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Subarachnoid Haemorrhage Following Caesarean Delivery in a Woman with Multiple Sclerosis: Case Report and Discussion

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Abstract

Background: Seizures and cerebral bleeding during pregnancy are uncommon but carry significant maternal and neonatal risks. When they occur postpartum, particularly in women with pre-existing neurological conditions such as multiple sclerosis (MS), diagnostic complexity increases.

Case presentation: We report a case of a 25-year-old primigravida with a longstanding diagnosis of MS, who underwent elective lower uterine segment caesarean section (LUSCS) at term. Shortly after delivery, she experienced a sudden severe headache, acute hypertension, and a generalised tonic-clonic seizure. Imaging revealed bilateral frontal SAH with no aneurysm or thrombosis. MRI also demonstrated a new pontine demyelinating lesion. She was managed conservatively with ICU support and early postpartum immunotherapy. Follow-up showed full clinical recovery.

Discussion: This case highlights the intersecting vulnerabilities of postpartum neurophysiology and chronic demyelinating disease. The differential diagnosis encompassed eclampsia, PRES, and seizure-induced SAH. The presence of MS may have predisposed to neurovascular fragility and cortical irritability in the postpartum setting.

Conclusion: Non-aneurysmal SAH in the early postpartum period remains a diagnostic and therapeutic challenge, particularly in women with chronic neurological disease. Multidisciplinary care and anticipatory planning are essential to optimise maternal outcomes in future pregnancies.

Keywords: Postpartum seizure; Subarachnoid haemorrhage; Multiple sclerosis; Pregnancy complications; Eclampsia; Demyelinating disease; PRES; Maternal stroke

Abbreviations: LUSCS: Lower Uterine Segment Caesarean Section; MS: Multiple Sclerosis; SAH: Subarachnoid Haemorrhage; PRES: Posterior Reversible Encephalopathy Syndrome; RCVS: Reversible Cerebral Vasoconstriction Syndrome; AVMs: Arteriovenous Malformations; CNS: Central Nervous System; RCVS: Reversible Cerebral Vasoconstriction Syndrome; CVST: Cerebral Venous Sinus Thrombosis; ICH: Intracerebral Hemorrhage; SMASH-U Structural vascular lesion, Medication, Amyloid angiopathy, Systemic disease, Hypertension, Undetermined; NCC: Neurocysticercosis

Introduction

In 1833, English poet and physician Erasmus Darwin noted the "disturbance of the mind and convulsions of the body" in a woman who seized after delivery—long before the formal identification of eclampsia [1]. Today, while seizures in pregnancy remain rare, they carry a disproportionately high risk for both mother and child [2]. When occurring in the immediate postpartum period, the diagnostic field widens to include both obstetric and non-obstetric aetiologies. Among these, subarachnoid haemorrhage (SAH) is a rare but serious event, responsible for a significant proportion of maternal morbidity and mortality worldwide [3,4].

Multiple sclerosis (MS) is an autoimmune demyelinating disorder predominantly affecting women of reproductive age. Historical records suggest that the earliest documentation of MS may date back to the 15th century in Holland, as recorded in the account of Saint Lidwina of Schiedam [5]. After breaking a rib while skating at age 16, she developed progressive neurological symptoms including paralysis and sensory disturbances—

considered retrospectively to be the earliest recorded account of MS. More recently, Betty Cuthbert (1938-2017), the renowned Australian sprinter and Olympic gold medallist became a public advocate for MS awareness, when she reflected on her experience of neurological decline and how it shaped her life after athletics, including its psychological toll and implications for independence and identity, emphasising the interplay between MS, women's health, and reproductive life stages [6]. Today, we recognize that pregnancy often confers a temporary immunological reprieve, with reduced relapse rates, particularly in the third trimester. However, the postpartum period is recognised as a time of increased relapse risk, coinciding with rapid hormonal and immunological shifts [7,8].

In this context, we present a unique case of SAH immediately following elective caesarean delivery in a woman with stable MS, managed conservatively with full recovery. The case raises important considerations around the vulnerability of the postpartum brain, the influence of chronic neurological disease on seizure thresholds, and the importance of interdisciplinary care planning in pregnancy.

Case Presentation

"I remember a light splitting behind my eyes. Then there was pain and panic, and then the tremors began. After that, I just remember waking feeling so very tired."

