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Review Article

Prevalence and Risk Factors of dengue fever in pregnant women

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Abstract

Background: Dengue infects an estimated 390 million people a year, and pregnancy obscures its warning signs because gestational haemodilution and overlapping obstetric symptoms mask the changes that ordinarily flag progression to severe disease [1,2].

Objective: This review synthesises the evidence on dengue prevalence among pregnant women, the risk factors for infection and severe disease, and the resulting maternal, fetal and neonatal outcomes, including vertical transmission.

Methods: We synthesised hospital-based screening cohorts, obstetric case series, matched case-control studies, registry-linkage analyses and systematic reviews addressing dengue in pregnant populations.

Findings: Dengue positivity among symptomatic pregnant women ranged from 14% to 32.7% across screening cohorts [8,9]; marked thrombocytopenia and elevated transaminases identified women at highest risk of death [14,15]; and Brazilian registry-linkage studies found symptomatic dengue independently associated with stillbirth and preterm birth [5,6], with vertical transmission concentrated near delivery [13,18].

Conclusions: Dengue in pregnancy carries a reproducible burden of maternal mortality, stillbirth, preterm birth and timing-dependent vertical transmission across distinct cohorts, and closing remaining gaps will require linked population-level surveillance as already shown in Brazil [7,16].

Keywords: dengue; pregnancy; vertical transmission; risk factors; maternal outcomes

1. Introduction

Dengue has moved, over the past three decades, from a disease confined to scattered tropical outbreaks to one that the most recent global mapping efforts estimate infects on the order of 390 million people a year across more than 125 countries [1]. Pregnant women are not a special risk group entomologically, since *Aedes aegypti* and *Aedes albopictus* bite pregnant and non-pregnant women alike, but pregnancy changes how the infection is recognised and managed once it occurs. Gestational haemodilution lowers the baseline platelet count and haematocrit against which dengue's warning signs are ordinarily read, the abdominal pain and vomiting of early warning-sign dengue overlap with obstetric emergencies from placental abruption to appendicitis, and the plasma leakage that defines severe dengue can be mistaken for, or can precipitate, pre-eclampsia [2,3]. A clinician who anchors on pregnancy-specific diagnoses first, in a woman actually developing dengue shock syndrome, loses the narrow window in which fluid management determines survival.

The literature on how often dengue occurs in pregnancy, and which women are most likely to acquire it or progress to severe disease, is more fragmented than the burden of the problem suggests. Much of what is known comes from single-centre cohorts in endemic countries, screening programmes nested within obstetric services, and a smaller number of large, linked-registry studies from Brazil that have compared thousands of dengue-exposed pregnancies against unexposed controls [4,5,6,7]. This review draws that literature together to characterise measured prevalence, recurring risk factors for infection and severity, and the resulting maternal, fetal and neonatal outcomes, including vertical transmission.

2. Global epidemiology and burden of dengue in pregnancy

Dengue's expansion has been driven by urbanisation, travel and a warming climate extending the range in

which *Aedes* mosquitoes complete their life cycle, and Bhatt and colleagues' widely cited modelling exercise placed the annual number of apparent and inapparent infections at roughly 390 million, with about 96 million manifesting some clinical severity [1]. Dengue is hyperendemic across much of South and Southeast Asia, Latin America and parts of sub-Saharan Africa, where fertility also remains comparatively high, so a very large number of pregnancies occur each year in settings of continuous or strongly seasonal transmission. Most surveillance systems do not routinely flag pregnancy status on a dengue notification, so the true denominator of pregnant women exposed in a given season is rarely known with precision.

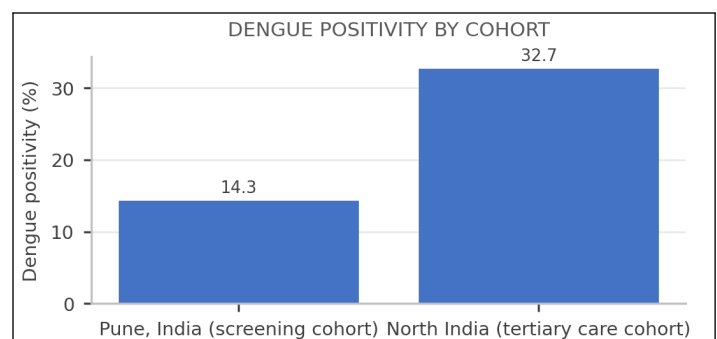


Figure 1: Dengue positivity among symptomatic pregnant women in two hospital-based screening cohorts, 14% (7 of 49) in a symptom-screening programme in Pune, India, against 32.7% (34 of 104) in a tertiary-care cohort in north India also screened for chikungunya and Zika virus, illustrating how strongly background transmission intensity and the testing threshold shape reported prevalence [8,9].

