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The acute respiratory distress syndrome after major cardiac surgery - Case report

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Introduction

Acute respiratory distress syndrome (ARDS) is a severe pulmonary disease first described in 1967 by Dr. Thomas L. Petty and colleagues as a syndrome of respiratory failure that presents with progressive arterial hypoxemia refractory to oxygen therapy, severe dyspnea caused by reduction of pulmonary compliance and diffuse bilateral pulmonary infiltrations. This clinical syndrome is characterised by acute, severe and life threatening respiratory insufficiency with high mortality rate [1-3]. The definition of ALI/ARDS was recommended by the American/European Consensus Conference since 1994 (Table 1). The standard definition classifies the patients with bilateral pulmonary infiltrates and arterial hypoxemia, who have the ratio of PaO_2 / FiO_2 less than 300 mmHg. However, if the ratio is less than 200 mmHg, the diagnosis of ARDS can be made. In patients with left heart disease, the pulmonary wedge pressure less that 18 mmHg is a criterion to distinguish ARDS form cardiac failure. Recently, new Berlin classification of ARDS was accepted in 2013, where the term ALI was abandoned and ARDS was categorized [3,4].

Pathophysiological, the arterial hypoxemia is based on accumulation of fluids and presence of noncardiogenic lung edema in distal air segments of the lungs leading to defects of blood gas exchange. There are several clinical disorders associated with the development of ARDS including sepsis, pneumonia, aspiration of gastric content, asphyxia, pulmonary contusion, major non-thoracic trauma, transfusion related acute lung injury, acute pancreatitis, opioid overdoses, drug reactions, amiodaron toxicity, narcotics, embolism and cardiopulmonary bypass [5-8]. The most common clinical disorder associated with ARDS is pulmonary or non-pulmonary infection, mostly pneumonia caused by bacteria, virus or fungi. In patients on mechanical ventilation there is a higher risk to develop ARDS in the presence of ventilator related pneumonia (VAP 48 hours on mechanical ventilation). The second most common reason of ALI development is non-pulmonary infection in addition to pneumonia. Intravascular volume overload, polytransfusions and in general volume resuscitation in shock, trauma or sepsis can secondary result in ALI/ARDS. Many patients with ARDS develop a clinical state like MODS with cardiovascular, renal, liver and hematologic impairments which also can be associated with multi transfusions of blood products and have a higher overall mortality risk [8]. Causal relationship between the transfusion-related acute lung injury (TRALI) is in immunomodulatory response such as patient's reaction to antigranulocyte antibodies, interaction between nonspecific inflammatory mediators as interleukine-8 and tumor-necrosis factor and may result in depressed immune response and infection. New studies report the neutrophile activation by insults as ischemia-reperfusion and transfusions of blood and blood derivatives. Many of studies shown close connection between multiple transfusions and higher mortality rate in critically ill patients [9].

Since the pathology of ARDS was first described in 1977, considerable progress has been made in understanding pathogenesis, pathophysiology and outcome of this acute lung injury. Acute lung injury (ALI) is caused by protein-rich pulmonary edema non-cardiogenic etiology primarily caused by neutrophil and platelet-dependent damage of the epithelial and endothelial membrane of the lung. The most important initial mechanism of ALI/ARDS is based on lung vascular injury, at the level of microcirculation where neutrophils become activated and release mediators of inflammation, cytocines, proteases and pro-coagulant molecules that lead to a loss of endothelial barrier function. Other experimental studies reported the correlation between ALI in patients on mechanical ventilation caused by high tidal volume and airway pressure which results in lung inflammation and endothelial and epithelial mechanical lung trauma. Another important factor is the virulence of some microorganism, especially the virulence of Pseudomonas aeruginosae as well Legionella, in determining the severity of ARDS, but the influence of genetic factor of the patients and the virulence factors as well the cigarette smoke exposure are not yet completely understood and require more research studies [10-12].

ARDS in cardiac surgery

ARDS is an important cause of postoperative respiratory failure with a mortality risk in general population of approximately 40% and 80% in population undergoing cardiac surgery [13]. The pathogenesis of ARDS in cardiac surgery patients is multifactorial. The increased risk of development ALI/ ARDS in these patients group has been associated with the use of cardiopulmonary bypass, need for blood transfusions and large volume overload, mechanical ventilation and major surgical trauma. Risk factors for development of ARDS are poor LVEF, age, smoking history, advanced COPB, complex surgery, emergency CABG, valve surgery, previous cardiac surgery, low cardiac output, arterial hypotension, massive blood transfusions, more than 3

Table 1. ARDS categorization.

