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Pressure sore incidence and treatment in Left Ventricular Assist Device (LVAD) -equipped patients: insights from a prospective series

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Abstract

Introduction: Left ventricular Assistance Device (LVAD) is indicated in patients with end-stage heart failure. Due to the non-physiologic blood flow, LVAD may favor pressure sores with a devastating risk of infection. This work shows prevalence and treatment of pressure sore in LVAD patients, to optimize their management.

Material and Methods: We retrospectively investigated all LVAD implantations at the Lausanne University Hospital (CHUV) from 2015 to 2019. We detected patients who developed a pressure sore, and evaluated timeline, management, and outcomes.

Results: 42 patients benefited from LVAD, among which 5 (12%) developed a stage III/IV pressure sore, within a mean time of 25 days. 4/5 patients were treated surgically and 1/5

conservatively, due to poor overall condition. Half of the patients treated with surgery had major complications requiring reoperation. Mean time to healing for patients after flap coverage was 6 weeks.

Discussion: The rapid development of deep pressure sores seen in 12% of patients may be a manifestation of the maladaptive blood flow induced by LVADs, combined with their bedridden condition. Initial signs of pressure sores should be considered seriously, as rapidly evolving and needing an aggressive surgical treatment whenever possible (80%). Complication rate was similar compared to standard pressure sore flap treatment. All patients benefiting from flap surgery achieved effective coverage after a mean follow-up of 24 months. No patient developed LVAD infection.

Conclusion: Surgery must be considered early in this population to prevent potential device infection.

Keywords: *Left Ventricular Assistance Device/LVAD/pressure sore/lumbosacral reconstruction/gluteal flap*

Introduction

Left ventricular assist device (LVAD) therapy is nowadays a well-established approach to improve heart function in patients suffering from end-stage heart failure (ESHF). It is generally used as destination therapy or bridge to cardiac transplantation [1, 2].

Particularly, bridge-to-transplant therapy has progressed since the implementation of new-generation continuous-flow LVADs (heartmate (HM) III™, Abbott, USA) with improvements in early and medium-term survival and quality of life for patients with ESHF [2]. Side effects need to be taken in account, such as gastro-intestinal bleeding, thromboembolic events (due to anticoagulation treatment/mistreatment), aortic insufficiency (due to the

continuous LVAD flow not matching physiological systolo-diastolic heart phases), as well as inflammatory and immune disorders. Importantly, infection remains the most feared complication of LVADs, as it can lead to pump-pocket sepsis, significantly impacting on patient's morbidity and survival [3, 4].

Interestingly, the treatment of wound complications such as bed pressure sores in ESHF patients with LVADs has never been investigated in literature, despite being a critical point, considering potential septicemia risks. Indeed, patients with cardiac failure are at already increased risk of developing pressure sores during their ICU stays and a non-pulsatile flow may further incite the development of pressure sores, affecting homeostasis, peripheral vascularization, end-organ function and wound healing[3, 5-7]. Moreover, In LVAD patients, the baseline systemic inflammatory response syndrome (SIRS) and associated cytokines profile may couple with the oxidative stress induced by microvascular dysfunction, enhancing the risk of developing pressure sores[8].

Despite the growing use of ventricular assistance devices, to our knowledge no reports exist on the management of decubital wounds in the specific population of LVAD-equipped patients, with no studies describing incidence and surgical management of such wounds, which represent a surgical challenge due to the increased risk of hemorrhage and the peculiarity of tissue perfusion.

This work analyzes a consecutive series of LVAD-equipped patients during both the immediate course after device implantation and in the long term, with a special focus on patients who developed a pressure sore. We discuss risk factors potentially leading to pressure sores, with the aim to optimize preventive measures in this specific population, and initiate prompt surgical management if indicated. An algorithm detailing our experience in

the management of this challenging complication is defined, including potential complications and drawbacks of both conservative and surgical approaches.

Patients and Methods

From November 1st 2015 to December 31st 2019, 42 patients with ESHF were consecutively treated using HM III LVAD in the Lausanne University Hospital cardiac surgery department, and included in a prospectively maintained database.