Where hospital-based screening has been done systematically, the proportion of febrile pregnant women found dengue-positive varies widely with transmission intensity, case definition and testing window. In a prospective symptom-screening programme in Pune, India, enrolling women with fever plus a second symptom during an active season, 7 of 49 women with completed testing (14%) were confirmed dengue-positive, and eye pain carried an odds ratio of 7.5 for a positive result, with conjunctivitis present in all seven confirmed cases [8]. A tertiary-care hospital in north India testing symptomatic pregnant women for dengue, chikungunya and Zika virus

found a substantially higher dengue positivity of 32.7% (34 of 104), with chikungunya positive in 9.6% and dual infection in 5.8%, no Zika detected, and dengue associated with higher hospitalisation and mortality than the other two arboviruses in the same cohort [9]. That roughly two-fold difference in positivity, shown in Figure 1, illustrates how strongly local transmission intensity and testing threshold shape the prevalence figure a given hospital reports, and cautions against treating any single study's percentage as representative more broadly.

3. Pathophysiology and clinical presentation in pregnancy

Dengue produces a spectrum from asymptomatic or mild febrile illness through dengue with warning signs to severe dengue, defined by the 2009 World Health Organization classification according to significant plasma leakage, severe bleeding, or severe organ impairment [10]. The pathophysiological event underlying progression is increased vascular permeability driven by the host immune response, and the most consistently reproduced risk factor for that progression, in pregnant and non-pregnant populations alike, is secondary infection with a heterologous serotype, in which pre-existing, sub-neutralising antibody is thought to enhance rather than block viral entry into Fc-receptor-bearing cells, generally termed antibody-dependent enhancement [11,3].

Pregnancy overlays this general risk architecture with features specific to gestation. Expanded plasma volume, a falling haematocrit, and a platelet count that runs somewhat lower than the non-pregnant reference range even in healthy pregnancies blunt the sensitivity of the haemoconcentration and thrombocytopenia signals clinicians rely on to detect early plasma leakage [2]. Basurko and colleagues, studying dengue in pregnancy in French Guiana, documented presentations of non-specific abdominal pain, vomiting and uterine contractions difficult to distinguish from obstetric causes

at first assessment, delaying dengue-specific management in a meaningful minority of cases [12]. Tan and colleagues, describing dengue across all three trimesters in Malaysia, reported that clinical severity and the risk of vertical transmission both varied with the trimester of infection, with infection close to delivery carrying the highest risk of the neonate acquiring detectable viraemia [13].

4. Risk factors for infection and progression to severe dengue

Three broad categories of risk factor recur across the pregnancy-specific literature: host immunological status, obstetric and clinical presentation, and access to and timing of care. Secondary heterologous infection has the strongest mechanistic and epidemiological support for progression to severe disease generally [11,3], though most pregnancy cohorts lack the paired acute and convalescent serology needed to establish infection sequence, limiting how directly this factor can be quantified in pregnant women specifically. Clinical predictors identified within pregnancy cohorts have instead been markers available at presentation. Naik and colleagues' screening study identified eye pain with conjunctivitis as a symptom-level predictor strong enough to justify adding it to routine screening alongside fever and rash [8]. Brar and colleagues, following 44 pregnant women with confirmed dengue in a large prospective Indian cohort, found that every woman who died had presented with a temperature between 100 and 102°F, a platelet count below 100,000/μL, and transaminases more than twice the upper limit of normal, a combination that functioned as a bedside marker for the women at highest risk of a fatal course [14]. Sutharsika and colleagues reported a related pattern from South India, where adverse maternal and perinatal outcomes concentrated specifically among women presenting with severe dengue or warning signs later in gestation [15], summarised together with the two preceding studies in Table 1.

Table 1: Clinical risk markers for dengue infection or progression to severe disease identified in three pregnancy-specific cohorts [8,14,15].

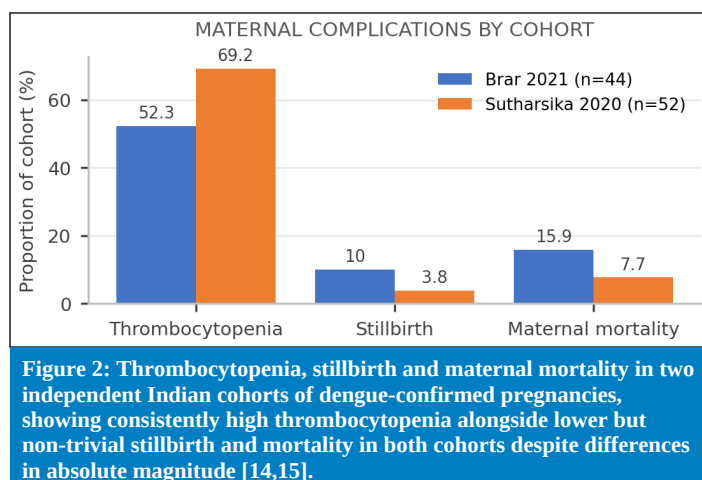
Study (setting, n)	Risk marker identified	Association reported
[8]	Eye pain / conjunctivitis at presentation	OR 7.5 for dengue positivity; conjunctivitis in 7/7 confirmed cases
[14]	Temperature 100-102F + platelets <100,000/uL + transaminases >2x ULN	Present in all maternal deaths (7/44, 15.9%)
[15]	Severe dengue / warning signs later in gestation	Adverse outcomes concentrated in this subgroup (65.3% of cohort)

Access-related factors compound these clinical markers. Because early warning-sign dengue mimics common obstetric complaints, women in settings with limited arbovirus testing capacity are more likely to have management delayed until warning signs are unmistakable, narrowing the window for fluid-management intervention [12,16]. Kallur and colleagues, reviewing a retrospective cohort at an Indian tertiary maternity hospital, argued explicitly for dengue-specific management guidelines within obstetric units, because existing general dengue protocols did not address pregnancy's altered fluid-balance requirements, and delayed recognition recurred as a contributor to their more severe outcomes [16]. Co-circulation of chikungunya and, in some regions, Zika virus, sharing the same vector and overlapping presentations, adds a further diagnostic risk, a gap Gupta and colleagues argued for closing through routine sentinel co-testing of symptomatic pregnant women [9].

5. Maternal outcomes

The maternal complication profile of dengue in pregnancy mirrors that in non-pregnant adults, thrombocytopenia, plasma leakage, haemorrhage and, in the most severe cases, shock and multi-organ dysfunction, but pregnancy adds obstetric complications on top. Brar and colleagues' cohort recorded thrombocytopenia in 23 of 44 women (52.3%), severe thrombocytopenia below 50,000/ μ L in 19 (43.2%), shock in 18 (40.9%), postpartum haemorrhage in 10 of 40 deliveries (25%), and maternal mortality in 7 of 44 women (15.9%), all of whom had presented with the

temperature, platelet and transaminase combination described above [14]. Sutharsika and colleagues' South Indian cohort of 52 dengue-confirmed pregnancies found a comparably high thrombocytopenia rate of 69.2% and a lower but still substantial maternal mortality of 7.7%, concentrated entirely among women with severe dengue [15]. These two cohorts, set out together in Figure 2, converge on one picture: maternal mortality clusters tightly around a small subset of women identifiable at presentation, rather than occurring unpredictably across the whole infected population.



Beyond acute severity, dengue in pregnancy has also been linked to obstetric complications independent of overall severity. Paixão and colleagues' Brazilian case-control analysis, matching over a thousand dengue-exposed pregnancies to unexposed controls using linked national health records, found symptomatic dengue associated with a significantly increased risk of stillbirth, persisting after adjustment for maternal age, parity and socioeconomic indicators [5]. A companion analysis from the same data linkage found symptomatic dengue independently associated with preterm birth among live births, strongest for infection occurring later in pregnancy [6,7]. Because these studies drew on routinely linked civil registration and notification data rather than a single hospital's cohort, they carry lower risk of referral-centre selection bias, strengthening the evidence for a genuine, if

moderate, increase in adverse maternal-fetal outcomes attributable to symptomatic dengue [4].

6. Fetal, neonatal outcomes and vertical transmission

Vertical transmission of dengue virus is well documented but depends strongly on the timing of maternal infection relative to delivery, with the highest risk when maternal viraemia is present at or immediately before birth. Tan and colleagues' Malaysian cohort found neonates born to mothers infected within days of delivery considerably more likely to show laboratory evidence of dengue than those born well after maternal recovery, establishing intrapartum timing as the dominant determinant of transmission risk [13]. Case reports continue to add detail: a recently published neonate developing fever and thrombocytopenia within four days of birth, born to a mother viraemic around the time of delivery, was interpreted as vertical transmission on the basis of concordant serotype and timing, reinforcing a low threshold for testing the neonate even after an uncomplicated delivery [17]. Basurko and colleagues' prospective matched cohort in French Guiana similarly found neonatal dengue concentrated among infants born to mothers symptomatic within the peripartum window, rather than distributed evenly across the trimester of maternal infection [18].

