Case Report

Unilateral neurogenic pulmonary oedema: An unusual cause for post-operative respiratory dysfunction following clipping of ruptured intracranial aneurysm

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ABSTRACT

A variety of central nervous system lesions like stroke, subarachnoid haemorrhage, trauma and seizure activity can result in neurogenic pulmonary oedema (NPE). Unilateral neurogenic pulmonary oedema is very rare. There are no reports of unilateral NPE with aneurysmal vasospasm. We present the case of a 55-year-old female who developed respiratory distress with unilateral pulmonary oedema and mild left ventricular dysfunction in the context of postoperative cerebral vasospasm following clipping of ruptured intracranial aneurysm. Neurogenic pulmonary oedema should always be in the differential diagnosis when patients with presumed neurogenic pathology develop respiratory compromise. The diagnosis of unilateral neurogenic pulmonary oedema requires a high index of suspicion. Early initiation of supportive treatment results in good outcome.

Key words: Cerebral vasospasm, intracranial aneurysm, post-operative, neurogenic pulmonary oedema, unilateral

INTRODUCTION

Acute neurogenic pulmonary oedema (NPE) is a common yet underdiagnosed clinical entity. It can occur after virtually any form of injury to the central nervous system (CNS). NPE is a potential contributor to the pulmonary dysfunction that occurs in these patients. Unilateral neurogenic pulmonary oedema (NPE) is a very rare occurrence. Search of medical databases [Pubmed (NLM) and Medline (Ovid)] using the keywords unilateral, neurogenic, pulmonary oedema and intracranial aneurysm did not reveal reports of the occurrence of this condition following clipping of aneurysm. The diagnosis requires a high index of suspicion, especially in the event of post-operative respiratory dysfunction. We report the case of a female aged 55 years who developed post-operative respiratory distress with unilateral pulmonary oedema in the context of neurological sequelae and clinical evaluation consistent with cerebral vasospasm.

CASE REPORT

A female aged 55 years presented with loss of consciousness of 4 h, altered sensorium of one day and headache of three days duration. She was a known hypertensive on regular treatment with oral Atenolol 50 mg/day for the last four years. She had no other associated co-morbid conditions. Her chest radiograph [Figure 1] and echocardiogram showed normal study. Computed tomography (CT) scan showed right sylvian fissure bleed and subarachnoid haemorrhage (SAH) with extension into the right cerebral hemisphere (Fisher grade 3). The clinical grade of SAH was world federation of neurological surgeons (WFNS) grade I. Cerebral four-vessel angiogram revealed anterior communicating artery aneurysm, and the remainder

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of the vasculature was normal with no vasospasm. All the other clinical and biochemical investigations were normal. Craniotomy and clipping of the aneurysm was contemplated. General anaesthesia was induced with Propofol 2 mg/kg body wt and endotracheal intubation was facilitated with vecuronium bromide 0.1 mg/kg body wt. Anaesthesia was maintained with air and oxygen in a ratio of 50:50, 1% inspired dial concentration of isoflurane. Propofol and fentanyl infusion were titrated to a mean blood pressure of 80 mmHg and vecuronium bromide infusion was administered for muscle relaxation. Central venous pressure (CVP) was maintained at 10 mmHg. Pterional craniotomy was performed in the supine position. The patient was stable haemodynamically throughout the procedure. Direct permanent clip was applied and the procedure was uneventful. At the end of the surgical procedure, the patient was extubated of trachea in the operating theatre after reversal of the residual neuromuscular blockade. The patient was shifted to the neurosurgical intensive care unit for monitoring and further management.

