Pulmonary oedema after airway obstruction due to bilateral vocal cord paralysis

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We report a case of pulmonary oedema which developed after airway obstruction due to bilateral vocal cord paralysis. The patient was a 52-yr-old woman undergoing craniotomy for an acoustic neuroma. Anaesthesia was uneventful. Spontaneous ventilation resumed after reversal of neuromuscular blockade. Following extubation she showed signs of airway obstruction and dyspnoea. The trachea was reintubated but she became hypoxic, PaO_2 – 36 mmHg, produced pink frothy secretions and the x-ray was typical of pulmonary oedema. The oedema cleared within 24 hr. Tracheostomy was performed one week later as the cords were still fixed, but the latter had recovered by three months and the tracheostomy was closed. The cause of the cord paralysis is unknown but probably was a result of surgical trauma to the brain stem.

On rapporte le cas d'un patient ayant développé un œdème pulmonaire aiguë après obstruction des voies aériennes suite à une paralysie bilatérale des cordes vocales. La patiente âgée de 52 ans a subi une craniotomie pour un neurinome acoustique. L'anesthésie fut accomplie sans incident. La ventilation spontanée fut reprise après antagonisme du blocage neuromusculaire. Après l'extubation, la patiente a démontré des signes d'obstruction des voies aériennes et de la dyspnée. La trachée fut réintubée mais la patiente devint hypoxique, PaO_2 – 36 mmHg, et on a observé des sécrétions spumeuses rosées et le rayon-x a démontré un œdème pulmonaire typique. L'œdème a régressé en dedans de 24 heures. Une trachéostomie fut faite une semaine

plus tard car les cordes vocales étaient encore fermées et la récupération s'ensuivit trois mois plus tard, c'est alors que la trachéostomie fut fermée. La cause de la paralysie des cordes vocales n'est pas connue mais serait due probablement au trauma chirurgical.

Airway obstruction may cause pulmonary oedema in both awake and anaesthetized patients.^{1–16} Acute airway obstruction produces a large subatmospheric transpulmonary pressure gradient elicited by attempts to breathe against a closed glottis, or narrowed airway.^{5,17–19} Reported cases included hanging or strangulation,^{1,14} laryngospasm during induction of or emergence from anaesthesia,^{2–4,8,10,12,15,16} laryngeal obstruction by tumour, tracheal obstruction due to goitre or neoplasm,^{1,14} croup, acute infectious pharyngitis or epiglottitis,^{6,13} and laryngeal haematoma.¹¹ Vocal cord paralysis also causes airway obstruction, but complete obstruction is rare in adults.^{20–24} In a recent review, there were no reports of pulmonary oedema occurring after vocal cord paralysis.²⁵ We report a case in which pulmonary oedema occurred after airway obstruction due to bilateral vocal cord paralysis in a patient undergoing cerebellopontine angle surgery.

Case report

A 52-yr-old female (weight of 45 kg) with regrowth of a right acoustic neuroma was scheduled for craniotomy and removal of the tumour. Her past medical and three surgical records revealed right facial palsy, nystagmus indicating the involvement of fifth, seventh and eighth cranial nerves but no previous anaesthetic complication. The patient also showed left-sided cerebellar gait disturbance and some signs of intracranial hypertension. The patient received diazepam, 10 mg, *po*, 90 min before arrival in the operating room. After an *iv* catheter, ECG and direct arterial blood pressure monitoring were provided, anaesthesia was induced with thiamylal, 5 mg · kg⁻¹ *iv*, and manual hyperventilation of the lungs was performed using enflurane, nitrous oxide and oxygen. Pancuronium 6 mg *iv* was given to facilitate tracheal

Key words

AIRWAY: obstruction;
LUNG: oedema.

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intubation together with lidocaine 70 mg to depress the associated cardiovascular responses. A tracheal tube (7.5 mm, profile cuff tube, Portex) was placed in the patient's trachea and the cuff was inflated with air. The vocal cords were difficult to visualize but intubation was performed "blindly" by direct laryngoscopy. The anaesthetic course was uneventful for the seven-hour operation during which the patient's condition was stable (systolic BP remained between 100 and 140 mmHg, heart rate 78–90 bpm, CVP 3–4 cmH₂O, body temperature 34.9–36.3° C, PaO₂ 160–178 mmHg, PaCO₂ 31–34 mmHg with 33% oxygen, 67% nitrous oxide and 0.6–1.5% enflurane). She received a total of 800 ml whole blood and 1500 ml of lactated Ringer's solution, and the blood loss and urine output during anaesthesia were 978 ml and 380 ml, respectively.

