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Hydropneumo- thorax - an unusual complication of lung lavage

Hydropneumothorax is an uncommon but potentially fatal complication for a patient undergoing positive pressure ventilation. The case of a 23-year-old woman with severe asthma requiring lung lavage is described. Twenty minutes after an uneventful left lung lavage, the patient experienced increased peak airway pressure, decreased oxygen saturation and hypercarbia, despite ventilation with 90 per cent oxygen. A chest x-ray revealed mediastinal shift and a left sided pneumothorax. Drainage was carried out, revealing air and clear fluid in the pleural space. The importance of technical problems such as patient and endotracheal tube positioning, elimination of cross-spilling and cardiopulmonary effects of lavage are discussed.

Unilateral lung lavage under general anaesthesia is a procedure utilized in patients with severe respiratory embarrassment, due usually to asthma or alveolar proteinosis. The procedure itself utilizes selective one lung ventilation and therefore stresses the patient's cardiopulmonary system. The complication of a hydropneumothorax in a patient undergoing controlled ventilation is a potentially fatal one and must be recognized early. The following case report describes the occurrence of a hydropneumothorax shortly after lung lavage and demonstrates

the problems involved with this unusual and rare complication.

Case report

A 23-year-old woman was transferred to Toronto General Hospital after four weeks in another hospital with status asthmaticus.

The patient had a ten-year history of asthma, with frequent admissions to hospital. She had been steroid dependent but never required intubation and ventilation. The haemoglobin concentration was $153 \text{ g}\cdot\text{L}^{-1}$. Serum electrolytes, urea nitrogen and creatinine were normal as were the chest x-ray and ECG. The FEV_1 was 600 ml and the FVC was 1200 ml. The patient was unable to tolerate full pulmonary function testing.

The patient was premedicated with salbutamol 0.5 ml, ipratropium 1 ml by inhalation, meperidine 75 mg IM, promethazine 25 mg IM and methylprednisolone 40 mg IV one hour preoperatively. Operating room monitoring included an electrocardiogram, blood pressure cuff, axillary temperature probe and pulse oximeter. An aminophylline drip was infusing at $25 \text{ mg}\cdot\text{hr}^{-1}$. An arterial line could not be established.

Induction included diazepam 10 mg, ketamine 150 mg, fentanyl 250 μg and pancuronium 8 mg. Intubation was carried out with a #35 French right sided National Catheter double lumen tube, without difficulty. The tube's position was checked by alternate clamping of the bronchial and tracheal portions of the connector and bilateral chest auscultation during ventilation. An underwater bubble test was done whereby the left lung port was clamped and connected to an underwater seal. This technique is explained in the discussion. Contralateral lung inflation failed to cause bubbling in the underwater seal at an airway pressure of 50 cm H_2O . This demonstrated the separation of the two

Key words

LUNG: lavage, alveolar proteinosis, asthma;
COMPLICATIONS: hydropneumothorax.

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lungs, even with positive pressure. The position of the tube and appropriate ventilation of the lungs was reconfirmed using fluoroscopy. Anaesthesia was maintained with oxygen and halothane 1.0 per cent.

Ventilation was controlled with a tidal volume of 600 ml at a frequency of 12 breaths·min⁻¹, using a circle system. Peak airway pressure was 35 cm H₂O. Since the left lung was to be lavaged the left lung tracheal port of the double lumen tube was clamped and connected to a "Y" tubing. One limb of the "Y" tubing was connected to a drainage bag on the floor, and the other was connected to an irrigation bag approximately 30 cm above the patient's mid axillary line. The irrigating solution was a three litre bag of warmed normal saline with 300 µg of added adrenaline. The patient was kept supine. To effect filling of the left lung, a clamp was removed from the irrigation line and placed on the drainage line. Each filling was continued until the stream of the solution slowed down (approximately 1500–1800 ml of solution). Each filling and drainage was accompanied by a physiotherapist doing chest percussions to facilitate return of the lavage fluid.

During the fourth and last drainage, the left lung was re-expanded with manual ventilation while the physiotherapist vibrated the chest. The lung was suctioned intermittently and was then re-expanded with a manual resuscitation bag delivering oxygen. This manoeuvre generated airway pressures between 50 and 60 cm H₂O on the non-lavaged side. When suction produced little return of fluid, the double lumen tube was replaced with a single lumen Portex #8.0 endotracheal tube. Throughout the procedure, the patient remained haemodynamically stable and maintained a haemoglobin saturation of 98 per cent. She was taken to the recovery room, and was electively ventilated on 90 per cent oxygen.

Approximately one half hour later, the pulse oximeter registered 82 per cent saturation. Airway pressures were as high as 60 cm H₂O. At this time the patient had a tachycardia of 130 beats·min⁻¹ but the blood pressure was normal. Blood gases revealed: pH 7.37, PO₂ 62 mmHg, PCO₂ 58 mmHg and HCO₃⁻ 34 mmol·L⁻¹ (FiO₂ = 0.9). The initial chest x-ray showed left sided pulmonary oedema but a repeat chest x-ray one half hour later revealed a small left pneumothorax and partial right lung collapse with shift of the mediastinum to the right. Physical exam showed no tracheal deviation,

or loss of breath sounds on the left side. A left-sided chest tube was inserted which drained considerable air and more than 500 ml of clear fluid. The oxygen saturation immediately rose to 97 per cent and peak airway pressure returned to 40 cm H₂O. Within two hours an arterial blood gas revealed: pH 7.46, PCO₂ 40 mmHg, PO₂ 163 mmHg, and HCO₃⁻ 30 mmol·L⁻¹ (FiO₂ = 0.7). The patient was extubated the next day. Blood gases during spontaneous respiration were: pH 7.46, PCO₂ 44 mmHg, PO₂ 75 mmHg and HCO₃⁻ 32 mmol·L⁻¹ (FiO₂ = 0.35). She was transferred to the ward for an uneventful recovery.

