ISSN: 3107-6513

Deconstructing Perinatal Health Disparities

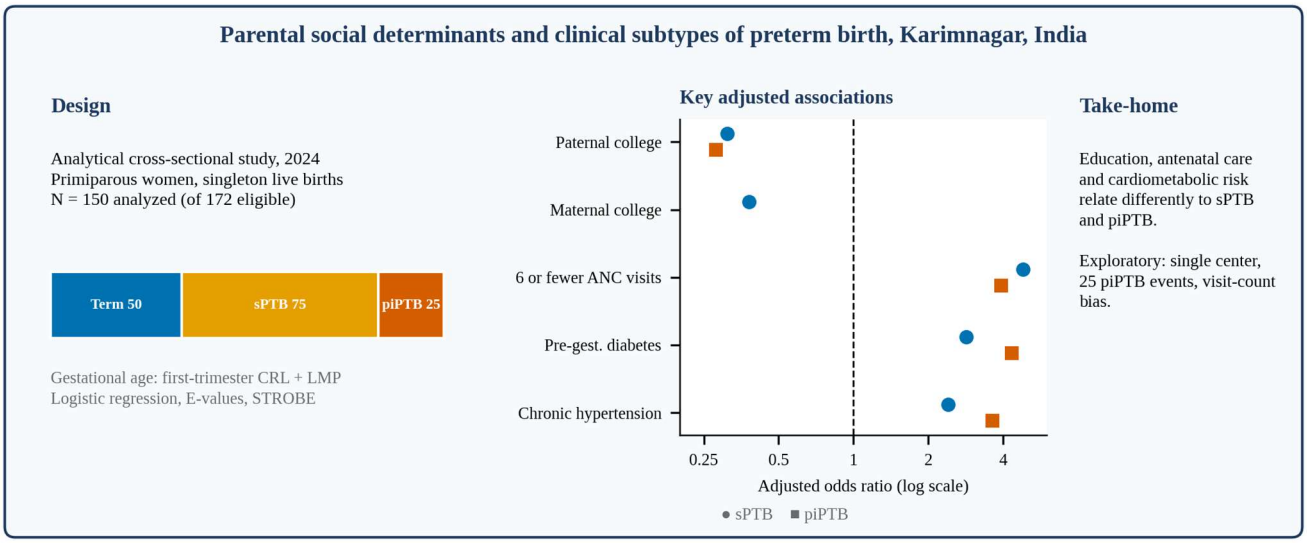
An Analytical Cross-Sectional Study of Parental Social Determinants and Clinical Preterm Birth Subtypes in Karimnagar, India

Atul Gopal Agrawal[#1,*], Ankita Agrawal[#2]

^#1^ Professor and Head, Department of Community Medicine, Surabhi Institute of Medical Sciences, Siddipet, Telangana, India (MBBS, MD Community Medicine, PGDAST, PGDMCH, PGDHHM, ACME, MBA, CCH, CAI, CSD)

^#2^ Consultant Physiotherapist and Researcher, Department of Physiotherapy, Shree Sathya Sai Sanjeevani Mother and Child Hospital, Yavatmal, Maharashtra, India (MPT, Orthopaedics)

*Corresponding author: dratulu9@gmail.com | ORCID: 0009-0007-6829-2766



Graphical abstract. Summary of design and key adjusted associations.

Abstract—

Background: Preterm birth, defined as delivery before 37 completed weeks of gestation, is a leading cause of neonatal and under five mortality. Spontaneous and provider initiated preterm birth arise through different clinical pathways yet are often analyzed together. We examined associations of maternal and paternal education, antenatal care utilization, pre-gestational diabetes and chronic hypertension with each subtype, using full term birth as the reference. Methods: This analytical cross sectional study was conducted at a tertiary care teaching hospital in Karimnagar, Telangana, India, from May to July 2024. Of 172 eligible primiparous women with singleton live births, 150 with complete paternal data were analyzed, comprising 50 full term births, 75 spontaneous preterm births and 25 provider initiated preterm births. Gestational age was established by first trimester crown rump length reconciled with the last menstrual period. Binary logistic regression, with a secondary multinomial model, produced crude and adjusted odds ratios with 95 percent confidence intervals, and E-values assessed robustness to unmeasured confounding. Results: Mean maternal age was 24.6 years. Maternal college education was associated with lower odds of spontaneous preterm birth, with an adjusted odds ratio of 0.38 and a 95 percent confidence interval of 0.16 to 0.92, but not of provider initiated preterm birth. Paternal college education was associated with lower odds of both subtypes. Six or fewer antenatal visits, pre-gestational diabetes and chronic hypertension were associated with higher odds of both subtypes, with adjusted odds ratios from 2.41 to 4.82. Severe pre-eclampsia or eclampsia was the leading indication for provider initiated delivery, accounting for 40 percent of cases. Conclusions: In this selected tertiary care cohort, education, antenatal care and cardiometabolic factors were differentially associated with preterm birth subtypes. Because of the single center design, the small provider initiated group and gestational age bias in visit counts, the findings are exploratory and associational. Larger prospective multicenter studies are warranted.

Keywords—preterm birth; spontaneous preterm birth; provider-initiated preterm birth; social determinants of health; antenatal care; paternal education; diabetes; hypertension; India

I. INTRODUCTION

Preterm birth (PTB), defined as delivery before 37 completed weeks of gestation, affected an estimated 13.4 million live births worldwide in 2020, roughly one in ten, with the largest absolute numbers in South Asia and sub-Saharan Africa [1], [2]. Complications of prematurity are the leading cause of death in children under five years [3]. India contributes more preterm births than any other country [1], [4], and analyses of national survey and facility data continue to highlight maternal, social and clinical correlates [5].

PTB is a clinically heterogeneous outcome. Spontaneous PTB (sPTB) follows spontaneous onset of preterm labor or preterm prelabor rupture of membranes (PPROM), whereas provider-initiated PTB (piPTB) follows induction or prelabor cesarean delivery performed because continuing the pregnancy poses maternal or fetal risk [6]–[8]. These pathways differ in etiology, risk factors and preventive opportunities, and pooling them can mask distinct determinants [7], [9].

