

Clinical Notes

Acute right heart strain due to lung contusion

Trauma remains an important problem of modern society. Severe blunt chest trauma may result in lung contusion, flail chest, pneumothorax, hemothorax, hemopneumothorax and multiple rib fractures. Acute right heart strain is well known in acute pulmonary embolism due to blood clot, fat, air, and amniotic fluid or tumor emboli. In the Hillsborough football stadium tragedy in the United Kingdom asphyxiation was described as an additional cause.¹ Electrocardiographic manifestations of acute right heart strain due to lung contusion have not been previously described. We report on an 18 year-old man who was brought to the Emergency Room 30 minutes after a Road Traffic Accident with sudden deceleration injury. He was conscious, alert but irritable (Glasgow Coma Scale 2). Abrasions were present in left ankle, right elbow and laceration in right occipital area. Pupils were equal, reactive to light. No focal neurological signs were present. He did not vomit and did not receive any medication until arrival. Airways were clear and he was breathing spontaneously with a respiratory rate of 22/minute. Pulse rate 115/minute. Blood pressure 130/90 mmHg. Neither cyanosis nor pallor were noted. Jugular venous pressure not raised. Heart sounds normal. No chest wall tenderness present. Chest wall expansion was slightly reduced on the right side. Inspiratory crepitations were present in the right infraclavicular and suprascapular area. Abdominal examination was normal. Chest x-ray showed diffuse patchy to homogenous shadowing in the right upper and middle lobe characteristic of lung contusion (Figure 1). Fracture of the left clavicle at the junction of medial two-thirds with lateral one-third with total malalignment but minimal displacement was present.



Figure 1 - Chest x-ray on admission.

Plain X-Rays of relevant suspicious areas (skull, pelvis, cervical spine, right humerus and abdomen) revealed no abnormality. Brain computerized tomography (CT) showed no skull fractures or intracranial abnormalities. Hemoglobin 7.5 mmol/L (8.3 - 10.6 mmol/L). Leucocytes $7.5 \times 10^9/L$ with normal differential count HCT 0.34. Platelets $350 \times 10^9/L$. Plasma electrolytes, urea, creatinine, prothromboplastin time were all normal. Arterial blood gases (ABG) breathing room air pH 7.44. PaCO₂ 4.0 kpa (30 mmHg). PaO₂ 8.26 kpa (62 mmHg). Saturation 92%. HCO₃ 21 mmol/L. Calculated alveolar arterial (A-o) gradient 4.1 kpa (31 mmHg). Electrocardiogram on admission (Figure 2a) showed QRS axis + 112° with S1 Q3 T3 pattern and anterior T-wave changes consistent with right heart strain. He was started on 1 l/mt oxygen by nasal cannula and 45 minutes later ABG pH 7.41, PaCO₂ kpa (32 mmHg). PaO₂ 12.26 kpa (92 mmHg). Saturation 99%. Six hours later his ECG showed return of QRS axis to normal (Figure 2b) ABG on air pH 7.36, PaCO₂ 4.30 kpa (32.3 mmHg)

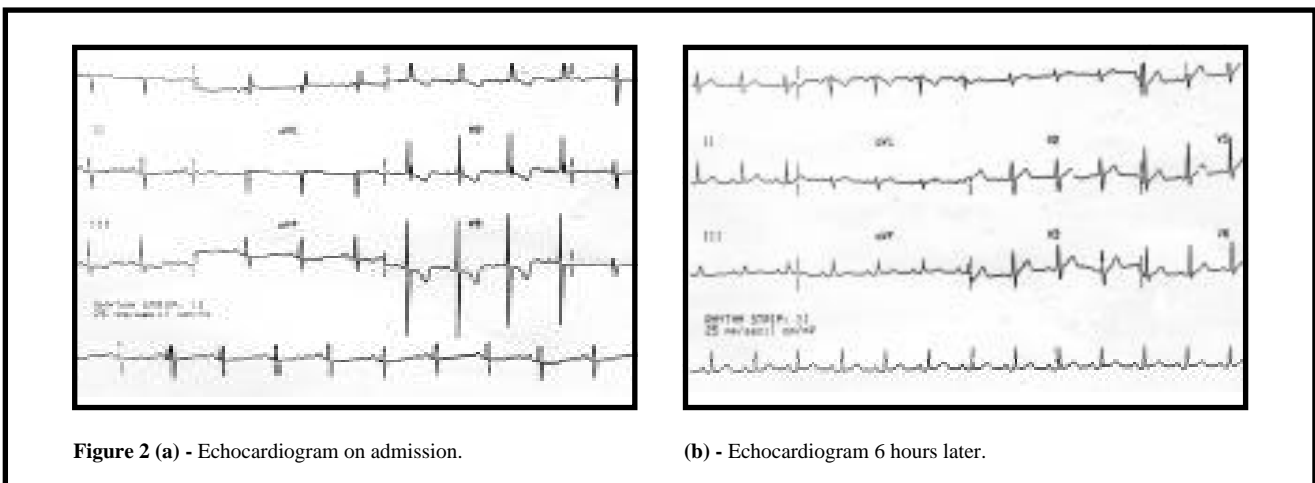


Figure 2 (a) - ECG on admission.

(b) - ECG 6 hours later.

PaO₂ 12.26 kpa (92 mmHg). Saturation 98%. Chest x-ray 24 hours following admission showed nearly complete resolution of lung contusion changes. Serial creatinine phosphokinase, myocardial isoenzyme, alkaline transaminase and lactate dehydrogenase were normal. He received analgesics and the clavicular fracture was strapped. He was quickly mobilized and discharged home 5 days later.

The exact pathophysiology of lung contusion is not well understood. Distention of airways by sudden pressure changes, interstitial shearing forces, spalling effect and disruption of liquid-gas interface are some of the speculated mechanisms.² In diagnosing lung contusion inhalation pneumonia, fluid overload and adult respiratory distress syndrome need to be carefully excluded.³ Computerized tomographic changes are known to precede x-ray changes with 100% pick-up rate for lung contusion at presentation.⁴ However, in our case chest x-ray changes were present on admission and therefore CT was unnecessary. Elevated pulmonary vascular resistance proportional to the degree of intrapulmonary shunting in patients with lung contusion has been shown by hemodynamic monitoring with Swan-Ganz catheter.⁵ PaO₂ and alveolar-arterial gradient were correlated with severity of contusion in the same study. The ECG changes of right heart strain resolved in 6 hours with restoration of normal oxygenation indicating resolution of intrapulmonary shunting and acute pulmonary hypertension. The rapid resolution in 24 hours of the extensive x-ray changes confirms lung contusion. Our patient did not have evidence of fat embolism or myocardial infarction. We believe that significant lung contusion was responsible for acute right heart strain pattern. Tachycardia, tachypnoea

together with such arterial blood gases picture and the ECG changes in the clinical setting of polytraumatized patient may be mistaken for pulmonary embolism. Anticoagulation in this category of patients is unnecessary and may lead to needless complications. We suggest that lung contusion should be considered as one of the additional causes of acute right heart strain.

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