

Case report

Management and clinical outcome of concomitant pulmonary embolism and paradoxical saddle aortic arch embolism

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SUMMARY

A 65-year-old man presented in a peri-arrest situation after collapse, he was found hypoxic with ischaemic arms. CT imaging showed massive bilateral pulmonary embolisms (PEs) and an aortic arch embolus extending from brachiocephalic trunk to left subclavian artery. Following intravenous thrombolysis, repeat imaging revealed that the aortic embolus had migrated distally into both axillary arteries and had occluded the right carotid from origin to skull base. Bilateral upper limb embolectomies were carried out from the brachial arteries together with forearm fasciotomies. Left hemianopia related to a right middle cerebral artery territory infarct was managed conservatively; forearm fasciotomy wounds were primarily closed and the patient was discharged on lifelong anticoagulation. A transoesophageal echocardiogram revealed a patent foramen ovale. This case demonstrates a very unusual presentation of concomitant PE and paradoxical saddle aortic arch embolism. A multidisciplinary approach has resulted in an excellent clinical outcome for this complex patient.

BACKGROUND

Concomitant thoracic aortic embolism and pulmonary embolism (PE) are rare conditions constituting only a few reports in the international literature, the majority of which are referred to the cardiothoracic surgeon for aortic and pulmonary thromboembolectomy under cardiopulmonary bypass with cardiocirculatory arrest.^{1 2} Presentation of these cases is a consequence of the symptoms of either pulmonary emboli or peripheral embolic episodes, and the management is dependent on the source of embolus, the site of embolus lodgement and the symptoms and condition of the patient.

Embolic events, in most cases of aortic thromboembolism, are considered to have cardiac origins, specifically from complications of atrial fibrillation and myocardial infarction.³ A minority of cases are related to paradoxical embolism from venous thrombosis and cardiac shunting.⁴ Aortic thrombosis can also be due to underlying atherosclerotic disease with superimposed acute thrombus or hypercoagulability states.⁵⁻⁷ Free floating thrombus in a diseased thoracic aorta complicated by thromboembolic episodes may be treated surgically by open thrombectomy, endovascular

techniques or non-operatively with anticoagulation or thrombolysis.⁸⁻¹¹

We report the management and outcome of a patient referred to a tertiary vascular centre presenting with simultaneous massive bilateral PE and aortic arch embolus resulting in bilateral upper limb ischaemia and cerebrovascular event.

We are not aware of any other reported cases of de novo PE and aortic arch embolism.

CASE PRESENTATION

A 65-year-old male patient was referred to our vascular service 24 hours after he presented to a spoke hospital in a peri-arrest situation after collapsing at home. On arrival in the emergency department, he was in cardiogenic shock with low saturations and signs of bilateral upper limb ischaemia.

No history of prodromal symptoms of chest pain, breathlessness or arm pain were pointed out by family when patient was admitted in accident and emergency department. He was resuscitated and a CT pulmonary angiogram was carried out. This demonstrated bilateral PEs and an aortic arch saddle embolus extending into the origin of the brachiocephalic trunk, across the right and left common carotid artery origins and into the left subclavian artery (figure 1). Only after full resuscitation, the patient started complaining of arm pain, tingling and coldness.

The patient had a medical history of mild hypertension, asthma, obesity, spinal surgery and neuropathic bilateral leg pain for which a spinal cord stimulator was implanted 2 months prior to this presentation. There was no history of thrombophilia, malignancy, recent travel, illicit drug or steroid usage.

He was managed in the intensive care unit in the spoke hospital with intravenous thrombolysis (100 mg alteplase) followed by an intravenous heparin infusion. An invasive ventilatory or circulatory support was not required at any point.

Although the patient's arm symptoms improved immediately following thrombolysis, this was not sustained and he was transferred to the vascular hub for revascularisation.