ISSN: 3107-6513

Social determinants of health, including education, household resources, health literacy and access to care, shape reproductive outcomes through cumulative pathways [10]. Lower socioeconomic position is associated with adverse birth outcomes including PTB [11], and lower maternal education has been linked to PTB in European cohorts [12]. Evidence from India includes community-based and hospital-based studies of socioeconomic and clinical risk factors [13]–[15]. Paternal characteristics are less often studied; systematic reviews suggest that paternal factors may relate to adverse birth outcomes, and paternal involvement may influence perinatal outcomes through household support and care-seeking [16], [17].

Antenatal care (ANC) offers opportunities for risk identification, counseling and referral; the World Health Organization (WHO) recommends at least eight ANC contacts, beginning in the first trimester [18]. However, interpreting visit counts is complicated by gestational-age bias, because early delivery truncates the opportunity to accumulate visits [19]. Pre-existing hypertension and diabetes increase maternal and fetal risk and may precipitate medically indicated early delivery [20]–[22]; India carries a high burden of both conditions [23].

We therefore evaluated associations of maternal and paternal educational attainment, ANC utilization, pre-gestational diabetes and chronic hypertension with sPTB and piPTB, using full-term birth as the reference, in primiparous women delivering at a tertiary-care hospital in Karimnagar, Telangana. We hypothesized that associations would differ by subtype, with clinical comorbidities more strongly related to piPTB.

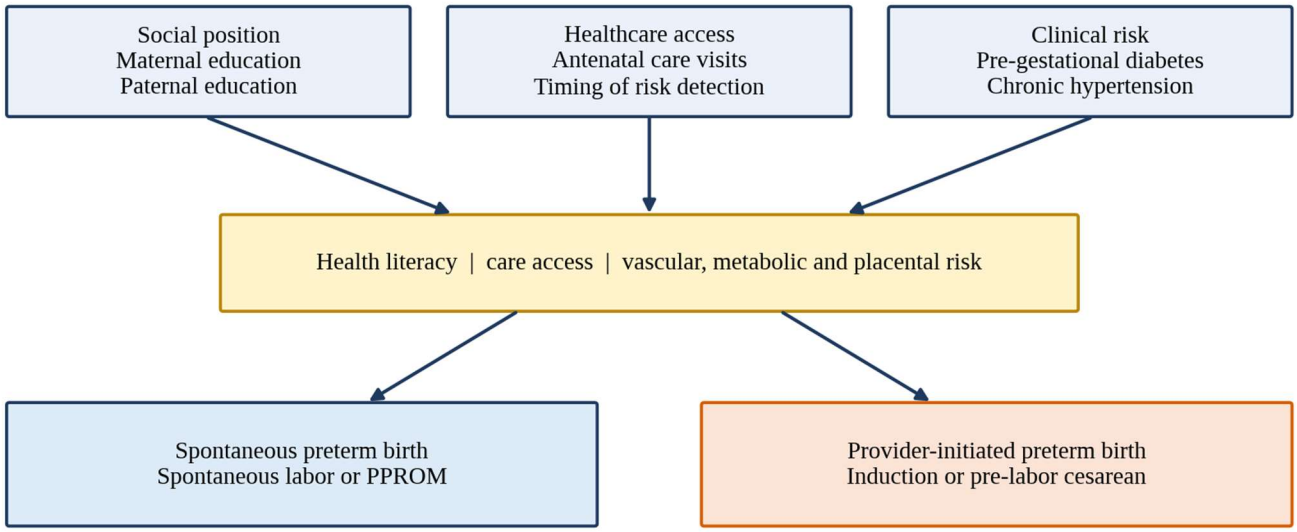


Fig. 1. Conceptual framework. Social position, healthcare access and clinical risk are hypothesized to act through maternal and fetal pathways on spontaneous and provider-initiated PTB. Arrows denote hypothesized pathways, not causal effects established here. PPROM: preterm prelabor rupture of membranes.

II. MATERIALS AND METHODS

A. Study Design, Setting and Reporting Standard

This analytical cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, in collaboration with the Department of Community Medicine, at Prathima Institute of Medical Sciences, a tertiary-care teaching hospital in Karimnagar, Telangana, India. Data were collected from 1 May to 31 July 2024. Reporting follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies [24]; the checklist is provided as Supplementary Table S2.

B. Participants and Selection

The target population was primiparous women aged 18–44 years admitted for delivery. Inclusion criteria were primiparous status, singleton pregnancy, a live-born infant, and verified parental education records with formal paternal acknowledgment. Exclusion criteria were multifetal pregnancy, major congenital anomaly or intrauterine fetal demise, unacknowledged paternity or missing paternal educational items, and emergency transfer without prior medical documentation. Singleton pregnancies were chosen to remove the dominant confounding effect of multifetal gestation on preterm labor. Of 172 eligible women screened, 22 were excluded because paternal information was missing or paternity was unacknowledged, leaving 150 for analysis (Fig. 2).

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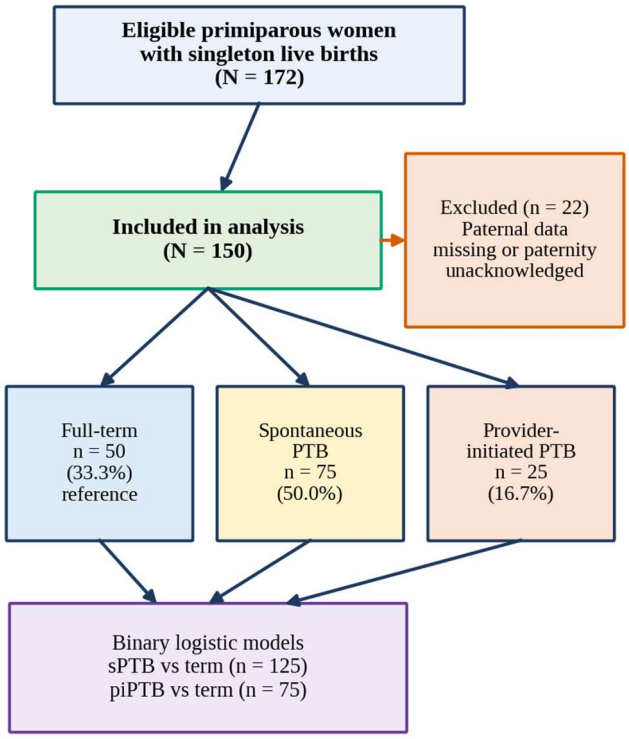


Fig. 2. STROBE participant flow. Derivation of the analytic cohort (N = 150) from 172 eligible primiparous women, with outcome groups and the model comparison sets. GA: gestational age.