Post-operatively, the patient was conscious, coherent and well oriented. There were no neurological deficits. The mean arterial pressure was maintained at 90–100 mmHg and the CVP was maintained at 10–12 mmHg. On the second post-operative day, the patient developed altered consciousness with drowsiness and was responding to tactile stimulation. She developed progressive dyspnoea, wheeze and crepitations. Her arterial saturation was 85%. Arterial blood gas analysis while on oxygen supplementation of 6 l with face mask showed hypoxemia with a PaO_2 of 50 mmHg, and required tracheal intubation and mechanical ventilation. The patient was afebrile, blood picture was normal and chest X-ray showed diffuse alveolar opacities on the right side [Figure 2]. CVP was 12 mmHg. An initial diagnosis of aspiration pneumonia was made. Tracheal aspirate and blood culture did not yield any growth of microorganisms. CT scan performed to establish the possible cause of neurological deterioration showed no fresh infarct and ventricles and cisterns were normal. She was haemodynamically stable, except for mild tachycardia. There were no fresh changes on the ECG. Echo cardiogram revealed diastolic dysfunction. A pulmonary artery (PA) catheter was placed and the pulmonary capillary wedge pressure (PCWP) was 28 cm H_2O. Repeat chest X-ray 6 h after intubation showed persistence of haziness of the lung fields on the right side. There was no evidence of infection, major cardiac dysfunction, fluid overload or any other detectable systemic cause for the pulmonary oedema. The high PCWP with no significant systolic dysfunction of the heart prompted us to contemplate the diagnosis of NPE. However, the cause for unilateral presentation could not be established.

The patient was managed with diuretics, dobutamine infusion and ACE inhibitors. Positive end expiratory pressure on the mechanical ventilator was increased to 10 cm H_2O. The patient responded to the treatment clinically in 2–3 h and radiologically over 24 h. However, the need for supportive therapy continued for about 36 h, as withdrawal of support resulted in recurrence. During this period, the neurological status also improved. The patient was gradually weaned off the ventilatory support and extubation of trachea was done on the fourth post-operative day. The

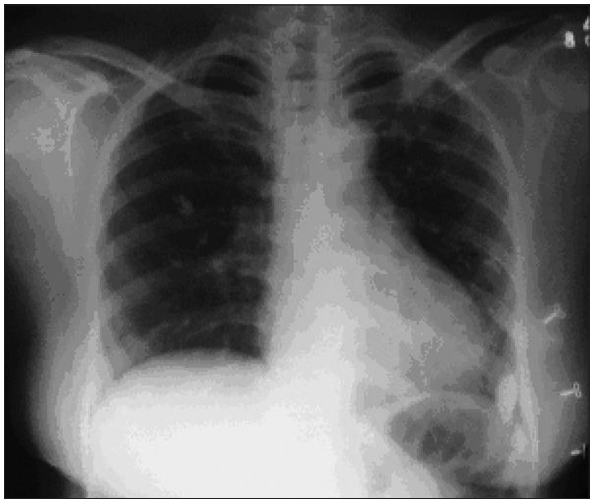

Figure 1: Pre-operative chest radiograph

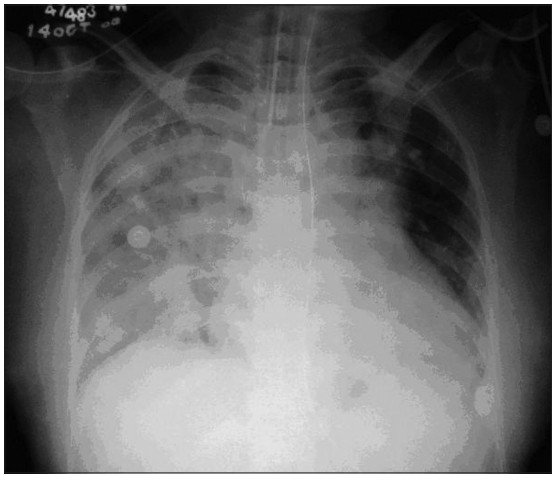

Figure 2: Chest radiograph showing unilateral pulmonary oedema on the right side

patient later received supportive respiratory treatment with continuous positive airway pressure. The NPE resolved [Figure 3] and the repeat ECHO revealed normal cardiac function.

