

Histological placental abnormalities and associated factors among malaria smear-positive women delivering at two tertiary hospitals in Uganda: a comprehensive multicenter cross-sectional study

Yumbi Lutula · Petrus Baruti · Marie Pascaline Sabine Ishimwe · Raymond Atwiine · Theodore Nteziyaremye · Anyanwu Godson Emeka · Theoneste Hakizimana

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Abstract

Malaria in pregnancy may disrupt placental structure and function, but evidence describing placental abnormalities among malaria-positive women in Uganda remains limited. This study determined the prevalence and patterns of gross and histological placental abnormalities and examined associated factors among malaria smear-positive women delivering at two Ugandan referral hospitals. A multicenter hospital-based cross-sectional study was conducted among 124 malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals between 1st May and 1st September 2023. Placentas underwent standardized gross examination, tissue sampling and haematoxylin-and-eosin histology. The primary outcome was the presence of any recorded histological placental abnormality. Multivariable logistic regression was used to identify independently associated factors. Histological placental abnormalities were identified in 79 of 124 placentas (63.7%; 95% CI 55.0–71.6). Placental weight below 500 g occurred in 93 (75.0%) placentas, while 98 (79.0%) had central umbilical-cord insertion and 84 (67.8%) had a diameter of 15–20 cm. Histological lesions were not mutually exclusive. Infarction was the most frequent lesion (62, 50.0%), followed by inflammation (43, 34.7%), fibrin deposition (39, 31.5%), increased syncytial knotting (38, 30.6%), thrombosis (35, 28.2%) and fibrinoid deposition (33, 26.6%). Maternal age < 20 years was associated with higher odds of histological abnormalities than age ≥ 20 years (aOR 14.54; 95% CI 3.64–58.05; $p < 0.001$). BMI ≥ 30 kg/m² was associated with lower odds than BMI < 25 kg/m² (aOR 0.19; 95% CI 0.04–0.83; $p = 0.027$). Histological placental abnormalities were common among malaria smear-positive women. Placental weight < 500 g was the predominant gross finding, while infarction was the most frequent histological lesion. Maternal age < 20 years and BMI ≥ 30 kg/m² were independently associated with the outcome. Malaria prevention and antenatal assessment should be strengthened, particularly for pregnant adolescents, alongside prospective studies with malaria-negative comparison groups.

Keywords Malaria · Pregnancy complications, parasitic · Placenta · Placenta diseases · Maternal age · Uganda

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Abbreviations

ANC:	Antenatal care
CDC:	Centers for disease control and prevention
HIV:	Human Immunodeficiency virus
HPF:	High power field
IPT:	Intermittent preventive treatment
ITNs:	Insecticide-treated nets (ITNs)
JRRH:	Jinja regional referral hospital
KIU-TH:	Kampala international university-teaching hospital
HRRH:	Hoima regional referral hospital
NMCD:	National malaria control division
SP:	Sulfadoxine-pyrimethamine
UMIS:	Uganda malaria indicator survey
WHO:	World Health Organization

Introduction

Malaria remains a major threat to maternal and newborn health, particularly in sub-Saharan Africa. In 2024, an estimated 282 million malaria cases and 610,000 malaria-related deaths occurred worldwide, with the WHO African Region carrying approximately 95% of this burden. In 33 African countries with moderate-to-high malaria transmission, malaria infection was estimated to affect approximately 13 million of 36 million pregnancies during the same year¹. Pregnancy therefore represents an important period of vulnerability, during which malaria can contribute to maternal anaemia, fetal growth restriction, preterm birth, low birth weight and stillbirth^{2,3}.

The placenta is central to the pathogenesis of *Plasmodium falciparum* malaria in pregnancy. Infected erythrocytes express the parasite-derived VAR2CSA protein, which mediates adhesion to chondroitin sulfate A within the placenta, enabling sequestration in the intervillous space⁴. This accumulation may initiate monocyte recruitment, inflammatory signalling, oxidative stress and disruption of placental angiogenesis, nutrient transport and villous development^{5,6}. Histological studies have consequently described intervillous inflammation, increased syncytial knotting, trophoblastic and basement-membrane changes, infarction and fibrin- or fibrinoid-related changes in malaria-exposed placentas^{5,7}. These changes may compromise the placenta's capacity to maintain maternal–fetal exchange and help explain the association between malaria in pregnancy and adverse birth outcomes.

Nevertheless, placental malaria and placental histological abnormality are not synonymous. Placental malaria is ordinarily defined by evidence of malaria parasites and/or haemozoin pigment in placental blood or tissue, whereas lesions such as infarction, thrombosis, inflammation, increased syncytial knotting and fibrin deposition may also occur in hypertensive, vascular, inflammatory and other obstetric conditions^{6,8}. Accordingly, peripheral blood-smear positivity confirms circulating parasitemia at the time of testing but does not demonstrate that every placental lesion was caused by malaria. Conversely, peripheral microscopy may fail to detect low-density, sub-microscopic or predominantly sequestered infections. Studies comparing peripheral blood, placental blood and placental tissue have shown substantial differences in detection according to the biological specimen and diagnostic method used⁹. Placental histology can provide additional evidence of parasites, inflammation and malaria pigment from current or previous infection and may correlate more closely with adverse birth outcomes than a single peripheral or placental blood test at delivery¹⁰.