C. Gestational-Age Ascertainment

Gestational age was established from first-trimester ultrasonographic crown–rump length (CRL) measured between 8+0 and 13+6 weeks, reconciled with a reliable last menstrual period (LMP) [25]. A 5-day discrepancy rule was applied: if LMP- and CRL-derived gestational ages differed by 5 days or less, LMP dating was accepted; if the difference exceeded 5 days, or the LMP was unreliable or unknown, the CRL estimate was taken as definitive. This threshold is stricter than the 7 days commonly applied to CRL measured at 9+0–13+6 weeks [26].

D. Outcome Definitions

PTB was delivery before 37+0 weeks; full-term birth was delivery at or beyond 37+0 weeks (reference). Subtypes were defined by the initiating pathway rather than by mode of birth (Table I) [6], [8], [9].

TABLE I
OPERATIONAL DEFINITIONS OF THE OUTCOME CATEGORIES

Category	Definition	n (%)
Full-term birth (reference)	Delivery at ≥37+0 completed weeks	50 (33.3)
Spontaneous PTB (sPTB)	<37+0 weeks following (i) spontaneous onset of preterm labor, whether vaginal birth or emergency intrapartum cesarean occurred, or (ii) PPROM followed by spontaneous labor or clinically managed delivery	75 (50.0)
Provider-initiated PTB (piPTB)	<37+0 weeks following planned medical induction or prelabor cesarean section for documented maternal indications (e.g., severe pre-eclampsia or eclampsia, uncontrolled pre-gestational diabetes with end-organ damage) or fetal indications (e.g., severe fetal growth restriction, oligohydramnios, acute fetal distress)	25 (16.7)

E. Exposures and Covariates

Maternal and paternal education were categorized as less than high school (reference), high school diploma or General Educational Development (GED), some college, and college degree or higher. ANC utilization was grouped as 12 or more visits (reference), 7–11 visits and 6 or fewer visits. Pre-gestational diabetes and chronic hypertension were coded yes or no. Maternal age (18–22, 23–34 and 35–44 years) was a covariate in all models.

F. Sample Size and Power

Sample size was calculated a priori with G*Power (version 3.1.9.7) [27] for logistic regression evaluating the primary social determinants (maternal and paternal education), with two-tailed $\alpha = 0.05$, target power $(1 - \beta) = 0.80$, an odds ratio of 0.35 for higher education, and a baseline event probability of 0.33 in the reference group; the authors report a required minimum of N = 138 and a post hoc power of 0.842 at N = 150. These calculations refer to the full cohort. The subtype-specific models used 125 (sPTB versus term) and 75 (piPTB versus term) observations and were therefore less powered; post hoc power is reported for completeness only and is not interpreted as evidence of adequacy.

G. Statistical Analysis

Analyses used IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as counts and percentages and compared with Pearson chi-square tests. Binary logistic regression estimated crude and adjusted odds ratios (AOR) with 95% confidence intervals (CI) for each subtype versus full-term birth (sPTB versus term, n = 125; piPTB versus term, n = 75). Adjusted models included maternal age, maternal education, paternal education, ANC visit strata, pre-gestational diabetes and chronic hypertension; a secondary multinomial logistic model was fitted with term as reference. Two-sided $p < 0.05$ was considered statistically significant. No correction for multiple testing was applied; 20 adjusted contrasts are reported, so isolated p-values near 0.05 should be interpreted cautiously. Calibration was assessed with the Hosmer–Lemeshow test, reported as $p > 0.20$ for all final models.

The piPTB model contains 25 events and the full covariate set implies roughly a dozen estimated parameters (about two events per parameter), well below usual events-per-variable guidance [28], [29]; the resulting estimates may be unstable or biased away from the null, which is considered under Limitations.

H. Sensitivity Analysis for Selection Bias

Included (n = 150) and excluded (n = 22) participants were compared on maternal age (independent-samples t-test) and gestational age at delivery (Mann–Whitney U test).

I. Post Hoc Verification and Robustness Analyses

To support transparency, the estimates were re-examined without access to participant-level data. (i) *Internal consistency:* for each adjusted estimate the Wald z-statistic was reconstructed from the reported OR and 95% CI (SE = [ln(upper) − ln(lower)] / 3.92), and the implied two-sided p-value was compared with the reported p-value; symmetry of the CI on the log scale was also checked. (ii) *Unmeasured confounding:* E-values [30] were computed for each statistically significant AOR. Because the outcome categories are common (>10%), odds ratios were converted to approximate risk ratios by the square-root transformation

ISSN: 3107-6513

before E-value calculation. These are derived quantities, not a re-analysis of the raw data.

J. Ethics

The study was approved by the Institutional Ethics Committee, Prathima Institute of Medical Sciences, Karimnagar, Telangana (IEC/PIMS/2024/27; 22 April 2024). Written informed consent was obtained from all participants, and confidentiality was maintained in accordance with the Declaration of Helsinki.

III. RESULTS

A. Participant Characteristics

The analytic cohort comprised 150 women (mean age 24.6 ± 3.8 years; range 18–41): 40.7% were aged 18–22, 52.0% aged 23–34 and 7.3% aged 35–44 years. Fifty births (33.3%) were full-term, 75 (50.0%) were sPTB and 25 (16.7%) were piPTB (Table II; Fig. 3). These proportions describe a selected tertiary-care cohort and must not be read as population prevalence.

TABLE II
BASELINE CHARACTERISTICS OF THE STUDY COHORT (N = 150)

Characteristic	Category	n	%	Characteristic	Category	n	%
Maternal age (years)	18–22	61	40.7	Paternal education	<High school	28	18.7
	23–34	78	52.0		High school/GED	62	41.3
	35–44	11	7.3		Some college	38	25.3
Delivery outcome	Full-term (≥37 wk)	50	33.3		College degree or higher	22	14.7
	Spontaneous PTB	75	50.0	Antenatal visits	≥12 (adequate)	55	36.7
	Provider-initiated PTB	25	16.7		7–11 (intermediate)	63	42.0
Maternal education	<High school	24	16.0		≤6 (inadequate)	32	21.3
	High school/GED	58	38.7	Pre-gestational diabetes	Yes	14	9.3
	Some college	42	28.0	Chronic hypertension	Yes	22	14.7
	College degree or higher	26	17.3				

Percentages are of the 150 analyzed women. GED: General Educational Development.

