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Bronchospasm/desaturation under anaesthesia: are they signs of pneumothorax?

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Bronchospasm/Desaturation Under Anaesthesia: Are They Signs of Pneumothorax?

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Introduction

The incidence of iatrogenic pneumothorax has increased as a result of aggressive invasive monitoring procedures, new modes of mechanical ventilation and invasive diagnostic/therapeutic techiques. The diagnosis of pneumothorax under anaesthesia can be difficult due to poor clinical manifestation (only signs may be cardiovascular changes, decreased oxygen saturation, or wheezes). If it is not diagnosed earlier and treated[1], it can lead to high mothidity and mortality. We are reporting two unusual cases of pneumothomx which occurred in otherwise healthy patients.

Case Report

Case 1

A 60 years old lady with a lump in her left breast which gradually increased in size over the last two years was admitted for modified radical mastectomy. Pre-operative assessment revealed an average height and body weight of 72 Kg. Physical examination did not reveal any systemic abnormality. She was normotensive, non-asthmatic and non-diabetic. All laboratoiy investigations, E.C.G. and X-rays chest were within normal limits. She was classified as ASA 11 because of relative obesity. The patient was advised a standard premedication of tab. midazolam 7.5 mg orally 2 hours before surgery. In the operating room, triple monitoring (non-invasive blood pressure, ECG and pulse oximetery) was started. She was induced with Thiopental sodium 4-5 mg/kg B. Wt. and tracheal intubation was facilitated with succinylcholine (1.5 mg/Kg B. Wt.). Patient was then placed on control mode of ventilation using IPPV on Siemen 710 ventilator, with a tidal volume of 650 ml and at a rate of 10/minute. Anaesthesia was maintained with a mixture of oxygen/N2O (40-60%) and halothane (0.5-1%). Relaxation was maintained with pancuronium bromide (1-2 mg on Pm bases) while analgesia was provided with intravenous pethidine (1 mg/Kg). Haemodynaniic parameters e.g. blood pressure (130/90 mm Hg) and heart rate (65/minute) increased temporarily at intubation and then stabilised towards the baseline values. Peak airway pressures were 25 cmH2O at this stage. Before proceeding with the surgery, it was decided to take a core needle biàpsy from the breast lump. The surgical resident made three attempts to take the tissue with the help of Trucut biopsy needle (Baxter Travenol 14G, 4/1/2 inches long). Fifty minutes after the biopsy, a sudden decrease in the tidal volume (from 650 to 550 ml) and a significant increase in peak airway pressures (from 25 cmH2O to 40-45 cmH2O was noticed. At this stage blood pressure rose to 155/108 mniHg, pulse rate went upto 125/minute and SaO2 decreased to 95% from 99-100%. Auscultation revealed presence of wheezes only on the right side. Left side could not be auscultated because of surgical field. A provisional diagnosis of bronchospasm was made and treatment was started with intravenous aminophylline (Bolus of 250 mg plus a continuous infusion at a rate of 0.6 mg/kg/hour). The wheezing sounds diminished and the air entry in the right side appeared to have improved. Still the airway pressures remained high (more than 40 cm ,H20). At this stage, to prevent baroirauma, it was decided to change the respiratory parameters. Tidal volume was decreased from 650 to 450 ml and the respiratory rate increased from 10 to 15 breaths/minute. The airway pressure remained below 40 cm H2O after these changes. Patient remained tachycardic and hypertensive during major part of the surgical time. When the resectionof breast tissue was completed, the surgical field was irrigated with normal saline, air bubbles were detected which were coming Out of a small hole in the

chest wall. There was no surgical emphysema. A diagnosis ofiatrogenicpneumothorax was made and a chest tube was passed before closing the wound. Withthe insertion of the chest tube, the blood pressure, heart rate and airway pressures came back towards normal baseline values. Oxygen saturation improved and became 99-100%. The patient was reversed with standard doses of atropine and prostigmine and extubated. A portable X-rays in the recovery room showed fuily expanded lungs and chest tube in proper position. Patient remained stable haemodynamically inthe recovery room and was shifted to the ward with the chest tube in-situ. She was discharged home 10 days after her stay in the hospital.