The susceptibility and placental consequences of malaria are also shaped by the timing, frequency and density of parasitemia and by maternal characteristics. In a high-transmission Ugandan cohort, repeated and higher-density parasitaemia during pregnancy increased the likelihood of histologically defined placental malaria, including among women with infections that were detectable only by molecular methods¹¹. Gravity influences

susceptibility because antibodies against placental-binding parasite variants accumulate across successive pregnancies, although age and gravidity are closely related and their effects may be difficult to separate in small studies⁵. Pregnant adolescents have also been shown to experience more clinical malaria and to have greater odds of peripheral and placental infection at delivery than adult women¹². Maternal comorbidities, nutritional status, antenatal-care attendance and malaria-prevention exposure may further influence placental findings, highlighting the need for careful assessment of potential confounding.

Malaria prevention during pregnancy therefore combines insecticide-treated nets, prompt diagnosis and effective treatment, and intermittent preventive treatment with sulfadoxine–pyrimethamine (IPTp-SP) in eligible women living in moderate-to-high *P. falciparum* transmission settings¹³. Despite these interventions, Ugandan studies demonstrate marked heterogeneity in placental malaria estimates. At Mulago National Referral Hospital, malaria was detected in 9.0% of peripheral smears, 11.3% of placental smears and 13.9% of placental histological examinations¹⁴. A subsequent Mulago study reported substantially different histological burdens according to IPTp-SP exposure¹⁵, while a high-transmission Ugandan trial cohort had a histology-based placental-malaria prevalence of 44.6%¹¹. More recently, a placental-blood rapid diagnostic test study in Lira District reported a prevalence of 4.4%¹⁶. These estimates should not be compared without considering differences in transmission intensity, malaria-prevention exposure, participant selection, timing of infection, diagnostic specimen and case definition.

Although previous Ugandan studies have provided important evidence on placental malaria, comparatively limited information is available on the broader spectrum of gross and microscopic placental findings among women with microscopy-confirmed peripheral malaria delivering at referral hospitals. Evidence is particularly limited for Jinja and Hoima Regional Referral Hospitals, which serve populations with differing malaria-transmission and obstetric referral profiles. Characterising these findings may improve understanding of placental pathology in malaria-positive deliveries, but the absence of a malaria-negative comparison group means that the observed lesions cannot be attributed specifically to malaria. This study therefore aimed to estimate the prevalence of histological placental abnormalities, describe gross and microscopic placental findings, and identify factors associated with the presence of any histological placental abnormality among malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals in Uganda.

Materials and methods

Study design and setting

This multicenter hospital-based cross-sectional study was conducted from 1 st May to 1st September 2023 at Jinja and Hoima Regional Referral Hospitals in Uganda. Jinja Regional Referral Hospital is a public tertiary referral facility in Jinja City, eastern Uganda, while Hoima Regional Referral Hospital is located in Hoima City, western Uganda. Both hospitals provide specialist obstetric and maternity services and receive referrals from surrounding districts and lower-level health facilities. Because placental histopathology services were unavailable at the participating hospitals during the study period, placental specimens were transported to the pathology laboratory at Mbarara Regional Referral Hospital for processing and examination.

Study population and eligibility

The study population comprised malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals. Women were eligible if they had a gestational age of ≥ 28 completed weeks, had malaria parasitaemia confirmed by peripheral blood microscopy, delivered at either participating hospital and provided written informed consent for participation and placental examination. Women who declined participation or placental

sampling were not enrolled. Recruitment and study procedures did not delay or otherwise influence routine obstetric care.

Sample size calculation

The sample was calculated using Kish–Leslie formula

$$N = \frac{Z^2 p(1-p)}{d^2}$$

Where N is the required sample size estimate and Z is the critical value for a normal distribution at the 95% confidence level, corresponding to 1.96. P= was the prevalence of placental abnormalities reported in Uganda of 30.3%¹⁵.

$$q=1-p \text{ Therefore, } N = \frac{1.96^2 * 0.303(1-0.303)}{0.05^2} = 324.5$$

Because the estimated source population was small, a finite-population correction was applied. The finite population ($N=200$) was estimated from delivery and laboratory records at Jinja and Hoima Regional Referral Hospitals, which documented approximately 200 malaria smear-positive pregnant women who would otherwise have met the study eligibility criteria during a comparable four-month period preceding recruitment.

$$n = \frac{n_0}{1 + \frac{(n_0-1)}{N}}$$

n_0 - initial sample size=325.

N - Adjusted sample size=200 at 2 study sites (JRRH and HRRH).

$$= n = 325 / [1 + (325-1)/200] = 123.98.$$

$n=124$ Participants.

Sampling and recruitment

Eligible women were recruited consecutively from the labour wards of the participating hospitals until the required sample size was attained. Trained research assistants identified potentially eligible women after their initial clinical assessment. The study procedures were explained in English, Lusoga or Runyoro, according to participant preference, and written informed consent was obtained before study-specific information or placental specimens were collected.

Data collection procedure

Data were collected using a structured, pretested, interviewer-administered questionnaire. The questionnaire captured sociodemographic characteristics, obstetric history, antenatal-care attendance, IPTp-SP exposure, insecticide-treated-net use and relevant medical history. Clinical and obstetric information was verified against antenatal records and hospital charts where available. Malaria microscopy results, gross placental characteristics and histological findings were recorded on standardized laboratory and placental-assessment forms.

