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DIFFERENT PATTERNS OF INFLAMMATION AFTER LEADLESS CARDIAC PACEMAKER IMPLANTATION – AN AUTOPSY STUDY

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BACKGROUND

Leadless cardiac pacemakers (LCPs) have fundamentally changed the field of device therapy. Lead and pacemaker pocket related complications - that affect between 2% and 12% of individuals with conventional pacemaker systems – can be effectively avoided with this new technology. The introduction of an LCP with VDD capabilities in 2020 will likely further increase the implantation rate of these devices in the future. However, there are certain drawbacks of LCP that have to be considered. Namely, the mode of extraction of LCP, especially years after implantation, in the case of a device infection or after battery depletion are issues that are still being debated. For extraction purposes, the Micra™ LCP has a knob on its tail that enables catching the device with a snare. Until now, there is still a lack of data on how much the Micra™ tends to be overgrown with tissue over time. Overgrowth with cardiac tissue may influence both the resistance to an infection with blood borne bacteria and the ability to grasp the device with a snare if extraction is needed. On the one hand, tissue could shield the device from bacteria, on the other hand this very tissue could cover the extraction knob and thus prevent a successful interventional extraction.

METHODS

We followed up on all patients who received a Micra™ LCP at our department. Survival status was determined by searching the hospital information system and by contacting the patients, relatives or their treating physicians. If no patient contact could be established, survival status was determined by contacting local registration authorities. If patients had deceased, efforts were made to identify the exact cause of death. As all pacemaker systems have to be removed before burial in Austria, we tried to find out whether the Micra™ LCP with the adjacent cardiac tissue block had been preserved and was still amenable to a thorough histopathological examination. If available, the following histological features were evaluated by two experienced pathologists in consensus: fibrin exudates on the LCP surface as well as fibrosis, inflammatory infiltrates, vascular proliferation and hemosiderin deposits in the myocardium adjacent to the LCP and its fixation tines. These findings were semi-quantitatively assessed as mild, moderate and marked depending on the degree and extent of histopathological changes. In addition to routine hematoxylin and eosin stains, Elastica-van-Gieson stainings were used to evaluate the extent of fibrosis and immunohistological stainings for CD3, CD20 and CD34 were used to identify T- and B-lymphocytes as well as endothelial cells, respectively.

RESULTS

Between December 2013 and July 2020, a Micra™ LCP was implanted in 283 patients (36.0% female) with a median age of 80.6 years (IQR: 76.5-85.1 years). During a median follow-up of 2.16 years (IQR: 1.17-3.96 years), sixty patients (21.2%) had died, predominantly from cardiovascular causes. The median survival time from implantation to death was 1.70 years (IQR: 1.02-2.98 years). Tissue blocks for histological analysis were available in eight patients (8/60, 13.3%) with a median survival time after LCP implantation of 379 days (IQR: 232-637 days; range: 18-1428 days). Fibrin capsules encoating the LCP as a whole or in part were identified in six (6/8, 75.0%, see Figure 1) patients who had a median implant duration of 295 days (IQR: 228-403 days, range: 18-576 days, septal position of the LCP in 2, apical position in 4 patients). In two (2/8, 25.0%) patients who died 697 and 1428 days after implantation, no fibrin coating was present (septal position of the LCP in both). Fibrin exudates were admixed with few neutrophils and lymphocytes, and in one patient few endothelial cells could be identified lining the fibrin coating. In all patients, fibrosis was found in the myocardium adjacent to the LCP and its tines. The degree of fibrosis ranged from mild (n=1) to moderate (n=7). Within the fibrotic areas, CD3-dominant inflammatory infiltrates and vascular proliferation were seen in all patients. Hemosiderin deposits were detected in four (4/8, 50.0%) patients (see Figure 2).

CONCLUSION

After Micra™ LCP implantation, a fibrotic tissue response with varying degrees of inflammation in the surrounding right ventricular implant area can be expected. Thereby, CD3-positive T-lymphocytes could predominantly be found. We could show that the LCP was covered with a fibrin capsule in many cases. The extent of overgrowth substantially varied between individuals but could be found as early as 18 days after implantation. Innovative physical methods of surface treatment might help to prevent the deposition of a fibrin cover on the Micra™ LCP. This would allow snaring of the LCP in all scenarios where extraction is required.

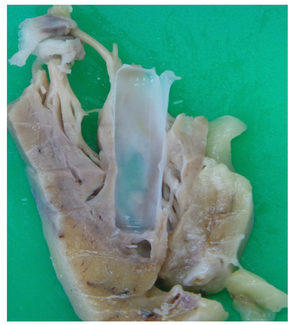

Figure 1: Fibrin capsule in a post mortem specimen.

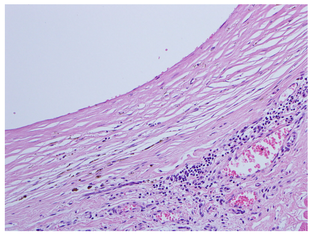

Figure 2: Hematoxylin eosin stain of the capsule with lymphocytes (blue dots).

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