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Cerebral palsy risk in relation to parental age: insights from a matched case-control study

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Abstract

Background Cerebral palsy, a prevalent childhood physical disability, exhibits complex multifactorial causes. This case-control study explores the role of parental age in cerebral palsy risk and associated patterns.

Methods A case-control study comparing children with cerebral palsy at ages 3 months to 3 years with age-matched healthy control subjects was conducted between January 2022 and August 2023 at Federal Medical Centre, Abeokuta, Nigeria. Cases were recruited from the pediatric physiotherapy outpatient clinic, while controls were selected from pediatric and children emergency wards. Data were collected retrospectively from electronic medical records. Risk factors were evaluated using conditional logistic regression models.

Results We studied 134 subjects with cerebral palsy and 134 age-matched control subjects. Significant risk factors for cerebral palsy included the maternal age 35–39 years (odds ratio 2.16, $P < 0.005$), maternal age ≥ 40 years (odds ratio 3.83, $P < 0.005$), paternal age 41–45 (odds ratio 2.00, $P < 0.05$), paternal age ≥ 46 (odds ratio 6.80, $P < 0.05$), primiparous (odds ratio 2.2, $P < 0.05$), paternal low income (odds ratio 2.49, $P < 0.05$), paternal primary education (odds ratio 24.61, $P < 0.05$), and maternal primary education (odds ratio 2.39, $P < 0.05$).

Conclusions This research contributes to our understanding of parental age as risk factors associated with cerebral palsy in children. The results also underscore the importance of demographic and socioeconomic factors, especially father's level of income, maternal parity, and parental education. These findings can guide future research and public health interventions aimed at reducing the burden of cerebral palsy.

Keywords Parental age, Cerebral palsy, Case control, Risk factors

Introduction

Cerebral palsy (CP) is the most common physical disability in childhood, with a prevalence of around 2.11 per 1000 live births globally [1]. The causes of CP are complex and multifactorial, with genetic and environmental factors playing a role. CP is a group of permanent movement disorders that affects a person's ability to move and maintain balance and posture. CP is caused by damage to the developing brain, which may occur before, during, or shortly after birth. This syndrome is the first cause of motor disability and the second cause of neurodevelopmental abnormalities in children after intellectual disability [1]. The exact causes of CP are still unknown, but research suggests that environmental and genetic factors may play a role. One of the potential environmental factors that have been linked to CP is parental age at the time of conception [2, 3]. There is increasing evidence to suggest

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that advanced parental age, particularly maternal age, is associated with an increased risk of CP in offspring. However, the relationship between parental age at conception and CP risk is not well understood, and few studies have explored the potential role of parental age patterns, which considers the age of both parents at the time of conception [4].

Parental age is a well-established risk factor for several adverse outcomes, including birth defects, intellectual disability, and autism spectrum disorders [5, 6]. It has been shown to have a significant impact on child health outcomes, including CP. Some studies have reported an increased risk of CP in children born to older parents [4, 7–9], while others have found no association [10]. However, the relationship between parental age patterns and CP risk remains unclear. Understanding these associations can provide valuable insights for both clinicians and researchers seeking to better identify risk factors and enhance prevention and management strategies for CP. This case-control study, which matches cases of CP with suitable controls, sets out to explore the patterns of parental age concerning the risk of CP. By scrutinizing data from individuals with CP and comparing it to data from matched individuals without the condition, we aim to discern whether advanced maternal or paternal age is correlated with a higher risk of CP. Additionally, we seek to ascertain if there are specific age thresholds at which this risk is more pronounced. The study's intent is to contribute to the broader understanding of risk factors for CP, offering insights that can guide families and healthcare providers in counseling and prevention strategies.

Methods

Study setting design

This study was conducted at the Federal Medical Centre, Abeokuta, Nigeria (FMCA), in its pediatric physiotherapy outpatient clinic, covering the period from January 2022 to August 2023. As the largest tertiary hospital in Ogun State, FMCA plays a crucial role in providing healthcare services to patients from neighboring towns in the region. The study's ethical considerations were thoroughly addressed, as it was officially approved by the Research Ethics Committee of the Federal Medical Center, Abeokuta, Nigeria (FMCA/470/HREC). Utilizing a matched case-control study design, we aimed to investigate the potential relationship between parental age and the risk of CP. The study participants were selected from our extensive electronic medical records system (EMR), which allowed for a comprehensive and retrospective identification of both cases, consisting of pediatric patients with CP, and control subjects, all meeting the specific study criteria.

Criteria for inclusion and the selection of both cases and controls

In our study, we identified cases as children with confirmed diagnoses of CP according to the International Classification of Diseases (ICD-10) (G.80.0-9) (WHO, 2016) and prior to study enrollment, while controls were selected from pediatric ward and children emergency ward. These cases were selected from the pediatric outpatient clinic within the physiotherapy department at FMCA.