Discussion

The discovery of a hydropneumothorax should alert the physician to an iatrogenic cause. Case reports of this unusual problem relate to some form of instrumentation or procedure. Hydropneumothorax occurs as a complication of small naso-enteral feeding tubes which inadvertently enter the airway and pierce the pleura.^{1,2} The possibility of a central venous catheter causing a hydropneumothorax is also established.³ A less well-known cause includes a pleural rupture during percutaneous nephrolithotomy.⁴ There is also a report of hydropneumothorax following rupture of the gastric fundus in a paraoesophageal hernia after gastropasty.⁵

If lung lavage becomes a more common procedure in the treatment of pulmonary disorders, hydropneumothorax may become a more common complication. Presently, lung lavage is a procedure done infrequently and in only a few centres. A rare complication such as hydropneumothorax may go unrecognized at first. At this centre the authors have had one previous case of pneumothorax with lung lavage (unpublished data). The efficacy of lung lavage for a variety of pulmonary diseases is well described elsewhere.^{6–12}

The technique of lung lavage presents difficulties as well as physiologic changes and complications. These will be addressed briefly. The major difficulties involve positioning the double lumen tube, positioning of the patient, cross spillage of fluid, fluid trapping, and circulatory disturbances.¹³ The proper positioning of the double lumen tube is essential in order to achieve complete separation of the lungs. Improper positioning can lead to cross spillage of fluid to the other lung which can lead to hypoxia and hypoventilation. It may also impede

drainage. A small change in tube position may correct this.¹⁴

Confirmation of tube position can be made by auscultation, image intensifier, chest x-ray, and a bubble test, as we have done. Also the use of a small fiberoptic bronchoscope down each lumen of the double lumen tube will confirm the position and complete separation of the lungs. As well, this technique allows for diagnosis of an overinflated bronchial cuff which can narrow the bronchial lumen and impede drainage of fluid and cause fluid trapping.¹⁵ A bubble test is performed by ventilating one lung while the double lumen port of the other lung is connected to an underwater seal (via rubber tubing). Application of 50 cm H₂O pressure to the contralateral lung should cause no bubbling via the tube if there is complete separation of the lungs. The test is done for both sides.¹⁵

Cross spillage can be identified by bubbles appearing in the lavage fluid, a decreased return of fluid, rales, rhonchi in the ventilated lung and by desaturation of an oximeter. Cross spillage is not only caused by improper endotracheal tube position but by inadequate inflation of balloon cuffs.¹⁴ Fluid trapping as well as being caused by overinflated bronchial balloon cuffs and tube malposition may also be a sign of hydropneumothorax or cross spillage.

The physiologic changes during lavage involve an improvement in PO₂ during the filling phases. This is due to a loss of pulmonary capillary perfusion secondary to liquid filling alveoli causing collapse of capillaries, which decreases shunt flow. During the drainage phase, this is relieved and pulmonary blood flow is increased in the unventilated lung and shunt is increased leading to a decrease in PO₂.^{16,17} The lavage of the lung also acts to remove a large quantity of surfactant material, leading to a stiff non-compliant lung. Ventilation for a few hours with PEEP until surfactant is replenished avoids respiratory failure secondary to atelectasis.¹⁹ Hypoxia and hypercarbia have been implicated as causes of premature ventricular contractions and bigeminy.¹⁴

Lung lavage, by washing out surfactant, makes the lung less compliant and more susceptible to barotrauma. This likely occurred during reinflation of the patient's lung during the final drainage of irrigation, and led to the hydropneumothorax. Tearing of pleural adhesions during the vigorous

manipulations of chest wall and lungs may also be a factor.

In conclusion, we want to stress that a chest x-ray after the procedure must always be done, to rule out barotrauma. In this case, a repeat examination was necessary before the process causing the hypoxia had progressed far enough to be identified.

Vigorous manual ventilation with a manual resuscitation bag is necessary to re-expand the lavaged lung. The barotrauma likely occurs as a result of the high airway pressures generated. It is the authors' recommendation that manometry of the lavaged lung during re-expansion be done so peak pressures can be limited to less than 50 cm H₂O. This may minimize the likelihood of this complication.

In our experience of over one hundred lung lavages hydropneumothorax has not occurred until this case. We also believe it has not been described in the literature. This diagnosis needs to be kept in mind since it is easy to ascribe hypoxia to the patient's disease process, or to the physiologic effects of the lavage, rather than to this potentially fatal complication.

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Résumé

L'hydropneumothorax est une complication peu fréquente mais potentiellement fatale pour un patient ventilé à pression positive. Le cas d'une femme de 23 ans présentant un asthme sévère et devant subir un lavage pulmonaire est décrit. Vingt minutes après un lavage du poumon gauche sans accident, la patiente présente une augmentation de la pression des voies aériennes, une diminution de la saturation en oxygène et une hypercarbie malgré une ventilation à 90 per cent d'oxygène. Le rayon-x révéla un déplacement médiastinal et un pneumothorax gauche. Le drainage effectué a démontré la présence d'air et de liquide clair dans l'espace pleural. L'importance des problèmes techniques tels que le positionnement de la patiente et du tube endotrachéal, l'élimination du "cross-spilling" ainsi que les effets cardiopulmonaires du lavage sont discutés.