Pneumopericardium and tension pneumopericardium after closed-chest injury

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Westaby, S. (1977). *Thorax*, 32, 91-97. Pneumopericardium and tension pneumopericardium after closed-chest injury. Three recent cases of pneumopericardium after closed-chest injury are described. The mechanism of pericardial inflation suspected in each was pleuropericardial laceration in the presence of an intrathoracic air leak. Deflation of the pericardium was achieved by underwater seal drainage of the right pleural cavity in the first patient, during thoracotomy for repair of tracheobronchial rupture in the second, and by subxiphoid pericardiotomy in the last. Haemodynamic changes after escape of air from the pericardium of the second patient confirmed the existence of tension pneumopericardium and air tamponade.

Pneumopericardium after a non-penetrating chest injury is rare. There are few previous reports of this lesion, and in the presence of additional injury its clinical importance remains poorly defined. This paper examines three recent cases in which operative deflation of the pericardium was performed. In one case release of air resulted in marked haemodynamic changes leading to a diagnosis of tension pneumopericardium.

Case 1

On 21 August 1974 a 37-year-old construction worker fell from scaffolding, bouncing from pole to pole and landing on a concrete floor 70 ft (21 m) below. He sustained multiple injuries. After resuscitation at the nearest hospital, where a pneumopericardium was noted on the chest radiograph (Fig. 1), the patient was transferred to the Birmingham Accident Hospital.

He was fully conscious but cyanosed with a raised jugular venous pressure, a blood pressure of 150/105 mmHg, and a tachycardia of 130 per minute. His many skeletal injuries included bilateral compound ankle fractures, fractures of the pelvis, crush fractures of L1 and L5 vertebrae, and subluxation at the L1/2 disc with cord transection at this level.

Examination of the chest revealed bilateral disruption of the first to fifth costochondral joints with paradoxical movement of the left second and third interspaces and an audible chondral

click. The right chest was resonant with decreased breath sounds. Precordial auscultation revealed a loud splashing sound or 'bruit de moulin'. Chest radiographs demonstrated a large pneumopericardium and a 50% pneumothorax on the right, with no mediastinal shift. Needle aspiration of the pericardium was performed through the fourth left interspace and 40 ml of air with frothy blood was expelled in a pulsatile manner into the syringe. This did not alter the size of the pneumopericardium radiologically. Insertion of an underwater seal drain into the right chest, however, resulted in expansion of the right lung and deflation of the pericardium which was complete in 24 hours.

Intermittent positive pressure ventilation with an initial inspired oxygen concentration of 50% was required to maintain adequate oxygenation, and 31 units of blood were transfused during the first 36 hours.

To facilitate further management, a tracheostomy and internal fixation of the ankle fractures were performed. His later course was complicated by profound hypoxia with chest radiographic changes of bilateral patchy consolidation. Recurrent bouts of paroxysmal tachycardia were controlled with digoxin and practolol, and an ileus, present on admission, resolved slowly over several days. The pleural drain was removed on the sixth day, and after 26 days he was successfully weaned off the ventilator. Unfortunately, the paraplegia showed no signs of recovery, and five weeks after

Fig. 1 Patient 1. (a) A posteroanterior chest radiograph showing the pneumopericardium shortly after admission to the Birmingham Accident Hospital. (b) Decubitus film showing shift of air within the pericardial sac, thus confirming its intrapericardial location.

the accident he was transferred to the spinal injuries unit at Oswestry.

Case 2

On 22 October 1975 a 23-year-old motorist sustained chest and head injuries when his car collided with a tree. Not wearing a seat belt he was thrown forward on to the steering wheel and through the windscreen. Remarkably, he walked one mile (1.6 km) to the nearest hospital where he complained of chest pain and bloodstained sputum.

Physical examination revealed lacerations of the face and scalp, tenderness over the lower third of the sternum, and gross surgical emphysema extending from the upper chest into his neck. He was cyanosed but normotensive and had a tachycardia of 120 per minute. Chest radiographs (Fig. 2) showed a sternal fracture at the junction of the middle and lower thirds and an extensive pneumopericardium and right pneumothorax. The right lung failed to expand after insertion of an underwater seal drain and there was a brisk air leak from the right pleural cavity. He was therefore transferred to the Regional Cardio-Thoracic Centre at Papworth Hospital.

