Cardiac lesions associated with cardiopulmonary resuscitation

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Abstract: Cardiac arrest is a major cause of morbidity and mortality across the world despite the fact that significant advances were made in basic and advanced life support techniques. Cardiac lesions during CPR are rare, and usually are not involved in thanatologic chains. They can however lead to severe or even lethal complications and may be difficult to differentiate from non-iatrogen trauma, especially in traumatic deaths. We present in this article four cases of cardiac lesions associated with resuscitated cardiac arrest, discuss their forensic significance and review the most important iatrogen cardiac lesions associated with cardiopulmonary resuscitation.

Keywords: cardiac contusion, myocardial lesions, cardiopulmonary resuscitation

Cardiac arrest is a major cause of morbidity and mortality across the world despite the fact that significant advances were made in basic and advanced life support techniques. Even though cardiopulmonary resuscitation has an overall success rate of 30-40%[1], over 50% of all resuscitated patients die before leaving the hospital, usually due to cardiovascular or CNS complications[2]; many successful resuscitations are in turn associated with a series of post cardiac arrest syndromes (PCASs) or iatrogenic traumas determined by the CPR technique.

According to Nolan et al there are three main types of post-cardiac arrest syndromes: (1) post-cardiac arrest brain injury, which encompasses impaired cerebrovascular auto-regulation, cerebral edema, and postischemic neurodegeneration and is clinically associated with coma, myoclonies, seizures, variable degree of cognitive dysfunctions, ischemic or hemorrhagic stroke, persistent vegetative state or brain death; (2) post cardiac arrest myocardial dysfunction, which encompasses global hypokinesis, reduced cardiac output, and ischemic heart disease and is clinically associated with acute cardio-circulatory insufficiency, hypotension, arrhythmias; (3) systemic ischemia/ reperfusion, which encompasses systemic inflammatory response syndrome, impaired vasoregulation, altered coagulation, adrenal suppression, impaired tissue oxygen delivery and utilization, impaired resistance to infection and is clinically associated with sepsis, hypotension, hyperglycemia, pyrexia, acidosis, and organ dysfunction[3-8]. Iatrogenic trauma may add to the severity of

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the PCASs but usually they are mild and, if found at the autopsy, have little relevance in tanathogenesys. Their medical-legal importance resides in the need of a differential diagnosis with either non-iatrogenic trauma, especially in violent deaths, or morphologically similar non-violent pathologies. [4-6,9-19]. CPR trauma can be divided in resuscitative injuries related to ventilation procedure (RIVP) and due to chest compression (RICC) [15]. RIVPs are usually associated with respiratory tract lesions (larynx, pharynx, trachea, lungs), but stomach lesions or bruises/abrasions on the face and neck can also be found. RICC are less often described, affecting the face (petechiae), eye (retinal hemorrhages, petechiae on the eyelids and conjunctiva), brain (subarachnoidal hemorrhages), skeletal structures (sternum and ribs fractures, bone marrow embolism), heart (cardiac rupture, myocardial hemorrhages, epicardial petechiae, air invasion into ventricles, coronary lacerations and hematomas), liver and spleen (ruptures, hematomas), hemo- or pneumoperitoneum, etc. We present in this article four cases of myocardial hemorrhages associated with resuscitated cardiac arrest and discuss their forensic significance.

Cases presentation
Case 1

A 32 years old female without a positive personal history of either cardiac or non-cardiac pathologies collapsed at work. The ambulance crew found it in ventricular fibrillation and successfully resuscitated her. She was admitted with the diagnosis of post CPR status, cardiac arrest under mechanical ventilation, and survived for another four days with a GSC=4. At the autopsy we found a left forth costal fracture and two contusive myocardial areas of 6/4/0.4 cm in the anterior wall of the left ventricle extended in the interventricular septum (Figure 1) and in an anterior left papillary muscle. A few epicardial petechiae were also identified. Histological examination revealed multiple myocardial contusions on the anterior and anteroseptal walls of the left ventricle (Figures 2, 3) and one papillary muscle, a small fibrino-hematic thrombus in the atriventricular node artery, and myxoid degenerescence of the mitral valve (Figure 4).
Case 2

