

Metastatic choriocarcinoma: A rare cause of stroke in the young adult

N Saad, YM Tang, E Sclavos and SL Stuckey

Department of Radiology, Princess Alexandra Hospital, Brisbane, Queensland, Australia

SUMMARY

A 20-year old woman presented with acute right hemiplegia 10 weeks after intrauterine fetal death at 34-weeks gestation (G1P0). A brain MRI showed a typical acute infarct in the left middle cerebral artery territory. A CT pulmonary angiography carried out 1 week later for sudden cardiopulmonary deterioration showed disseminated pulmonary metastases. The CT was extended to the abdomen and pelvis, which showed a uterine mass with bilateral theca lutein cysts. The radiological diagnosis of metastatic choriocarcinoma was made and was subsequently confirmed with the markedly increased serum β -human chorionic gonadotrophin. A serum β -human chorionic gonadotrophin is therefore considered worthwhile in any young female adult who presents with stroke.

Key words: *choriocarcinoma; metastases; magnetic resonance imaging; stroke.*

INTRODUCTION

The increased risk of cerebral infarction during pregnancy and in puerperium has been well documented.^{1,2,3} In many of these cases, the precise aetiology cannot be identified. In those where a pregnancy-associated cause can be identified, the most common is eclampsia. We present a rare case of metastatic choriocarcinoma with an initial presentation of stroke.

CASE REPORT

A 21-year-old female presented with acute onset of right arm and leg weakness, 2 months after an intrauterine fetal death at 34-weeks gestation. This was rapidly followed by a right facial palsy and dysphasia. A CT scan showed a large acute left middle cerebral artery (MCA) territory infarct. The MRI axial fluid-attenuated inversion recovery and diffusion-weighted imaging confirmed the acute infarction in the left MCA territory (Fig. 1a,c). It also disclosed small foci of abnormally high-T2 signal in the contralateral hemisphere (Fig. 1b,d). This raised concern for a vasculitic aetiology to her stroke and a full vasculitic screen was carried out, all of which was normal. Magnetic resonance angiography confirmed left MCA occlusion (Fig. 1e).

A week later, she had a witnessed cardiopulmonary arrest, was defibrillated and returned to normal cardiac rhythm and output.

A chest X-ray showed multiple bilateral pulmonary masses (Fig. 2a), which were seen to a better advantage on a CT pulmonary angiography carried out to exclude pulmonary embolism. Extensive pulmonary masses with multiple bilateral pulmonary arterial and venous filling defects were also depicted (Fig. 2b–d). These findings favoured cannon ball pulmonary metastases and tumour emboli in the pulmonary arteries and veins.

Computed tomography of the abdomen and pelvis showed a large, heterogeneous uterus with large bilateral adnexal cysts (Fig. 3). The findings were highly suggestive of a malignant trophoblastic uterine neoplasm with bilateral theca lutein cysts, innumerable pulmonary deposits and both arterial and venous tumour thrombi. An urgent serum β -human chorionic gonadotrophin (β -HCG) was grossly increased at 1.4 million IU/L, confirming the diagnosis.

Following anticoagulation, the patient's admission was complicated by haemorrhagic transformation of the large MCA

N Saad MB ChB, MS, MD, FRANZCR; **YM Tang** MB BS, FRANZCR; **E Sclavos** MB BS, FRANZCR; **SL Stuckey** MB BS, FRANZCR, MMed.

Correspondence: Dr Nivene Saad, Department of Radiology, Princess Alexandra Hospital, Ipswich Road, Woolloongabba, Qld 4102, Australia.