On arrival he was haemodynamically stable, self-ventilating, alert and orientated. His symptoms were bilateral arm pain with weakness and



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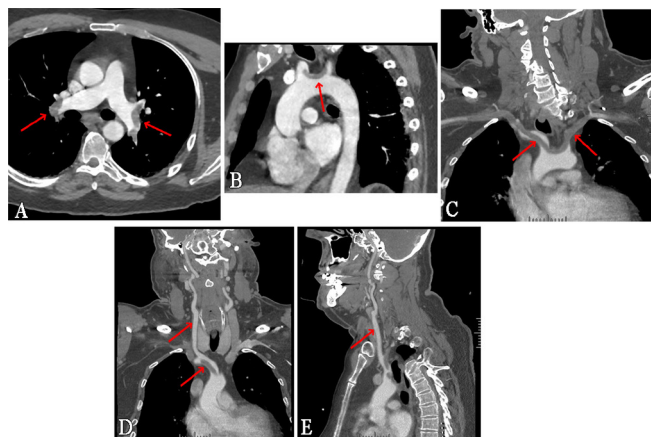


Figure 1 CT pulmonary angiogram on presentation, prethrombolysis. This CT scan was performed at the spoke hospital on arrival into the emergency department to rule out pulmonary embolisms which represented the most likely diagnosis. (A) Bilateral pulmonary embolisms. (B) Saddle embolus sitting in the aortic arch. (C) Saddle embolus involving both subclavian artery and aortic arch. (D) Involvement of brachiocephalic trunk and full patency of carotid arteries. (E) Patent right carotid artery.

numbness of the hands and fingers. On examination, there were no palpable upper limb pulses and doppler signals were damped monophasic.

INVESTIGATIONS

Repeated haematology and biochemistry, on arrival at our tertiary centre, shown in [table 1](#).

A full thrombophilia screen was carried out, including paroxysmal nocturnal haemoglobinuria screen, JAK2 mutation, lupus, protein S and C activity, cardiolipin antibodies, immunoglobulin dosing, activated protein C sensitivity ratio and electrophoresis. No abnormalities were detected. Tumour markers (CA 125, CEA, PSA) were also negative.

As part of preoperative planning, a repeated *CT angiography* (head to toes) was performed to confirm findings highlighted at the spoke hospital and to rule out undiagnosed malignancy as part of thrombophilia full workup.

Table 1 Full blood count and biochemical results on arrival at tertiary centre

White cell count	11.2×10 ⁹ /L
Red blood cell	4.60×10 ¹² /L
Haemoglobin	133 g/L
Platelet count	199×10 ⁹ /L
Sodium	138 mmol/L
Potassium	4.1 mmol/L
Calcium	2.17 mmol/L
Adjusted calcium	2.35 mmol/L
Total CK	120 U/L
Troponin I	201 ng/L
Urea	7.2 mmol/L
Creatinine	92 µmol/L
Alkaline phosphatase	108 U/L
Albumin	36 g/L
Alanine transaminase	33 U/L
Bilirubin	8 U/L

Bilateral large PEs were confirmed. The thrombus previously seen in the aortic arch moved distally. There was then an occluded right common carotid artery with thrombus extending to the skull base (complete circle of Willis with no thrombus seen within the circle of Willis). Right axillary artery was occluded. A tongue of thrombus was extending into the left vertebral artery from an occluded left axillary artery. A non-occlusive thrombus was seen in the distal external carotid artery extending into the maxillary artery.

No cardiac shunt was identified and no features of right ventricle (RV) strain were reported. Chest was clear with no enlarged thoracic or axillary lymph nodes. No filling defects within the abdominal or pelvic veins to suggest deep vein thrombosis (DVT); however, the left external iliac vein was enlarged with adjacent stranding concerning for thrombus here. Except for renal cysts there was normal appearance of the solid abdominal organs with no destructive bone lesions ([figure 2](#)).

A preoperative bedside transthoracic echocardiogram (TTE) demonstrated mildly reduced left ventricular function with significant RV strain. There was no evidence of intracardiac shunting or a patent foramen ovale (PFO).

The ECG showed a normal sinus rhythm with no signs of ischaemia or RV strain.

