Review Article

Long-Term Ventricular Assist Devices – Main Complications in Contemporary Clinical Practice

Dayanna Machado Pires Lemos,[1,2] Gustavo Paes Silvano,[1] Kely Regina da Luz,[1] Marco Aurélio Lumertz Saffi,[1,2] Marcus Vinicius Przepiorka Vieira,[1] Fernando Luis Scolari,[1] Lívia Adams Goldraich[1,2]

Hospital de Clínicas de Porto Alegre,1 Porto Alegre, RS – Brazil

Programa de Pós-Graduação em Ciências da Saúde: Cardiologia e Ciências Cardiovasculares, Universidade Federal do Rio Grande do Sul,[2] Porto Alegre, RS – Brazil

Abstract

Advanced heart failure (HF) is associated with reduced quality of life and high hospitalization and mortality rates. Ventricular assist devices (VADs) promote an increase in cardiac output, and consequently improvements in body functions, functional capacity and patient survival. However, the use of VAD may be associated with complications and require systematic and specialized care. Ischemic and hemorrhagic stroke is among the most feared complications and its occurrence is related to thrombus formation in the pump. The connection between the driveline and the external power source is a potential source of infection that may extend to the mediastinum. Management of bleeding caused by anticoagulation therapy may be challenging, since discontinuation of the treatment may lead to thrombus formation. Aortic insufficiency and right ventricular dysfunction may occur, particularly in prolonged periods of support, requiring optimization of VAD parameters and clinical management. Although uncommon, mechanical failure of the VAD may occur and require replacement of the pump or even heart transplant. Thus, identification and management of the main complications of VAD in patients with advanced HF is needed, so that strategies for prevention and rigorous clinical follow-up can be implemented. This review aims to summarize the main adverse events in patients with long-term VAD.

Introduction

Stage D advanced heart failure (HF) is characterized by abnormalities in cardiac structure that lead to tissue hypoperfusion, target-organ damage, cachexia, and limiting symptoms.[1,2] It is estimated that 5-25% of patients with HF will develop the advanced stage of the disease, which is associated with high hospitalization and mortality rates, even among those under optimized drug therapy.[3-5] Also, a large number of patients will require advanced therapies.[6] In this context, heart transplant (HT) is usually the surgical treatment of choice; however, the feasibility of this treatment is limited by the low availability of organs and the potential clinical complications of the procedure.[7] Therefore, long-term ventricular assist devices (VADs) represent an important therapeutic alternative that allows patient to get back to daily life activities, promoting higher quality of life and survival.

VAD is a surgically implanted mechanical pump that provides circulatory support in patients with severe systolic dysfunction, restoring cardiac output and reducing left ventricular (LV) work.[8] The VAD has inflow cannulas positioned in the left ventricle, and a mechanical pump connected to the external power source. Today, VAD with two different technologies, named second- and third-generation devices are used. Second-generation axial-flow devices, like the HeartMate II (HMII; Abbott Labs), were widely used for about 15 years, but its use has decreased worldwide. Likely, the commercialization of the centrifugal-flow HeartWare Ventricular Assist Device (HVAD; Medtronic), which uses a combination of hydrodynamic and magnetic levitation, has been discontinued recently. The HeartMate III (HMIII; Abbott Labs) accounts for 77% of the implants today.[9,10] It consists of a magnetically levitated cardiac pump, with wider blood-flow paths and pulsatility and has been associated with better outcomes of stroke-free survival and reintervention due to malfunctioning of the pump.[11]

In the last decade, approximately 25 thousand patients have undergone VAD implantation.[10] In 2019, 3,198 VADs were implanted in the USA, which is the highest registered by the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS).[10] VAD technology has improved substantially and its use for the treatment of advanced HF has increased tremendously.[12] Also, the 1- and 2-year survival between the years 2015 and 2019 has improved compared with 2010 to 2014 (82.3% in the first year and 73.1% in the second year *vs.* 80.5% in the first year and 69.1% in the second year).[10] Currently, median survival rate of patients with VADs is nearly five years.[9]

