One Lung Anesthesia In A Patient With Severely Reduced Diffusion Capacity: Reviewing The Contribution Of Ventilation Perfusion Mismatch

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Abstract

Diffusion capacity of the lung for carbon monoxide (DLCO) is an independent predictor of estimating operative risk for patients undergoing major lung surgery. DLCO may be decreased in a variety of conditions such as non-perfusion of ventilated alveoli (e.g. pulmonary vascular disease), anemia, pneumonia, interstitial infiltrative disorders, alveolar proteiosis, etc. In fact, any process that creates maldistribution of blood flow can be considered to decrease the “effective” alveolar surface area. However, most of the hypoxemia previously attributed to a diffusion block across a thickened membrane is now thought to be due to ventilation perfusion (V/Q) mismatch, as is the case with interstitial lung disease. Ventilation perfusion (V/Q) mismatch can be corrected to a great extent by an increase in the inspired oxygen concentration (FiO2) or an increase in alveolar ventilation. We report the peri operative course of a patient with severely compromised lung function parameters and a very low diffusion capacity [DLCO <3 %]. He was suffering from an advanced interstitial lung disease and pneumothorax and underwent an uneventful decortication and pleurectomy with one lung anesthesia. We reviewed the literature in an effort to analyze the underlying cause of the uneventful intraoperative and postoperative course for the patient.

INTRODUCTION

The term interstitial lung disease [ILD} describes a diverse group of parenchymal disorders that are characterized by a reduction in lung volume and lung compliance, and an increase in lung recoil pressure at a given absolute lung volume. The patients suffering from ILD are known to pose a high risk of perioperative morbidity. Diffusion capacity of the lung for carbon monoxide (DLCO), also known as the transfer factor of the lung for carbon monoxide (TLCO), is a measure of the ease of transfer for carbon monoxide (CO) molecule from alveolar gas to the hemoglobin of the red blood cells in the pulmonary circulation. The transfer of the gas molecule is limited by both perfusion and diffusion. DLCO was first shown in the late 1980's to be a strong independent predictor of pulmonary complications and operative mortality after major lung surgery [1]. But an absolute value of DLCO may not be a good guide for prediction of intraoperative hypoxaemia especially in a patient with ILD. The reduction in DLCO in patients with ILD is primarily due to ventilation perfusion (V/Q) mismatch and the severity of the reduction in DLCO doesn't correlate with the disease stage [2].

CASE HISTORY

A 53 years old, 85 kgs, nomotensive, nondiabetic male patient was admitted in our hospital with history of recurrent left pneumothorax. He had a history of bronchial asthma and advanced ILD on home oxygen therapy at 2 liter/ min for the last 3 years. He was on azathioprine, wysolone, N-acetylcysteine, calcium tablets and nebulization with ipratropium bromide, levosalbutamol sulphate and Fluticasone. One and a half months ago, he had a sudden aggravation of breathlessness without any improvement by oxygen. He was admitted to a local hospital with the diagnosis of left sided pneumothorax. An intercostal drain (ICD) was inserted to relieve the pneumothorax. But, no signs of improvement were noticed and another ICD was put a week later. After ten days, both ICDs were removed and patient was discharged from the hospital.

Four days after discharge, he started having swelling all over the body, with respiratory distress. He was re-admitted to the hospital and an ICD was inserted in the left 2 nd intercostal space. He was redischarged from that hospital with an ICD in situ. He consulted the thoracic surgeon of our hospital for

further management.