The pooled evidence on adverse fetal and neonatal outcomes, summarised in Table 2 alongside the Brazilian registry-linkage findings, is now substantial enough to have supported two systematic reviews and meta-analyses, both finding dengue infection during pregnancy associated with increased risks of preterm birth, low birth weight, and stillbirth relative to unexposed pregnancies, with effect sizes broadly consistent despite heterogeneity in the individual estimates [4,19]. Rathore and colleagues' more recent synthesis reached a similar conclusion while flagging that thrombocytopenia, postpartum haemorrhage and intensive-care admission

were consistently over-represented among dengue-affected pregnancies, mirroring the individual-cohort findings of Brar and Sutharsika above [20,14,15]. Naz and colleagues, reviewing vertical transmission specifically, emphasised that although confirmed neonatal dengue cases remain modest in number, the consistency of the intrapartum-timing pattern across geographically distinct case series and cohorts argues for a real, mechanistically plausible phenomenon rather than an artefact of ascertainment [21]. A cohort following pregnant women prospectively in a dengue-endemic region of Kenya, rather than relying on symptomatic presentation to trigger testing, likewise found dengue exposure during pregnancy, including infections that symptom-based surveillance would miss, associated with measurable effects on birth outcomes, suggesting hospital-based figures understate the true burden by missing pauci-symptomatic and asymptomatic maternal infections [22].

Table 2: Adverse fetal and neonatal outcomes reported across hospital-based cohorts and a Brazilian registry-linkage case-control study [14,15,5,6].		
Study	Design / n	Key fetal-neonatal finding
[14]	Prospective cohort, n=44	Preterm birth 37.5%, low birth weight 32.5%, stillbirth 10%
[15]	Retrospective cohort, n=52	Stillbirth 3.8%; no preterm births observed in this cohort
[5]	Matched case-control, linked national registry	Symptomatic dengue independently associated with increased stillbirth risk
[6]	Retrospective cohort, linked national registry	Symptomatic dengue independently associated with preterm birth

7. Discussion

Several conclusions are reasonably well corroborated across independent settings. First, prevalence among symptomatic pregnant women varies substantially, from roughly 14% to over 30% in hospital-based screening cohorts, driven primarily by transmission intensity and testing threshold rather than by biological differences between populations [8,9]. Second, secondary

heterologous infection remains the best-supported mechanistic risk factor for severe disease generally, but pregnancy-specific cohorts have more consistently identified bedside markers, a narrow febrile range, marked thrombocytopenia, and elevated transaminases, as practical predictors of the women most likely to die or suffer major morbidity, a pattern reproduced independently across Indian cohorts [14,15]. Third, registry-linkage studies from Brazil, less vulnerable to referral bias than single-centre series, corroborate the hospital-based finding that symptomatic dengue is associated with increased stillbirth and preterm birth risk [4,5,6,7]. Fourth, vertical transmission is real but strongly timing-dependent, concentrated among infections close to delivery, consistent enough across settings to be treated as an established clinical principle rather than an open question [13,18,17,21].

Important gaps remain. Few cohorts had paired serology distinguishing primary from secondary infection, so the strongest general risk factor for severe dengue could not be directly tested against outcomes in pregnant women, leaving open the possibility that the Indian bedside markers partly proxy for secondary-infection status [14,11]. Most prevalence data also come from symptomatic, hospital-presenting women, and the one prospective community-based cohort identified here suggests pauci-symptomatic infection contributes meaningfully to the true burden [22]. Co-circulation of chikungunya and Zika virus means studies relying on clinical case definitions alone risk misclassifying cases, a concern Gupta and colleagues addressed by testing for all three viruses and finding dengue disproportionately more morbid [9].

8. Conclusion

Dengue in pregnancy sits at an uncomfortable intersection of two clinical pictures never designed to be read together: a systemic viral illness whose warning signs depend on haematological and vascular parameters that

pregnancy itself perturbs, and an obstetric course whose normal complications can mask, or be mistaken for, those same signs. The evidence reviewed here indicates that prevalence among symptomatic pregnant women in endemic, hospital-presenting populations commonly falls between roughly 14% and 33%, that secondary infection and a recognisable cluster of bedside markers mark the women at highest risk of severe disease and death, and that both maternal mortality and adverse fetal outcomes, stillbirth, preterm birth and vertical transmission concentrated near delivery, are real and reproducible across geographically distinct cohorts rather than artefacts of any single study design [14,15,4,5,13]. Closing the remaining gaps around asymptomatic infection, paired serology for infection sequence, and dengue-specific obstetric management protocols will require the kind of linked, population-level surveillance that Brazil's registry studies have already shown is achievable, extended to the other high-burden regions where most dengue-affected pregnancies occur [7,16].

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11. Conflict of Interest

The authors declare no conflict of interest.

12. Author Contributions

Bruno L conceived the review and drafted the manuscript. Gallo F performed the literature search and data extraction. Romano C critically revised the manuscript for intellectual content. All authors approved the final version.

13. Ethics Statement

This is a narrative review of previously published, publicly available literature and did not require ethics committee approval or informed consent.

14. Data Availability

All data discussed are available in the cited published sources.

15. References

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