MANAGEMENT OF SEPTIC SHOCK AND HYPOXIA INDUCED CONJUGATED HYPERBILIRUBINAEMIA IN CASE OF MAJOR CHEST TRAUMA – CASE REPORT

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Abstract

This case report describes a 50-year-old male trauma patient with multiple crushed thoracic lesions: flail chest, bilateral hemopneumothorax, myocardial infarction and pulmonary contusion. In the ICU he developed ARDS and pulmonary infection which lead to septic shock. Hypoxia due to inadequate ventilation and reduced visceral perfusion due to shock induced elevated levels of conjugated bilirubin. Continuous veno-venous hemofiltration (CVVH) sessions in combination with CytoSorb® filters and MARS® were needed. After surgical fixation of the flail segment ventilatory parameters improved significantly.

Keywords: Hyperbilirubinaemia flail crush myocardial contusion

Introduction

Blunt thoracic trauma is frequent and varies from superficial contusions to crushing of the thoracic wall, compromising completely the respiratory function. Many life-threatening injuries can appear: airway obstruction, hemopneumothorax, injuries of the large vessels, cardiac tamponade or contusion, multiple rib fractures, flail chest and massive pulmonary contusion (Jagodzinski, Weerasinghe, & Porter, 2010) (Yamamoto, Schroeder, Morley, & Beliveau, 2005).

I.
Case report

A 50-year-old man was crushed by a tractor while that had flipped over on a seep road. During ambulance transport the patient had unmeasurable blood pressure, acute respiratory failure followed by ventricular tachycardia (VT) witch responded to resuscitation. At admission to the emergency department, the patient was intubated and receiving mechanical ventilation, oxygen saturation (SpO2) 70%, BP 80/60 mmHg with inotropic support. The first laboratory results were: creatine kinase (CK) 4781 U/L, potassium 7.51 mmol/L, creatinine 1.76 mg/dL, hemoglobin (Hgb) 13.3 mg/dL, hematocrit (Hct) 39.7%, troponin I 0.104 ng/ml, myoglobin > 1000 ng/ml. Electrocardiogram signs of anterolateral myocardial infarction were present. The full-body scan showed bilateral hemopneumothorax, comminuted sternal fracture, multiple bilateral rib fractures with left anterolateral flail segment, massive bilateral pulmonary contusion (Figure 1), bilateral iliac wing and left ischiopubic ramus fractures, retroperioneal hematoma. Bilateral thoracostomy was performed and the patient was admitted to the intensive care unit (ICU). On the 2nd day severe acute respiratory distress syndrome (ARDS) developed (PaO2/FiO2=94.4), on day 3 tracheostomy was performed and for the next 17 days the patient was oncontrolled mechanical ventilation (IPPV – FiO2 70%, PEEP 8 cmH2O) with continuous sedation and curarization. Potassium levels normalized in day 4, CK peaked on day 3 with a level of 13288 U/L and normalizes after 7 days. Intensive fluid resuscitation was continued, maintaining urine output at 2 mL/kg/hr. Serum creatinine levels peaked on the second day (1.86 mg/dL). In the first 21 days a total of 13 packed red blood cells, 19 fresh frozen plasma and 7 platelet units were administered to correct anemia (Hgb 8.5mg/dL, Hct 26%) and coagulopathy. On the 3th day Acinetobacter baumannii and Staphylococcus aureus MSSA have been isolated from the respiratory secretions. Sistemic anibiotics based on antibiogram and local treatment (bronhoscopic bronhoalveolar lavage) was initiated. In the second week the patient developed severe sepsis and septic shock (C-reactive protein 182.4 mg/L) caused by pulmonary infection, with a

progression to multiple organ dysfunction (MODS): creatinine clearance 63 mL/min/1.73m2, urea serum levels above 100 mg/dL. Conjugated and total bilirubin levels rose progressively in the first week reaching a peak of 16 mg/dL (80% conjugated) on day 8. In next 2 weeks the patient received 4 continuous veno-venous hemofiltration (CVVH) sessions in combination with CytoSorb® filters and MARS® (Molecular Adsorbent Recycling System) system, reducing bilirubin levels to 4.04 mg/dL (86% conjugated) in the next 12 days. In this period hepatic transaminase levels were mildly elevated (less than five times the upper limit of normal). Alkaline phosphatase levels were normal, CT and ultrasound excluded obstructive jaundice. On day 19 sedation and miorelaxation was halted and mechanical ventilation was changed to assisted mode (CPAP) for about 48 hour. During this period the patient became respiratory decompensated asociated with hemodynamic deterioration, extreme bradycardia followed by successfully converted VT. On the next day the patient underwent external chest wall fixation using 3 Blades metal plates (Figure 2). After surgery the blood gas values gradually normalized (Figure 3). Starting from day 3 the patient became stabile without inotropic support. Before surgery bilirubin levels rose again reaching a second peak (conjugated bilirubin 10.64 mg/dL) on the day of surgery, needing 3 more CVVH sessions in order to decrease bilirubin levels (Figure 4). On postoperative day 10 mechanical ventilation was changed to T-piece breathing, and on day 14 the tracheostomy tube was removed. The patient is discharged on day 47 after admission to the ICU.

