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ISSN No. 2394-3971

Case Report

ACUTE CORONARY SYNDROME AS A CAUSE OR A CONSEQUENCE OF A SEVERE THORACIC BLUNT TRAUMA – A CASE REPORT

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Submitted on: February 2017
Accepted on: March 2017
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Abstract

Background: Acute coronary syndrome associated with severe thoracic trauma may be a concomitance, a coincidence or a subsequent event, requiring specific adaptation of emergency management.

The purpose of the study- to highlight the special circumstances of an ACS occurrence in a severe thoracic blunt trauma

Material and method: Clinical case presentation - A 29 years old patient with severe thoracic blunt trauma, secondary brought to a level I trauma center ED. First assessment - serious chest trauma, malignant cardiac dysrhythmias, severe head trauma, spinal cord and pelvic fracture, progressive shock. Increased levels of cardiac biomarkers with atypical progression.

Several hypotheses for the acute cardiac damage etiology considered, as toxic, ACS both causing or following the accident, but, taking into account the kinematics and severity of thoracic trauma, it was also considered the possibility of cardiac contusion occurred. Considering circumstances, the management has been orientated to life-threatening lesions and damage control and advance imagistic to confirm cardiac injury causes.

Conclusions: The strong suspicion for ACS associated with significant thoracic blunt trauma poses questions about his etiology (coronary artery disease, aortic or coronary artery traumatic disrupture), and, subsequently, about possible primary reperfusion procedures required.

The concomitance of brain injury, hemorrhage and pulmonary contusion limited treatment of a potential ACS from CAD, fast volume replacement and ventilation strategies, in the context of shock and risk of secondary brain and spinal cord injury.

HEMS has to be involved with a primary mission to evacuate directly to a level I trauma center this type of patients.

Keywords: acute coronary syndrome, cardiac contusion, thoracic trauma, shock, pulmonary contusion, blunt trauma, HEMS

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Introduction

Car accidents and multiple injured patients is a constant issue of the regular emergency medical services activity. However, generating irreproducible circumstances, kinematics of trauma, the presence of especially associated elements gives uniqueness, profile and joint lesions, evolutionary perspectives and absolutely private management strategy requiring continuous adaptation and specific therapeutic means.

For these reasons, all of the alternatives of generation, association, summative effects, enhancement or removal of the signs and symptoms of certain types of injuries must be considered, investigated, certified or excluded for optimal management.

Material and Methods

We are presenting a clinical case study of a 29 years old male patient, driver, in a high-speed car crash victim - frontal collision followed by rolling, with air bag, without a seatbelt. Two other passengers declared dead on the scene.

The patient was transported by an ordinary ambulance, under superficial sedation, to a level III medical center with altered mental status, GCS 10 to 6 over 2 hours, confused, respiratory rate = 34/', SpO2 = 81%, tachycardia (HR = 145 - 173/'), ET CO2 = 59mmHg, BP = 100/73 mmHg .

After 2 hours it is requested advice transferring the patient to a regional trauma center.30' later, transferred by HEMS to a level II trauma center in GCS 6 points coma, progressive shock (BP - 73/35), paroxysmal heart rhythm disorders (atrial fibrillation with rapid ventricular response spontaneously converted to sinus rhythm, recurrence of atrial fibrillation and association of severe sinus bradycardia with HR below 40 / min). In preparation for evacuation endotracheal intubation practiced under general anesthesia - crush induction (fentanyl, diazepam, etomidate, atracurium), IPPV mechanically ventilation initiated (FiO_2 - 1, RR - 14 breaths /', Tidal volume – 8 ml/kg, 600ml), inotropic and vasopressor support (dopamine 7.5 micrograms / kg. /', increased at 20 minutes to 14 micrograms / kg./'). Subsequently, maintaining a poor hemodynamic condition determinate norepinephrine administration (12micrograms/'). The hemodynamics slowly recovered, but the patient continued to have paroxysmal dysrhythmias.

During this time the patient presents two cardiopulmonary arrests through pulseless activity, (ROSC in 4'and 3'), one of them occurred during air evacuation.

CT scan identified extensive bilateral posterior, apical and pulmonary contusions, in hilum area, cerebral contusions and subarachnoid bleeding, pericardial fluid in small amounts, multiple spinal fractures (C5 with 0,6 cm displacement between C5 – C6, vertebral canal diameter C5 – C6 = 1,22cm; spinal process fracture of C 6 spine; left transverse process fracture of D spine 10,11, L1, 2; unstable, complex body fracture of L1), multiple rib and pelvic fractures (sacrum, iliac and ischiopubic joints).