Despite advances in the VAD design and in clinical treatment, 30-day adverse events still occur in 31% of patients.[13] According to INTERMACS, 72% of patients are hospitalized at least once within 12 months after

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Mailing Address: Lívia Adams Goldraich •
Rua Ramiro Barcelos, 2350. Postal Code 90035-903, Porto Alegre, RS – Brazil
E-mail: lgoldraich@hcpa.edu.br
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implantation.[10] Thus, the use of VAD has been associated with complications that can increase morbidity and mortality and hence require a close follow-up for better outcomes of the intervention (Figure 1).[11,14]

Several measures should be taken to promote safety and minimize potential adverse treatment events for the patients using VADs.[15,16] This review aims to summarize the main adverse events in patients in long-term mechanical circulatory support.

Case report

Here we report a clinical case of a female patient, 54 years old, history of dilated cardiomyopathy and severe mitral insufficiency for Chagas disease, who underwent HMII implantation in 2018 due to inotrope dependence and immune hypersensitivity, which reduces the possibility of HT. The post-implantation was complicated with bleeding, sepsis, severe abdominal distension and difficult anticoagulation control. After about 10 months of follow-up, the patient developed sustained elevation of power and

Stroke
Anticoagulation
Thrombolytics (selected cases)
Endovascular therapy

Aortic insufficiency
Clinical management
Optimization of the VAD parameters (ramp study)
Valve replacement

Pump thrombosis
Clinical management
Intensification of anticoagulation
Thrombolytics (selected cases)
Device replacement or heart transplant

Right ventricular dysfunction
Clinical management
Optimization of the VAD parameters

Mechanical problems / alarms
Revision of the log files
Optimization of the VAD parameters
Device replacement

Infection
Antibiotics
Debridement
Device replacement

Bleeding
Management of anticoagulation and antithrombotics
Clinical management/transfusions
Endoscopic or surgical treatment (selected cases)

Figure 1 – *Main complications of the ventricular assist device and summary of treatments.*

important VAD flow variations, clinical signs of hemolysis – hemoglobinuria and hemoglobin fall, requiring blood transfusion, peak lactate dehydrogenase (LDH) of 2557 U/L and loss of renal function. The patient underwent replacement of the VAD pump, due to clinical suspicion of thrombosis, which was confirmed intraoperatively. After two years with a good clinical course, persistent low-flow VAD alarms occurred, and the patient developed progressive signs of HF and cardiogenic shock. Due to clinical suspicion of subocclusive thrombus in the outflow cannula and aortic insufficiency (AoI), the patient underwent another surgery. Surgical findings revealed dense fibrous tissue around the Dacron and polytetrafluoroethylene (PTFE) patch of the outflow cannula, and presence of inflammatory exudate within the cannula, causing extrinsic compression (Figure 2). The cannula was reimplanted after removal of the fibrous layer and aortic valve replacement was performed. This case illustrates some of the challenges faced in the follow-up of patients with VAD, related to bleeding monitoring, occurrence of hemolysis, and identification of mechanical changes in the cannulas and changes in valvular changes. Trained, multidisciplinary teams are essential for better outcomes.

We will now describe the main adverse events related to the use of VADs and a brief discussion of their management.

Main adverse events of long-term VAD

Stroke

Devastating neurological events such as ischemic (thromboembolic) or hemorrhagic stroke affect nearly 10% of patients with VAD within one year.[10] These events are the main cause of long-term mortality after VAD implantation. However, the growing number of centrifugal-flow HMIII device has caused a reduction in these events. In a two-year clinical follow-up, HMIII was associated with a lower incidence of any stroke, and an estimated two strokes could be prevented for every 10 patients who receive HMIII implant.[17]

The risk of stroke in patients with VADs is associated with several factors. Patient-related factors may be related to higher odds of cerebrovascular events, such as age, female sex, severity of HF, history of diabetes, hypertension, atrial fibrillation, hypercoagulability, and

