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Stroke associated with pregnancy and the postpartum period: global incidence, risk factors, and preventive measures

Ictus asociado al embarazo y puerperio: incidencia global, factores de riesgo y prevención aplicable

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Pregnancy-related stroke (PRS) remains an infrequent but disproportionately devastating neurological event, situated at the intersection of obstetric physiology and vascular pathology [1-3]. Its clinical relevance has increased in parallel with shifts in maternal demographics and the growing prevalence of cardiometabolic risk factors among women of reproductive age [2,4]. Yet, despite this growing recognition, PRS continues to be approached as a fragmented entity (part obstetric complication, part neurological emergency) without a fully integrated conceptual framework.

Recent global evidence synthesizing more than 270 million pregnancies estimated a pooled incidence of approximately 25 cases per 100,000 pregnancies, confirming that PRS, although uncommon, is far from negligible [1]. In absolute terms, this corresponds to a low individual probability; however, the risk increases substantially in the presence of specific clinical conditions, particularly hypertensive disorders, where the relative risk may rise several-fold compared to normotensive pregnancies.

This epidemiological signal is neither homogeneous nor stable. Marked regional variability (ranging from lower estimates in several European settings to substantially higher burdens in parts of Asia and the Americas) suggests that PRS is shaped not only by biological susceptibility but also by healthcare structures [1,4]. Differences in access to prenatal care, early detection of hypertensive disorders, postpartum surveillance, and availability of neuroimaging likely influence both incidence and reporting. Accordingly, global estimates should be interpreted as robust summaries rather than universally transferable figures. This reinforces the need to interpret PRS not only as a biological complication, but as a condition embedded within healthcare systems and shaped by structural determinants of maternal health.

Beyond incidence, the clinical weight of PRS becomes more apparent when considering its disproportionate contribution to maternal morbidity and mortality. Although absolute risk remains low, the relative increase in risk associated with hypertensive disorders, coagulopathies, and cardiometabolic conditions is clinically meaningful, particularly in vulnerable populations. This underscores that PRS is not merely a rare complication, but a high-impact event concentrated within identifiable high-risk profiles, in which relatively small absolute probabilities translate into clinically meaningful risk when viewed in the context of maternal morbidity and mortality.

A critical, and often underrecognized, feature of PRS is its temporal distribution. The postpartum period consistently emerges as the phase of greatest vulnerability, extending beyond the conventional six-week window emphasized in obstetric care [1,5,6]. This extended risk horizon challenges traditional care models, which tend to prioritize delivery-centered outcomes while underestimating delayed cerebrovascular events. The implication is clinically consequential: postpartum care should evolve from a short-term recovery paradigm into a longitudinal surveillance strategy, particularly for women with vascular, hypertensive, or thrombotic risk profiles. This temporal reframing has direct implications for care delivery, suggesting that postpartum cerebrovascular risk should be approached as a prolonged and dynamic process rather than a time-limited obstetric concern.

From a pathophysiological perspective, PRS does not represent a mere extension of stroke mechanisms observed in the general population. Pregnancy induces a transient but profound vascular state characterized by hypercoagulability, endothelial activation, altered cerebral autoregulation, and significant hemodynamic stress [2,3,7]. These changes, while physiologically adaptive in minimizing hemorrhagic risk during childbirth, simultaneously increase susceptibility to both thrombotic and hemorrhagic events. Notably, hemorrhagic stroke appears to contribute proportionally more in pregnancy than in non-pregnant young adults, a pattern consistent with the central role of hypertensive disorders such as preeclampsia and eclampsia in destabilizing cerebral vasculature [1,2,7].

In this context, PRS should be understood not as an isolated neurological event, but as the clinical expression of systemic vascular dysregulation occurring within a uniquely vulnerable biological window. Framing these mechanisms within a unified model not only clarifies pathogenesis, but also provides a clinically relevant basis for risk stratification and anticipatory care.

Risk stratification further reinforces this integrative perspective. Hypertension (both chronic and pregnancy-related) remains the most consistent and clinically consequential determinant, but it rarely acts in isolation. Among these, hypertensive disorders stand out not only for their consistency but for their magnitude of effect, representing one of the strongest drivers of increased stroke risk during pregnancy and the postpartum period. Migraine, coagulopathies, cardiovascular disease, obesity, smoking, alcohol exposure, and broader cardiometabolic vulnerability collectively shape a multidimensional risk profile [1,2,4,7]. Importantly, several of these factors are identifiable before conception or early in gestation, suggesting that part of the burden of PRS reflects missed opportunities for anticipatory care.