Blood gases at resuscitation team arrival: pH = 7,184, paO2 = 67,3mmHg, PaCo2= 54,7mmHg, BE = -7,9mmol/l, HCO3 =16,4mmol/l, blood lactate level= 2,7mmol/l, normal level of electrolytes; (at ED Craiova pH = 7,226, paO2 = 88,7mmHg, PaCo2= 41,9mmHg, BE = -4,6mmol/l, HCO3 =18,3mmol/l, blood lactate level= 1,8mmol/l), hemoglobin level 10,22g/dl, after 2h - 8,4 g/dl, GPT = 389u/l, GOT = 479u/l, total amylase level= 150u/l.

FAST examination – very low quantity of free pericardial fluid, no abdominal fluid.

ECG – fast AF - 130 - 170/'., alternating with severe sinus bradycardia, minor right bundle branch block.

Increased levels of CK MB (74,5ng./ml) and myoglobin (over 1000 ng./ml).

Qualitative toxicological examination of saliva - negative.

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The patient was evacuated by air to a – level I trauma center after about 4 hours from the accident.

Results and Discussion

Particular aspects of the case were represented by:

Determinism, evolution, and consequences of cardiac injury were governed by suspicion by both the acute coronary syndrome and cardiac contusion (justified by the kinematics and severity of trauma, lung contusion severity and extension, the presence of pericardial effusion, but unviewed by chest CT). The needs to have emergency angiographic certification was, in the first instance, to establish the etiology of acute coronary syndrome (CAD, or coronary artery traumatic lesion, as a dissection, intimal disruption with subsequent thrombosis, or even aortic disruption with coronary arterial trunk affected – intimointimal intussusception)[1] with primary reperfusion therapy was, in fact, one of the strongest arguments for final disposal to a trauma center with both interventional spinal and cardiology facilities, taking into account the absolute contraindication to anticoagulation. Dynamics of cardiac enzymes suggested the occurrence of a coronary event preceding the car accident taking into account that the increased CKMB level was found in approx. 2,5 h after the accident (rising tendency - maintained for another 5 hours. It should have been expected a rapid increase after the event in case of a myocardial contusion, although it is not excluded the combination of those reflected in this dynamics of enzymes. Myoglobin level had a much larger increase in the first hour, but its specificity is much weaker in this context because of multiple other muscle contusive injuries. On the other hand, cardiac ultrasound did not reveal isolated areas of hypokinesia and the cardiac dysrhythmias were materialized into supraventricular tachyarrhythmias and sinus bradycardia, which would not specific characterize an acute coronary syndrome, but not exclude it. So, it considered that aortic or coronary artery disruption by deceleration should be investigated as a cause of the acute coronary syndrome following trauma.The pericardial fluid sustains that, but equally, cardiac contusion may be involved too. It was taken into account that the origin of dysrhythmias could be induced by drugs, but toxicological examination invalidated this hypothesis. Under these conditions, with myocardial function disturbed by myocardial primary suffering (any of these reasons or a combination of them), by secondary suffering induced by both the neurogenic shock and hypoxemia and also maintained by arrhythmias, the attempt to recover the shock had encountered difficulties in rapid refill[7], requiring administration of blood (2 units) and association of a strong and sustained positive inotropic support. To the level I trauma center both, a critical occlusion on the descending interventricular artery produced by an intimal disruption of the descending coronary artery and a myocardial contusion has been confirmed, but no attempt of reperfusion performed. In chromatography laboratory, the patient reproduced cardiorespiratory arrest, and two attempts of percutaneous intervention under LUCAS device placement failed. Prolonged open chest resuscitation practiced, with no effect. Patient declared dead into the operating room.

The presence of severe head trauma – imposed the absolute need to limit the augmentation of intracranial pressure without compromising cerebral flow pressure; this led to the fact that the rapid filling to be restricted during the fluid therapy, replaced by blood administration, vasoactive support and, ventilatory strategy (hyperventilation for 2 hours, small discontinuous PEEP, RR - 14 -15/'). The secondary brain injury caused by shock, agitation, pain and ventilation dysfunction, on which it was added the post-resuscitation secondary suffering was also an element of

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generating a vicious circle within the brain functioning, and also within the myocardial and respiratory function. The issue of a controlled hypothermia with generic indication in case of a serious head trauma[5], after resuscitation [2,.9] or acute coronary syndrome [6] also required a delicate decision in the context of subarachnoid haemorrhage and pulmonary contusion, thus for the first 6 hours this therapy has not been applied.

