Heart failure while on ventricular assist device support: A true clinical entity?

Insuffisance cardiaque chez les patients sous assistance circulatoire mécanique : une vraie entité clinique?

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Summary Ventricular assist devices (VADs) have become an established therapeutic option for patients with end-stage heart failure. The appearance of heart failure in VAD patients seems unexpected. Nevertheless, this phenomenon is not rare. We report six cases of VAD patients with clinical presentation of heart failure at different times after implantation and describe the mechanisms involved. The aetiology of this heart failure, like its clinical presentation, varies and has yet to be identified.

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Résumé L’assistance circulatoire mécanique (ACM) de longue durée est devenue un traitement de l’insuffisance cardiaque terminale. Elle améliore la qualité de vie et la survie de ces patients. L’apparition d’une insuffisance cardiaque chez les patients assistés est inattendue. Pourtant ce phénomène n’est pas rare. Il est important d’identifier cette insuffisance cardiaque et de préciser les mécanismes afin de proposer un traitement adapté. Les objectifs

Abbreviations: BIVAD, biventricular assist device; CI, cardiac index; CVP, central venous pressure; HF, heart failure; INTERMACS, Interagency Registry for Mechanically Assisted Circulatory Support; LV, left ventricular; LVAD, left ventricular assist device; NYHA, New York Heart Association; PCWP, pulmonary capillary wedge pressure; rpm, revolutions per minute; RV, right ventricular; RVAD, right ventricular assist device; RVF, right ventricular failure; TAH, total artificial heart; VAD, ventricular assist device.

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Background

Ventricular assist devices (VADs) have become an established therapeutic option for patients with end-stage heart failure (HF) [1,2]. VADs support either the left ventricle (LVAD) or both ventricles (BiVAD) [3]. The output of these devices is able to provide a systemic flow close to normal values at rest. Owing to this capability, the occurrence of HF during mechanical support is not expected.

HF is defined as a clinical syndrome characterized by specific symptoms (dyspnoea and fatigue) in the medical history and by signs of fluid retention (pulmonary congestion and peripheral oedema) on physical examination [4,5]. The usual laboratory tests can help to confirm the diagnosis but their use is limited in VAD patients [6]. The assessment of filling pressure in echocardiography remains difficult because the validity of standard variables is uncertain in severe dilated cardiomyopathy [7] and difficult to prove in LVAD patients when an inflow cannula is implanted in the left ventricle. The use of pulmonary artery catheterization (PAC) can be dangerous in BiVAD patients and is contraindicated in continuous BiVAD and total artificial heart (TAH) patients because it is potentially fatal [8,9]. We considered acute episodes of left HF, right HF and global HF. The diagnosis was made when dyspnoea with rales and pulmonary congestion was present on X-ray examination (with no indication of infection or acute lung injury) and/or peripheral oedema. The diagnosis was consolidated with increased central venous pressure (CVP) and/or pulmonary capillary wedge pressure (PCWP) when right catheterization or CVP measurements were possible [10].

If HF occurs, its potential impact on patient outcome can be very significant, in terms of prolonged mechanical ventilation plus its consequences, as well as increased risk of infection multiorgan failure, delayed rehabilitation and longer duration of hospitalization. There are no data in the literature on HF in patients with VADs.

We report six cases in which VAD patients presented HF at different times after implantation and try to describe the different causes of the clinical presentation.

Clinical summaries

HF within the first week post-VAD insertion

Patient 1: HeartWare LVAD — multifactorial HF

A 66-year-old man with ischaemic cardiomyopathy and low ejection fraction had presented New York Heart Association (NYHA) stage IV symptoms since 2009 (Tables 1 and 2). He had chronic renal insufficiency (creatinine 1.8 mg/dL). PAC in June 2009 showed a cardiac index (CI) of 1.6 L/min/m². In November 2010, the patient received a HeartWare LVAD as destination therapy because he developed new cardiogenic shock with progressive decline on inotropic drugs (Interagency Registry for Mechanically Assisted Circulatory Support [INTERMACS]) 2). INTERMACS levels are clinical stages that were developed and implemented into the first year of data collection for the INTERMACS [11]; they are helpful for evaluating the prognosis of patients after VAD implantation. Each level defines clinical status: level 1, critical cardiogenic shock; 2, progressive decline; 3, stable but inotrope dependent; 4, recurrent advanced HF; 5, exertion tolerance; 6, exertion limited; and 7, advanced NYHA III [11].

