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Association of Pathological Cardiotocograph with Adverse Fetal Outcomes

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ABSTRACT

Background: Cardiotocography (CTG) is a standard, non-invasive mode of intrapartum monitoring of the fetus. Pathological CTG patterns are consistently correlated with perinatal morbidity/mortality and fetal distress. While the clinical significance of CTG has been validated, local studies examining the association are not common, particularly in resource-poor areas. **Objective:** To compare the frequency of adverse fetal outcomes in pathological and normal cardiotocograph. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study.** This study was conducted from July to December 2024 at the Department of Obstetrics and Gynaecology, POF Hospital, Wah Cantt. **Methodology:** A total of 378 women in labor with singleton pregnancies beyond 36 weeks were enrolled. CTG findings were categorized as normal or pathological based on predefined criteria. Adverse fetal outcomes assessed included 5-minute Apgar score <5, neonatal resuscitation, and early neonatal death (within 48 hours). **Results:** Pathological CTG was observed in 37 women (9.8%). Among them, 15 (40.5%) neonates had low Apgar scores, 11 (29.7%) required resuscitation, and 14 (37.8%) experienced early neonatal death. In contrast, among 341 women (90.2%) with normal CTG, the rates were significantly lower: low Apgar in 47 (13.8%), resuscitation in 35 (10.3%), and early neonatal death in 4 (1.2%) ($p < 0.001$). **Conclusion:** Pathological cardiotocography is strongly associated with adverse fetal outcomes and should be considered a critical component of intrapartum monitoring, especially for high-risk pregnancies.

INTRODUCTION

Cardiotocography (CTG) refers to a real-time, non-invasive monitoring technique for the assessment of the health of the fetus during pregnancy and labor.[1] CTG, simultaneously, plots the uterine contraction and the fetal heart rate (FHR) and overlies them to observe how the fetus, particularly under stress during labor, experiences the intrauterine milieu.[2] CTG proves to be a useful aid to the recognition of signs of fetal distress, enabling proper clinical intervention at the correct moment to reduce perinatal morbidity and mortality.[3] CTG interpretation, while often broadly classified as normal, suspicious, and pathological patterns, becomes an important factor as part of the decision of the obstetrician.[4]

A pathological cardiotocograph demonstrates abnormal features, which are associated with compromised fetal oxygenation.[5] Such a pattern includes persistent bradycardia, reduced or absent baseline activity, recurrent late deceleration, and prolonged deceleration.[6] Such features are associated with fetal acidosis/hypoxia and need immediate evaluation and possibly intervention, e.g., expedited delivery. While CTG is highly sensitive, it can be relatively nonspecific, which can lead to overdiagnosis and unjustified cesarean delivery, but a certainly pathological CTG remains a prominent feature of adverse perinatal outcome.[7]

Poor perinatal outcomes associated with abnormal CTG are low Apgar scores, reflecting a compromised physical condition of the newborn at birth.[8] An Apgar score of less than 7 at 5 minutes has been utilized as a marker of neonatal distress and intrapartum hypoxia.[9] Such newborns may have reduced muscle tone, depressed reflexes, or weak respiratory effort, all of which may be the consequence of prolonged or severe labor-related oxygen lack.[10] Abnormal CTG allow the clinician to foretell such scenarios and prepare for speedy delivery and postnatal therapy, yet despite such prompt therapy, a number of such neonates might still have sequelae as the consequences of the severity or longer periods of hypoxic events.[11]

In addition to low Apgar scores, neonates following a pathological CTG may require resuscitation urgently, e.g., with positive pressure ventilation or advanced life support methods at the delivery room.[12] Such a situation often requires NICU admission for ongoing observation and management.[12] Unfortunately, rarely involving severe prolongation of intrauterine hypoxia or occult compromise, neonatal death may follow despite

appropriate clinical management.[13] This is a reminder of the need for appropriate interpretation and prompt action with CTG findings to reduce perinatal morbidity and mortality.[13] For this reason, ongoing education in the interpretation of CTG and the use of adjunct devices such as fetal scalp pH or analysis of the ST segment could further enhance the use of CTG to inform obstetric practice.[13]