Study variables

The primary outcome was the presence of any histological placental abnormality, coded as present when at least one prespecified lesion was identified on histological examination and absent when no lesion was recorded. The assessed lesions were infarction, inflammation, fibrin deposition, increased syncytial knotting, thrombosis and fibrinoid deposition. Individual lesions were recorded separately, and more than one lesion could occur in the

same placenta. Gross placental characteristics included trimmed placental weight (<500, 500–600 or >600 g), maximum placental diameter (<15, 15–20 or >20 cm) and umbilical-cord insertion (central or non-central).

Potential explanatory variables included maternal age (<20 or ≥ 20 years), marital status, education level, residence, occupation, body mass index (18.5–24.9, 25.0–29.9 or ≥ 30.0 kg/m²), gravidity, antenatal-care attendance, IPTp-SP doses, insecticide-treated-net use, HIV status, diabetes mellitus, chronic hypertension, peripheral parasite density and mode of delivery. Parasite density was categorized as 10–99, 100–1,000 or 1,000–10,000 parasites/ μ L. During the study period, Ugandan guidance recommended initiation of IPTp-SP in the second trimester after 13 weeks of gestation, with subsequent doses administered at each scheduled antenatal-care visit until delivery, provided that successive doses were administered at least one month apart^{1,17}.

Malaria microscopy

Peripheral malaria infection was assessed using thick and thin Giemsa-stained blood films. Thin films were fixed with absolute methanol, whereas thick films were air-dried without fixation. Slides were stained with 10% Giemsa for 40 min and examined by qualified laboratory technologists using a $\times 100$ oil-immersion objective. A smear was classified as positive when asexual malaria parasites were detected.

Parasite density was determined by counting asexual parasites against 200 leukocytes, extending the count to 500 leukocytes when fewer than 10 parasites were detected. Density was calculated as the number of parasites counted multiplied by 8,000 and divided by the number of leukocytes counted. A smear was considered negative after examining 100 high-power fields without detecting asexual parasites. Parasite density was categorized as 10–99, 100–1,000 or 1,000–10,000 parasites/ μ L.

Placental collection and gross examination

Placentas were collected within 30 min of delivery and assigned unique participant identification codes. Each placenta was fixed in 10% neutral-buffered formalin for 24 h and subsequently stored in 70% ethanol at room temperature before transportation for histological processing. Gross examination was performed using a standardized placental assessment form. The membranes and umbilical cord were trimmed before placental weight was recorded. Placental weight was categorized as <500, 500–600 or >600 g. Placental diameter was recorded in centimeters and categorized as <15, 15–20 or >20 cm. Umbilical-cord insertion was initially recorded as central, eccentric or marginal. For statistical analysis, eccentric and marginal insertions were combined as non-central insertion.

Placental tissue sampling and histological examination

A minimum of five standard tissue blocks was prepared from each placenta: one membrane roll, two transverse umbilical-cord sections obtained proximal and distal to the placental insertion, one section incorporating the chorionic plate and villous parenchyma, and one section incorporating the basal plate and villous parenchyma. Additional blocks were obtained from macroscopically visible lesions where present¹⁰. Tissues were dehydrated through graded ethanol, cleared in xylene, embedded in paraffin wax, sectioned using a rotary microtome and stained with haematoxylin and eosin. Histological findings were assessed using standardized morphological criteria. Infarction was defined as localized coagulative necrosis of chorionic villi; inflammation as inflammatory-cell infiltration of intervillous or villous tissue; fibrin deposition as eosinophilic fibrin within intervillous or perivillous spaces; increased syncytial knotting as excessive aggregates of densely packed syncytiotrophoblast nuclei relative to gestational age; thrombosis as an intravascular or intervillous fibrin-containing thrombus; and fibrinoid deposition as homogeneous eosinophilic fibrinoid material replacing or surrounding villous tissue^{8,18}. Each finding was recorded separately as present or absent, and more than one finding could occur in the same placenta.

Quality control

Research assistants received standardized training in participant recruitment, informed-consent procedures and questionnaire administration. The questionnaire was pilot-tested among approximately 10% of the calculated sample at Kampala International University Teaching Hospital, and pilot participants were excluded from the main study. Completed questionnaires were reviewed daily by the principal investigator for completeness and consistency. Unique study codes were used to link questionnaires, malaria microscopy results and placental specimens.

Placental collection, fixation, tissue processing and haematoxylin-and-eosin staining followed standardized laboratory procedures. All slides were initially examined by one pathologist, and a randomly selected 10% were independently reviewed by a second pathologist. Discordant interpretations were resolved by joint review and consensus. Formal blinding to clinical information was not documented, and interobserver agreement statistics were not calculated.

Malaria smears were prepared, stained and examined by qualified laboratory technologists according to the participating hospitals' standard operating procedures. Smear and staining quality were checked before interpretation. Before analysis, the database was checked for completeness, duplicate records, implausible values and internal inconsistencies.