The PAC measurements on the day of implantation while the patient received inotropic drugs were: CI, 2.26 L/min/m²; CVP, 4 mmHg; and PCWP, 12 mmHg. On the day of surgery the patient received 5570 L of fluids and diuresis was 3350 L. After LVAD implantation, the patient was immediately extubated but ventilation performance was impaired. The estimated flow of the LVAD was 4.9 L/min at 3900 revolutions per minute (rpm). The CVP was 18 mmHg and the PCWP was 27 mmHg. X-ray examination indicated alveolar-interstitial oedema. The patient was treated by non-invasive ventilation and intravenous diuretics. The status of the patient deteriorated further and he needed mechanical ventilation. The patient was finally extubated after 15 days. He was rehabiliated successfully and discharged after a total of 6 weeks of hospital support.

The symptoms could be explained by impaired renal function, relative volume overload and the long period of low cardiac output syndrome.

Patient 2: Biventricular Thoratec VAD — multifactorial HF

A 41-year-old man presented with a 6-month history of dilated cardiomyopathy and a thrombus in the left ventricle. The thrombus was evacuated by ventriculotomy at the end of September 2010 but weaning from the bypass was impossible. The patient received venoarterial extracorporeal membrane oxygenation. Haemodynamic status and echocardiographic findings precluded weaning. The patient received a biventricular Thoratec paracorporeal VAD (BiVAD) because of biventricular dysfunction at the beginning of November 2010; his respiratory status improved and he was extubated after 3 days. Nevertheless, he needed oxygen and developed peripheral oedema. The right ventricular assist device
Table 1. Summary of patients.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>INTERMACS</th>
<th>Type of VAD</th>
<th>Timing of appearance</th>
<th>Clinical presentation</th>
<th>Aetiology of HF</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66</td>
<td>2</td>
<td>LVAD HeartWare</td>
<td>1 week</td>
<td>Pulmonary oedema</td>
<td>Multifactorial</td>
<td>Discharged</td>
</tr>
<tr>
<td>2</td>
<td>41</td>
<td>1</td>
<td>BiVAD Thoratec</td>
<td>1 week</td>
<td>Pulmonary and peripheral oedema</td>
<td>Multifactorial</td>
<td>Discharged</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>3</td>
<td>LVAD</td>
<td>2 weeks</td>
<td>Pulmonary oedema</td>
<td>RVF</td>
<td>Discharged</td>
</tr>
<tr>
<td>4</td>
<td>41</td>
<td>1</td>
<td>BiVAD HeartWare</td>
<td>1 month</td>
<td>Pulmonary oedema</td>
<td>Pulmonary overflow of RVAD and tamponade</td>
<td>Multifactorial</td>
</tr>
<tr>
<td>5</td>
<td>58</td>
<td>2</td>
<td>TAH</td>
<td>2 months</td>
<td>Pulmonary and peripheral oedema</td>
<td>Device malfunction</td>
<td>Death</td>
</tr>
<tr>
<td>6</td>
<td>72</td>
<td>—</td>
<td>LVAD VentrAssist</td>
<td>1 year</td>
<td>Pulmonary and peripheral oedema</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

BiVAD: biventricular assist device; HF: heart failure; LVAD: left ventricular assist device; RVAD: right ventricular assist device; RVF: right ventricular failure; TAH: total artificial heart; VAD: ventricular assist device.