On arrival his condition was found to have deteriorated; the blood pressure was 100/60 mmHg and pulse-rate 140 per minute. Surgical emphysema had spread into the face. On auscultation of the chest a loud splashing sound could be heard with muffling of heart sounds over the precordium. There was a continued brisk air leak from the right pleural cavity. Bronchial rupture was diagnosed and bronchoscopy confirmed a large tear at the origin of the right main bronchus, extending into the carina and lower trachea posteriorly. At thoracotomy extensive mediastinal emphysema was encountered and the pericardium was seen to be bulging and tensely inflated with air. Incision of the pericardial sac resulted in expulsion of a large quantity of frothy, bloodstained fluid under considerable pressure, with a subsequent rise in blood pressure from 110/70 to 130/90 mmHg. On inspection of the deflated pericardial sac, a 3 mm tear on the posterior wall adjacent to the bronchial tear was demonstrated. It was postulated that a valve mechanism had resulted in pericardial inflation and air tamponade. After repair of both bronchial and pericardial tears the chest was closed with underwater seal drainage of the right pleural cavity. The patient continued to have a tachycardia of 120 per minute which gradually settled

over a 10-day period. Air was absent from the pericardium 24 hours after operation.

Case 3

On 29 November 1975 an 18-year-old motorist sustained multiple injuries after collision with an articulated lorry. For an hour he remained trapped and unconscious in the driver's seat and on arrival at the nearest hospital was unresponsive, cyanosed, and profoundly hypotensive. Clinical assessment revealed facial fractures with loss of teeth and bleeding into the mouth and pharynx, bilateral fractured pubic rami, fractures of the left ulna and radius, and several rib fractures on the right side. Chest radiographs showed a small right pneumothorax with diffuse, patchy shadowing throughout the right lung field and a large pneumopericardium. He was resuscitated with blood and dextran and ventilated artificially, and after an initial improvement laparotomy was performed for suspected intra-abdominal bleeding. This revealed a large intrapelvic haematoma related to the fractured pubic rami.

After this there was an improvement in his conscious state but considerable deterioration in respiratory function. Unfortunately, his lungs were ventilated with 100% oxygen for four days, during which time the chest radiograph (Fig. 3) showed increasing patchy opacities in both lung fields, the development of bilateral pneumothoraces, and a progressive increase in size of the pneumopericardium. He became restless, febrile, and increasingly hypoxic and developed surgical emphysema of the chest and neck. At this stage bilateral intercostal underwater seal drains were inserted and he was transferred to the Cardio-Thoracic Surgical Unit at Papworth Hospital.

His condition on arrival was critical, a tension pneumothorax having developed en route due to kinking of the right intercostal drain. He was deeply cyanosed with tachycardia of 140 per minute and a blood pressure of 200/110 mmHg. Precordial auscultation revealed a loud splashing sound audible over the base of the heart, and the heart sounds were muffled. The chest radiograph showed a ground-glass appearance over both lung fields with a large pneumopericardium, mediastinal emphysema, and peribronchial interstitial emphysema. A clinical diagnosis of ruptured bronchus was made but repeated attempts at bronchoscopy were interrupted due to profound hypoxia and slowing of the heart rate. No bronchial tear could be identified. In view of the balloon-like appearance of the pericardium on the

chest radiograph, and a central venous pressure of 18 cm of water, elective drainage of the pericardium was performed via a subxiphoid approach. On incision of the bulging pericardium, a large amount of gas and yellow fluid was expelled under pressure. An underwater seal drain was left *in situ*

through which a brisk air leak persisted for 11 days.

Further surgical intervention was deferred in view of his poor overall condition. His course was complicated by persistent severe hypoxia and decreasing pulmonary compliance requiring ventilatory

Fig. 2 Patient 2. (a) Posteroanterior radiograph taken shortly after arrival at hospital showing a pneumopericardium, pneumomediastinum, right pneumothorax, and surgical emphysema of the neck and chest wall. (b) Detail of the pneumopericardium.

(a)

(b)

Fig. 3 Patient 3. (a) Anteroposterior chest radiograph showing pneumopericardium, pneumomediastinum, and bilateral pulmonary opacities four days after admission to hospital. Bilateral pleural underwater seal drains have been inserted and a tracheostomy performed. Surgical emphysema has spread into the neck. (b) Detail of the pneumopericardium.

tion pressures in excess of 60 cm of water. He suffered recurrent pneumothoraces, bronchopneumonia, and sputum retention requiring bronchoscopic clearance. Chest radiographs showed pulmonary interstitial emphysema with multiple air cysts throughout both lung fields. Two episodes of septicaemia were successfully treated with antibiotics and he was resuscitated after cardiac arrest 18 days after the accident. Despite the severity of the lung changes—for which he received a high dosage of methyl prednisolone—he made a remarkable recovery and was discharged from hospital 12 weeks after admission.