Data management and analysis

Data from questionnaires, laboratory forms and placental histology reports were entered into Microsoft Excel 2016 and analysed using Stata version 14.2 (StataCorp, College Station, TX, USA). Continuous variables were summarized using means with standard deviations or medians with interquartile ranges, as appropriate, while categorical variables were summarized using frequencies and percentages. The prevalence of histological placental abnormalities was calculated as the proportion of examined placentas with at least one recorded abnormality, with a 95% confidence interval. Associations with histological placental abnormalities were assessed using binary logistic regression. Candidate variables were selected based on biological plausibility and/or univariable screening at $p < 0.20$. The final multivariable model included maternal age, body mass index, parasite-density category, antenatal-care attendance, diabetes mellitus and chronic hypertension. Results were reported as crude and adjusted odds ratios with 95% confidence intervals and category-specific p -values. All tests were two-sided, and statistical significance was defined as $p < 0.05$.

Results

A total of 300 women were screened for malaria; 126 were smear-positive and two declined participation, leaving 124 participants for the final analysis. Their median age was 21 years (IQR 19–26), and 80 (64.5%) were aged ≥ 20 years. Most participants resided in rural areas (82, 66.1%), were married (63, 50.8%), had secondary education (64, 51.6%) and were housewives (103, 83.1%) (Table 1).

Prevalence of histological placental abnormalities among malaria smear-positive women ($N = 124$)

Among the 124 malaria smear-positive women, 79 had histological placental abnormalities, corresponding to a prevalence of 63.7% (95% CI 55.0–71.6).

Table 1 Sociodemographic characteristics of malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals, Uganda (N= 124).

Characteristic	Frequencies (n)	Percentages (%)
Maternal age, years		
<20	44	35.5
≥20	80	64.5
Marital status		
Married	63	50.8
Not married	61	49.2
Education level		
No formal education	25	20.2
Primary	29	23.4
Secondary	64	51.6
Tertiary	6	4.8
Residence		
Urban	42	33.9
Rural	82	66.1
Occupation		
Housewife	103	83.1
Employed	9	7.2
Other ¹	12	9.7

¹Other comprised students (n=4) and occupations recorded as ‘other’ without further specification (n=8).

Table 2 Gross placental characteristics and histological findings among malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals, Uganda, (N= 124).

Placental characteristic or finding	Frequencies (n)	Percentages (%)
Gross placental characteristics		
Placental weight, g		
<500	93	75.0
500–600	22	17.7
>600	9	7.3
Umbilical-cord insertion		
Central	98	79.0
Non-central	26	21.0
Placental diameter, cm		
<15	35	28.2
15–20	84	67.8
>20	5	4.0
Histological placental findings		
Infarction	62	50.0
Inflammation	43	34.7
Fibrin deposition	39	31.5
Increased syncytial knotting	38	30.6
Thrombosis	35	28.2
Fibrinoid deposition	33	26.6

Histological findings were not mutually exclusive; more than one finding could be recorded in the same placenta. Percentages were calculated using all 124 examined placentas as the denominator. Histological findings were not mutually exclusive; more than one finding could be recorded in the same placenta. Percentages were calculated using all 124 examined placentas as the denominator.

Gross and histological placental findings among malaria smear-positive women

Placental weight below 500 g was observed in 93 of the 124 placentas (75.0%). Most placentas had central umbilical-cord insertion (98, 79.0%), while 26 (21.0%) had non-central insertion. A placental diameter of 15–20 cm was recorded in 84 placentas (67.8%), whereas 35 (28.2%) measured less than 15 cm and five (4.0%) measured more than 20 cm. Histological findings were not mutually exclusive; therefore, more than one lesion could be recorded in the same placenta. Infarction was the most frequently recorded lesion (62, 50.0%), followed by inflammation (43, 34.7%), fibrin deposition (39, 31.5%), increased syncytial knotting (38, 30.6%), thrombosis (35, 28.2%) and fibrinoid deposition (33, 26.6%) (Table 2).

Factors associated with histological placental abnormalities among malaria smear-positive women (N = 124)

In univariable logistic regression, maternal age below 20 years, being not married, having no formal education, BMI ≥ 30 kg/m², parasite density of 1,000–10,000 parasites/ μ L and fewer than four antenatal-care visits had category-specific *p*-values below 0.20 and were considered for multivariable analysis. Diabetes mellitus and chronic hypertension were additionally considered because of their clinical plausibility. The *p* < 0.20 threshold was used only for candidate-variable screening and did not define statistical significance (Table 3).

After adjustment for potential confounding factors, maternal age below 20 years and BMI ≥ 30 kg/m² remained independently associated with histological placental abnormalities. Women aged below 20 years had significantly higher adjusted odds of histological placental abnormalities than women aged ≥ 20 years (aOR = 14.54; 95% CI: 3.64–58.05; *p* < 0.001). Conversely, women with BMI ≥ 30 kg/m² had 81% lower adjusted odds than women with BMI 18.5–24.9 kg/m² (aOR = 0.19; 95% CI: 0.04–0.83; *p* = 0.027). Parasite density, antenatal-care attendance, diabetes mellitus and chronic hypertension were not independently associated with the outcome (Table 4).

Discussion

This study found that histological placental abnormalities were common among malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals. Overall, 79 of 124 women had at least one recorded abnormality, corresponding to a prevalence of 63.7% (95% CI 55.0–71.6). Gross examination showed that 75.0% of placentas weighed below 500 g, 79.0% had central umbilical-cord insertion and 67.8% had a diameter of 15–20 cm. The predominant microscopic findings were infarction, inflammation, fibrin deposition, increased syncytial knotting, thrombosis and fibrinoid deposition. After adjustment, maternal age below 20 years was associated with substantially higher odds of the composite histological outcome, whereas BMI ≥ 30 kg/m² was inversely associated with it.