Table 2. Biological variables.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Creatinine (mg/dL)</th>
<th>BUN (mg/dL)</th>
<th>Plasma protein (g/dL)</th>
<th>ASAT (IU/L)</th>
<th>ALAT (IU/L)</th>
<th>PT (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Before</td>
<td>1.8</td>
<td>62</td>
<td>6.1</td>
<td>27</td>
<td>31</td>
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<tr>
<td></td>
<td>At the time of HF</td>
<td>1.9</td>
<td>74</td>
<td>6.0</td>
<td>87</td>
<td>15</td>
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<tr>
<td>2</td>
<td>Before</td>
<td>2.1</td>
<td>205</td>
<td>4.5</td>
<td>17</td>
<td>23</td>
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<tr>
<td></td>
<td>At the time of HF</td>
<td>1.8</td>
<td>180</td>
<td>4.5</td>
<td>14</td>
<td>2</td>
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<tr>
<td>3</td>
<td>Before</td>
<td>0.81</td>
<td>13</td>
<td>6.2</td>
<td>28</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>At the time of HF</td>
<td>0.7</td>
<td>30</td>
<td>6.2</td>
<td>73</td>
<td>64</td>
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<tr>
<td>4</td>
<td>Before</td>
<td>1.3</td>
<td>79</td>
<td>6.6</td>
<td>5830</td>
<td>2910</td>
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<tr>
<td></td>
<td>At the time of HF</td>
<td>0.64</td>
<td>32</td>
<td>5.9</td>
<td>115</td>
<td>92</td>
</tr>
<tr>
<td>5</td>
<td>Before</td>
<td>2.1</td>
<td>157</td>
<td>5.6</td>
<td>293</td>
<td>220</td>
</tr>
<tr>
<td></td>
<td>At the time of HF</td>
<td>1.3</td>
<td>100</td>
<td>6.1</td>
<td>36</td>
<td>22</td>
</tr>
<tr>
<td>6</td>
<td>Before</td>
<td>0.86</td>
<td>30</td>
<td>7.4</td>
<td>33</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>At the time of HF</td>
<td>3.1</td>
<td>116</td>
<td>6.7</td>
<td>751</td>
<td>746</td>
</tr>
</tbody>
</table>

ASAT: aspartate aminotransferase; ALAT: alanine aminotransferase; BUN: blood urea nitrogen; HF: heart failure; PT: prothrombin time.

(RVAD) flow was 4.8 L/min and the LVAD flow was 6.6 L/min. Systolic arterial pressures were about 90 mmHg and CVP was 20 mmHg. Renal function was impaired (creatinine 2.1 mg/dL) and plasma protein was decreased (4.5 g/dL). Echocardiography eliminated tamponade and radiography showed alveolar-interstitial oedema. Oedema decreased after 4 days of intravenous high-dose diuretics. Thirty days after the operation, the patient was discharged with BiVAD support.

The clinical presentation could be explained by the long period of low cardiac output syndrome, impaired renal function, poor nutritional status and vasoplegia induced by the two cardiac surgical interventions.

HF within the first month post-VAD insertion

Patient 3: HeartWare LVAD — right ventricular (RV) failure

A 24-year-old man was diagnosed with dilated cardiomyopathy and low ejection fraction (< 25%) present for 6 weeks and of unknown origin. He was hospitalized at the beginning of January 2011 for a first episode of cardiogenic shock with impossibility of weaning from inotropic drugs (INTERMACS 3). PAC (with inotropic drugs) before the implantation revealed: Cl, 2.68 L/min/m²; PCWP, 13 mmHg; and CVP, 5 mmHg. The patient was a drug addict and we decided to implant a HeartWare LVAD as a bridge to a decision.
At day 8, the patient presented respiratory insufficiency; CVP was 16 mmHg and the flow of the LVAD was unchanged at around 5 L/min (for a speed of 2800 rpm). Transthoracic echocardiography showed that the left ventricle was not dilated but that the right ventricle was dilated with severe impairment of systolic function.

After reduction of the LVAD flow (2600 rpm) and inotropic drugs, the patient’s status improved. Transthoracic echocardiography showed that the RV diameter was decreased and the left ventricular (LV) diameter was increased.

We deduced RV failure (RVF) worsened by too high a flow speed.

