Stroke during induction of labour in a patient with carotid aneurysm and prior multiple venous thromboses

Q1 **Sujata Datta, Jeremy S Stern and Bode Williams**

Q2 Frimley Park Hospital, Surrey, UK

Summary: A case of stroke during induction of labour in a pregnant patient at term anticoagulated for prior venous thrombosis is reported. The cause was a middle cerebral artery embolism, originating from a false dissecting aneurysm of the internal carotid artery. Investigations and causes of stroke in a pregnant patient are briefly outlined.

Q3 **Keywords:**

A 30-year-old women developed acute weakness and sensory disturbance in the left arm and leg during induction of labour at 40 weeks gestation.

This was her third pregnancy. Prior to her first pregnancy, she was on warfarin as she had three episodes of deep vein thrombosis and a pulmonary embolism. She had no identifiable thrombophilic trait or any family history of venous thrombosis. Her first pregnancy resulted in therapeutic termination at 8 weeks gestation due to unintended conception on warfarin. Her second pregnancy ended in a spontaneous miscarriage at 9 weeks gestation. Lupus anticoagulant was negative.

She sought pre-pregnancy counselling following her miscarriage and was switched from warfarin to therapeutic low-molecular-weight heparin (LMWH) throughout pregnancy. She had joint obstetrics and haematology antenatal care and her pregnancy remained problem free. Heparin was omitted 36 hours prior to planned induction of labour.

Five hours following the initial dose of vaginal prostaglandin, she complained of a severe headache, tingling face sensation, weakness and numbness of the left upper and lower limbs. There was no associated visual or speech disturbance. She was normotensive with no proteinuria. Brain magnetic resonance imaging (MRI) showed an acute infarct in the right frontoparietal area (Figure 1) and appearances consistent with a dissecting aneurysm of the right internal carotid artery (Figure 2).

Over the following 24 hours, she made a substantial neurological recovery. Delivery was expedited by caesarean section with spinal anaesthesia. Postnatally, LMWH was re-commenced immediately and she was transferred to a stroke unit for rehabilitation. Trans-oesophageal echocardiogram and 24-hour ECG were normal. In addition, thrombophilia tests including antithrombin III, protein C and S, factor 8, activated protein C, prothrombin mutation, methylene tetrahydrofolate reductase (C677T), factor V Leiden mutation, lupus anticoagulant, anticardiolipin antibodies and Janus kinase-2 mutation analysis for myeloproliferative disorders were also negative either in the past or present.

She was discharged home 11 days after delivery on therapeutic LMWH and with no persisting neurological deficit. Five weeks later, she was admitted to the regional neuroscience unit where MRI and computed tomography angiogram confirmed the presence of thrombus within the lumen of a dissecting aneurysm of the right internal carotid artery. Expansion of the right bony petrous apex indicated that the aneurysm was longstanding. It was felt that conservative management was most appropriate. She was re-commenced on warfarin and had a levonorgestrel intrauterine device fitted for contraception. A year later, the base of the aneurysm was enlarged by 2 mm and remains under annual MRI monitoring. She has been advised to avoid another pregnancy as we feel that her next pregnancy and delivery will be associated with an increased risk of thrombotic stroke upon withdrawing anticoagulation.

Correspondence to: Bode Williams, Consultant Obstetrician and Gynaecologist
Q2 **Email:** sirbodie@yahoo.co.uk

DISCUSSION

Stroke during pregnancy is a rare event. The true incidence is not known because of the varied populations studied. The excess or attributable risk of stroke during and within six weeks of pregnancy is 8.1 strokes per 100,000 pregnancies. Furthermore, stroke is more likely to occur after delivery.[1] The relative risk of cerebral infarction in the antenatal period is 0.7, rising in the postpartum period to 8.7[1] and the perinatal period is also associated with a higher risk of venous embolism, including cortical venous thrombosis.[2]

Our patient had previous recurrent venous thrombosis outside pregnancy, followed by a stroke when anticoagulation was withdrawn prior to delivery. Unusually, this was in association with a second lesion, an apparently longstanding dissecting carotid false aneurysm whose presence was not associated with any previous trauma. It is likely that the cerebral embolic event secondary to thrombus within the aneurysmal lumen was provoked by the interruption of anticoagulation during the induction of labour.