TREATMENT

Bilateral embolectomies were carried out from the brachial arteries, with bilateral forearm fasciotomies under general anaesthesia. A large volume of thrombus was removed from each side and distal pulses were restored bilaterally. Postoperatively, he was maintained on an intravenous heparin infusion.

On postoperative day 6, he developed transient visual disturbances with left hemianopia. CT brain showed an acute right middle cerebral artery territory infarct mainly involving the branches within the right parieto-occipital regions together with a small perforator infarct within the right internal capsule and caudate head ([figure 3](#)). This is in keeping with embolic infarcts secondary to large thrombus load within the right carotid artery.

Carotid duplex showed near occlusion of the right common, internal and external carotid arteries. The thrombus in the right internal carotid artery extended 1.4 cm distal to the carotid bifurcation with some evidence of recanalisation. The left carotid

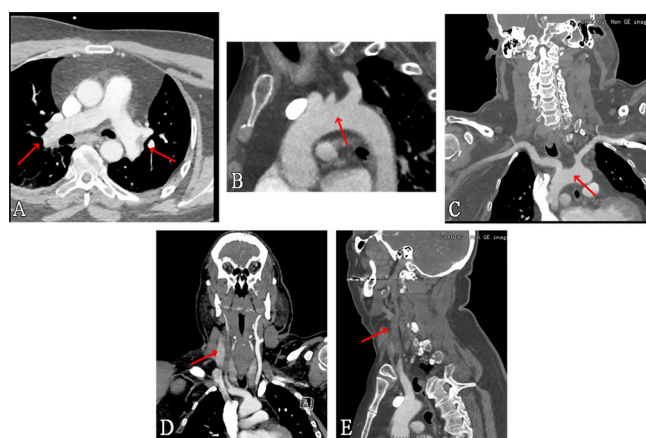


Figure 2 Post-thrombolysis CT angiogram. A repeated CT scan was performed in order to review the evolution of the embolic event and plan surgical treatment. (A) Resolving pulmonary embolisms. (B) Aortic arch free of embolus. (C) Proximal subclavian arteries and aortic arch free of embolus. (D) Embolic occlusion of the right carotid artery. (E) Embolic occlusion of the right carotid artery.

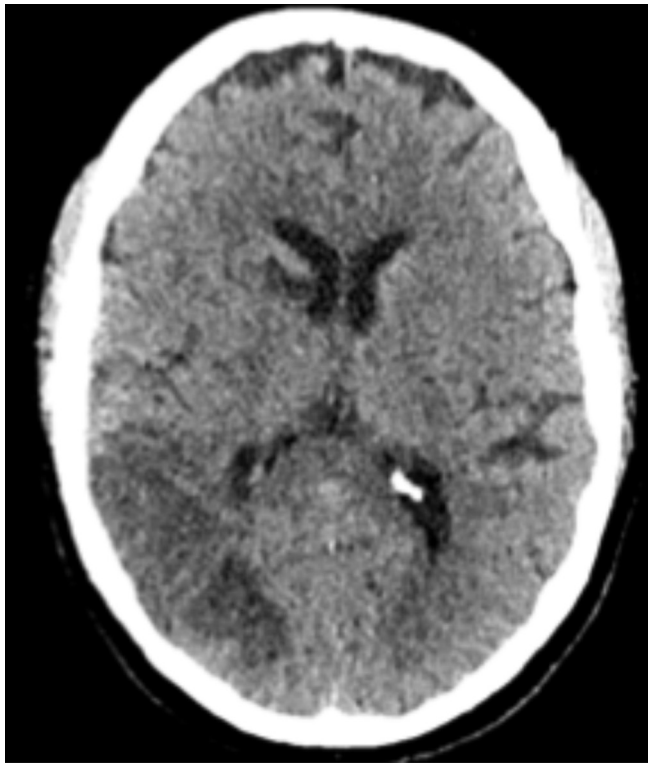


Figure 3 CT head showing right middle cerebral artery (MCA) embolic infarct.

system was normal without stenosis and both vertebral arteries had normal antegrade flow. It was decided that conservative management rather than carotid endarterectomy or embolectomy was appropriate.

