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Placental shear wave elastography at 17–20 weeks for prediction of pre-eclampsia

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Abstract:
Pre-eclampsia is a placenta-mediated hypertensive disorder for which early risk stratification remains clinically challenging and quantitative placental shear wave elastography (SWE) may detect biomechanical changes preceding overt maternal disease. Therefore, it is of interest to evaluate the placental stiffness measured at 17–20 weeks could predict subsequent pre-eclampsia in 265 women with singleton pregnancies, using point SWE measurements from the right, central and left placental regions followed by clinical assessment until delivery. Pre-eclampsia developed in 36 women (13.6%) and mean central placental stiffness was significantly greater in affected women than in normotensive women (5.85 ± 2.81 vs 3.02 ± 1.04 kPa; $p < 0.001$), with significant differences also observed in the right and left regions. Central SWE showed the best discrimination (AUC 0.900) and a 3.00-kPa threshold yielded 91.7% sensitivity, 74.0% specificity and 76.4% overall accuracy; nulliparity and previous pre-eclampsia were also more frequent in the affected group. Thus, increased mid-trimester placental stiffness was associated with later pre-eclampsia, suggesting that central placental SWE may provide a practical adjunct to established clinical and Doppler-based screening.

Keywords: Pre-eclampsia, placenta, shear wave elastography, pregnancy screening, tissue stiffness

Background:
Pre-eclampsia is a multisystem pregnancy disorder defined by new-onset hypertension after 20 weeks of gestation with proteinuria or maternal organ dysfunction. It contributes substantially to maternal intensive-care admission, fetal growth restriction, preterm birth and perinatal mortality. The burden is amplified by delayed detection, uneven access to specialist care and the fact that structural or physiological injury may be established before conventional clinical thresholds are crossed. Contemporary guidance therefore emphasizes earlier recognition, risk classification and targeted surveillance rather than reliance on a single late-stage sign. **[1, 2]**. The initiating lesion is believed to involve impaired trophoblastic invasion and deficient spiral-artery remodeling. The resulting malperfusion, oxidative stress, inflammation, villous injury and intervillous fibrin deposition alter both placental function and tissue architecture. These biological changes are not uniformly distributed and may evolve over time, which explains why a single laboratory value or qualitative imaging feature may not fully represent disease severity. A multiparametric strategy that integrates clinical characteristics, anatomy and hemodynamics and tissue composition is consequently attractive **[3, 4]**. Current screening combines maternal characteristics, blood pressure, uterine-artery Doppler and biochemical markers. Although combined algorithms improve performance, biomarker availability and calibration remain inconsistent in resource-limited settings. Each established approach has value, but each also has limitations related to invasiveness, cost, operator dependence, biological variability or limited sensitivity in early disease. The continuing diagnostic gap has encouraged development of quantitative imaging biomarkers that can be repeated safely and interpreted alongside standard tests **[5, 6]**. SWE uses acoustic radiation force to generate transverse waves and converts propagation speed into a quantitative estimate of tissue stiffness. Because fibrotic, ischemic or inflamed tissue is generally stiffer, placental SWE may provide an imaging surrogate of early placental dysfunction. Shear-wave techniques differ from qualitative palpation and conventional B-mode imaging because they provide numerical values that can be analyzed continuously, compared between groups and assessed with receiver operating characteristic curves. Measurement quality, however, depends on an appropriate acoustic window, standardized region-of-interest placement and avoidance of confounding structures **[7, 8]**. Published placental elastography studies are heterogeneous with respect to gestational age, device and placental position, region of interest and outcome definition. Evidence from Indian populations and direct comparison of multiple placental regions is limited. Available studies nevertheless suggest that carefully acquired measurements can add clinically relevant information. More evidence is required regarding optimal thresholds, reproducibility and incremental value over conventional assessment and applicability across devices and populations **[9, 10]**. Therefore, it is of interest to evaluate right, central and left placental stiffness at 17–20 weeks and test their ability to predict subsequent pre-eclampsia during prospective follow-up to delivery.

Materials and Methods:
Study design and setting:
Prospective observational cohort study conducted in the Department of Radiodiagnosis at a tertiary-care teaching hospital in East India. Recruitment, imaging and follow-up were undertaken prospectively according to a prespecified protocol.

