Left atrial metastasis presenting as recurrent embolic strokes

N CHALMERS, I W CAMPBELL

From the Victoria Hospital, Kirkcaldy, Fife, Scotland

SUMMARY Tumour emboli from an endocardial metastasis of an endometrial stromal sarcoma caused recurrent and ultimately fatal strokes in a woman of 40. Endocardial metastases are rare. They are known to embolise to the pulmonary and systemic arteries but not to cause recurrent cerebral emboli.

Case report

A 40 year old woman who gave a 3–4 year history suggestive of migraine was admitted with a right hemiparesis and aphasia of sudden onset. She smoked 15 cigarettes per day. She had had unilateral headache usually on the left occurring every few months. On several recent occasions these had been associated with complete loss of vision lasting 20 to 30 minutes. Eighteen months previously a hysterectomy had been performed for menorrhagia. At operation a hard inflammatory mass was found in the sigmoid colon. Histological examination of the uterus revealed stromal adenomyosis which was described as extremely cellular with occasional mitotic figures. A barium enema showed moderate diverticular disease of the sigmoid colon only. Fibreoptic sigmoidoscopy showed no abnormality and the chest radiograph showed a sharply defined circular lesion behind the heart in the left lower lobe of the lung. This was interpreted as a benign cyst.

At admission there was no lymphadenopathy. Blood pressure was 120/80 mm Hg and pulse 80 per minute and regular. No cardiac murmurs or vascular bruits were heard. Examination of the chest and abdomen was normal. There was complete aphasia and a dense right hemiparesis. There was pronounced hyperreflexia on the right with knee and ankle clonus and an extensor right plantar response. After admission she rapidly recovered and 48 hours later had only a mild residual dysphasia.

Initial investigations showed a mild neutrophilia but otherwise normal full blood count and plasma viscosity. Urea, electrolytes, liver function tests, and plasma glucose were normal. Serum cholesterol and triglyceride concentrations were also normal. An electrocardiogram showed only right bundle branch block and no abnormality was seen on the chest radiograph, the previously noted left lower lobe lesion being completely obscured by the cardiac shadow on this occasion. Skull radiographs showed calcification in the falx. Computed axial tomography showed a low density area in the territory of the left middle cerebral artery, indicating an infarct. Treatment with aspirin was started because there appeared to have been a spontaneous arterial occlusion with no obvious precipitating cause.

Four months later she was readmitted with a further episode of transient right hemiparesis and dysphasia. Again there was a rapid recovery. On this occasion the retrocardiac shadow was noted on the chest radiograph and in addition there was a mass at the right hilum. Bronchoscopy was normal. Three weeks later, after a third transient right hemiparesis, she suddenly sustained a dense left hemiparesis associated with loss of consciousness and several grand mal convulsions. There was no improvement in level of consciousness and she died two days later.

A large cystic infarct in the left cerebral hemisphere and a recent haemorrhagic infarct in the right frontoparietal region were found at necropsy. There was a 3.5 cm diameter secondary tumour at the hilum of the right lung and a 2 cm tumour in the left lower lobe. In addition there was a 5 cm diameter tumour in the left atrium (fig 1). There was also a large tumour mass in the pelvis; it involved both ovaries and the rectosigmoid junction. Histological
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examination showed that these were secondary deposits of an endometrial stromal sarcoma with the same features as the hysterectomy specimen obtained two years before. There was an organised infarct in the spleen but the liver and kidneys were normal. There were atheromatous plaques at the origins of both internal carotid arteries. A final diagnosis of recurrent cerebral emboli from a left atrial metastasis of an endometrial stromal sarcoma was made.

Discussion

Whereas the myocardium and pericardium are not infrequently the site of metastatic tumours, the endocardium is rarely the site of secondary deposits.1-3 In 1950 Coller et al found only nine cases in published reports.4 This is presumably because of the rapid blood flow within the heart coupled with the relatively small surface area of the endocardium compared with, say, the vascular bed of the lungs. It may be that endocardial metastases result from tumour cells lodging in subendocardial capillaries, rather than implanting directly on the endocardial surface. In most of the cases in Coller’s series the valves were affected, and it has been suggested that valve damage may be a prerequisite for tumour implantation. Thomas et al have reported three cases of left atrial metastases, all associated with underlying abnormalities of the mitral valve, of which two had intra-atrial thrombus. They proposed that circulating tumour cells initially lodge in the thrombus.5 In our case, however, the valves were normal and the atrium contained no thrombus. It is also recognised that an intracardiac secondary tumour can exist within the right atrial cavity as a tumour “culture” without vascular attachment to the endocardium.6 In the present case the tumour adhered firmly to the endocardium.

Systemic arterial tumour embolism producing effects by vascular occlusion is also rare. It was described in left atrial myxoma7-9 and as a complication of primary sarcoma of the heart8 and of left atrial metastases.5 There are also several reports of systemic embolisation of pulmonary secondaries and primary bronchogenic carcinomas. Embolisation of the latter is liable to occur perioperatively,10-11 but can be the presenting feature of a bronchogenic neoplasm.12 Fatal pulmonary embolism of a right atrial metastasis of osteogenic sarcoma has been reported.6,13 In addition there have been reports of cases of pulmonary metastases directly spreading to the left atrium along a pulmonary vein that were associated with systemic embolisation14-16 and also a case of recurrent bilateral femoral artery embolism of a metastasis of an osteogenic sarcoma invading the aorta at its bifurcation.17 Lastly, there is a report of a patient with left ventricular and left atrial endocardial metastases who died of myocardial rupture after tumour embolus to a coronary artery.18

The present case had pulmonary as well as intracardiac metastases, so it is conceivable that the pulmonary lesions were the source of the cerebral emboli. There was no evidence of spread along pulmonary veins, however, and we consider it much more likely that the emboli arose from the intracardiac lesion. No tumour emboli were found in the cerebral circulation so we do not know whether the cerebral emboli were fragments of tumour or of an adherent clot. There was no evidence of adherent clot at necropsy and it seems more likely that the small tumour fragments in the cerebral circulation had either dispersed or were simply not visible on the sections examined histologically.

Echocardiography was not performed in this case because a cardiac source of emboli was never suspected from the history and clinical findings. An echocardiographic examination would probably have enabled the diagnosis to be made antemortem; however, this is unlikely to have been of much benefit to the patient. It does, however, emphasise the point that echocardiography is valuable in cases of suspected systemic or cerebral emboli, even in the absence of overt cardiac disease.19,20

We believe that recurrent cerebral emboli from an intracardiac metastasis has not been reported before. Intracardiac metastases are rare, but occult cardiac disease can cause transient cerebral ischaemic attacks and echocardiography should be included in the investigation of patients in whom there is no obvious source of emboli.

Figure A 5 cm diameter metastasis arose from the endocardium of the left atrium (arrowed).

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