Exclusion criteria for cases and controls

We implemented specific exclusion criteria, excluding children with postnatal CP (Fig 1). Furthermore, we excluded individuals with incomplete or unreliable medical records to maintain data accuracy.

Matching

This hospital-based case-control study was matched by patient's age. We employed frequency matching based on patient's age group to ensure a balanced distribution of age in both the case and control groups. Controls were meticulously selected to replicate this age distribution. Matching in case-control studies is done to reduce the potential for confounding variables, thereby strengthening the validity of study findings [11]. It ensures that cases and controls are more comparable in terms of specific characteristics, such as age, gender, or other potential confounders, which allows for a more accurate assessment of the exposure-disease relationship [12]. By matching, we enhanced the ability to detect true associations between exposures (parental age) and outcome (CP) while minimizing the influence of variables that could distort the results, leading to more reliable and interpretable research findings.

Sample size

The sample size calculation was based on an 18% prevalence of older women with children having cerebral palsy [13]. To achieve a statistical power of 80% for detecting an odds ratio (OR) of 2.0 with a 95% confidence interval, we adopted a 1:1 ratio of cases to controls. Consequently, the determined sample size is 268 patients, comprising 134 cases and 134 controls.

Study variables

The dependent variable in this study was the clinical diagnosis of CP, whereas the independent variables encompassed sociodemographic characteristics (such as patient's age and sex) and parental characteristics

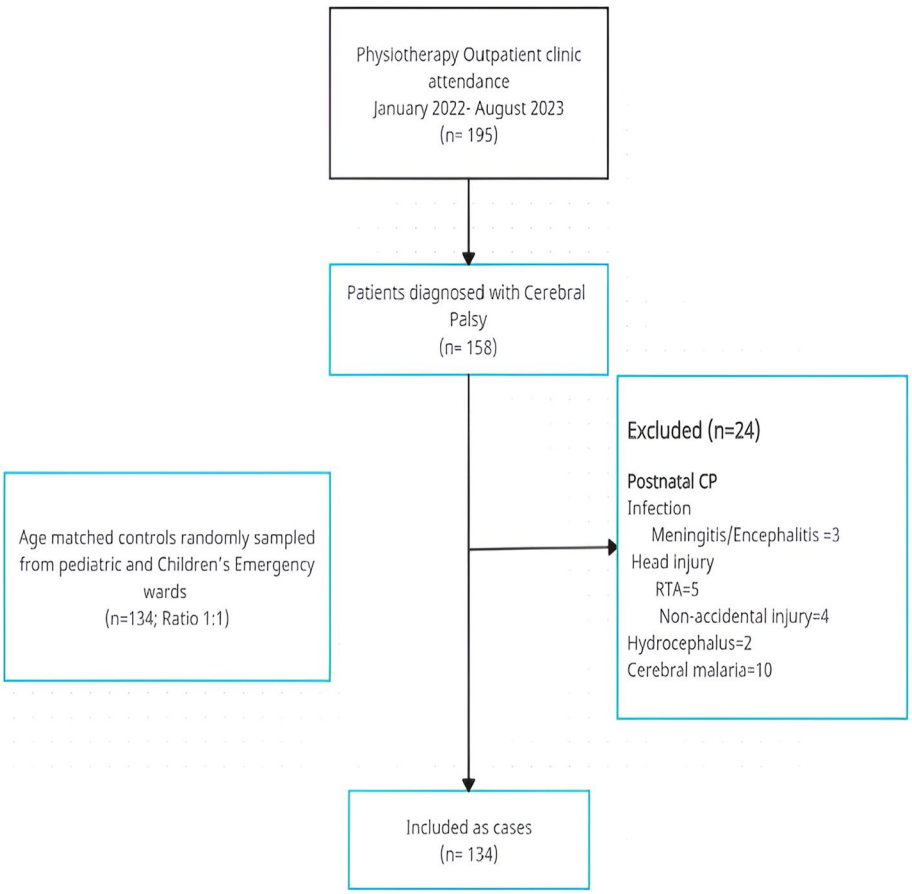

Fig. 1 Flowchart of cases and controls

(including age, religion, level of income, and education status).

Statistical analysis

Data were collected, revised, coded, and entered to the Statistical Package for Social Science (IBM SPSS) version 26. The quantitative data were presented as mean and standard deviations, while qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done using chi-square test. The confidence interval was set to 95%, and the margin of error accepted was set to 5%. The *P*-value was considered significant as the following: *P*-value < 0.05, significant (S). In bivariate analysis, odds ratio with 95% confidence interval (CI) was calculated. Risk factors with a *P*-value of ≤ 0.05 in the bivariate analysis were retained in the multivariate analysis. The multivariate analysis was conducted to verify the associations between different independent variables and CP and interpreted as significant at a *P*-value of < 0.05 and 95% CI.

Results

This case-control study was conducted on two-hundred and sixty-eight (268) children. Among 134 cases and 134 controls, 167 (62.3%) were males, while 101 (37.7%) were females. There were 90 (33.6%) of the males who had CP, while only 44 (16.4%) were females. The child's age distribution among cases is in Table 1. A higher proportion of the children were first born in cases group (24.3%) and controls (20.5%).

