Subendocardial hemorrhages in a case of extrapercardial cardiac tamponade – A possible mechanism of appearance

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SUMMARY

Introduction Subendocardial hemorrhages are grossly visible bleedings in the inner surface of the left ventricle, the interventricular septum, and the opposing papillary muscles and adjacent

columnae carneae of the free wall of the ventricle. These are commonly seen in sudden profound hypotension either from severe blood loss from "shock" in the widest sense and, even more often, in combination with brain injuries.

Case Outline We present a case of a 38-year-old man, injured as a car driver in a frontal collision, who died c. 45 minutes after the accident. The autopsy revealed severe chest trauma, including multiple right-sided direct rib fractures with the torn parietal pleura and right-sided pneumothorax, several right lung ruptures, and a rupture of one of the lobar bronchi with pneumomediastinum, and prominent subcutaneous emphysema of the trunk, shoulders, neck and face. The patchy subendocardial hemorrhage of the left ventricle was observed. The cause of death is attributed to severe blunt force chest trauma. **Conclusion** We postulate pneumomediastinum leading to extrapericardial tamponade as the underlying mechanism of this subendocardial hemorrhage.

Keywords: forensic pathology; subendocardial hemorrhage; extrapericardial cardiac tamponade; pneumomediastinum; subcutaneous emphysema; lung injury

INTRODUCTION

Autopsy reveals superimposed images of both injuries and organ changes generated by the injuries. Our task is to put these images in chronological order, and to make the reconstruction of the injury event. In order do it properly, a good understanding of the pathophysiological mechanisms and injury patterns is required.

Subendocardial hemorrhages are grossly visible bleedings in the inner surface of the left ventricle, the interventricular septum, and the opposing papillary muscles and adjacent columnae carneae of the free wall of the ventricle. The hemorrhages are flame-shaped and confluent, not petechial. They can appear extremely rapidly, within a few heartbeats [1], and can even be found in trauma deaths with nearly immediate circulatory arrest [2]. These are commonly seen in sudden profound hypotension, either from severe blood loss or from "shock" in the widest sense [1, 3], and, even more often, in combination with brain injuries [4]. These hemorrhages were sometimes termed "shock lesions", or named after Sheehan [5].

In the case under review here, we present an intriguing mechanism of subendocardial hemorrhages appearance.

CASE OUTLINE

A 38-year-old man was injured as a car driver in a frontal collision. He died c. 45 minutes after the accident, on the way to the hospital. An autopsy was performed the following day.

The deceased was 176 cm tall and weighed approximately 75 kg. Along with multiple small skin excoriations and bruises of the extremities, the external examination revealed prominent subcutaneous emphysema of the trunk, shoulders, neck and face (Figure 1). The internal examination showed multiple right-sided direct rib fractures (fracture en dedans) with the torn parietal pleura and right-sided tension pneumothorax. The right lung was partially loose and collapsed. On the lateral side of the right lung, there were three ruptures up to c. 2 cm in depth, which corresponded to the fractured ribs. A relatively small amount of free blood (c. 300 ml) was found in the right pleural cavity. There was a rupture in the right lung hilus with one of the lobar bronchus lacerated (Figures 2a and 2b), while the mediastinal connective tissue was crepitant to the touch. Left lung, pericardium, heart and great thoracic vessels were intact and without any visible injuries. Patchy subendocardial hemorrhage of the left ventricle was observed (Figure 3). The autopsy also revealed a longitudinal liver laceration, on the lower side of the right lobe with c. 500 ml of free blood in the abdominal cavity, and a right-sided parietal skin laceration with the isolated contrecoup fracture of the left bony orbit and several left frontal lobe brain contusions. The victim's blood and urine alcohol concentrations were found to be 1.01 g/l and 1.17 g/l, respectively. The toxicological analysis

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Figure 1. Subcutaneous emphysema extended to the face. Eyelids were inflated.



Figure 2. a) The rupture in the right lung hilus; b) The laceration of the lobar bronchus (arrow); RULB – right upper lobe bronchus, RLLB – right lower lobe bronchus.

of the blood, urine, and vitreous humor showed no traces of drugs (head-space gas chromatography).

The cause of death was attributed to severe blunt force chest trauma.

