

# Unilateral Pulmonary Oedema Following Total Hip Arthroplasty

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## Abstract

The occurrence of unilateral pulmonary oedema in post operative patients is very uncommon (11). It has been described in urological (8) and cardiothoracic surgery (13) but to the best of our knowledge it has not been reported following orthopaedic surgery. Pulmonary oedema can occur due to congestive cardiac failure, its unilateral presentation being rare. We present a patient with pre-existing left ventricular dysfunction who developed unilateral pulmonary oedema (down lung syndrome) as a postoperative complication of total hip replacement in lateral decubitus. The condition was diagnosed retrospectively and managed with a satisfactory clinical outcome. The risk factors, pathophysiology, differential diagnosis and treatment of this uncommon condition are discussed.

## CASE REPORT

A 69 year old male weighing 75kgs, height 1.63m and Body mass index (BMI) 28 underwent elective primary total hip arthroplasty for osteoarthritis of his right hip. Significant medical history included ischemic heart disease, congestive cardiac failure presently well controlled on regular furosemide and atrial fibrillation which was rate controlled with digoxin. Clinical examination was normal, lung fields were clear on auscultation, and airway evaluation was a modified Mallampatti class 3 (<sub>15</sub>) All the preoperative investigations including haemoglobin and renal function tests were normal. In view of his ischaemic heart disease an echocardiogram was done, which revealed an ejection fraction of 35%, marginally elevated pulmonary artery pressure and left ventricular wall hypokinesia.

Considering the comorbid illness, the patient was classified as belonging to the American society of Anaesthesiologists (ASA) physical status 3 (<sub>16</sub>) and due to poor functional activity, cardiac effort tolerance status was New York Heart Association (NYHA) class 3 (<sub>17</sub>).

Prior to induction of anaesthesia, 3 lead electrocardiography (ECG), pulse oximetry and non invasive blood pressure monitoring was started. Balanced anaesthesia was induced using midazolam 3 mg, fentanyl 50 microgram and propofol 100 mg. Laryngeal mask size 4 (LMA Classic®, Intavent Orthofix, Berkshire) was inserted and patient allowed to breathe spontaneously. Central venous line and radial artery

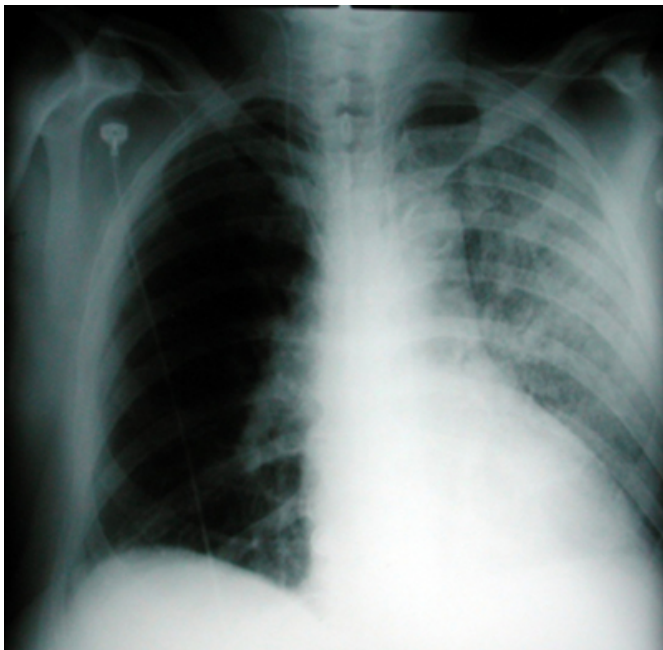
catheter were inserted for intraoperative hemodynamic monitoring. Patient also had right lumbar plexus block using 20cc of 0.5% bupivacaine in the left lateral position. Intraoperative monitoring included end tidal gas analysis, inspired oxygen concentration (FiO<sub>2</sub>), spirometry-flows and volumes, airway pressure, temperature, invasive arterial and venous pressures. Anaesthesia was maintained using oxygen nitrous oxide mixture and end tidal sevoflurane was maintained between 1.2-2% and gas flows used were between 1-4 litres/min. Patient was then positioned left lateral and a right primary cemented total hip arthroplasty performed. 45 minutes after the start of the operation around the time of femoral cementing, the patient started desaturating. SpO<sub>2</sub> steadily dropped from 97% to 88%, there was no evidence of obstructed breathing and patient maintained spontaneous respiration and end tidal carbon dioxide remained between 5.5 to 6.5 Kpa. FiO<sub>2</sub> was increased to 100%, ABG done at the time showed a pH of 7.32, PaO<sub>2</sub> 8.3kpa, PaCO<sub>2</sub> 4.3 kpa. There was bilateral air entry and no rhonchi heard in both lung fields, the airway pressures remained normal and lung compliance on hand ventilation was normal. Spirometry showed adequate tidal & minute volumes. We clinically ruled out common causes like hypoventilation due to obstructed breathing or opioids, pneumothorax and anaphylaxis but it was not possible to rule out aspiration, atelectasis, pulmonary edema and cement embolisation. The temporal sequence of events related to the cement favoured embolisation and ventilation perfusion