Concerning the parental demographics, majority of the mothers (35.1%) were in the age group of 30–34 years contributing 19.4% among cases and 15.7% among controls. This study also reported a higher percentage for the fathers (32.5%) between the ages of 31–35, with 17.2% attributed to cases and 15.3% to controls. The age groups 31–35 also had 32.1% for the mean parental age which was calculated by summing up both father and mother age and divided by two. Multiparous mothers (60.8%) reported a higher frequency as compared to primiparous mothers (39.2%). More than 65% and 70% of the fathers and mothers were low-income earners respectively. Also concerning level of education, about 171 (63.8%) of the

Table 1 Child, maternal, and parental characteristics with and without CP

Variables	Children without CP (N1 = 134) n1 (%)	Children with CP (N2 = 134) n2 (%)	χ^2	*p*-value
Child's age (months)			-	-
< 6	3 (2.2)	3 (2.2)		
6–12	20 (14.9)	20 (14.9)		
12–24	44 (32.9)	44 (32.9)		
24–36	59 (44.0)	59 (44.0)		
36 and above	8 (6.0)	8 (6.0)		
Child gender			8.011	0.005
Male	57 (21.3)	35 (13.1)		
Female	77 (28.7)	99 (36.9)		
Child position in family			2.232	0.508
1	55 (20.5)	65 (24.3)		
2	36 (13.4)	30 (11.2)		
3	27 (10.1)	28 (10.4)		
4 and above	16 (6.0)	11 (4.1)		
Maternal age (years)			11.036	0.026
20–24	23 (8.6)	15 (5.6)		
25–29	43 (16.0)	26 (9.7)		
30–34	42 (15.7)	52 (19.4)		
35–39	22 (8.2)	31 (11.6)		
≥ 40	4 (1.5)	10 (3.7)		
Mean ± SD	31.1 ± 5.8	29.6 ± 5.7		
Parity			9.787	0.002
Multiparous	94 (35.1)	69 (25.7)		
Primiparous	40 (14.9)	65 (24.3)		
Paternal age			13.243	0.021
≤25	4 (1.5)	4 (1.5)		
26-30	29 (10.8)	11 (4.1)		
31-35	41 (15.3)	46 (17.2)		
36-40	44 (16.4)	42 (15.7)		
41-45	11 (4.1)	22 (8.2)		
≥ 46	5 (1.9)	9 (3.4)		
Mean ± SD	36.3 ± 6.5	34.9 ± 6.4		
Mean parental age			12.191	0.016
Mean ± SD	33.9 ± 5.9	32.6 ± 5.7		
Religion			0.982	0.322
Christianity	82 (30.6)	80 (31.0)		
Islamic	52 (19.4)	60 (22.4)		
Parity			9.877	0.002
Multiparous	69 (25.7)	94 (35.1)		
Primiparous	65 (24.3)	40 (14.9)		
Father's level of income			5.976	0.049
Low	99 (36.9)	81 (30.2)		
Medium	31 (11.6)	44 (16.4)		
High	4 (1.5)	9 (3.4)		
Mother's level of income			3.428	0.180
Low	95 (35.4)	96 (35.8)		
Medium	22 (8.2)	29 (10.8)		
High	17 (6.3)	9 (3.4)		

Table 1 (continued)

Variables	Children without CP (N1 = 134) n1 (%)	Children with CP (N2 = 134) n2 (%)	χ^2	*p*-value
Father's level of education			40.525	0.000
Primary	4 (1.5)	26 (9.7)		
Secondary	77 (28.7)	94 (35.1)		
Tertiary	53 (19.8)	14 (5.2)		
Mother's level of education			7.606	0.022
Primary	17 (6.3)	17 (6.3)		
Secondary	80 (29.9)	98 (36.6)		
Tertiary	37 (13.8)	19 (7.1)		

CP cerebral palsy: χ^2 chi-square: *p*-value level of significance; *SD* standard deviation

fathers and 178 (66.4%) of the mothers had secondary education respectively. These findings are summarized in Table 1.

Bivariate analysis

According to Table 1, there was statistically significant differences according to the bivariate analysis between cases and control group, in respect of maternal age ($p = 0.026$), parity ($p = 0.002$), paternal age ($p = 0.021$), mean parental age ($p = 0.016$), father's level of income ($p = 0.049$), father's level of education ($p = 0.000$), and mother's level of education ($p = 0.022$).

Univariate analysis

Table 2 shows the crude (univariate) and adjusted (multivariate) odds ratios and 95% confidence intervals for CP. Compared with offspring of mothers between 20 and 24 years age, CP risk was 7% lower among children of mothers between 25 and 29 years of age (*COR*: 0.93, 95% confidence interval (CI), 0.41 to 2.09) and 89% higher among children of mothers within the 30–34 years of age (*COR*: 1.89, 95% confidence interval (CI), 0.88 to 4.09). Also relative to mothers between 20 and 24 years of age, mothers between 35 and 39 years of age and above 40 years of age were two times and three times as likely to give birth to a child with CP respectively. Primiparous mothers were significantly more likely to give birth to a child with CP ($p = 0.02$).