Patient 4: Biventricular HeartWare VAD — pulmonary overflow of the RVAD and tamponade

A 41-year-old woman with dilated cardiomyopathy of unknown aetiology presented in October 2010 with refractory cardiogenic shock (INTERMACS 1). HeartWare BIVAD implantation was performed because of biventricular dysfunction and the primary postoperative course was uneventful.

At day 21, the patient had pulmonary insufficiency without sign of infection. RVAD flow was 5.2 L/min (2400 rpm) and LVAD flow was 6.6 L/min (2600 rpm). The measurement of CVP showed a pressure of 16 mmHg. X-ray examination showed alveolar-interstitial pulmonary oedema. Echocardiography eliminated tamponade.

We concluded that there was pulmonary overflow of the RVAD and the course was favourable with an increase in left pump flow (2900 rpm) and 2 days of intravenous diuretics.

After 4 weeks (day 29), the patient developed dyspnoea with orthopnoea a few hours after withdrawal of the epicardial electrode. The flows decreased: RVAD flow was 3.3 L/min (2400 rpm) and LVAD flow was 3.9 L/min (2900 rpm). CVP increased to 19 mmHg. X-ray investigation showed cardiomegaly (Fig. 1). Tamponade was suspected and confirmed by echocardiography. The patient underwent emergency surgery, the haematoma was evacuated and haemodynamic status stabilized immediately (RVAD flow 4.6 L/min and LVAD flow 5.8 L/min). The patient was rehabilitated.

Patient 5: CardioWest total artificial heart — multifactorial HF

A 56-old-man presented with a 15-year history of valvular cardiomyopathy with aortic stenosis and severe LV dysfunction. He had chronic renal insufficiency (creatinine 2.1 mg/dL). In September 2010, clinical deterioration led to implantation of biventricular support; a CardioWest TAH was selected due to severe calcifications of the ascending aorta. Immediate follow-up was complicated by tamponade, which required new surgical intervention, and by cerebral bleeding. For cerebral protection, the patient received hypertonic serum (mannitol) for 3 days and diuretics were stopped. One week after the neurological event, the patient developed clinical signs of HF with dyspnoea and peripheral oedema. The TAH flow was unchanged at 8.6 L/min (with a vacuum of 9 mmHg and a fixed beat of 130/min). CVP was 24 mmHg. X-ray investigation showed alveolar-interstitial pulmonary oedema. Thoracic computed tomography eliminated tamponade. After 3 days of treatment with intravenous diuretics, the signs disappeared. We deduced volume overload worsened by renal failure.

One month later, the patient again presented clinical signs of HF with dyspnoea and peripheral oedema. The TAH flow was 8.2 L/min (with a vacuum of 9 mmHg and a fixed beat of 130/min). CVP was 19 mmHg. X-ray investigation showed alveolar-interstitial pulmonary oedema. Renal function was impaired. Blood pressure was not completely controlled (systolic arterial pressure around 130 mmHg). Symptoms improved after diuretic therapy and antihypertensive agents.

The clinical presentation could be explained by impaired renal function and uncontrolled blood pressure. The patient was finally discharged with TAH support.

HF after the first year post-VAD insertion

Patient 6: HeartMate II LVAD — device malfunction

A 72-year-old man received a HeartMate II LVAD as destination therapy in 2009 for ischaemic cardiomyopathy. Follow-up was uneventful and the patient lived with his wife at home. The patient was readmitted on December 2010 due to stage IV dyspnoea, oedema and ascites. Pump flow was low (3.6 L/min for 2900 rpm) with a decreased pulsatility index (13) and normal power (3.5 watts). PAC showed a CI of 1.5 L/min/m², a CVP of 27 mmHg and a PCWP of 31 mmHg. X-ray examination showed cardiomegaly with alveolar-interstitial oedema. The international normalized ratio was 6.6. Renal and hepatic functions were impaired. Lactate dehydrogenase was high (793 IU/L) whereas free haemoglobin was normal. Echocardiography showed dilatation and severe dysfunction of both ventricles and an opened aortic valve. A mechanical dysfunction of the pump was
suspected but it was decided not to operate and to attempt medical treatment (inotropic drugs, diuretics and haemodilution). The patient died.