A study conducted in Karachi reported that 6.35% of neonates were delivered with pathological cardiotocography, among whom 18.18% exhibited a low APGAR score.[14] Similarly, research by Waheed N et al. documented a low APGAR score in 20.51% of cases with pathological CTG.[15] Salahuddin N et al. also demonstrated a notable association between pathological CTG and adverse neonatal outcomes, with a low APGAR score observed in 18.9% of cases compared to 9.7% in those with normal CTG. Additionally, the need for neonatal resuscitation was higher in the pathological CTG group (15.6% vs. 7.3%), and the rate of early neonatal deaths was significantly greater (13.9% vs. 4.1%).[16]

Despite the accessible evidence linking pathological cardiotocography to grim fetal outcomes, local data are limited, particularly for resource-poor regions where immediate treatments are a need. Differences in practices of obstetric practices, neonatal facility availability, and CTG interpretation expertise also need local studies. By conducting the current work, we are able to establish the predictive potential of pathological CTG among our population and guide clinical practice toward improvement of perinatal health. We are also able to contribute to the standardization of intrapartum monitoring and immediate management of fetal distress protocols.

METHODOLOGY

This descriptive study was conducted over a six-month period, from July to December 2024, in the Department of Obstetrics and Gynaecology at POF Hospital, Wah Cantt. A total of 378 women in labour were enrolled. The sample size was calculated using the WHO software, employing a 95% confidence interval and a 2% margin of error, based on an anticipated early neonatal death rate of 4.1%.[16]

Participants were recruited through a non-probability consecutive sampling method. Women aged 18 to 45 years with a singleton pregnancy beyond 36 weeks of gestation, as confirmed on ultrasound, and who were in active labour—defined by regular uterine contractions occurring at least twice every 10 minutes, each lasting 30 seconds or more and resulting in cervical dilation beyond 4 cm—were considered eligible for inclusion. All parity groups were included. Women were excluded if they had fetuses with known congenital anomalies, or if they had a documented history of cardiovascular disease, hypertension, or diabetes mellitus.

After obtaining ethical clearance, written informed consent was secured from each participant, with assurance of confidentiality and no risk associated with participation. Relevant demographic and clinical data, including age, parity, gestational age, residential and socioeconomic status, and cardiotocograph (CTG) classification, were recorded.

CTG monitoring was performed intermittently during labour, with each session lasting approximately 20 minutes. A tracing was classified as normal when baseline fetal heart rate (FHR), beat-to-beat variability, presence of accelerations, and absence of decelerations were all reassuring. A CTG was categorized as pathological when there were late decelerations in at least 30% of contractions, a silent trace for 30 minutes, prolonged variable decelerations (≤80 beats/min lasting ≥60 seconds), sustained bradycardia (<100 beats/min for ≥3 minutes), or tachycardia (≥180 beats/min for ≥30 minutes).

Newborns were assessed immediately after delivery for adverse outcomes. A 5-minute Apgar score below 5 was considered low. Neonatal resuscitation was deemed necessary if the newborn exhibited signs such as rapid breathing above 60 breaths per minute, oxygen saturation below 90%, arterial oxygen pressure under 50 mmHg, or carbon dioxide levels exceeding 60 mmHg. Early neonatal death was defined as death occurring within the first 48 hours of life.

All data were entered and analyzed using SPSS version 29. Quantitative variables, such as maternal age, gestational age, and parity, were summarized using means and standard deviations or medians and interquartile ranges, depending on the data distribution, which was tested using the Shapiro-Wilk test. Categorical variables, including CTG type and each adverse fetal outcome, were reported as frequencies and percentages. The association between CTG findings and adverse outcomes was evaluated using chi-square or Fisher's exact test, with a p-value of ≤0.05 considered statistically significant. Stratification was carried out for maternal age, gestational age, parity, socioeconomic and residential status, followed by post-stratification statistical testing to assess effect modification.