A 25-year-old primigravida presented for elective caesarean delivery following an otherwise uncomplicated pregnancy. She carried a longstanding diagnosis of multiple sclerosis (MS), managed by a private neurologist in South Australia. Her MS, though generally stable, had necessitated intermittent corticosteroid therapy for antenatal flare-ups at 9- and 18-weeks gestation. Prior to conception, she had completed a course of Cladribine (Mavenclad), with plans to commence Natalizumab postpartum as prophylaxis against flare recurrence.

Throughout pregnancy, she remained under close neurological review, with intermittent symptoms—dizziness, visual disturbances, and non-focal headaches—attributed to vestibular migraines. Cardiac evaluation for syncope episodes demonstrated benign findings, including rare ventricular and supraventricular ectopic beats. An antenatal MRI showed stable lesions with no acute concerns. Her mental health was concurrently supported with low-dose sertraline.

In line with advice from her neurologist, an elective lower uterine segment caesarean section (LUSCS) was scheduled to mitigate peripartum stress and fatigue. The procedure proceeded uneventfully under spinal anaesthesia. A healthy male infant was delivered, and intraoperative blood loss was modest (350mL).

However, during postoperative transfer—just minutes after birth—she complained of a sudden, severe frontal headache and neck pain, accompanied by nausea. Her blood pressure, which had previously been normal, spiked to 180/107mmHg. Within moments, she developed focal left-hand twitching, which rapidly evolved into a generalized tonic-clonic seizure.

The anaesthetic team initiated emergency management. Midazolam was administered; her airway was protected. Recovery of consciousness occurred within fifteen minutes, accompanied by postictal fatigue, but with full orientation and no residual deficits. Magnesium sulphate was commenced empirically for suspected eclampsia, despite no prior hypertensive or proteinuric history. Laboratory work-up revealed a mildly elevated protein/creatinine ratio and transaminases but otherwise unremarkable parameters.

Urgent non-contrast CT brain imaging revealed small-volume subarachnoid haemorrhage (SAH) in the superior right frontal and high left frontal lobes, with no signs of mass effect or hydrocephalus. A neurology consultation suggested a non-aneurysmal etiology, with differential considerations including eclampsia, posterior reversible encephalopathy syndrome (PRES), cerebral venous sinus thrombosis, and reversible cerebral vasoconstriction syndrome.

Subsequent MRI confirmed bilateral SAH and demonstrated minor progression of MS lesions in the temporal lobes with a new pontine focus suggestive of demyelination. CT angiography and venography excluded aneurysm and sinus thrombosis. No surgical intervention was warranted.

Her management included ICU monitoring, supportive care, and continuation of her planned postpartum Natalizumab therapy. Antiepileptic prophylaxis was initially withheld but later initiated with carbamazepine due to seizure risk.

Echocardiography showed normal cardiac structure and function. She was transferred to the maternity ward on postoperative day two and discharged home on day four in stable condition.

Follow-up imaging over the ensuing six weeks demonstrated resolution of haemorrhagic changes with residual haemosiderin staining, and no further lesions. Clinically, she remained well, with no recurrent seizures and stable MS.

Discussion

Seizures complicate fewer than 1% of pregnancies, yet they are associated with significantly increased maternal and perinatal morbidity and mortality, both acute and long-term [1]. Among these, cerebrovascular events—particularly intracerebral haemorrhage and subarachnoid haemorrhage (SAH)—remain a rare but serious threat [2]. In Australia, maternal stroke occurs in approximately 34 per 100,000 deliveries and accounts for an estimated 5–12% of maternal deaths [3]. These events are most often haemorrhagic and most likely to occur during the early

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postpartum period, typically within the first six weeks following delivery. The third trimester and postpartum period carry the highest risk, with underlying causes frequently involving one or more of the following: preeclampsia/eclampsia, cerebral venous sinus thrombosis, or reversible cerebral vasoconstriction syndrome (RCVS) [4,5].

In pregnancy, early signs of neurological compromise are often attributed to obstetric causes—headache to dehydration, seizure to eclampsia—without exploration of co-existing or alternative diagnoses. Similarly, postpartum seizures are often assumed to be eclamptic, but the presence of chronic neurological disease or other aetiologies demands a readiness to question presumptions [6].