Case 2

A 52 years old lady came to the hospital with the complaints of feeling of heaviness, pain and appearance of a lump inherrightbreast. She was admitted for modified radical mastectomy also. Preoperative assessment revealed a non-diabetic, non-asthmatic and normotensive patient with normal ECG, chest X-rays and biochemistry. She was graded as ASA 11 because of her relative obesity (height 153 cm and body weight 70 Kg). A standard oral premedication of tab. midazolam7.5 mgwasgiven2 hours before surgery. Thebasic triple monitoring (non-invasive blood pressure, ECG and pulse oximetery) was started before induction while capnograph was attached aftertracheal intubation. Patient was induced with Thiopental Sodium (4-5 mg/Kg I.V.). Trachea was intubated with a portex Et tube of 7.5 size. Muscle relaxation was achieved with pancuronium bromide (0.1 mg/Kg). Patient's lungs were ventilated with a mixture of Oxygen/N2O (40/60%) and halothane (0.5-1%). Intravenous Pethidine (1 mg/Kg) was given for analgesia. Patient was placed on a control mode of ventilation (IPPV) using Servo-Manley MS 2000 Ventilator. The biopsy of the lump was taken by a resident using Trucut biopsy needle (Baxter Travenol 14 G, 4-1/2 inches long). The surgical procedure for modified radical mastectomy was started and it lasted for three and a half hours while duration of anaesthesia was about four hours. The intraoperative course was unremarkable. She remained haemodynamically stable, normocapnic and well oxygenated (SaO2 98-99%). At the end of surgery muscle relaxation was reversed by giving standard doses of reversal drugs (atropine plus prostigmine) and patient's trachea extubated. In the recovery room pulse oximeter showed a SaO2 of 86-87%, while blood pressure and heart rate was normal. Oxygen flow was increased from 4L/minute to 10 L/minute via face mask. Temporarily the oxygen saturation improved upto 92%. After 15 minutes the saturation again dropped to. 89% and there was no improvement despite an increase of oxygen flow rate to 15L/minute. The patient was stable haemodynamically. On clinical examination, there was markedly decreased air entry on the right side. There was no surgical emphysema. An x-ray chest was advised which showed a right sided pneumothorax. A chest tube was inserted and air gushed out of the tube. Oxygen saturation improved immediately (SaO2 99%). Rest of her stay in the recovery room and hospital remained uneventful.

Discussion

During the last decade the incidence of iatrogenic pneumothomx has increased as a result of invasion of major vessels for monitoring, prolonged mechanical ventilationand external cardiac massage[2]. New methods of diagnostic and therapeutic procedures such as percutaneous liver and renal biopsy, laparoscopy, amniocentesis, has resulied in new causes of pneumothorax[3]. The incidence of pneumothorax is l%during invasive momtonng[4], 14%inthepatientswhowere treated with high levels of PEEP during controlled ventilation[5], while it is 8-10% when percutaneous needle aspiration biopsy of lung was done[6]. The clinical presentation of pneumothorax is dependent upon the patient's level of consciousness, associated cardiopulmonary condition and age[3]. Detection of pneumothomx in the anaesthetized patient is a very difficult task, as it may mimic that of bronchospasm or as non-specific

cardiovascular changes. The frequent earlier signs might be tachycardia, hypotension, increased airway pressures, wheezes, pmgressive hypoxia and cardiac arrhythinias[4,7]. In our first patient, the signs were increased airway pressures, decreased tidal volume, decreased air entry, wheezes, decreased oxygen saturation, tachycardia and hypertension. A misdiagnosis ofbronchospasm was made and treated. Misdiagnosis is not un- common underanaesthesia as beth the conditions may present with similar signs. Delay in diagnosis could be dangerous and may result in a fatal out-come[3]. In our second patient the only sign was decreased oxygen saturation as detected on pulse oximetery in the recovery room. This hypoxia was refractory to the increase in flow of oxygen fmm 4 to 1 5L/minute. Pneumothomx must be considered if there is unexplained cardio-respiratory detenoration4, in the recovery room[3]. Desaturationas detected by pulse oximetery must alert the anaesthetist about the possibility of pneumothorax[4] especially if a pneumothorax prone procedure hasbeendone onthe patient. Inbothourcases small open type pneumothoraces occurred due to the Trucut biopsy needle. Insertion of a chest tube before closure of the surgical wound treated the problem in case 1. In the second patient, no significant problems were encountered during anaesthesia and surgery. The reason for trouble free anaesthesia course might be due to the fact that it was a small open type Pneumothorax. In a report on two cases undergoing laparoscopic fundoplication, workers suspected pneumothomx due to minor reduction in oxygen saturation[8]. They did not face any other significant problems until muscle relaxation was reversed and spontaneous respiration allowed to resume as was the case inQurpatient. Pulse oximeteiy could detect the initial sign of pneumothorax prior to haemodynaniic instability'. In our second patient desaturation and refractory hypoxia without haemody namic instability was due to a delayed pneumothorax. The open type of pneumothorax was probably converted into pneumothorax type 11 due to closure of the external outlet at the muscle and skin level. The discontinuation of IPPV, reversal of muscle relaxation and return of spontaneous respiration might have enlarged the pneumothomx/collapse of the lung due to the changes in the intrapleural pressure from a mean positive to a mean negative. Based onour cases and review of the literature we suggest that if there is unexpected and unexplained desaturation/bronchospasm intraopemtively or in the postoperative period, pneumothorax should be suspected. It should be highon the list of differential diagnosis especially if the patient has undergone a pneumothorax prone procedure. We also recommend that the patients with increased chances of iatrogenic pneumothorax should be monitored for ECG, blood pressure, pulse oximeteiy and airway pressures. Also we should consider avoidance of nitrous oxide in such cases. The biopsy'should be takenby an expert person only. Multiple attempts to get the biopsy by a junior surgeon should be discouraged.

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