RESULTS

In this study assessing the association of pathological cardiotocograph (CTG) with adverse fetal outcomes, a total of 378 patients were evaluated with a mean maternal age of 29.87 ± 7.70 years, mean gestational age of 37.56 ± 1.50 weeks, and mean parity of 1.81 ± 1.33. Among the participants, 37 (9.8%) had pathological CTG while 341 (90.2%) had normal CTG findings (as shown in Table 1).

Table 1

Patient Demographics

Demographics		Mean ± SD
Age (years)		29.87±7.70
Gestational Age (weeks)		37.56±1.50
Parity		1.81±1.33
CTG Type	Pathological n (%)	37 (9.8%)
	Normal n (%)	341 (90.2%)

Adverse fetal outcomes were reported as follows: low Apgar scores in 62 neonates (16.4%), resuscitation required in 46 cases (12.2%), and early neonatal deaths in 18 cases (4.8%) (as shown in Table 2)

Table 2
Frequency of Adverse Fetal Outcomes

Adverse fetal outcomes	Frequency	% age
Low Apgar	62	16.40%
Resuscitation	46	12.20%
Early Neonatal Death	18	4.80%

Pathological CTG showed a statistically significant association with all adverse outcomes. Low Apgar scores occurred in 15 (40.5%) neonates with pathological CTG compared to 47 (13.8%) with normal CTG ($p < 0.001$). Similarly, neonatal resuscitation was needed in 11 (29.7%) cases with pathological CTG versus 35 (10.3%) with normal CTG ($p < 0.001$). Early neonatal death was reported in 14 (37.8%) newborns with pathological CTG, compared to only 4 (1.2%) in those with normal CTG ($p < 0.001$, Fisher Exact Test) (as shown in Table 3)

Table 3
Comparison of CTG Type with Adverse Fetal Outcomes

	Low Apgar		P-value
CTG Type	Yes n(%)	No n(%)	
Pathological	15 (40.5%)	22 (59.5%)	<0.001
Normal	47 (13.8%)	294 (86.2%)	
Resuscitation			
CTG Type	Yes n(%)	No n(%)	
Pathological	11 (29.7%)	26 (70.3%)	<0.001
Normal	35 (10.3%)	306 (89.7%)	
Early Neonatal Death			
CTG Type	Yes n(%)	No n(%)	
Pathological	14 (37.8%)	23 (62.2%)	<0.001*
Normal	4 (1.2%)	337 (98.8%)	

*Fischer Exact Test

Stratified analysis of maternal factors further supported these findings. Low Apgar scores were significantly more frequent among women aged ≤30 years (47/199; 23.6%) compared to those >30 years (15/179; 8.4%) ($p < 0.001$), with no significant difference based on gestational age ($p = 0.568$), but a significant association with parity ≤2 (54/287; 18.8%) versus >2 (8/91; 8.8%) ($p = 0.024$). Resuscitation was also more common among younger mothers aged ≤30 years (34/199; 17.1%) versus >30 years (12/179; 6.7%) ($p = 0.002$), and in parity ≤2 (42/287; 14.6%) compared to >2 (4/91; 4.4%) ($p = 0.009$, Fisher Exact Test), though not significantly different by gestational age group ($p = 0.334$). Early neonatal deaths did not differ significantly by age group ($p = 0.800$) or gestational age group ($p = 1.000$), but were more frequent in women with parity ≤2 (18/287; 6.3%) compared to none in those with parity >2 (0/91; 0.0%) ($p = 0.019$, Fisher Exact Test) (as shown in Table 4 and Graph 1)