All preoperative data including primary cause of heart failure, patients' demographic data (age, BMI) and comorbidities (such as diabetes, renal failure, metabolic syndrome and malnutrition), were collected from patients' ICU and anesthesia records. Operative protocols, hospital letters, and outpatient reports were used to evaluate the hospital stay and the follow-up period.

We recorded major cardiac-related complications after LVAD implantation, including pump failure or thrombosis, bleeding, stroke, infection, cardiac failure or dysrhythmia. In patients developing pressure sores we recorded onset of pressure sore after LVAD implantation, negative-wound pressure therapy, details on management (surgical vs conservative), time to healing, hospital stay and follow-up. In patients requiring surgical management, postoperative complications were divided in cardiac-related and flap-related. Flap-related complications were considered major if patients needed to get back to theater of if a supplementary reconstructive procedure had to be performed. Minor complications included wound dehiscence requirement conservative treatment for over a month.

All patients granted informed consent for medical and photographic documentation and use for research purpose. The study was conducted according to the declaration of Helsinki.

Surgical technique for LVAD implantation

LVAD (Heartmate (HM) III™, Abbott, USA) was implanted between the left ventricular apex and the descending aorta via either median sternotomy (n=34), double mini-thoracotomy (n=7), or left anterolateral thoracotomy (n=4) [2] (Figure 1).

All patients had central or peripheral cardiopulmonary bypass. Aortic cross-clamping was performed if a concomitant left-sided cardiac procedure was required (bioprosthetic aortic valve replacement (n=2), outflow graft anastomosis (n=1). An apical sewing ring was sutured to the left ventricular apex.

In all patients with bridge-to-transplantation strategy, pericardium was closed in front of the device using a GoreVR PrecludeVR pericardial membrane (W. L. Gore & Associates Inc., Flagstaff, AZ, USA) to facilitate sternal re-entry. Driveline was placed according to the double tunnel technique [9, 10]. The postoperative cardiac surgery course included pressure point mobilisation by nurses every 2 hours. After surgery, dressings were changed 3 times a week. Prophylactic anticoagulation was started 6 hours after surgery with intravenous heparin and raised to therapeutic levels after drain removal, then maintained as long as the LVAD was in place.

Surgical technique for the reconstruction of pressure sore

Surgical management included extensive debridement of infected and devitalized tissues associated with multiple samples for microbiology analysis [11]. Bulky and well-vascularized tissues were used to obliterate dead space and ensure stable coverage. Local reconstructive options such as V-Y advancement/rotation gluteal fasciocutaneous flaps, or gluteus maximus musculocutaneous flap were favored over more complex procedures due to the overall poor condition of patients. When needed, flaps were partially deepitheliazed to cover the dead space according to our previous experiences [12]. Quilting sutures were used to minimize dead space and protect from shearing forces. After surgery patients were bedridden for 3 weeks with change in decubitus every 2 hours, alternating dorsal, lateral and ¾ lateral decubitus.

Ambulation started with a physiotherapist at 3 weeks post-operatively, and was progressively increased. Prophylactic anticoagulation was started 6 hours after surgery with intravenous heparin, quickly increased to therapeutic dose. Patients receive antibiotics intraoperatively after multiple microbiology samples were sent for analysis. Postoperatively, antibiotics were first empiric, then adapted to microbiology results, for 4 to 6 weeks based on the infectious disease consultant advice and presence of osteitis. Osteitis was suspected clinically, and confirmed either radiologically, or by intraoperative bone sampling. Time to healing was defined as the time when stitches were removed. Follow-up was defined as the last visit in the outpatient clinic within the study period, with a minimum of 6 months.

Statistical Analysis

Patients' data were analysed using GraphPad Prism (version 8.0, GraphPad software, La Jolla, CA). Descriptive statistics included calculation of mean, standard deviation, standard error of the mean and range for continuous variables.

Results

During the study period, 42 patients benefited from LVAD implantation. Preoperative demographics and characteristics are summarized in table 1.

LVAD was implanted either as bridge to transplantation (n=31) (74%), destination therapy (n=7) (17%), or bridge to candidacy (n=4) (9%). Intraoperative cardiopulmonary bypass time averaged 73 ± 23 minutes (mean +/- SD).

