Research Highlight

VAD infections: the lead, the graft and the pump

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Ventricular assist device (VAD) implantation has become an established treatment in the case of end-stage heart failure. One of the most important limitations is represented by the need of an extra-corporeal power supply and controller connected to the pump through a percutaneous cable. This may lead to local or even systemic infections that can jeopardize post-operative results. In the case of cable infections, medical treatment is usually less effective because of the presence of biofilms reduces the probability of complete pathogen eradication with specific antibiotic therapy (1). Nowadays ultrasound (US) imaging and computed tomography (CT) are recognized to be the best diagnostic tools to detect and define VAD specific infections. These diagnostic tools allow to identify the presence of infective effusions around the implanted components of the VAD.

Hannan *et al*. have tried to standardize the definition of different types of infections that can occur in VAD patients (2). The main objective was to establish concepts for a correct clinical diagnosis of infections and to identify predisposing risk factors, allowing a more practical prevention and management of infection related complications. In VAD assisted patients, three main categories of infections can be identified: VAD-specific infections, VAD-related infections and non-VAD infections.

The first group is the most important and is characterized by infections related to the device implanted components, such as pump, inflow or outflow cannula, pocket (defined as the space where the pump is housed) and percutaneous driveline (2). In this case, infection diagnosis is based on the modified Duke's criteria, usually applied for infective endocarditis.

The most common infections that affect patients with VAD are percutaneous driveline infections (PDI). Hannan *et al*. classified PDI as superficial or deep, depending on the depth of the infection (2). Frequently, these infections are very superficial and limited to the cable exit-site. These conditions can occur at the beginning because of an injury during VAD implant (tunnelling of the driveline) and subsequently as a result of excessive driveline traction and movement (3). Careful tunnelling and fixing methods of the cable are essential in preventing PDI.

Our group has recently published the use of a non-invasive device to fix the driveline to the skin. The purpose of this fixing method is to move the fulcrum of the lever a few inches away from the cable exit-site. The result is a safe and effective driveline stabilization that promotes correct healing of the wound (4).

The device consists of two components: a StatLock system, commonly used to fix peripherally inserted central catheters (PICCs), and a silicone suture wing taken from a central venous catheter (CVC) set.

From November 2010 to May 2014 at our institution, 23 patients underwent HeartWare LVAD (HVAD) implantation for end-stage heart failure. All patients were INTERMACS II or III. Mean age was 60.9 years (range, 39-70 years). Twenty patients were male and three patients were female. HVAD were implanted either through median sternotomy (n=20) or left thoracotomy (n=3). Median sternotomy is the routine cardiac access while the left thoracotomy is the preferred surgical approach for patients with previously operations. In case of sternotomy, the driveline is tunnelled on the right side of the abdominal wall, otherwise the exit-site is on the left side. The tunnelling procedure, according to Slaughter *et al*. (5), consists of two steps: first, the cable is positioned from the pericardium to the abdomen (above the umbilicus) running between the rectus abdominis muscle and its deep fascia. At

this level, the cable is fixed onto the superficial fascia of the rectus in order to create a physical barrier for "ascending" infections. As second step, the driveline is tunnelled under the skin towards the exit-site.

Since April 2013, we have used our self-made fixing device for all patients, even for those previously implanted. An analysis of patients' data before April 2013 showed a 0.21 PDI/patient/year incidence. Maintaining the same medication protocol, after the introduction of our device, no PDI occurred in any patients. In addition, no episodes of cable infection have been detected within the first four months after VAD implantation. This is probably due to reduced mobility of the patient in the early postoperative period which prevents the driveline to be solicited through the skin exit-site. In the long term follow-up, patients treated with the fixation kit showed a trend towards a significant lower PDI incidence compared to the control population.

In conclusion, PDI is still one of the major limitations of VAD application. Gradual improvement of the driveline characteristics, such as a flexibility, biocompatibility, and decreased cable diameter, together with improved surgical tunnelling technique have led to a significant reduction in PDI incidence. The introduction of driveline fixing tools could further improve outcomes, while waiting for total implantable mechanical assisted devices. Additional studies are needed in order to confirm these findings.

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