Pulmonary edema in the neuroradiology suite: a diagnostic dilemma

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Purpose: To present the case of an initially unexplained complication of sudden pulmonary edema in a patient during stenting of the carotid artery in the interventional neurology suite.

Clinical Features: A 46-yr-old woman (ASA III) having an intracavernous carotid artery angioplasty and stenting procedure under neurolept anesthesia developed sudden pulmonary edema on completion of an otherwise uneventful stenting procedure. The patient responded well to initial management of pulmonary edema although the cause of the edema remained unclear. On retrospective examination of data and with the evolution of clinical signs it became apparent that the patient had suffered a subarachnoid hemorrhage which eventually resulted in her death.

Conclusions: Clinical signs of intracerebral events may be slow to evolve. The cause of sudden pulmonary edema in patients undergoing an interventional neuroendovascular procedure is likely to be neurogenic in origin despite the initial lack of neurological signs.

Objectif : Présenter le cas d'une complication, d'abord inexpiquée, d'œdème pulmonaire pendant la pose d'un tuteur à l'artère carotide.

Éléments cliniques : Une femme de 46 ans (ASA III) admise pour une angioplastie du siphon carotidien et la pose d'un tuteur sous neuroleptanesthésie, a développé un œdème pulmonaire soudain à la fin de la pose du tuteur, par ailleurs sans incident. La patiente a bien réagi au traitement initial de l'œdème pulmonaire même si la cause en est restée obscure. À la suite d'un examen rétrospectif des données et de l'évolution des signes cliniques, il est devenu évident que la patiente a subi une hémorragie sous-arachnoïdienne qui a finalement causé sa mort.

Conclusion : Les signes cliniques d'événements intracérébraux peuvent présenter une lente évolution. La cause d'un œdème pulmonaire soudain chez des patients qui subissent une intervention neuroendovasculaire peut être d'origine neurogène malgré l'absence initiale de signes neurologiques.

THERE have been many reports of sudden pulmonary edema secondary to cardiac failure, intracerebral¹ events and obstruction of the airway.^{2,3} We report a case where a patient undergoing carotid artery stenting as a neuro-radiological procedure developed sudden pulmonary edema, the cause of which became apparent only after retrospective review of clinical data.

Case report

A 46-yr-old, poorly compliant insulin dependent, diabetic, woman (ASA III) presented for stenting of the intracavernous portion of the right carotid artery as an interventional neuroradiological procedure. Three years earlier the patient had a left sided cerebrovascular accident (CVA) resulting in right upper limb weakness which resolved with rehabilitation therapy. The following year she had multiple mini-strokes manifested by weakness and tingling of the left arm. This progressed to a right sided CVA resulting in profound left upper limb weakness. Doppler examination at this time showed a 90% stenosis of the left common carotid artery (CCA) but no evidence of stenosis in the right CCA.

The patient had a 15 yr history of diabetes. This was initially managed with oral hypoglycemics but more recently, in the last eight months, she had needed treatment with insulin. She was noted to have signs of diabetic retinopathy and peripheral neuropathy. Other medical problems included arthritis for which she had uneventful knee surgery six years previously. There was no clinical evidence of cardiac disease, hypertension or respiratory disease although she did have a history of shortness of breath with minimal exertion. Preoperative examination showed an alert and orientated but overweight woman (Wt.90 kg, Ht 157 cm, BMI 36 kg·m⁻²) with some weakness of the left upper limb but normal power to the leg and right side; full visual fields; no focal cranial nerve deficits; loud bruit over the left and a faint bruit over the right carotid arteries. Auscultation of her chest revealed no signs of failure. Her blood pressure was 110/60 mmHg. In addition to her obese stature, she was noted to have slight limitation to mouth opening, and a thyromental distance of 6 cm, indicating a potentially difficult intubation. Chest X-ray was unremarkable. Her ECG showed a sinus tachycardia of 108 beat·min⁻¹ with a RBBB and prolonged QT interval which was unchanged from previous admissions. A recent random blood glucose was 18.2 mmol·L⁻¹ and an accucheck on the morning of surgery was 15.9 mmol·L⁻¹. Electrolyte concentrations were within normal range. The patient had been given her usual dose of insulin on the morning of surgery and an

intravenous glucose infusion was being administered.

The patient was booked for an angiogram and stenting of the intracavernous portion of the right carotid artery in the interventional radiology suite with neurolept anesthesia. In keeping with routine practice for our institution, oxygen was administered by facemask at five L·min⁻¹ throughout the procedure. She was monitored with a Lead II electrocardiogram (ECG), pulse oximeter and non invasive blood pressure monitor. Spontaneous respiratory rate and end tidal CO₂ were monitored via the face mask. She received a total dose of 2 mg midazolam and 150 µg fentanyl over the one hour 45 min procedure. Anticoagulation was with 8000 units *iv* heparin at the start of the procedure and a further 1000 units one hour later. Local anesthetic was infiltrated into the right groin region by the neuroradiologist and the right femoral artery was catheterized for the angiogram and stenting.

