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BIOCHEMICAL AND PHYSIOLOGICAL CHANGES IN MOTHERS WITH CHORIOAMNIONITIS AND THEIR IMPACT ON NEONATAL OUTCOMES

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ABSTRACT

Background: Chorioamnionitis is a condition in which the fetal membranes and amniotic cavity become inflamed, which can cause significant changes to happen in the mother and to the baby. Identifying biochemical and physiological abnormalities in the mother early in pregnancy could be used to identify babies who are at greater risk of poor outcome.

Objective: To evaluate biochemical and physiological changes in mothers with chorioamnionitis and determine their impact on neonatal outcomes.

Methodology: This prospective observational study took place in Kabir Medical College, Peshawar from January 2025 to June 2025. 82 pregnant women with clinical chorioamnionitis (0.26% of all deliveries) were enrolled using the consecutive non-probability sampling method. Physiological parameters of the mother were recorded such as temperature, heart rate, respiratory rate, blood pressure and fetal heart rate. Laboratory tests performed were complete blood count, neutrophil-to-lymphocyte ratio, C-reactive protein, procalcitonin and serum lactate. The neonatal outcomes were Apgar score, early-onset sepsis, respiratory distress, NICU admission, requirement for respiratory support, stillbirth, and early neonatal death. The appropriate statistical tests were used to assess associations and multivariable logistic regression was used to determine independent predictors of adverse neonatal outcomes.

Results: Maternal fever was present in 82.9% of participants, maternal tachycardia in 73.2%, leukocytosis in 67.1%, raised C-reactive protein in 78.0%, elevated procalcitonin in 46.3%, and serum lactate above 2 mmol/L in 25.6%. NICU admission occurred in 52.4% of neonates, early-onset sepsis in 32.9%, respiratory distress in 30.5%, and perinatal mortality in 9.8%. Prolonged rupture of membranes, fetal tachycardia, raised maternal procalcitonin, elevated lactate, and lower gestational age were independently associated with adverse neonatal outcomes.

Conclusion: Maternal chorioamnionitis was associated with substantial inflammatory and physiological disturbances and a high frequency of neonatal complications. Combined clinical and biochemical assessment may support early risk stratification and timely maternal and neonatal management.

KEYWORDS: Chorioamnionitis, maternal inflammation, procalcitonin, C-reactive protein, neonatal sepsis, NICU admission

INTRODUCTION

Chorioamnionitis refers to an acute inflammatory process in the chorion, amnion, amniotic fluid, placenta or any combination of these structures. It is almost always due to a bacterial infection which has ascended from the lower genital tract but can also be caused by hematogenous seeding or by invasive obstetric care. It is more common in cases of prolonged rupture of membranes, prolonged labour, multiple vaginal examinations, preterm delivery and intrauterine instrumentation. Chorioamnionitis is an important cause of maternal and neonatal morbidity as the inflammatory process can progress quickly [1-3].

Physiological and biochemical changes occur in the mother in response to chorioamnionitis. A systemic inflammatory response may be indicated by fever, tachycardia, uterine tenderness, hypotension, and increased respiratory rate. Laboratory abnormalities are often present and include leukocytosis, neutrophilia, raised C-reactive protein, raised

procalcitonin, and raised serum lactate. Some of these are also associated with changes during uncomplicated labour but significant or persistent abnormalities could represent more severe infection and an increased risk of maternal sepsis. The use of clinical observations in conjunction with biochemical markers may thus enhance the ability to recognize disease severity [4-6].

The fetal and neonatal morbidity and mortality from chorioamnionitis are notable. Intra-amniotic inflammation can lead to fetal inflammatory response syndrome, preterm labour, impaired placental gas exchange and early-onset neonatal infection. Exposed neonates have a higher risk of low Apgar score, respiratory distress syndrome, pneumonia, sepsis, seizures, NICU admission and mortality. The vulnerability of premature and low-birth-weight neonates is due to their immature respiratory and immune systems. These complications can be minimised by timely antibiotic administration, close monitoring of the foetus, appropriate planning of delivery and early support for the newborn [7-9].

Despite the clinical importance of chorioamnionitis, the predictive value of individual maternal physiological and biochemical changes remains variable. Fever and leukocytosis may be sensitive but lack specificity, whereas procalcitonin and serum lactate may provide additional information regarding bacterial activity and systemic severity. Evidence from local healthcare settings regarding the relationship between these maternal markers and neonatal outcomes remains limited. Therefore, this study was conducted to evaluate the biochemical and physiological changes occurring in mothers with chorioamnionitis and to determine their association with adverse neonatal outcomes.

METHODOLOGY

This prospective observational study was conducted at Kabir Medical College, Peshawar, from January 2025 to June 2025. The study was designed to evaluate biochemical and physiological changes in mothers diagnosed with chorioamnionitis and to determine their relationship with neonatal outcomes. A total of 82 pregnant women fulfilling the eligibility criteria were enrolled through consecutive non-probability sampling. The sample size was calculated using an anticipated frequency of adverse neonatal outcomes among mothers with chorioamnionitis, a 95% confidence level, and an acceptable margin of error. All eligible women presenting during the study period were approached for participation until the required sample size was achieved.

Singleton pregnancies were included if the woman had been diagnosed with clinical chorioamnionitis at 24 weeks gestation or later during labour or before delivery. The clinical diagnosis was chorioamnionitis if the mother had a fever (38°C or higher) and had one or more of the following symptoms: fetal tachycardia, maternal tachycardia, uterine tenderness, foul-smelling amniotic fluid, or maternal leukocytosis. Women with multiple pregnancies, major fetal congenital anomalies, chronic renal disease, chronic hepatic disease, autoimmune disease, pre-existing systemic infection or incomplete laboratory records were excluded. To minimize the possibility of confounding, mothers receiving long-term antibiotic therapy before admission or who delivered outside of the study hospital were also excluded.

Demographic and obstetric data was obtained from the mother by a structured data collection form after enrolment. The following variables were included: maternal age, gravidity, parity, gestational age, booking status, duration of labour, duration of rupture of membranes, number of vaginal examinations, presence of meconium-stained or foul-smelling amniotic fluid, uterine tenderness, mode of delivery and use of intrapartum antibiotics. The physiological measurements made included maternal temperature, pulse rate, respiratory rate, systolic blood pressure, diastolic blood pressure, oxygen saturation, and fetal heart rate. Maternal fever was considered to be a temperature of at least 38°C, maternal tachycardia a pulse rate more than 100 beats per minute, fetal tachycardia a fetal heart rate of more than 160 beats per minute, and prolonged rupture of membranes as a rupture of membranes of 18 hours or more.

Maternal venous blood samples were obtained at the time of diagnosis and before the administration of antibiotics whenever clinically feasible. Laboratory tests performed were hemoglobin concentration, total leukocyte count, absolute neutrophil count, platelet count, neutrophil-to-lymphocyte ratio, C-reactive protein, serum procalcitonin, serum lactate, renal function tests, liver function tests, and blood culture. Leukocytosis was defined as total leucocyte count $> 15 \times 10^9$/L in the blood and elevated lactate > 2 mmol/L in blood. Postnatal parameters were documented after delivery such as birth weight, neonatal sex, neonatal intensive care unit (NICU) admission, requirement for resuscitation, respiratory support, respiratory distress syndrome (RDS), early onset neonatal sepsis (EONS), blood culture positivity, neonatal pneumonia, hypoglycemia, seizures, stillbirth, and early neonatal death. An adverse neonatal outcome was considered to be an early onset neonatal sepsis, a NICU admission, need for respiratory support, 5-minute Apgar score less than 7, stillbirth, or death within 7 days of birth.

The data was entered and analyzed via IBM SPSS Statistics. Data were presented as mean (SD) or median (IQR) depending on the distribution of the data for continuous variables and as frequencies and percentages for categorical variables. Continuous maternal variables were compared between the neonates with and without adverse outcomes using the independent-samples t-test or Mann–Whitney U test. Correlations between maternal factors (categorical

variables) and neonatal outcomes were conducted using the chi-square test or Fisher's exact test. Those with p value < 0.20 from univariable analyses were included in a multivariable binary logistic regression model to determine independent predictors for adverse neonatal outcomes. Odds ratios (ORs) with 95% confidence intervals (CIs) were presented, and a p value <0.05 was deemed statistically significant.

RESULTS

The study included a total of 82 mothers with chorioamnionitis and their neonates. The average maternal age was 28.7 years ± 5.4 years (range 18-41 years). Most participants, 47 (57.3%), were aged between 20 and 29 years, while 25 (30.5%) were between 30 and 39 years. 12.2% of the mothers were either below 20 or 40 years and above. The average gestation at delivery was 35.8 ± 3.1 weeks. 46 (56.1%) cases were delivered preterm, that is, prior to 37 complete weeks. The number of primigravida mothers was 31 (37.8%) and multigravida mothers 51 (62.2%). In 49 (59.8%) mothers the rupture of membranes was prolonged for 18 hours or more. The average time of membrane rupture was 21.6 ± 10.8 hours. In 29 (35.4%) cases, the amniotic fluid was meconium stained and in 34 (41.5%) cases the amniotic fluid was foul smelling. Thirty-nine (47.6%) mothers had tender uterus. The number of mothers undergoing vaginal delivery was 45 (54.9%) and 37 (45.1%) mothers underwent cesarean delivery.

Table 1. Maternal demographic and obstetric characteristics (n = 82)

Variable	Frequency (%) or Mean ± SD
Maternal age, years	28.7 ± 5.4
Age <20 years	6 (7.3)
Age 20–29 years	47 (57.3)
Age 30–39 years	25 (30.5)
Age ≥40 years	4 (4.9)
Primigravida	31 (37.8)
Multigravida	51 (62.2)
Gestational age, weeks	35.8 ± 3.1
Preterm delivery	46 (56.1)
Prolonged rupture of membranes ≥18 hours	49 (59.8)
Meconium-stained amniotic fluid	29 (35.4)
Foul-smelling amniotic fluid	34 (41.5)
Uterine tenderness	39 (47.6)
Vaginal delivery	45 (54.9)
Cesarean delivery	37 (45.1)

The most common physiological abnormality was maternal fever, which was seen in 68 (82.9%) women. 60 (73.2%) of the cases had tachycardia with mean maternal heart rate of 109.6 ± 13.8 beats per minute. In 28 (34.1%) mothers increased respiratory rate was noted and in 12 (14.6%) mothers, hypotension was noted. In 44 (53.7%) cases, fetal tachycardia (FHR>160 bpm) was observed. Severe maternal sepsis developed in 16 (19.5%) participants, with 4 (4.9%) of those admitted to intensive care unit (ICU).

Table 2. Maternal physiological findings at diagnosis

Physiological variable	Frequency (%) or Mean ± SD
Maternal temperature, °C	38.4 ± 0.6
Maternal fever ≥38°C	68 (82.9)
Maternal heart rate, beats/minute	109.6 ± 13.8
Maternal tachycardia >100 beats/minute	60 (73.2)
Respiratory rate, breaths/minute	21.8 ± 4.3
Increased respiratory rate	28 (34.1)
Maternal hypotension	12 (14.6)
Fetal tachycardia >160 beats/minute	44 (53.7)
Maternal sepsis	16 (19.5)
Maternal intensive care admission	4 (4.9)

The mean total leukocyte count was 16.9 ± 5.2 × 10^9/L, and leukocytosis exceeding 15 × 10^9/L was present in 55 (67.1%) mothers. The mean neutrophil-to-lymphocyte ratio was 8.7 ± 3.9. Thirty-eight (46.3%) mothers had elevated serum PCT levels (52.6 ± 31.4 mg/L) and 21 (25.6%) mothers had lactate levels above 2 mmol/L. In 18 (22.0%) cases, maternal blood cultures were positive. Escherichia coli was the most commonly isolated organism (7 isolates),

followed by group B Streptococcus (4 isolates), Staphylococcus aureus (3 isolates), Klebsiella pneumoniae (2 isolates) and other organisms (2 isolates).

Table 3. Maternal biochemical and hematological findings

Laboratory parameter	Frequency (%) or Mean ± SD
Hemoglobin, g/dL	10.8 ± 1.4
Total leukocyte count, ×10⁹/L	16.9 ± 5.2
Leukocytosis >15 ×10⁹/L	55 (67.1)
Absolute neutrophil count, ×10⁹/L	13.7 ± 4.8
Neutrophil-to-lymphocyte ratio	8.7 ± 3.9
Platelet count, ×10⁹/L	218.5 ± 72.6
C-reactive protein, mg/L	52.6 ± 31.4
Raised C-reactive protein	64 (78.0)
Procalcitonin, ng/mL	1.42 ± 1.76
Raised procalcitonin	38 (46.3)
Serum lactate, mmol/L	1.9 ± 1.1
Lactate >2 mmol/L	21 (25.6)
Positive maternal blood culture	18 (22.0)

The mean neonatal birth weight was 2.42 ± 0.68 kg. Low birth weight below 2.5 kg was observed in 45 (54.9%) neonates, while 13 (15.9%) had a birth weight below 1.5 kg. There were 44 (53.7%) male and 38 (46.3%) female neonates. There were 32 neonates (39.0%) with an Apgar score of < 7 at one-minute and 19 neonates (23.2%) with an Apgar score of < 7 at five minutes. 28 (34.1%) cases needed neonatal resuscitation. Forty-three (52.4%) neonates required admission to the neonatal intensive care unit. Twenty-seven (32.9%) had early onset neonatal sepsis, of which 13 (15.9%) were culture-proven sepsis. Twenty-five (30.5%) neonates had respiratory distress syndrome and 21 (25.6%) required CPAP or mechanical ventilation. A diagnosis of neonatal pneumonia was made in 12 (14.6%) infants, neonatal hypoglycemia in 14 (17.1%) and neonatal seizures in 7 (8.5%). Three infants (3.7%) were stillborns, five (6.1%) were early neonatal deaths. The overall perinatal mortality rate thus was 9.8%.

Table 4. Neonatal characteristics and clinical outcomes

Neonatal variable	Frequency (%) or Mean ± SD
Birth weight, kg	2.42 ± 0.68
Low birth weight <2.5 kg	45 (54.9)
Very low birth weight <1.5 kg	13 (15.9)
Male sex	44 (53.7)
Female sex	38 (46.3)
Apgar score <7 at 1 minute	32 (39.0)
Apgar score <7 at 5 minutes	19 (23.2)
Neonatal resuscitation	28 (34.1)
NICU admission	43 (52.4)
Early-onset neonatal sepsis	27 (32.9)
Culture-confirmed neonatal sepsis	13 (15.9)
Respiratory distress syndrome	25 (30.5)
Respiratory support required	21 (25.6)
Neonatal pneumonia	12 (14.6)
Hypoglycemia	14 (17.1)
Neonatal seizures	7 (8.5)
Stillbirth	3 (3.7)
Early neonatal death	5 (6.1)
Overall perinatal mortality	8 (9.8)

The composite adverse neonatal outcome had high prevalence (49 (59.8%) neonates): early onset sepsis, NICU admission, respiratory support, 5 minute Apgar score below 7, stillbirth, and early neonatal death were seen. The mothers who had a prolonged rupture of membrane had a significantly higher incidence of adverse outcomes than those who had a shorter rupture of membrane (71.4% vs. 42.4%, p=0.009). Fetal tachycardia also was associated with adverse neonatal outcomes (75.0% vs 42.1%, p = 0.003). Leukocyte counts were significantly higher in mothers with

neonates that had adverse outcomes compared to those with neonates that had favourable outcomes (18.5 ± 5.1 versus 14.5 ± 4.3 × 10⁹/L, p < 0.001). Maternal C-reactive protein was also higher in the adverse-outcome group (64.8 ± 32.7 versus 34.4 ± 18.9 mg/L, p < 0.001). The mean PCT level was 1.88 ± 1.91 ng/mL for mothers whose neonates had adverse outcomes, 0.73 ± 1.12 ng/mL for mothers whose neonates did not have adverse outcomes (p = 0.003). There was also a higher level of lactate in the adverse-outcome group (2.2 ± 1.2 versus 1.4 ± 0.7 mmol/L, p = 0.002).

Table 5. Association of maternal factors with composite adverse neonatal outcomes

Maternal factor	Adverse outcome n/N (%)	No adverse outcome n/N (%)	p-value
Prolonged rupture of membranes ≥18 hours	35/49 (71.4)	14/49 (28.6)	0.009
Membrane rupture <18 hours	14/33 (42.4)	19/33 (57.6)	
Maternal fever ≥38°C	44/68 (64.7)	24/68 (35.3)	0.083
Maternal temperature <38°C	5/14 (35.7)	9/14 (64.3)	
Maternal tachycardia	41/60 (68.3)	19/60 (31.7)	0.010
No maternal tachycardia	8/22 (36.4)	14/22 (63.6)	
Fetal tachycardia	33/44 (75.0)	11/44 (25.0)	0.003
No fetal tachycardia	16/38 (42.1)	22/38 (57.9)	
Raised maternal CRP	43/64 (67.2)	21/64 (32.8)	0.012
Normal maternal CRP	6/18 (33.3)	12/18 (66.7)	
Raised procalcitonin	29/38 (76.3)	9/38 (23.7)	0.004
Normal procalcitonin	20/44 (45.5)	24/44 (54.5)	
Maternal lactate >2 mmol/L	18/21 (85.7)	3/21 (14.3)	0.006
Maternal lactate ≤2 mmol/L	31/61 (50.8)	30/61 (49.2)	