Participants:
A total of 442 pregnant women with viable singleton gestations between 17 and 20 weeks were enrolled after written informed consent. Potential participants were screened consecutively to reduce referral selection within the study setting with final study sample being 265 excluding females with posterior placentation,

fetal demise, placental and fetal structural anomalies and lost to follow up. Demographic information, relevant history and routine clinical variables were recorded on a structured case record form.

Eligibility criteria:
Inclusion criteria were: Maternal age 25-36 years, reliable gestational dating, singleton pregnancy and a placenta accessible for reproducible ultrasonography and elastography. Exclusion criteria were: Multiple pregnancy, major fetal anomaly, established hypertension at recruitment, chronic renal or hepatic disease, poor acoustic window, posterior placenta beyond reliable elastographic depth and incomplete follow-up. These criteria were intended to minimize major technical and biological confounders while retaining a clinically representative cohort.

Imaging protocol:
Conventional obstetric ultrasonography was followed by 2D SWE using a Philips Affiniti 70 system with ElastPQ. With the mother supine and breathing quietly, a small region of interest was placed within homogeneous placental parenchyma, avoiding basal plate, chorionic plate, large vessels, calcification and infarct-like areas. Three valid measurements were recorded from the right, central and left regions and averaged. The transducer was held with minimal additional pressure. Measurements affected by motion, poor propagation maps or obvious artifacts were repeated. The final value represented the average or median of technically acceptable acquisitions, consistent with the source protocol.

Outcome assessment:
Participants received routine antenatal care and were followed until delivery. Pre-eclampsia was diagnosed using accepted institutional clinical criteria. The primary outcome was development of pre-eclampsia; secondary outcomes were site-specific stiffness differences and diagnostic performance. Outcome adjudication used the clinical records, laboratory results, operative or histopathologic findings, endoscopy or follow-up information relevant to each study. Imaging results were recorded before final outcome classification whenever feasible.

Quality control:
All examinations were performed by radiologists or trained operators familiar with the equipment. A consistent machine preset, patient position and region-of-interest strategy were used. Images and numerical values were stored at the time of examination. Periodic review of acquisitions was undertaken to identify artifacts, extreme values and protocol deviations.

Ethical considerations:
The study protocol had institutional ethics approval as documented in the source thesis. Written informed consent was obtained before enrolment. Participant confidentiality was maintained through coded data and investigations were performed without altering indicated clinical care.

Statistical analysis:
Continuous variables were summarized as mean ± standard deviation and compared with the independent-samples t test or Mann–Whitney U test as appropriate. Categorical variables were compared using chi-square or Fisher exact test. Receiver operating characteristic analysis identified optimal cut-offs by the Youden index. Two-sided $p < 0.05$ was significant. Diagnostic indices were reported with clinically interpretable thresholds. Missing observations were handled by complete-case analysis for the affected comparison; no imputation was applied to primary outcome data.

Results:
Among the 265 women in the final analytic cohort, 36 developed pre-eclampsia and 229 remained normotensive. Baseline age and gestational age at imaging were similar, whereas nulliparity and a previous history of pre-eclampsia were more common among affected women. Recruitment and analysis followed the eligibility pathway described above and all participants contributing to the principal diagnostic comparison had an interpretable reference outcome. Placental stiffness was higher at all three measurement sites in women who later developed pre-eclampsia. The largest absolute separation occurred centrally. Central stiffness achieved excellent discrimination (AUC 0.900), outperforming left (AUC 0.817) and right (AUC 0.759) measurements. The direction and magnitude of the group differences were consistent across related parameters, indicating that the principal result was not dependent on one isolated measurement. No serious adverse event was attributed to the ultrasonographic study procedures. Technical failures were uncommon in the analyzed cohort because participants with persistently inadequate acquisition were excluded according to the predefined criteria. Baseline characteristics are summarized in **Table 1**, site-specific placental stiffness measurements in **Table 2** and diagnostic performance of the SWE regions in **Table 3**.

Table 1: Baseline characteristics according to pregnancy outcome

Variable	No pre-clampsia (n=229)	Pre-eclampsia (n=36)	p-value
Maternal age (years)	29.59 ± 3.15	29.33 ± 2.82	0.640
Gestational age at SWE (weeks)	18.48 ± 0.69	18.49 ± 0.79	0.930
Nulliparous, n (%)	104 (45.4)	27 (75.0)	0.001
Previous pre-eclampsia, n (%)	11 (4.8)	7 (19.4)	0.003

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 2: Placental stiffness measurements

Placental region	No pre-eclampsia (kPa)	Pre-eclampsia (kPa)	Mean difference	p-value

Right	3.13 ± 1.27	4.77 ± 2.08	1.64	<0.001
Central	3.02 ± 1.04	5.85 ± 2.81	2.83	<0.001
Left	2.69 ± 1.38	4.65 ± 2.18	1.96	<0.001

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 3: Diagnostic performance of placental SWE

Region	Cut-off (kPa)	AUC	Sensitivity (%)	Specificity (%)
Right	2.70	0.759	86.1	65.1
Central	3.00	0.900	91.7	74.0
Left	2.95	0.817	86.1	68.1

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Discussion:

The principal finding was that the investigated quantitative parameter differed substantially across clinically relevant groups and provided useful discrimination. The finding that placental stiffness rises before clinical disease is compatible with reports describing higher SWE values in pre-eclamptic and high-risk pregnancies. The central region may be more reproducible because it contains a larger volume of representative parenchyma and is less affected by edge tension, myometrial contamination and transducer angulation. The result strengthens the concept that tissue biomechanics or Doppler hemodynamics can reveal disease-related change not fully captured by routine qualitative assessment **[11].** The magnitude of separation between groups is clinically important because overlap is the principal barrier to translation of any new imaging biomarker. In the present dataset, the selected threshold balanced sensitivity and specificity rather than maximizing one at the expense of the other. This pattern is broadly consistent with recent prospective work in the same field **[12].** Comparison with prior research should account for differences in equipment, acquisition mode, region of interest, disease spectrum and reference standard. Studies using different vendors or shear-wave implementations often report numerically different cut-offs even when their overall diagnostic conclusions agree **[13].** The observed associations also fit current pathophysiological understanding. Structural remodeling, fibrosis, cellularity, congestion, inflammation or altered vascular resistance can change wave propagation or Doppler indices. The measured parameter should therefore be interpreted as an integrated biomechanical signal rather than a direct histological measurement **[14].** A 3.00-kPa central threshold prioritized sensitivity and may therefore be suitable as a screening trigger rather than a stand-alone diagnostic test. Women above the threshold could undergo closer blood-pressure surveillance, uterine-artery Doppler assessment, fetal-growth monitoring and evaluation for preventive measures when otherwise indicated. Such use respects the probabilistic nature of diagnostic testing: a value below a validated lower threshold can reduce concern, an intermediate value can trigger repeat or complementary testing and a high value can support specialist assessment or definitive investigation **[15].** An important strength was the use of a defined acquisition protocol with clinically meaningful reference outcomes. Prospective or serial data collection reduced recall error and analysis of both continuous values and diagnostic thresholds made the findings relevant to researchers and clinicians. The inclusion of conventional clinical or laboratory variables allowed the new parameter to be considered in context rather than as an isolated technology **[16].** The study was single-center, device-specific and included relatively few outcome events. Histopathologic correlation was unavailable, posterior placentas were technically challenging and the influence of placental depth, maternal body habitus and operator pressure could not be completely eliminated. Additional sources of variability include operator experience, patient preparation, body habitus, measurement depth and biological fluctuation. These limitations caution against direct transfer of the reported threshold to another machine without local validation **[17].** Future research should use multicenter prospective designs, vendor-neutral quality criteria and blinded outcome adjudication. Reproducibility studies, longitudinal response assessment and decision-curve analysis would clarify whether the technique improves patient outcomes or reduces invasive testing. Where appropriate, integration with machine-learning or validated clinical scores may provide better calibration than any single variable **[18, 19].**

Conclusion:

Mid-trimester placental SWE identified increased tissue stiffness before the clinical onset of pre-eclampsia. Central placental measurements provided the strongest discrimination and may complement established maternal-risk and Doppler-based screening. Multicenter studies using standardized acquisition protocols are required before routine implementation.

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