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**Leclercia adecarboxylata infective endocarditis in a man with mitral stenosis : A case report and review of the literature**

A case report and review

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## Abstract

### BACKGROUND

Infective endocarditis (IE) is a rare disease with a high mortality rate. *Leclercia adecarboxylata* (*L. adecarboxylata*) is a dynamic gram-negative bacillus of enterobacteriaceae, a rare pathogen, which is often affecting immunodeficient patients. There are about three cases of immunocompetent patients with monomicrobial *L. adecarboxylata* infection. There are only three reported cases of IE caused by *L. adecarboxylata* in the world. The mitral valve is often affected in IE, and the prognosis for IE with mitral valve lesions is often poor.

### CASE SUMMARY

A 51-year-old man was found to have moderate to severe mitral stenosis on echocardiography. He came to our Cardiothoracic Surgery Department for surgical management. A diastolic murmur was heard on auscultation of the heart in the mitral region. On the second day of hospitalisation, he presented with slurred speech, reduced muscle strength in the left limb and acute cerebral infarction on cranial computed tomography scan (CT). Surgical treatment was decided to postpone. On ninth day of admission, the patient developed a sudden high fever and shock and was transferred to the Cardiac Intensive Care Unit (CICU), where echocardiogram revealed an anterior mitral valve leaflet vegetation. After empirical anti-infective treatment with vancomycin (1g q12h), an emergency valve replacement was performed. Bacterial culture identified *L. adecarboxylata* infection. Anti-infective treatment with piperacillin-tazobactam (4.5g q8h) was added for 4 wk. Follow-up echocardiography showed normal bioprosthetic valve function after mitral valve replacement.

### CONCLUSION

We report the first case of *L. adecarboxylata* IE in China, and clinicians should pay attention to this pathogen.

**Key Words:** Key words: *Leclercia adecarboxylata*; Infective endocarditis; Mitral valve; Vegetation; Cerebral infarction; Case report

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**Core Tip:** *Leclercia adecarboxylata* (*L. adecarboxylata*) is a ubiquitous microorganism often found in water and soil and a rare pathogen can be isolated from specimens such as blood. We present a rare case of IE caused by *L. adecarboxylata* that occurred in an immunocompetent male patient. He presented with clinical symptoms after diarrhoea. *L. adecarboxylata* is the only pathogen and intestine may be the portal of entry for the infection. Immunosuppression is a significant risk factor for *L. adecarboxylata* infection. *L. adecarboxylata* is usually the only pathogen cultured in immunosuppressed individuals. Bacteria crossing the intestinal mucosal barrier may cause bacteremia.

## **INTRODUCTION**

*Leclercia adecarboxylata* (*L. adecarboxylata*) is a movable gram-negative rod-shaped bacterium in the Enterobacteriaceae family, most commonly found in water and soil<sup>[1, 2]</sup> and rarely isolated from clinical specimens, with most of the Enterobacteriaceae characteristics, such as partly anaerobic, oxidase-negative, mesophilic, peripheral flagellate bacilli<sup>[3]</sup>. Infective endocarditis (IE) caused by *L. adecarboxylata* is a very rare disease in clinic. Only three cases of *L. adecarboxylata* endocarditis have been reported in the existing literature<sup>[4-6]</sup>. This time, we report a case of *L. adecarboxylata* endocarditis complicated with severe mitral stenosis, atrial fibrillation and acute cerebral infarction. *L. adecarboxylata* strains were sensitive to antibiotics other than amoxicillin, cephalosporin and compound sulfamethoxazole. After surgery and 4-week antibiotic therapy, the patient recovered without sequelae.

## CASE PRESENTATION

### *Chief complaints*

A 51-year-old male patient presented to the Cardiovascular Surgery Department with a complaint of moderate to severe mitral stenosis combined with mild to moderate regurgitation on echocardiography for 1 mo.

### *History of present illness*

The patient presented to the local hospital 1 mo ago with chest tightness and was diagnosed with "atrial fibrillation". Echocardiography showed combined valvular disease including moderate to severe mitral stenosis combined with mild to moderate regurgitation, mild aortic valve insufficiency, mild tricuspid valve insufficiency, decreased left ventricular function and a small amount of pericardial effusion. The patient was admitted to our hospital for mitral valve replacement.

### *History of past illness*

The patient had a history of atrial fibrillation. Prior to admission to our hospital, the patient had a short bout of diarrhoea lasting approximately 3 days, which improved with the use of "anti-diarrhoea medication" (not specified).

### *Personal and family history*

None.

### *Physical examination*

The patient had temperature of 36.5 °C, heart rate of 91 beats per minute (bpm), respiratory rate of 15 cycles per minute (cpm) and blood pressure of 131/79 mmHg on admission. Cardiac auscultation revealed a severe diastolic murmur in the mitral region and a systolic murmur in the tricuspid valve. Cardiac percussion showed enlargement of the cardiac border to both sides.

### *Laboratory examinations*

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Blood analysis, blood biochemistries, prothrombin and partial thromboplastin times, d-dimers, as well as arterial blood gas were normal.