Discussion

Pneumopericardium is a rare sequel to closed-chest injury and still rates highly in terms of radiological curiosity. Shackelford (1931) collected nine such cases from the world literature. Pericardial laceration was present in five patients, suspected in one, but excluded by postmortem examination in three. Cargill *et al.* (1973) reported a further two cases, the first associated with rupture of the thoracic aorta and the second discovered at necropsy with pericardium intact. McCaughey and King (1975) described one case associated with tracheal rupture, and Borrie and Lichter (1974), reporting four cases of pericardial rupture after non-penetrating chest injury, found air in the pericardium of one. The latter authors state that pneumopericardium implies pleuropericardial rupture, a potentially lethal condition which may lead to herniation and strangulation of the heart.

Intrapericardial air in the absence of pericardial laceration, a finding also reported after prolonged positive pressure ventilation (Cohen and Lockhart, 1970) and acute asthma (Toledo *et al.*, 1972), led Rosen *et al.* (1963) to suggest that, following a sudden rise in intrathoracic pressure such as may result from a severe blow on the chest, alveoli may rupture, releasing air into the interstitial tissues of the lung. This air may track along the sheaths of the pulmonary vessels to the mediastinum, neck, and retroperitoneal tissues and even to the pericardial cavity itself. This pathway has been demonstrated by anatomical dissection.

There have been no previous reports of air tamponade after closed-chest injury. The haemodynamic derangements caused by tension pneumopericardium in the presence of multiple injuries requiring resuscitation with large volumes of fluid are difficult to assess. Decreased atrial and ventricular filling, leading to increased central

venous pressure and pulsus paradoxus, have been demonstrated during experimental pericardial inflation (Adcock *et al.*, 1940), in pneumopericardium after positive pressure ventilation in hyaline membrane disease (Sagel *et al.*, 1973), and in pneumopericardium as a sequel to subtotal pericardiectomy (Khan, 1974). Adcock *et al.* (1940) showed that when intrapericardial pressure is raised from 145–265 mm water a proportionate rise in venous pressure occurs. Above this level the rise is no longer proportional and signs of tamponade develop. In order to maintain the circulation, venous pressure must exceed pericardial by 35–40 mm water.

The second and third patients were observed under the relatively controlled conditions of general anaesthesia with positive pressure ventilation. Inspection of the pericardium at operation in both cases revealed that it was bulging and tensely inflated with air. Incision was followed by expulsion of gas and froth under pressure. In the second patient this resulted in marked haemodynamic changes, and there is little doubt that air tamponade existed in this case.

The mechanism of pericardial inflation proposed in the present series is pleuropericardial laceration in the presence of tracheobronchial or pulmonary air leak, thus creating a pleuropericardial fistula. Evidence for this process is the brisk evacuation of pericardial air after intercostal drainage of the right pleural cavity in the first patient, the operative findings of a pericardial tear adjacent to the tracheobronchial rupture in the second, and the persistent pleural and pericardial air leak after surgical drainage of the pericardium in the third patient. As in tension pneumothorax a valve mechanism may develop in a pericardium already distended with air, allowing inflation during inspiration and air trapping in expiration. Positive pressure ventilation must exacerbate this problem if the pericardium is not drained. Anatomically, the right main bronchus and transverse sinus of the pericardium are closely related, and it is not surprising that traumatic laceration in this region may result in communication.

Diagnosis in suspected pneumopericardium is not difficult. The loud precordial splashing or 'bruit de moulin', as described by Bricheteau (1844), with muffled heart sounds is characteristic. Radiography provides confirmation. Cimmino (1967), however, suggested that pneumopericardium was overdiagnosed and mistaken for pneumomediastinum. In pneumopericardium air does not rise above the upper border of the pericardium in the erect film, and decubitus films are useful

show shift of air in the pericardial sac. Mediastinal air will not move in the short interval between films (Fig. 1).

Air tamponade must be suspected if the chest radiograph shows marked ballooning of the pericardium and the venous pressure is raised. In these circumstances evacuation of the pericardial air is advisable, particularly if positive pressure ventilation is required.

The dangers of pleuropericardial laceration must be considered since herniation with strangulation of the heart may occur several days after the initial injury. Bronchoscopy and thoracotomy may be required for diagnosis and repair of bronchial and pericardial tears.

Exposure of the pericardial sac to damaged air passages may result in infection and suppurative pericarditis. The patients described received antibiotics for reasons other than their pericardial tear, and pericardial infection did not occur. In two patients troublesome supraventricular tachycardia followed release of air, and in one this required digitalisation and beta blockade. In the presence of anoxia and cardiac trauma the significance of this arrhythmia is difficult to define.

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