mismatch (V/Q) causing hypoxemia. Since it was not possible to intubate the patient in the lateral position under the drapes, intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP) was delivered through the LMA after muscle relaxation with vecuronium. His oxygen saturations improved to 93-95% with FiO<sub>2</sub> 0.6.

Intraoperative blood loss was approximately 400 ml; patient had 1.5 litres of Hartmanns solution and 500 mls of Gelofusine® (B. Braun Melsungen AG) and no blood was transfused. There were no hypotensive episodes. Central venous pressure monitoring throughout the operation was normal between 7 to 12 mmHg and urine output was 70-80ml per hour. At the end of the operation, muscle relaxant reversed with neostigmine and glycopyrrolate, the patient was sat up to 45°, patient reverted to spontaneous respiration, his oxygen saturations improved to 95% with PEEP 5, ABG showed PaO<sub>2</sub> 12kpa, PaCO<sub>2</sub> 5.9kpa. Once patient was awake, Laryngeal mask was removed, the undersurface of which showed no features suggestive of aspiration. A chest radiograph (CXR) was taken in the recovery unit which revealed alveolar and interstitial shadows on the left lung suggestive of pulmonary oedema with unilateral presentation (fig 1).

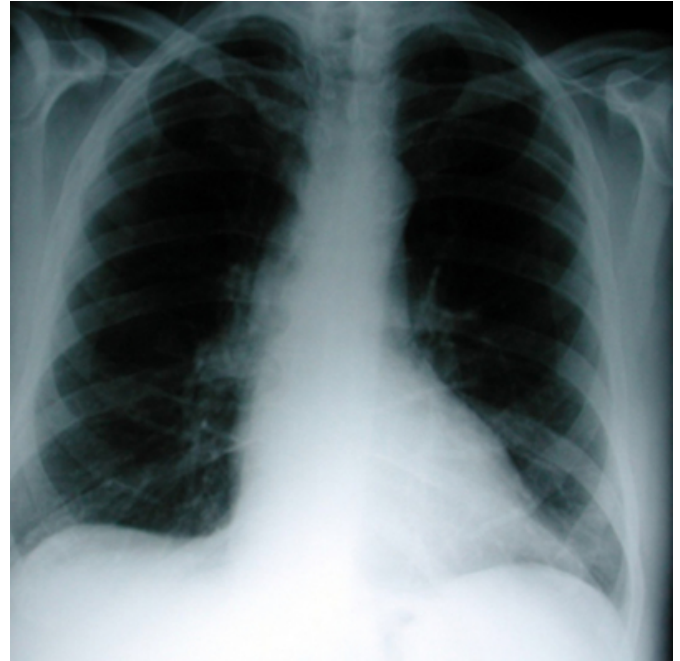
**Figure 1**

Figure 1 : Chest radiograph- Unilateral Pulmonary oedema



**Figure 2**

Figure 2: Chest radiograph- After treatment



The patient was maintained in the propped up position; Continuous positive airway pressure (CPAP) of 5cms of water, FiO<sub>2</sub> 40%, fluid restriction, Furosemide 40mg 8th hourly and chest physiotherapy were instituted. Pulmonary oedema resolved rapidly in 12 hours and the patient had an uneventful post operative recovery (figure 2)