Post-mortem analysis of the pump showed a massive thrombosis of the apical cannula (Fig. 2).

Discussion

The six case reports illustrate the different possible aetiologies of a clinical presentation of HF in patients with a VAD. In the postoperative course of VAD implantation, a clinical presentation of HF is not rare. The mechanisms may be complex, with multiple contributing factors [12].

Different mechanisms of oedema syndrome

Cardiogenic shock is accompanied by the production of numerous inflammatory cytokines (interleukin-1β, interleukin-6, interleukin-8, tumour necrosis factor-α, C-reactive protein, soluble adhesion molecules, complement system) due to expression of inducible nitric oxide synthase [13,14]. This activation of the inflammatory response causes an alteration of vascular permeability and participates in the formation of oedema. These signs are worsened by vasoplegic syndrome, which occurs after cardiac surgery with an incidence ranging between 5% and 25% [15,16]. Vasoplegic syndrome is due to a systemic inflammatory response and is characterized, among other things, by low systemic vascular resistance [17].

Moreover, in the early postoperative period, LVAD results in decreased RV function. RV preload is reduced because of the improvement in cardiac output. The afterload increases due to the septal shift (causing increased RV wall stress), secondary to LVAD implantation, pulmonary vasoactivity, blood transfusion and inflammation induced by surgery [18–20]. The decreased RV function contributes to the appearance of HF signs.

Preoperatively, patient status was often poor, with cardiogenic shock accompanied by multiple organ failure, particularly renal and hepatic failure [1,21]. After VAD implantation, the impaired organs need time to recover. In a study conducted by Friedel et al., in 61 patients assisted by a BiVAD (n = 54), an LVAD (n = 5) and a TAH (n = 2) in order to bridge to transplantation, functional recovery of hepatic and renal failure was observed in 90% and 95% of cases [22]. The mean duration of organ recovery after mechanical circulatory support was between 10 and 15 days. Nevertheless, in this study, 21 patients died because of multiorgan failure or septic complications.

Impaired renal function helps to maintain and worsen the signs of HF after VAD implantation. Heart and kidney performance are closely inter-related physiologically and pathophysiologically, both in health and in disease [23–25]. Before VAD implantation, for the patient with HF and volume overload, the combination of high pulmonary artery or central venous pressure with low systemic pressure alters the renal perfusion pressure. Neurohormonal activation plays a major role in these phenomena. Mediated by activation of arterial baroreceptors and intrarenal sensors, it results in abnormal activation of the renin-angiotensin-aldosterone system, activation of the sympathetic nervous system and activation of the arginine-vasopressin system. All of these phenomena are responsible for increased preload and afterload (caused by vasoconstriction and sodium retention) and activation of nitric oxide synthase, leading to initiation of an inflammatory response and secretion of proinflammatory cytokines with negative inotropic effects. This vicious cycle damages the heart and kidneys. After VAD implantation, renal function remains impaired for a few weeks and leads to increases in preload and afterload. Impaired renal function is also responsible for a maladaptive response to overload volume [26].

Congestive liver dysfunction induces elevation of hepatic enzymes and hypoalbuminaemia [27]. In fact, hypoalbuminaemia is multifactorial and is caused by malnutrition, inflammation, cachexia, haemodilution, protein-losing enteropathy, increased transcapillary escape rate and nephrotic syndrome. According to Starling’s law, hypoalbuminaemia leads to a low plasma oncotic pressure, which induces a fluid shift from the intravascular to the interstitial space; it facilitates the onset of cardiogenic pulmonary oedema [28–30].

Finally, oedema can be caused by high hydrostatic pressure resulting from fluid overload during the perioperative period, which is usually mediated by neurohormonal renal salt and water retention. Systemic venous pressure increases and contributes to the cycle of oedema formation [12].

Nevertheless, the symptoms of HF can persist or appear later, whereas the patients recover normal hepatic and renal function. Other causes more specific to VAD patients can explain these presentations.

The load sensitivity of VAD

VADs are preload dependent and afterload sensitive [31–35].