On examination, he was awake, alert and oriented, on oxygen therapy by binasal cannula at the rate of 2 lit/min, afebrile, without any pallor or edema. He had clubbing of all the fingers .The ICD was in situ. He had a blood pressure of 122/ 78 mmHg, heart rate 90/min and SpO2 94%. On chest auscultation, widespread crepitations were heard with inspiratory accentuation. Loud P2 was audible. No significant clinical findings were elicited during examination of other organs. On routine laboratory investigations hemogram, LFT and KFT were normal. Protrombin time: 14.8 sec [C=12sec] was mildly deranged, with normal INR 1.2 and PTTK 28 sec. A 12 lead electrocardiogram showed T wave inversion in V1, V2 and V3 leads. Echocardiography revealed normal size of cardiac chambers, no regional wall motion abnormalities, good left ventricular systolic function and minimal pericardial effusion. Arterial blood gas analysis showed hypoxemia [pH 7.4, Pco2 41.6, Po2 46.4, SO2 78.8, HCO3 25.5, BE 1.2 mmol/L]. Pulmonary function tests were grossly deranged. Forced expiratory volume in 1 second (FEV1) 26%, Mean Expiratory Flow [MEF] 31%, total lung capacity [TLC- He] 47% and reduction in DLCO to 3% of predicted. No significant reversibility was noticed after bronchodilator nebulization therapy with salbutamol.

A diagnosis of interstitial lung disease with entrapped lung and large loculated left sided pneumothorax was made. The patient was planned for left thoracotomy, with decortication, lung biopsy and pleurectomy under high risk consent. Preoperatively azathioprine was stopped. In the operation theater non-invasive blood pressure measurement showed a reading of 118/ 80 mmHg and SpO2 was 92% on binasal cannula with 2-liter/min oxygen. The respiratory rate was 22/min. A peripheral intravenous cannula was secured with a 14G cannula and left radial artery was cannulated for continuous arterial blood pressure monitoring. Preoxygenation was done for three minutes (SpO2 improved to 98%) and midazolam 1 mg, glycopyrrolate 0.2 mg and fentanyl 150 ?g given intravenously. Patient was induced with propofol 80 mg, ketamine 15 mg, atracurium 45 mg and isoflurane. Intubation was carried out with a 37 FG left sided disposable (Bronchocath) double lumen endobronchial tube. Tube positioning was checked and adequate lung isolation could be achieved.

After positioning the patient in the right lateral position, thoracic epidural catheter was secured at the level of T5 –T6 interspace. Inj buprinorphine 150 ?g injected epidurally after 3 ml test dose of lignocaine 2% and adrenaline 1:2 lac solution. Left thoracotomy incision was made and one lung ventilation [OLV] was started using 350 ml tidal volume, 16/min respiratory rate and 5 cmH2O positive end expiratory pressure. Peak airway pressure was between 27 to 30 cmH2O during OLV and SpO2 was 99 to 100 % at a FiO2 0.6. Anaesthesia was maintained by oxygen, isoflurane, fentanyl infusion and intermittent doses of atracurium.

Surgeons performed decortication, lung biopsy and pleurectomy. The patient maintained haemodynamic stability with blood pressure between 100 to 130 mmHg systolic without any inotropic support and SpO2 97 to 100%. Muscle relaxation was reversed by neostigmine 2.5 mg and glycopyrrolate 0.4 mg and patient was extubated on table. Post extubation the patient was pain free and had a blood pressure of 130/ 84 mmHg, heart rate 88/min, SpO2 97% and respiratory rate of 20/min. He was shifted to the intensive care unit (ICU) on oxygen by ventimask.

In the ICU, the patient remained haemodynamically stable and was kept on BIPAP intermittently alternating with oxygen by nasal cannula at 4 lit/min. Arterial blood gases started improving in the subsequent days (Table: 1) and he was discharged from the ICU on the third postoperative day (POD) after an uneventful hospital course. Thoracotomy tubes were removed on the fourth POD and he was discharged from the hospital on the 5 th POD with 2-lit/min oxygen via binasal cannula.

