Cerebrovascular emergencies in pregnancy

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Caring for pregnant and postpartum patients with neurological disease carries specific challenges. In performing a diagnosis, it is often difficult to differentiate between true pathology and neurological symptoms resulting from normal pregnancy physiology. Treating the pregnant patient can be problematic as well. Providers need to be aware of the possible untoward effects of maternal treatments on the developing fetus, but not withhold therapies that reduce disease-related morbidity and mortality. Given the complexities of conducting trials during pregnancy, few treatments are based on high-quality data; observational data and clinical expert opinion often guide treatments. With the exception of preeclampsia/eclampsia, neurological diseases typically do not warrant early delivery in the absence of fetal distress. Multidisciplinary care, utilizing the expertise of anesthesiology, critical care medicine, emergency medicine, maternal–fetal medicine, neurology, and radiology, is essential in ensuring prompt diagnosis and treatment.

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Introduction

Neurological symptoms in pregnant women are common. Practitioners must differentiate pathological etiologies from preexisting or pregnancy-specific neurological conditions. Unique pathophysiological changes of pregnancy can increase the risk for neurological complications. For example, estrogen-stimulated production of clotting factors increases the risk of thrombosis, especially postpartum [1]. Furthermore, venous compliance, capillary leakage, and vasogenic edema rise along with progesterone levels [2].

Patients presenting with acute neurological complaints warrant immediate attention, as timely intervention can be lifesaving for both mother and fetus. Eclampsia, for instance, carries maternal mortality rates of 0.5–14%, with the lowest rates in patients who receive regular prenatal care [3,4]. Physicians caring for gravid patients must understand how treatment differs from the treatment of non-gravid patients. Additionally, practitioners must be mindful when selecting medications and interventions that the fetus is exposed to minimal risk, while not compromising on maternal treatment. Applying expertise from multiple disciplines — anesthesiology, critical care medicine, emergency medicine, maternal—fetal medicine, neurology, and radiology — is key in achieving prompt and accurate diagnosis, and in initiating treatment.

When coordinating care, gestational age, fetal viability, and mode of delivery need to be considered. Planning for a safe delivery often adds another layer of clinical complexity, as providers consider the mode of delivery (cesarean section, unassisted vaginal delivery, or vaginal delivery assisted with a vacuum or forceps), anesthetic options, hemodynamic changes specific to the trimesters of pregnancy and parturition, and hemostasis.

In this review, we focus our attention on neurological complications during and just after pregnancy, including preeclampsia/eclampsia, reversible cerebral vasoconstriction syndrome (RCVS), posterior reversible encephalopathy syndrome (PRES), stroke, intracranial hemorrhage (ICH), and cerebral venous sinus thrombosis (CVT). The authors recognize that there are many neurological diseases that can be encountered in the gravid population; however, this section focuses on the neurological emergencies that carry the most significant morbidity and mortality.

Preeclampsia/eclampsia

Preeclampsia is unique to pregnancy and the puerperium. It is characterized by new-onset hypertension (>140/90 mm Hg on two occasions at least 4 h apart) and proteinuria exceeding 300 mg in 24 h, developing after 20 weeks of gestation. Preeclampsia is often heralded by headache, peripheral edema, restlessness, and hyper-reflexia. Preeclampsia progresses to eclampsia in approximately 0.5% of pregnancies. In eclampsia, patients have the same findings as in preeclampsia, with the addition of generalized tonic–clonic seizures. Simultaneous goals of treatment include blood pressure control, prevention of cerebral edema, hemorrhage, infarction, and edema and seizure prevention. Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome and PRES are additional complications often seen with preeclampsia/eclampsia, and both necessitate delivery [3–8].

Magnesium sulfate infusion, given to preeclamptic patients with severe features, reduces seizure risk by >50% [3,9–13]. Refer to the “Hypertensive Disorders in Pregnancy” chapter for details on the management of preeclampsia, dosage of magnesium sulfate, and treatment of hypertension. Despite magnesium treatment, seizures may persist in 10% of patients [3].

