CASE REPORT

Cardiac arrest following arteriovenous fistula manipulation: a cautionary note

Miles Gandolfi, Bimbi S Fernando

Royal Free London NHS
Foundation Trust, London, UK

Correspondence to
Dr Miles Gandolfi,
miles.gandolfi@nhs.net

Accepted 23 May 2017

SUMMARY

Arteriovenous fistulas can lead to a number of different chronic complications. We describe a case where a patient developed a thrombosis within her brachio basilic arteriovenous fistula, which was manually manipulated in order to restore fistula flow. This resulted in a pulseless electrical activity cardiac arrest within a few minutes. After ten minutes of chest compressions and intubation, there was return of spontaneous circulation. No epinephrine was given nor shocks administered. Patient was extubated within minutes and was alert, orientated and haemodynamically stable. CT pulmonary angiogram showed extensive bilateral pulmonary emboli. Manual manipulation of the arteriovenous fistula lead to significant amounts of thrombus embolising to the pulmonary arteries, and resultant cardiac arrest due to circulatory compromise. Chest compressions likely dislodged these emboli, allowing circulation to recommence. We publish this as a cautionary note of a rare but potentially fatal complication.

BACKGROUND

Arteriovenous fistulas can lead to a number of chronic complications such as stenosis, thrombosis, lymphoedema, aneurysm and infection.¹ It is important that clinicians who deal with these on a regular basis are aware of potential complications, and the risks that these pose. This case highlights a rare, life-threatening, potentially avoidable complication, which we cannot find mentioned in case reports elsewhere, and the immediate management of this complication.

CASE PRESENTATION

A 71-year-old female presented for her regular haemodialysis, and it was found that her left brachio basilic arteriovenous fistula had no bruit or thrill. She had successfully had dialysis 3 days previously. She was sent to a walk-in clinic the same day and seen by a consultant transplant surgeon.

She had a history of thrombus in the same fistula the previous year, which had been managed by fistuloplasty, and by increasing her regime of regular tinzaparin 750 units three times a week to 1250 units three times a week. The fistula had been imaged with ultrasound the previous month, showing a good flow of 950 mL/min within the fistula, and a small amount of hyperplasia in the basilic vein just above the anastomosis causing increased flow velocities (>4 m/s). The patient had required dialysis for four and a half years due to

end-stage renal failure, and also had a background of insulin-dependent diabetes mellitus, hypertension, atrial fibrillation and multiple pulmonary embolisms (PEs). She had been fitted with an inferior vena cava filter and was on regular low molecular weight heparin.

The fistula was manually manipulated with restoration of the flow, and the patient immediately stated that she felt unwell and faint. After approximately 2 min, she lost consciousness, had a weak thready pulse and a cardiac arrest team was called to the clinic area.

The patient lost cardiac output and chest compressions were started. The rhythm strip showed pulseless electrical activity (PEA), but there were difficulties in getting vascular access, which meant no epinephrine or other intravenous medications were given within the first 10 min. The patient was intubated and remained in PEA for 10 min, after which she had return of spontaneous circulation, with a heart rate of 160 beats per minute and a blood pressure of 140/80. By this time a cannula had been inserted, and the arterial blood gas which had been performed during chest compressions returned. This showed her potassium was 7.5 mmol/L.

The patient was started on treatment for her hyperkalaemia and transferred to the emergency department where she started pulling at the endotracheal tube and making respiratory effort. She was haemodynamically stable, so was extubated and was fully alert following this. Repeat potassium after treatment was 4.6 mmol/L.

CT pulmonary angiogram (CTPA) was performed which showed extensive PEs.

Patient was started on a heparin infusion and a permacath line was inserted the next day to enable haemodialysis, as the fistula was still not fully functional. This fistula was not re-imaged during her inpatient stay as it was clinically evident to be non-functioning. She remained in hospital for 4 days, and was then discharged home on daily low molecular weight heparin injections.

INVESTIGATIONS

Arterial blood gas during the arrest showed a potassium of 7.5 mmol/L, a lactate of 8 mmol/L, a pH of 7.08 and a base excess of -16 mmol/L. The pCO₂ was 5.8 kPa and the pO₂ was 23 kPa on 100% oxygen.

ECG following the arrest showed p wave pulmonale, right bundle branch block and tachycardia, with no changes consistent with hyperkalaemia (figure 1). ECG 8 hours following the arrest for diffuse chest pain showed that these changes were resolving (figure 2).

CrossMark

To cite: Gandolfi M, Fernando BS. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-220179

Figure 1 ECG immediately following return of spontaneous circulation.

CTPA was performed which showed extensive bilateral pulmonary emboli extending from the main pulmonary arteries into all lobes of the lung. There was an occlusive thrombus in the right middle lobe pulmonary artery. There was evidence of right heart dysfunction with dilated right atrium and ventricle, and the main pulmonary trunk dilated to 3.2 cm (figure 3).