The LVAD can be compared to a ‘normal heart’ in terms of preload dependence. For the same pump speed, when a VAD patient receives fluid, the flow/output of the LVAD is increased. From a certain threshold, when the right ventricle has failed, the LVAD cannot be filled; the flow decreases and clinical symptoms of HF appear [32,35].
Heart failure while on VAD support

With a BiVAD, RVF should not develop because the right ventricle is assisted. But when the flow of the RVAD is too high, the blood cannot be evacuated by the LVAD and pulmonary oedema can occur [34]. This situation is reversible after an increase of LVAD flow. Nonaka et al. recommended maintaining certain conditions during rotary pump BiVAD implantation to avoid this pulmonary congestion: a ratio between right and left VAD flow of < 1 and/or an average pulmonary artery flow rate < 160 mL/kg/min and systolic pulmonary artery pressure < 50 mmHg [33].

The use of continuous-flow HeartWare pumps as implantable BiVADs is more recent. In a study of eight patients, Hetzer et al. described overflow pulmonary oedema in one patient who died as a result [36]. LVAD and RVAD at the same speed can have different flows due to the difference in pulmonary vascular resistance and systemic vascular resistance. Thus, the right pump in a normal pulmonary resistance circuit would pump more volume than the left and induce pulmonary oedema.

All VADs, especially those with centrifugal and axial flow pumps, are sensitive to afterload [36]. Patients with implanted VADs recover sufficient output and can develop arterial hypertension [37]. From a certain level of blood pressure, the flow will decrease and pulmonary congestion can appear [31,35]. A mean arterial blood pressure > 75 mmHg leads to the opening of the aortic valve and inadequate functioning of the LVAD [38].

Specificity of the CardioWest TAH: heart insufficiency with a higher VAD flow

The CardioWest TAH is a biventricular orthotopic pneumatic pulsatile blood pump, which replaces the failing atria and ventricles of the heart as well as the proximal portion of each great vessel; it is driven by an external console. For each ventricle, the length of the blood-flow path is shorter and the inflow and outflow valves are larger than in any other bridge-to-transplant device, resulting in greater blood flow at smaller preload. The console settings include left ventricular and RV pressures of 190 and 70 mmHg, respectively. The settings of the TAH are relatively fixed: the beat ranges between 120 and 130/min; stroke volume is constant at 70 mL; the vacuum averages 9 mmHg; and the percentage systole averages 53%. The CardioWest TAH therefore has an output range of 7–9 L/min [39,40].

Orthotopic positioning eliminates concerns about problems resulting from the native heart, such as right HF, arrhythmias, problems with native and prosthetic heart valves, clots within the native ventricles, ventricular septal defects, rejection and infarction and stone heart. Nevertheless, signs of HF can happen. In the immediate follow-up after implantation, complications such as atrial compression and tamponade have to be eliminated. Later, the aetiology is multifactorial. Impaired renal function with inadequate diuresis can contribute to clinical signs of fluid retention. Like other VADs, the TAH is preload and afterload dependent. Due to its high output, the TAH can induce arterial hypertension, which may increase afterload and signs of HF. In summary, there are numerous mechanisms responsible for clinical signs of HF in TAH patients with high flow.

Oedema can be controlled with diuretic therapy, which has to be chronic.

Heart insufficiency and low VAD flow

In a clinical presentation of HF with decreased VAD flow, three diagnoses should be investigated: tamponade, RVF and VAD malfunction.

Tamponade

A clinical presentation of HF makes it necessary to exclude tamponade by echocardiography or thoracic computed tomography, especially if CVP is elevated without oedema syndrome. Tamponade is not rare after VAD implantation (up to 30%) and often occurs in the first days after implantation [41,42]; it increases the mortality of VAD patients [42].

Right ventricular failure

RVF is a severe problem in LVAD patients; its incidence varies from 7% to 50% and it is responsible for increased perioperative and 1-year mortality (19% to 43%) [19,43–45].