Concerning father's age, CP risk was 62% lower among children of fathers between 26 and 30 years of age as compared to fathers less than 25 years of age. Fathers whose age fall between 31 and 35 years were 12% more likely to have children with CP, and those within 36–40 years of age were 5% less also likely. The odds of having a child with CP is higher among fathers between 41 and 45 years of age as compared to fathers less than 25 years by two times. Fathers who were greater than 46 years were

Table 2 Conditional logistic regression analysis to estimate the odds (odd ratio, OR) of CP

	COR (95% *CI*)	AOR (95% *CI*)
Maternal age		
20–24	1	1
25–29	0.93 (0.41–2.09)	3.36 (0.72–15.71)*
30–34	1.89 (0.88–4.09)	8.14 (1.39–47.29)*
35–39	2.16 (0.92–5.05)*	16.66 (2.23–121.69)*
≥ 40	3.83 (1.01–14.49)*	73.33 (4.11–1307.11)*
Parity		
Multiparous	1	1
Primiparous	2.2 (1.34–3.66)*	3.76 (1.81–7.81)*
Paternal age		
≤ 25	1	
26–30	0.38 (0.08–1.78)*	1.73 (0.233–12.86)
31–35	1.12 (0.26–4.78)	10.31 (1.19–88.88)*
36–40	0.95 (0.22–4.07)	5.63 (0.67–47.29)
41–45	2.00 (0.42–9.55)	6.29 (0.87–121.42)*
≥ 46	6.80 (3.31–10.52)	10.30 (0.22–171.15)
Joint age	1.04 (0.99–1.09)*	0.85 (0.72–1.01)*
Fathers level of income		
High	1	1
Medium	1.91 (0.81–4.50)	1.58 (0.69–3.59)
Low	2.49 (0.94–6.63)*	0.39 (0.14–1.13)*
Father's level of education		
Primary	24.61 (7.37–82.21)*	0.29 (0.09–0.95)*
Secondary	4.62 (2.39–8.96)*	0.1 (0.10–0.17)*
Tertiary	1	1
Mother's level of education		
Primary	2.39 (1.27–4.47)*	5.84 (3.21–9.88)
Secondary	1.95 (0.82–4.65)	1.87 (0.75–4.71)
Tertiary	1	1

CP, cerebral palsy; *COR*, crude odds ratio; *AOR*, adjusted odds ratio: *$p < 0.05$

six times more likely to have children with CP. This study also puts into consideration the mean of the father and mother's age (mean FM age). For a unit increase in mean FM age, the odds of CP were 1.04 times.

As regards the level of income, fathers with medium level of income were 1.91 times more likely to have children with CP as compared to those with high level of income. The odd of having children with CP was two times higher in fathers with low-income level compared to reference group. The odds of CP were higher in fathers with primary level of education (*COR*: 24.61, 95% confidence interval (CI), 7.37 to 82.21) and lesser in fathers with secondary level of education (*COR*: 4.62, 95% confidence interval (CI), 2.39 to 8.96). Mothers with secondary education were 2 times more likely to have children with CP, while those with primary education were 1.95 times more likely.

Multivariable analysis

In the fully adjusted model (Table 2), all maternal age groups, primiparous (*AOR*: 9.49, 95% *CI*: 1.31–68.88), unknown maternal education status (*AOR*: 18.64, 95% *CI*: 2.15–161.73), CNS infection in infancy (*AOR*: 0.02, 95% *CI*: 0–0.58), and neonatal admission (*AOR*: 0.13, 95% *CI*: 0.03–0.61) remained statistically significant. All maternal age groups were statistically significant with an increase in gradient risk of having children with CP. Mothers with advanced age were statistically more likely to have children with CP ($P < 0.05$). Fathers age between 31–35 years and 41–45 years of age were also more likely to have children with CP ($P < 0.05$). Mothers experiencing their first childbirth (AOR: 3.76, 95% CI, 1.81 to 7.81) were also found to have an increased likelihood of having children with cerebral palsy.

Discussion

CP is one of childhood neurological disorders that impose a significant burden on families as well as society, which needs continuous care and multiple financial resources. Paternal and maternal age have become one of the most discussed risk factors in paternal-maternal-fetal medicine, and its multidimensional impact on the developing fetus has made it one of the main topics of epidemiological research. This study aimed to evaluate the parental age patterns and risk for CP at FMCA. A hundred and thirty-four (134) children with CP as cases and 134 children without CP as controls of age up to 3 years were recruited and matched with child's age.