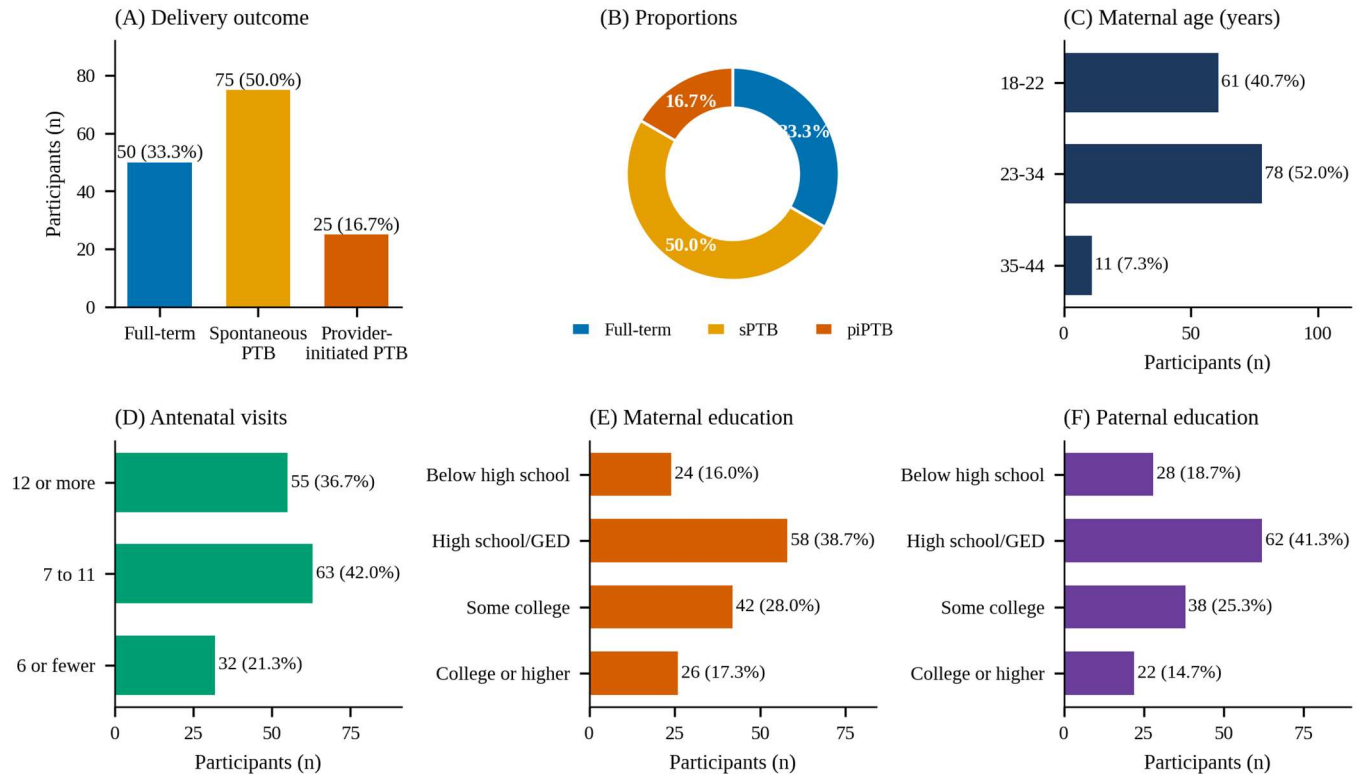


Fig. 3. Cohort profile. (A) Counts and (B) proportions of full-term, spontaneous PTB (sPTB) and provider-initiated PTB (piPTB); distribution of (C) maternal age, (D) antenatal visits, (E) maternal education and (F) paternal education (N = 150).

B. Educational Attainment and PTB Subtypes

Compared with maternal education below high school, college education or higher was associated with lower adjusted odds of sPTB (AOR 0.38, 95% CI 0.16–0.92; p = 0.032) but not piPTB (AOR 0.85, 0.20–3.61; p = 0.824). Paternal college education was associated with lower adjusted odds of both sPTB (AOR 0.31, 0.12–0.81; p = 0.016) and piPTB (AOR 0.28, 0.08–0.94; p = 0.039) (Table III; Figs. 4 and 6A–B). Intermediate education levels showed point estimates below 1 but confidence intervals spanning the null, a pattern compatible with a graded association that this sample is too small to resolve.

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TABLE III
EDUCATIONAL ATTAINMENT AND PTB SUBTYPE: CRUDE AND ADJUSTED ODDS RATIOS

Exposure level	Outcome (model n)	Crude OR (95% CI)	AOR (95% CI)	p
Maternal education (reference: <high school)				
High school/GED	sPTB (125)	0.62 (0.24–1.58)	0.65 (0.24–1.75)	0.392
	piPTB (75)	0.85 (0.26–2.78)	0.88 (0.25–3.12)	0.845
Some college	sPTB (125)	0.48 (0.18–1.28)	0.52 (0.18–1.48)	0.218
	piPTB (75)	0.72 (0.20–2.58)	0.76 (0.19–3.04)	0.698
College degree or higher	**sPTB (125)**	**0.35 (0.12–0.98)**	**0.38 (0.16–0.92)**	**0.032***
	piPTB (75)	0.82 (0.21–3.22)	0.85 (0.20–3.61)	0.824
Paternal education (reference: <high school)				
High school/GED	sPTB (125)	0.72 (0.28–1.85)	0.75 (0.27–2.08)	0.582
	piPTB (75)	0.78 (0.23–2.64)	0.81 (0.22–2.98)	0.751
Some college	sPTB (125)	0.45 (0.16–1.24)	0.48 (0.16–1.42)	0.184
	piPTB (75)	0.58 (0.15–2.22)	0.61 (0.14–2.62)	0.508
College degree or higher	**sPTB (125)**	**0.28 (0.09–0.85)**	**0.31 (0.12–0.81)**	**0.016***
	piPTB (75)	**0.25 (0.05–1.18)**	**0.28 (0.08–0.94)**	**0.039***

*p < 0.05. Reference outcome: full-term birth. AOR: odds ratio adjusted for maternal age, maternal education, paternal education, ANC strata, pre-gestational diabetes and chronic hypertension. sPTB: spontaneous PTB; piPTB: provider-initiated PTB.