The pathophysiology of RVF is complex. In the long-term, LVAD improves RV function by LV unloading and decreasing LV filling pressure and pulmonary venous return. Nevertheless, in the early postoperative period, many mechanisms may contribute to RVF, as described at the beginning of discussion [18–20]. RVF leads to liver and renal failure, oedema and ascites [41,44]; it induces underfilling of the left ventricle and the pump with potential arrhythmia and cardiac failure [19,43–45].

Prediction of RVF after LVAD placement would lead to more precise patient selection and optimal device selection. Many studies have tried to identify factors that predict RVF after LVAD implantation [19,43–45]. Clinical and biological factors such as sex (female), dilated cardiomyopathy, previous cardiac surgery and biological factors reflecting hepatic and renal failure were associated with postoperative RVF in LVAD patients. Some haemodynamic and echocardiographic indices were assessed but the results were discordant [19]. Because of the multiplicity of factors contributing to RVF after LVAD implantation, some scores were developed [44,45]. However the prediction of postoperative RV dysfunction remains difficult.

Despite its effectiveness in the treatment of severe RVF, the use of biventricular mechanical support for end-stage congestive HF remains controversial because patients who require prolonged support with a BiVAD have a lower survival rate than recipients of a LVAD. In addition, BiVAD recipients have higher rates of major adverse events, such as thromboembolism, device infections and mechanical complications [19,43]. RVF after LVAD can also be managed medically with pulmonary vasodilators, including nitroprusside, nitric oxide and iloprost, and inotropes or with a temporary RVAD [46].

Device malfunction

Device malfunction is principally due to mechanical failure or thrombosis [42,44,47,48]; its incidence increases with time, with a cumulative probability of 6% at 6 months and 64% at 2 years [47]. Device malfunction should be considered when VAD flow is low with HF presentation. Sometimes,
the clinical presentation is associated with haemolysis and an increase in the power of the device. Device malfunction leads to death in <5% of cases but otherwise does require replacement of the mechanical circulatory support [33,34,36].

Management

Before the VAD implantation

Optimization of patient status before implantation should correct additional factors responsible for fluid retention. This involves optimization of liver and renal function, assessment of nutritional status and improvement, when necessary, of nutritional and metabolic status. Optimization of status before VAD surgery, however, is often not possible in these patients because implantation is often an emergency procedure (INTERMACS 1, 2 or 3). Some studies suggest that the risk of RVF can be decreased by preoperative optimization of nutrition, haemodynamics and organ function, and by minimization of RV preload with inotropic drugs and an intra-aortic balloon pump. Limiting bleeding and transfusion, avoiding surgical RV injury and distension, tricuspid annuloplasty, early cessation of positive pressure ventilation and RV afterload reduction could also improve RVF after LVAD [28,47].

After the VAD implantation

After the diagnosis of HF symptoms in VAD patients, it is important to discount complications that could require emergency surgery, such as tamponade or device malfunction.

Concerning RVF, when echocardiography has shown that the LV diameter is small, the aortic valve is closed and the right ventricle is dilated, a reduction in LVAD speed can improve RV function by increasing preload and decreasing septal shift. Overload volume should be avoided or corrected by diuretic therapy. Control of the afterload includes control of blood pressure. Mean arterial blood pressure in the range of 65 to 75 mmHg is recommended throughout LVAD support to avoid opening of the aortic valve and inadequate functioning of the LVAD [38]. Most patients show a good and fast response to intravenous diuretic therapy.

Finally, VAD patients should have strict blood pressure control and small doses of diuretics to avoid volume overload.

Conclusion

Our six case reports illustrate the different causes of signs of HF in VAD patients. In the postoperative course after VAD implantation, a clinical presentation of HF is not rare. VAD patients need time for recovery after implantation because they often have multiple organ failure and inflammatory reaction due to cardiogenic shock and cardiac surgery. Later, the appearance of HF should prompt examination for RVF or for complications associated with the use of a VAD. Registries such as European Registry for Patients with Mechanically Assisted Circulatory Support (EUROMACS) and INTERMACS could increase understanding of this important issue and assess the repercussions.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

Heart failure while on VAD support


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