Transcatheter aortic valve replacement prosthetic valve *Listeria monocytogenes* infective endocarditis. A case report

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ABSTRACT

Background: *Listeria monocytogenes* is known to cause gastrointestinal tract, meningeal, and occasionally blood stream infections in immunocompromised patients. It is often overlooked as a cause of infective endocarditis, more so in patients with prosthetic valves.

Case Presentation: We present an 85-year-old Caucasian man who was admitted with unexplained symptoms of predominantly left-sided heart failure. He was found to have *L. monocytogenes* bacteremia on routine admission blood cultures. A follow up trans-esophageal echocardiography revealed aortic valve vegetations. He markedly improved with complete symptom resolution after 6 weeks of intravenous ampicillin and gentamicin without surgical intervention.

Conclusion: Clinicians should maintain a high index of suspicion for infective endocarditis in elderly patients with prosthetic valves presenting with *L. monocytogenes* bacteremia, as prompt echocardiographic evaluation and prolonged combination antibiotic therapy can prevent complications.

Keywords: *Listeria monocytogenes*, TAVR prosthetic valve, infective endocarditis, left-sided heart failure.

Type of Article: CASE REPORT **Specialty:** Internal Medicine

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Introduction

Listeria monocytogenes is often an overlooked cause of infective endocarditis, especially in patients with prosthetic valves. Generally, clinicians have lower tendencies to perform surveillance echocardiographies in cases of *Listeria* bacteremia, as compared to cases involving gram-positive cocci bacteremia [1]. The consequence is usually under treatment and eventual rebound bacteremia with progression of valvular destruction [2]. The case we describe is an 85-year-old man who was diagnosed with *Listeria monocytogenes* transcatheter aortic valve replacement (TAVR) valve endocarditis after presenting with signs of left-sided heart failure. His clinical course is summarized in Table 1.

Case Presentation

The patient we present was an 85-year-old Caucasian man whose past medical history was significant for paroxysmal atrial fibrillation, non-obstructive coronary artery disease, severe aortic stenosis (status post TAVR 5 years prior), compensated heart failure with preserved ejection fraction (left ventricular ejection fraction = 55%–60% a year prior), symptomatic bradycardia (status post permanent pacemaker placement 11 years prior), essential hypertension and hyperlipidemia.

His home medication included: apixaban, atorvastatin, diltiazem, ferrous sulfate, losartan, metoprolol succinate, omeprazole, spironolactone, and torsemide.

For this admission, he presented to the emergency department via emergency medical services (EMS) which he called himself because he felt unwell. He reported that on stopping to take his daily pills while on a trip from another state, he suddenly felt lightheaded and believed he lost consciousness. He was unsure if the event happened when he tried to get up from his car seat or change position. He was also unsure how long he lost consciousness for but acknowledged that on regaining consciousness, he did not have memory loss, had no tongue bites, and had no stool or urine incontinence. He reported no preceding chest pain, shortness of breath, diaphoresis, fever, chills, flu-like symptoms, vomiting, diarrhea, headaches, or focal neurological symptoms. After the event, he reported no focal body weakness or numbness. He reported being independent in terms of performing activities of daily

living but acknowledged lately being easily fatigued with occasional orthopnea but no cough, limb swelling, abdominal swelling, or new onset wheeze. As per EMS, at the point of contact, his oxygen saturation by pulse oximetry (PSO2)- was 70%–75% prompting initiation of supplemental oxygen by nasal cannula.

Physical examination findings

On arrival to the emergency room, he was hypoxic with PSO2 of 89% on 3 l of oxygen with a respiratory rate of 22 breaths per minute, blood pressure of 124/76mmHG, and a pulse rate of 98 beats per minute. He was sick looking and in mild distress but able to speak in full sentences, preferred to have the head of the bed raised, and was afebrile. He was fully oriented in person, place, time, and situation. He did not have pedal edema, had no digital clubbing, no skin rash, no nail changes, joint swelling, or visible bruising. He had a regular heart rate with a normal volume pulse and no jugular venous distention. The point of maximal impulse was in the fifth intercostal space, mid-clavicular line with a grade II pansystolic apical murmur and a grade II right upper parasternal area systolic murmur radiating to the right carotid artery. Breath sounds were diminished in the right mid to basilar region with

Table 1. *summary of the patient's clinical course.*

Timeline	Procedures	Events
Admission		-Admitted with hypoxic respiratory failure following an episode of unwitnessed syncope
Day 2-post admission	Transthoracic echocardiography: Aortic valve leaflets were mildly thickened/calcified with mild valvular aortic stenosis. Follow up trans-esophageal echocardiography: TAVR valve nodular echo-density at the right coronary cusp -non-coronary cusp junction	-Admitted to the intensive care unit for intubation and mechanical ventilation due to worsening hypoxic respiratory failure. - Started on intravenous ampicillin and gentamicin
Post discharge		Complete symptom resolution after 6 weeks of IV antibiotics without surgical intervention

Table 2. *Laboratory test results taken at admission.*

Test	Result	Normal ranges
White blood cell count	11,200 cells/ul (neutrophils- 934 cells/ul, lymphocytes- 470 cells/ul, Eosinophils-10 cells/ul)	4.5-11 × 10^3 cells/ul
Hemoglobin	13 g/dl	13.5-18 g/dl
Platelet count	244,000 cells/ul	150,000-450,000 cells/ul
Serum sodium	135 mmol/l	135-145 mmol/l
Serum potassium	4.5 mmol/l	3.5-5.3 mmol/l
serum bicarbonate	29 mmol/l	21-32 mmol/l
Anion gap	8	8-12
Blood urea nitrogen	55 mg/dl	7-23 mg/dl
Serum creatine	2 mg/dl (1 mg 4 months prior)	0.5-1.2 mg/dl
BUN/Creatine ratio	28	
Random blood glucose	138 mg/dl	70-140 mg/dl
Serum calcium	8.4 mg/dl	8.4-10.4 mg/dl
Aspartate transaminase (AST)	30 U/l	<59 U/l
Alanine transferase (ALT)	22 U/l	<35 U/l
Alkaline phosphatase (ALP)	165 U/l	38-126 U/l
Serum albumin	4.1 g/dl	3.5-5 g/dl
D-dimer	910 ng/ml	
serum Lactate	0.9 mmol/l	
serum procalcitonin-	0.29 ng/ml	<0.25 ng/ml
N-Terminal Pro-brain natriuretic peptide (pro-BNP)-	10,100 pg/ml	
Troponin I	Index: 1.08 ng/ml (<0.034 ng/ml) second troponin: 1.09 ng/ml Third troponin: 1.07 ng/ml All assays done within 24 hours	
2 sets of blood cultures and urine cultures taken	Pending at admission	

fine crepitations over the left basilar area. The abdominal exam was unremarkable. He had no focal neurological deficits. Orthostatic vital signs were negative for orthostatic hypotension.

Laboratory evaluation

His initial laboratory tests are summarized in Table 2.

Imaging studies

A chest X-ray and chest computed tomography (CT) scan done at admission are shown in Figure 1. Bedside point of care ultrasound showed no right ventricular strain with follow up compression venous ultrasound scans resulting negative for deep venous thromboses.

The admission differential diagnosis was: 1 – Cardiogenic syncope possibly due to orthostatic hypotension, 2 – Hypoxic respiratory failure secondary to decompensated heart failure, and 3 – acute kidney injury likely due to type 1 cardiorenal syndrome. He was started on intravenous furosemide, the dose of diltiazem and metoprolol succinate was reduced, and he was continued on oxygen supplementation by nasal cannula with initial improvement.

Hospital course and follow-up investigations

On the second day of admission, a transthoracic echocardiography (TTE) done showed-mildly increased left ventricular wall thickness with normal systolic function (ejection Fraction of 55%-60%). There was a pacemaker lead in the right ventricle. The aortic valve leaflets were mildly thickened/calcified with mild valvular aortic stenosis. A bedside thoracentesis was done, and a transudative pleural effusion was drained from the right pleural cavity. Due to progressive hypoxic respiratory failure and mental status changes, he was intubated and admitted to the intensive care unit (ICU). On the same day, blood cultures resulted in positive for *L. monocytogenes* in both culture bottles. The subsequent gram-positive blood culture panel by Nucleic acid amplification as well resulted in positive for *L. monocytogenes* DNA. Due to mental state changes, a repeat brain CT was performed, which was normal, and a lumbar puncture was performed to rule out *L. monocytogenes* meningitis. Both opening pressure and cerebral-spinal fluid parameters were within normal limits.

Because an earlier TTE showed thickening of the aortic valves but no definitive vegetations, a follow up trans-esophageal echocardiogram (TEE) was done while in the ICU, which showed the presence of a TAVR aortic valve prosthesis with a nodular echo-density at the right coronary cusp non-coronary cusp junction. No clear vegetation was noted on remaining native valves or on pacemaker leads (Figure 2).

At that point, a diagnosis of decompensated congestive heart failure with preserved ejection fraction due to *Listeria monocytogenes* TAVR aortic valve prosthesis infective endocarditis was made. His renal function had returned to baseline (creatine 1.0 mg/dl). He was started on a 6-week course of Intravenous (IV) gentamicin and ampicillin. Repeat blood cultures after 2 days of antibiotics were negative. The cardiothoracic surgery team recommended no surgical intervention at the time. He was eventually extubated, discharged after 4 weeks in the hospital.