Email: nivene_f_saad@health.qld.gov.au

Submitted 10 November 2004; accepted 1 March 2006.

doi: 10.1111/j.1440-1673.2006.01631.x

© 2006 The Authors

Journal compilation © 2006 Royal Australian and New Zealand College of Radiologists

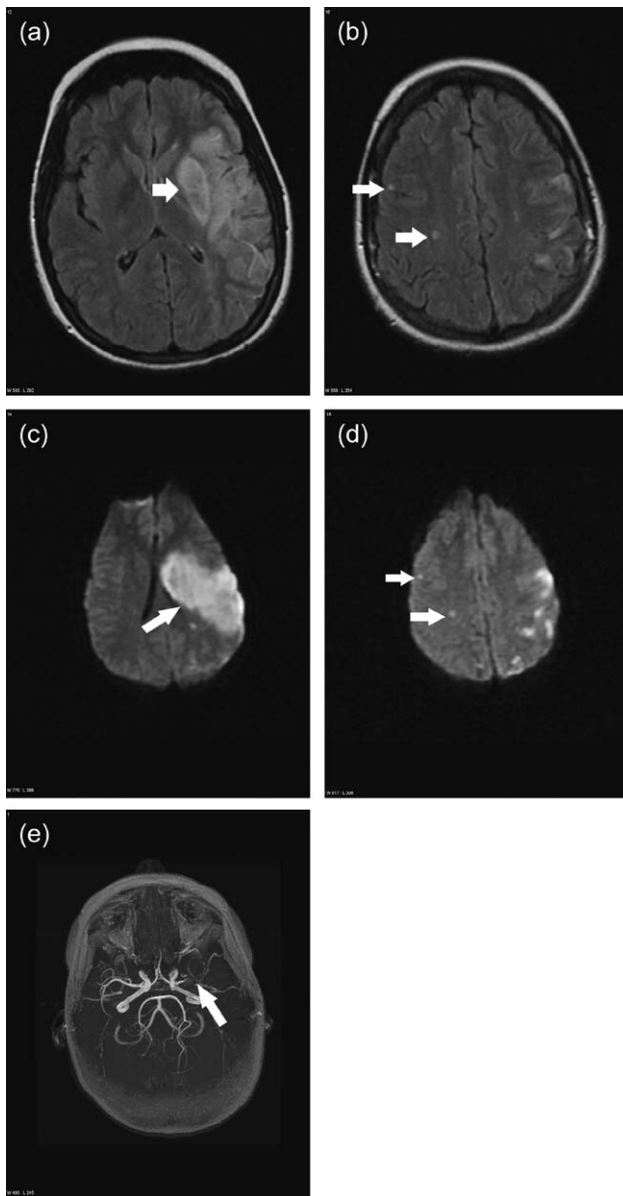


Fig. 1. (a) Axial T2 fluid-attenuated inversion recovery (FLAIR). Abnormal hyperintense signal in the distribution of the left middle cerebral artery (MCA) (arrow). (b) Axial T2 FLAIR. Several foci of increased signal are also present in the distribution of the right MCA (arrows). (c) Axial diffusion-weighted imaging (DWI). Corresponding restricted diffusion in the left MCA territory (arrow). (d) Axial DWI. Foci in the right MCA territory were also noted (arrows). (e) Time of flight MR angiography shows occlusion of the M1 portion of the left MCA (arrow).

infarct, shown on a CT scan (Fig. 4a). A repeat MRI with intravenous contrast confirmed the haemorrhagic transformation, yet did not suggest the presence of cerebral metastases (Fig. 4 b–d).

The patient was thereafter managed conservatively and has made a slow but incomplete neurological recovery. Chemotherapy was commenced and her serum β -HCG level continues to decline.