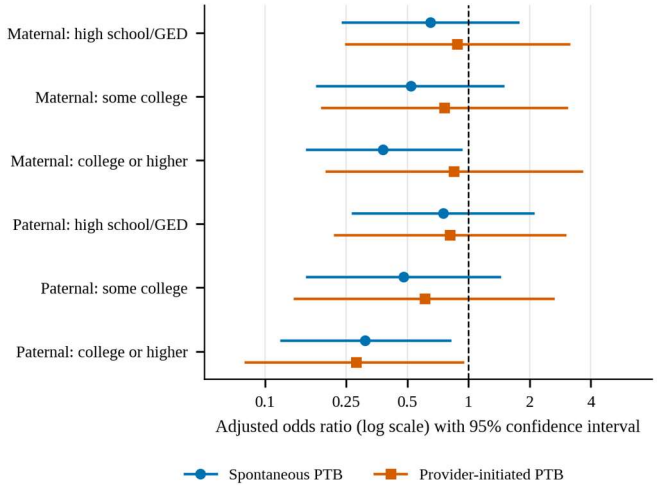


Fig. 4. Forest plot: education. Adjusted odds ratios (log scale) with 95% CIs for sPTB and piPTB by maternal and paternal education (reference: below high school).

C. Antenatal Care and Clinical Comorbidities

Relative to 12 or more visits, 6 or fewer ANC visits were associated with higher adjusted odds of sPTB (AOR 4.82, 1.85–12.56; p = 0.001) and piPTB (AOR 3.94, 1.22–12.71; p = 0.022); 7–11 visits showed no significant association (Table IV; Figs. 5 and 6C). Pre-gestational diabetes was associated with sPTB (AOR 2.85, 1.05–7.74; p = 0.040) and piPTB (AOR 4.35, 1.28–14.78; p = 0.018), and chronic hypertension with sPTB (AOR 2.41, 1.02–5.68; p = 0.045) and piPTB (AOR 3.62, 1.15–11.39; p = 0.028). Point estimates were larger for piPTB than sPTB for both comorbidities, although the confidence intervals overlap broadly and no formal test of difference between subtypes was performed.

TABLE IV
ANTENATAL CARE, PRE-GESTATIONAL DIABETES, CHRONIC HYPERTENSION AND PTB SUBTYPE

Predictor (reference)	Outcome (model n)	Crude OR (95% CI)	AOR (95% CI)	p
7–11 visits (ref ≥12)	sPTB (125)	1.85 (0.82–4.18)	1.92 (0.81–4.55)	0.138
	piPTB (75)	1.62 (0.52–5.05)	1.68 (0.51–5.53)	0.391
≤6 visits (ref ≥12)	**sPTB (125)**	**4.42 (1.75–11.16)**	**4.82 (1.85–12.56)**	**0.001***
	piPTB (75)	**3.65 (1.18–11.28)**	**3.94 (1.22–12.71)**	**0.022***
Pre-gestational diabetes (ref no)	**sPTB (125)**	**2.65 (1.02–6.88)**	**2.85 (1.05–7.74)**	**0.040***
	piPTB (75)	**4.12 (1.25–13.58)**	**4.35 (1.28–14.78)**	**0.018***
Chronic hypertension (ref no)	**sPTB (125)**	**2.28 (1.01–5.15)**	**2.41 (1.02–5.68)**	**0.045***
	piPTB (75)	**3.45 (1.12–10.62)**	**3.62 (1.15–11.39)**	**0.028***

*p < 0.05. Reference outcome: full-term birth. Covariates as in Table III.

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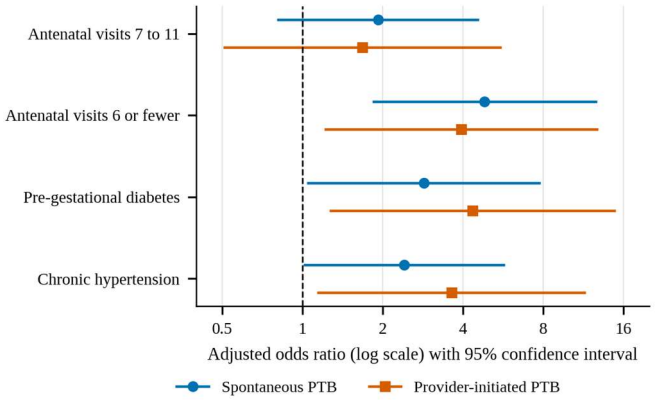


Fig. 5. Forest plot: antenatal care and comorbidities. Adjusted odds ratios (log scale) with 95% CIs for sPTB and piPTB.

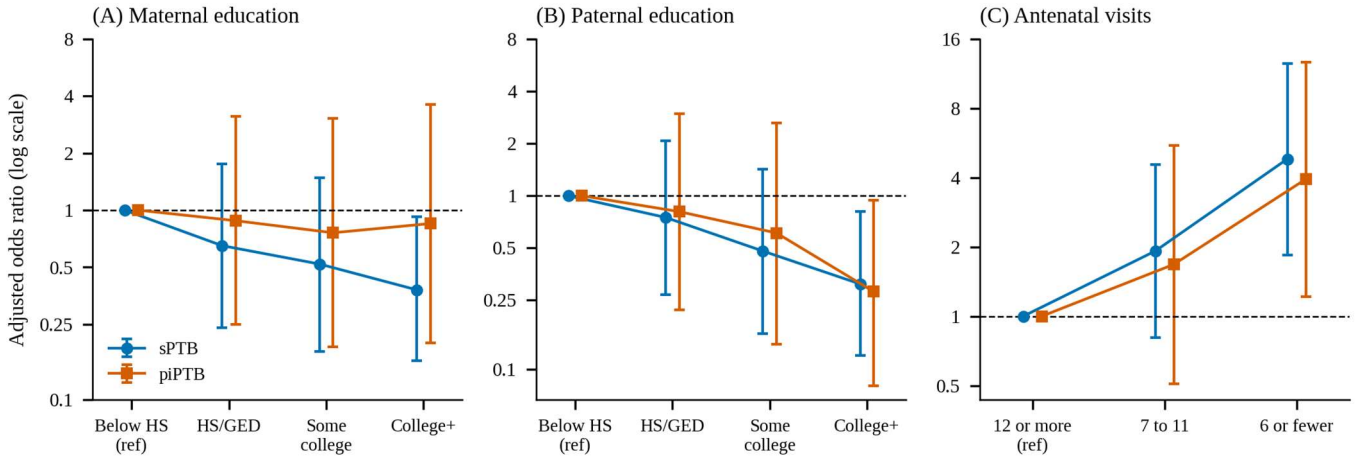


Fig. 6. Exposure–response patterns. Adjusted odds ratios across ordered categories of (A) maternal education, (B) paternal education and (C) antenatal visits. HS: high school. Intermediate categories are not individually significant.