A nineteen old female, was found at home in cardiac arrest; after a successful resuscitation she was hospitalized in a third degree coma. She was previously healthy but had a history of birth control pills for a couple of months before the event. She was transferred to an emergency hospital where she died seven days later. During hospitalization the evolution was extremely severe, with hypoxic encephalopathy, acute renal failure, hepatic dysfunction, acute cardio-circulatory insufficiency on maximal vasopressor and inotrope support, and CID. The autopsy proved inconclusive for the cause of death. Gross pathological examination revealed a partially organized thrombus in the left auricle, dated about seven days before death (Figure 5), a subendocardial hemorrhagic necrosis of the left ventricle, occurring most likely two days before death (Figure 6,7), a few epicardial petechiae, a pulmonary artery thrombosis dated also about two days before death, bronchopneumonia and renal infarct. All except the auricle thrombosis and possibly the epicardial petechiae were considered to be terminal events, associated with MOSF and CID. The auricle thrombosis was considered to be either the cause of the initial cardiac arrest or a resuscitation - related lesion. As the patient hadn’t a personal history of atrial fibrillation, chronic mitral valve pathology or other predisposing cardiac, hematological or thrombophilia disease, the cause of the thrombosis was related to CPR.

![Figure 5. Thrombosis of the left auricula (mixture of fibrin, erythrocytes and leucocytes), HE, 5x](image1)

![Figure 6. Extensive subendocardial hemorrhagic necrosis](image2)

![Figure 7. Extensive subendocardial hemorrhagic necrosis, HE & Lie, 5x](image3)

Case 3

A 28 years old male was admitted to the hospital with the diagnosis of acute hemorrhagic and necrotic pancreatitis. During hospitalization he developed a deep coma (GCS=3), fever, acute respiratory, renal, and cardio-circulatory insufficiency. On the seventh day, after an unsuccessfully resuscitated cardiac arrest, is pronounced dead. Coagulative status was slightly decreased but fibrinogen levels were normal until the day he died (332 mg/dl). During the necropsy cardiac examination revealed multiple subepicardial petechiae with the absence of petechiae in other locations which made us conclude they were CPR-related.

Case 4

A 57 years old male was hospitalized for undetectable arterial tension, dizziness, and severe altered mental state. The symptoms had started eight days before the admission. Clinical examination revealed
peritoneal irritation, diminished vesicular murmur, breathing difficulties, gastric stasis. Acute abdomen is suggested by clinical, radiological and echographic investigations, but the patient developed bradycardia and asystole before a surgical intervention could be performed. The patient was resuscitated but developed a second, irresuscitable cardiac arrest after one hour and a half. Gross pathological examination found the cause of death to be acute hemorrhagic and necrotic pancreatitis. Secondary to CPR attempts the patient had costal fractures on the right (5th-7th ribs) and a hemorrhagic myocardial area in the of 1/0.8/0.8 cm below the right side coronary artery at about 1 cm from the origin caused by the CPR attempts.

**Discussions**

Cardiac lesions during CPR are rare, and usually not involved in thanatologic chains. Krischer [20], on a study of 705 deaths after CPR found cardiovascular lesions to occur in 10.6% of cases (a total of 72 lesions), the majority of which were hemoperitoneum (8.4%), epicardial hematoma (2.7%), and myocardial hemorrhages (1.3%), including ventricular subendocardial contusions and atrial lesions. He also noticed in six patients great vessel complications – bubbles in the vena cava, laceration of small abdominal vessels, aortic adventitial hemorrhage, superior vena cava perforation and inferior vena cava contusion[20].

Hemopericardium is, according to Krischer, the most frequent cardiac complication following CPR, and may be caused by small myocardial contusions, tears in coronary branches, rib fractures with posterior displacement tearing the pericardial sac, etc. The most frequent cause however seems to be the administration of cardiac injections during CPR. Davison found, from 53 patients which suffered CPR, 17 cases with pericardial effusion during echography and 8 (out of 28) cases of hemopericardium during the autopsy[21]. Usually the hemopericardium in these cases is small, only rarely being cited quantities of over 100 ml[21,22]. Fatal cases are also cited but they are extremely rare [23]. Schonefelder for example found four fatal hemopericardium cases out of 340 autopsies with a history of intra-cardiac injections as a resuscitative measure[23].