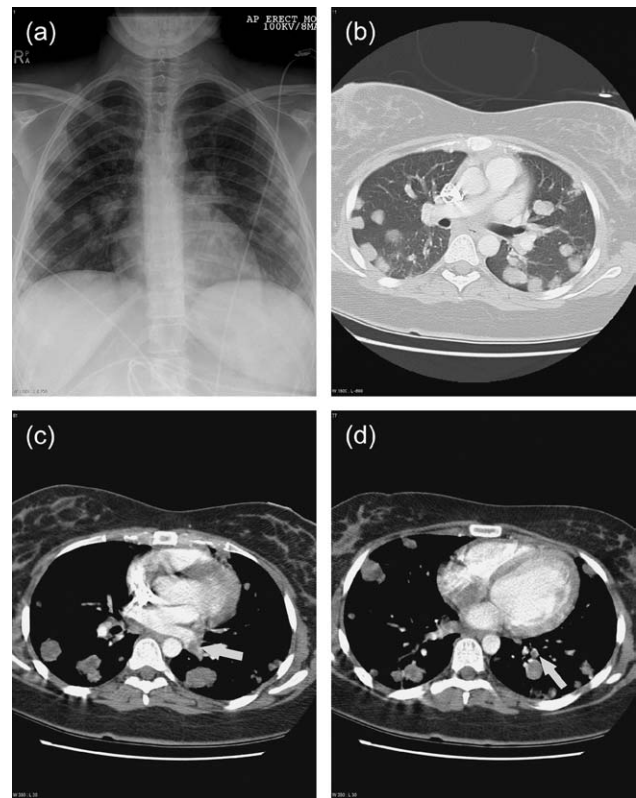


Fig. 2. (a) Chest X-ray showing multiple bilateral pulmonary masses. (b) Computed tomography chest in lung window also shows multiple pulmonary masses. (c) Computed tomography pulmonary angiography shows filling defects within the pulmonary veins (arrow). (d) Computed tomography pulmonary angiography also identifies filling defects in the pulmonary arteries (arrow).



Fig. 3. Computed tomography abdomen. (a) Abnormal heterogeneous density within the uterine cavity (arrowhead) and a left adnexal cystic mass (arrow). (b) Image at a lower level again displays abnormal heterogeneous density within the uterine cavity (arrowhead) and a cystic mass posterior to the uterus, likely the right ovary in the pouch of Douglas (arrow), suggestive of theca lutein cyst. These bilateral ovarian cysts were confirmed on an ultrasound scan but not pathologically proven.

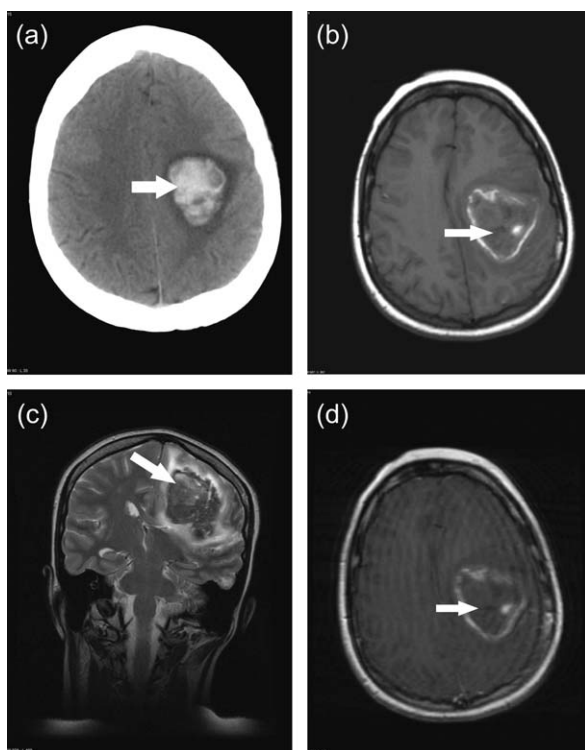


Fig. 4. (a) Axial non-contrast CT brain carried out 3 days later when the patient's neurological state deteriorated. Haemorrhagic transformation of the left middle cerebral artery (MCA) infarction was evident (arrow). (b) Axial T1-MRI confirmed the low-signal acute haemorrhagic component (arrow). (c) Coronal T2-MRI shows the low-signal rim of the haemosiderin (arrow) as well as heterogeneous signal centrally. (d) Axial T1 with gadolinium at the same level as Figure 4b. There was no abnormal enhancement in either of the MCA territories. The central low-signal area (arrow) was unchanged.