DISCUSSION

NPE is a potential complication of CNS insults such as intracranial haemorrhage, uncontrolled generalized seizures, head trauma, tumours and neurosurgical procedures.[1] They result in cardiopulmonary sequelae because of the interplay between enhanced sympathetic tone and inflammatory cytokine release. NPE develops as a result of altered hydrostatic pressure due to pulmonary venoconstriction or transient elevation in left-sided cardiovascular pressure and permeability defects.[2]

NPE is one of the serious complications that result in poor clinical outcome after SAH.[3] The incidence of NPE is 8%. Higher-grade patients, radiologically severe bleed and ruptured vertebral artery aneurysm tend to be more frequently complicated by NPE.[3,4] Early surgery of ruptured intracranial aneurysms is also associated with a higher incidence of NPE.[5] The onset of NPE can either be acute (3 h after injury) or slow to develop (4 days later). Intraoperative NPE has also been reported.[6,7] Some reports suggest that endovascular treatment of aneurysm with Guglielmi Detachable Coils may improve NPE.[8] However, Brewer *et al*. reported two cases of NPE following angioplasty for refractory cerebral vasospasm following SAH.[9] Surgery and post-operative cerebral vasospasm in our patient could have resulted in delayed appearance of NPE during the post-operative course.

Bilateral alveolar opacities are the usual radiological feature of NPE, but a unilateral presentation is uncommon. The unilateral presentation is usually misdiagnosed in clinical practice. Previous reviews of this subject have discussed the different aetiologies. Rapid re-expansion of the collapsed lung, obstruction of a bronchus, down lung syndrome (gravitational oedema) with or without variations of pulmonary venous pressure, systemic-to-pulmonary arterial shunts, compression or occlusion of the pulmonary vasculatures, disturbances of the neurogenic control of capillary size and permeability, pleural pathologies and impairment of vascularization of one lung are the hypotheses put forward to explain the unilaterality of the distribution. Unilateral NPE is a very rare occurrence. It has been reported after multiple sclerosis[10] and

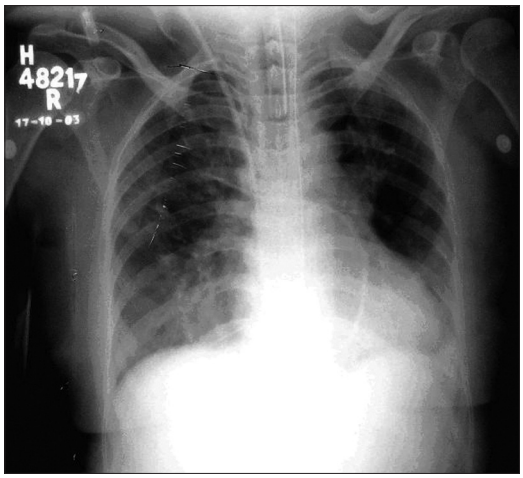

Figure 3: Chest radiograph showing resolution of pulmonary oedema with treatment. X-ray shows pulmonary artery catheter in place

vascular posterior fossa lesion with stroke.[11,12] It has not been reported in aneurysmal SAH. Diagnosis requires a high index of suspicion, especially in the case of post-operative respiratory decompensation. In this case, a female patient aged 55 years developed respiratory distress with unilateral pulmonary oedema in the context of neurological sequelae and clinical evaluation consistent with cerebral vasospasm. The differential diagnosis included excessive vascular filling, infectious pneumonia, gastric fluid aspiration oedema and cardiogenic pulmonary oedema. There was no other detectable cause for respiratory dysfunction and pulmonary oedema. In pulmonary oedema of unknown origin with neurological conditions, the possibility of NPE should be considered. However, the cause for unilateralism could not be established in this case.

This case was reported to highlight the conflicting goals of management of pulmonary oedema and cerebral vasospasm. The complications of most therapeutic modalities suggested for cerebral vasospasm, like triple "H" therapy, nimodipine and magnesium include pulmonary oedema, systemic hypotension and reduction in cardiac output due to vasodilatation. This patient received supportive treatment and recovered completely from this event. Early recognition and timely use of PEEP and vasoactive drugs, together with judicious fluid replacement, are important in the management of patients. In addition, the placement of pulmonary artery catheter is crucial for assessing the cardiac function and fluid status.

CONCLUSION

Unilateral NPE is a very rare occurrence.[11] Its

diagnosis requires a high index of suspicion, especially in the case of respiratory decompensation following a clipping of ruptured intracranial aneurysm. Early initiation of definitive treatment is required for resolution of pulmonary oedema.

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