Hemodynamic Measurements During A Tension Pneumothorax In A Patient With An Acute Myocardial Infarct

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Citation

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Abstract

Pulmonary barotrauma (PB) is a frequent complication of mechanical ventilation. Tension pneumotorax is the most severe form of PB being early diagnosis and treatment important due to the high mortality. Most registered hemodynamic changes consistent with tension pneumothorax have occurred in animals, reports in human beings are rare.

We present a patient admitted to intensive care unit because of cardiogenic shock due to a myocardial infarct who developed a tension pneumothorax on mechanical ventilation, mimicking a myocardial infarct refractory to drugs. Hemodynamic and blood gas changes were observed before and after tube thoracostomy.

INTRODUCTION

Pulmonary barotrauma (PB) refers to the development of extra-alveolar air due to increase of the intrathoracic pressure. Tension pneumothorax (TP), the most severe form of PB, usually occurs in patients with pulmonary necrotizing processes, aspiration pneumonia, chronic obstructive pulmonary disease and those patients requiring ventilatory support (1). Early diagnosis and treatment are important due to the high TP mortality rate which is even higher when it is associated to other severe processes (2). We present a female patient who was admitted to intensive care unit (ICU) because of cardiogenic shock by acute myocardium infarct, who required orotracheal intubation and mechanical ventilation, developing TP.

CASE REPORT

An 80-year old female patient was admitted to emergency room due to a 12-hour dyspnea. On physical examination, her systolic pressure was 65 mmHg, tachypnea and there were bilateral basal rales in more than 50% of the pulmonary fields and no cardiac murmur was heard.

The electrocardiogram showed sinusal tachycardia with subendocardic lesion in the anterolateral leads.

Arterial blood gases showed: pH:7.21, Pco2 60 mmHg, Po2 56 mmHg, HCO3- 24 mEq/l, So2 81%.

Orotracheal intubation and mechanical ventilation with FIO2 of 1.0 was performed. The mode of ventilatory support was controlled mechanical ventilation, tidal volume of 700 ml, respiratory rate 13/min, with peak airway pressure of 45 cmH2O. No positive end-expiratory pressure was instaured. Po2 increased to 183 mmHg and pH became normal.

At the ICU, the chest radiography taken after the intubation only showed a generalized bilateral alveolar-interstitial infiltrate.

Although the patient was given intravenous dopamine and dobutamine reaching 17 m g/Kg/min and 15 m g//kg/min respectively, the systolic blood pressure remained 70 mmHg.

An echocardiogram was performed which only showed septal hypokinesia and moderate contractility decrease.

A Swan-Ganz catheter was introduced via the right subclavian vein to measure pressures. On posterior physical examination, hypoventilation in the left hemithorax was detected, with no subcutaneous emphysema.

Immediatly, a chest radiography was taken showing a left TP and and the thermodilution catheter on the same side. A thoracostomy tube was placed.

The hemodynamic, lactic acid and arterial blood gas data before and after 20 minutes of thoracic drainage are described in the table 1.

Figure 1

Table 1: Arterial blood gas (FIO2 0.8), lactic acid, and hemodynamic data recorded during tension pneumothorax, and after thoracic drainage.

VARIABLES	PNEUMOTHORAX	THORACIC DRAINAGE
pH	7.33	7.37
Po2 (mmHg)	35.1	121
Pco2 (mmHg)	45.8	34
SO2 (%)	62.3	97
SBP (mmHg)	70	153
RAP (mmHg)	12	10
PASP (mmHg)	26	27
PADP (mmHg)	19	14
PAOP (mmHg)	18	10
Lactic acid (mmol/l)	3.3	1.7

SO2: Arterial oxygen saturation.
SBP: Systolic blood pressure.
RAP: Right atrial pressure.
PASP: Pulmonary artery systolic pressure.
PADP: Pulmonary artery diastolic pressure.
PAOP: Pulmonary artery occlusion pressure.

Before of thoracic drainage, cardiac output was not measured due to disease severity. The cardiac index derivaded data and mixed venous gases after thoracic drainage are described in the table 2.

Figure 2

Table 2: Cardiac index derivaded data, and mixed venous gases after thoracic drainage.

