Reminder of important clinical lesson

Idiopathic stroke in a normal parturient

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Summary

A 34-year-old Chinese woman was admitted to the labour suite for induction of labour. She had no history of hypertension, vascular disease or coagulapathy. She was induced for less fetal movement at 38 weeks of gestation. Labour progressed smoothly, but immediately after delivery the patient had symptoms of facial neurological deficit, slurred speech and heaviness in the left side of the body. After completion of the third stage of labour, her condition soon deteriorated with left-sided facial palsy and left-sided dense hemiplegia. The patient was then transferred to the intensive care unit where an urgent CT scan was arranged for her. She was diagnosed as a case of right-sided stroke with dense left-sided hemiplegia and left facial palsy. She was placed under the care of the neurosurgical team for 2 weeks and was discharged on regular physiotherapy treatment. She had completely recovered 6 months later.

BACKGROUND

Stroke is an unusual but potentially devastating occurrence during pregnancy and the postpartum period. Maternal mortality is reported to be as high as 26% and survivors may face long-term neurological sequelae. In addition, associated fetal mortality and morbidity remain high. Although strokes are unavoidable, early interference can be key to saving lives and maintaining brain function in some patients. Therefore even if the probability that an obstetrician will ever encounter a patient who has a stroke is very low, it is critical that the signs are recognised, diagnosis is made swiftly and neurological and other consultations are obtained as appropriate.

CASE PRESENTATION

A 34-year-old woman, para 1+0, who had previously delivered a normal healthy baby by caesarean section for breech presentation in 2001 was admitted at 23 weeks of pregnancy into the peripheral clinic with blood pressure of 114/ 77 mm Hg. She was having abnormal glucose tolerance test (GTT) results and was recommended an appropriate diet. Family history was not significant. She was admitted at 38 weeks with less fetal movement and was induced with a prostaglandin E2 pessary, and labour progressed normally. The patient had seven antenatal visits, and over these visits her systolic blood pressure ranged from 114 to 120 mm Hg and her diastolic blood pressure ranged from 70 to 75 mm Hg. At the fully dilated cervix stage her pulse rate increased to 160 beats/min with blood pressure of 120/80 mm Hg. There was impaired consciousness with an inability to concentrate. The patient had a normal vaginal delivery and a healthy baby boy was born. Immediately after delivery, the patient presented with slurred speech with facial asymmetry and left-sided body weakness. The patient was transferred to the intensive care unit (ICU) where she was examined by the on-call doctor and diagnosed as a case of right-sided stroke with dense left-sided hemiplegia and left facial palsy. An urgent CT scan was arranged and the patient was transferred to a specialised hospital where the CT scan showed an intraparanchymal bleed in the right upper parietal region. The bleed measured approximately 55×22 mm, with slight brain oedema. The lesion caused a slight mass effect and a slight shift to the right side. Ventricles were within normal limits. Accordingly, a CT angiogram was performed to exclude arteriovenous malformation. The result showed no evidence of arteriovenous malformation, aneurysm malformation or any vascular abnormality.

The patient was kept under observation under the care of a neurosurgeon and a neuromedical team where they advised no surgical intervention unless there was a deterioration of symptoms and clinical signs. A follow-up nonenhanced CT scan of the brain showed an intracerebral haematoma in the right parietal region with surrounding perifocal oedema causing mild compression of the ipsilateral ventricle and a mild midline shift to the left side. There was also obliteration of the right sylvian fissure with effacement of the cortical sulci over the right convexity. In comparison to the previous CT scan, the right parietal intracerebral haematoma showed no change in size with slightly decreased density.

The patient was managed conservatively and discharged after 2 weeks of hospital stay to regular physiotherapy treatment, which she underwent for 6 months. The patient made a full recovery.

INVESTIGATIONS Radiological findings CT of the brain

This was performed on day 1 (9 January 2009). An acute intraparenchymal bleed could be seen in the right upper parietal region; the bleed measureed approximately 55×22 mm, with slight brain oedema around the bleed. The lesion caused a slight midline shift to the left side and the ventricle was within normal limits (figure 1).