The observed prevalence was numerically higher than estimates from several Ugandan studies of histologically confirmed placental malaria. Namusoke et al. reported placental malaria in 13.9% of women by histology at Mulago National Referral Hospital¹⁴, while Briggs et al. reported a prevalence of 44.6% in a high-transmission setting in eastern Uganda¹¹. At Mulago Hospital, Odongo et al. found histological placental malaria in 12% of women who had received the specified IPTp-SP exposure and 48% of those who had not¹⁵. By comparison, Ezebialu et al. (2012) reported histological evidence of placental malaria in 69.6% of 365 parturients in southeastern Nigeria¹⁸.

These estimates are not directly comparable because they represent different outcomes and study populations. Most previous studies defined placental malaria by identifying parasitized erythrocytes and/or malaria pigment and, in some cases, classified infection as acute, chronic or past. The present study instead defined its primary outcome as the presence of at least one of six recorded histological lesions. Moreover, all participants were malaria-positive by peripheral blood-smear microscopy, whereas several comparator studies enrolled women regardless of malaria status. Differences in malaria-transmission intensity, gravidity, IPTp-SP exposure, infection timing and diagnostic sensitivity may further explain the variation^{5,6}.

Diagnostic method is particularly important. Anchang-Kimbi et al. demonstrated markedly different detection rates when peripheral blood, placental blood and placental tissue were examined in the same women⁹. Cardona-Arias and Carmona-Fonseca reported that 92% of placental infections detected using placental blood smear and qPCR in Colombia were submicroscopic¹⁹, while Arango et al. found that submicroscopic infections were associated with villitis and intervillitis²⁰.

In Uganda, repeated microscopy and ultrasensitive qPCR demonstrated that the frequency, density and timing of parasitaemia during pregnancy influenced the risk of histologically confirmed placental malaria¹¹. The absence

Table 3 Univariable logistic-regression analysis of factors associated with histological placental abnormalities among malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals, Uganda ($N=124$).

Variable	Category	Abnormality absent, n (%)	Abnormality present, n (%)	cOR (95% CI)	p -value
Maternal age, years	<20	3 (6.8)	41 (93.2)	15.11 (4.32–52.81)	<0.001
	≥20	42 (52.5)	38 (47.5)	1.00 (Reference)	—
Marital status	Married	34 (54.0)	29 (46.0)	1.00 (Reference)	—
	Not married	11 (18.0)	50 (82.0)	5.33 (2.35–12.09)	<0.001
Education level	No formal education	4 (16.0)	21 (84.0)	5.25 (0.77–35.98)	0.091
	Primary	7 (24.1)	22 (75.9)	3.14 (0.51–19.25)	0.216
	Secondary	31 (48.4)	33 (51.6)	1.06 (0.20–5.68)	0.942
	Tertiary	3 (50.0)	3 (50.0)	1.00 (Reference)	—
Residence	Urban	15 (35.7)	27 (64.3)	1.00 (Reference)	—
	Rural	30 (36.6)	52 (63.4)	0.96 (0.44–2.09)	0.924
Occupation	Housewife	39 (37.9)	64 (62.1)	1.00 (Reference)	—
	Employed	3 (33.3)	6 (66.7)	1.22 (0.29–5.15)	0.788
	Other	3 (25.0)	9 (75.0)	1.83 (0.47–7.17)	0.387
Body mass index, kg/m ²	18.5–24.9	13 (39.4)	20 (60.6)	1.00 (Reference)	—
	25.0–29.9	26 (31.7)	56 (68.3)	1.40 (0.60–3.24)	0.432
	≥30	6 (66.7)	3 (33.3)	0.32 (0.07–1.53)	0.156
Diabetes mellitus	Yes	1 (16.7)	5 (83.3)	2.97 (0.34–26.28)	0.327
	No	44 (37.3)	74 (62.7)	1.00 (Reference)	—
Parasite density, parasites/ μ L	10–99	24 (45.3)	29 (54.7)	1.00 (Reference)	—
	100–1,000	17 (33.3)	34 (66.7)	1.66 (0.75–3.66)	0.214
	1,000–10,000	4 (20.0)	16 (80.0)	3.31 (0.98–11.23)	0.055
	Yes	3 (33.3)	6 (66.7)	1.15 (0.27–4.84)	0.848
HIV status	No	42 (36.5)	73 (63.5)	1.00 (Reference)	—
	Yes	1 (20.0)	4 (80.0)	2.35 (0.25–21.67)	0.452
Chronic hypertension	Yes	1 (20.0)	4 (80.0)	2.35 (0.25–21.67)	0.452
	No	44 (37.0)	75 (63.0)	1.00 (Reference)	—
Gravidity	Primigravida	33 (37.9)	54 (62.1)	1.00 (Reference)	—
	Multigravida	10 (34.5)	19 (65.5)	1.16 (0.48–2.80)	0.739
	Grand	2 (25.0)	6 (75.0)	1.83 (0.35–9.62)	0.474
	Multigravida				
ANC visits	<4	33 (42.3)	45 (57.7)	0.48 (0.22–1.07)	0.072
	≥4	12 (26.1)	34 (73.9)	1.00 (Reference)	—
IPTp-SP doses	<3	3 (25.0)	9 (75.0)	1.80 (0.46–7.02)	0.397
	≥3	42 (37.5)	70 (62.5)	1.00 (Reference)	—
Insecticide-treated-net use	No	6 (40.0)	9 (60.0)	0.84 (0.28–2.52)	0.750
	Yes	39 (35.8)	70 (64.2)	1.00 (Reference)	—
Mode of delivery	Caesarean delivery	11 (45.8)	13 (54.2)	0.61 (0.25–1.50)	0.282
	Vaginal delivery	34 (34.0)	66 (66.0)	1.00 (Reference)	—
Placental weight, g	<500	30 (32.3)	63 (67.7)	1.45 (0.56–3.78)	0.442
	500–600	9 (40.9)	13 (59.1)	1.00 (Reference)	—
	>600	6 (66.7)	3 (33.3)	0.35 (0.07–1.76)	0.201
Umbilical-cord insertion	Central	35 (35.7)	63 (64.3)	1.00 (Reference)	—
	Non-central	10 (38.5)	16 (61.5)	0.89 (0.36–2.17)	0.796
Placental diameter, cm	<15	12 (34.3)	23 (65.7)	1.18 (0.52–2.69)	0.695
	15–20	32 (38.1)	52 (61.9)	1.00 (Reference)	—
	>20	1 (20.0)	4 (80.0)	2.46 (0.26–23.01)	0.430