D. Clinical Indications for Provider-Initiated PTB

Among the 25 piPTB cases, severe pre-eclampsia or eclampsia was the leading indication (10 cases, 40.0%), followed by pre-gestational diabetes with end-organ or placental compromise (6, 24.0%), severe fetal growth restriction with oligohydramnios (5, 20.0%) and acute fetal distress or placental abruption (4, 16.0%) (Table V; Fig. 7). Maternal indications therefore accounted for 16 of 25 cases (64.0%) and fetal or placental indications for 9 (36.0%), a distribution compatible with the larger point estimates seen for hypertension and diabetes in the piPTB models.

TABLE V
PRIMARY CLINICAL INDICATIONS FOR PROVIDER-INITIATED PTB (N = 25)

Primary indication	Domain	n	%
Severe pre-eclampsia or eclampsia	Maternal	10	40.0
Pre-gestational diabetes with end-organ or placental compromise	Maternal	6	24.0
Severe fetal growth restriction with oligohydramnios	Fetal/placental	5	20.0
Acute fetal distress or placental abruption	Fetal/placental	4	16.0
Total		**25**	**100.0**

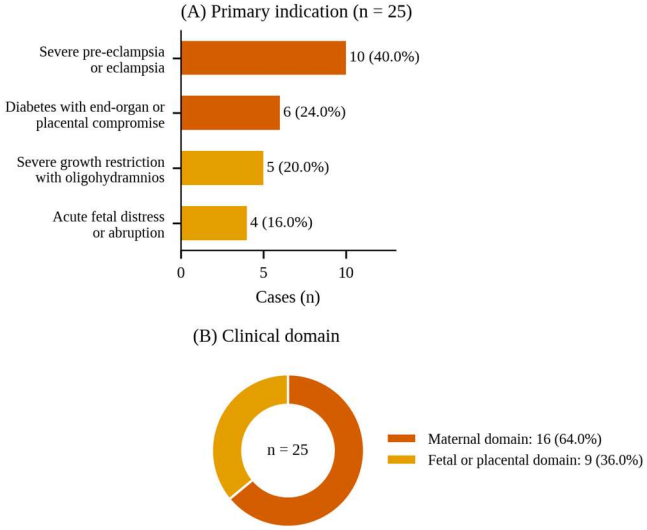


Fig. 7. Indications for provider-initiated PTB. (A) Distribution of primary indications (n = 25); (B) maternal versus fetal or placental domains.

E. Comparison of Crude and Adjusted Estimates

Adjusted estimates were very close to crude estimates for every association (Fig. 8), including after simultaneous adjustment for maternal and paternal education. In three estimates (maternal college and sPTB, paternal college and sPTB, and paternal college and piPTB) the adjusted confidence interval was narrower than the crude interval, an uncommon

ISSN: 3107-6513

pattern when covariates are added to a logistic model that is acknowledged as a limitation (Supplementary Table S1).

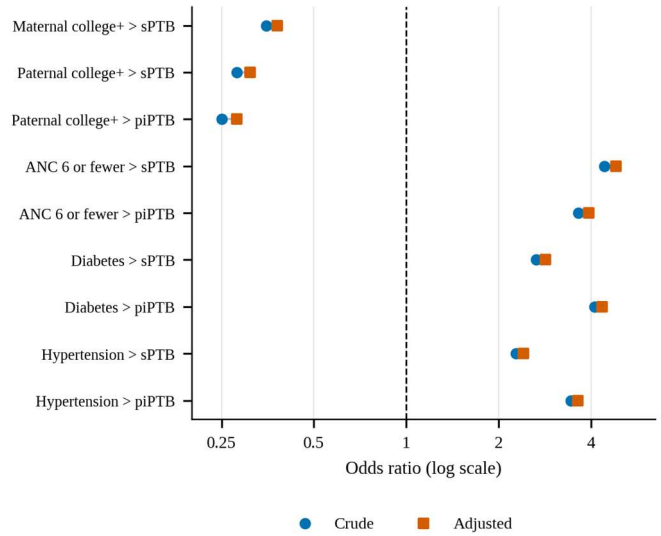


Fig. 8. Crude versus adjusted estimates. Odds ratios (log scale) for the statistically significant associations. ANC: antenatal care.

F. Sensitivity Analysis: Included Versus Excluded Participants

Included and excluded participants did not differ significantly in maternal age (24.6 ± 3.8 vs 24.1 ± 4.1 years; t = 0.573, p = 0.567) or gestational age at delivery (35.8 ± 2.9 vs 35.2 ± 3.2 weeks; Mann–Whitney U = 1482.5, Z = −0.831, p = 0.406) (Table VI). This addresses two measured variables only and does not exclude selection bias related to paternal-data completeness.

TABLE VI
INCLUDED VERSUS EXCLUDED PARTICIPANTS

Variable	Included (n = 150)	Excluded (n = 22)	Test	p
Maternal age, years (mean ± SD)	24.6 ± 3.8	24.1 ± 4.1	t = 0.573	0.567
Gestational age at delivery, weeks (mean ± SD)	35.8 ± 2.9	35.2 ± 3.2	U = 1482.5; Z = −0.831	0.406

G. Robustness to Unmeasured Confounding

E-values for point estimates ranged from 2.48 to 3.82; E-values for the confidence limit nearest the null were close to 1 for several associations (Table VII; Fig. 9), meaning that modest unmeasured confounding could move those intervals to include the null. The strongest signal was for 6 or fewer ANC visits and sPTB (E-value 3.82; 2.06 for the CI limit), which is also the estimate most exposed to gestational-age bias (Section IV-C).

TABLE VII
E-VALUES FOR STATISTICALLY SIGNIFICANT ADJUSTED ASSOCIATIONS

Exposure	Outcome	AOR (95% CI)	E-value (estimate)	E-value (CI limit)
Maternal college+	sPTB	0.38 (0.16–0.92)	2.63	1.25
Paternal college+	sPTB	0.31 (0.12–0.81)	2.99	1.46
Paternal college+	piPTB	0.28 (0.08–0.94)	3.19	1.21
≤6 ANC visits	sPTB	4.82 (1.85–12.56)	3.82	2.06
≤6 ANC visits	piPTB	3.94 (1.22–12.71)	3.38	1.44
Pre-gestational diabetes	sPTB	2.85 (1.05–7.74)	2.77	1.18
Pre-gestational diabetes	piPTB	4.35 (1.28–14.78)	3.59	1.52
Chronic hypertension	sPTB	2.41 (1.02–5.68)	2.48	1.11
Chronic hypertension	piPTB	3.62 (1.15–11.39)	3.21	1.35

E-values computed from reported AORs using the square-root approximation for common outcomes [30]. college+: college degree or higher.