Mean hospital stay after LVAD implantation was 53 ± 54 days (mean +/- SD) (range 18– 287 days; median 29 days).

Mean follow-up was 20 ± 13 months (mean +/- SD) (range 1–48 months). After LVAD implantation, 13 patients received a heart transplant (31%), 5 patients died (12%), while 25

patients were still on LVAD at the end of the follow-up period (60%). Cause of death was either cardiac arrhythmia, respiratory, renal or multiple organ failure. Actuarial survival after device implantation was 94.8 ± 3.6%, 88.4 ± 5.5%, and 84.4 ± 6.6% at 6, 12 and 24 months, respectively.

Decubital ulcers in LVAD population

Five patients (12%) developed a pressure sore. Among these, 4 patients had LVAD as bridge to transplantation and one as destination therapy.

Detailed characteristics of the patients' series are listed in table 2 including demographic data, etiology of heart failure, indication to LVAD, Intensive Care Unit stay and complications after LVAD implantation. Characteristics, management and outcomes of pressure sore in patients on LVAD are listed in table 3, including pressure sore timeline, size and location, length of stay and follow-up. Mean time from LVAD implantation to development of pressure sore was 25 +/- 8.2 days (mean +/- SEM). Overall, 4/5 patients were treated surgically (debridement + gluteal fasciocutaneous rotational flap (2/4), gluteal musculocutaneous rotational flap (1/4), debridement with VAC treatment (1/4)), and 1/5 conservatively with ointments.

Microbiologic data including microbiologic findings and treatment (antibiotic type and duration) are listed in table 4. Microbiology samples were sterile in the patient treated conservatively and positive in all patients who underwent surgery, with bone biopsies confirming osteitis.

Time between onset of the pressure sore to reconstruction ranged from 1 to 3 months (mean 64 +/- 19 days (mean +/- SEM)). 3/4 of patients treated surgically benefited from negative pressure wound therapy (NPWT) for 2 months (patient no. 1), and 3 weeks (patient no. 3).

Patient no. 4 was treated with debridement (without coverage) 2 months after the onset of the pressure sore, and negative pressure wound therapy for 4 months. He slowly healed within 6 months.

Among patients who benefited from flap surgery, just one had a single-stage procedure, while others had debridement and delayed coverage at one week. 2/3 patients who benefited from debridement and flap coverage had major complications requiring reoperation (1 hematoma, 1 major dehiscence) (appendix 1). Patient no. 1 had a cardiac arrest during the reconstructive procedure during preoperative mobilization to lateral decubitus, from which he completely recovered without sequelae. Further, due to early mobilization on POD 7 combined with therapeutic anticoagulation, he developed a hematoma, requiring surgical drainage and flap readvancement in lateral decubitus. Anticoagulation was progressively reintroduced after surgery and strict bedrest was advised for three weeks. Despite these protective measures, he presented a new wound dehiscence three weeks after surgery. We finally raised a contralateral gluteus maximus fasciocutaneous rotation flap to cover the defect without tension. Surgery was performed in ventral decubitus with uneventful healing (figure 2).

Patient no. 2 was mobilized intensively 3 weeks after surgery with a physiotherapist, including indoor cycling, and developed a wound dehiscence requiring flap readvancement for wound closure. He then healed uneventfully.

Mean time to healing in patients treated with flap coverage was 6 +/- 1.5 weeks (mean +/- SEM). The patient who received a conservative treatment did not heal until he died of cardiac-related complications after 3 months.

Global average hospital stay in patients treated with debridement +/- flap coverage was 4.7 +/- 0.4 months (mean +/- SD). Mean hospital stay after pressure sore coverage by reconstructive procedures of 2.7 +/- 0.4 months (mean +/- SD). Mean follow-up was 24 +/-

8.1 months (mean +/- SEM) (range 12-48 months). Patient no. 5 who died prematurely due to cardiac-related condition was excluded from calculation of this mean.

Discussion

Due to the decline in organ donations and scarcity of healthy donor hearts, the treatment of End Stage Heart Failure (EHSF) with mechanical circulatory support devices such as LVADs is gaining popularity. Despite technical advances, LVAD is unable to simulate the systolo-diastolic physiologic pulse that is replaced by a continuous blood flow. Moreover, infection remains a considerable complication after implantation affecting 95% of LVAD patients, among which 50% were specifically LVAD-related [2]. Since infection significantly increases morbidity and mortality to LVAD patients, it should be prevented and treated promptly if present [13].