Although the use of magnesium sulfate in preeclamptic patients with severe features is well established, its use in cases of mild preeclampsia is less compelling. Severe features of preeclampsia include systolic blood pressures of ≥160 mm Hg, diastolic blood pressures of ≥110 mm Hg, thrombocytopenia, impaired liver function, renal insufficiency, pulmonary edema, and new-onset cerebral or visual disturbances. Two double-blinded, randomized controlled trials evaluated magnesium treatment in patients with mild preeclampsia. Neither showed any significant reduction in eclampsia or progression to severe disease [11,14]. In a trial that randomized >10,000 preeclamptic women to receive magnesium sulfate or placebo, the number needed to treat to prevent one seizure in patients with mild disease was 109, compared to 63 patients with severe features [9]. Currently, there is insufficient data to recommend magnesium sulfate use in patients without severe features of...
Preeclampsia [8]. Although the use of magnesium sulfate in antepartum and immediately postpartum preeclamptic patients with severe features is well established, the use of magnesium is not as well established for patients presenting after the immediate postpartum period with features consistent with preeclampsia. Nevertheless, many experts recommend magnesium for seizure prophylaxis for postpartum patients presenting with elevated blood pressure and neurological symptoms such as headache or visual disturbances [15].

Eclampsia is one of the few true obstetrical emergencies. A patient who presents with convulsive seizures near term should be considered eclamptic until proven otherwise. Up to 16% of patients with eclampsia are normotensive, but this subset of patients should be managed in the same fashion as their hypertensive counterparts [3,18]. Brain imaging via noncontract computed tomography (CT) or magnetic resonance imaging (MRI) is recommended to rule out intracranial pathology for patients with atypical presentations, for instance, gravid patients with seizures and no clear signs of preeclampsia, persistent focal neurological deficits, and no history of epilepsy [17]. Once seizures and hypertension have been stabilized, delivery should be expedited, regardless of gestational age. After all, the definitive treatment for eclampsia is delivery, although not necessarily by cesarean section. The fetal heart rate tracing is often non-reassuring during the seizure and immediately after during the recovery phase. However, the fetus status typically recovers in parallel with maternal stabilization. Most physicians delay delivery until the patient is oriented, seizure activity has ceased, and the fetal heart rate has become reassuring. Ultimately, providers need to take into account gestational age, cervical status, maternal status, fetal presentation, and pregnancy history when making recommendations regarding mode of delivery [3,8,16].

Reversible cerebral vasoconstriction syndrome

RCVS, sometimes referred to as postpartum angiopathy when presenting postpartum, carries significant morbidity and mortality [17–21]. Given its rarity, the exact incidence is unknown; however, the syndrome is more commonly seen in patients with preeclampsia and autoimmune disorders [20]. Hallmarks of this syndrome are sudden onset of a severe thunderclap headache (often multiple thunderclap headaches) and segmental vasoconstriction of cerebral arteries documented on brain imaging. Approximately two-thirds of peripartum cases occur post-delivery [20,22,23]. RCVS can mimic eclampsia, as seizures are found in up to 28% of patients. RCVS has also been reported in patients with preexisting preeclampsia [20,24]. Although the exact pathophysiological process resulting in RCVS is unknown, vasoactive agents, postpartum state, and physical and sexual activity have been implicated as inciting factors [22,25]. The mainstay in diagnosis is MRI or CT angiography, which can be normal in the first days of the process [27]. Transcranial Doppler can be used to follow the course of the disease [26].

To date, there is no consensus on the optimal treatment of RCVS. Treatment is guided by case series, and it is limited to retrospective and observational data, as there are no randomized prospective clinical trials. Symptomatic relief and eliminating precipitating factors are the first steps in treatment [22,27]. Calcium channel blockers are often used to treat RCVS with good effect [19,28–30]. Contrary to historical belief, most authorities now recommend against the use of steroids in patients with RCVS [19,22]. In severe cases, including those with new-onset neurological deficit or worsening imaging findings, aggressive interventions are often employed. The internal carotid artery infusion of milrinone, verapamil, or nimodipine has been reported with varying degrees of success [18,20,31–33]. Balloon angioplasty has also been used in treating severe cases, but carries significant risk [20,31,34]. The use of intravenous prostacyclins has been reported, but has not been validated, in the pregnant population [35]. It is our practice to initiate treatment with a calcium channel blocker, and to hydrate intravenously to counteract the hypotensive property of the calcium channel blocker. We emphasize the importance of multidisciplinary care, utilizing the expertise of our critical care, neurology, and maternal–fetal medicine colleagues to ensure proper care.