Graph 1
Stratification of Maternal Factors with Adverse Fetal Outcomes

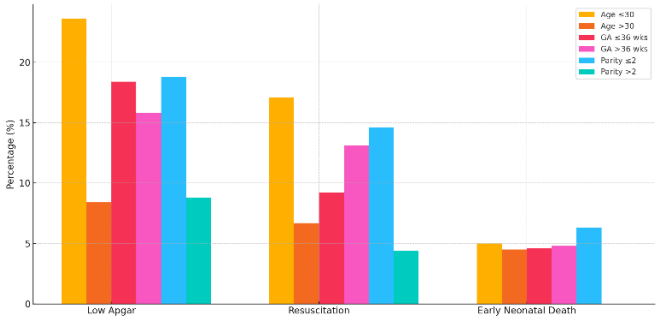

Table 4
Association of Maternal Factors with Adverse Fetal Outcomes

Maternal Factors		Low Apgar score		P-value
		Yes n(%)	No n(%)	
Age Group	≤30	47 (23.6%)	152 (76.4%)	<0.001
	>30	15 (8.4%)	164 (91.6%)	
Gestational Age Group	≤36	16 (18.4%)	71 (81.6%)	0.568
	>36	46 (15.8%)	245 (84.2%)	
Parity Group	≤2	54 (18.8%)	233 (81.2%)	0.024
	>2	8 (8.8%)	83 (91.2%)	
Resuscitation				
		Yes n(%)	No n(%)	
Age Group	≤30	34 (17.1%)	165 (82.9%)	0.002
	>30	12 (6.7%)	167 (93.3%)	
Gestational Age Group	≤36	8 (9.2%)	79 (90.8%)	0.334
	>36	38 (13.1%)	253 (86.9%)	
Parity Group	≤2	42 (14.6%)	245 (85.4%)	0.009*
	>2	4 (4.4%)	87 (95.6%)	
Early Neonatal Death				
		Yes n(%)	No n(%)	
Age Group	≤30	10 (5.0%)	189 (95.0%)	0.800
	>30	8 (4.5%)	171 (95.5%)	
Gestational Age Group	≤36	4 (4.6%)	83 (95.4%)	1.000*
	>36	14 (4.8%)	277 (95.2%)	
Parity Group	≤2	18 (6.3%)	269 (93.7%)	0.019*
	>2	0 (0.0%)	91 (100.0%)	

*Fischer Exact Test

DISCUSSION

The finding of this analysis strongly supports a significant association of pathological cardiotocography (CTG) with undesirable fetal outcomes, including low Apgar scores, neonatal resuscitation, and early neonatal mortality. The higher prevalence among cases with pathological CTG suggests that the abnormal fetal heart patterns are likely manifestations of pre-existing fetal distress, presumably a result of compromised placental oxygenation or uteroplacental insufficiency. The higher frequency of undesirable outcomes among younger mothers (≤30 years) and lower parity (≤2) may reflect relative labor inexperience, suboptimal maternal adaptation, or lower uterine and placental maturity. Although the association with gestational age was not statistically significant, the tendency for a higher frequency of undesirable outcomes at ≤36 weeks probably reflects the lower neonatal physiological stores and immature organs. These establish the clinical significance of CTG as an effective antecedent indicator of fetal compromise and validate the need for incorporating maternal demographic and obstetric attributes as risk factors as part of the intrapartum monitoring process.

Among adverse outcomes, low Apgar scores occurred in 16.4%, neonatal resuscitation was required in 12.2%, and early neonatal death occurred in 4.8%. A significantly higher proportion of these outcomes occurred in the pathological CTG group: 40.5% had low Apgar scores (vs 13.8% in normal CTG), 29.7% required resuscitation (vs 10.3%), and 37.8% resulted in early neonatal death (vs 1.2%) ($p < 0.001$ for all). These findings strongly align with those of Salahuddin et al. [16], who also reported a significantly higher frequency of low Apgar scores at both 1 and 5 minutes in the abnormal CTG group (64.4% vs 37.9% and 46.7% vs 28.2%, respectively), along with

increased need for resuscitation and NICU admissions. The greater percentage of poor outcomes in Salahuddin's study may be attributed to the inclusion of only high-risk pregnancies, compared to our broader inclusion criteria.