### *Imaging examinations*

The echocardiogram of our hospital showed severe mitral stenosis with mild mitral regurgitation, mild and moderate tricuspid regurgitation, mild aortic regurgitation, and mild pulmonary hypertension. A chest computed tomography scan (CT) showed an enlarged cardiac shadow and a small amount of pericardial effusion. The electrocardiogram showed atrial fibrillation.

### *Disease changes*

On the second day of admission, the patient was found to have slurred speech, a shallow left nasolabial fold and grade 4 muscle strength in the left limb.

### *Imaging examinations*

Cranial CT showed acute cerebral infarction in the right frontotemporal occipital lobe with a small amount of post-infarct blood leakage. Considering the patient's sudden acute cerebral infarction complicated by cerebral haemorrhage, it was discussed and decided to postpone the surgical treatment.

### *Recurrence of disease changes*

On the 9th day of hospitalization, the patient developed a sudden high fever with a temperature of 38.6 °C. The examination showed heart rate of 170 bpm, blood pressure of 90/60 mmHg and respiratory rate of 25 cpm. The patient was transferred to the Cardiac Intensive Care Unit (CICU) for further treatment.

### *Further diagnostic work-up*

After admission to the CICU, blood count analysis showed a white blood cell count of 7010/mm<sup>3</sup> (85.6% neutrophils) and blood gas analysis showed a lactate level of 2.8 mmol/L and a calcitonin level of 0.98 Pg/L. Bedside echocardiogram showed a vegetation attaching to the anterior leaflet of mitral valve (Figure 1A, Figure 1B).

### ***Preliminary diagnosis***

The initial diagnosis in this case was severe mitral stenosis with mild regurgitation, moderate tricuspid regurgitation, mild aortic regurgitation, mild pulmonary hypertension, pericardial effusion, atrial fibrillation, acute cerebral infarction, acute cerebral haemorrhage, shock, and acute IE.

### ***Identification of pathogenic microorganisms***

Appropriate microbiological analysis was used to determine the etiology and susceptibility to antibiotics in patients with acute IE. Gram stain, antacid bacillus stain and culture for aerobic and anaerobic bacteria were performed on blood and intraoperatively resected valve vegetation. <sup>3</sup> Samples were first enriched in thioglycolic acid broth, incubated overnight and then inoculated on blood agar plates, McConkey agar plates, Sabouraud agar plates, anaerobic growth media and Löwenstein-Jensen medium. Blood cultures and superfluous cultures were suggestive of positive Gram-negative bacilli and identified as *L. adecarboxylata* infection. The organism was sensitive to a variety of antibiotics, including ampicillin, amikacin, aztreonam, ciprofloxacin, cefazolin, cefepime, cefotaxime, gentamicin, imipenem, meropenem, and piperacillin.

### **FINAL DIAGNOSIS**

The final diagnosis in this case was severe mitral stenosis with mild regurgitation, moderate tricuspid regurgitation, mild aortic regurgitation, mild pulmonary hypertension, pericardial effusion, atrial fibrillation, acute cerebral infarction, acute cerebral haemorrhage, septic shock, and acute IE due to *L. adecarboxylata*. The patient's IE diagnosis was based on the two main criteria (echocardiography finding vegetations

in the anterior lobe of the mitral valve, and positive cultures of blood and vegetation) and three minor criteria (history of mitral stenosis, fever of 38.6°C and embolic infarction) of modified duke criteria.

### **TREATMENT**

Immediately after admission to the CICU, fluid replacement, volume expansion and norepinephrine (18 µg/min) was given to maintain blood pressure and correct shock, and vancomycin (1g q12h) was administered empirically after blood cultures were retained for anti-infective treatment. Considering that the patient had acute IE with shock, which was an indication for emergency surgery, emergency surgery was performed. The patient underwent cardiac lesion resection, mitral bioprosthetic valve replacement, tricuspid valvuloplasty, radiofrequency ablation of atrial fibrillation and left atrial auricular resection under general anaesthesia. Postoperatively, levosimendan (1 mg/h) was given to enhance cardiac function, norepinephrine (24µg/min) to maintain blood pressure and circulatory stability, vancomycin (1g q12h) for experienced anti-infection and warfarin (2.5mg qd) for anticoagulation. After 24 h of these treatments, the patient's circulation and respiration were relatively stable, but he was still febrile. Levosimendan and norepinephrine were discontinued. 2 tubes of blood were obtained for pre-operative blood cultures, suggesting *L. adedecarboxylata* infection, and anti-infective therapy with piperacillin tazobactam (4.5g q8h) in combination with vancomycin (1g q12h) was administered on the second day after surgery. The patient's fever resolved and the neutrophil percentage decreased to normal within 48 h of the combined anti-infective treatment. Blood cultures were performed on the first, second, third, fifth and seventh postoperative days respectively (2 tubes of blood per day). Blood cultures were positive on the first three days after surgery, and negative on the fifth and seventh postoperative days. On the 17th day of hospitalization, the patient returned to the general ward of the cardiovascular surgery department and continued anti-infective treatment with vancomycin (1g q12h) and piperacillin tazobactam (4.5g q8h) for a total of 4 wk of the dosing cycle.