After residual neuromuscular blockade with pancuronium was reversed by neostigmine, 2.5 mg, and atropine, 1.0 mg, the patient regained consciousness and responded to commands regarding grasp and movement of the extremities. During the emergence from anaesthesia her systolic BP increased from 110–120 mmHg to 146–176 mmHg and the tracheal tube was removed without difficulty. Immediately after extubation, paradoxical chest motion was noted, and the patient became progressively dyspnoeic. Manual ventilation with 100% oxygen by a mask did not improve her condition. Two to three minutes after extubation arterial blood gas analysis showed PaO₂ 309 mmHg, PaCO₂ 68 mmHg, and pHa 7.265. Laryngospasm was suspected, but the patient was alert and was able to attempt several deep breaths with marked suprasternal and subcostal retractions on command. A few minutes later the trachea was reintubated. There was no gag reflex and no reaction to laryngoscopy and tracheal intubation. Five minutes later blood gas analysis showed PaO₂ 36 mmHg, PaCO₂ 64 mmHg while the patient breathed spontaneously on room air. Immediate suction produced a mass of pink frothy secretions through the tracheal tube. A portable CXR showed a normal heart size but diffuse and fluffy bilateral alveolar infiltration which was prominent in right upper lobe regions, and obliteration of the hilar shadows. A sample of the pulmonary oedema fluid, a total of 132 ml collected for about 30 min, showed a total protein content of 6.3 mg · dl⁻¹ and a pulmonary oedema fluid-serum protein ratio of 0.92 (Table).

During positive-pressure ventilation of the lungs with end-expiratory pressure (PEEP) and intermittent manual hyperinflation, PaO₂ gradually improved over next six hours and the amount of suctioned pulmonary oedema fluid progressively decreased. Approximately 18 hr after the onset of pulmonary oedema, PaO₂ was 154 mmHg and PaCO₂ was 39 mmHg with a tidal volume of 350 ml

TABLE Components of pulmonary oedema fluid and serum, 15 min after the onset of pulmonary oedema

Component	PE fluid	Serum*	(Normal range)	
			in serum	
Total protein	6.3	6.7	6.5 ~ 8.2	d · dl ⁻¹
Albumin	3.6	3.7	3.1 ~ 5.1	g · dl ⁻¹
A/G	1.33	1.23	1.4 ~ 2.2	
Calcium	9.9	9.0	8.2 ~ 10.8	mg · dl ⁻¹
Inorganic phosphate	4.8	4.3	2.5 ~ 5.5	mg · dl ⁻¹
Sodium	161	141	134 ~ 147	mEq · L ⁻¹
Chloride	130	109	96 ~ 110	mEq · L ⁻¹
Potassium	9.1	3.1	3.7 ~ 5.0	mEq · L ⁻¹
Urea nitrogen	15.9	12.0	8.0 ± 20.0	mg · dl ⁻¹
Creatinine	0.7	0.7	0.4 ~ 1.1	mg · dl ⁻¹
Uric acid	1.3	3.9	2.0 ~ 6.0	mg · dl ⁻¹
CPK	211	105	0 ~ 165	IU · L ⁻¹
CPK-MB	255	18	5 ~ 30	IU · L ⁻¹
SGOT	64	22	0 ~ 40	IU · L ⁻¹
SGPT	0	6	0 ~ 40	IU · L ⁻¹
LDH	2699	390	120 ~ 520	IU · L ⁻¹
Alkaline phosphatase	353	87	50 ~ 230	IU · L ⁻¹
Choline esterase	45	277	170 ~ 420	IU · L ⁻¹
Amylase	590	105	40 ~ 240	IU · L ⁻¹
Bilirubin	1.1	1.3	0.2 ~ 1.0	mg · dl ⁻¹
Osmolarity	340	296	275 ~ 295	mOsm · L ⁻¹

	PE fluid	Blood*
WBC (× 10 ³)	2.1	12.8
RBC (× 10 ⁶)	0.19	4.54
Hb (g · dl ⁻¹)	1.1	14.5
Ht (%)	1.7	42.4
Platelet (× 10 ³)	5.7	139
Haemolysis	58	0

*Results of simultaneous arterial blood sample.

and 30% inspired oxygen using CPAP of 5 cmH₂O. Repeated CXR showed that the pulmonary infiltrates had resolved. Since she was alert and her general condition was very stable, her trachea was extubated, but she complained of difficulty in breathing; she breathed with a very husky voice and inspiratory dyspnoea was noted. Soon after she became cyanosed and the trachea was reintubated. Examination of the larynx and vocal cords with a fiberoptic bronchoscope found no laryngeal oedema, but revealed that both true cords were fixed in the midline with a 1–2 mm airway. Bilateral vocal cord paralysis due to recurrent laryngeal nerve palsy was suspected.