Historically, postpartum seizures were regarded with equal parts wonder and fear. In classical antiquity, Hippocratic texts attributed convulsions to imbalances of bodily humours or the accumulation of toxins—especially in the context of retained lochia or disrupted menstruation—implying a form of systemic "poisoning" that affected the brain. Such ideas persisted well into the early modern period. In the 19th century, many such episodes were labelled "hysteria," with limited differentiation from epilepsy. It was not until the 20th century that distinctions between eclampsia, stroke, and seizure disorders in women became more rigorously understood [7]. Contemporary medicine now recognises the pathophysiology underlying postpartum seizures—ranging from eclampsia and posterior reversible encephalopathy syndrome (PRES) to rarer structural or infectious causes [8].

The differential diagnosis of seizure and intracranial haemorrhage in the postpartum period includes arteriovenous malformations (AVMs) and ruptured aneurysms as the most common culprits in cases of spontaneous subarachnoid haemorrhage (SAH) [9]. However, a comprehensive evaluation must also include metabolic disturbances—such as hypoglycaemia or hyponatraemia—as well as central nervous system (CNS) infections, which may present acutely with seizure or altered mental status in pregnancy [10].

Structural brain lesions, including gliomas or other space-occupying processes, represent rare but critical considerations, particularly when focal neurological deficits or persistent postictal confusion are present. Imaging in such cases may reveal mass effect or edema, guiding urgent neurosurgical referral. Ischaemic and haemorrhagic stroke causes in pregnancy and postpartum are often categorised into three key groups: reversible cerebral vasoconstriction syndrome (RCVS), preeclampsia/eclampsia, and cerebral venous sinus thrombosis (CVST) [10]. Notably, RCVS and eclampsia are increasingly recognised as overlapping syndromes, both marked by vasoconstriction, endothelial dysfunction, and hypertensive surges [11]. While they are typically treated as distinct entities, it may be more clinically meaningful to view them along a shared neurovascular spectrum—one that may be further destabilised in women with underlying neurological conditions such as multiple sclerosis [12].

It is also important to recognise that pre-existing epilepsy may at times be misinterpreted as new-onset seizure activity—or vice versa—particularly in the absence of a clear history, recent imaging, or continuity of neurological care [13]. Although the patient in this case had no prior history of epilepsy, the broader context of maternal neurology calls for careful preconception and antenatal planning in women with known seizure disorders, ensuring that emergent presentations are not misattributed, overlooked, or mistreated during the complex physiological transitions of pregnancy and the postpartum period [14].

To aid diagnostic clarity in cases of intracerebral hemorrhage (ICH), the SMASH-U framework (Structural vascular lesion, Medication, Amyloid angiopathy, Systemic disease, Hypertension, Undetermined) offers a structured heuristic that may be adapted to the peripartum context to include obstetric-specific triggers [15]. In particular, medication-induced vascular instability should be noted; agents such as vasoconstrictive pharmacotherapy and methylergometrine (methergine), a uterotonic occasionally used during the third stage of labour, have been implicated in triggering RCVS and subsequent seizure [16].

In multicultural settings such as Australia, clinicians must also consider globally prevalent conditions such as Neurocysticercosis (NCC), caused by the larval form of *Taenia solium*, a common parasitic infection of the central nervous system and a leading global cause of acquired epilepsy [17]. While not endemic in Australia, it is increasingly relevant due to migration from endemic areas including Latin America, South and Southeast Asia, sub-Saharan Africa, and parts of China. In peripartum women from these regions, NCC may be misattributed to eclampsia or postpartum exacerbations of MS, particularly when hypertension and proteinuria are absent. Neuroimaging often reveals parenchymal cysts or calcified lesions, and diagnosis depends on a high index of suspicion, detailed travel and dietary history, and appropriate serological testing [17,18].

In this background, our case underscores the intricate interplay between chronic neurological illness, the adaptive physiology of pregnancy, and the dynamic vulnerabilities of the postpartum state. It demonstrates the convergence of seizure, acute hypertension, and non-aneurysmal SAH, prompting a broad differential diagnosis that spans obstetric, neurological, and vascular domains. The precise sequence of causality—whether seizure preceded haemorrhage or vice versa—is difficult to disentangle. The postpartum brain may be conceptualised as a threshold organ—having just traversed the hypercoagulable, hypertensive, and hormonally dynamic terrain of late pregnancy. Each of these forces, individually and in concert, may render the neurovascular interface more susceptible to injury [19].