cOR, crude odds ratio; CI, confidence interval; ANC, antenatal care; IPTp-SP, intermittent preventive treatment of malaria in pregnancy with sulfadoxine-pyrimethamine. Reference categories are indicated. A category-specific $p<0.20$ was used for candidate-variable screening and did not define statistical significance.

Table 4 Multivariable logistic-regression analysis of factors associated with histological placental abnormalities among malaria smear-positive women delivering at Jinja and Hoima Regional Referral Hospitals, Uganda ($N=124$).

Variable	Category	aOR (95% CI)	<i>p</i> -value
Maternal age, years	<20	14.54 (3.64–58.05)	<0.001*
	≥20	1.00 (Reference)	—
Body mass index, kg/m ²	18.5–24.9	1.00 (Reference)	—
	25.0–29.9	0.77 (0.20–3.00)	0.705
	≥30	0.19 (0.04–0.83)	0.027*
Parasite density, parasites/μL	10–99	1.00 (Reference)	—
	100–1,000	1.55 (0.60–4.04)	0.368
	1,000–10,000	2.41 (0.56–10.43)	0.240
Antenatal-care visits	<4	0.49 (0.19–1.27)	0.143
	≥4	1.00 (Reference)	—
Diabetes mellitus	Yes	2.67 (0.20–35.86)	0.458
	No	1.00 (Reference)	—
Chronic hypertension	Yes	0.70 (0.06–8.71)	0.785
	No	1.00 (Reference)	—

aOR, adjusted odds ratio; CI, confidence interval. The model was adjusted simultaneously for all variables shown. Reference categories are indicated.

* $p < 0.05$.

of molecular testing and longitudinal assessment in the present study may therefore have limited characterization of low-density, earlier and recurrent infections.

Three-quarters of the placentas weighed below 500 g. This finding may indicate reduced placental growth in some women but should not, by itself, be interpreted as pathological placental smallness. Placental weight varies with gestational age, fetal size and maternal characteristics and is affected by whether the cord and membranes are removed before weighing. The Amsterdam Placental Workshop Group recommends standardized trimming, weighing and gestational-age-specific interpretation of placental weight⁸. In a small Kenyan retrospective cohort, women with malaria–pre-eclampsia comorbidity had lower mean placental and neonatal weights than women with healthy pregnancies. However, because malaria and pre-eclampsia occurred together, their individual contributions could not be separated²¹. Consequently, the current finding is best reported descriptively as “placental weight below 500 g,” particularly because gestational-age-specific placental-weight percentiles and a malaria-negative comparison group were unavailable.

Most placentas had central umbilical-cord insertion, whereas 21.0% had non-central insertion. Clinically important abnormal insertion is generally defined more specifically as marginal or velamentous insertion. In a population-based study of 634,741 pregnancies, marginal and velamentous insertion together occurred in 7.8% of singleton pregnancies and was associated with increased risks of preterm birth, pre-eclampsia, placental abruption, low birth weight and other adverse outcomes²². A systematic review found that included studies reported associations between abnormal cord insertion and preterm birth, small-for-gestational-age birth, low birth weight, intrauterine fetal death and emergency caesarean delivery. However, inconsistent definitions limited quantitative synthesis, and the clearest pooled evidence concerned emergency caesarean delivery²³. Direct comparison with the present 21.0% is inappropriate because “non-central” combined eccentric and marginal insertions, while velamentous insertion was not reported separately. Eccentric insertion is not equivalent to marginal or velamentous insertion; therefore, the cord-insertion result should remain descriptive and should not be attributed to malaria.

Most placentas measured 15–20 cm in diameter, while 28.2% measured below 15 cm. As with placental weight, diameter varies with gestational age, fetal growth, specimen completeness and measurement procedure. It should ideally be interpreted alongside placental thickness, weight, gestational age and neonatal birth weight. The present study therefore describes the distribution of placental dimensions but cannot determine whether malaria caused reduced placental size.