The procedure was uneventful and the patient remained alert and neurologically unchanged until soon after completion of stenting when she was noted by the radiologists to be agitated. She became slightly hypertensive with an increase in blood pressure from 140/80 to 170/98 mmHg. Her heart rate increased from 100 to 110 beat·min⁻¹. Small incremental boluses of propofol were given to control her agitation over five minutes. Repeated questioning and examination revealed no change in neurological status and the patient did not complain of any headache or chest pain but was dyspneic. There was concern that the patient had airway obstruction although the capnography tracing did not change. She then began to cough, producing clear frothy sputum and her O₂ saturation decreased to 75%. An X-ray of her thorax confirmed the diagnosis of fulminant pulmonary edema. The cause of the pulmonary edema was considered to be related to myocardial ischemia, pulmonary embolus or even airway obstruction. An arterial blood gas sample showed PaO₂ of 48 mmHg. She responded to a change to the sitting position, additional O₂ via facemask and 40 mg furosemide and 5 mg morphine *iv*. After the pulse oximeter reading increased to 95%, and the patient seemed more comfortable clinically, an end of procedure angiogram was completed and assessed as satisfactory by the neuroradiologist. Heparin was not reversed. The femoral catheter was left *in situ* with instructions for its removal at six hours after the last dose of heparin.

The patient was transferred to the intensive care unit (ICU). She remained in the sitting position breathing oxygen 50% via facemask and fully monitored for O₂ saturation, non invasive blood pressure and ECG. A

portable chest X-ray confirmed pulmonary edema. Throughout this period the patient remained neurologically intact with a Glasgow coma scale of 15, orientated and conversing despite her shortness of breath. After this initial improvement clinically, the patient's oxygen saturation readings decreased again and she required continuous airway pressure (CPAP) of 5 cm H₂O via a CPAP mask. She then became progressively more drowsy, and at four hours after the initial event, her neurological state deteriorated. The trachea was then intubated and the lungs were mechanically ventilated. A CT scan at this time showed a diffuse subarachnoid hemorrhage most prominent in the right Sylvian fissure with considerable midline shift. At this time, review of the original post stent angiogram was done and it showed dissection of a branch of the middle cerebral artery with a small amount of extravasation of blood. A repeat angiogram showed vessels that were patent but in spasm.

The patient's level of consciousness remained depressed. At 11 hr post event, an emergency ventricular drain was sited. The initial intracranial pressure (ICP) reading was 40 mmHg. Cerebrospinal fluid was drained and ICP corrected to 20 mmHg. Despite this, she developed profound and rapid cardiovascular compromise, culminating in an episode of ventricular tachycardic arrest from which she was resuscitated. In view of her poor neurological prognosis coupled with her ongoing brittle cardiovascular state, and after discussion with the family, further treatment was withdrawn and she died in the ICU three days after the initial procedure. Autopsy showed evidence of subarachnoid hemorrhage, bilateral lung congestion and small bilateral serosanguinous pleural effusions. There was no evidence of myocardial infarction or pulmonary emboli.

Discussion

Carotid endarterectomy is considered the treatment of choice for occlusive disease at the carotid bifurcation. Studies^{4,5} comparing surgery with medical therapy have found a considerable reduction in stroke rates in the surgery group. These studies have served to popularise a surgical approach to treatment of symptomatic and asymptomatic disease over medical therapy. However, endovascular treatment of carotid artery atherosclerotic disease is being used more frequently as an alternative to surgery.⁶ Whilst initially this involved only angioplasty, more recently, stent placement has been gaining popularity. The first percutaneous therapy for peripheral vascular stenosis simply involved opening a stenotic lesion with a guidewire.⁷ This was followed, a decade later, by the development

of balloon angioplasty and by 1979 there was a report of the first coronary angioplasty series in human patients.⁸ Techniques developed further and by 1983 Bockenheimer and Mathias⁹ had used primary balloon angioplasty for carotid artery stenosis in four patients.

The advantages of a percutaneous procedure include avoidance of a neck incision and dissection of the carotid bifurcation and its concomitant complications of hypoperfusion during clamping and hyperperfusion following clamp relaxation. The procedure requires passing a guide wire across the narrowing to enable plaque stenting or dilation—a process associated with a higher risk of embolization than from dissection of the vessel. The most recent survey of major interventional centres reported on 5210 endovascular carotid stent procedures performed worldwide.⁶ The overall 30 day post procedure mortality rate was 0.86% and a combined minor/ major stroke and procedure related death rate 5.07%. Randomized trials to compare endovascular procedures with endarterectomy are in progress.¹⁰ The only one published at present, was stopped due to an unacceptably high morbidity rate in the endovascular group.¹¹ Other studies and reviews¹² have also concluded that surgery remains the better treatment option for most patients presenting with a symptomatic severe carotid artery stenosis but a subset of patients may qualify as candidates for angioplasty and stenting.¹³⁻¹⁵ These are patients considered to have the greatest perioperative cardiac or cerebrovascular risk factors.