ARDS Severity	PaO_2/FiO_2*	Mortality**
Mild	200 – 300	27%
Moderate	100 – 200	32%
Severe	< 100	45%

*on PEEP 5+; **observed in cohort

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packed of red blood cells. Not all of cardiac surgery procedures carry out the same level of risk for ARDS. Many studies show an increasing risk to 16% for aortic surgery, while valve surgery shows 8.1%. The use of cardiopulmonary bypass starts a cascade of systemic inflammatory response with complement activation which activates neutrophyles in pulmonary circulation leading to lung injury. Addition to low cardiac output, splanchnic hypoperfusion and ischemia results in bacterial endotoxin translocations. Heparin inactivation by protamine which is known as a complement activator increases the risk for ARDS in cardiac surgery [14]. TRALI in cardiac patients is defined as the occurrence of hypoxemia and bilateral pulmonary infiltrations after 6 hours after blood transfusions. Improving surgical practice, limiting the time of CPB, intraoperative ultrafiltration have shown benefits in decreasing the risk for ARDS as well the cerebral hypoperfusion consequences in cardiac patients, called postperfusion syndrome or pump head characterised by neurocognitive impairments [15]. Using of fast track extubation protocols in off pump techniques to prevent VAP, using prone position and lung protective ventilatory strategies, restrictive blood transfusions and avoiding fluid overload are approved beneficial therapeutic options for ARDS patients. Lung protective strategies are the gold standard in management of ARDS: Low idal volume ventilation (6 ml/kg of ideal body weight), limitation of plateau pressure to 28-30 cm H_2O, appropriate PEEP levels in BIPAP/CPAP mode are crucial to prevent further lung injury during ARDS [16,17].

Case report

A 51 years old male was hospitalized at Clinic for Cardiac Surgery of Clinical Center University of Sarajevo for operative treatment under the diagnose of three vessel coronary disease, aneurysm of ascendant aorta and aortic valve insufficiency (gr. II/III). In the medical history, the patient informed us about severe long-lasting pain occur four months ago. He also complained over the fatigue in the rest and dyspnea without physical effort, as well as dizziness and swelling of the lover limbs. Lately, time he has had sleep disorder and hard time sleeping on the back. Recently, he was hospitalized at the Cardiology Clinic where cardiac catheterisation was performed and it shown three vessel coronary disease, LAD stenosis of 70%, RCA stenosis 70% and CX stenosis 50%, and echocardiography findings shown a good ejection fraction of the left ventricle of 60%, the left ventricle enlargement, a medium to severe aortic valve insufficiency with AR+3, regurgitation on the tricuspid valve TR+3, also aneurismatic ascendant aorta. The color Doppler of the carotid and iliaco-femoral circulation proved that the arterial vessels had been altered due to atherosclerosis without hemodynamic disorders. Cardiological exams shown unstable angina pectoris, arterial hypertension without a history of diabetic disease. Over the last year he used antihypertensive therapy, antianginal medications and statins. Anticoagulant therapy was stopped by protocol. The patient is a passionate smoker, over 60 cigarettes in last 40 years and obese with a BMI 33.1(obesity class I). The additive EuroSCORE calculated at the admission at our clinic was 9, and the Logistic EuroSCORE (mortality %) was 14.22%.

In the operating room, non-invasive/invasive hemodynamic monitoring was placed to the patient. Arterial line was inserted in the left radial artery. The maintenance of general endotracheal balanced anesthesia by our house protocol was achieved by high dose of opioid, Propofol and inhalatory anesthetic Sevoflurane. We provided complete antibiotic therapy by protocol for cardiac surgery. The Bentall procedure was performed and coronary artery bypass grafting with mamarial artery and venous graft. During a complicated and 8.5 hours, lasting procedure the patient lost a large amount of blood which was been replaced with 4 doses of deplasmated erythrocytes and 5 doses of fresh frozen plasma to substitute coagulation factors and three doses of concentrated thrombocytes to correct the trombocytopenia. The aorta cross clamping time was 183 minutes and the duration of extracorporeal circulation was 254 minutes (Table 2). During the operative procedure, the hemodynamic of the patient was very unstable. The maintenance of the blood pressure was achieved with high dose of catecholamines (Dobutamine, Adrenalin, Noradrenalin) and continuously saline administration. The heart rate was controlled with amiodaron. The patient was admitted to the intensive care unit with high dose of multiple inotropic support, with preserved but hematuric urine output. In the acidobase status we evidenced respiratory and metabolic acidosis (pH 7.20, Base excess -9.9 mEq/l, Blood glucoses > 17 mmol/l, Creatinin 118 umol/l, Lactate 11.2 mg/dl).