Incidence of pressure ulcer in patients after cardiac surgery is reported to reach a global incidence of 11%, with 80% of stages I-II and 20% of stages III or IV[14]. These values differ significantly from those experienced in our series of patients requiring circulatory assistances using LVADs, with a 6-times folds increase of *surgical* pressure sores.

General risk factors for pressure sores include length of stay in the ICU > 3 days, vasoactive agents, need for transfusion, mechanical ventilation, prolonged sedation, need for mechanical circulatory support, lack of adequate repositioning [15, 16]. Preventive measures include load reduction with frequent mobilization, air-mattress, global optimization of patient conditions (reduction in vasopressors, improved nutrition and hygiene). Patient optimization before and after LVAD implantation is crucial to prevent pressure sore and promote wound healing. Nutritional evaluation by a dietetician is routinely performed. High-protein oral nutritional supplements and substitution of deficiencies, including micronutrients (vitamin A, B12, C, E, K, Zinc, Selenium, Copper, Manganese, Magnesium, and Phosphorus) can decrease the

incidence of pressure sore by 25%, and increase healing rate. Daily recommended protein intake ranges from 1.25 to 1.5 g/kg/day, moving up to 1.5-2 g/kg/day in patients with stage III-IV pressure sores [17]. Preoperative optimization includes smoking cessation ideally 3 months prior to LVAD implantation, even though cessation is tolerated up to 1 month before surgery. Recommended hemoglobin prior to LVAD implantation is 100 g/l. After flap coverage, we also aim a hemoglobin > 100 g/l according to current literature[18]. Other comorbidities including chronic renal failure, diabetes control, chronic pulmonary disease must be optimized before LVAD surgery to minimize the risk of postoperative complications. Patients undergo a full clinical and biological workup before LVAD implantation and strict follow-up during their hospital stay[19].

However, no consensus or clear guidelines exist regarding optimal pressure sore management and timing, especially in the ICU or patients undergoing cardiac surgery. It should be noted that prevention is often not optimal since mobilization is usually limited in this population shortly after cardiac surgery, due to potential hemodynamic instability and poor overall condition.

Our experience with rapid development of pressure sores seen in the LVAD population may be a manifestation of the maladaptive blood flow induced by LVADs, combined with the bedridden condition of these patients after LVAD implantation. Interestingly, patients who developed pressure sore complications did not present concomitant LVAD hemodynamic dysfunctions. In physiologic conditions, arterial pulsatility intrinsically delivers kinetic energy to the blood flow improving peripheral microcirculation, lymphatic flow and tissue perfusion. Moreover, shear stress induced by pulsatile flow has positive effects on the structure and function of blood vessels [20]. Pulsatile flow stimulates the release of nitric oxide and other vasoactive substances that induce vasodilatation, and allows blood to reach

peripheral tissues such as skin [20]. Although LVAD improves central hemodynamics compared to preoperative characteristics of patients with severe heart failure (who already have a poor peripheral vascular function compared to control healthy subjects), it worsens peripheral vascular function [20]. A study measured finger plethysmography in patients with severe heart failure before and after LVAD implantation [21]. It showed significantly lower values of reactive hyperemia in the LVAD group, a value that has been shown to correlate with higher level of vascular endothelial dysfunction and vascular abnormality [21]. Moreover, LVAD has a mechanical effect on blood producing cellular debris that contribute to endothelial dysfunction, promote inflammation and lead to reduced capacity of skin and muscle for wound healing [21].

Pressure ulcer can be a frightful complication as infection and potential may severely impact LVAD-equipped patients.

Pressure sore stage I-II are usually treated conservatively, while stage III-IV usually require surgery which prevents infection and protein loss [22, 23]. In our population, we observed a rapid progression of skin necrosis towards stage III-IV pressure ulcers after initial signs of skin sufferance, despite preventive measures. Chen et al. recommend an intensive multidisciplinary optimization of the patient and meticulous wound care for 1 to 2 weeks before performing reconstructive surgery [24]. Negative Pressure Wound Therapy (NPWT) is usually used as a bridge to final closure, until wound is clean and granulating, without recommendation on optimal duration of the therapy [25]. In our series, one patient was successfully treated by NPWT, not requiring flap surgery, despite long healing time.