VARIABLES	VALUES
CI (l/min/m2)	3.1
SVRI (dyne.sec/cm5)	1831
PVRI (dyne.sec/cm5)	472
DO2 (ml/min/m2)	525
VO2 (ml/min/m2)	131
O2E (%)	0.24
PvO2 (mmHg)	42
SvO2 (%)	74%

CI: Cardiac index.
SVRI: Systemic vascular resistance index.
PVRI: Pulmonary vascular resistance index.
DO2: Oxygen delivery index.
VO2: Oxygen consumption index.
O2E: Oxygen extraction fraction.
PvO2: Mixed venous oxygen pressure.
SvO2: Mixed venous oxygen saturation.

Maximal creatine phosphokinase was 3594 UI/l with MB isoenzyme of 19%. Dopamine could be at first diminished to 8 m g/Kg/min and later both drugs were withdrawn and the patient was discharged from ICU after a few days.

DISCUSSION

Almost all the TP studies reported with hemodynamic monitorization have usually been performed in animals. In 1983, Gustman et al.3 observed that sheep with TP that were breathing spontaneously showed no decrease in the cardiac index or blood pressure, whereas the cardiac index diminished significantly in those which were on mechanical ventilation. This is due to the fact that the increase in the negative intrathoracic pressure swings, responsible for the increased venous return in the spontaneous breathing, were abolished. In human beings, Steier et al.2 described 74 patients who developed tension pneumothorax during the mechanical ventilation, with tachycardia, hypotension, as well as hypoxemia and hypercapnia. In this serie, these authors found an increasing mortality incidence as the delay in having the radiological findings increased, (from 30 minutes to 8 hours) ranging from 31% to 7% when it was a clinical diagnosis.

In 1978 McLoud et al.4 reported on 3 patients (2 on mechanical ventilation) a rise in PADP consistent with the

development of pneumothorax. In 1990, Yu and Lee5 reported an increase only in PADP and they considered it could be due to the transmission of the intrapleural pressure (PPL) to the pulmonary vasculature (interrupting the pulmonary blood flow during the diastole), changing the position of Swan-Ganz catheter (in the same side of the pneumothorax) from the position 3 to 2 of West, and therefore equalizing PPL and alveolar pressure. If PPL exceed PADP, blood flow during diastole ceases and the pulmonary artery catheter diastolic pressure tracing reflects the PPL rather than the pressure in the pulmonary artery. Thus in our case the thermodilution catheter showed the PPL during the diastole without varying the PASP when the pleural pressure was higher than PADP.

To our knowledge, Connolly6 reported the first and only description of a patient with TP in whom all the hemodynamic and blood gases measurements were made. This author described the onset of hypoxemia, acidosis, increased central venous, pulmonary artery (systolic and diastolic) and of pulmonary occlusion pressures, and decrease of the cardiac output, consistent with the pneumothorax but maintaining the blood pressure at the expense of increased systemic vascular resistance. In our case, consistent with Connolly_s report, we found increased RAP, PADP and PAOP, without any increase in PASP, which could be explained by Yu and Lee hypothesis5, since in our patient the thermodilution catheter was placed in the collapsed lung.

Arterial hypoxemia and increased lactic acid levels clearly improved after the pulmonary collapse was solved. In our case, we believe, that these hemodynamic alterations may be due to pulmonary arteriolar vasocostriction secondary to alveolar hypoxia and the vascular compression exerted by the pneumothorax. Systemic hipotension and hipoxemia induced by TP may impair coronary blood flow, decrease significantly the myocardial oxygen supply and increase the myocardial ischemia.

We conclude that the diagnosis of TP remains a special challenge in ventilated patients. The presence of cardiogenic shock non-responsive to drugs in patients with an acute myocardium infarct undergoing mechanical ventilation makes us think not only in the mechanical complications and/or contractility deficit but also in the occurrence of a tension pneumothorax.

ABBREVIATIONS

FIO2 : Fractional inspired oxygen.
PB: Pumonary barotrauma.
TP: Tension pneumothorax.
ICU: Intensive care unit.
SBP: Systolic blood pressure.
RAP: Right atrial pressure.
PASP: Pulmonary artery systolic pressure.
PADP: Pulmonary artery diastolic pressure.
PAOP: Pulmonary artery occlusion pressure.
CI: Cardiac index.
SVRI: Systemic vascular resistance index.
PVRI: Pulmonary vascular resistance index.
DO2: Oxygen delivery index.
VO2: Oxygen consumption index.
O2E: Oxygen extraction.
PPL: Intrapleural pressure.

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