Simple treatment, including maintaining hypertension, hypervolemia, and hemodilution (triple-H), with and without oral nimodipine, has been used successfully postpartum. In one report, hypervolemia was achieved by infusing 5% dextrose in normal saline at 400 cc/h in combination with 12.5 g albumin every 6 h. This regimen resolved the severe headache, and the treatment was weaned over the
ensuring days. As severe hypertension can result from hypervolemia, this approach is probably best avoided in pregnant patients [23,25].

RCVS is a rare disorder, with many symptoms overlapping with other disease processes. The exact pathophysiology remains unknown, and at this point, there is no clear consensus on the treatment. The goal of the treatment is to relieve cerebral vasoconstriction in hopes of mitigating potential neurological sequelae, and this should be accomplished in a tertiary, multidisciplinary care setting.

**Posterior reversible encephalopathy syndrome**

Preeclampsia, eclampsia, severe hypertension, and RCVS can all be complicated by PRES. A clinical diagnosis of PRES includes the presence of headaches, seizures, encephalopathy, and visual disturbances, as well as radiological findings of focal reversible vasogenic edema. The edema is best seen on MRI of the brain, most commonly involving the parietal–occipital lobes, followed by the frontal and temporal lobes, and least commonly involving the cerebellum [36]. Symptoms develop without prodrome, and progress over 12–48 h [28]. Visual symptoms often resolve in hours to days, but edema often lags behind clinical symptoms. In fact, one of the distinctive characteristics of PRES is the reversibility of the clinical and radiological abnormalities after appropriate treatment of precipitating factors [37].

Recommendations regarding the treatment of PRES are limited, not only because of the various etiologies leading to this diagnosis but mainly because of the lack of well-designed clinical trials. Although PRES is reversible when treatment is instituted, delayed diagnosis and treatment can result in chronic neurological sequelae. A 2005 study followed eight patients diagnosed over 4 years with PRES at one institution. Of the eight patients, five had hypertensive encephalopathy during pregnancy, two had eclampsia during the postpartum period, and one patient with chronic renal failure developed symptoms after immunosuppressive treatment. In all but one patient, neurological and radiological abnormalities resolved after appropriate treatment [38]. Potential inciting factors should be recognized and treated. If medications or cytotoxic immunosuppressive therapy is thought to be causative, the dosage should be decreased or stopped. As hypertension does occur in most patients with PRES, blood pressure should be lowered, often resulting in concomitant clinical improvement. Seizures are usually treated with phenytoin and other antiepileptic medications, unless the patient has eclampsia — in which case, magnesium sulfate is recommended. As symptoms and imaging studies improve, antiepileptic medications can be tapered, as patients do not appear to be at risk for a chronic seizure disorder. Lastly, as electrolyte disturbances, fluid overload, uremia, and sepsis may contribute to the development of PRES, these too should be treated [6,39].

**Stroke**

Cerebral vascular accidents are uncommon in pregnancy, occurring in 10.7 per 100,000 deliveries, but account for >12% of all maternal deaths. During pregnancy, the rate of stroke appears to increase, with reported incidence ranging from four to 34 per 100,000. The majority of strokes occur within 3 days of delivery in the postpartum period [40]. Risk factors for ischemic stroke include preeclampsia, eclampsia, chronic hypertension, migraines, cesarean delivery, sickle cell disease, systemic lupus erythematosus, thrombocytopenia, drug use (especially cocaine), African-American race, older age, greater parity, and multiple gestation [41].

It is critical when a stroke occurs to establish as quickly as possible whether it is ischemic or hemorrhagic. Brain imaging with MRI or CT should be done as soon as possible. MRI is the preferred imaging modality in pregnancy, with potentially better sensitivity at identifying small infarcts, but CT is often the first imaging study done as it is more readily obtainable. Gadolinium-enhanced MRI contrast should be avoided unless absolutely necessary because of the lack of data regarding safety to the fetus.

**Acute ischemic stroke**

The initial management of pregnant patients with stroke does not differ from that of nonpregnant patients. After confirming adequate oxygenation, circulatory integrity, and achieving euglycemia, goals
of therapy include defining the underlying vascular lesion, reperfusion, and preventing recurrence [42,43]. Avoiding hypotension and marked hypertension is important to preserve cerebral perfusion pressure. The mainstays of treatment include thrombolytic agents, antiplatelet agents, and anticoagulation [42]. Testing for various causes of thrombophilia is recommended to help determine potential underlying etiologies. Echocardiography and some form of cerebrovascular imaging are modalities that can help determine the underlying risk factors and etiology if initial workup is unrevealing.