Among 134 cases, 65.7% of male children and 34.3% of female children were observed which shows the significant difference in the association between the genders. This implies a male predisposition among children with CP which is in congruent with other studies [14–16]. This correlation was explicitly supported in the research conducted by Hagberg et al. in 2001, which affirmed that the female gender exhibited a lower risk of developing CP compared to the male gender [16]. Likewise, a study supported this observation, revealing that the rate of CP per 1000 male births exceeds that among females by about 30% [17]. The male sex represents a risk factor for most neurodevelopmental disabilities such as intellectual disability, autism, attention deficit and hyperactivity disorder, and, mainly, CP, with males representing up to 70% of all affected children [18–20]. This biological vulnerability of the male sex in CP and other neurodevelopmental disorders has been explained by several factors such as a different brain organizations [14, 21], genetic predisposition [21, 22], and the influence of female hormones on a possible reduction of the consequences of brain damage [20]. Therefore, sex has been reported to have an effect on increasing the risk of CP [19], but it is not entirely clear whether it may also affect the severity of the outcome, the development of comorbidities, and the response to treatments. The male embryo is suggested at a greater risk of damage or death [23]. Additionally, stillbirth, premature birth, congenital deformities, perinatal brain damage, and neonatal adverse outcomes are more common in male [24].

Our study highlights the significant association between maternal age and CP, shedding light on the varying risk levels among different age groups. Among these groups, the risk is at its lowest between the ages of 25–29 and escalates dramatically for mothers aged 35 and above as compared to 20–24 years of age. This age-dependent risk factor mirrors the findings of many previous studies, affirming that maternal age plays a pivotal role in the occurrence of CP [24, 25]. The variations in CP risk among different maternal age groups can be attributed to several underlying factors. Firstly, it is well-established that as women grow older, the likelihood of experiencing various health complications during pregnancy increases [26–28]. These complications, such as gestational diabetes and hypertension, can have a direct impact on the developing fetus. Furthermore, older mothers may be more prone to certain genetic mutations that can contribute to CP in their offspring [29–31]. On the other end of the spectrum, younger mothers generally have a lower risk of CP, which can be attributed to factors such as their reduced likelihood of preexisting health conditions, lower rates of chronic diseases, and potentially healthier lifestyles. Additionally, early childbearing often coincides with a shorter reproductive history, potentially resulting in fewer opportunities for adverse events affecting fetal development [31]. The sharp increase in CP risk beyond the age of 35 is a subject of particular concern, as the biological processes in older mothers, such

as the progressive decrease in the number and quality of oocytes, along with the natural aging process of matured ova, can create an environment that is less favorable for the developing fetus [32]. The cumulative effect of these age-related factors significantly raises the likelihood of CP in babies born to mothers aged 35 and older.

Findings from the binary logistic regression also revealed that paternal age is a risk factor for CP. The findings suggest that younger fathers (26–30) might have a reduced risk, while older fathers (41–45 and ≥ 46) appear to face an elevated risk. Young fathers aged 26 to 30 might experience a relatively lower risk of CP. One possible explanation for this reduced risk among younger fathers is their overall better health and potentially more robust sperm quality [33]. These factors could contribute to a healthier fetal development, which, in turn, might lower the risk of CP. Conversely, the situation appears quite different for older fathers, particularly those aged 41 to 45 and those aged 46 or above. The data point towards an elevated risk of CP in these age groups. The reasons behind this increased risk are multifaceted. Advanced paternal age has been associated with a higher likelihood of genetic mutations, as older sperm is more prone to carrying genetic abnormalities [34–36]. These mutations can be a contributing factor to the development of CP in offspring. A study revealed a direct correlation between paternal age and decreased sperm quality and testicular function [36]. Genetic abnormalities, such as DNA mutations and chromosomal aneuploidies, and epigenetic modifications, such as the silencing of essential genes, have all been linked to the father's advancing years [37]. Advanced paternal age has demonstrated an impact on reproductive and fertility results, influencing the efficacy of procedures like in vitro fertilization (IVF) and intracytoplasmic sperm injection (ICSI), as well as the incidence of preterm births [36]. A study was conducted on extensive sequencing analysis and found that germline single base-pair alterations increased at a rate of about two base pairs per year with increasing paternal age [38].

Our study also revealed that primiparous mothers were two times more likely to give birth to children with CP. This heightened risk among first-time mothers could be attributed to their relative lack of experience and understanding when it comes to child care, which frequently leads to a multitude of stressors and difficulties. As Nan et al. (2020) have noted, this lack of experience can be particularly overwhelming for first-time mothers, potentially influencing the health outcomes of their children [39]. Furthermore, the unique challenges faced by primiparous mothers may extend to various aspects of child-rearing, from prenatal care to postnatal support, ultimately impacting their children's risk of developing CP. Also, first-time mothers often lack experience in child care and may not recognize early signs of developmental issues [40]. In their early journey into parenthood, these mothers may not be as skilled at deciphering the subtle cues, which refer to the small, often nonverbal signals or hints that babies give to express their needs, feelings, or discomfort [41]. These cues can include changes in facial expressions, body movements, or sounds that experienced parents may readily understand. This lack of experience can result in delayed detection of potential issues and hinder timely interventions. Based on our research, it was evident that fathers with lower income had a twofold increased likelihood of having children with CP. This observation can be elucidated by the cultural context in many African settings, where fathers often bear the role of primary breadwinners, shouldering significant financial responsibilities, including healthcare expenses [42–44]. Consequently, this financial burden, more acutely felt by fathers with limited income, might inadvertently lead to delays in accessing quality healthcare services and timely medical interventions for their children, potentially contributing to the increased risk of CP in offspring.

