Short Communication

Traumatic Pneumopericardium-Case Report and Review of Literature

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Abstract

Introduction: Pneumopericardium is a rare entity that is defined as the collection of air within the confines of the pericardial sac. It is seen as a consequence of chest trauma or mechanical ventilation.

Presentation of case: A 55-year-old man was admitted to The Canberra Hospital following a collision between a bus and a pushbike. He sustained head, chest and abdominal injuries. An unusual feature of his chest injury was the presence of a pneumopericardium.

Discussion: Pneumopericardium is associated with 58% mortality even in the absence of a tamponade and 37% will develop a tension pneumopericardium. There are no clearly accepted management guidelines for the treatment of this rare condition. Intervention is guided by clinical and radiographic features of haemodynamic compromise with all cases otherwise managed by close observation.

Conclusion: The available published literature on pneumopericardium is scant, and accordingly, incidence rates of this condition remain unclear. In particular, clinical trials investigating the optimal management of this pathological entity are non-existent. It is crucial to recognize that though a relatively benign condition at times, it is a serious complication of trauma with potentially fatal consequences. An all-inclusive approach to management is crucial with judicious intensive care management coupled to early and appropriate imaging and serial reassessments.

INTRODUCTION

Pneumopericardium is a rare entity defined by the collection of air within the confines of the pericardial sac [1]. The description stems from observations of 18th century anatomical pathologists who noted gaseous efflux from the pericardial cavity during autopsy [1]. Clinical descriptions were not availed till the description of the \textit{bruit de Moulin} murmur by Bricheteau in 1844 [1,2]. Pneumopericardium is a complication that has most often been described in the context of chest trauma-blunt or penetrating—and mechanical ventilation [3]. Pneumopericardium presents with distinctive signs and include auscultatory and imaging findings and non-specific electrocardiographic changes [1]. In the absence of tension, symptoms of pneumopericardium are non-characteristic with even asymptomatic presentations noted in literature [1]. Despite the available literature on pneumopericardium, neither incidence rates nor clinical trials are available [4]. The presence of pneumopericardium thus begs two questions-1) Is it an epiphenomenon guiding clinicians to the nature and severity of the trauma? 2) Is there merit in prophylactically decompressing the pericardium?

CASE PRESENTATION

A 55-year-old man was retrieved to the Emergency Department following a frontal impact motor vehicle and pushbike collision. Immediately, apparent multi-system injuries included cerebral contusions, subarachnoid haemorrhage, pneumopericardium, right pneumothorax, multiple rib fractures, bilateral lung contusions and a rectus haematoma. In view of the severity of the chest trauma he was monitored for cardiac contusion.

Reassuringly, he did not succumb to a cardiac arrest, thus not necessitating an emergency thoracotomy on scene or on arrival to the emergency department. No evidence of major vessel injury was noted (Figure 1,2). There was no history of suicidal intent. However, the patient had a history of palpitations that were not previously investigated. On retrieval, he was intubated for a
Glasgow Coma Scale (GCS) of 7. Clinical examination suggested a right-sided pneumothorax and a large bore intercostal catheter were inserted with drainage of air. FAST scanning revealed no evidence of major intra-abdominal haemorrhage. An admission ECG reflected a sinus bradycardia and there was no auscultatory evidence of major pneumothoraces as evidenced in our patient. The gaseous efflux tracks along the aforementioned weakened junctions to terminate in the pericardial reflection. A systematic analysis by Adcock and colleagues in 1940 noted that haemodynamic stability was maintained until the critical mean intrapericardial pressure reached 145mmH2O. Symptoms of cardiac tamponade developed as pressure approached 270mm H2O [1]. Varying studies have described a range of pericardial volume tolerance - 90 to 1000 ml - without clinical evidence of a tamponade provided the rate of insufflation was slow [1,3].

Trauma is seen to be the leading cause of pneumopericardium. Cummings and associates found that trauma accounted for 62% of their 154 cases [1]. Blunt trauma with associated increased intrathoracic pressure resulting in tracheobronchial-pericardial communication is seen in 85% of cases [1]. Other trauma related mechanisms include, penetration along the pulmonary venous perivascular sheaths from ruptured alveoli to the pericardium and pleuropericardial tear [7]. Some of these mechanisms are the likely cause in our patient given the concomitant presence of pneumopericardium and pneumothorax.

Disease processes in contiguous organ systems, closely follow trauma with an incidence of 25% [1]. Erosive sequelae from the gastrointestinal tract or lungs, either from an infective, neoplastic, or post-operative inflammatory change has been postulated. Septicaemic seeding with resultant gas forming pericarditis was evident in 4% [1]. In 9% of patients studied, pneumopericardium was the result of diagnostic and therapeutic interventions [1]. Thoracocentesis, endotracheal intubation and rectal barotrauma are described examples [1,8].

The clinical features of pneumopericardium are varied and non-specific. The classical symptoms are dyspnoea and precordial pain [1,9]. On review of published literature, a culmination of non-specific symptoms such as palpitations, altered phonation, dysphagia, neck crepitus, decreased cardiac dullness, Hamman’s sign - the heart beating against air filled tissues produces a rasping sound synchronous with the heart beat - or rarely, signs of pericardial tamponade have been described [1,4,5,9,10]. According to Cummings and associates, precordial shifting tympany and bruit de Moulin murmur are characteristic findings with the consistent presence of the mill-wheel murmur primarily in complicated pneumopericardium [1].

However, in the presence of only a small amount of pericardial condition and a 58% mortality rate in the absence of a tamponade has been reported [1]. There are no accepted management guidelines for the treatment of this rare condition.

The mechanism of pneumopericardium is variable. Mansfield et al noted a site of potential weakness at the juncture of the parietal reflections and pulmonary veins ostia, where microscopic dissection of air into the pericardial sac occurs [1,5]. The junction is histologically flawed given the absence of collagenous tissue.

The pathogenesis of pneumopericardium has been eluded to by way of the Macklin effect. The latter was derived from the study of feline pulmonology and involves the rupture of alveolar walls as a result of increased intra-alveolar pressure from raised thoracic pressure stemming commonly from high impact blunt trauma [1,5]. The latter culminates commonly in rib fractures and pneumothoraces as evidenced in our patient. The gaseous efflux tracks along the aforementioned weakened junctions to terminate in the pericardial reflection [5,6]. A systematic analysis by Adcock and colleagues in 1940 noted that haemodynamic stability was maintained until the critical mean intrapericardial pressure reached 145mmH2O. Symptoms of cardiac tamponade developed as pressure approached 270mm H2O [1]. Varying studies have described a range of pericardial volume tolerance - 90 to 1000 ml - without clinical evidence of a tamponade provided the rate of insufflation was slow [1,3].

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DISCUSSION

Pneumopericardium can imply a serious life threatening...
air, cardiac examination may be normal, as seen in our patient. A low-grade febrile illness was evidenced in 33% of cases with mild leukocytosis in a further 50% of individuals [10].

Pneumopericardium is confirmed with varying imaging modalities. Computerized tomography has its merits given high sensitivity in detecting pericardial air and its value in the trauma setting (Figure 2-4) [11]. The latter identifies critical pathologies and insults in the circumstance of limited history or restricted examination. It should be noted however, that in the trauma setting, many of pathologies have a temporal relationship and thus require regular reassessments. In the trauma setting it is not uncommon that the patient is haemodynamically unstable and thus their management precludes imaging [2]. A thorough review of the use and value of roentograms was undertaken by Ciammino in 1967 [1]. Lateral films are more sensitive than posteroanterior radiographs (PA) as they allow clearer visualization of the restrosternal accumulation of air [10]. PA typically reveals radiolucency between the left heart border and the mediastinal pleura. Additionally, a continuous diaphragm sign can be seen as is evidenced in Figure (1). Furthermore, radiolucent features such as prominence of the aortic knob, halo and small heart sign can be appreciated. Limitations of roentogram in the trauma setting include the need to take films in a supine position [2,5]. It has been shown that at least 50% of cases will be missed on PA [10].

Ultrasonography (US) in trauma is well established but is directed solely at identifying the presence of pericardial fluid and thus not used in identifying a possible pneumopericardium owing to air being a bad medium sonographically [12]. The value of echocardiography however is formidable owing to its ability to undertake real-time visualization to ascertain the presence of altered anatomy and myocardial architecture and physiology [11]. Trans-Oesophageal Echocardiography (TOE) is seen to be superior albeit invasive to Transthoracic Echocardiography (TTE) but may not be appropriate early on in a trauma resuscitation and assessment [4]. Given the findings of a normal left ventricular size with an ejection fraction of 70% with normal global ventricular systolic and diastolic filling pressures and function with nil sonographic valvular dysfunction, there was no need to follow with the invasive TOE or right heart catheterization as there were no concerns of acoustic shadowing artifact impeding acquisition of images.

Maurer and investigators noted the persistence of bradycardia with non-specific electrocardiographic (ECG) manifestations when a critical intrapericardial pressure is reached [1]. There are no pathognomonic ECG changes in pneumopericardium, although ST-segment or T-wave changes, cardiac axis deviation and dysrhythmias have been described [10]. The presence of pericardial air and hence its interference with electrical conduction results in non-specific ECG changes [10]. The isolated sinus bradycardia noted in our case coupled to significant Troponin I titres with no specific ECG changes highlights the presence of myocardial contusion in addition to the pneumopericardium.

There lacks descriptive guidelines to mold the diagnostic and therapeutic interventions for pneumopericardium patients with no terminal sequelae. The value of prophylactically undertaking a needle or tubal pericardiostomy has never been investigated despite the mortality reported by Cummings and associates to be 58% in the absence of a tamponade [1]. A minute number of case series have advised a conservative approach to managing pneumopericardium patients in the absence of haemodynamic compromise - short hospitalization with the appropriate use of intensive care medicine can limit the progress of complications; regular and timely reassessment; analgesia and resting in a comfortable position and oxygen therapy where indicated [5]. The latter lacks objective efficacy though it’s been advocated by some that the use of high flow 100% oxygen promotes the rapid absorption of free air via a washout of nitrogen [5,10]. 80% of patients who died have been noted to succumb to infectious complications and thus the use of antimicrobial therapy has been advocated [5].

With the aim to monitor progress and note evolving complications, Korean investigators have suggested the importance of follow-up chest radiography within 12 to 24 hours [10]. Review of literature establishes the ability of patients to return to full activity following resolution of signs and symptoms and reassuring investigations - echocardiography and chest radiography [5]. Some researchers have noted the resolution of pericardial free gas within 1-2 weeks [5,10].

CONCLUSION

This case highlights several issues when faced with such a rare thoracic emergency given the absence of standardized literature on its management strategies. Further studies are required to ascertain the value of prophylactic pericardiostomy given the associated mortality.

ACKNOWLEDGEMENTS

The authors have no information to disclose in relation to the use of any writing assistance.

The Authors would like to acknowledge the Radiology Department for the provision of radiological images used in this manuscript.

Written informed consent was obtained from the patient for publication of this case report and its accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

CONFLICT OF INTEREST

The authors have no financial and personal relationships with other people or organizations that could inappropriately influence (bias) this submission.

REFERENCES