Similar findings were observed by Bhuvaneshwari and Rekha [17], who reported that 48.7% of neonates with pathological CTG had an Apgar score of 4–6 at 1 minute compared to only 15.2% in the reactive CTG group ($p < 0.001$), and 52% required resuscitation versus 4.5% of the reactive CTG group. These rates are notably higher than in our cohort, likely due to their specific focus on term pregnancies presenting with reduced fetal movements—an indicator of compromised fetal well-being. In contrast, Mustafa [18] also demonstrated significantly worse outcomes in pathological intrapartum CTG, with 63.8% requiring resuscitation and high NICU admissions (79.1%). The extremely poor outcomes in the intrapartum CTG group compared to antenatal CTG in that study further emphasize the timing and acuity of fetal compromise.

Our study reported early neonatal death in 37.8% of the pathological CTG group, a finding that is consistent with Mustafa's [18] observation of 6.9% neonatal mortality in pathological intrapartum CTG and that of Naeem et al. [20], who reported stillbirth/neonatal mortality in 10.7% of cases with pathological CTG versus none in the normal CTG group ($p = 0.003$). Although our early neonatal death rate is higher, differences in clinical protocols, timely access to emergency obstetric care, and variations in defining early neonatal death versus stillbirth may explain the discrepancy. Moreover, the findings of Naeem et al. [19] further support our conclusion that pathological CTG is significantly associated with increased NICU admission and adverse perinatal outcomes.

Khan et al. [20] found a 16.7% rate of Apgar <7 at 5 minutes and NICU admission in 20% of abnormal CTG cases, which is similar to our 16.4% rate of low Apgar at 1 minute but lower than our NICU proxy (resuscitation 12.2%). Their study, focused solely on abnormal CTG at term, reinforces the link between pathological CTG and adverse outcomes, though the severity of compromise in our sample appears more pronounced, possibly due to higher inclusion of compromised cases or delays in intervention.

Waheed et al. [15] reported a very high frequency of Apgar <7 at 1 minute in 61.5% of pathological CTG cases and 20.5% persistence at 5 minutes. Their findings also align with ours and reinforce the predictive nature of pathological CTG for low Apgar scores, though unlike our study, their stratified analysis did not find significant associations with maternal age, parity, or gestational age. This discrepancy could be due to differences in population characteristics or study design, as our larger sample size allowed greater power for subgroup comparisons.

Khalid et al. [21], in a large series of 240 women with pathological CTG, found 43.8% had a 5-minute Apgar ≤7. This is higher than our overall rate but corresponds closely with the 40.5% low Apgar rate among our pathological CTG cases. Their study noted that adverse outcomes were not exclusively tied to pathological CTG, suggesting variability in fetal response to distress and the limitations of CTG interpretation, a point also reflected in our results and supporting the need for adjunctive diagnostic modalities.

With the foregoing finding, the reproducible linkage of abnormal cardiotocograph with undesirable endpoints by various studies, such as the current, lends credence to the use of cardiotocograph as a non-invasive screen during the second stage of labor. But variation in sensitivity, interpretation, and therapeutic thresholds emphasizes the need for situational decision-making while treating the abnormal CTG finding. Though our result does not contradict previous records, the differential frequency of endpoints highlights the influence of institutional practices, clinician expertise, and interval of intervention as predictors of the fate of the fetus.

This study, however, has several limitations. Being a single-center study, the finding might not become applicable to the other health facilities which have different patient population or different treatment approaches. Additionally, the adoption of CTG as the sole monitoring device without employing adjunctive tests such as fetal scalp blood sampling or Doppler studies could have led to overdiagnosis or misassessment of fetal distress. Also, interobserver variation when interpreting CTG was not ruled out, hence possibly introducing the issue of classification bias.

CONCLUSION

Our study has identified that CTG is highly related to undesirable fetal outcomes, like decreased Apgar scores, increased need for neonatal resuscitation, and neonatal mortality during the first neonatal interval. Our result gives credence to the relevance of CTG clinically as a useful intrapartum monitoring tool for the prediction of fetal compromise in the perinatal interval. Early detection and relevant intervening management after the onset of pathological CTG patterns can contribute to improved perinatal survival, specifically higher risk pregnancies.

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