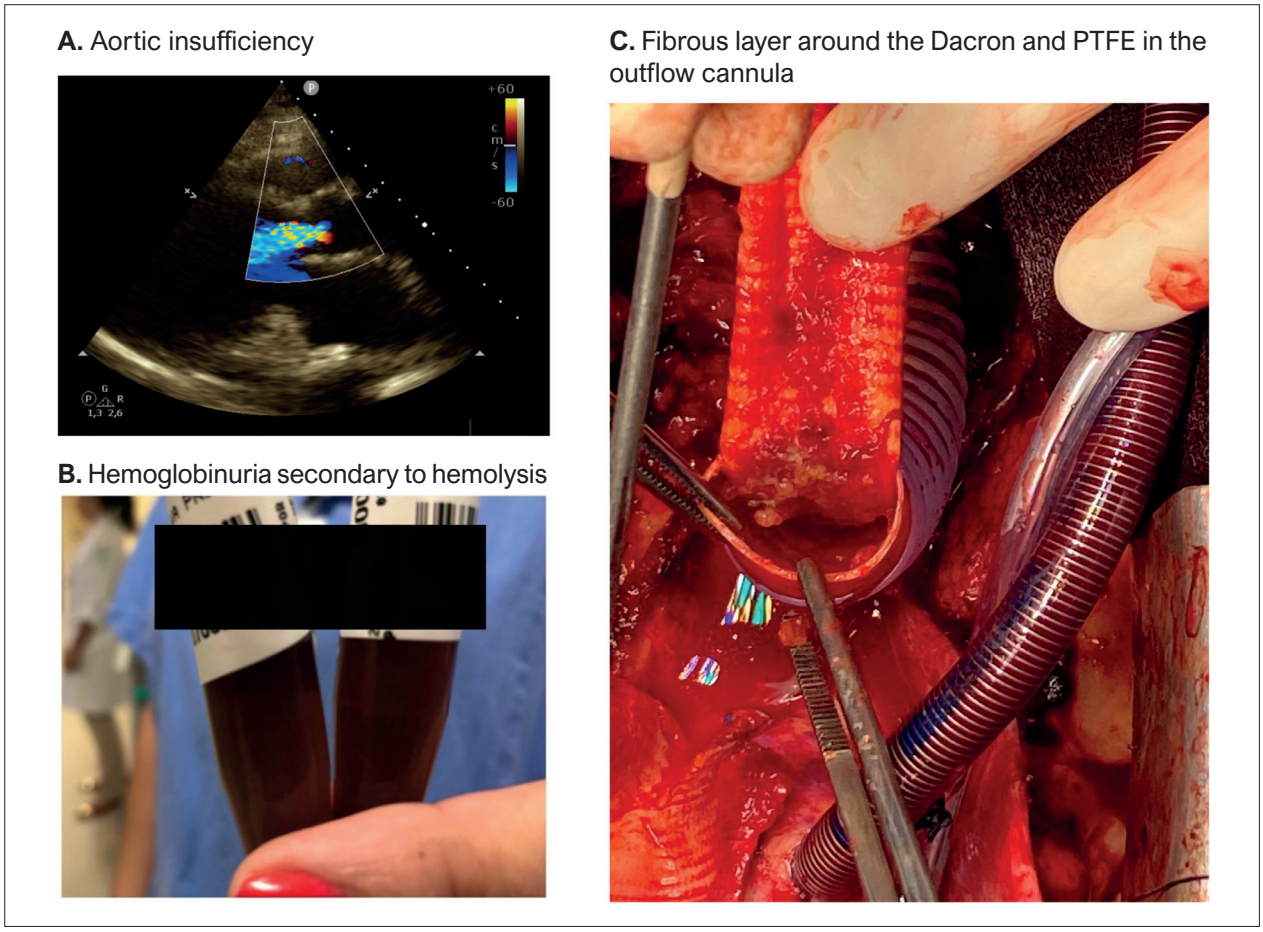

Figure 2 – *Complications in a patient using ventricular assist device. (A) Doppler echocardiography showing moderate-to-severe aortic insufficiency (B) Hemoglobinuria secondary to hemolysis caused by pump thrombosis. (C) Fibrous tissue layer around the Dacron and the polytetrafluoroethylene (PTFE) patch, causing extrinsic compression in the outflow cannula.*

infections unrelated to the implant. Besides, perioperative (aortic clamping and cardiac arrest with cardioplegia) and postoperative (duration of mechanical support, infection, subtherapeutic anticoagulation and hypertension following VAD implantation) factors, and those related to the VAD (infection, hemolysis and pump thrombosis) also influence the rate of stroke.[17,18]

During patient assessment, attention should be paid to the precise onset of neurologic manifestations and their course, using preferably the National Institute of Health and Stroke Scale (NIHSS),[19] and computed tomography angiography of the brain and intracranial vessels. Magnetic resonance is contraindicated due to the VAD metallic components, which can make difficult the early detection of ischemic events.