The use of multivariate logistic regression was used to identify independent factors associated with an adverse neonatal outcome. When maternal age was adjusted for, as were gestational age and mode of delivery, prolonged rupture of membranes was an independent risk factor for adverse neonatal outcomes with an adjusted odds ratio of 2.86. Fetal tachycardia was more than tripled the risk of a poor neonatal outcome. Poor neonatal outcome was also independently associated with raised procalcitonin, and serum lactate levels >2mmol/L in the mother. There was a protective effect for increasing gestational age; for every extra completed week, the risk of an adverse neonatal outcome dropped by about 21%.

Table 6. Multivariable logistic regression for predictors of adverse neonatal outcomes

Predictor	Adjusted odds ratio	95% confidence interval	p-value
Prolonged rupture of membranes ≥18 hours	2.86	1.08–7.57	0.034
Maternal tachycardia	2.43	0.87–6.78	0.090
Fetal tachycardia	3.41	1.24–9.37	0.017
Raised maternal CRP	2.18	0.71–6.67	0.173
Raised maternal procalcitonin	3.09	1.08–8.83	0.035
Maternal lactate >2 mmol/L	3.77	1.01–14.10	0.048
Gestational age, per additional week	0.79	0.66–0.94	0.008
Cesarean delivery	1.29	0.48–3.48	0.611

Overall, maternal chorioamnionitis was accompanied by marked physiological and biochemical disturbances, particularly fever, tachycardia, leukocytosis, raised C-reactive protein, increased procalcitonin and elevated serum lactate. These abnormalities were associated with a substantial burden of neonatal complications, including prematurity, early-onset sepsis, respiratory distress, NICU admission and perinatal mortality.

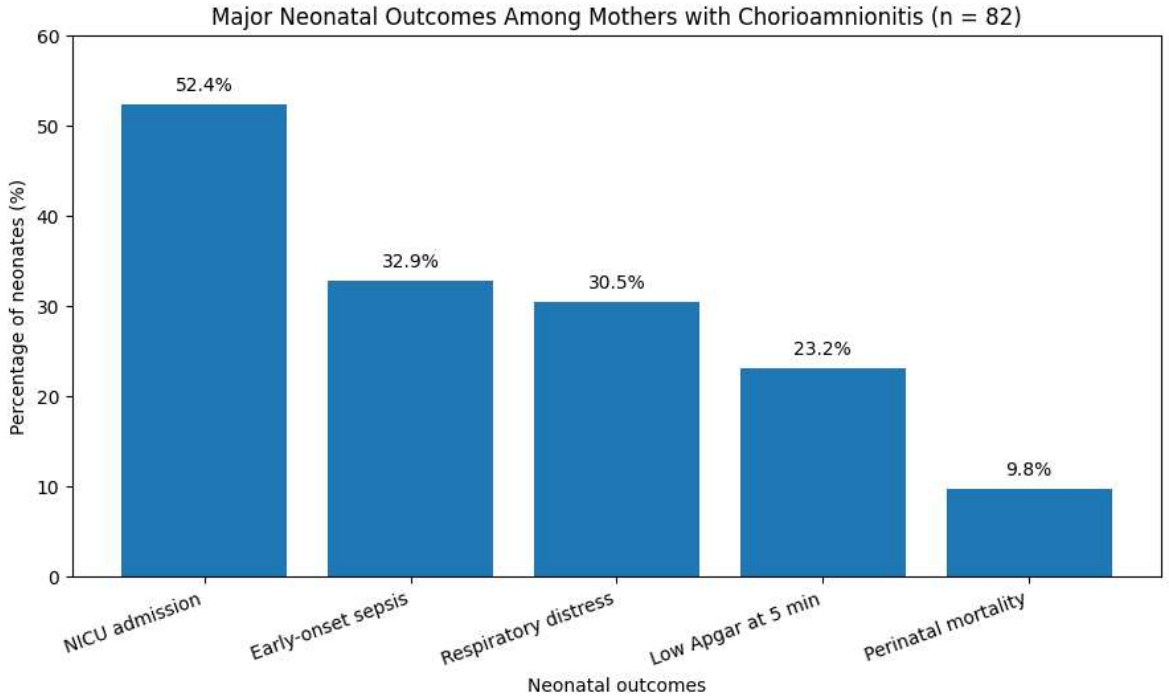

Figure 1. Major neonatal outcomes among mothers diagnosed with chorioamnionitis (n = 82).
NICU admission was the most frequent adverse outcome (52.4%), followed by early-onset neonatal sepsis (32.9%), respiratory distress syndrome (30.5%), a five-minute Apgar score below 7 (23.2%), and perinatal mortality (9.8%).

DISCUSSION

The present study demonstrated that maternal chorioamnionitis was associated with substantial biochemical and physiological disturbances and a high frequency of adverse neonatal outcomes. Maternal fever, tachycardia, leukocytosis, elevated C-reactive protein, raised procalcitonin, and increased serum lactate were commonly observed at the time of diagnosis. More than half of the neonates required admission to the neonatal intensive care unit, while early-onset sepsis, respiratory distress syndrome, low Apgar scores, and perinatal mortality were also documented. These findings indicate that chorioamnionitis is not limited to localized intra-amniotic infection but may produce a systemic maternal inflammatory response with clinically important consequences for the newborn [10-12].

Physiological abnormalities most common in the study were maternal fever and maternal tachycardia. The changes were probably related to intraamniotic infection/inflammation. Fetal tachycardia also occurred more frequently and was significantly associated with composite adverse neonatal outcomes. Persistent fetal tachycardia is a result of maternal pyrexia, fetal inflammatory activation, diminished placental oxygen exchange and/or early fetal compromise. Fetal tachycardia had a higher rate of adverse outcomes which indicates fetal heart rate should be considered an important warning sign. Therefore, careful intrapartum monitoring and timely obstetric intervention could help minimise the risk of deterioration in the neonate [13-15].

The biochemical results also confirmed the existence of a strong inflammatory response. Leucocytes, C-reactive protein concentration, procalcitonin level and serum lactate level were significantly higher in mothers whose neonates developed adverse outcomes than in mothers whose neonates developed favourable outcomes. The levels of leukocytosis and C-reactive protein are raised during infection but are also raised during labour and other inflammatory processes and can be less specific. However, procalcitonin levels raised and serum lactate levels elevated were not found to be modifier-independent determinants of poor neonatal outcomes in the adjusted analysis. Bacterial inflammatory activity may be better reflected by a rise in procalcitonin, and impaired tissue perfusion or more severe systemic infection may be reflected by an increase in lactate. The use of multiple markers together may therefore more accurately determine a state of health or disease than a single laboratory parameter [16-18].

One other important determinant of poor neonatal outcome was prolonged rupture of membranes. Mothers with membranes that had been ruptured for more than 18 hours had a significantly increased risk of adverse outcomes. Any prolonged rupture of the membrane can allow ascending microorganisms to contaminate the amniotic cavity, thus raising the risk of placental inflammation, fetal infection and early onset neonatal sepsis. Prematurity and low birth weight were also common in this study, and likely played a role in the greater incidence of respiratory distress, NICU

admission and neonatal infection. The only variables that were significantly associated with a poor neonatal prognosis were the combined effect of infection and fetal immaturity (as indicated by the regression analysis), which showed that the longer the gestation period, the more likely the baby was to survive [19, 20].

The study has several limitations. It was carried out in one institution with a relatively small sample, and may not be representative. Antibiotic use prior to sample collection may have affected some laboratory markers and Culture results. Not all participants had histopathological confirmation of chorioamnionitis nor had placental cultures performed, and there were no long-term assessments of neonatal neurodevelopment. In spite of these constraints, clinical predispositions for neonatal complications are found in the mother. Further multi-centre studies are warranted to assess the combined diagnostic and prognostic value of maternal temperature, fetal heart rate, procalcitonin, lactate and inflammatory indices, as well as to investigate neonatal outcomes after the first few days.

CONCLUSION

Chorioamnionitis was associated with significant maternal physiological and biochemical abnormalities and a considerable burden of neonatal morbidity and mortality. Prolonged rupture of membranes, fetal tachycardia, raised maternal procalcitonin, elevated serum lactate, and lower gestational age were important predictors of adverse neonatal outcomes. Early recognition of these high-risk features, prompt administration of antibiotics, close fetal surveillance, timely delivery when clinically indicated, and immediate neonatal assessment may improve maternal and neonatal outcomes. Combined clinical and laboratory evaluation appears more useful than reliance on any single marker for identifying pregnancies at greatest risk.

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