DIFFERENTIAL DIAGNOSIS

Initially when the patient went into cardiac arrest, the feeling was that this could either be due to pulmonary embolus or hyperkalaemia.

The arterial blood gas potassium of 7.5 mmol/L was concerning, but as a patient with chronic renal failure this was not dissimilar to previous blood results.

Figure 2 ECG 8 hours following return of spontaneous circulation.

Figures 3 (A, B, C, D) CT pulmonary angiogram slices showing extensive bilateral pulmonary emboli.

The fact that chest compressions alone bought about return of spontaneous circulation was more suggestive of a mechanical obstruction (PE) which had been mechanically shifted than an electrolyte disturbance, which we were initially unable to treat due to lack of vascular access.

The ECG showed no hyperkalaemic changes, but showed a tachycardia with p wave pulmonale and right bundle branch block, suggesting a PE was a more likely cause of the arrest. CTPA scan confirmed that PEs were present. Given this patient's history of recurrent PE, it is important not to jump to assuming that PE findings on CTPA will be the cause of the arrest, but given the history leading up to the arrest, and the right heart strain found, this is highly likely.

TREATMENT

- ▶ Cardiopulmonary resuscitation
- ▶ Briefly intubated
- ▶ Calcium chloride 10 mL 10% intravenous, 10 units insulin in 50 mL 50% dextrose for hyperkalaemia
- ▶ Heparin infusion for PE, later converted to low molecular weight heparin for discharge. This was chosen over tinzaparin or a NOAC as the patient was on dialysis, and as the patient was haemodynamically stable after resuscitation, immediate thrombolysis was decided against after weighing up risks and potential benefits.

OUTCOME AND FOLLOW-UP

Patient was fully conscious and haemodynamically stable an hour following her cardiac arrest. She was admitted to hospital for 4 days in order to organise her haemodialysis, given that her fistula was not working, and organise her anticoagulation. She was discharged home on low molecular weight heparin anticoagulation.

DISCUSSION

Given the fact that the embolisation of clot and subsequent haemodynamic compromise appears to be a rare occurrence, we speculate that this occurred in this patient due to the clot

being freshly formed, and hence easily dislodged. We also speculate that the subsequent arrest was likely related to the background frailty of the patient.

In addition to highlighting the risks of manual manipulation of an arteriovenous fistula, this case also shows successful cardiopulmonary resuscitation in a patient with PEA without intravenous epinephrine (currently under investigation in the PARAMEDIC2 trial). We propose that this success was likely due to mechanically shifting the embolus within the pulmonary arteries. The case also highlights the importance of comparing hyperkalaemia (or other investigation findings) with a patient's previous results. Patients with end-stage renal failure often tolerate much higher serum potassium levels.

On literature review for similar cases, no case report was found where manual manipulation of arteriovenous fistula lead to PE. One report covered the thrombectomy of a thrombosed arteriovenous fistula and resultant PE, and cautioned that venous emboli may result from fistula manipulation.² Another two reports cover cases of massive PE resulting from using angioplasty and percutaneous thrombolysis to attempt to declot arteriovenous fistulas.^{3 4} These reports show that removing the thrombus from an arteriovenous fistula is not without risk, regardless of the setting.

Contributors Both MG and BSF were involved in producing this paper by making substantial contributions to the conception or design of the work. Drafting the work and revising it critically for important intellectual content, giving final approval of the

Learning points

- ▶ Thrombus can form in an arteriovenous fistula without warning or obvious cause, despite anticoagulation.
- ▶ Manual or percutaneous manipulation of an arteriovenous fistula can lead to embolic disease, which can have life-threatening consequences.
- ▶ Just good quality chest compressions and airway management can lead to return of spontaneous circulation.
- ▶ Importance of comparing investigation results with patient baseline to formulate diagnosis and management plan.

version to be published. They are 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.

Competing interests None declared.

Patient consent Consent obtained from next of kin.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2017. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- 1 Stolic R. Most important chronic complications of arteriovenous fistulas for hemodialysis. *Med Princ Pract* 2013;22:220–8.
- 2 Grebenyuk LA, Marcus RJ, Nahum E, *et al.* Pulmonary embolism following successful thrombectomy of an arteriovenous dialysis fistula. *J Vasc Access* 2009;10:59–61.
- 3 Sadjadi SA, Sharif-Hassanabadi M. Fatal pulmonary embolism after hemodialysis vascular access declotting. *Am J Case Rep* 2014;15:172–5.
- 4 Shah A, Ansari N, Hamadeh Z. Cardiac arrest secondary to bilateral pulmonary emboli following arteriovenous fistula thrombectomy: a case report with review of the literature. *Case Rep Nephrol* 2012;2012:1–6.

Copyright 2017 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow