TandemHeart as a Bridge to Recovery in Legionella Myocarditis

Legionnaires’ disease is the designation for pneumonia caused by the Legionella species. Among the rare extrapulmonary manifestations, cardiac involvement is most prevalent, in the forms of myocarditis, pericarditis, postcardiotomy syndrome, and prosthetic valve endocarditis. Mechanical circulatory support has proved to be a safe and effective bridge to myocardial recovery in patients with acute fulminant myocarditis; however, to our knowledge, this support has not been used in infectious myocarditis specifically related to Legionellosis.

We describe a case of Legionella myocarditis associated with acute left ventricular dysfunction and repolarization abnormalities in a 48-year-old man. The patient fully recovered after left ventricular unloading with use of a TandemHeart percutaneous ventricular assist device. In addition, we review the English-language medical literature on Legionella myocarditis and focus on cardiac outcomes. (Tex Heart Inst J 2015;42(4):357-61)

Legionnaires’ disease is the designation for pneumonia caused by the Legionella species. In addition to its pulmonary involvement, Legionella has been isolated in the heart, brain, lymph nodes, spleen, liver, and kidneys in autopsy studies. Extrapulmonary manifestations are rare; of these, cardiac involvement is most prevalent, in the forms of myocarditis, pericarditis, postcardiotomy syndrome, and prosthetic valve endocarditis. Reported arrhythmias include bradycardia, tachycardia, and conduction abnormalities. The severity of the illness depends on the organ systems involved; it is typically severe with cardiac involvement, especially in myocarditis.

We report the case of a patient who had Legionella myocarditis associated with acute left ventricular (LV) dysfunction and repolarization abnormalities, and we describe his treatment with use of a TandemHeart® percutaneous ventricular assist device (pVAD) (CardiacAssist, Inc.; Pittsburgh, Pa). In addition, we review the medical literature on Legionella myocarditis and focus on cardiac outcomes.

Case Report

In April 2013, a 48-year-old previously healthy man presented at a hospital with a 10-day history of cough, subjective fever, chills, myalgias, and generalized body aches. He was instructed to take oral antibiotics. However, he was readmitted the same night with worsening dyspnea, headaches, and hypoxemic respiratory failure that necessitated intubation. A chest radiograph and computed tomographic scans of the chest revealed extensive multilobar alveolar consolidation within the right lung (Fig. 1). Legionella infection was diagnosed on the basis of a positive urine antigen test. All other microbiological tests were negative, including bronchoalveolar lavage (gram stain, bacterial and viral cultures, and acid-fast bacilli) and Streptococcus pneumoniae urine antigen. Multiple viral studies were also negative: human immunodeficiency virus, parvovirus, Coxackievirus A and B, influenza, and herpes simplex virus 1 and 2. Despite broad-spectrum antibiotic therapy that included levofloxacin and azithromycin, the patient’s condition rapidly deteriorated, and his clinical course was complicated by acute renal and heart failure. A transthoracic echocardiogram (TTE) revealed dilated cardiomyopathy with severely depressed LV systolic function and severe mitral regurgitation. The patient’s LV internal dimensions in systole and diastole were 4.8 cm and 5.8 cm, respectively. The estimated LV ejection fraction (LVEF) was 0.10. Cardiac biomarkers were noted to be positive. The patient then developed atrial flutter with variable atrioventricular block (Fig. 2A), followed by...
reccurrent torsades de pointes; he underwent defibril-
lation twice and emergency intra-aortic balloon pump
placement for hemodynamic support. At that time, he
was started on anticoagulation with use of intravenous
unfractionated heparin.

The patient was transferred to our institution in re-
fractory cardiogenic shock for further treatment. He was
electrically unstable, with recurrent torsades de pointes
that was probably related to severe diffuse myocardial
injury and metabolic derangements. Marked QTc pro-
longation (550 ms) with intraventricular conduction
abnormalities precluded the use of amiodarone, so li-
docaine was used to suppress the ventricular tachyar-
rhythmias (Fig. 2B). The patient’s antibiotic therapy
was changed to doxycycline, and he was given 4 doses
of intravenous immunoglobulin (IVIG). In view of the
refractory cardiogenic shock and electrical instability
secondary to torsades de pointes, the patient was placed
on a TandemHeart pVAD. Subsequently, still on the
day of admission, TTE revealed severe systolic dysfunc-
tion (LVEF, 0.20–0.25), severe global hypokinesis with
paradoxical septal motion, and an akinetic inferior wall.
Coronary angiograms excluded coronary artery disease.
The patient improved after continued LV unloading
and hemodynamic support, and he was successfully
extubated and weaned from the TandemHeart after 4
days of total support. Because of atrial flutter, the anti-
coagulation was changed to warfarin (international nor-
malized ratio target, 2–3). The patient was discharged
<table>
<thead>
<tr>
<th>Reference</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Basis of Legionella Diagnosis</th>
<th>ECG</th>
<th>LVEF</th>
<th>During Event</th>
<th>After Event</th>
<th>Cardiac Biomarkers</th>
<th>Antibiotic Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nelson DP, et al. (1985)</td>
<td>52/M</td>
<td>Pericarditis</td>
<td>Serology</td>
<td>No</td>
<td>0.35</td>
<td>NA</td>
<td>Normal</td>
<td>Negative</td>
<td>Aspirin</td>
<td>NA</td>
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<tr>
<td>Armengol S, et al. (1992)</td>
<td>43/M</td>
<td>Myocarditis</td>
<td>Serology</td>
<td>Yes</td>
<td>Decreased</td>
<td>Normal</td>
<td>Sinus tachycardia and LBBB</td>
<td>Erythromycin</td>
<td>NA</td>
<td>Antibiotics</td>
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<tr>
<td>de Lasance, A, et al. (1994)</td>
<td>56/F</td>
<td>Myocarditis</td>
<td>DFA in sputum</td>
<td>Yes</td>
<td>0.46</td>
<td>Normal</td>
<td>Sinus tachycardia and LBBB</td>
<td>Vasopressors, antibiotics</td>
<td>NA</td>
<td>Antibiotics</td>
</tr>
<tr>
<td>Bodur H, et al. (2006)</td>
<td>43/M</td>
<td>Myocarditis</td>
<td>Urine antigen</td>
<td>Yes</td>
<td>0.55–0.60</td>
<td>NA</td>
<td>Sinus tachycardia and LBBB</td>
<td>Vasopressors, antibiotics</td>
<td>NA</td>
<td>Antibiotics</td>
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<tr>
<td>Antonarakis ES, et al. (2006)</td>
<td>45/M</td>
<td>Myocarditis</td>
<td>Urine antigen</td>
<td>Yes</td>
<td>0.15–0.20</td>
<td>0.40–0.45 at 6 mo</td>
<td>TWI in lateral leads</td>
<td>Positive</td>
<td>Negative</td>
<td>Antibiotics</td>
</tr>
<tr>
<td>Burke PT, et al. (2009)</td>
<td>50/F</td>
<td>Perimyocarditis</td>
<td>Urine antigen</td>
<td>No</td>
<td>0.25</td>
<td>NA</td>
<td>ST-segment elevation in lateral leads</td>
<td>Vasopressors, antibiotics, and NSAIDs</td>
<td>Positive</td>
<td>Antibiotics</td>
</tr>
<tr>
<td>Ishimaru N, et al. (2012)</td>
<td>32/M</td>
<td>Myocarditis</td>
<td>Urine antigen</td>
<td>Yes</td>
<td>0.37</td>
<td>Normal</td>
<td>Sinus tachycardia and LBBB</td>
<td>Antibiotics and IVIG</td>
<td>NA</td>
<td>Antibiotics and IVIG</td>
</tr>
<tr>
<td>Current case</td>
<td>48/M</td>
<td>Myocarditis</td>
<td>Urine antigen</td>
<td>Yes</td>
<td>0.10</td>
<td>0.60</td>
<td>AF with variable AV block, Qtc (550 ms), and intra-atrial conduction abnormalities</td>
<td>Antibiotics and IVIG</td>
<td>Positive</td>
<td>Negative</td>
</tr>
</tbody>
</table>

AF = atrial flutter, AV = atrioventricular, BAL = bronchoalveolar lavage, DFA = direct fluorescent antibody, ECG = electrocardiogram, LBBB = left bundle branch block, NSAIDs = nonsteroidal anti-inflammatory drugs, NA = not applicable, pVAD = percutaneous ventricular assist device, TWI = T-wave inversion.

*All patients except one survived.
from the hospital 3 weeks after admission, with instructions to take angiotensin-converting enzyme inhibitor and β-blocker medications. At that time, TTE revealed a normal LVEF (0.60) with normalization of LV internal dimensions in systole and diastole (3.3 and 5.3 cm, respectively), and mild mitral regurgitation. Six months later, the patient’s electrocardiographic results were normal, and he subsequently remained asymptomatic.

Discussion

We describe what we think is the first successful use of the TandemHeart as a bridge to recovery in *Legionella* myocarditis. In this uncommon presentation of *Legionella* infections, morbidity is usually high. Endomyocardial biopsy is usually recommended in the presence of myocarditis, but biopsies are performed infrequently because of the perceived risks and the lack of a widely accepted histologic standard. Although the results of a myocardial biopsy would have strengthened the diagnosis of *Legionella* myocarditis beyond the clinical, microbiologic, and echocardiographic findings, the risk to our patient was too high. Urinary antigen tests for the microbiologic, and echocardiographic findings, the risk to our patient was too high. Urinary antigen tests for *Legionella* have sensitivities from 70% to 100% and specificities approaching 100%. Their major disadvantage is their unreliable detection of organisms other than *Legionella pneumophila* serogroup 1.5

Myocarditis is a severe disorder and a frequent cause of heart failure. When myocarditis occurs sporadically, a viral or immune pathogenesis is usually suspected. Accordingly, high doses of IVIG have been given, but with discordant results. Some authors of case series have reported beneficial outcomes; however, the results of the prospective, randomized Intervention in Myocarditis and Acute Cardiomyopathy (IMAC) trial revealed that IVIG does not substantially improve LVEF in comparison with placebo in patients who have recent-onset idiopathic dilated cardiomyopathy or myocarditis.7 Hence, the main therapy is standard heart-failure medication, such as angiotensin-converting enzyme inhibitors or angiotensin receptor blockers and β-blockers. On the other hand, previous reports have described the use of the TandemHeart as a safe and effective bridge to myocardial recovery in patients with acute fulminant myocarditis.7

To our knowledge, this is the first case of a pVAD’s successful use as a bridge to recovery in a patient whose acute LV dysfunction and repolarization abnormalities were secondary to *Legionella* myocarditis. Our search of PubMed with use of the key term “Legionella myocarditis” yielded 13 articles. We analyzed the 7 that were accessible and in English (Table I). 5,10-12 Five cases were reported as myocarditis,5,10-12 one as perimyocarditis,12 and one as pericarditis.12 Half the patients who had acute LV dysfunction during presentation still had LV dysfunction after appropriate therapy. Of note, one patient still had decreased LV function 6 months later (LVEF, 0.40–0.45). Of 5 patients with abnormal electrocardiographic findings,5,10-12 only one had repolarization abnormalities (QTC, 500 ms), similar to our patient. None had been given mechanical circulatory support as a bridge to recovery. As in our case, one patient was given IVIG as part of his medical therapy, and he had a complete LV functional recovery.3

Risk is posed by the pVAD itself in the presence of sepsis, because the known risk factors of hemolysis, bleeding, and limb ischemia13 can overlap with disseminated intravascular coagulation caused by sepsis. In addition, the use of a pVAD implies anticoagulation, which substantially increases the bleeding risk in an already coagulopathic host. Therefore, strict clinical monitoring is essential, and adequate microbiological management is necessary. During 4 days of pVAD support with anticoagulation, no such sequelae were noted in our patient.

In conclusion, although myocarditis is a rare complication of *Legionella* infection, it can be serious or fatal. In patients with myocarditis that leads to cardiogenic shock and electrical instability, short-term mechanical circulatory support should be considered early as a bridge to recovery, in combination with optimal heart-failure medical management and appropriate antibiotic therapy. Further studies are needed to predict LV recovery in this clinical situation.

References