Infarction was the most frequent microscopic lesion, occurring in 50.0% of placentas. Placental infarction represents loss of viable villous tissue following impaired maternal intervillous perfusion. Inflammation, intervillous fibrin deposition and infarction have been described in malaria-affected placentas, and active infection has been associated with reduced villous area and vascularity²⁴. However, infarction is not specific to malaria and may

also reflect maternal vascular malperfusion, chronic hypertension or pre-eclampsia. Without a malaria-negative comparison group, malaria cannot be considered the sole explanation for the high frequency of infarction.

Inflammation was recorded in 34.7% of placentas. Sequestration of *Plasmodium falciparum*-infected erythrocytes within the intervillous space can promote maternal monocyte recruitment, cytokine production, oxidative stress and trophoblast injury^{6,25}. Primary histological studies have documented intervillous inflammatory infiltration in active placental malaria, frequently accompanied by fibrin deposition, basement-membrane thickening and increased syncytial knotting⁷. Nevertheless, placental inflammation has several infectious and non-infectious causes. Because the anatomical compartment, inflammatory-cell type and lesion severity were not separately reported, this finding should remain “placental inflammation” rather than being labelled malaria-associated intervillitis.

Fibrin deposition and fibrinoid deposition were recorded in 31.5% and 26.6% of placentas, respectively. Fibrin-related changes have been documented following trophoblast injury in malaria-exposed placentas. Ismail et al. (2000) reported increased fibrin deposition and fibrinoid necrosis in active placental malaria⁷, while Crocker et al. (2004) described syncytiotrophoblast disruption, increased syncytial knotting and fibrin-type fibrinoid deposition in malaria-affected placentas²⁶. These findings provide biological context for the fibrin-related changes observed in the present study. However, the two variables should be retained separately only if the pathology protocol confirms that they had distinct operational definitions and were independently recorded.

Increased syncytial knotting and thrombosis were present in 30.6% and 28.2% of placentas, respectively. Syncytial knots may increase with accelerated villous maturation and impaired placental oxygenation, while placental thrombi may reflect vascular stasis, endothelial injury or maternal or fetal vascular malperfusion. Both findings are biologically compatible with placental injury during malaria, but neither is malaria-specific. Obiri et al. demonstrated that placental histological lesions, including increased syncytial knotting, and active or past placental malaria were related but independent factors associated with pre-eclampsia²⁷. Human placental observations and experimental evidence further suggest that malaria-associated inflammation may be accompanied by increased coagulation activity, reduced fibrinolysis and excessive fibrin deposition, although these mechanisms were not assessed in the present study²⁸.

Taken together, the microscopic findings are compatible with inflammatory, trophoblastic and vascular injury previously described in malaria-exposed placentas. These changes could interfere with maternal–fetal exchange and placental growth. Placental-malaria-associated inflammation has been linked to disruption of the insulin-like growth factor axis, providing a plausible pathway to impaired fetal growth²⁹. Acute histologically confirmed *P. falciparum* placental infection has also been associated with lower maternal haemoglobin, shorter gestation, lower birth weight and stillbirth³⁰. However, these clinical consequences cannot be inferred from the present study because maternal and neonatal outcomes were not systematically related to individual histological lesions.

After adjustment, women aged below 20 years had substantially higher odds of the composite histological outcome than women aged 20 years or older (aOR 14.54; 95% CI 3.64–58.05; $p < 0.001$). This finding is consistent with broader evidence that pregnant adolescents experience a greater malaria burden. In an individual-participant-data meta-analysis of 5,804 women from five sub-Saharan African countries, adolescents experienced more clinical malaria and had higher odds of peripheral and placental infection at delivery than adult women¹². Bihoun et al. similarly found that younger age was associated with histologically confirmed placental malaria among women with peripheral malaria in Burkina Faso³¹.

Younger women may have less cumulative antimalarial immunity and are more likely to be in their first or second pregnancy, before acquiring sufficient pregnancy-specific immunity against placental-binding parasite variants. Infection timing may also differ by gravidity: longitudinal Ugandan evidence suggests that early infection is particularly relevant among Primigravida, whereas later infection is more strongly associated with placental malaria among multigravida³². Nevertheless, the magnitude of the present association requires caution. Histological abnormalities occurred in 41 of 44 women aged below 20 years, leaving only three without the outcome and producing a wide confidence interval. Gravidity was also not retained in the final model; therefore, the independent effects of young age and early gravidity cannot be fully separated.

Women with BMI ≥ 30 kg/m² had lower adjusted odds of the composite histological outcome than women with BMI 18.5–24.9 kg/m² (aOR 0.19; 95% CI 0.04–0.83; $p=0.027$). No comparable association was observed for BMI 25.0–29.9 kg/m², indicating no clear dose–response pattern. Only nine participants had BMI ≥ 30 kg/m², of whom three had the outcome, making the estimate vulnerable to sparse-data bias, residual confounding and random variation. The finding should therefore not be interpreted as evidence that obesity protects against placental injury.

