Clinical implications of the morphological features of central pulmonary artery thromboemboli shown by transoesophageal echocardiography

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Abstract

Objectives—To illustrate the use of transoesophageal echocardiography in the detection of the morphological features of central pulmonary artery thromboemboli and their clinical implications.

Design—Review of five cases of central pulmonary artery thromboemboli detected by transoesophageal echocardiography.

Setting—University teaching hospital.

Patients—Five patients (three men and two women) admitted under general medical units.

Results—Central pulmonary artery thromboemboli were detected by the use of transoesophageal echocardiography in all the patients presented. Presentations were acute, subacute, or chronic. The morphological features of the thromboemboli on transoesophageal echocardiography were used to correlate with the time course of the illness, and to guide treatment. Two patients received thrombolytic treatment, one patient was treated with anticoagulation alone, and two patients had inferior vena caval filters implanted.

Conclusions—Transoesophageal echocardiography is an alternative diagnostic tool in the detection of central pulmonary artery thromboemboli. Morphological features of central pulmonary thromboemboli on echocardiography can provide useful information that may help to guide treatment.

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Central pulmonary artery thromboembolism is a life threatening but treatable condition. The presentation can be acute, subacute, or chronic. The decrease in the cross sectional lumen in the pulmonary artery leads to an abrupt rise in the pulmonary arterial pressure,¹ then right heart pressure overload and a drop in cardiac output. If the condition deteriorates, the patient can go into cardiogenic shock. If the patient survives the acute phase, the thrombus can either fragment and resolve through the endogenous fibrinolytic system, or it can be endothelialised in the central pulmonary artery. Chronic pulmonary hypertension and right heart failure can be the end result.² Therefore, prompt diagnosis and appropriate treatment are important in the acute phase and may influence long-term prognosis.

A number of investigations are currently available in the diagnosis of thromboemboli in the central pulmonary artery. Pulmonary angiography and nuclear ventilation-perfusion lung scan have been the most usual modes of imaging used.³ Echocardiography has recently been used in the diagnosis of pulmonary embolism. Direct visualisation of central pulmonary artery emboli with transthoracic echocardiography (TTE) has only been reported for a few cases.^{4,5} This is because the central pulmonary artery and its main branches cannot be imaged clearly from the transthoracic approach. Transoesophageal echocardiography (TOE), however, offers the unique opportunity to image clearly the main pulmonary artery, its bifurcation, and a long section of the right main pulmonary artery. There have been several case reports on the use of transoesophageal echocardiography in the detection of central pulmonary artery emboli.^{6,7}

We present five patients to show the use of TOE in direct visualisation of central pulmonary artery thromboemboli. We also studied the morphological features of the thromboemboli on echocardiography in an attempt to differentiate acute from chronic thromboemboli. The information obtained was taken into account to decide on treatment.

Case reports

CASE 1

A 68 year old man with documented rectal adenocarcinoma and pelvic metastasis was admitted with a sudden onset of severe dyspnoea. Physical examination showed respiratory distress and raised jugular venous pressure, but no focal signs in the chest. There was no clinical evidence of deep venous thrombosis. A chest x ray film was normal.

Transthoracic echocardiography showed mild right ventricular dilatation. No abnormalities were detected in the pulmonary artery. When TOE was performed a long, thin, mobile thromboembolus in the right pulmonary artery was found, the appearance of which was consistent with a cast from a deep calf vein (fig 1 (A)). A separate thromboembolus was seen in the right atrium (fig 1 (B)). Nuclear ventilation perfusion lung scans showed several mismatched defects consistent with pulmonary embolism.

Thrombus morphology on echocardiography suggested an acute thromboembolic event. The patient was treated systemically

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Figure 1 (A) Transoesophageal echocardiography showing type A thromboembolus (TH) in the right pulmonary artery (RPA). (B) Thromboembolus in the right atrium (RA). SVC, superior vena cava; ASC AO, ascending aorta; MPA, main pulmonary artery.

with streptokinase. This was followed by considerable clinical improvement. Repeat TOE performed three days later showed complete resolution of the pulmonary and the intracardiac emboli.

CASE 2

A 59 year old woman who sustained a traumatic fracture of her right fibula was treated with a plaster below the knee. Three days later, she presented to the hospital with an episode of collapse followed by left sided pleuritic chest pain, right calf pain, and dyspnoea. Physical examination showed tachypnoea and

a swollen right leg. Cardiac and chest examinations were unremarkable. Electrocardiography showed signs of right heart strain and arterial blood gases showed moderately severe hypoxaemia.