From a clinical standpoint, this multidimensional risk architecture is best understood not as a collection of isolated variables, but as the convergence of interacting domains that together define maternal vulnerability. In practice, the identification of high-risk patients emerges from the coexistence of key elements, including hypertensive disorders, cardiometabolic dysfunction, and contextual factors such as limited access to structured prenatal and postpartum care. Rather than relying on single-factor thresholds, risk assessment should be approached as an integrative process in which the accumulation and interaction of these variables signal a higher probability of adverse cerebrovascular events. This perspective allows clinicians to move beyond checklist-based identification toward a more pragmatic and individualized recognition of risk across the continuum of care.

Preventive strategies should therefore operate across multiple levels, but not all interventions carry the same feasibility or impact across settings. At the individual level, early identification and aggressive management of hypertensive and metabolic disorders represent the most immediately actionable and high-yield strategies [3,7]. At the system level, priorities should focus on scalable interventions, including standardized screening pathways, improved access to prenatal care, and structured postpartum follow-up, particularly in resource-constrained settings where delayed diagnosis and limited surveillance disproportionately affect outcomes [1,4,5].

At the interface between neurology and obstetrics, heightened clinical vigilance for focal neurological deficits, atypical headache patterns, seizures, or altered mental status remains critical, as timely

recognition often determines prognosis more than the availability of advanced interventions. Framing prevention in terms of prioritization rather than mere comprehensiveness allows for more realistic implementation, especially in settings where resources are limited and the margin for effective intervention is narrow.

Despite these advances, the current evidence base reveals a set of unresolved and clinically relevant gaps that require more explicit definition. Key limitations include the absence of standardized diagnostic criteria for PRS, variability in the temporal definition of the postpartum period, and substantial heterogeneity in study design and case ascertainment across settings [1,4]. In addition, the persistent underrepresentation of low- and middle-income countries introduces a structural bias that limits the global applicability of existing estimates [1,4].

These limitations highlight clear priorities for future research: the development of standardized definitions, the establishment of prospective and globally representative cohorts, and the generation of data capable of supporting more consistent and generalizable risk estimation. Framing these challenges explicitly is essential to move the field from descriptive heterogeneity toward cumulative and comparable evidence.

Current evidence does not yet provide sufficiently granular, patient-level predictive tools to guide individualized decision-making in PRS. Most available data are derived from heterogeneous populations and non-standardized definitions, limiting their direct applicability to real-world clinical settings. Addressing this gap will require prospective, globally representative studies and the development of integrative models capable of capturing the complex interplay between biological, clinical, and systemic determinants.

Beyond epidemiology and prevention, PRS also poses a broader conceptual challenge: how to translate heterogeneous, population-level evidence into individualized clinical decisions. Unlike conditions governed by stable algorithms, PRS requires interpretation under uncertainty, where timing, mechanism, maternal comorbidity, obstetric context, and healthcare resources must be considered simultaneously [2,3]. In this sense, evidence application in PRS is inherently interpretative rather than purely procedural.

Ultimately, stroke associated with pregnancy and the postpartum period should be understood as a dynamic and context-dependent clinical condition, in which biological vulnerability, healthcare structures, and temporal evolution of risk converge. This perspective challenges reductionist interpretations and underscores the need for deeper integration across neurology, obstetrics, vascular medicine, and public health [3,5].

These considerations also carry important implications for medical education. The complexity of PRS (characterized by heterogeneous evidence, context-dependent risk, and time-sensitive decision-making) demands analytical competence that extends beyond traditional training. From undergraduate medical students to general practitioners, residents, neurologists, obstetricians, and other neuroscience-related specialists, there is an urgent need to strengthen education in research methodology, critical appraisal, and clinically oriented evidence-based reasoning [8,9].

Without this foundation, even high-quality evidence may be misinterpreted, overgeneralized, or applied without sufficient contextual judgment. In a condition where diagnostic and therapeutic interpretation can directly influence maternal outcomes, cultivating a scientifically literate and critically engaged workforce is not optional, it is a clinical necessity.

Conflicts of interest

The authors declare no conflict of interest.

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Data, Materials, and Code Availability

Not applicable.

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