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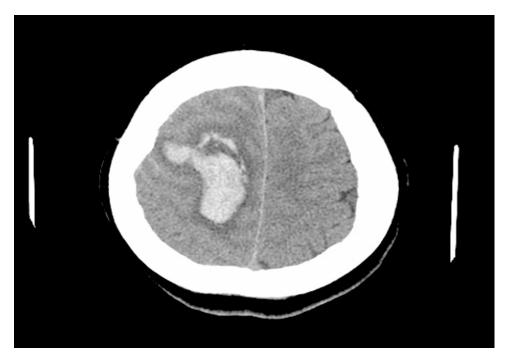


Figure 1 CT scan showing intraparenchymal bleed in the right upper parietal region (day 1).



Figure 2 Angiocerebral CT showing no evidence of arteriovenous malformation, aneurysm formation or any other vascular abnormality (day 4).

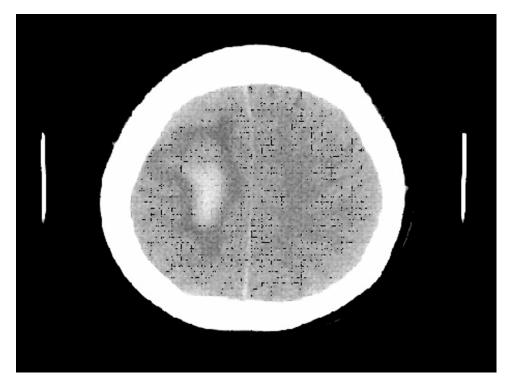


Figure 3 Non-enhanced CT scan of the brain showing intracerebral haematoma with no change in size with slightly decreased density (day 12).

Angiocerebral CT

This was performed on day 4 (14 January 2009). There was no evidence of arteriovenous malformation (AVM), aneurysm formation or any other vascular abnormality (figure 2).

Repeated CT of the brain

This was performed on day 12 (21 January 2009). A follow-up non-enhanced CT scan of the brain showed intracerebral haematoma in the right parietal region with surrounding perifocal oedema causing mild compression of the ipsilateral ventricle and a midline shift to the left side. There was also obliteration of the right sylvian fissure with effacement of the cortical sulci over convexity. In comparison to the previous CT scan, the right parietal intracerebral haematoma showed no change in size with slightly decreased density (figure 3).

Obstetric ultrasound

This was performed at 8 weeks and 27 weeks of gestation.

Haematological findings Full blood count

- ► Haemoglobin, 9.7 g/dl
- ▶ White blood cells, 5.1×10^3 cells/µl
- ► Platelet count, 254×10³ cells/µl

Coagulation profile

- ► Prothrombin time, 11.7 s
- ► International normalised ratio, 0.90
- ► Activated partial thromboplastin time, 30.1 s
- ► Fibrinogen, 505 mg/dl
- ▶ D-Dimer test, 8.68 mg/dl

Biochemistry findings Renal function tests

- ► Urea, 26 mg/dl
- Creatinine, 0.6 mg/dl

Immunological findings Thrombophilia screening

- ► Activated protein C resistance, 123.5 s (normal)
- ► Antithrombin III, 96% (normal)
- ► Protein C, 135% (normal)
- ▶ Protein S, 53% (normal)

Antibodies

- ▶ Anti-DNA negative
- ► Anti-nuclear factor positive
- Anti-cardiolipin antibody IgG and IgM negative

Microbiological findings

Midstream urine for culture and sensitivity; no growth.

DIFFERENTIAL DIAGNOSIS

- Complications of pregnancy such as pre-eclampsia, eclampsia.
- Migraine with an accompanying aura if there is an evolving neurological deficit.
- Septic aneurysm.
- Arterial dissection.
- ► Cerebral AVM.
- ▶ Dural arteriovenous fistula.
- Vascular lesion around the spinal cord.

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TREATMENT

The patient was placed on conservative management during her 2-week hospital stay and then underwent 6 months of physiotherapy treatment.