**Thrombolytic therapy**

In the nonpregnant patient, thrombolytic therapy within the first 4.5 h of symptom onset is the most effective intervention for salvaging ischemic brain tissue [42]. Only recombinant tissue plasminogen activator (tPA) is approved for use in acute stroke in the nonpregnant patient [44]. There are reports of >200 pregnant woman who have received thrombolytic therapy for various indications including myocardial infarction, pulmonary embolus, superior vena cava syndrome, and ischemic stroke [45–48]. A 2006 report reviewed eight pregnant patients with ischemic stroke treated with thrombolytic therapy. Seven patients made a good recovery. One patient died from arterial dissection resulting from a complication of angiography. Three patients had miscarriages, three terminated the pregnancies, and there were deliveries of two healthy newborns [49].

tPA is FDA category C. It does not cross the placenta, and animal studies have not indicated a risk of teratogenicity [50]. Gravid or not, complications of thrombolytic therapy include ICH, systemic bleeding, and angioedema. Thrombolytic treatment in pregnancy may result in maternal hemorrhage, which could lead to preterm labor, placental abruption, fetal demise, or intra-partum hemorrhage. These risks need to be weighed against the benefits of thrombolytic therapy in reducing maternal mortality; however, about half of patients who survive pregnancy-related stroke retain residual neurological deficits [51]. The 2013 AHA stroke treatment guidelines still list pregnancy as an exclusion criterion. Therefore, treating a pregnant woman having a stroke with tPA requires a very careful discussion with the patient and family about risks and benefits, and it should involve the expertise of a stroke specialist [42].

When tPA is given, aspirin therapy (81 mg daily) for secondary stroke prevention should not be started until 24 h after the initial event [52]. In nonpregnant patients with ischemic stroke, aspirin initiated within 48 h improves long-term outcomes, and it reduces the risk of recurrent ischemia without the risk of major hemorrhagic complications [53]. If possible, aspirin should be stopped 7–10 days before delivery, as a slight increase in postoperative bleeding has been reported in some studies in patients who continued aspirin until the day of surgery [54].

Anticoagulation is not recommended in acute treatment of thrombotic stroke, and it should be delayed if feasible for 7–10 days to avoid ICH complications. In a patient with a preexisting indication for anticoagulation, most physicians will continue anticoagulation throughout pregnancy and for at least 6–8 weeks postpartum. The 2012 American College of Chest Physician guidelines recommend anticoagulation therapy for at least 6 weeks postpartum, and for a minimum total duration of 3 months, for pregnant women with acute venous thromboembolism [55]. The 2014 American Heart Association guidelines recommend that patients with ischemic stroke and an inherited thrombophilia be evaluated for deep vein thrombosis, which is an indication for potentially both short-term and long-term anticoagulation [56]. The prolonged postpartum anticoagulation course is recommended given the increased risk of venous thromboembolism in the postpartum state [1].

**Pregnancy-specific considerations with ischemic stroke**

If ischemic stroke occurs in pregnancy, treatment should be directed at protecting brain tissue, preventing complications, and controlling modifiable factors that worsen prognosis. Before 24 weeks, pregnancy termination can be considered, but it is unclear whether termination improves the overall maternal outcome. If maternal and fetal statuses are stable, patients should be expectantly managed until delivery can be planned later on in the pregnancy, preferably as close to a 39-week gestation as possible.
Recurrence of stroke

Patients who have had a stroke in a prior pregnancy often present for preconception counseling. There is sparse data to guide counseling, mainly in the form of case series and expert opinion. Case series suggest an overall low recurrence rate of 1 in 143, or 0.7%, especially if the causative vascular lesion has been treated [57–59]. A recurrent stroke is more likely to occur during the postpartum period than during the antepartum period [59]. In patients with an ongoing thrombophilia (e.g., Factor V Leiden), the risk of recurrence is higher, approaching 20% [57].

Underlying medical disorders including diabetes, hypertension, smoking, hyperlipidemia, and diabetes need to be addressed and optimized. For stroke recurrence prevention in pregnancy, American Heart Association recommendations are based on two scenarios: either a high-risk condition (i.e., mechanical heart valves) requiring anticoagulation outside of pregnancy or a lower-risk situation in which antiplatelet therapy would be the treatment recommendation outside of pregnancy [56]. Except for cardioembolic stroke, the effectiveness of heparin in preventing recurrent stroke has not been studied. Therefore, low-dose aspirin, unfractionated heparin (UFH), low molecular weight heparin (LMWH), or no treatment could be acceptable in subsequent pregnancy. As there are little data regarding the risk–benefit ratio of secondary prevention of non-cardioembolic stroke in the first trimester, the American Academy of Neurology has no consensus on this issue. Approximate percent recommendations in a 2009 American Academy of Neurology survey were 40% for aspirin 81 mg, 25% no treatment, and 10% unfractionated weight heparin or LMWH [60].

Intracranial hemorrhage

ICH is uncommon in pregnancy, with an incidence of five per 10,000, but when it occurs, mortality rates are as high as 40% [61]. The relative risk of ICH during pregnancy and 5 weeks postpartum is 5.6 times higher than in nonpregnant women [62]. Fourteen to 55% of hemorrhagic strokes in pregnancy result from severe preeclampsia, eclampsia, or HELLP [63]. Most often, however, pregnancy-related subarachnoid hemorrhage (SAH) and ICH are caused by aneurysmal rupture or bleeding from a vascular malformation [64]. Management involves airway evaluation and possible intubation, clot stabilization, deep venous thrombosis prophylaxis, blood pressure reduction, cerebral edema management, control of temperature and seizures, and definitive treatment of the aneurysm if one is found [65].

Cerebral aneurysms

Despite gestational vasodilation, plasma volume expansion, and increased hypertension, pregnancy does not appear to increase the risk of cerebral aneurysm rupture [66,67]. Typical presentation is with a thunderclap headache, vomiting, seizures, or reduced consciousness. Aneurysmal SAH is most common in the third trimester, and up to 6 weeks postpartum. This propensity for a rupture is likely due to increased vascular stress from expanded circulating blood volume and increased cardiac output. Moreover, hormonally driven hyperplasia of the intimal smooth muscle wall, with loss of elasticity, increases vulnerability to aneurysmal rupture [64,68,69].

Unruptured, asymptomatic, stable aneurysms typically do not require intervention during pregnancy. If aneurysmal SAH occurs during pregnancy, treatment with endovascular coiling or neurosurgical clipping improves maternal and fetal outcomes [70–73]. Once an aneurysm has been treated, the pregnancy can typically proceed to term.

Arteriovenous malformations

The prevalence of arteriovenous malformations (AVMs) in the general population has been reported to be 18 per 100,000 [74]. In a recent review of 58,429 deliveries, ischemic strokes developed in 21 patients, 11 had hemorrhagic strokes, and four had sinus thrombosis. Within the 11 hemorrhagic strokes, four were definitively attributed to AVM rupture [75]. Although rare, ICH during pregnancy is not uncommonly attributed to AVM rupture. Evidence regarding the risk of AVM rupture in pregnancy
is controversial. A retrospective study in 1990 indicated a 3.5% risk of hemorrhage during pregnancy, similar to age-matched, nonpregnant women [76]. A 2014 review also found an annual hemorrhage rate of 3.11% [77]. However, several case series and case reports have demonstrated a trend toward greater rates of hemorrhage from AVMs during a woman's reproductive years. A 2012 retrospective review described an 8.1% AVM rupture rate during pregnancy compared with the overall annual hemorrhage rate of 4.8% [78]. Another 1993 review reported a 9.3% rate of hemorrhage during pregnancy compared with 4.5% during the remainder of childbearing years [79]. Overall, there is no clear evidence to recommend women with unruptured AVMs to avoid pregnancy [80].

The same hemodynamic and endocrine changes of pregnancy that contribute to aneurysmal rupture likely also contribute to AVM hemorrhage. Treatment and intervention is the same as in nonpregnant women. In a review of 154 cases of spontaneous ICH during pregnancy, 23% were attributable to AVM rupture. Of 22 women who were observed with ruptured AVMs, there was a 32% maternal mortality rate and a 23% fetal mortality rate. The maternal mortality rate was 23% in cases where women required surgical intervention, and there were no reports of fetal death [64]. Microsurgical excision is the standard for treating symptomatic AVM, often with presurgical embolization, but endovascular embolization with coiling should also be considered [80]. Untreated vascular malformations are prone to re-bleeding. In one study, the annual rate of recurrent hemorrhage in women with an AVM was roughly 31% in the first year following an initial hemorrhage, and 6% in subsequent years. If possible, AVMs that have previously bled should be treated before a subsequent pregnancy [81].

**Delivery following ICH**

If the cerebral aneurysm or AVM has been definitively treated (e.g., clipped or coiled), and the cause of the cerebral hemorrhage has been treated, most sources indicate that patients may undergo labor and a vaginal delivery [82]. Vaginal delivery has not been reported to be associated with an increased risk of maternal bleeding complications. One recent study observed a 1.4% risk of ruptured aneurysm with pregnancy and 0.05% with delivery, comparable to that of the general population [76,79]. Vaginal delivery is not advised in the case of a ruptured, unsecured aneurysm, neurosurgery performed within a week of delivery, or incompletely treated AVM. Options then include a cesarean section or an assisted second stage of labor with forceps or a vacuum extractor, with the goal to avoid increased strain with valsalsva [83,84]. Whether delivery is planned via a cesarean section or an assisted vaginal delivery, controlling modifiable risk factors during labor and delivery, such as hypertension, is strongly recommended.

**Cerebral venous sinus thrombosis**

World statistics estimate cases of CVT to be 40–50 cases per 100,000 live births. The rate of death with CVT from all causes is 2–10%, but less when associated with pregnancy [85,86]. The occurrence of CVT has been related to the hypercoagulability of pregnancy, although an exact etiology is unknown, and likely multifactorial [85,87]. CVT has a variety of clinical presentations, depending on the site involved. Occlusion of the cerebral cortical veins can lead to focal neurological signs or seizures. Occlusion of the major venous sinuses can lead to intracranial hypertension, headache, vomiting, and papilledema. Cavernous sinus thrombosis can lead to a painful eye or exophthalmos [85,88]. Risk factors for CVT are hypertension, advanced maternal age, cesarean delivery, infections, and excessive vomiting. The treatment of CVT consists of anticoagulation, typically with UFH or LMWH [89]. 2014 American Heart Association guidelines recommend therapeutic anticoagulation throughout pregnancy, and continuing postpartum for at least 6 weeks, for a total minimum duration of 6 months. Seizures and intracranial hypertension are common, and they may also need to be treated [43,56].

**Summary**

Expeditious diagnosis and treatment of pregnant patients with acute neurological symptoms is crucial in reducing morbidity and mortality. Neurological emergencies include those unique to
pregnancy, as well as disease processes that occur in the general population. MRI and/or cerebrovascular imaging may be necessary for diagnosis when CT is non-diagnostic. Therapies are often the same as those used to treat nonpregnant patients, and they are most effective when initiated in a timely manner, as delay in treatment can lead to significant long-term neurological deficits. Immediate delivery is rarely necessary, although in cases in which delivery reduces disease severity (for instance, eclampsia), delivery is recommended after maternal stabilization.

Efficient multidisciplinary management, coordinating recommendations from specialists in neurology, critical care, neurosurgery, obstetrics, emergency medicine, anesthesiology, radiology, and neonatology, is necessary. Large randomized controlled trials investigating treatment options during pregnancy are greatly needed. Until there is more data specifically pertaining to the treatment of neurological emergencies in pregnancy, many interventions will be based on observational case reports or conclusions drawn from studies in the general population.

### Practice points

- During pregnancy, providers need to be aware of the effects of therapies and interventions on the developing fetus.
- An early delivery is often not indicated if there is no evidence of fetal distress, except for severe preeclampsia or eclampsia.
- Multidisciplinary care is needed when caring for pregnant patients with neurological emergencies.
- Significant hypertension in pregnancy can be treated with hydralazine, labetalol, or nifedipine.
- Magnesium sulfate is given to patients with severe preeclampsia or eclampsia for seizure prophylaxis.
- Tissue plasminogen activator can be considered to treat acute ischemic stroke in gravid patients, followed by low-dose aspirin.

### Research agenda

- Determine clinical application of angiogenic factors for preeclampsia
- Establish a registry of patients with stroke in pregnancy
- Ensure safety of thrombolytic therapy for ischemic stroke in pregnancy
- Determine best approach to treating aneurysms and intracranial vascular malformations in pregnant patients.

### Conflict of interest

None.

### References


