-3 maternal histopathological CA, no association between ROM and CA was observed.

Clinical findings for diagnosing clinical CA include maternal fever, uterine tenderness, maternal or fetal tachycardia, and purulent amniotic fluid or discharge. In our study, maternal clinical symptoms were strongly associated with the diagnosis of clinical CA, even when further grouped by stage 2-3 maternal inflammatory response ($p<0.001$) (Table 4) and fetal inflammatory response ($p<0.01$) (Table 5). While isolated symptoms are not strongly linked to CA, their combination suggests intrauterine infection [3]. Foul-smelling discharge, though rare, often indicates a prolonged or organism-specific infection [3].

Histopathological CA is more common than clinical CA, making it a more sensitive (83-100%) but less specific (23-52%) predictor of CA [3]. In our study, 55% of mothers diagnosed with clinical CA also had histopathological CA indicating maternal inflammatory response. Smulian JC et al. [11] found similar results, with 62% of mothers diagnosed with clinical CA showing histological findings.

Infants born to mothers with clinical chorioamnionitis had significant respiratory and gastrointestinal symptoms, which remained significant when classified by maternal stage 2-3 inflammatory response and fetal stage 2-3 inflammatory response. Fetal inflammatory response syndrome (FIRS) is known to be associated with fetal and neonatal complications [3,12].

The use of blood cultures and antibiotics was observed in infants with maternal clinical CA along with MIRS stage 2-3. NICU stays were significant for infants of mothers with clinical CA, both with and without histological findings. In our study, 45% of infants with maternal clinical CA had respiratory symptoms, compared to a 20% incidence of respiratory distress syndrome (RDS) in a previous study [13]. Chorioamnionitis is also linked to early onset sepsis; however, our study found no association between positive blood cultures and maternal CA [14].

Chorioamnionitis is a risk factor for long-term

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neurodevelopmental issues. Although our study did not focus on long-term outcomes, prevention and early diagnosis of CA are crucial to prevent neonatal outcomes in Neonatal Intensive Care Unit (NICU). Early administration of antibiotics to mothers suspected of having CA has been shown to improve perinatal and neonatal outcomes [15,16].

In our study, the association of neonatal symptoms with maternal clinical CA remained significant even after adjusting for histopathological findings, suggesting that clinical CA is a better predictor of infant outcomes. Further studies with larger sample sizes and varied gestational ages are needed to corroborate these findings.

Conclusion

The presence of clinical chorioamnionitis (CA) in mothers is associated with poorer neonatal outcomes compared to those without clinical CA. This is further emphasized by the fact that even when stratified for histopathological findings indicative of stages 2-3, infants born to mothers with clinical chorioamnionitis had significantly worse outcomes, particularly in terms of respiratory distress and gastrointestinal symptoms.

Conflict of Interest Disclosures (includes financial disclosures): The authors declare that there are no conflicts of interest.

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