Pulmonary edema secondary to upper airway obstruction has been well documented.^{1,2} It is thought to result from the high subatmospheric intrapulmonary pressures (-50 cm water or more) that can develop as a result of strong inspiratory effort against an obstructed upper airway. The resulting elevation in hydrostatic pressure above oncotic pressure favours transudation of fluid into the peri-alveolar spaces.

Post anesthetic pulmonary edema in otherwise healthy people, has been cited to be caused by post extubation laryngospasm,¹⁶ excessive endotracheal suction¹⁷ or by failure of a drowsy patient to switch from nasal to oral breathing.¹⁸ It occurs at a frequency of 0.05- 0.1% of all anesthetics¹⁹ and often goes unrecognized or undiagnosed.

Neurogenic pulmonary edema (NPE) is a recognised complication of a neurological event, commonly subarachnoid bleeds and major head injuries.¹ It is thought to be related to a central sympathetic discharge as a consequence of acute hypothalamic dysfunction and medullary ischemia. Mayer *et al.*²⁰ described echocardiographic evidence of reduced left ventricular function, border line CK-MB elevation,

various ECG changes and persistent T wave inversions in their series of five patients with SAH and NPE.

For our patient it was initially thought that the most likely causes for the sudden pulmonary oedema were myocardial ischemia, pulmonary emboli or even upper airway obstruction secondary to the boluses of propofol given for agitation to a patient with a short thick neck. Closer examination showed no obvious signs of airway obstruction, either clinically or registered on the monitoring equipment and there were no appreciable changes in the patient's neurological state. This, together with an apparently normal end of procedure cerebral angiogram, accounted for the initial confusion in diagnosis. The patient responded well to initial standard emergency treatment for pulmonary edema. There were no signs of a primary cardiovascular event, no ECG changes, no chest or leg pains and at that stage the only hemodynamic change was a slight increase in the patient's blood pressure. Thus, both myocardial infarction and pulmonary emboli were ruled out. Our attention was then turned to neurogenic causes. The negative angiogram was falsely reassuring, once again favouring an airway or cardiovascular event as the cause. There were no good reasons for reversing the heparin and, in fact, keeping the possibility of a pulmonary embolus in mind it seemed a good indication for maintaining an anticoagulated state. The onset of neurological symptoms was slow and initially the decreased level of consciousness was attributable to hypoxia and exhaustion. However, the patient continued to deteriorate neurologically and the subarachnoid hemorrhage progressed to become a terminal event causing the hydrocephalus and cardiovascular compromise from which she never recovered.

L'e Orme *et al.*²¹ reported a similar case where a patient presented to the emergency department with unexplained pulmonary edema and was initially treated for presumed myocardial ischemia. Only after a full history became available from the patient did a cerebral angiogram confirm dissection of the right vertebral artery. Magnetic resonance imaging showed multiple small areas of infarction in both cerebellar hemispheres and the right side of the medulla.

Minimally invasive radiological procedures are well tolerated with local or neurolept anesthesia and, hence, are an ideal way of treating medically compromised patients whose risks under general anesthesia would be unacceptably high. The awake and responding patient provides the best intraoperative monitor of cardiovascular and neurological perfusion. However, in this case, the absence of signs of neurological deterioration suggested that the problem was extracranial. Perversely the most sensitive warning system failed and, in fact, confounded the true diagnosis.

Arguably, had the patient been anesthetised and the trachea intubated for this procedure the possibility of an airway obstruction would not have been an issue. However she would probably have been kept ventilated and sedated whilst further cardiovascular investigations were performed to determine the cause of the apparent congestive heart failure, not unreasonable in a poorly compliant diabetic. There would have been no appreciable deterioration in the patients neurological state and perhaps the subarachnoid hemorrhage responsible for her inevitable demise would only have been a post mortem finding.

This is a report of NPE occurring in an young awake patient having an endovascular procedure for symptomatic carotid artery stenosis. There was some initial confusion in establishing the cause for the sudden onset pulmonary edema. The diagnosis of acute subarachnoid hemorrhage became apparent through a process of elimination of other possible causes. It illustrates that endovascular procedures carry with them a risk of intracranial events and serves as a reminder that neurogenic pulmonary edema is a likely cause of sudden pulmonary edema in the interventional neuroradiology suite.

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