For stage III-IV pressure ulcers, after debridement of devitalized tissues, standard coverage options include local advancement, rotational or V-Y advancement gluteal flaps (fasciocutaneous or musculocutaneous) [26, 27]. Occasionally, perforator flaps such as lumbar

artery perforator flap, or paraspinal perforator flap can be used for lesions higher than the sacral area. Simple and effective coverage options should be favored if patients are in poor overall condition [28, 29].

In our experience, initial signs of pressure sores in LVAD-equipped patients should be taken in consideration with particular attention, as rapidly evolving into surgical pressure sore stage and needing an aggressive surgical treatment in the majority of the cases (80%). 66% of patients who underwent reconstruction of the pressure sore developed complication, comparably to complication rates after pressure sore surgical coverage in the literature[14]. Unfortunately, there are no specific research focusing on outcomes of pressure sore in the cardiac surgery population to our knowledge. Pressure sore recurrence after surgery can reach 80% in some series, mainly in nonambulatory patients after spinal cord injury[30]. In our study, all patients requiring flap surgery finally achieved effective wound closure and could be kept on transplant list.

Limitations of the study are related to the relatively low number of patients in this specific population, despite being the largest HeartMate LVAD series in the literature to our knowledge. Based on our experience, we present a management algorithm for pressure sore in this specific population (figure 3).

Conclusion

Considering the potentially devastating risk of pressure sore-related sepsis in patients equipped with LVAD, surgery should be considered early to treat deep pressure sore in this population.

Disclosure

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Ethical approval

Not required

Declaration of Competing Interest

None declared

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Table 1. LVAD population preoperative demographics and characteristics

Preoperative data		
Age	57 +/- 12 (14-74)	mean +/- SD (range)
Gender - Male - Female	39 (93%) 3 (7%)	
Indication to LVAD - Bridge to transplantation - Destination therapy - Bridge to candidacy	31 (74%) 7 (17%) 4 (9%)	
Etiology of heart failure • Ischemic heart disease o Acute myocardial infarction < 3 months o Dilated ischemic cardiomyopathy • Primary cardiomyopathy o Dilated	26 (62%) 6 20 15 (36%) 14 1 1 (2%)	

○ Hypertrophic Valvular heart disease		
Previous cardiac surgery - Coronary artery bypass surgery - Mechanical mitral valve replacement - Mechanical aortic valve replacement	8 (19%) 5 (12%) 2 (5%) 1 (2%)	
History of stroke	7 (17%)	
NYHA stage - III - IV	17 (41%) 25 (59%)	
Laboratory - Albumin (g/l)	35 +/- 9	mean +/- SD

Table 2. Demographic data of patients who developed a pressure sore on LVAD

Patient number	**Age**	**Etiology of heart failure**	**Indication to LVAD**	**Comorbidities**	**ICU stay**	**Complications after LVAD**
1	51	Valvular, hypertensive and metabolic	Bridge to transplantation	PAD, metabolic syndrome, hyperuricemia	6	Cardiac arrest
2	54	Cardiac arrest after obstruction of a triple	Destination therapy	PAD, metabolic syndrome,	25	Cardiac arrest, hemothorax, hepatitis,

		coronary artery bypass		smoking, chronic renal insufficiency, obstructive sleep apnea		gastritis, gastrointestinal hemorrhage, urosepsis, stroke, polyneuropathy
3	52	Failure of revascularization after NSTEMI	Bridge to transplantation	DM2, sp traumatic splenic rupture, costal fractures, AKI	27	Bilateral pleural effusion, hemothorax, VAP, hepatitis, hemorrhagic and vasoplegic shock
4	62	S/p multiple coronary bypass	Bridge to transplantation	Metabolic syndrome, obstructive sleep apnea	20	LVAD cable infection (MSSA)
5	57	Idiopathic dilated cardiomyopathy	Bridge to transplantation	Obesity, DM2, chronic renal insufficiency, obstructive sleep apnea	75	Multiple hemorrhagic shock, VAP, hepatitis, urosepsis