Discussion

Crush injuries occur most commonly after collapse of structures during natural disasters. In our country crush injuries are rare and in most of the cases are caused by traffic accidents. Traumatic crush syndrome is a result of a static compression on skeletal muscles leading to rhabdomyolysis, leading to an efflux of potassium and myoglobin into the systemic circulation, leading to a range of life-threatening conditions including hyperkalaemia and acute renal failure (ARF) (Jagodzinski, Weerasinghe, & Porter, 2010). At admission to the ICU, our patient presented signs of rhabdomyolysis: elevated myoglobin, CK and potassium levels in association with renal dysfunction. Aggressive fluid resuscitationstarted duringrescue and continued at the ED and ICU, played a key role in preventing myoglobin-induced ARF (Parekh, Caro, & Tainter, 2012).

Flail chest is a life-threatening condition which invariably is associated with pulmonary contusion, affecting the alveolar gas exchange (Yamamoto, Schroeder, Morley, & Beliveau, 2005). Conservatory treatment consists in correct air-way management, supplementary oxygen therapy in order to maintain PaO2 above 100 mmHg, positive pressure mechanical

ventilation, analgesia and bronhoscopic bronhoalveolar lavage (Yamamoto, Schroeder, Morley, & Beliveau, 2005) (Jing-Qing Xu, 2015). Surgical fixation of the mobile segment reduces ventilation requirement and intensive care stay (Marasco SF, 2013).

Myocardial contusion or infarction is a rare complication in trauma, but it is responsible for 25% of deaths (Skinner, Laing, Rodseth, L, Hardcastle, & Muckart, 2015). In blunt chest traumas the heart is compressed between the sternum and spine, in most cases causing right ventricular lesions. Left ventricle lesions may occur following coronary dissection. Symptoms are often masked: precordial pain by chest pain due to multiple rib fractures, hypotension and hypoxia may be the consequence of hemodynamic collapse following myocardial lesions or other severe injuries likehemorrhage, pulmonary or neurological injuries (Sybrandy, Cramer, & Burgersdijk, 2003). Therefore it is very important to combine several diagnostic methods, such as myocardial enzymes, electrocardiogram or echocardiography. CK-MB, troponin I and T, are the most frequently used myocardial enzymes. CK-MB is not suitable in trauma because muscle injuries lead to elevation of enzyme levels, especially in lesions of the tongue and diaphragm (muscles rich in MB isoenzyme). Troponin I or T are more specific for myocardial injury, and therefore are recommended for positive diagnosis of acute myocardial infarction (Skinner, Laing, Rodseth, L, Hardcastle, & Muckart, 2015) (Sybrandy, Cramer, & Burgersdijk, 2003) (Kaye & O'Sullivan, 2002). Transthoracic echocardiography is often limited because of painand chest injuries, so if possible, transesophageal ultrasound should be performed (Skinner, Laing, Rodseth, L, Hardcastle, & Muckart, 2015) (Sybrandy, Cramer, & Burgersdijk, 2003) (Kaye & O'Sullivan, 2002). At the emergency room our patient had elevated troponin I levels (0.104 ng/ml) at 3 hours afterthe accident. The ECG showed sinus rhythm, 100 bpm, ST segment elevation in leads DI, DII, aVL, aVF, V2-6. Echocardiography could not be performed because of multiple rib fractures. In subsequent reevaluation on ECG were highlighted QS complexes in leads V1-V6. Echocardiography showed left ventricle apical akinesia and an ejection fraction of 40%. Based on these facts we can say that the patient suffered an acute myocardial infarction. Our patient had no history of ischemic heart disease, but presents several cardiovascular risk factors: abdominal obesity, hypertension, chronic smoking (56 pack-years). Therefore myocardial infarction mechanism is unknown: it may be the direct consequence of trauma or coronary atherosclerotic plaque lesion. In order to clarify the mechanism and determining prognosis, coronary angiography should be performed. During hospital transport the patient presented cardiac arrest due to pulseless ventricular tachycardia. Arrhythmias due to myocardial contusion most frequently occur within 24 hours after the trauma

(Sybrandy, Cramer, & Burgersdijk, 2003). At the same time in case of trauma arrhythmias may have multiple mechanisms.