Transoesophageal echocardiography showed a long, thin, mobile thromboembolus in the right pulmonary artery. Peripheral pulmonary embolism was confirmed by the presence of bilateral mismatched defects on nuclear lung scans.

Clinical data and thrombus morphology on echocardiography suggested an acute embolic event. The patient was treated with intravenous streptokinase. Repeat TOE one week later showed complete resolution of the central thromboembolus. This coincided with resolution of the peripheral perfusion defects on nuclear lung scans.

CASE 3

A 61 year old man presented with a five week history of cough, dyspnoea, and pleuritic chest pains. Physical examination showed signs consistent with chronic obstructive airways disease. Cardiovascular examination showed a raised jugular venous pressure and a palpable right ventricular impulse. There was no clinical evidence of deep venous thrombosis. A chest x ray film showed a prominent right hilum which, on computed tomography, was shown to be caused by dilatation of the proximal right pulmonary artery.

Transthoracic echocardiography showed a dilated and hypertrophied right ventricle. The pulmonary artery was considerably dilated with the right pulmonary artery measuring 3.5 cm in diameter. Doppler echocardiography showed pulmonary hypertension. Transoesophageal echocardiography showed a thromboembolus in the right pulmonary artery (fig 2). It seemed to be immobile and was adherent to the anterior wall of the right pulmonary artery, extending from 1.5 cm distal to the bifurcation of the pulmonary artery to as far as could be seen. Thrombus morphology on echocardiography suggested an organised old thromboembolus or a new thrombosis that originated in the pulmonary artery. The patient was reluctant to undergo surgical pulmonary thromboendarterectomy and was therefore treated with a long-term anticoagulant.

CASE 4

A 69 year old man with known left upper lobe cavitation and mycetoma after a staphylococcal infection of a previous left upper lobe pulmonary infarct presented with recurrent haemoptysis and right heart failure. Electrocardiography showed right axis deviation, P pulmonale, and right bundle branch block. Nuclear lung scans showed a matched ventilation-perfusion defect in the left upper lobe consistent with a cavity and multiple small mismatched defects.

Transthoracic echocardiography showed dilated right heart chambers, right ventricular hypertrophy, paradoxical septal motion, and pulmonary hypertension. Transoesophageal

Figure 2 Type B thromboembolus adherent to the anterior wall of the right pulmonary artery. SVC, superior vena cava; ASC AO, ascending aorta; MPA, main pulmonary artery.

echocardiography showed a large thrombus extending from the bifurcation of the pulmonary artery into the right pulmonary artery as far as could be seen (fig 3). It completely occluded the dilated right pulmonary artery. Colour flow Doppler showed evidence of canalisation within the thrombus suggesting that it was old. Venography showed occlusion of the right external iliac vein. Because anticoagulation treatment was contraindicated and the patient was judged to be unfit for surgical pulmonary thromboendarterectomy, he was treated with implantation of an inferior vena caval filter.

Figure 3 Type B thromboembolus occluding the right pulmonary artery near the bifurcation of the main pulmonary artery. SVC, superior vena cava; ASC AO, ascending aorta; MPA, main pulmonary artery.

CASE 5

A 57 year old woman presented with a sudden onset of left hemiparesis and hemianaesthesia. She had no respiratory symptoms. Electrocardiography was normal. Computed tomography showed a cerebral infarct in the right lentiform nucleus extending into the adjacent posterior limb of the internal capsule and corona radiata.

Transoesophageal echocardiography was performed to exclude a cardiac source of cerebral embolism. It unexpectedly showed a mobile laminated thrombus in the pulmonary artery starting at the bifurcation, extending 6–7 cm into the right pulmonary artery. The pulmonary artery pressure was high. A patent foramen ovale with right to left shunting was also found. Nuclear ventilation-perfusion lung scans showed multiple mismatched defects consistent with pulmonary embolism.

Thrombus morphology on echocardiography implied recent embolisation. The discovery of the right to left shunt on echocardiography strongly suggested the possibility of paradoxical embolism as the cause of her cerebral event. She was therefore treated with implantation of an inferior vena caval filter, then long term anticoagulation.