A previous imaging test and a careful clinical examination can help in the diagnostic and decision-making processes. VAD parameters should be analyzed for signs of the VAD malfunction or thrombosis.[17] Patients seen in hospitals without a circulatory support program should be followed by the VAD referring team, revising the therapeutic plan and determining the need for emergent transfer.[17] The most common events are the ischemic ones, caused mainly by embolism, whose management may be challenging depending on the extension of cerebral infarction. The balance between the risk of hemorrhagic transformation and the need of anticoagulation should be considered in the decision making. In the absence of hemorrhage, the selective use of thrombolytic agents and endovascular interventional neuroradiology should be considered in those with early presentation and clinical indication. However, it should be mentioned that these recommendations derive from clinical trials with patients without VAD.[20]

In cases where embolism is secondary to VAD thrombosis, systemic thrombolysis was shown to be safe in case reports.[17] However, a meta-analysis of observational studies did not show superiority of thrombolytic treatment over conventional pharmacological treatment, and the use of thrombolytics was associated with higher risk of major hemorrhage.[21] A randomized study is needed to evaluate whether the routine use of thrombolytics is beneficial in this condition. In hemorrhagic stroke, anticoagulation discontinuation or reversal with prothrombin complex concentrate is recommended for patients with INR ≤ 1.5. The time when anticoagulation should be resumed must be discussed with the neurovascular team.

Aortic insufficiency

Nearly 25% of patients that undergo VAD implantation develop severe-to-moderate AoI or aggravation of pre-existing AoI. This is a relatively high rate, considering that the prevalence of severe AoI prior to VAD implantation is 0.6%.[10]

Elevations in the pump flow lead to intermittent or permanent closure of the aortic valve and eventual commissural fusion.[9] This is the main risk factor for AoI, which is associated with high morbidity.[22] Also, the site and angle of the outlet graft anastomosis on the aortic wall contribute to AoI progression. AoI, in turn, causes a cascade of events – part of the cardiac output generated by the VAD returns to the left ventricle due to valvular incompetency, resulting in a fall in cardiac output and an increase in filling pressure.

The assessment of the aortic valve is essential for determining AoI severity and the planning of treatment.[23] Anatomic characteristics, such as the number of cuspids, remodeling degree, area of calcification and presence of aortic root dilation are also important, as well as the measurement of the regurgitation jet size.[23] Traditional echocardiographic parameters usually underestimate the severity of AoI; the regurgitant jet is present throughout the whole cardiac cycle since the left ventricle cannot compensate the flow during systole.[24] Therefore, even small regurgitant orifices can represent severe AoI.[23,24] Novel echocardiographic parameters have been used for grading AoI severity. Some of these include the systolic-to-diastolic velocity (S/D) ratio of the VAD outflow cannula, and the diastolic acceleration of the VAD outflow cannula, that are inversely and directly proportional to the AoI severity, respectively. A S/D ratio $<5 cm/s^2$ and a diastolic acceleration >49 cm/s^2 indicate moderate-to-severe regurgitation. However, these findings should be analyzed along with LV dilation, aortic valve remodeling and permanent closure, continuous regurgitant jet and aortic root dilation.[23] Such analysis requires an experienced professional and systematic reassessment. One of the strategies to prevent this complication is the flow velocity titration, guided by echocardiography, or maintenance of a pulsatile flow (native or generated by the device), allowing the intermittent opening of the aortic valve.[9] The benefit of this intervention, though, needs to be confirmed by clinical trial.[25]