In the case of trauma patient massive blood transfusion, hematoma resorption and liver dysfunction in general lead to unconjugated hyperbilirubinaemia. In hypovolemic shock or other cases of hypoxia, due to splanchnic circulation changes, the hepatocyte's energetic metabolism is disrupted. The hepatocyte's most energy-dependent process is the excretion of conjugated bilirubin against concentration gradient from hepatocytes into the biliary tract, so it is highly sensitive to hypoxia (Kors, Spoelstra-de Man, & Girbes, 2009) (Nakatani T, 1991). High production of bilirubin through post-transfusional accelerated hemolysis (500 mg bilirubin/L) and hematoma resorption (5g bilirubin/L) will elevate the level of unconjugated bilirubin leading to increased conjugation and excretion overload. Renal dysfunction can increase the level of jaundice due to decreased excretion of conjugated bilirubin (Kors, Spoelstra-de Man, & Girbes, 2009). Pre-existing liver pathology (inactive viral hepatitis B and hepatic steatosis) were factors that most likely contributed to the development of hyperbilirubinemia (Kors, Spoelstra-de Man, & Girbes, 2009). Endotoxins and proinflammatory cytokines released in sepsis intervene in bilirubin excretion by altering the hepatocyte's cytoskeletal arhitecture and downregulating transporter proteins (Nicolas Nesseler, 2012). In our patient's case, elevated bilirubin levels were closely related with septic shock and hypoxia. After MARS® dialysis on day 10, conjugated bilirubin levels droped around 6 mg/dL.

Conclusion

In trauma cases it is important to recognize signs of rhabdomyolysis in order to initiate intensive fluid replacement therapy for prevention of hyperkalaemia and acute renal failure. In patients with flail chest and pulmonary contusion in order to achieve good oxygenation levels early stabilization with plate fixation is beneficial. Myocardial contusion or infarction is a rare complication, but a correct diagnosis and early treatment is vital. Continuous veno-venous hemofiltration (CVVH) sessions in combination with CytoSorb® filters and MARS® seemed to be very helpful in lowering conjuncted bilirubin.

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Figures:

Figure 1

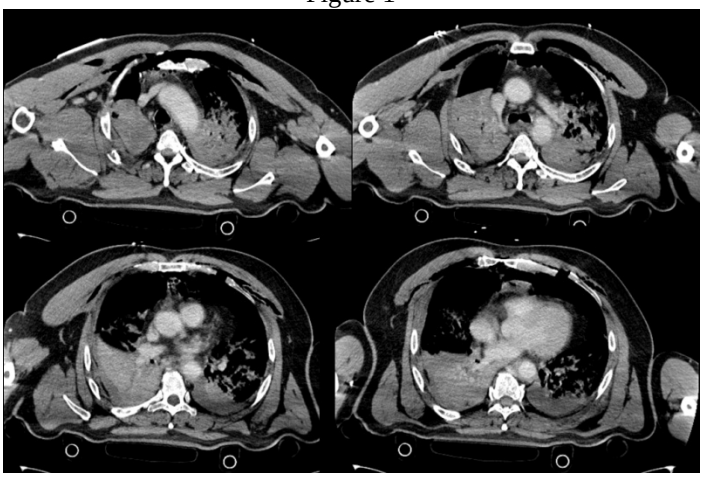

Figure 2

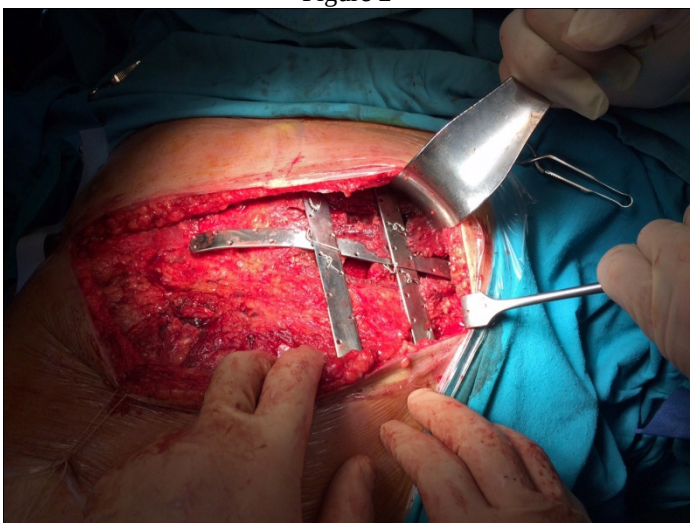

Figure 3

Day	**21**	**22**	**surgery**	**24**	**25**	**26**	**27**
V. mode	CPAP	CPAP	IPPV	BiPAP	BiPAP	CPAP	CPAP
FiO_2	60	100	60	60	60	50	50
pH	7.43	7.22	7.35	7.44	7.47	7.48	7.5
pCO_2	35	43	35	40	41	33	28
pO_2	125	67	89	197	206	175	134
Lac	1.4	2.1	2.5	1.7	1.2	1.5	1.2

Figure 4