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The hypercoagulable state of pregnancy persists into the puerperium, an evolutionary adaptation to minimise postpartum bleeding, but one that increases the risk of thrombotic and endothelial complications, including cerebral venous sinus thrombosis and PRES [20]. The hypertensive lability of the peripartum period also warrants attention as women may experience abrupt haemodynamic change and fluid shifts following placental separation, which can provoke transient but significant surges in blood pressure [21]. These vascular perturbations form a milieu in which eclampsia and related cerebrovascular events can erupt unexpectedly [22].

Furthermore, the hormonal recalibration of the postpartum state—marked by rapid declines in estrogen, progesterone, and cortisol—has profound neurological implications. These changes can influence seizure thresholds, emotional regulation, and immune responsiveness [23]. In the context of demyelinating disease such as multiple sclerosis (MS), the postpartum period is a well-established window of heightened vulnerability, as the relative immune quiescence of pregnancy gives way to reactivation and disease rebound [24].

In this patient, these combined forces—coagulopathic, vascular, and neuroendocrine—appear to have breached a fragile neurological threshold. The presence of chronic neurological disease, particularly one characterised by disrupted white matter integrity, likely lowered her seizure threshold and heightened susceptibility to neurovascular compromise [25]. Emerging literature suggests that the blood–brain barrier in MS patients may respond atypically to peripartum shifts, further predisposing to PRES-like events and potentially contributing to SAH [26].

Multiple Sclerosis and Pregnancy

MS is not, in itself, a contraindication to vaginal birth [27]. Relapse rates tend to diminish in the third trimester, secondary to immunomodulatory adaptation, which means that exacerbation of symptoms provoked by the physical demands of labour may not be a fait accompli [28]. In this case, antenatal care reflected best-practice interdisciplinary collaboration, including the cessation of Cladribine prior to conception and planned recommencement of Natalizumab postpartum [29]. Neurological symptoms such as dizziness and headaches were evaluated thoroughly and attributed to vestibular migraine, with no focal signs to suggest active disease.

Nonetheless, the postpartum period remains a known inflection point for disease recurrence [30]. In this context, the decision to proceed with elective lower uterine segment caesarean section (LUSCS) was based on neurologist advice to minimise maternal fatigue and labour stress—both of which are potential triggers for MS flare. In retrospect, this setting proved fortuitous: the sudden onset of severe frontal headache, followed by focal seizure and generalised tonic-clonic activity, occurred in a highly monitored environment, enabling immediate intervention. This contrasts with other reported cases in which neurological complications arose during or following spontaneous labour [31]. It reinforces that the mode of delivery in women with known or suspected intracranial pathology should be determined on a case-by-case basis, rather than applied reflexively [32].

Non-Aneurysmal Subarachnoid Haemorrhage and Diagnostic Imaging

The immune reconstitution or rebound phase after pregnancy is a turbulent window in MS and may also have a contributory role in the pathogenesis of other neurovascular syndromes, including PRES and non-aneurysmal SAH [33]. Non-aneurysmal SAH in the peripartum setting may arise from several mechanisms. One hypothesis is that seizure-induced rupture of delicate subpial vessels occurs, particularly in a brain with demyelination [34]. Another is the vasogenic endothelial dysfunction seen in PRES, wherein fluctuating blood pressure and impaired autoregulation provoke microvascular injury [35].

In this patient, the modest volume of SAH, absence of aneurysm on CTA, and lack of mass effect or midline shift suggest a self-limited vascular event. MRI did, however, reveal a new pontine lesion suggestive of active demyelination. Whether this lesion was present prior to the seizure or whether it contributed to cortical irritability and lowered the seizure threshold remains speculative. It is plausible that the seizure and the lesion represent parallel manifestations of postpartum immune reactivation [36].

The case reinforces the importance of timely neuroimaging. Although the patient was postpartum, clinical hesitation toward neuroimaging persists in many peripartum contexts. This reflects a residual culture of caution—particularly during breastfeeding or immediately post-delivery. This caution must now be weighed against the increasing complexity of maternal neurology [37]. Brain CT without contrast remains the first-line investigation for suspected SAH, especially when speed and availability are essential [38]. While MRI is superior in suspected demyelinating lesions and posterior circulation pathology—as was ultimately critical in this case—it may not always be accessible. In resource-variable settings, CT combined with vascular imaging remains a defensible first step, particularly when clinical suspicion is high [39].