Mean	55				30	
SEM	1.98				11.6	

*AKI: acute kidney injury; DM2: diabetes metillus type 2; HTA: hypertension; ICU: intensive care unit; metabolic syndrome: HTA, DM2, dyslipidemia, obesity; MSSA: methicilline-sensitive staphylococcus aureus; PAD: peripheral arterial disease; VAP: ventilator-associated pneumonia

Table 3. Characteristics and outcomes of pressure sore in patients on LVAD

Patient no.	**Onset of pressure sore after LVAD (POD)**	**Size, stage and location of sore**	**Management**	**NPWT**	**Outcome**	**Length of stay**	**Follow-up**
1	19	Sacral stage IV (13x15 cm)	Debridement, flap coverage	2 m	Complicated but final complete healing	5.5 m	18
2	10	Sacral stage III (10x12 cm)	Debridement, flap coverage	-	Complicated but final complete healing	4.5	18
3	40	Sacral	Debridement,	3 w	Favourable	4	12

		stage IV (7x6 cm)	flap coverage				
4	6	Ischiatic stage III (8x6 cm)	Debridement without coverage	4 m	Slowly favourable	3	48
5	48	Sacral stage IV (10x8 cm)	Conservative with local wound care	-	Death from cardiac-related condition (3 m)	(3)	(3)
Mean	**25**					**4.7**	**24**
SEM	8.2					0.4	8.1

*m: month; no.: number; NPWT: negative pressure wound therapy; POD: post-operative day; w: week

Table 4. Microbiological findings of pressure sore on LVAD

Patient no.	**Microbiological findings**	**Treatment (type and duration)**
1	Chronic osteomyelitis Intra-operative bone sample: Bacteroides	Piperacilline-Tazobactam, Cefepime, Trimethoprim Sulfamethoxazol 6 weeks

	Fragilis	
2	Chronic osteomyelitis Primary coverage, deep tissue: Pseudomonas Aeruginosa, Enterococcus Faecalis. Take back, bone sample : Proteus Vulgaris, Enterococcus Faecalis	Meronem, Trimethoprim Sulfamethoxazol, 6 weeks after flap coverage
3	Deep tissue, intra-operative sample, Escherichia Coli, Klebsellia Pneumoniae Bone sample: sterile	Amoxicillin-Clavulanic Acid, 7 days after flap coverage
4	LVAD cable infection, MSSA Intra-operative deep tissue sample:	Flucloxacilline then doxycycline (for LVAD cable infection) Suppressive until LVAD withdrawal