The presence of medullary trauma created the possibility to add on top, the component of neurogenic to cardiogenic and hemorrhagic shock resulting in aggravation of the multiple organ suffering by persistent associating hypovolemia and hypoxia. In addition, the danger of excessive fluid replacement, with both pulmonary and cerebral edema risk augmentation was considered.

Pulmonary contusion - bilateral expanded, late compensated (by setting up general anesthesia and ventilatory support), increased the secondary brain and affected the spinal injury prognosis, but also constituted a right ventricular afterload augmentation which affected the effectiveness of volume loading in an attempt to compensate the shock. Also, the need for establishing PEEP in order to optimize the oxygenation of contusion areas was limited on one hand because of the poor venous return and on the other hand was limited by the levels of high intracranial pressure; thus, it has been used discontinuous, low levels of PEEP (4 - 6), as the shock was controlled.

The risk of "occult lesions "- had to be taken into account in the presence of so many injuries generating a complex shock, in the presence of altered mental status[3] to a patient who came from a violent event with two deceased on the scene. The retroperitoneal lesions and the spine lesions were particularly targeted. Systematic assessment, extension and dynamic of the biological and imagistic investigation were means of achieving a definitive sum of injuries.

The issue of general anesthesia. It is of interest for the progress of the case that the establishment of general anesthesia was done very late (after 4 hours from the accident, practically at the resuscitation team arrival), which allowed the development of worsening side reacted events. The rapid sequence of anesthetic induction targeted patient anesthesia (pivot morphine), deep sedation by using drugs that suppress not significant the hemodynamic, followed by relaxation (after tracheal intubation)[11] and setting controlled ventilation. Etomidat[10] was the hypnotic chosen for both the benefits of intracranial pressure control and also for those regarding hemodynamic stability.

Air evacuation decision - required adaptation of ventilatory flow rates and pressures, and also the adaptation of the behavior of shock management and increased intracranial pressure management, all of those being sensitive elements and showing vulnerability of the patient during variation in atmospheric conditions at takeoff, landing and during the flight with an unpressurized aircraft (helicopter). Also the decision of sequential secondary evacuation versus single - phase but with incomplete management has generated controversy, since this air transfer takes above 60', at a critical patient[4], for which there is not a general consensus regarding what is supposed to be the best option in terms of impact on survival, unless it is about a primary evacuation[8].However, the immediate air evacuation from the scene by a high-level competency medical team and directing the patient as a primary intervention to the appropriate hospital (level I trauma center) remain the best option in terms of survival chain, in condition to respect, "golden hour" standards.

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Conclusions

Acute coronary syndrome was, from the beginning, a strong diagnosis suspicion, but, his etiology certification was mandatory in a traumatic context, severe bleeding, and brain injury. In this situation, none of the classical decision as controlled hypothermia, pharmacologic reperfusion, was effective. Even at this age, CAD could be involved, coronary arterial trunk appearance too, toxic circumstances should be excluded, so chromatography are the right emergent decision.

In the same time, taking into account the kinematics and severity of thoracic trauma, myocardial contusion was on the possible diagnostic hypotheses, but it was also considered too, the possibility of an association of acute coronary syndrome, myocardial contusion and the spinal cord damage as associated causes of shock and cardiac rhythm disturbance. The association of brain injury, hemorrhage, and extensive pulmonary contusion limited fast volume replacement and the acceptable ventilation strategies, in the context of shock and risk of secondary brain and spinal injury.

Specifying these contingencies, directing the patient to a highest level trauma center with facilities for both advanced imaging facilities and primary reperfusion facilities combined with advanced management for neurotrauma possibilities would be optimal. We appreciate that the dispatch center performance in appreciating the HEMS primary intervention needs in the field was unsatisfactory and affected the survival chain of this patient.

The main conclusion does not underestimate and carefully evaluating the possibility of a coronary event preceding the traumatic event when shock, electrical instability or enzyme dynamics are not fully corresponding with trauma context his severity or evolution even in the presence of myocardial contusion, but, at the same time do not forget that acute coronary disruption may be a cause requiring not only high-performance imaging to identify but immediate cardiovascular surgery, so HEMS should be from the beginning involved.

Acknowledgments – nothing to declare

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Medico Research Chronicles, 2017