A right atrial mass: thrombus, tumour or other?

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INTRODUCTION

Strokes are commonly caused by cardiac abnormalities. This article describes a patient with systemic and pulmonary emboli and a right atrial mass.

DISCUSSION

Cardiac abnormalities have been noted in over half of the strokes of uncertain origin (Horstkotte et al, 1995) and may result from embolization of thrombi, tumours or vegetations.

Thrombi are most commonly the result of underlying cardiac disease. Ventricular thrombi occur in the setting of systolic dysfunction as a result of dilated cardiomyopathy or ischaemic heart disease. Atrial thrombi occur in the setting of atrial fibrillation or mitral valve disease. Thrombi are less commonly caused by hypercoagulable states such as the antiphospholipid antibody syndrome, pregnancy and certain malignancies.

Cardiac tumours commonly occur in the setting of metastatic malignancy, usually from breast, lung or malignant melanoma. Primary cardiac tumours occur much less frequently and are usually benign (about 75%). Of these benign tumours almost half are myxomas (Colucci et al, 1997). If multiple myxomas are found, first-degree relatives should be screened with echocardiography as 10% of myxomas are familial (autosomal dominant) and multiple myxomas frequently occur in these patients (Haught et al, 1991). Primary malignant tumours include angiosarcomas, rhabdomyosarcoma, mesothelioma, fibrosarcoma and lymphoma.

This case highlights the difficulty that can occur in differentiating intracardiac masses in vivo, particularly between thrombi and tumours (vegetations differ to the other masses as they are closely related to the valves). This is crucial as the treatment of thrombi usually involves anticoagulation while the management of

CASE REPORT

A 71-year-old man was admitted with right lower lobe pneumonia, right-sided pleural effusion and congestive cardiac failure. He had been well until a left occipito-parietal embolic stroke (documented on computed tomography; CT) 2 years previously. Chronic atrial fibrillation and hypertension were noted and he was commenced on digoxin, warfarin and amlodipine. A transthoracic echocardiogram showed normal left ventricular contractility, mild mitral regurgitation and a severely dilated left atrium. He made a full neurological recovery.

In the intervening 2 years he suffered three transient ischaemic attacks, all in different cerebral territories. Two months before this admission, he suffered a haemorrhagic right basal ganglia stroke (documented on CT) with left hemiparesis. Carotid artery duplex imaging showed only minor stenoses. Warfarin was ceased and he was transferred to a nursing home, being dependent for all activities of daily living.

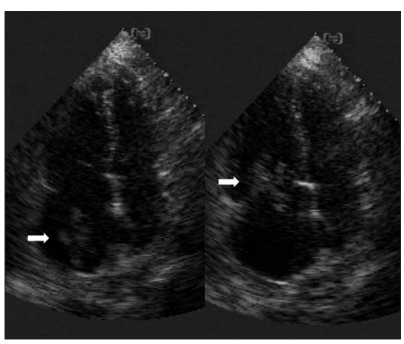
This admission he presented with a productive cough, dyspnoea and wheeze. Examination revealed right lower lobe consolidation with effusion, mild cardiac failure and new right leg weakness. Full blood count and biochemistry were normal except for leucocytosis (15.6x10^9/litre). The electrocardiogram did not show any evidence of ischaemia and the chest X-ray confirmed right lower lobe consolidation with effusion and pulmonary venous congestion. Blood and sputum cultures were negative for pathogens and a pleural aspirate revealed an exudate but no malignant cells. Intravenous frusemide, cefotaxime and metronidazole were started with moderate symptomatic improvement.

A transthoracic echocardiogram showed a lobulated right atrial mass with a pedicle attached to the septum (*Figure 1*). This mass prolapsed through the tricuspid valve on some beats and was thought most likely to be a tumour. Left ventricular contractility was moderately impaired and there was moderate tricuspid incompetence and moderate pulmonary hypertension. No intracardiac shunt was evident.

A cerebral CT showed a new left temporoparietal haemorrhagic infarct. The risks of surgical removal of the right atrial mass were considered too high and antiplatelet therapy with aspirin and dipyridamole was commenced. Three days later, he became acutely confused, hypoxic and dyspnoeic, and then unresponsive. Cardiopulmonary resuscitation was unsuccessful.

Post-mortem revealed a right atrial thrombus adherent to the interatrial septum (*Figure 2*) and scattered emboli in the medium-sized pulmonary arteries. There was no intracardiac shunt or left atrial thrombus. The cause of death was attributed to a combination of pulmonary embolus, aspiration pneumonia and congestive cardiac failure. The multi-territory strokes were postulated to arise from emboli from either left atrium thrombi (which had already embolized) or from atheromatous plaques in the aorta or carotids.

Figure 1. Right atrial mass (arrow) seen on transthoracic echocardiography during systole (left) and prolapsing into the right ventricle in diastole (right). An MPEG of this echocardiogram can be seen on www.hospitalmedicine.co.uk



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tumours usually involves a search for other masses and often resection.

Figure 2. Right atrial thrombus found at post-mortem.

Echocardiography is the current mainstay for diagnosis and differentiation of intracardiac masses, with the transoesophageal approach being more sensitive and specific in detecting atrial masses than the transthoracic approach (Alam et al, 1995). Myxomas usually present as a solitary mass, occur commonly in the left atrium with an identifiable stalk attached to the atrial septum and are rarely calcified (Lobo et al, 2000). Thrombi tend to have highly heterogenous distributions of pixel intensities rather than clumps of high or low pixel intensities. Sometimes, however, it is impossible to reliably distinguish the two. In a study by Gerber et al (2000) involving 20 experienced echocardiographers, images were correctly classified as thrombus or tumour in 71% of cases. Magnetic resonance and computed tomography imaging may further assist in assessing cardiac masses but have limited availability and lack portability. HM

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