	Bacteroides fragilis	
5	Bedside sample: sterile	-

*MSSA: methicilline-sensitive Staphylococcus Aureus

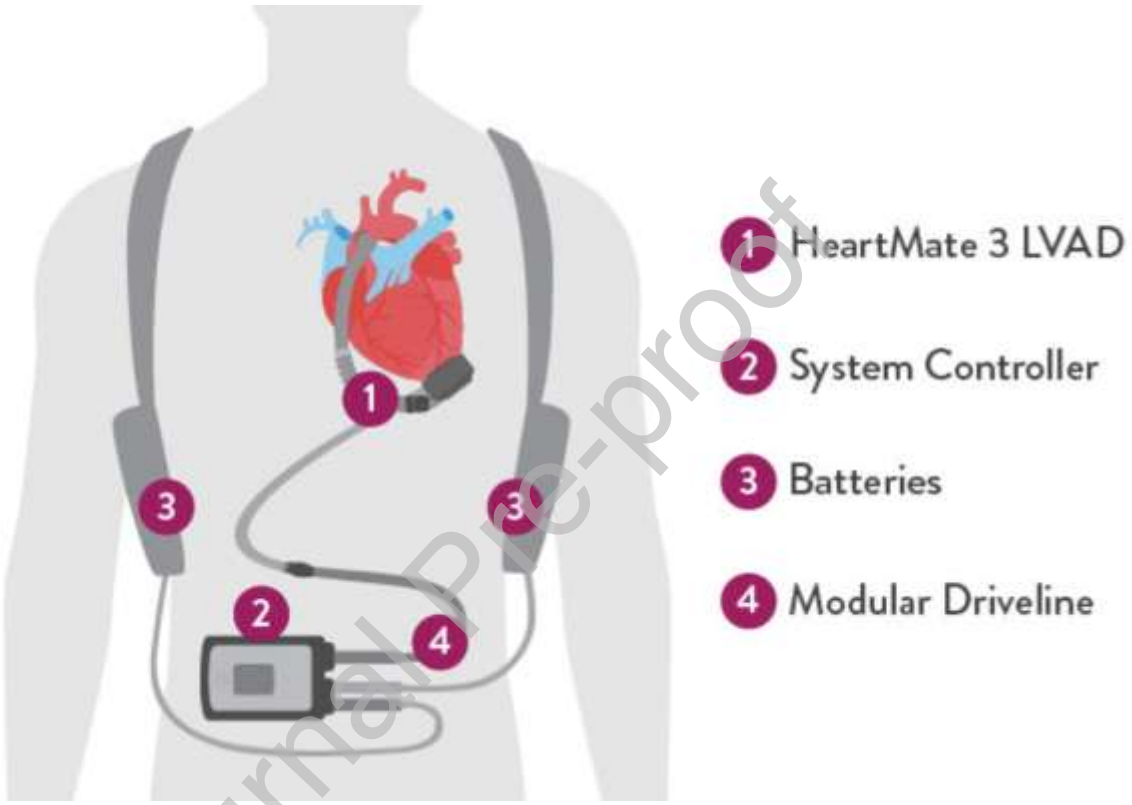

Figure 1. The Left Ventricular Assistance Device (LVAD) (Heartmate (HM) III□, Abbott, USA) is implanted between the left ventricular apex and the descending aorta. Image available at www.cardiovascular.abbott/us

Figure 2. Long-term stable coverage was finally achieved in patient no. 1 after 18 months follow-up

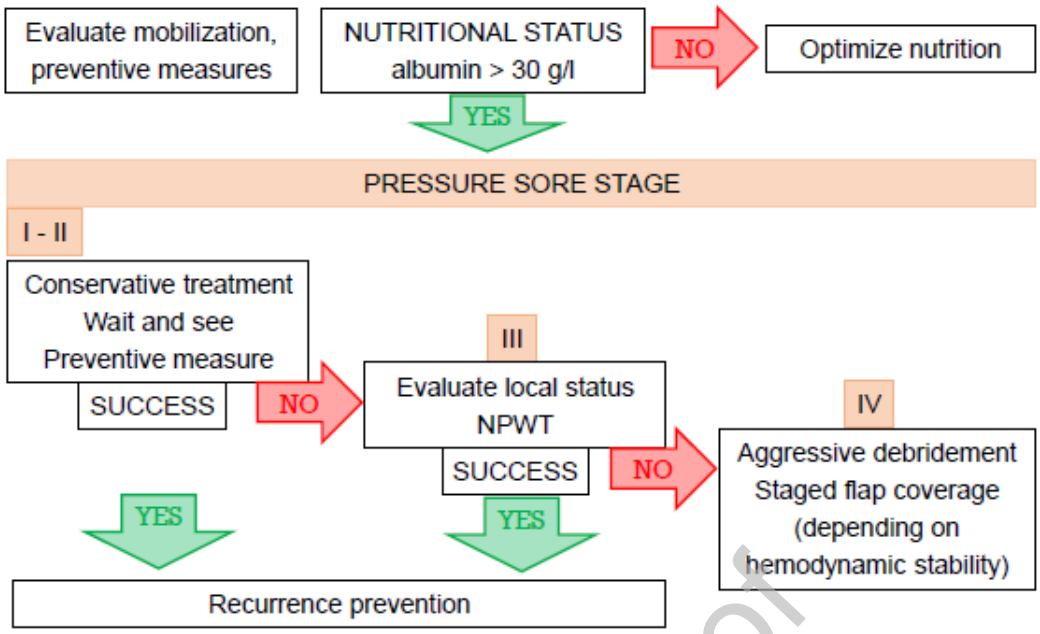

Figure 3. Management algorithm of pressure sore in patients on LVAD