The findings from our study suggest a strong association between the father's level of education and the risk of CP. Specifically, fathers with primary education had a significantly elevated risk. This observation underscores the importance of husband's education and the woman's health behavior [45–47]. This possibility is supported by the Whitehall studies in the UK, which found that a woman's health behaviors corresponded more closely to her husband's social class than to her own [48]. Fathers with higher educational attainment are often better equipped to make informed decisions regarding maternal and child health. In the United States, a father's socioeconomic information may represent the couple's social position more completely than the mother's, given the persistent gender gap in occupation and income [49], even among college graduates [50]. As a marker of relative social status, paternal education may represent the father's ability to contribute time, energy, and resources to support the mother's health before, during, and after pregnancy.

Mothers with a primary level of education were found to be twice as likely to have children with CP compared to mothers with tertiary education. Consistent with this, a study indicated that a low level of education might be linked to CP, with half of the cases reporting that their mothers were illiterate [43]. In our study, 12.6% of the mothers had only completed primary education, while 73.1% had attained a secondary school education. The low educational level of mothers stands out as a preventable risk factor for CP, similar to many other diseases, and can be addressed through appropriate legislative

policies. Elevating the educational attainment of girls holds the potential to foster the emergence of educated mothers and the birth of healthier children. Moreover, an increase in education levels is associated with enhanced health literacy, enabling parents to acquire more knowledge about maintaining a healthy pregnancy and proper pregnancy follow-up [11, 51, 52].

Limitations, bias, and generalizability

The study relied on clinically collected data from the EMR which limited the risk factors we could evaluate. For example, reliable information on antenatal risk factors was not available: maternal eclampsia, maternal hypertension, maternal diabetes, physical problem (e.g., fall down), maternal viral diseases, and low birth weight (< 2500 g). Also, drugs used in pregnancy, maternal nutritional status during pregnancy, and maternal alcohol consumption during pregnancy could not be considered in our analysis.

Conclusion

Our case-control study revealed that both maternal and paternal age are significant risk factors for the occurrence of CP in children. Being a primiparous mother, along with the father's income level, mother's income level, and the educational status of both parents, also predicts the likelihood of having children with CP. The establishment of a nationwide CP register system in Nigeria is essential for gaining a comprehensive understanding of the condition, and it is imperative for enhancing healthcare services for children with CP.

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Authors' contributions

AOF, AOD, OPS, and OMT made substantial contributions to the conception and design of the study. MOO, AE, BEU, and OT participated in the data collection on EMR. AOF analyzed and interpreted the data. AOF and OMT revised the article critically for important intellectual content. The authors read and approved the final manuscript.

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Declarations

Ethics approval and consent to participate

The Health Research Ethics Committee of the Federal Medical Centre Abeokuta (FMCA) (FMCA/470/HREC) granted approval for the study. Official permission was also obtained from the Head of Physiotherapy Department FMCA.

Competing interests

The authors declare that they have no competing interests.