Post discharge

He continued to improve and completed the 6-week course of intravenous antibiotics. However, 5 months later, he was admitted to another tertiary hospital with the recurrence of heart failure symptoms. Re-evaluation for recurrent endocarditis was negative, but he was found to have a new onset of TAVR aortic prosthetic valve failure with stenosis and severe regurgitation. Subsequently, he underwent valve-in-valve transfemoral TAVR.

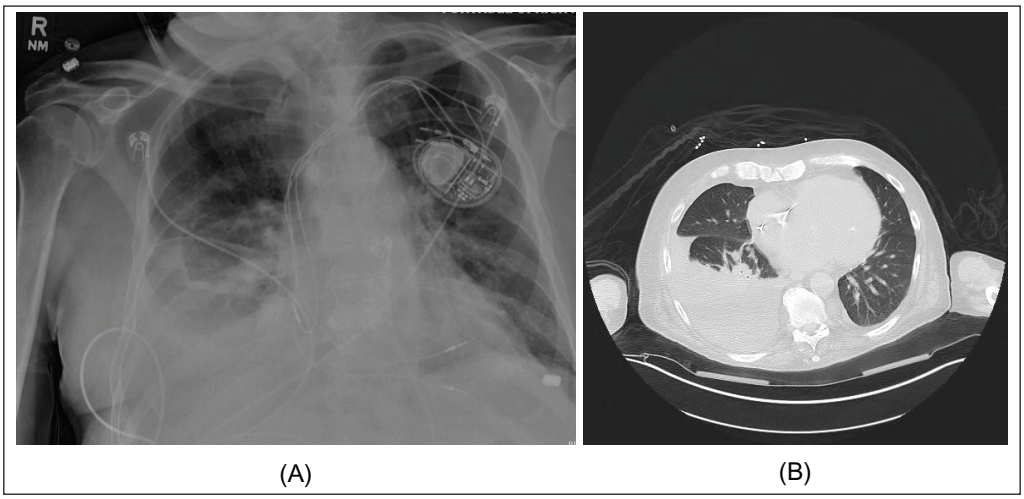

Figure 1. *Chest imaging performed at admission.*

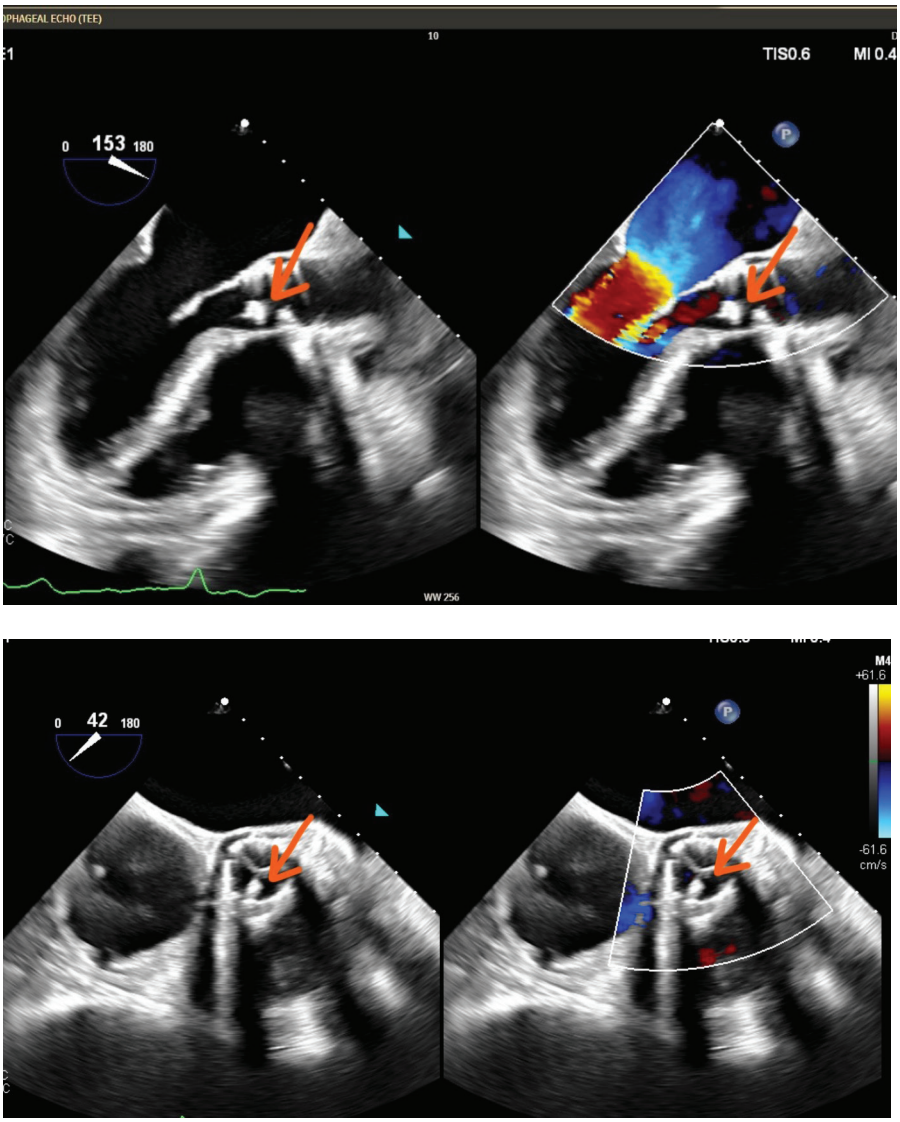

Figure 2. *Trans-esophageal echocardiography showing TAVR aortic prosthetic valve vegetations. The orange arrow shows a nodular echodensity on the TAVR aortic valve.*

Discussion

We present a case of IE involving a TAVR prosthetic valve in an elderly man with unexplained progressive symptoms of left-sided heart failure. Commonly, clinicians will have a high index of suspicion for endocarditis with gram-positive cocci bacteremia, prompting surveillance TTE with follow up TEE in high-risk cases. In usual practice, this is not the case for *L. monocytogenes,* which typically is known to cause gastrointestinal tract disease, meningeal disease, and rarely, blood stream infections in susceptible patients such as immunosuppressed patients, individuals at the extremes of age (neonates and older adults), and pregnant patients [3].

The main identifiable risk factor for *Listeria* infection was the patient's advanced age of 85 years. The index TTE showed aortic valve thickening but no definite vegetation or new onset stenosis/ regurgitation. He also did not have most minor criteria for IE. The decision to perform a follow up trans-esophageal echocardiography was based on the unexplained persistent hypoxemic respiratory failure from pulmonary edema (left-sided heart failure), as well as a high degree of suspicion otherwise; the inclination was to treat for listeria bacteremia with 2 weeks of antibiotics. In a published meta-analysis of 64 cases of *Listeria* IE, the majority (54%) of the patients had prosthetic valves [2]. Accurately making a diagnosis with a timely echocardiography has clinical implications, as this would mean a longer course of intravenous antibiotics.

Optimum treatment involves the use of ampicillin and gentamicin for synergy, for 6 weeks. The main challenge to this regimen is the nephrotoxicity as well as ototoxicity of gentamicin [4]. This requires close monitoring of renal function and, in some cases, audiometric evaluations. Alternative regimens, including IV sulfamethoxazole and trimethoprim (Bactrim), meropenem, and vancomycin, have proved not as effective with higher recurrence rates. Our patient had just recovered from acute kidney injury when he started gentamicin, but he was able to tolerate it for up to 6 weeks [5].

Conclusion

Clinicians should maintain a high index of suspicion for infective endocarditis in elderly patients with prosthetic valves presenting with *L. monocytogenes* bacteremia, as prompt echocardiographic evaluation and prolonged combination antibiotic therapy can prevent complications.

What is new?

Listeria monocytogenes is a documented uncommon cause of infective endocarditis, more so in patients with prosthetic valves.

Acknowledgments

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List of Abbreviations

ALP	Alkaline phosphatase
ALT	Alanine transferase
AST	Aspartate transaminase
CoNs	Coagulase Negative Staphylococcus aureus
CSF	Cerebrospinal fluid
CT	Computed Tomography
DNA	Deoxyribonucleic Acid
EMS	Emergency Medical Services
ICU	Intensive Care Unit
IE	Infective Endocarditis
IV	Intravenous-
TAVR	Transcatheter Aortic Valve Replacement
TEE	Trans-esophageal echocardiography
TTE	Trans-thoracic echocardiography

Conflict of interest

The authors declare that they have no competing interests.

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Consent for publication

Written informed consent for publication of this case report and identifying images was obtained from the participant.

Ethics approval and consent to participate

Ethical approval was provided by the United Health Services institutional review board, IRB number: 15012, August/19th/2025.

Authors' contributions

JZ participated in the care of the patient. FM, HW, SM, CP, MR, and JZ reviewed the patient's records as well as participated in manuscript writing.

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Summary of the case

1	Patient (gender, age)	Male, 85 years
2	Final diagnosis	Transcatheter aortic valve replacement prosthetic valve *Listeria monocytogenes* infective endocarditis
3	Symptoms	Exertional intolerance
4	Medications	Ampicillin, Gentamicin
5	Clinical procedure	Trans-esophageal echocardiography
6	Specialty	Internal medicine, Infectious diseases