Petechiae are small hemorrhages produced by rupture of small vessels (usually venules)[24]; epicardial petechiae are usually caused by abrupt increases in intravascular pressure with subsequent over distension and rupture. Hashimoto found them in 40% of autopsies in cases which previously suffered CPR [15], located as follows: 52% on the anterior side of the heart, 17% on the posterior wall and in 30% on both the anterior and the posterior side of the heart. Epicardial hematomas are rare [20,25-28] and usually determined by the force applied by the ribs/sternum moving towards the epicardium, but may be caused by cardiac injections as well[21]. Myocardial contusions and hemorrhages are usually caused by a direct mechanism, as described above. There are however cases in which the water-hammer effect may play an important role [29,30], as are the eight cases described by Hashimoto [15] with septal myocardial hemorrhages, and possible subendocardial hemorrhages. CPR with associated myocardial contusions seem to have a poorer prognosis than the ones without myocardial contusions [15] as it is leads to additional hypotension, arrhythmias, hypoxia, etc. [30]

Cardiac rupture or laceration occur only exceptionally [1, 15, 22, 25, 31-35], and are usually associated with a preexistent myocardial pathology [15]. A greater prevalence seems to be associated with open chest direct cardiac massage [36] – Baldwin et al found them in six out of 16 cases analyzed. In patients without preexistent myocardial pathology the rupture/laceration may be caused by a fractured sternum/rib [13,37-39], or by an acute hemorrhage when there is a decrease in blood quantity in the heart chambers, permitting a stronger compression of the heart between the anterior thoracic wall and the vertebral column[15]. Other possible mechanism for CPR-associated right ventricular rupture is the increased pressure in the right cardiac chambers when compressed pulmonary valves affect the blood flow during resuscitation[15]. Increased risk for CPR-associated cardiac rupture seems to be associated with the use of active compression-decompression devices like the Cardio-Pump® [40,41]

Valvular lesions are only exceptionally described. Buschmann et al [41] for example found a rupture of a highly calcified mitral valve ring after CPR with subsequent migration of the calcified fragments into the myocardium and cardiac tamponade. Chatson described the development of a ventricular pseudoaneurism associated with CPR six weeks after mitral valve replacement [42] Gerry et al. described two cases of rupture of a papillary muscle followed by a tricuspid insufficiency following CPR [43].
Cardiac thrombosis associated with CPR was previously described by Milstoc and Berger[44]; they described a patient with cor pulmonale which had a thromboembolic mass in the right atrium, right ventricle and pulmonary artery. The thromboembolic mass contained bone marrow emboli and bone spicules probably due to a fractured rib after external cardiac massage. Barringer et al described a bone marrow embolus adherent to a mural thrombus in the right atrium after CPR.[45] Andriessen et al. described a CPR complicated with cardiac air emboli and a vena cava thrombosis which occluded the entry of the right ventricle.[46] As in the above described situations our case had a predisposing condition (the use of birth control pills) which could favor an increased thrombotic state. The particularity of our case is however the lack of direct or local causes for the development of the atrial thrombosis.

Another cardiac structure which is sometimes affected by CPR but is often overlook in routine autopsy cases is the cardiac electrical conduction system (ECS). Rossi et al found a hemorrhage in the bundle of His associated with CPR and suggested as a possible mechanism an excessive squeezing of the heart between the sternum and the vertebral column[47]. Frink and Rose described direct blunt injuries of the coronary arteries or the ECS in 44% (35 cases) of patients who received CPR prior to their death[48]. Nishida found lesions of the ECS in seven out of 80 patients, suggesting their frequency is much higher than previously suggested[49]; the distribution of hemorrhages in his study is as follows – six cases had hemorrhages in the conduction tissue (three had hemorrhages in the sinus node, one had a hemorrhage in the proximal bundle of His, two in the distal left branch), and one had a focal dissection of the atrioventricular node artery.

The histological pattern of CPR related hemorrhages is similar to the classical traumatic cardiac contusion morphology, and is very similar to the one found in myocardial infarction, with a more preeminent and individualized hemorrhagic area. Histological examination reveals contraction band necrosis, myocardial cell segmentation, bundles of contracted myocardium alternating with bundles of distended myocardium, a widening of the intercalated disks, granular myocardial disruption [30], fibrillar eosinophilia, elongated nuclei and sarcomers, depleted troponin I, C, and myosin [50].

Acknowledgements

This work was supported by CNCSIS –UEFISCSU, project number PNII – IDEI 2642/2008

References

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