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References

1. Potcovaru CG, Salmen T, Chitu MCC, Dima V, Mihai MB, Bohiltea RE, et al. Cerebral palsy: review of epidemiology, etiology, clinical features, classification and prevention. Roman J Pediatr. 2022;71(S2):18–22.
2. Curley JP, Mashoodh R, Champagne FA. Epigenetics and the origins of paternal effects. Hormones Behav. 2011;59(3):306–14.
3. Sellier E, Uldall P, Calado E, Sigurdardottir S, Torrioli MG, Platt MJ, et al. Epilepsy and cerebral palsy: characteristics and trends in children born in 1976–1998. Europ J Paediatr Neurol. 2012;16(1):48–55.
4. Sellier E, Surman G, Himmelmann K, Andersen G, Colver A, Krägeloh-Mann I, et al. Trends in prevalence of cerebral palsy in children born with a birthweight of 2,500 g or over in Europe from 1980 to 1998. Europ J Epidemiol. 2010;25(9):635–42.
5. McGrath JJ, Petersen L, Agerbo E, Mors O, Mortensen PB, Pedersen CB. A comprehensive assessment of parental age and psychiatric disorders. JAMA Psychiatry. 2014;71(3):301.
6. Bray I. Advanced paternal age: how old is too old? J Epidemiol Commun Health. 2006;60(10):851–3.
7. Frans EM, Sandin S, Reichenberg A, Lichtenstein P, Långström N, Hultman CM. Advancing paternal age and bipolar disorder. Archiv General Psychiatry. 2008;65(9):1034.
8. Bellver J, Garrido N, Remohí J, Pellicer A, Meseguer M. Influence of paternal age on assisted reproduction outcome. Reproduct BioMed Online. 2008;17(5):595–604.
9. Reiss AL, Kesler SR, Vohr B, Duncan CC, Katz KH, Pajot S, et al. Sex differences in cerebral volumes of 8-year-olds born preterm. J Pediatr. 2004;145(2):242–9.
10. Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, et al. Proposed definition and classification of cerebral palsy April 2005. Dev Med Child Neurol. 2005;47(8):571–6.
11. Kahneman D. Control of spurious association and the reliability of the controlled variable. Psycholog bullet. 1965;64(5):326–9.
12. Niven D, Berthiaume L, Fick G, Laupland K. Matched case-control studies: a review of reported statistical methodology. Clinical Epidemiol. 2012;4:99–110.
13. Schneider RE, Ng P, Zhang X, Andersen J, Buckley D, Fehlings D, et al. The Association Between Maternal Age and Cerebral Palsy Risk Factors. Pediatr Neurol. 2018;82:25–8.
14. Chounti A, Hägglund G, Wagner P, Westbom L. Sex differences in cerebral palsy incidence and functional ability: a total population study. Acta Paediatrica. 2013;102(7):712–7.
15. Christensen D, Van Naarden Braun K, Doernberg NS, Maenner MJ, Arneson CL, Durkin MS, et al. Prevalence of cerebral palsy, co-occurring autism spectrum disorders, and motor functioning–Autism and Developmental Disabilities Monitoring Network, USA 2008. Develop Med Child Neurol. 2014;56(1):59–65.
16. Hagberg B, Hagberg G, Beckung E, Uvebrant P. Changing panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year period 1991-94. Acta Paediatrica. 2007;90(3):271–7.
17. Jarvis S. Case gender and severity in cerebral palsy varies with intrauterine growth. Archiv Disease Childhood. 2005;90(5):474–9.
18. Johnston MV, Hagberg H. Sex and the pathogenesis of cerebral palsy. Develop Med Child Neurol. 2006;49(1):74–8.
19. Himmelmann K, Uvebrant P. The panorama of cerebral palsy in Sweden. XI. Changing patterns in the birth-year period 2003–2006. Acta Paediat. 2014;103(6):618–24.
20. Vasileiadis GT, Thompson RT, Han VKM, Gelman N. Females follow a more "compact" early human brain development model than males. A case-control study of preterm neonates. Pediatric Res. 2009;66(5):551–4.
21. Chen C, Chen C, Chen H, Liu W, Shen I, Lin K. Potential predictors of changes in gross motor function during various tasks for children with cerebral palsy: a follow-up study. Res Develop Disab. 2013;34(1):721–8.
22. Bi D, Chen M, Zhang X, Wang H, Xia L, Shang Q, et al. The association between sex-related interleukin-6 gene polymorphisms and the risk for cerebral palsy. J Neuroinflam. 2014;11(1):100.

