G25 Adipositas Cordis and Iatrogenic Death: Fatal Complication or Medical Error?

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After attending this presentation, attendees will learn some basic information regarding the risk of pacing maneuvers, and the role of fatty infiltration of the right ventricle in causing delayed cardiac laceration.

This presentation will impact the forensic science community by discussing the utility of an integrated analysis of clinical, radiological and histological data for identifying any eventual medical error during pacing maneuvers.

It is well-known that the hearts of most adults in western countries contain varying physiological amounts of fat, found mainly in the subepicardial region of the anterolateral wall of the right ventricle. In the normal heart the boundary between the inner myocardium and the outer subepicardial fat is usually distinct, although a slight fuzzy border may be observed. On the contrary, in the fatty infiltration of the right ventricle irregular islands of adipose tissue may extend from the epicardium to the endocardium with the interposition of only few muscle fibers.

In such cases the risk of cardiac rupture after myocardial infarction as well as the risk of ventricular laceration after cardiac surgery is notably increased.

The case of a 70-year-old woman who died of an acute pericardial tamponade due to a delayed laceration of the right ventricle after pacemaker implantation is reported. The autopsy finding of a severe fatty infiltration of the right ventricle, its causal role in determining the fatal pericardial effusion and the legal responsibilities of the physicians who performed the implantation are critically discussed under a forensic point of view.

Myocardial perforation by pacing electrodes or Implantable Cardioverter-Defibrillator (ICD) leads is a well-known and documented complication, occurring at a rate of about 0.4-2.0%. The largest part of the injuries are clearly related to the impacting maneuvers peculiar to the manipulation of pacing catheters and are recognized intraoperatively or in the early postoperative period. Even if the complication is misdiagnosed or the rupture is delayed, due to the “self-sealing” properties of the myocardium and to the fact that generally the lead closes up the ventricular perforation (avoiding a massive bleeding), life- threatening pericardial or pleural effusions are rare.

In our case, the presence of an extended fatty infiltration of the lateral wall of the right ventricle (35% of the myocardium was displaced by adipose tissue) forced the operator to move the implantation lead back and forth to obtain a valid electric signal. In that manner, because of the enhanced fragility of the right ventricle, the surgeon produced three micro-perforations, one of them localized on the lateral wall above the insertion of the anterior papillary muscle, and two of them localized near the apex. All the perforations were of small dimensions and had “self- sealed” soon after the lead damage because the echocardiography performed thirty minutes after the implantation did not reveal pericardial effusion and the patient was totally asymptomatic during the afternoon and the evening of the operative day.

Clinical and radiological data suggest that the fatal ventricular laceration has formed during the late evening or night. Indeed, the granulocyte infiltration along the margins of the tear dates the lesion between four and six hours before death.

Considering the size and morphology of the injury as well as the extensive transmural fatty infiltration observed in that point of the ventricle, the most probable explanation is that the micro-perforation, produced by the lead, progressively enlarged due to the presence of multiple adipose cells that reduced the adhesion forces between the myocytes. Therefore, the fatty infiltration not only favored the lead- related injuries, but also played a key-role in causing the rapid and fatal pericardial bleeding.

Regarding the site and method of pacemaker implantation as well as the post-operative clinical monitoring, it is believed that several questionable choices have been made.

Attempting multiple maneuvers (i.e., making several punctures) to find a site to place an active fixation lead at the apex is extremely dangerous, above all if the patient suffers from a fatty infiltration of the right ventricle.

Moreover, even if the echocardiography performed thirty minutes after the intervention did not reveal any pericardial effusion, considering the complicated implant procedure, the patient should have been cautiously monitored in a coronary unit, instead of being transferred to an internistic department. A proper postoperative surveillance would have prevented the fatal outcome with a high degree of probability. **Fatty Infiltration of the Right Ventricle, Delayed Cardiac Rupture, Hemopericardium**

 ** Presenting Author*