The inverse BMI association also contrasts with Tanzanian evidence showing higher odds of histology-positive placental malaria among overweight or obese women than among women with normal BMI³³. This discrepancy may reflect differences in outcome definition, participant selection, malaria-transmission intensity, timing of BMI assessment and control of confounding. If BMI was measured near delivery, gestational weight gain, oedema and pregnancy-related tissue mass may have affected classification. Restriction of the study to peripheral smear-positive women may also have influenced the observed relationship. The BMI finding is therefore best considered hypothesis-generating.

Parasite density increased directionally across exposure categories, but the adjusted estimates were imprecise and were not statistically significant. This differs from the Ugandan study by Briggs et al., in which the frequency, density and timing of parasitaemia predicted histologically confirmed placental malaria¹¹. The difference may reflect single-time-point assessment of parasite density, limited sample size and use of a composite lesion outcome rather than parasite- or pigment-defined placental malaria. Antenatal-care attendance, diabetes mellitus and chronic hypertension were also not independently associated with the outcome. These results indicate insufficient statistical evidence of independent associations; they do not establish the absence of clinically meaningful relationships, particularly because diabetes and chronic hypertension were uncommon and their estimates were imprecise.

Strengths and limitations

This multicenter study provides evidence from two Ugandan regional referral hospitals serving geographically distinct populations. Consecutive recruitment reduced selection among eligible women, while combined gross and microscopic placental examination enabled comprehensive assessment of placental findings. Individual histological lesions were recorded separately, allowing overlapping abnormalities, and a prespecified composite outcome supported consistent prevalence estimation and regression analysis. The multivariable model was kept parsimonious relative to the 79 outcome events.

Several limitations should be considered. The cross-sectional design prevents assessment of temporality or causality. Because only malaria smear-positive women were recruited and no malaria-negative comparison group was included, the findings cannot be generalized to all pregnant women or used to attribute the observed lesions specifically to malaria. Infarction, inflammation, thrombosis, syncytial knotting and fibrin-related changes may also occur in other obstetric conditions. Peripheral microscopy may not fully characterize infection across pregnancy, and molecular testing, placental-blood testing, timing of IPTp-SP doses and recent malaria treatment were unavailable. The composite outcome combined heterogeneous, non-mutually exclusive lesions and may have obscured lesion-specific associations. The modest sample size and small numbers in some categories produced imprecise estimates and possible sparse-data bias, particularly for maternal age and body mass index. Residual confounding is also possible because some maternal, socioeconomic and clinical variables were unavailable or incompletely measured. Finally, maternal and neonatal outcomes were not systematically evaluated; therefore, the clinical consequences of the placental findings could not be established.

Conclusions and recommendations

Histological placental abnormalities were common among malaria smear-positive women delivering at the two Ugandan referral hospitals, affecting nearly two-thirds of participants. Low placental weight was the predominant

gross finding, while infarction, inflammation, fibrin deposition and increased syncytial knotting were the most frequent histological lesions. Maternal age below 20 years was independently associated with higher odds of placental abnormalities, whereas body mass index ≥ 30 kg/m² showed an inverse association that should be interpreted cautiously because of the small number of women in this category. These findings do not establish that malaria caused the observed lesions because the study was cross-sectional, the abnormalities were not malaria-specific, and no malaria-negative comparison group was included. Prospective studies incorporating comparison groups, molecular malaria diagnostics, standardized placental assessment and maternal and neonatal outcomes are needed to confirm these associations and determine their clinical significance.

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Author contributions YL, PB and TH designed and developed the proposal. MPSI, TN performed the data collection and entry. TH and RA performed the statistical analysis. YL, TN, PB and MPSI drafted the initial manuscript. TH contributed to reviewing and revising the manuscript. The final manuscript was read and approved by all the authors.

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Data availability The dataset that was utilized in this study is not publicly available due to ethical considerations. Upon reasonable request, the dataset used can be accessed with the permission of the corresponding author Dr Theoneste Hakizimana (email: theonestehakizimana5@gmail.com).

Declarations

Competing interests The authors declare no competing interests.

Human ethics and consent to participate Ethical approval was obtained from the Kampala International University Research Ethics Committee (KIU-REC-2022-023), and administrative permission was obtained from Jinja and Hoima Regional Referral Hospitals. Participants aged 18 years or older provided written informed consent. Participants younger than 18 years who met the Research Ethics Committee criteria for mature or emancipated minors provided written informed consent on their own behalf; otherwise, written assent and permission from a parent or legal guardian were obtained. Participation was voluntary, and refusal or withdrawal did not affect the care provided. Privacy and confidentiality were maintained using unique study codes and restricted access to study records. The study was conducted in accordance with the principles of the Declaration of Helsinki.

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Authors and Affiliations

Yumbi Lutula¹ · Petrus Baruti¹ · Marie Pascaline Sabine Ishimwe² · Raymond Atwiine³ · Theodore Nteziyaremye⁴ · Anyanwu Godson Emeka⁵ · Theoneste Hakizimana¹

✉ Theoneste Hakizimana
theonestehakizimana5@gmail.com

¹ Department of Obstetrics and Gynecology, Kampala International University, Ishaka, Uganda

² Department of Pediatrics and Child Health, Kampala International University, Ishaka, Uganda

³ Department of Pathology, Mbarara University of Science and Technology, Mbarara, Uganda

⁴ Department of sciences, University of Rwanda, Kigali, Rwanda

⁵ Department of Anatomy, Faculty of Biomedical Sciences Kampala, International University, Ishaka, Bushenyi, Uganda