DISCUSSION

Initially, it was thought that subendocardial hemorrhages might be due to rupture of congested subendocardial blood vessels in cases of sudden hypotension: the intraventricular pressure drops precipitously, and the existing blood pressure in the coronary system is then unsupported across the endocardium by the equal pressure within the ventricular lumen, which in turn leads to the rupture of the superficial vessels [1]. Also, mechanical damage to the left ventricular endocardium caused by the vigorous contractions of the relatively empty left ventricle seems to be a possible causative factor in the origin of subendocardial hemorrhages [6]. Recent studies suggest that subendocardial hemorrhages are mediated by hypersecretion of catecholamines [3, 7]. Finally, some animal experiments suggest a combination of these two mechanisms [2]. The subendocardial hemorrhages are a manifestation of a generalized cardiovascular lesion occurring in various conditions which in varying ways lead to systemic hypoxia [8].

In cases of cardiac tamponade, the pressure exerted by the pericardial fluid will eventually equal diastolic pressure within the heart chambers. The first structures to be affected



Figure 3. The patchy subendocardial hemorrhage of the left ventricle

are the right atrium and the right ventricle, where diastolic pressures are normally the lowest. Compression by pericardial fluid interferes with the right atrial filling during diastole, resulting in systemic venous congestion and symptoms of right-sided heart failure. Decreased atrial filling leads to decreased ventricular filling, decreased stroke volume, and reduced cardiac output, which may result in life-threatening circulatory collapse [9]. However, cardiac tamponade can also occur due to the extrapericardial compression of the heart. The most common cause of this are large pleural effusions. The increased intrapleural pressure is transmitted to the pericardial space and impairs ventricular filling, thus mimicking the hemodynamic abnormalities of cardiac tamponade. There are case reports of extrapericardial cardiac compression resulting from anterior mediastinal hematomas, masses, ascites, and pneumomediastinum [10, 11, 12].

Pneumomediastinum or mediastinal emphysema is the presence of extra-alveolar air in the mediastinum. Most commonly, free air leaks from ruptured trachea or bronchi, dissecting along the bronchovascular sheaths towards the mediastinum, and eventually into the subcutaneous tissue. Posttraumatic pneumomediastinum can originate from the mediastinal propagation of subcutaneous emphysema, or due to a direct air leak from a tension pneumothorax through a small tear in the mediastinal pleura [13]. Mediastinal emphysema is accompanied by massive subcutaneous emphysema, and vice versa in a quarter of cases, and it can compress the venae cavae, causing extrapericardial tamponade and shock [13]. The tension pneumothorax produced by bronchial rupture also compresses the large veins with the same effects of extrapericardial tamponade as mediastinal emphysema [13].

In the presented case, the laceration of lobar bronchus (Figure 2b) is followed by right-sided tension pneumothorax, mediastinal and subcutaneous emphysema, all exacerbated by cardiopulmonary resuscitation attempts. The tension pneumothorax and mediastinal emphysema compressed the large heart veins, causing extrapericardial tamponade. This condition led to decrease of atrial and ventricular filling, stroke volume, and reduction in cardiac output and shock, which most probably was the cause of subendocardial hemorrhage that occurred. Also, the blood in the right pleural space, as well as in the abdomen, would have contributed to the shock, primarily caused by pneumomediastinum.

It is possible that the rise of coronary pressure due to the excessive endogenous secretion of the catecholamines stemming from the accident, administration of adrenaline during resuscitation attempt, as well as coincided abrupt

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drop of intraventricular pressure due to acute extrapericardial cardiac compression, and relatively mild traumatic brain injury, could have been the contributing pathophysiological mechanisms of subendocardial hemorrhages in the presented case.

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Могући механизам настанка субендокардних крварења код екстраперикардне тампонаде срца – приказ случаја

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КРАТАК САДРЖАЈ

Увод Субендокардна крварења у пределу леве коморе најчешће виђамо у случајевима где је узрок смрти акутни хиповолемијски шок – пре свега искрварење, или повреда мозга. Наравно, и неке друге повреде могу бити узрок настанка ових крварења.

Приказ случаја Ради се мушкарцу старом 38 година, који је повређен као возач у чеоном судару. Обдукцијом је установљена тешка повреда грудног коша: серијски прелом десних ребара и руптура десног плућног крила и бронха,

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са развојем тензионог пнеумоторакса, пнеумомедијастинума и поткожног емфизема. Обдукцијом су установљена и субендокардна крварења у левој комори.

Закључак Могући патофизиолошки механизам настанка ових крварења у срцу, у конкретном случају, јесте и настанак и развој ектраперикардне тампонаде срца.

Кључне речи: судска медицина; субендокардна краварења; екстраперикардна тампонада срца; пнеумомедијастинум; поткожни емфизем; повреда плућа

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