## DISCUSSION

Lateral decubitus is a routinely used position in elective hip replacement surgery. LMA and spontaneous ventilation is a very common practice in anaesthesia for hip replacements as followed in our institution. It is not uncommon to experience episodes of desaturation in patients in lateral position, spontaneous ventilation and during cement application. Common differential diagnosis includes hypoventilation, aspiration, atelectasis, bronchospasm, anaphylaxis, cardiac, neurogenic, negative pressure pulmonary oedema (NPPO), pneumothorax and cement/fat embolisation. Unilateral pulmonary oedema is a rare cause of intraoperative desaturation in patients in lateral position, the diagnosis in our case was made retrospectively on postoperative CXR after excluding all other causes.

There are different factors associated with development of pulmonary oedema in surgical patient. Some of the reported causes include those associated with lung reexpansion<sup>(10)</sup>, obstructed breathing<sup>(6,9,14)</sup>, neurogenic pulmonary oedema<sup>(3)</sup>, unilateral pulmonary oedema associated

with acute cardiac event (myocardial infarction, ventricular septal rupture, chordae tendinae rupture (1)) and those associated with congestive cardiac failure (7,12). Lung re-expansion pulmonary oedema is more common after tumour excision (10), and intrathoracic surgeries (10). Unilateral pulmonary oedema due to obstructed breathing is common with inadvertent single lung intubation, improperly placed laryngeal mask (14) or post extubation laryngospasm. In our patient the possibility of improper placement of laryngeal mask was unlikely because there was no evidence of obstructed breathing through out the procedure, end tidal carbon dioxide trace, oxygen saturation and tidal/minute ventilation remained normal through out the procedure. Unilateral pulmonary oedema following acute intraoperative cardiac event was unlikely due to normal troponin T, absent ECG changes and postoperative echocardiogram showing no new changes. Aspiration of gastric contents into the dependant lung is a likely possibility but the timing, absence of contamination of the undersurface of the LMA, generalized rather than localized infiltrates on the CXR, onset and resolution of changes in a rather short span of time go against the diagnosis.

Fat embolism (20) and cement embolism (19) causing ARDS have also been reported but unilateral presentation of ARDS is extremely rare. The absence of other clinical findings, short latency period of findings in Xray, unilateral & homogenous presentation goes against the diagnosis though their contribution to development of pulmonary oedema cannot be undermined. Bronchioalveolar lavage analysis for fat droplets may have been helpful but the rapid improvement in the clinical condition of the patient did not warrant such an evaluation.

In the lateral position the normal ventilation and perfusion relationships are changed (2). The nondependent or up lung is relatively hypoperfused and hyperventilated and the down lung is relatively hyperperfused and hypoventilated. Ventilation of the down lung is further impaired by the weight of the mediastinal structures, induction of anaesthesia and reduced functional residual capacity causing a ventilation/perfusion (V/Q) mismatch (18). The pulmonary circulation is a low pressure circuit; the Starling forces at the alveolocapillary membrane are altered due to the raised pulmonary arterial and venous pressures in the dependant lung & compounded by the effects of Hypoxic pulmonary vasoconstriction (HPV) due to V/Q mismatch on the pulmonary arterial and venous pressures (3). This alteration of Starling forces in the dependent lung in a person with pre-

existing cardiac dysfunction and raised pulmonary arterial and venous pressure possible led to dependant unilateral pulmonary oedema (down lung syndrome). Cement and fat embolisation may also contribute to the raised pulmonary arterial and venous pressures (4). The temporal sequence of events in relation to the cementing of the femoral prosthesis suggests cement embolisation may have contributed to left ventricular dysfunction and V/Q mismatch. Our patient had all the risk factors namely lateral decubitus position, cardiac dysfunction, cement use and hypoxemia.

In conclusion, unilateral pulmonary oedema due to left ventricular dysfunction (down lung syndrome) is a rare cause of unilateral infiltrates on chest xray. It is a diagnosis of exclusion after all the common causes have been considered. A high index of suspicion is necessary, the key lies in the early onset of radiologic findings and its good response to treatment.

### CONFLICTS OF INTEREST

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