Endocarditis and purulent cardiac tamponade after pacemaker implantation

L. Álvarez-Acosta*, R. Romero-Garrido, and J. Hernández-Afonso

Arrhythmology Department, Hospital Universitario Nuestra Señora de la Candelaria, Santa Cruz de Tenerife, Spain

* Corresponding author. Tel: +34 922602963; fax: +34 922602337. E-mail address: luisalvaco@gmail.com

We report a patient with a severe complication after pacemaker implantation. Two months after the implant, he developed cardiac tamponade secondary to severe purulent pericardial effusion. A pouch infection led to lead endocarditis and the purulent pericardial effusion. During surgery and heart mobilization the lead perforated the ventricular wall at the level of the outflow tract, close to the anterior descending artery.

We report a case of a patient with a pacemaker pouch infection and secondary ‘endopericarditis’ presented as a secondary cardiac tamponade to severe purulent pericardial effusion 2 months after implantation. The patient was a 50-year-old male without any relevant conditions with a paroxysmal third degree atrioventricular block. A 2-year experienced electrophysiologist performed the procedure and cefazolin was used as perioperative antibiotic prophylaxis. Two leads were implanted [a right atrial appendage and a right ventricular outflow tract (RVOT)]. The pouch and the device were followed up 1 week after the implant and no haematoma or signs of infection were observed. The patient began with fever 45 days after the implant and then at Day 60 arrived at our emergency room with septicaemia and cardiac tamponade signs. Pouch infection, large lead vegetations were also observed (Figure). Flora seeding from blood, pouch, and lead vegetations were proved positive for Staphylococcus aureus. Late RVOT perforations are exceptional but late cardiac tamponade, pneumopericardium, or progressive atrial perforations have been described. To our knowledge, pericardial infections have only been described in the setting of epicardial pacemakers. We postulate that the infection itself could have caused this atypical presentation. Endocardial inflammatory damage caused by the bacterial infection at the level of the lead tip and the subsequent epicardial contiguity infection may have led to this almost fatal outcome. Another possibility is that micro perforations of the active fixation lead may have caused a pericarditis and the concurrent endocarditis led to the infection of the effusion. The complete externalization of the lead occurred only during surgery and fortunately the cable did not cause any damage to the anterior descending artery (Figure).
References