DISCUSSION

The risk of cerebral infarction during pregnancy and in puerperium is increased, although the precise pathogenesis remains poorly understood.¹ Postulated mechanisms include the hypercoagulable state and vessel wall changes associated with pregnancy. The main pregnancy-associated cause is eclampsia. Other pregnancy-specific causes include amniotic fluid embolism and choriocarcinoma.² Choriocarcinoma may metastasize to the cerebral blood vessels, causing thrombosis or aneurysm formation and resulting in ischaemic stroke or intraparenchymal haemorrhage, respectively.^{3,4} However, in a significant number of patients the cause remains undetermined.⁵

The incidence of arterial ischaemic strokes associated with pregnancy or early puerperium is approximately 3.8–5 in 100 000 pregnancies, which is not dissimilar to that in women of child-bearing age.¹ This is less common than the incidence of cerebral venous thrombosis, which is estimated as 10–20 per 100 000 deliveries in occidental countries (higher rates of 200–500 per 100 000 have been reported in India).⁵

Gestational choriocarcinoma is a malignant neoplasm that arises from placental trophoblastic tissue. It has a tendency to early metastases, especially to the lungs, brain, liver and vagina.⁶ The clinical presentation of cerebral involvement include raised intracranial pressure, intracerebral haemorrhage and cerebral ischaemic infarction.

The diagnosis of choriocarcinoma is made by measuring the hormone β -HCG, which is secreted by trophoblastic tissue. Histopathological proof of choriocarcinoma is not required to initiate therapy,⁷ especially when the levels are increased relative to term.⁸ Approximately half of all the choriocarcinomas are preceded by a molar pregnancy, whereas 25% follow term pregnancies and the remaining 25% follow an abortion or ectopic pregnancy.⁷ This tumour is very sensitive to chemotherapy, with high cure rates, even in advanced stages with cerebral metastases.⁹ Surgical therapy is only required in cases of haemorrhage in patients with isolated tumours not responding to chemotherapy.⁷

CONCLUSION

This case report emphasizes the importance of considering the diagnosis of choriocarcinoma in a woman of child-bearing age who presents with a stroke. History of previous pregnancy should be actively sought. Serum β -HCG level estimation should also be obtained, allowing prompt diagnosis and initiation of therapy for a curable disease.

REFERENCES

1. Leys D, Lamy C, Lucas C *et al*. Arterial ischaemic strokes associated with pregnancy and puerperium. *Acta Neurol Belg* 1997; **97**: 5–16.
2. Mas JL, Lamy C. Stroke in pregnancy and the puerperium. *J Neurol* 1998; **245**: 305–19.
3. Gurwitt LJ, Ling JM, Clarh RE. Cerebral metastatic choriocarcinoma: a postpartum cause of stroke. *Obstet Gynecol* 1975; **45**: 583–8.
4. Komeichi T, Igarashi K, Takigami M, Saito K, Isu T, Itamoto K. A case of metastatic choriocarcinoma associated with cerebral thrombosis and aneurysmal formation. *No Shinkei Geka* 1996; **24**: 463–7.
5. Lamy C, Sharshar T, Mas JL. [Cerebrovascular diseases in pregnancy and puerperium.] *Rev Neurol (Paris)* 1996; **152**: 422–40. (In French.)
6. Ilancheran A, Ratnam SS, Baratham G. Metastatic cerebral choriocarcinoma with primary neurological presentation. *Gynecol Oncol* 1988; **29**: 361–4.
7. Flam F, Holtz A. Case report: choriocarcinoma presenting with brain metastases. *Neurol Res* 1994; **16**: 403–4.
8. Picone O, Castaigne V, Ede C, Fernandez H. Cerebral metastases of a choriocarcinoma during pregnancy. *Obstet Gynecol* 2003; **102**: 1380–83.
9. Baertschi E, Notter M, Miranov A, Wernli M, Bargetzi MJ. Cerebral metastases in choriocarcinoma: a case report. *Schweiz Rundsc Med Prax* 2003; **92**: 763–8.