Figure 1

Table 1: Trend of arterial blood gas values in the peri operative period

Time	FiO2	pH	PCO2	PO2	SO2	HCO3	Base excess
Preoperative	0.28	7.4	41.6	46.4	78.8	25.5	1.2
Intraoperative	0.6	7.34	35.6	232	97	24.5	0.2
Immediate postoperative	0.6	7.4	47.9	69.0	80.7	25.5	0.5
10 hrs Postop	0.4	7.3	35.7	142	99.5	16.8	- 8.5
1st POD	0.28	7.3	45.8	74.9	94.1	21.8	-4.1
2nd POD	0.28	7.4	49.5	59.5	91.3	26.2	0.7
3rd POD	0.28	7.4	49.0	63.2	92.3	30.7	5.7

DISCUSSION

The best assessment of respiratory function comes from a history of the patient's quality of life [3]. The most useful test of the gas exchange capacity of the lung that also correlates

with the total functioning surface area of alveolar capillary interface is the DLCO . A predicted postoperative DLCO (ppoDLCO) less than 40% correlates with both increased respiratory and cardiac complications and is relatively independent of the forced expiratory volume in 1 sec (FEV1) [4] . Although the ability of the preoperative lung to perform gas exchange (measured by DLCO) may seem to be more important than its mechanical behavioral properties (shown by FEV1) in determining surgical outcome, impaired DLCO and not FEV1, is shown to be a poor prognostic predictor of postoperative quality of life [5] .

However, once the patients with increased risk are defined, assessment of the cardiopulmonary interaction represented by maximum oxygen consumption (VO2 max), is considered the “gold standard” for assessing the two interrelated organ systems [6] . The ILD represents a variety of conditions involving the lung parenchyma – the alveoli, the alveolar epithelium, the capillary endothelium, and the intervening spaces. The reduction in DLCO in patients with ILD is due primarily to ventilation perfusion (V/Q) mismatch [2] . Lung regions with reduced compliance as a result of either fibrosis or cellular infiltration may be poorly ventilated but may still maintain adequate blood flow (i.e. perfusion is intact). V/Q mismatch in these regions may behave like true venous admixture. However, the severity of the reduction in DLCO doesn't correlate with the disease stage [2] . Hypoxemia due to V/Q mismatch generally responds to an increase in inspired oxygen concentration (FiO2) or an increase in total alveolar ventilation [7] as depicted by the formula: Alveolar ventilation = Respiratory rate x Tidal Volume – dead space volume.

Combined anaesthesia [general anaesthesia with thoracic epidural anaesthesia (TEA) by local anesthetics produces a larger increase in the pulmonary shunt fraction during OLV than does intravenous general anaesthesia alone [8] . The probable causes in such reduction in PaO2 in TEA group are attenuation of protective hypoxic pulmonary vasoconstriction (HPV), decrease in heart rate, mean arterial pressure, stroke volume and cardiac output by TEA induced sympathectomy and also of systemic effects of the local anesthetics absorbed (such as a decrease in cardiac output) [9] .

Although HPV is inhibited by all the volatile anaesthetic agents; isoflurane , sevoflurane and desflurane have less inhibitory effect than halothane and enflurane [10] . Propofol 6-12 mg/kg/hr infusion does not abolish HPV during OLV in humans. It causes a greater reduction in cardiac index and right ventricular ejection fraction than isoflurane anaesthesia [11] . Propofol infusion in combination with remifentanyl is probably the technique of choice for a stable OLV with no effect on HPV [12] . In the present case, we did not use both these agents. Remifentanyl was not available at our set up .We used Isoflurane as it has been documented that inhaled anesthetics have no effect on HPV at about one MAC especially when used for short duration.

CONCLUSION

The management of patients for high-risk thoracic surgery remains to be one of the most difficult challenges for the anaesthesiologist. OLV adds to the complexity of the anaesthetic technique. Various investigators have agreed that no single variable has the sufficient power to predict pulmonary complications or death in a thoracic surgical population. Even though some variables correlate with complications; overall, none of them predict pulmonary complications successfully in high-risk patients. Analyzing the nature of the primary lung pathology and manipulating the anaesthetic technique accordingly may improve the perioperative outcome.

Thus proper understanding of the basic lung pathology, its impact on lung function, the corrective nature of surgery and physiology of one lung ventilation is necessary for optimum management of such seemingly complicated pulmonary surgical cases.

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