We would postulate that our patient has an as yet undetermined underlying thrombophilic disorder given her repeated venous and

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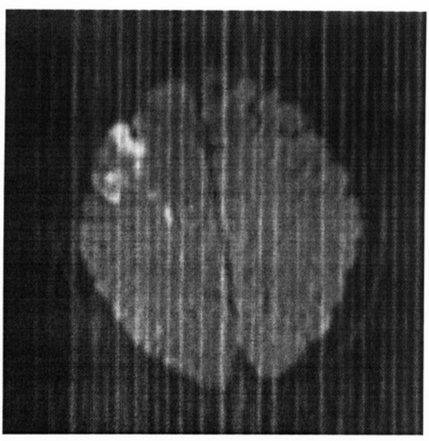

Figure 1 Diffusion-weighted brain magnetic resonance imaging showing acute frontoparietal infarct

now an arterial event. Furthermore, the stroke probably resulted from an unusual combination of first inherent thrombophilic factors additionally exacerbated by the state of late pregnancy and second an arterial vascular thrombogenic lesion.

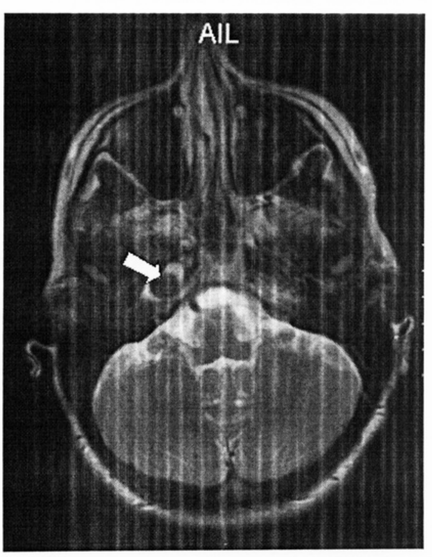

Figure 2 T2-weighted brain magnetic resonance imaging showing expanded internal carotid artery containing thrombus

The causes of thromboembolism include acquired and genetic thrombophilic disorders, valvular cardiac disease, bacterial endocarditis, arrhythmias, vasculitis, myeloproliferative disorders and dissection of vessels.[3–6] Further risk factors within pregnancy include pre-eclampsia, caesarean delivery and postpartum infection.[2,7]

Anticoagulation in an at-risk pregnancy is a challenge for obstetricians and physicians. Chronically anticaogulated patients on warfarin require carefully coordinated pre-pregnancy and antenatal care among all health-care professionals. The standard practice is to switch from warfarin to therapeutic doses of LMWH pre-pregnancy or in the early first trimester to minimize the risk of warfarin embryopathy.[8] Patients with a prior history of stroke would also benefit from concomitant administration of low-dose aspirin prophylaxis (60–150 mg) during pregnancy to further minimize the recurrent risk of a cerebrovascular thrombo-embolic event.[9] However, the need to interrupt LMWH prior to planned induction or caesarean section in order to time regional anaesthesia must be balanced against the increased risk of thromboembolic complications. The anticoagulation free interval must be kept to a minimum and treatment resumed as soon as possible after delivery. The consensus view is that patients on LMWH thromboprophylaxis should have epidural needle placement 12 hours after the last dose. Patients receiving higher or treatment doses of LMWH should have epidural needle placement delayed for at least 24 hours after the last dose to ensure normal haemostatsis at the time of needle insertion.[9] If anticoagulation free interval is prolonged due to unexpected delays in establishing labour or delivery complications, then low-dose unfractionated subcutaneous heparin administration could be considered to bridge the gap as prophylaxis against venous thrombosis.[10]

Although in the case presented here two apparently separate aetiologies for stroke were identified, in fact it would not be unusual for no such further factor to be identified in association with a stroke in pregnancy. However, thorough investigation is always required to identify possible underlying causes to reduce maternal morbidity.

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