The degree of AoI may not be reduced by clinical treatment and requires surgical intervention. Although there is still not a consensus on the best surgical approach, the Park stitch (a central coaptation stitch for leaflets) and the aortic valve replacement stand out. Both procedures can be performed either concomitantly or after the VAD implantation, in the presence of hemodynamically important AoI. Percutaneous aortic valve replacement for this indication has been noted in case reports, further studies are still needed.[9]

Right HF

Previous right ventricular (RV) dysfunction, associated with pulmonary hypertension and acute hemodynamic changes, facilitate the occurrence of right HF in the post-left VAD implantation in approximately 15-25% of patients.[26,27] Although its mechanism is still not clear, it is believed that changes in chamber geometry are caused by a sudden increase in LV outflow and RV preload. After the left VAD implantation, RV dysfunction is associated with impairments in body functions, longer hospital stays and higher mortality.[28]

The adequate screening for candidates for the VAD implantation by risk prediction is essential to identify those

patients with right HF that could benefit from a left VAD.[13] From the diagnostic point of view, electrocardiogram, echocardiogram, cardiac biomarkers, magnetic resonance, and right heart catheterization are complementary tests.[29] In the immediate post-operative period, patient monitoring using invasive parameters, such as the measurement of RV work, central venous pressure and serial echocardiograms is fundamental.[28]

Although most patients with RV dysfunction respond to inotropic therapy and optimization of VAD parameters, the early implementation of temporary RV circulatory support shows prognostic benefit.[30] New less invasive techniques for left VAD implantation seem to be associated with lower incidence of post-implantation RV dysfunction. However, late RV dysfunction may also occur, leading to a worse prognosis.[28] In these subacute and chronic contexts, increased pump velocity and flow can overload an already compromised right ventricle at any time after the transplantation. RV dysfunction may also occur secondary to ventricular arrythmias, pulmonary embolism, persistent pulmonary hypertension, and new or aggravated tricuspid regurgitation.[17,31]

Patients with right HF may develop hemodynamic deterioration, implantable cardioverter defibrillator, and even cardiac arrest with ventricular tachycardia or fibrillation caused by impaired filling and inadequate flow in the VAD. Echocardiography should be performed to exclude cardiac tamponade and to analyze ventricular filling and dimensions. Clinical treatment includes vasoactive therapy, diuretics and inotropic support, preferably with milrinone, and should be guided by invasive hemodynamic monitoring with pulmonary artery catheterization. In case of significant pulmonary hypertension, pulmonary vasodilator therapy should be considered, and percutaneous RV support may be less relevant.[17]

Pump thrombosis and cannula obstruction

Pump thrombosis has an incidence of 8% in the first year after VAD implantation and consists one of the main causes (up to 50%) of replacement of the device. According to the INTERMACS, pump thrombosis affects 5.5% of patients with HMII. In this regard, magnetic levitation devices, like the HMIII, provide a safer design, with an incidence of only 1% in a 24-month follow-up.[11]

Although the etiology of pump thrombosis has not been fully elucidated, it is known to be multifactorial and show variations depending on the device.[31] Associated factors include heat generated from the pump rotor, shear stress with platelet aggregation, thrombosis at cannulation site, impaction of the outflow cannula and migration or malposition of the inflow cannula. In addition, patient-related factors including a history of atrial or ventricular thrombus, atrial fibrillation, presence of left mechanical prosthetic valve, ventricular dysfunction degree and hypovolemia, and factors related to the management of the patient, like subtherapeutic anticoagulation, absence of antiplatelet therapy, low rotation, and control of infections.[31]

Patients with pump thrombosis usually present elevation in the pump speed and power, decreased flow, and different degrees of hemolysis and HF.[31] Fibrin deposition on the pump components causes flow delay, which requires compensation by an increase in the pump power to maintain the speed. The turbulent flow increases the shear stress, leading to hemolysis, which is manifested by hemoglobinuria, jaundice, increased serum LDH, free hemoglobin, total and indirect bilirubin, and decreased haptoglobin levels.[17] When this complication is suspected, the patient should be urgently transferred to a VAD-capable center. In patients with hemodynamic instability, intensive monitoring, anticoagulation and HF treatment should be immediately initated.[31]

Therapeutical strategies include anticoagulation and antiplatelet agents, thrombolysis and/or device replacement. The selection of the initial therapy is a complex decision, based on several factors, including patient clinical presentation. While pump replacement has been associated with an increase in perioperative mortality, clinical treatment is more likely to be unsuccessful, and to higher rates of recurrence or need for pump replacement or HT. Also, mortality is found to increase with every pump replacement.[31]

In case of improvement in clinical outcomes with unfractionated heparin and/or direct thrombin inhibitors, an increase in the antithrombotic regimen (AAS 200 mg or 325 mg/day and warfarin and target INR between 2.5 and 3.0), and eventually, dual antiplatelet therapy. If symptoms persist, aggressive antithrombotic therapy with direct thrombin inhibitors such as bivalirudin and argatroban should be considered, but data on their efficacy are still limited. Thrombolysis with recombinant tissue plasminogen activator (e.g. alteplase) should be considered only after cranial computed tomography to exclude eventual ischemic events and hemorrhagic transformation. It is important to mention that the evidence of the benefits of these therapies is still uncertain and based on case series, and the risk of severe hemorrhagic complications cannot be ruled out. For this reason, the therapies should be implemented with caution and be restricted to patients who are not candidates for surgical treatment.[31]

Surgical replacement of the pump for thrombosis is considered the definite (and gold-standard) treatment. Preoperative evaluation by computed tomography scan of the chest with contrast and echocardiography can be performed to detect possible anatomical causes of thrombosis. Suggestive findings of malpositioning of the inflow cannula and dynamic obstruction, kinking or compression of the outflow cannula are indications for the replacement of the VAD by median sternotomy due to limited access via the subcostal approach. The subcostal approach is the preferred route as it allows better access to the LV apex for manipulation of the pump and inflow connection. It can be performed with extracorporeal circulation (ECC) via peripheral cannulation or without ECC, depending on the ventricular reserve and hemodynamic stability of the patient. Good results have been reported with the subcostal approach in experienced

centers, with a 30-day mortality of 6.5% in patients with HMII.[31]

Emergency HT is a therapeutical option for patients without contraindications, considering that the estimated waiting time is not long, the management of the HF is feasible, and that hemolysis does not have important repercussions, such as the need for multiple transfusions or severe renal insufficiency. Favorable results of the management of outflow cannula stenosis with percutaneous stent implantation and intravascular ultrasound to distinguish between thrombosis from external compression have been reported.[9] Explantation of the VAD is usually the treatment of choice for patients with recovery of ventricular function.[31]

Bleeding

Although changes in the VAD design have caused a reduction in the incidence of bleeding, this is still one of the most common complications. The contemporary rate of bleeding is 1.4 events per patient-year within 90 days after the implantation, and 0.3 events per patient-year in the late follow-up period. According to INTERMACS, only 67% of patients are free from major bleeding in the first year of therapy. In addition, severe bleeding is the cause of 2% of deaths in patients with VADs.[10] In a two-year clinical follow-up, patients with HMIII showed lower rates of bleeding in comparison with patients with HMII, probably due to the pump design that promotes a lower interaction between VAD and blood.[11,32]

Perioperative bleeding is the most common immediate complication after VAD implantation, affecting up to 80% of patients. Besides the sternum, the most common site of bleeding is the outflow cannula anastomosis. Its preoperative prevention includes nutritional and hemodynamic optimization (especially for reversal of hepatic and renal dysfunction and related coagulopathies), suspension of anticoagulant and antiplatelet therapy. The risk of bleeding may be reduced by improvements of surgical techniques, appropriate reversal of heparin anticoagulation, and use of pro-hemostatic agents and factor concentrates as appropriate.[31]

In the postoperative period, gastrointestinal bleeding is the most prevalent, especially in elderly patients with a history of this condition.[9,10] Although its pathophysiology remains unclear, factors like low pulsatility, acquired von Willebrand disease secondary to shear stress, angiodysplasia (abnormal small blood vessels) in the gastrointestinal tract and anticoagulation therapy seem to be related.[9,33] The most common sources of bleeding are arteriovenous malformations in the stomach and duodenum, and inflammatory changes and ulcerous lesions in the digestive tract.[17] However, in many cases, the origin of bleeding cannot be identified. Endoscopic and colonoscopic evaluations are recommended to identify the bleeding source; it is worth pointing out, though, that the site of bleeding may be in the small bowel, which would reduce the diagnostic value of these procedures.[31]

The treatment of gastrointestinal bleeding includes volemic resuscitation, proton pump inhibitors and endoscopic approach. Either suspension or reversal of anticoagulation therapy yields modest benefits, with a recurrence rate of up to 9%, besides increasing the risk of severe thromboembolic events. Blood component transfusions may be required, but should be considered cautiously, as they add risk of immune hypersensitivity in candidates for HT.[9,31]

Epistaxis is the second most common hemorrhagic complication in patients with VAD. Its initial management consists of local vasoconstriction, cautery and tamponade. Percutaneous intervention including arterial embolization should be needed in severe cases, and evaluation by an otorhinolaryngologist is recommended.[31]

Infections

Infection is a common complication and an independent predictor of mortality in patients with VADs.[10] Risk factors include trauma in the driveline, obesity, duration of support, aging, diabetes, renal insufficiency, and malnutrition.[34] In 2011, a work group of the International Society for Heart and Lung Transplantation worked on the standardization of definitions of these infections and classified them as VAD-specific infections, VAD-related infections, and non-VAD-related infections.[35]

VAD-specific infections may occur in the pump, cannulas, pump pocket or driveline. An early identification and an aggressive treatment are essential in the infection control, which may require the removal of the device.[35] VAD-related infections refer to those that may also occur in patients who do not have VADs, but may have different characteristics or require specific care in patients with VADs, as in cases of infectious endocarditis and mediastinitis. Non-VAD infections are not affected by the presence of the VAD, such as pneumonia and urinary tract infection. In the INTERMACS registry, 42% of patients using VAD developed an infection at a median of 69 (interquartile range 12 to 272) days. Most were non-VAD infections (49%), followed by VAD-related (26%) and VAD-specific infections (25%).[36]

Gram-positive cocci, especially *Staphylococcus aureus* and coagulase-negative staphylococcus account for more than 50% of infections. Gram-negative bacilli may also be present, particularly *Pseudomonas aeruginosa*.[37] Fungal infections are less common but have a significantly worse prognosis; most infections are caused by *Candida spp*.[38] The identification of the causal agent is extremely important. Blood culture collection prior to antibiotic therapy and analysis of samples collected from exudates are essential in the assessment of patients with suspected or confirmed infection in any segment of the VAD.

Due to the smaller size and surface of contact, continuous-flow VADs are associated with lower rates of infection than pulsatile-flow VADs (e.g., 0.38 versus 0.62 driveline infections per patient-year with HMII and HeartMate XVE, respectively).[39] However, driveline infections remain a significant problem after the

device insertion, particularly in the first 30 days after implantation.[36] Its clinical manifestations include general malaise, fever may occur, and when so, are usually associated with higher impairment in functional capacity and abscess formation.[39-41]

Regarding laboratory data, VAD infections are marked by high white blood cell counts, and increased C-reactive protein levels.[42] In case of suspicion, echography and computed tomography of the abdomen and abdominal wall can detect from thickening of adjacent tissues to formation of organized collections.[42,43]

Controlling the source of the infection should be made whenever possible and includes drainage and debridement. Local debridement of the driveline exit site may be needed in the presence of fluctuant, hard, or necrotic tissue, and eventually, the driveline is relocated to another site, distant far from the infection. In patients with deep infection, surgical drainage and vacuum-assisted closure should be considered.[44,45] The benefits of negative-pressure wound therapy include removal of debris, edema reduction, improvement of blood, and granulation tissue formation.[42] Other local interventions with potential benefits are the use of antibiotic beads and omental or muscular transposition flaps.[46,47]