Neurological examinations revealed anisocoria (right > left), bilateral horizontal nystagmus, bilateral VII nerve paresis, left VI palsy, depressed gag reflex, ataxia, and aphonia. A CT examination revealed left-sided shift of the brain stem due to the CP angle mass and low density area around the rostral medulla oblongata and caudal pons, and some clots in the fourth ventricle.

One week later, laryngoscopy revealed bilateral vocal

cord fixed and 14th postoperative day, the extubation was unsuccessful. Tracheostomy was performed under local anaesthesia.

The course of rehabilitation was uneventful. Three months later, the tracheostomy cannula was removed because the movement of vocal cords recovered. The patient discharged although the neurological deficits remained.

Discussion

Laryngospasm and laryngeal oedema are the most serious immediate airway problems following extubation of the trachea. In the present case, we postulated that the airway obstruction after extubation of tracheal tube was initially due to laryngospasm and then due to laryngeal oedema.^{24,26} Laryngospasm is unlikely if the depth of anaesthesia is sufficient during extubation of the trachea or the patient is allowed to awaken before the extubation. Laryngeal oedema, though rare, occurs especially in children²⁴ or after neck surgery. Difficulty in the visualization of the larynx and vocal cords prevent them from being differentiated.

Bilateral vocal cord paralysis following tracheal intubation²⁰⁻²³ or without previous intubation^{27,28} is a rare cause of airway obstruction. After four attempts at extubation resulted in acute airway obstruction we suspected recurrent laryngeal nerve palsy. Although rare, it has been recognized after tracheal intubation but usually does not cause airway obstruction.²⁰⁻²³ It may have occurred in this patient following compression of the peripheral anterior branches of recurrent nerve^{21,23} by an over-inflated cuff or following vocal cord trauma due to "blind" intubation.

There are reports of bilateral vocal cord paralysis leading to acute airway obstruction occurring in patients with brain stem ischaemia²⁷ and stroke.²⁸ In this patient postoperative neurological and brain CT examinations suggested local ischaemia of lower cranial neuronal regions. It is possible that the surgical intervention of the brain stem region may have caused the paralysis. The nucleus ambiguus occupies much of the rostral medulla and caudal pons and the axons of the motor neurones innervating the pharynx, larynx and upper oesophagus pass via the 9th, 10th, and 11th cranial nerves. The neurons serving abduction are phylogenetically newer and fewer than the adductor neurons,²⁸ and are more susceptible to injury.²⁸ Also, since the adductors have three times more muscle mass than the abductors, injury to the abductor neurons can be responsible for cord spasm in adduction. The anterior branch of the recurrent laryngeal nerve innervates the cord adductors and is vulnerable to compression between an expanded cuff and the overlying thyroid cartilage. Thus, cord paralysis due to tracheal tube

cuff compression usually leaves them in the intermediate position and thus hoarseness, but not airway obstruction, is more likely.²¹ Therefore the bilateral vocal cord paralysis in this patient was more likely to have been due to insult of the brain stem than to local recurrent laryngeal nerve palsy.

The development of pulmonary oedema as a complication of upper airway obstruction is rare but well-recognized. Mechanisms for its development are not well understood but it is suggested that increased permeability is the principal cause. The large negative intrathoracic pressure favours transudation of fluid from the pulmonary capillaries into the alveolar space.^{4,5,10,14,16,18} It is possible that pulmonary oedema occurred from neurogenic mechanisms associated with the surgery as has been reported in traumatic cerebral impact, cerebral compression, and seizures.²⁹⁻³¹ This is unlikely because there was no evidence of sympathetic overactivity, or increased intracranial pressure.

The high protein content of the oedema fluid (pulmonary oedema fluid/serum ratio of 0.92) of the present patient suggests that the pulmonary oedema followed increased permeability.^{32,33} Experimentally, high oedema fluid/serum protein ratio has been reported in permeability oedema (0.98 ± 0.05), whereas lower values have been reported in neurogenic oedema ($0.48 \sim 0.84$) and in haemodynamic oedema (0.54 ± 0.04) in dogs.³²

We have not previously experienced bilateral vocal cord paralysis as a complication of cerebellopontine surgery. Surgical procedures in this region may be associated with a variety of potential, rare complications.³⁴ Indeed, a recent report described respiratory obstruction which occurred due to bilateral abductor vocal cord paralysis in infants with increased intracranial pressure with the Arnold-Chiari malformation.³⁵ Postanaesthetic laryngospasm due to upper airway obstruction is the most frequent cause of the pulmonary oedema,¹⁶ but bilateral vocal cord paralysis should be considered as a potential cause of acute airway obstruction leading to pulmonary oedema.

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