Discussion

Detection of central pulmonary artery thromboemboli by TOE has previously been reported in a few cases. In the report of Wittlich *et al*, which compares TOE with other methods such as pulmonary angiography, computed tomography, and surgical findings in the detection of central pulmonary artery thromboemboli in patients with severe pulmonary embolism, TOE had a sensitivity of 96.7% and a specificity of 86%. Only 60% of patients with established severe pulmonary embolism had central pulmonary artery thromboemboli, however, in his series.⁸

Transoesophageal echocardiography offers some distinct advantages when used as a diagnostic tool if severe pulmonary embolism is suspected. It can be performed promptly in the emergency room or intensive care unit in critically ill patients with unstable haemodynamics without the need to interrupt treatment and transfer them to the radiology or nuclear medicine departments for angiography or lung scans. Transoesophageal echocardiography can provide a rapid definitive diagnosis of central pulmonary artery thromboemboli, therefore allowing the managing physician to make a decision on whether the patient should receive medical treatment such as a thrombolytic agent or emergency surgical embolectomy. It may also be performed intraoperatively. Deleuze *et al* reported a case in which pulmonary embolectomy was performed under intraoperative TOE guidance without cardiopulmonary bypass.⁹ As shown in cases 1 and 2, the response to thrombolytic treatment can be monitored by repeating the TOE.

Apart from diagnosing central pulmonary artery thromboembolism, TOE provides use-

ful information on pulmonary artery pressure, valvar pathology, and ventricular function. Other conditions with similar presentation such as pericardial tamponade and aortic dissection can be excluded. As shown in case 5, TOE can also detect the presence of occult intracardiac shunt. This finding had significant clinical implications in that patient.

Transoesophageal echocardiography has clear superiority over TTE in patients with suspected central pulmonary artery thromboembolism. Transthoracic echocardiography provides mainly indirect evidence such as pulmonary hypertension and signs of right ventricular pressure overload. Thromboemboli in transit in the right heart can sometimes be detected by TTE.¹⁰ Direct visualisation of central pulmonary artery thromboemboli is, however, a rare phenomenon.

The use of TOE in the diagnosis of central pulmonary artery thromboemboli has limitations. Only the extrapulmonary portion of the pulmonary arteries can be seen. Views of the left pulmonary artery in particular, are often limited to the first 2–3 cm. Therefore, absence of emboli on TOE does not necessarily exclude the diagnosis. If, however, no emboli are seen in the central portion of the pulmonary artery the patient is usually not critically compromised, so there is time for the diagnosis to be made by other modes of imaging.

The cases presented show the importance of differentiating various morphological types of central pulmonary artery thromboemboli as detected by echocardiography. Wittlich *et al*⁸ described two types. Type A thrombi are long, thin, mobile structures. They move independently of the pulmonary artery as can be shown by M mode imaging. They represent emboli that originate from a deep venous thrombosis. As shown in cases 1 and 2, their presence seems to imply recent, acute embolic events. Aggressive treatment is often necessary. By contrast type B thrombi are immobile structures, often firmly adherent to the arterial wall. They represent organised embolic material that has been in the pulmonary artery for some time. They might even have originally developed in the pulmonary artery as a result of chronic pulmonary hypertension. As shown in case 4, partial recanalisation of the embolus can occur with blood flowing through these channels as detected by colour flow Doppler. This feature can be used to differentiate between a chronic thromboembolus and a very large acute embolus occluding the pulmonary artery. Therefore, thrombus morphology on echocardiography can be used to assess the time course of the embolic event and has important clinical implications in the decision about treatment.

The most appropriate treatment for massive pulmonary embolism caused by type A embolus remains unclear from the reported evidence. Thrombolytic treatment is supported by some reports. Vestraete *et al* reported a 6% mortality in patients with acute massive pulmonary embolism treated with recombinant tissue type plasminogen activa-

tor.¹¹ The time, however, to thrombolytic treatment was from 27 to 31 hours. Knowing that the mortality from massive pulmonary embolism reported by Mattox *et al* was 50% within 30 minutes, 70% within one hour, and 85% within six hours,¹² such a delay in treatment may have selected the subgroup of patients most likely to survive regardless of the form of treatment given.

In patients with massive pulmonary embolism and severe haemodynamic compromise who are too ill to be given thrombolytic treatment, or when thrombolysis is either contraindicated or too slow in producing benefit, surgical pulmonary embolectomy remains an effective alternative option, especially in patients with no coexisting medical conditions that raise the surgical risk. The surgical mortality has been reported as ranging from 24% to 64%.^{12,13} With advances in cardiopulmonary support and techniques in extracorporeal circulation, and the improvement in the speed of reaching a definitive diagnosis before the patient's condition deteriorates, the surgical mortality is likely to approach the 24% figure in the future.