Few hours after admission to ICU relative hemodynamic stability was achieved on high dose of inotropic and vasoactive support of Dobutamin, Adrenalin and Noradrenalin. ABS were corrected. Body temperature of the patient began to increase to 39.4°C, sedated on mechanical ventilation. In the following hours, the patient lost another 600 ml of blood in thoracic tubes, which was substituted with 2 doses of DE and 2 doses of FFP. At the first and second postoperative day on the wake up attempt he shown psychomotoric agitation without cognitive contact attained but with preserved motoric function. Arterial blood gas analyses shown a significant decrease of PaO_2 and increase of $PaCO_2$. We set the ventilator mode on supportive BIPAP mode of controlled mechanical ventilation. The respiratory signs on mechanical ventilation shown worsening as well as the blood gas analyses, also creatinine and lactate status. Peripheral oxygen saturation decreased from 85% to 71% on high oxygen supply. The chest radiography shown typical bilateral infiltrations, left side dominated. Inflammatory parameters rose, the C-reactive protein to 337 mg/L, Leukocytes on $2*10^9$/L. Microbiological series of hemoculture, urinoculture, pericardial content taken intraoperatively, were sterile, while in the content of the broncho aspiration was isolated Pseudomonas aeruginosae. Since bacterial infection was detected we immediately decided to treat the VAP by dual antibacterial and antifungal therapy according to the antibiogram with Imipenem, Vancomycin and Flukonazol. Additional dose of FFP was given. As there the ARDS was diagnosed based on continuously worsening of respiratory parameters, low PaO_2 < 27 mmHg, $PaCO_2$ > 80 mmHg, the PaO_2/FiO_2 ratio fell down to the value of 50 / severe type of ARDS/. The chest radiography taken daily shown constant worsening of bilateral infiltrations. On following days, efforts were put into BIPAP mode reprograming on setting for ARDS patients with next modalities: low tidal volume 6ml/kg of the ideal body weight, respiratory rate at 12 to maximum of 17 respirations per minute, pressure control to 30 mbar, the PEEP up to 10, as long patients hemodynamic parameters tolerate this values of PEEP, the FiO_2 was initial 70 % with the tendency to decrease at the time of rising oxygen saturation of patient, PASB 10 mbar and the final setting was to inverse the inspirum and experium in the 3:1 ratio. After next 48 hours on this type of mechanical ventilation the respiratory parameters were on the way of increasing values, PaO_2 > 70 mmHg, $PaCO_2$ 35 mmHg, SaO_2 97%. After cancelation of the sedation the next

Table 2. Laboratory findings.

	Hct (%)	Hgb (gm/dl)	pO_2 (mmHg)	pCO_2 (mmHg)	SO_2 (mmHg)
Initial lab	0.42	138	88.4	36.4	96.6
Operating theatre lab	0.30	90	79.1	50.9	92.6
ICU lab	0.26	87	74.7	57.6	90.5

day, the patient was conscious, carried out simple commands, but still respiratory insufficient to be detached from controlled mechanical ventilation, which we switched to the CPAp mode on FiO_2 0.45. The blood inflammatory parameters were in decline. The patient became afebrile to sub-febrile. Urine output was satisfactory as well the values of arterial blood creatinin and urea levels. Hemodynamic signs were stable. The seventh postoperative day, the patient was completely conscious, carried out all of the commands. He was passive mobilised with the help of physiotherapist, breathing spontaneously with oxygen support by a catheter achieving optimal oxygen saturation SpO_2 98%. Extubation criteria were met and the patient was successfully extubated on the eight days after surgery. The chest radiography shown regression of the infiltrations on both pulmonary side. The following days after respiratory and fully hemodynamic stabilisation without inotropic support and satisfactory values of inflammatory parameters and optimisation of renal values the patient was discharged from the Intensive care unit to postintensive care where he spent next six days fully mobilised until discharged home in good condition with control echo findings which shown good systolic function of left ventricle and mild mitral and tricuspid regurgitation.

Discussion

Acute respiratory distress syndrome (ARDS) is a serious complication in cardiac surgery which despite therapeutic innovation still has high mortality rate. Multiple factors are included in the pathogenesis of this pulmonary disorder. The most important pathological factor for development of ARDS in patients are ventilator associated pneumonia after 48 hours of mechanical ventilation in the ICU as we had in our case where Pseudomonas pneumoniae was diagnosed by microbiological test taken postoperatively. The importance of administering adequate goal directed dual antibacterial therapy according to the antibiogram and advised respiratory therapy with advanced ventilator settings were crucial. As the standard SIMV ventilatory modes in case of ARDS did not generate satisfactory results, supportive BIPAP mode with ARDS patient adjusted parameters give excellent results. As the inflammatory parameters decreased and renal values came in reference range, the conditions were met for improvement of the respiratory status. The modification of BIPAP ventilatory parameters by the latest guidelines for ARDS patients was the crucial moment in the therapy. The extend time of extracorporeal circulation left repercussions on patient's neurocognitive decline as in many studies shown. The neurocognitive status of the patient shows positive response to achievement of cardiorespiratory stabilisation.

Conclusion

Crucial moments in management of ARDS are in time administering of adequate antibiotic therapy in patients who develop early signs of respiratory infection on mechanical ventilation as well as early recognition of ARDS in risk group of cardiac patients. Advised adjustment of mechanical ventilation settings individual to every patient providing concept of the lung protective regime in the early beginnings of ARDS are important steps on the way of clinical recovery and conditioning of blood gas analyses. Providing the hemodynamic stability and optimisation of the acidobase milieu in ARDS patients leads to repairing the cognitive and motor deficit in cardiac surgery patients and creating condition for successful cerebral recovery.

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