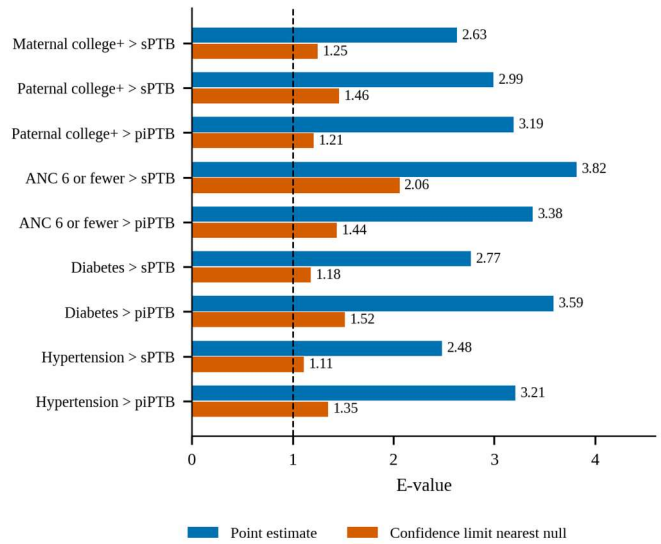


Fig. 9. E-values. Minimum strength of association an unmeasured confounder would need with both exposure and outcome to explain away the point estimate (blue) or move the confidence limit to the null (orange).

H. Internal Consistency of Reported Estimates

For all 20 adjusted estimates, p-values reconstructed from the reported OR and CI agreed with the reported p-values (largest absolute difference 0.006, for maternal some-college education and sPTB: implied p = 0.224 versus reported 0.218), and every reported estimate lay near the geometric midpoint of its CI (largest log-scale deviation 0.021). These checks support the internal coherence of the reported values but do not substitute for re-analysis of the source data (Supplementary Table S1).

IV. DISCUSSION

A. Principal Findings

In this tertiary-care cohort of primiparous women, social and clinical factors showed partly distinct associations with spontaneous and provider-initiated PTB. Higher paternal education was associated with lower odds of both subtypes, maternal college education with lower odds of sPTB only, low ANC attendance with higher odds of both, and pre-gestational diabetes and chronic hypertension with both subtypes, with larger point estimates for piPTB. The subtype distinction is clinically relevant because spontaneous labor or PPROM and medically indicated delivery call for different preventive and service responses [7], [8]. Provider-initiated delivery has been

ISSN: 3107-6513

reported to account for up to about half of PTB in many high-income settings [9]; in this cohort it represented a smaller share (16.7% of births, 25 of 100 preterm births), which underlines why subtype-specific analysis matters when results are compared across health systems.

B. Education and the Social Gradient

The inverse association between maternal education and sPTB agrees with evidence linking lower education and socioeconomic disadvantage with PTB [11], [12] and with Indian studies of socioeconomic correlates [13]–[15]. Education may act through health literacy, household resources, occupational exposures, autonomy and care access. The finding that paternal education was associated with both subtypes, including piPTB where maternal education was not, is notable but should be treated cautiously: paternal education may proxy household socioeconomic context and support [16], [17], and the piPTB estimates rest on only 25 events with wide intervals. These mechanisms were not measured, and no causal interpretation is warranted.

C. Antenatal Care and Gestational-Age Bias

Low ANC attendance was strongly associated with both subtypes, in line with the rationale for the WHO recommended minimum of eight contacts [18] and with hospital-based Indian data that list inadequate antenatal care among the commonly associated conditions [15]. However, early delivery shortens the time available to accumulate visits, so a low count may partly be a consequence rather than a cause of preterm birth (Fig. 10) [19]. The magnitude of the 6-or-fewer-visit associations is therefore likely inflated, and the data cannot show that increasing visit counts alone would prevent PTB. Future studies should record timing of booking, content and continuity of care, and model visits relative to gestational opportunity.

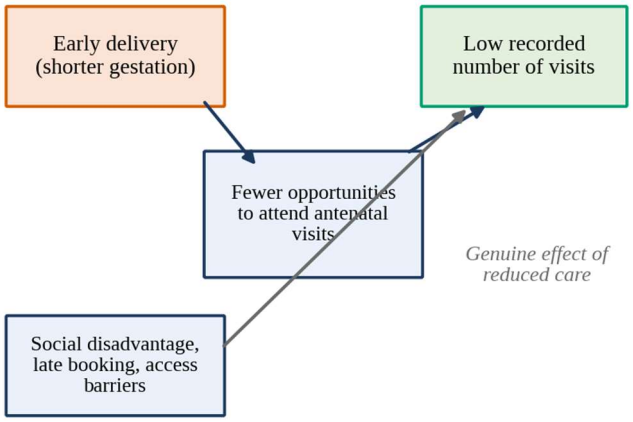


Fig. 10. Gestational-age bias in ANC visit counts. Earlier delivery reduces the opportunity to accumulate visits, producing a spurious or inflated association between few visits and PTB, alongside any genuine effect of reduced care.

D. Comorbidities and Provider-Initiated PTB

Chronic hypertension and pre-existing diabetes raise maternal and fetal risk and are established reasons for indicated early delivery [20]–[22]. In this cohort, 64.0% of piPTB cases had a maternal indication, led by severe pre-eclampsia or eclampsia (40.0%). Larger point estimates for piPTB are therefore biologically plausible; trial evidence indicates that treating chronic hypertension to a target below 140/90 mm Hg reduces a composite of adverse outcomes that includes medically indicated early preterm birth [21], and glycemia exhibits a continuous relationship with adverse perinatal outcomes [22]. Nevertheless, disease severity and glycemic or blood-pressure control were not recorded, and the indication data are primary-indication counts only, so the pathway-specific interpretation remains provisional. Given the burden of diabetes and hypertension in India [23], preconception screening and optimization are plausible priorities.