23. Smith J, Wells L, Dodd K. The continuing fall in incidence of hypoxic-ischaemic encephalopathy in term infants. BJOG: An Int J Obstetr Gynaecol. 2000;107(4):461–6.
24. Zhou L, Meng Q, Von Ehrenstein OS, Xiao J, Gao Y, Wu YW, et al. Parental age and childhood risk for cerebral palsy in California. J Pediatr. 2023;255:147–53.
25. Ogawa K, Urayama KY, Tanigaki S, Sago H, Sato S, Saito S, et al. Association between very advanced maternal age and adverse pregnancy outcomes: a cross sectional Japanese study. BMC Pregn Childbirth. 2017;17(1):349.
26. Strøm MS, Tollånes MC, Wilcox AJ, Lie RT, Forthun I, Moster D. Maternal chronic conditions and risk of cerebral palsy in offspring: a national cohort study. Pediatrics. 2021;147(3):e20201137.
27. Vandekerckhove M, Guignard M, Civadier M-S, Benachi A, Bouyer J. Impact of maternal age on obstetric and neonatal morbidity: a retrospective cohort study. BMC Pregn Childbirth. 2021;21(1):732.
28. Schulkey CE, Regmi SD, Magnan RA, Danzo MT, Luther H, Hutchinson AK, et al. The maternal-age-associated risk of congenital heart disease is modifiable. Nature. 2015;520(7546):230–3.
29. Zaidi AA, Wilton PR, Su MS-W, Paul IM, Arbeithuber B, Anthony K, et al. Bottleneck and selection in the germline and maternal age influence transmission of mitochondrial DNA in human pedigrees. Proceed Nat Academ Sci. 2019;116(50):25172–8.
30. Yatsenko AN, Turek PJ. Reproductive genetics and the aging male. J Assist Reproduct Genet. 2018;35(6):933–41.
31. Sujan AC, Rickert ME, Class QA, Coyne CA, Lichtenstein P, Almqvist C, et al. A genetically informed study of the associations between maternal age at childbearing and adverse perinatal outcomes. Behav Genet. 2016;46(3):431–56.
32. Mills TA, Lavender T. Advanced maternal age. Obstet Gynaecol Reproduct Med. 2011;21(4):107–11.
33. Wyrobek AJ, Eskenazi B, Young S, Arnheim N, Tiemann-Boege I, Jabs EW, et al. Advancing age has differential effects on DNA damage, chromatin integrity, gene mutations, and aneuploidies in sperm. Proceed Nat Academ Sci. 2006;103(25):9601–6.
34. Jenkins TG, Aston KI, Carrell DT. Sperm epigenetics and aging. Translat Androl Urol. 2018;7(S3):S328–35.
35. Gourinat A, Mazeaud C, Hubert J, Eschwege P, Koscinski I. Impact of paternal age on assisted reproductive technology outcomes and offspring health: a systematic review. Androl. 2023;11(6):973–86.
36. Kaltsas A, Moustakli E, Zikopoulos A, Georgiou I, Dimitriadis F, Symeonidis EN, et al. Impact of advanced paternal age on fertility and risks of genetic disorders in offspring. Genes. 2023;14(2):486.
37. Bhatia-Dey N, Csoka AB, Heinbockel T. Chemosensory ability and sensitivity in health and disease: epigenetic regulation and COVID-19. Int J Molec Sci. 2023;24(4):4179.
38. Kong A, Frigge ML, Masson G, Besenbacher S, Sulem P, Magnusson G, et al. Rate of de novo mutations and the importance of father's age to disease risk. Nature. 2012;488(7412):471–5.
39. Nan Y, Zhang J, Nisar A, Huo L, Yang L, Yin J, et al. Professional support during the postpartum period: primiparous mothers' views on professional services and their expectations, and barriers to utilizing professional help. BMC Preg Childbirth. 2020;20(1):402.
40. Willis E, Godbold R. Children's complex health: maternal experiences of care and decision making. J Child Health Care. 2023:136749352311584.
41. Myers MJU. A big brother: new findings on how low-income fathers define responsible fatherhood. J Fam Econ Issues. 2013;34(3):253–64.
42. Sato M, Oshitani H, Tamaki R, Oyamada N, Sato K, Nadra AR, et al. Father's roles and perspectives on healthcare seeking for children with pneumonia: findings of a qualitative study in a rural community of the Philippines. BMJ Open. 2018;8(11):e023857.
43. Forthun I, Lie RT, Strandberg-Larsen K, Solheim MH, Moster D, Wilcox AJ, et al. Parental education and the risk of cerebral palsy for children: an evaluation of causality. Develop Med Child Neurol. 2020;62(10):1176–81.
44. Abdul-Kader M. Low health literacy (LHL): a conniving opponent of patient treatment compliance. SIASAT. 2023;8(1):33–9.
45. Blumenshine P, Egerter S, Libet M, Braveman P. Father's Education: An Independent Marker of Risk for Preterm Birth. Matern Child Health J. 2010;15:60–7.
46. Meng Y, Groth S. Fathers Count: The Impact of Paternal Risk Factors on Birth Outcomes. Matern Child Health J. 2018;22:401–8.
47. Mashoodh R, Habrylo I, Gudsnuk K, Pelle G, Champagne F. Maternal modulation of paternal effects on offspring development. Proceedings of the Royal Society B: Biological Sciences; 2018. p. 285.
48. Bartley M, Martikainen P, Shipley M, Marmot M. Gender differences in the relationship of partner's social class to behavioural risk factors and social support in the Whitehall II study. Soc Sci Med. 2004;59(9):1925–36. (PDF) Father's Education: An Independent Marker of Risk for Preterm Birth. Available from: https://www.researchgate.net/publication/41041782_Father's_Education_An_Independent_Marker_of_Risk_for_Preterm_Birth. Accessed 28 Oct 2023.
49. Holzer H, Neumark D. Assessing affirmative action. J Econ Lit. 2000;38(3):483–568.25.
50. Zhang L. Gender and racial gaps in earnings amongrecent college graduates. Rev High Educ. 2008;32(1):51. (PDF) Father's Education: An Independent Marker of Risk for Preterm Birth. Available from: https://www.researchgate.net/publication/41041782_Father's_Education_An_Independent_Marker_of_Risk_for_Preterm_Birth. Accessed 28 Oct 2023.
51. Yu S. The influence of mothers' educational level on children's comprehensive quality. J Educ Human Soc Sci. 2023;8:1264–72.
52. Ying K, Rostenberghe HV, Kuan G, Mohd Yusoff MHA, Ali SH, Yaacob NS. Health-related quality of life and family functioning of primary caregivers of children with cerebral palsy in Malaysia. Int J Environ Res Pub Health. 2021;18(5):2351.

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