A transoesophageal echocardiogram (TOE) with air contrast was used to detect the presence of cardiac shunting and a small PFO was demonstrated.

On postoperative day 15, the arm fasciotomies were closed under local anaesthesia. At this point, the intravenous heparin infusion was switched to oral anticoagulation with warfarin and international normalised ratio (INR) target between 2 and 3. This will be required lifelong.

OUTCOME AND FOLLOW-UP

The patient was discharged home fully independent with all activities of daily living. Total length of hospital stay was 26 days. Follow-up with cardiology has been arranged to consider elective closure of the PFO, as well as with neurology and vascular surgery teams.

We reviewed the patient in our planned outpatient clinic at 2 months following discharge. Embolectomy and fasciotomy wounds were healing with no sign of complications. Neurological signs affecting the visual field were still present with some degree of improvement.

DISCUSSION

This case demonstrates a very unusual presentation of concomitant PE and paradoxical saddle aortic arch embolism resulting in bilateral arm ischaemia that required embolectomies to revascularise the upper limbs and fasciotomies of the forearms. The patient also sustained a cerebrovascular event, which was managed conservatively.

It is apparent from repeated imaging that the treatment with intravenous thrombolysis, initiated due to haemodynamic instability in the setting of PE, also served to alter the morphology of the arch embolus. While this seemed to improve upper limb symptomatology initially, this was not sufficient to avoid revascularisation surgery. However, this procedure was considerably simplified by the embolus moving distally out of the aortic arch into the axillary arteries thus allowing for embolectomies to be carried out from bilateral brachial approaches with minimal risk to both carotid artery origins (the right being occluded already with no neurological symptoms at that time).

The early identification of the need for forearm fasciotomies, closed at an appropriate time under local anaesthetic, resulted in a full restoration of upper limb function and minimal morbidity.

Paradoxical aortic emboli, as represented in this case, do not require surgical repair of the aorta, contrary to primary thrombotic events of the thoracic aorta which should be considered for surgical intervention.^{8 12} Acute peripheral emboli can be treated when clinically indicated. The associated presence of PE and the absence of pre-existing arterial disease made the diagnosis of paradoxical emboli in this case the most likely one.

We note that the original echocardiogram carried out in this patient did not show a PFO and this was a TTE. In a setting where there is genuine concern for paradoxical emboli, a TOE with air contrast is the most reliable diagnostic tool and is recommended.¹ These findings do however raise the question as to whether, in the setting of massive PE, the increased right heart pressure has opened up a PFO rather than this being the underlying pathology. We feel this is unlikely due to the simultaneous presentation of collapse, related to the PE, and arm pain associated with the arterial embolus.

A multidisciplinary approach over a hub and spoke network has facilitated the excellent recovery of a patient with a complex and rare condition.

Learning points

- ▶ Intravenous thrombolysis is the key treatment in unstable patients presenting with massive pulmonary embolism. In the context of paradoxical embolism, it also helps to stop the progression of the embolic event.
- ▶ Between initial medical management (thrombolysis and intravenous heparin) and surgical treatment, repeated imaging is mandatory to refine operative planning.
- ▶ It is important to differentiate between thrombotic and embolic events of the aorta.
- ▶ Embolic events do not require surgical repair of the aorta in contrary to primary thrombotic events which should be considered for surgical intervention with excision of the affected aortic segment.
- ▶ The early identification of the need for forearm fasciotomies helps in achieving full restoration of upper limb function with minimal morbidity.
- ▶ Transoesophageal echocardiogram is more sensitive and better able to characterise abnormalities of the inter-atrial septum such as patent foramen ovals and atrial septal defects (ASDs) and should be used when there is a high degree of suspicion following negative transthoracic echocardiogram (as in this case).

Contributors DRH is the main supervisor of this article, being in charge of contents and objectives. EM gave the substantial contributions in writing, designing the paper and analysis of the available data. APW, consultant radiologist, provided imaging and reviewed related drafts. JRB critically revised the work for important intellectual

content. Final approval of the published was shared by every author after individual revision, in agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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