Patients with chronic pulmonary hypertension due to unresolved pulmonary embolism of type B morphology are usually refractory to anticoagulants and thrombolytic treatment. Type B thrombi can sometimes be managed with pulmonary thromboendarterectomy. Moser *et al* reported 42 cases with a surgical mortality of 16.6%. Surgical success leads to a dramatic reduction of symptoms, with sustained reduction in pulmonary hypertension, reduction in the size of the pulmonary artery, right ventricle, right atrium, and inferior vena cava, with a normalisation of the interventricular septal position.

In conclusion, TOE is an alternative diagnostic tool in the detection of central pulmonary artery thromboemboli in patients with suspected severe pulmonary embolism. It has some distinct advantages over other modes of investigation. It is ideal clinically where a rapid diagnosis is vital in a critically ill patient who is unfit to be transferred elsewhere to undergo invasive angiography or lung scans. It offers vital information on ventricular function, the degree of pulmonary hypertension, and the presence of any relevant structural cardiac abnormalities. The echocardiographic morphological features of the thromboemboli provide additional information that can be used to assess the time course of the embolic event and help to guide treatment. Therefore, TOE should be considered as an imaging option in this clinical setting and should be included in the physician's armamentarium.

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1 McIntyre KM, Sasahara AA. The haemodynamic response to pulmonary embolism in patients without prior cardiopulmonary disease. *Am J Cardiol* 1974;28:288–94.

2 Rich S, Levitsky S, Brundage BH. Pulmonary hypertension from chronic pulmonary thromboembolism. *Ann Intern Med* 1988;108:425–34.

- 3 The PIOPED Investigators. Value of ventilation/perfusion scan in acute pulmonary embolism: results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). *JAMA* 1990;263:2753-9.
- 4 Kasper W, Giebel A, Tiede N, Hoffmann T, Memertz T, Just H. Echocardiographic diagnosis of pulmonary embolism. *Herz* 1989;63:649-80.
- 5 Weston MJ, Wilde P. Echocardiographic diagnosis of massive pulmonary embolism. *Br J Radiol* 1989;62:751-3.
- 6 Nixdorff U, Erbel R, Drexler M, Meyer J. Detection of thromboembolus of the right pulmonary artery by transesophageal two dimensional echocardiography. *Am J Cardiol* 1988;61:448-9.
- 7 Klein SL, Steward WL, Cosgrove DM, Mick MJ, Salcedo E. Visualisation of acute pulmonary emboli by transesophageal echocardiography. *J Am Soc Echocardiogr* 1990;3:412-5.
- 8 Wittlich N, Erbel R, Eichler A, Schuster S, Jakab H, Iversen S, *et al.* Detection of central pulmonary artery thromboembolism by transesophageal echocardiography in patients with severe pulmonary embolism. *J Am Soc Echocardiogr* 1992;5:515-24.
- 9 Deleuze P, Saada M, De Paulis R, Brochard L, Mazzucotelli JP, Rotman N, *et al.* Intraoperative transesophageal echocardiography for pulmonary embolectomy without cardiopulmonary bypass. *Ann Thorac Surg* 1991;52:137-8.
- 10 The European Working Group on Echocardiography. The European cooperative study on the clinical significance of right heart emboli. *Eur Heart J* 1989;10:1046-59.
- 11 Verstraete M, Miller GSH, Bounameaux H, Charbonnier B, Colle JP, Lecorff G, *et al.* Intravenous and intrapulmonary recombinant tissue-type plasminogen activator in the treatment of acute massive pulmonary embolism. *Circulation* 1988;77:353-60.
- 12 Mattox KL, Feldtman RW, Beall SL, DeBakey ME. Pulmonary embolectomy for acute massive pulmonary embolism. *Ann Surg* 1981;195:726-31.
- 13 Bottzann T, Vejlsted H, Albrechtsen O. Pulmonary embolectomy using extracorporeal circulation. *Thorac Cardiovasc Surg* 1981;29:320-2.
- 14 Moser KM, Daily PO, Peterson K, Dembitsky W, Vapnek J, Shure D, *et al.* Thromboendarterectomy for chronic, major-vessel thromboembolic pulmonary hypertension. Immediate and long-term results in 42 patients. *Ann Intern Med* 1987;107:560-5.