It is also worth noting that iodine-based CT contrast agents, while safe in most postpartum contexts, carry some theoretical risk in pregnancy due to potential neonatal thyroid suppression [40]. Nonetheless, when vascular pathology is suspected, contrast-enhanced CT or CTA should not be withheld when clinically indicated. Transcranial Doppler offers a non-invasive modality for monitoring cerebral vasospasm and could be considered in ongoing surveillance, particularly where vasoconstrictive syndromes like RCVS are suspected [41]. Finally, if CT is inconclusive yet clinical suspicion persists, lumbar puncture remains essential for detecting xanthochromia or residual blood. In rare cases, tissue biopsy or neurosurgical consultation may be required to resolve

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persistent diagnostic uncertainty [42].

Other Considerations in Differential Diagnosis

The most common causes of haemorrhagic stroke in pregnancy include arteriovenous malformations, cerebral aneurysms, and hypertensive disorders such as eclampsia [43]. Despite this patient's previously normal blood pressures, the sudden hypertensive episode and seizure immediately postpartum raise the possibility of fulminant preeclampsia. Magnesium sulphate was appropriately commenced. However, subsequent blood and urine analyses (including protein/creatinine ratio and liver function) were not consistent with classical preeclamptic patterns.

It must be acknowledged that eclampsia can present atypically, with minimal or absent laboratory hallmarks [44]. Clinical vigilance must remain high even in women without prior hypertension or classical biochemical features. If, however, seizures persist after magnesium therapy, suspicions of other non-eclamptic causes should escalate [45].

While SAH can occur as a complication of eclampsia, the clinical and radiographic features in this case—modest volume of blood, absence of vasogenic oedema, and lack of systemic features—point toward a separate, non-hypertensive vascular event. It is plausible that this was triggered by postpartum stress or immune-mediated shifts affecting vulnerable cerebral vasculature, particularly in the context of underlying demyelinating disease [46].

Antiepileptic Therapy and Postpartum MS Care

Initial withholding of antiepileptic prophylaxis was reasonable, given the single seizure episode in a provoked context [47]. However, the subsequent initiation of carbamazepine was justified in light of cortical blood irritation and the evolving neuroimaging findings. Natalizumab was recommenced early, in line with consensus guidelines for postpartum relapse prevention, offering an important layer of protection during this immunologically dynamic time [48].

Speculative Aetiology

The precise aetiology of the subarachnoid haemorrhage (SAH) in this patient remains elusive; yet, several physiologically plausible mechanisms warrant consideration. It is conceivable that a transient episode of postpartum hypertension—whether related to incipient preeclampsia or nonspecific vascular lability—led to endothelial injury and vessel rupture [49]. In this hypothesis, the acute rise in blood pressure would represent a prodromal vascular insult.

Alternatively, the haemodynamic surge may have been reactive—a physiological response to sudden neurological distress, pain, or seizure. In this reading, the headache was a sentinel symptom of haemorrhage, with hypertension and seizure following in rapid sequence [50].

Another plausible mechanism considers the role of underlying multiple sclerosis, and specifically, the neurovascular fragility associated with postpartum immune reconstitution. Inflammatory remodelling and altered blood–brain barrier permeability may have lowered the seizure threshold or predisposed the vessel to rupture, especially in the context of a demyelinating lesion in the brainstem [51]. Seizure activity itself, particularly when focal and intense, may generate sufficient cortical pressure to induce rupture of fragile subpial vessels [52].

These hypotheses do not need to be mutually exclusive. More likely, they converged: a "perfect storm" of postpartum vascular reactivity, neurological vulnerability, and immune rebound. Together, they rendered the neurovascular interface fragile enough that an otherwise minor instability—be it haemodynamic or electrical—tipped the balance toward haemorrhage.

Ultimately, this case serves as a reminder that postpartum neurological events do not always conform to linear causality. Rather, they emerge from threshold states—where vascular, immune, and neural forces are delicately poised—and where the clinician must remain attuned not only to what is seen, but to what may be silently unfolding beneath [53].