OUTCOME AND FOLLOW-UP

The patient was diagnosed as a case of right-sided stroke with dense left-sided hemiplegia and left facial palsy. She was placed under the care of the neurosurgical team for 2 weeks in a specialised hospital and was discharged on regular physiotherapy treatment. She had completely recovered 6 months later.

DISCUSSION

Pregnancy-related stroke is fortunately a rare event. However, when it occurs there may be implications for the management of the patient and the delivery of the child. The prevalence of intracranial haemorrhage during pregnancy ranges from 0.01% to 0.05% but results in high maternal mortality of 40% to 83% and accounts for 5% to 12% of all maternal deaths during pregnancy.

Pregnancies (and the perperium) are prothrombotic states. During pregnancy and for approximately 6 weeks after birth, all women are at increased risk of thromboembolic disease. Indeed in the last report on the confidential enquiries into maternal death in the UK, thromboembolism was the leading direct cause of maternal death. The reported incidence of pregnancy related stroke varies widely but probably lies between 11 and 26 deliveries per 100 000.²

One study³ showed a rate of ischaemic strokes of 11/100 000 deliveries, with the postpartum period representing the greatest period of risk. The same study also showed that intracerebral haemorrhage occurred at a rate of 9/100 000 of deliveries, again with the highest risk occurring in the postpartum period.

It is important to recognise that strokes occur in young women of childbearing age at a rate of 10.7 per 100 000.⁴ The risk is elevated during pregnancy for a number of reasons including hypercoagulability, venous stasis and blood pressure fluctuation. Indeed, some estimate that the risk of stroke is 13-fold higher in pregnant than in non-pregnant women, although the rarity of the condition makes the true prevalence a matter of debate.⁴

As in other patients, it is important when a stroke does occur to establish as quickly as possible if this is due to cerebral ischaemia or haemorrhage. Clinical history or physical examination is insufficiently sensitive to answer this question, so brain imaging in the form of CT or MRI should be carried out as quickly as possible after the onset of symptoms.

Stroke in younger adults is typically categorised as primarily ischaemic or haemorrhagic. Transient ischaemic attacks are rare at a young age. Haemorrhagic strokes are relatively more common. The cause can be found in 55% to 93% of young patients. 5-7

MRI is the preferred imaging option in pregnancy. CT scanning can expose the fetus to radiation; however if MRI is not available, the benefit of CT scanning greatly outweigh the risks in this situation and it should be performed.

As is the case with all younger patients with stroke, there should be an extensive diagnostic assessment including vascular studies of the extracranial vessels, cardiac investigations and thrombophilia screening. Intracranial haemorrhage has been estimated to constitute 2% to 7% of the total cases of neurological disorder related to pregnancy.² In many cases this relates to uncontrolled hypertension.

Haemorrhage from either a subarachnoid bleed or intracerebral bleed takes place most frequently in the second trimester and the postpartum period. A bleed from an underlying aneurysm can take place with equal frequency in all trimesters and the perperium, while bleeding from an underlying AVM occurs most frequently after the first trimester and in the postpartum period.

Subarachnoid haemorrhage (SAH) mostly from aneurysm accounts for 3% of all strokes. A saccular aneurysm will be responsible in approximately 85% of patients with SAH. In 10% of patients, SAH is secondary to non-aneurysmal perimesencephalic haemorrhage.²

There are various ischaemic stroke risk factors that include hypertension, diabetes, sickle cell disease, thrombophilia, smoking, heart disease, multiple gestations and greater parity. In addition, caesarean delivery, alcohol and recreational drug abuse, mainly cocaine, also represent other risk factors. Furthermore, complications of pregnancy such as pre-eclampsia, eclampsia, hyperemesis and disturbance of the electrolyte and fluid balance are major risk factors for stroke.

A study made on a large Swedish cohort of more than 650 000 women with more than 1 million deliveries over an 8-year time period concluded that the greatest risk of ischaemic and haemorrhagic cerebral events was in the 2 days before and 1 day after delivery, with an increased but declining risk over the subsequent 6-week postpartum period. It has been estimated that there is a mortality of 10% to 13% following pregnancy-related stroke, and that this is disproportionately higher in black women, in older patients and in those with no parental care. 89

Antiphospholipid syndrome can be triggered by pregnancy. This may present with stroke, which may be either arterial or venous. Diagnosis rests on the detection of one or more anti-phospholipid antibodies; lupus anticoagulant, anti-cardiolipin antibody and anti-B2 glycoprotein.¹⁰

Women who are pregnant or in the postpartum period account for 10% to 25% of patients with thrombotic thrombocytopaenic purpera (TTP). TTP may present with a wide variety of neurological symptoms including strokes, transient ischaemic attacks, fluctuating neurological symptoms, headaches, seizures and confusion. ¹⁰

Peripartum or postpartum angiopathy, a cerebral dysregulation syndrome affecting large and medium sized cerebral artery, may be associated with haemorrhagic or ischaemic strokes. The clinical picture typically occurs in women aged 20–50 years and is usually one of abrupt onset with severe thunderclap headaches, seizures and focal neurological deficit. The imaging findings may often be suggestive, as white matter changes have a posterior emphasis and are usually not as extensive compared with eclampsia. Diffusion-weighted MRI may help in differentiation. ^{11–14}

Learning points

- Although uncommon, the development of stroke during pregnancy and purperium can be a challenging diagnostic and management problem. For this reason we advise that such patients are managed in a specialised multidisplinary unit with rapid access to expertise in obstetric, neurological, neuroradiological and rehabilitation services.
- Strokes can happen at any time. The clinical presentation is similar to that seen in patients who are not pregnant; however, these symptoms can mimic those seen in pre-eclampsia and eclampsia, and because of that the possibility of stroke may be overlooked.
- It is prudent to counsel all patients and especially those with relevant risk factors to seek care for any symptom that may be associated with stroke: headaches, visual changes, epigastric pain, seizures, nausea, vomiting or neurological defects (focal or global). Severe hypertension and widened pulse pressures are also symptomatic of stroke.

Competing interests None.

Patient consent Obtained.

REFERENCES

- Philip MM, Van VH, Adil MM, et al. Endovascular treatment of cerebral artery aneurysms during pregnancy: report of three cases. Am J Neuroradiol 2000;21:1306–11.
- Davie CA, O'Brien P. Stroke and pregnancy. J Neurol Neurosurg Psychiatr 2008;79:240–5.
- Sharshar T, Lamy C, Mas JL. Incidence and causes of strokes associated with pregnancy and puerperium. A study in public hospitals of lle de France. Stroke in Pregnancy Study Group. Stroke 1995;26:930–6.
- 4. **Baha S.** Stroke in pregnancy and the postpartum period. *OB/GYN News* 2005.
- Adams HP Jr, Butler MJ, Biller J, et al. Nonhemorrhagic cerebral infarction in young adults. Arch Neurol 1986;43:793–6.
- Grindal AB, Cohen RJ, Saul RF, et al. Cerebral infarction in young adults. Stroke 1978;9:39

 –42.
- Toffol GJ, Biller J, Adams HP Jr, Nontraumatic intracerebral hemorrhage in young adults. Arch Neurol 1987;44:483–5.
- James AH, Bushnell CD, Jamison MG, et al. Incidence and risk factors for stroke in pregnancy and the puerperium. Obstet Gynecol 2005;106:509–16.
- Chang J, Elam-Evans LD, Berg CJ, et al. Pregnancy-related mortality surveillance—United States, 1991–1999. MMWR Surveill Summ 2003;52:1–8.
- Austin S, Cohen H, Losseff N. Haematology and neurology. J Neurol Neurosura Psychiatr 2007;78:334

 –41.
- Call GK, Fleming MC, Sealfon S, et al. Reversible cerebral segmental vasoconstriction. Stroke 1988;19:1159–70.
- Raps EC, Galetta SL, Broderick M, et al. Delayed peripartum vasculopathy: cerebral eclampsia revisited. Ann Neurol 1993;33:222–5.
- Donaldson JD. Eclampsia and postpartum cerebral angiopathy [editorial]. J Neurol Sci 2000;178:1.
- Singhal AB. Postpartum angiopathy with reversible posterior leukoencephalopathy. Arch Neurol 2004;61:411–6.

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