E. Strengths and Limitations

Strengths include a clearly defined tertiary-care setting, restriction to singleton first live births, first-trimester CRL dating with an explicit discrepancy rule, documented clinical indications for piPTB, written informed consent, separation of spontaneous and provider-initiated PTB, inclusion of paternal education, an included-versus-excluded comparison, and transparent verification of reported estimates.

Limitations include: (i) single-center, hospital-based sampling with a high proportion of preterm births (66.7%), reflecting a selected referral population, so prevalence and generalizability are limited; (ii) a small piPTB group (n = 25), implying wide intervals, limited power and a risk of unstable or overfitted estimates [28], [29]; (iii) a full covariate set of about a dozen parameters fitted to 25 piPTB events (about two events per parameter) and simultaneous adjustment for correlated maternal and paternal education, which can yield unstable estimates and wide intervals, together with residual confounding by body mass index, tobacco exposure, household income, infection, assisted conception and disease severity; (iv) gestational-age bias in ANC visit counts; (v) possible selection bias from excluding 22 women with missing paternal data, which age and gestational-age comparisons cannot exclude; (vi) multiple comparisons without correction; (vii) the cross-sectional, outcome-defined sampling, which precludes causal inference and temporal ordering; (viii) power calculated for the full cohort rather than for the 125- and 75-observation subtype models; (ix) piPTB indication data limited to primary indications; and (x) three adjusted confidence intervals that were narrower than their crude counterparts. Findings should be regarded as exploratory.

F. Implications

- **Practice:** early registration and continuous, quality ANC with risk-based follow-up, with explicit attention to women with limited education.
- **Preconception and chronic disease:** screening and optimization of diabetes and hypertension before conception, and structured follow-up in pregnancy.
- **Family-inclusive education:** engaging partners in antenatal counseling and household health literacy.
- **Research:** larger prospective multicenter cohorts recording delivery indications, booking gestation, ANC content and quality, dating method and relevant confounders, with subtype-specific multinomial modeling and penalized estimation where events are few [28], [29].

V. CONCLUSION

In this 150-participant tertiary-care cohort, maternal and paternal education, ANC attendance, pre-gestational diabetes and chronic hypertension were associated with spontaneous and provider-initiated PTB in partly different ways. Given the small single-center sample, the small number of piPTB events relative to covariates and gestational-age bias in visit counts, these results are exploratory and hypothesis-generating. Prospective multicenter studies are needed before programmatic conclusions are drawn. The direction and strength of all 20 adjusted associations are summarized in Fig. 11.

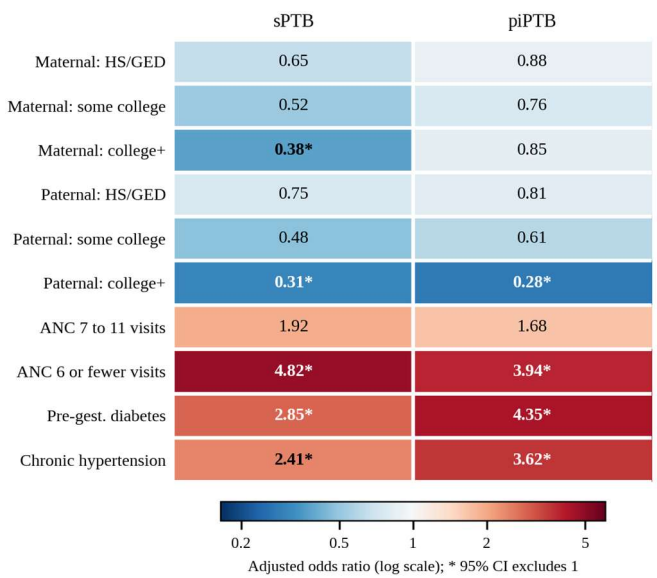


Fig. 11. Heat map of adjusted odds ratios for sPTB and piPTB. Blue cells indicate lower and red cells higher odds relative to the reference category; asterisks mark associations whose 95% CI excludes 1. HS: high school; ANC: antenatal care.

DECLARATIONS

Ethics approval and consent to participate: Approved by the Institutional Ethics Committee, Prathima Institute of Medical Sciences, Karimnagar, Telangana, India (IEC/PIMS/2024/27; 22 April 2024). Written informed consent was obtained from all participants.

Funding: This research was self-funded by the authors. No external financial support, grants or sponsorships were received for the design, execution, analysis or drafting of this study.

Competing interests: The authors declare that they have no competing financial, personal or non-financial interests that could inappropriately influence the content of this manuscript.

Author contributions: A.G.A.: conceptualization, study design, methodology, clinical data acquisition, statistical modeling, G*Power calculations and primary drafting. A.A.: data validation, literature review, visual graphics design, STROBE flowcharting, editing and critical revision. Both authors read and approved the final manuscript.

Data availability: De-identified participant datasets, statistical coding and SPSS output supporting the conclusions are available from the corresponding author on reasonable academic request, subject to institutional ethics data-sharing agreements.

ACKNOWLEDGMENT

The authors acknowledge the clinical and nursing staff of the Departments of Obstetrics and Gynaecology and Community Medicine, Prathima Institute of Medical Sciences, Karimnagar, for institutional support during data collection.

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ISSN: **3107-6513**

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TABLE S1
STROBE CHECKLIST (CROSS-SECTIONAL STUDIES): LOCATION OF ITEMS

STROBE domain	Content	Location
Title and abstract	Design in title; structured abstract	Title, Abstract
Background/rationale; objectives	Scientific background; hypothesis	Section I
Study design; setting; participants	Design, site, dates, eligibility, flow	II-A, II-B; Fig. 2
Variables	Outcomes, exposures, covariates	II-D, II-E; Table I
Data sources/measurement	Gestational-age dating and discrepancy rule	II-C
Bias	Selection bias, gestational-age bias, confounding	II-B, II-H, IV-C, IV-E
Study size	G*Power calculation	II-F
Statistical methods	Logistic and multinomial models; verification analyses	II-G, II-I
Participants; descriptive data	Flow; characteristics	III-A; Table II; Fig. 3
Outcome data; main results	Crude and adjusted ORs; piPTB indications	III-B to III-D; Tables III–V; Figs. 4–7
Other analyses	Sensitivity analysis; E-values; consistency check	III-E to III-H; Tables VI–VII; Figs. 8–9, 11; Table S1
Key results; limitations; interpretation; generalizability	Summary, limitations, implications	IV-A to IV-F; V
Funding; ethics	Declarations	II-J; Declarations