Future Pregnancies

While the outcome in this case was ultimately favourable, it is important to acknowledge that similar presentations can lead to profound morbidity or even mortality. Recent data from Finland—a high-resource setting with robust maternal healthcare—revealed a maternal mortality rate of 12.5% in comparable cases, with over 20% of survivors sustaining significant long-term neurological disability [54]. In this light, the patient's full neurological recovery is not only fortunate, but clinically remarkable**.** Other reported cases have involved progression to palliative care, underscoring the emotional and ethical weight carried by these presentations [55].

Although the tone of this report is intentionally hopeful, a balanced discussion must also reflect the devastating outcomes that can follow perinatal seizures—particularly when delays in diagnosis or limited access to neurocritical care exist. This contrast highlights the importance of timely imaging, multidisciplinary collaboration, and anticipatory planning in maternal neurology [56].

Future care would prioritise optimal MS management, with particular attention to blood pressure monitoring and the preservation of vascular integrity. While the decision for elective caesarean delivery ensured close clinical oversight in this instance, it should not be viewed as universally applicable to all women with MS. For this patient, however, a repeat caesarean section would be a prudent choice in future pregnancies, offering predictability, continuity, and the reassurance of multidisciplinary preparedness [57].

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Conclusion

This case demonstrates the profound complexity of neurological events in the postpartum setting, particularly in women with pre-existing demyelinating disease. While seizures and hypertension immediately following delivery raise the spectre of eclampsia, alternative vascular and neurological causes must be urgently explored. In this patient, non-aneurysmal subarachnoid haemorrhage likely reflected a convergence of postpartum vascular reactivity, seizure vulnerability, and underlying MS pathology.

Importantly, her outcome was favourable due to close multidisciplinary collaboration, early recognition, and proactive postpartum management. For future pregnancies, the emphasis must be placed on individualised planning, maternal neurological stability, and delivery in a setting equipped for acute neuro-obstetric care.

Lessons to be Learnt

This case offers several key insights for clinicians caring for women with chronic neurological disease during the peripartum period.

a) **Do not assume seizure = eclampsia** – Even in the presence of hypertension, alternative diagnoses such as subarachnoid haemorrhage, PRES, or MS flare must remain in view. This reinforces the importance of considering a broad range of diagnostic options in postpartum neurological events. While seizures and hypertension following delivery may initially suggest eclampsia, alternative or concurrent diagnoses—such as subarachnoid haemorrhage—must remain on the radar, particularly when clinical features are atypical or do not respond as expected.

b) **MS increases postpartum neurovascular fragility** – Immune rebound is not just relapse risk; it represents a lowering of seizure threshold and structural vulnerability within the central nervous system. The case highlights the unique neurological susceptibilities of women with multiple sclerosis in the early postpartum phase. The abrupt withdrawal of pregnancy hormones, resurgence of immune activity, and vascular lability together create a threshold state where demyelination, seizure activity, and vascular injury may coalesce.

c) **MRI is safe postpartum and often essential**. Timely imaging can be lifesaving. Clinicians must move beyond hesitancy, particularly when neurological symptoms are atypical or progressive.

d) **Multidisciplinary coordination is critical**. This case highlights the importance of rapid collaboration between obstetrics, neurology, ICU, radiology, and anaesthetic specialties. Optimal management of subarachnoid haemorrhage, even when non-aneurysmal or self-limiting, requires a high level of clinical vigilance and infrastructure. Best-practice guidelines recommend that nimodipine is the agent of choice for preventing delayed cerebral ischaemic in subarachnoid haemorrhage. This was not considered in our case, which may reflect the rapid resolution of symptoms but could represent a point for future protocol alignment**,** especially in cases with a greater haemorrhagic volume or vasospasm risk. Current recommendations also advocate that SAH should be managed in tertiary neurovascular centres, with admission to a dedicated stroke unit for neurologically stable patients. While this patient benefited from coordinated specialist input, escalation to include stroke unit involvement underscores the standard of care such presentations warrant.

e) **Postpartum care must be anticipatory, not reactive**. Clear escalation plans, early reintroduction of disease-modifying therapy, and follow-up imaging are crucial in women with MS or similar conditions to optimise outcomes and reduce the risk of relapse. This case also invites further exploration into the postpartum neurovascular threshold—and how clinicians may better safeguard women who live near its edge.

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