Infections of the surgical cavity refer to those in the pump pocket which, similar to the driveline infections, occur in the long term. First- and second-generation VADs required a large cavity between the abdominal wall or pericardium and the diaphragm and were more prone to therapeutic failure because of poor vascularization.[37] Modern devices are usually placed in the intrathoracic or preperitoneal space, and some of them do not require a surgical pocket.[44,45]

Bloodstream infections affect up to 30% of patients using VADs, especially in the first three months after the implant surgery.[48,49] These infections are normally related to the driveline, pump pocket or the pump, but other sources of infection (e.g. implantable cardioverter defibrillator and infectious endocarditis) should be investigated and controlled.[17] In most cases, a prolonged treatment with oral antibiotics is required.[45]

Mechanical failure

Mechanical dysfunction of the VAD occurs in up to 6% of patients in the first year.[10] Although the literature has focused on the pump failure, its incidence is on 13% only, and may be related to thrombosis, as previously discussed in this article. Other device components are potentially subject to malfunctioning, such as the controller (30%), driveline (14%) and battery (19%), with fatal and non-fatal repercussions.[50] Yet, the incidence of deaths due to device malfunctions has decreased from 3.9% to 1.4%,[10] and obesity was considered an independent predictor of mechanical dysfunction of the pump.[50] In 2021, after extensive use, the HVAD was removed from commercial distribution by the manufacturers because of events of delay or failure to restart after elective or accidental discontinuation of pump operation.[51]

The *short to shield* phenomenon occurs when stresses applied to the driveline with repeated stretching, bending, or twisting beyond the limits of robustness of the driveline causes fracture of the internal ground shielding, which can damage power data transmission, leading to pump stoppage.[50] Driveline failure frequently requires external repair or pump replacement in cases when the portion of the driveline that fails is close to the skin exit site or at its junction with the VAD. In the HMIII, the external segment of the driveline that connects the controller was improved with the addition of another connector that allows the non-surgical driveline replacement in case of damages. Also, failure of other external components may occur when the patient inappropriately connects the drivelines, damaging the connecters. Failure of the VAD controller may be caused by software issues, exposure to water or fluids, and damage from dropping, which reinforces the importance of always keeping a spare controller available. The inadequate use of the device and traumas can damage the battery damage, which reduces its expected life and affects its full recharge.[50]

Periodic VAD interrogation is essential for identification of failures. Registries of critical alarms and flow changes, pulsatility index and peak circulatory power should be recorded. A member of the VAD team should send log files for analysis by clinical engineers whenever appropriate.[17] VAD auscultation is not a reliable method to detect malfunctioning due to its low specificity.[31] In addition to the signs and symptoms of HF, physical examination of the patients should provide hints about the VAD malfunctioning. In most patients using VADs, peripheral pulse cannot be palpated due to reduced pulse pressure. Thus, mechanical dysfunction should be considered in patients with a palpable radial pulse. Examination of the sclera for icterus and the conjunctiva for small hemorrhages can also add information on hemolysis.[31]

Standardized preclinical tests and medical device engineering have been developed to prevent these failures. Patients, caregivers and health care professionals should receive systematic instructions about how to care for the VADs. However, there are challenges in real life that cannot be predicted by laboratory tests, and devices may be less robust in the long term for reasons not necessarily related to lack of care.[50]

Final considerations

In a relatively short time, VADs have become a well-established treatment for advanced HF, with an increasing number of adults being supported with VADs as destiny therapy, bridge to transplant, bridge to transplant eligibility and, less frequently, as bridge to recovery. Although the risks of adverse events are still significant, improvements in survival rates and reduction in morbidity tend to progress with advances in technology and patient selection and follow-up. Besides, there is a growing number of studies evaluating strategies for prevention, diagnosis and management of complications, despite the observational design in most of them. The continuous review of adverse events of VADs and the identification of unique aspects of their diagnosis and management become paramount as novel devices are developed and implemented in the clinical practice.

Author Contributions

Conception and design of the research, Acquisition of data, Analysis and interpretation of the data, Writing of the manuscript and Critical revision of the manuscript for intellectual content: Lemos DMP, Silvano GP, Saffi MAL, Vieira MVP, Scolari FL, Goldraich LA.

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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