

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Surgery Quiz – Case 18

A 76-year-old male patient was ambulance transferred to the emergency department with undifferentiated ATLS stage 3 shock after an AAST grade I chest injury (non-displaced posterior fracture of the left 9th and 10th rib) sustained from a low height fall. The patient had a previous history of thrombolysed anterior STEMI with associated: (a) Long standing persistent atrial fibrillation treated inter alia with rivaroxaban; and (b) ACC/AHA stage C congestive heart failure. In admission the patient had systolic blood pressure 85 mmHg, heart rate of 122/min, breathing rate of 34/min, confusional to lethargic level of consciousness and lactic acidosis. Physical examination revealed cool skin, mottled extremities, irregular and rapid peripheral pulses, peripheral edema without jugular venous distention or crackles in the lungs and distant heart sounds. Upper airway was managed adjunctively with nasopharyngeal airway, adequate oxygenation was provided with simple oxygen face mask at flow rates of 4 L/min, vascular access was gained by a subclavian introducer catheter and a Foley catheter was placed. 1 L of lactated Ringer's was initially administered with transient response. Anteroposterior chest radiograph revealed no signs of hemopneumothoraces, and cardiomegaly with perihilar haze and bilateral blunting of the lateral costophrenic angles. FAST revealed (a) perisplenic fluid accumulation without the presence of fluid in hepatorenal, pelvic and pericardial spaces, and (b) a smooth, linear disruption

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2019, 36(2):285–286

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to the upper pole splenic echotexture indicating an AAST grade III splenic injury. As the patient was transient hemodynamically stable, a contrast-enhanced thoracic and abdominal computed tomography (CT) performed, which revealed: (a) Cardiomegaly with central pulmonary venous congestion and small bilateral pleural effusions less than 3 cm in anteroposterior depth (fig. 1a), and (b) a smooth, linear low-density upper pole laceration without active extravasation of contrast media and low-density (10–15 HU) perisplenic fluid accumulation (fig. 1b).

What is your diagnosis?

- (a) Exclusive cardiogenic shock
- (b) Exclusive hemorrhagic shock
- (c) Combined hemorrhagic and cardiogenic shock
- (d) Other types of shock

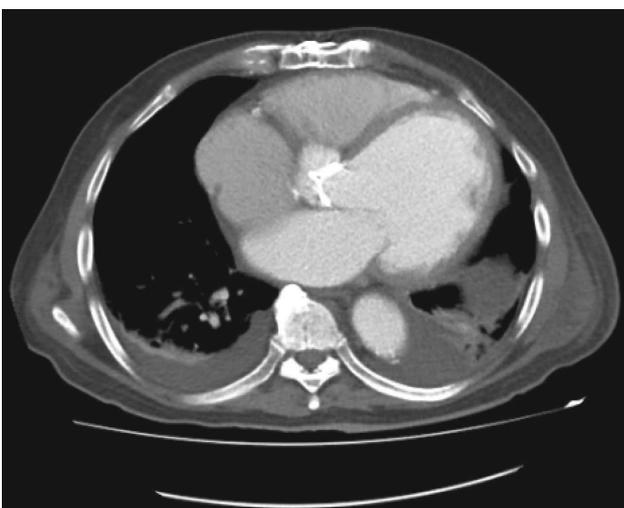


Figure 1a

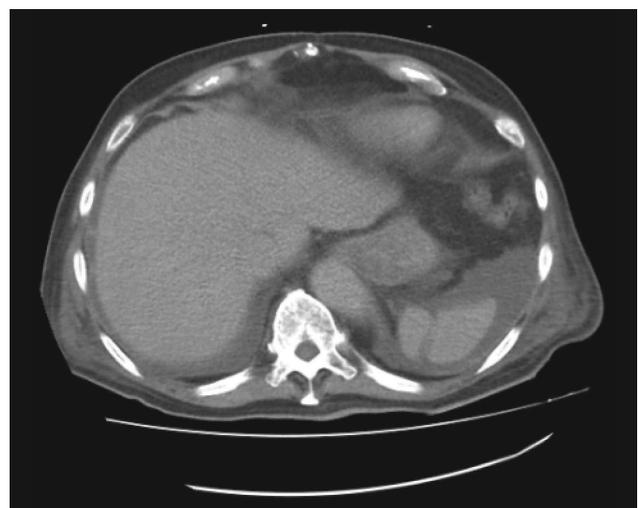


Figure 1b

Comment

Question 1 – What was the etiology of shock? As the patient was on compensated reversible shock, prompt evaluation focusing on rapid identification of underlying shock etiology for definitive treatment guidance is essential. The most prominent diagnoses were cardiogenic shock and combined hemorrhagic and cardiogenic shock. On the one hand, the patient's history of congestive cardiomyopathy and the tachyarrhythmia (atrial fibrillation with rapid ventricular response) were suggestive of cardiogenic shock. On the other hand, spleen injury had to be excluded as the imaging finding of splenic laceration with perisplenic-only fluid accumulation, although hypodense on CT was suggestive of hemorrhagic shock. Diagnostic peritoneal lavage performed to assess the character of the intraperitoneal fluid which was non-hemorrhagic light yellow. Consequently, the key element which led to the beyond-doubt diagnosis of exclusive cardiogenic shock was spleen injury exclusion by diagnostic peritoneal lavage. The imaging finding of spleen laceration seemed to represent an unusual but otherwise normal anatomic variant of a deep long fissure on the diaphragmatic surface of spleen.

Question 2 – What should be the appropriate resuscitation? The aim of initial non acute myocardial infarction cardiogenic shock management is to restore adequate oxygen delivery to peripheral tissues. The key to a good outcome is an organized approach with rapid diagnosis and prompt initiation of pharmacologic therapy to maintain adequate blood pressure, cardiac output and to reverse the underlying cause. All patients require admission to an intensive care setting. Placement of central and arterial lines is required. Invasive monitoring with pulmonary artery catheterization may be helpful in guiding fluid resuscitation in situations in which left ventricular

preload is difficult to determine. Ventilatory and hemodynamic support is critical. Patients should be treated with noninvasive or invasive ventilation with a saturation objective of >95%. Fluid resuscitation should only be indicated in patients with preload insufficiency. In patients with fluid overload and ventricular dysfunction, loop diuretics should be used to restore euvolemic state. A MAP of at least 65 mmHg should be reached using inotropic treatment and or vasopressor treatment, or higher when there is a history of hypertension. Norepinephrine should be used to restore perfusion pressure as first line treatment. Combination of norepinephrine and dobutamine should be used in refractory hypotension. Epinephrine can be a therapeutic alternative to the combination of dobutamine and norepinephrine, but it is associated with a greater risk of arrhythmia, tachycardia, and hyperlactatemia. Finally, all curable causes of cardiogenic shock, such as fluid and electrolyte disturbances, arrhythmias, thromboembolic disorders should be treated.

References

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