## OUTCOME AND FOLLOW-UP

The patient's vital signs were stable and blood cultures were negative after surgery and anti-infective therapy. At follow-up visit, <sup>3</sup> one month after surgery, the patient was asymptomatic, and a new echocardiography showed that the patient had normal biologic valve function after mitral valve replacement, and no abnormal echogenicity was detected around the valve.

## DISCUSSION

<sup>1</sup> The first case of aortic valve disease combined with endocarditis was reported by Lazare Rivière<sup>[7]</sup> in 1646, and in 1806 Jean-Nicolas Corvisart<sup>[8]</sup> introduced the term 'vegetation', Jean Baptiste Bouillaud<sup>[9]</sup> called the inner layer of the heart the "endocardium" and the term "endocarditis" came into use in 1835<sup>[10]</sup>. IE is a rare disease with a high mortality rate, ranging from 1.5 to 11.6 per 100,000 people worldwide each year. However, despite significant advances in antibiotic therapy and surgery, the in-hospital mortality rate for IE still exceeds 20%, with 1-year mortality rates still as high as 30%<sup>[11]</sup> and 5-year mortality rates as high as 45%<sup>[12]</sup>. In the real clinic, these figures may be underestimated<sup>[13]</sup>.

Although the incidence varies little numerically between countries, there are significant differences in patterns. <sup>1</sup> Historically, rheumatic heart disease and dental surgery were considered to be the major contributing factors to IE. In recent years, the incidence of rheumatic heart disease has declined in high-income countries, and the incidence of heart disease and cardiac surgery has increased as the average human age increases<sup>[14]</sup>, with invasive procedures, prosthetic valves, and cardiac implant devices emerging as risk factors for IE. Long-term intravenous drug use (IDU-IE) is also an increasingly common cause of IE. Degenerative valve disease and congenital heart disease are also risk factors for IE<sup>[15, 16]</sup>. Currently, the most common susceptibility disease in IE is valvular heart disease<sup>[17]</sup>, and with an ageing population there is an increasing incidence of degenerative valve disease such as mitral and aortic regurgitation<sup>[18]</sup>. Of

these, mitral valve prolapse is the predominant structural abnormality in native valve endocarditis (NVE). Nosocomial infections are also an important factor in disease, with hospital-acquired staphylococci being the main source of infection in high-income countries, mainly affecting the elderly<sup>[11]</sup>.

As a typical lesion of IE, the vegetation is composed of a large number of platelets, fibrin, microorganisms and a small number of inflammatory cells. The pathogenesis of IE usually begins with damage to the endothelium<sup>[19]</sup>. Endothelial damage triggers thrombosis caused by fibrin and platelet deposits<sup>[20]</sup>. When bacteraemia occurs, bacteria can reach and colonise these damaged areas. <sup>6</sup> After colonisation, the surface is rapidly covered with another layer of platelets and fibrin, suitable for further colonisation, leading to progressive bacterial infection. In general, the endothelial layer and valves of the heart are resistant to infection. However, very deadly microorganisms such as *Staphylococcus aureus* are able to infect normal heart valves<sup>[21]</sup>.

IE includes both natural valve endocarditis and IE caused by intracardiac prosthetic materials. The latter includes prosthetic valve endocarditis<sup>[22]</sup> and IE associated with pacemakers, implantable cardiac defibrillators (ICD) and ventricular assist devices.

Early and accurate diagnosis of IE is essential, as delayed treatment can affect the prognosis of the disease<sup>[23, 24]</sup>. The diagnosis of IE is widely made using microbiological techniques and imaging techniques (echocardiography, <sup>1</sup> CT and nuclear imaging). <sup>9</sup> Histological findings from postoperative excised tissue can also be used for diagnosis. The clinical diagnosis of IE is based on the modified Duke criteria, which are included in the guidelines for IE<sup>[22]</sup>. The Modified Duke Criteria of 2000 classify the diagnosis <sup>1</sup> into 'major' and 'minor' criteria and classify cases as "definite", "possible" or "rejected"<sup>[25]</sup>. A positive blood culture is one of the main diagnostic criteria. When atypical infection is clinically suspected, routine serological screening should be performed. If all serology is negative, the sample should be checked for antinuclear and antiphospholipid antibodies. The cornerstone of the modified Duke criteria is the echocardiography.

In any case of suspected IE, echocardiography is the imaging technique of choice<sup>[26]</sup>. Echocardiography is the most important tool for the detection of endocardial lesions as it can reveal vegetations, abscesses, perforations, fistulas, valvular aneurysms and pseudoaneurysms.

8 The clinical features of IE are highly variable and depend largely on the infecting organism and the medical history of heart disease or not<sup>[27, 28]</sup>. The clinical presentation may be acute, subacute or chronic<sup>[29]</sup>. Approximately 90% of infected patients present with fever, and IE is also an important cause of unexplained fever<sup>[30-32]</sup>. It is accompanied by various systemic symptoms such as appetite loss and weight loss<sup>[33]</sup>. About 85% of patients have heart murmurs<sup>[34]</sup>. Clinical features also include neurological signs and symptoms<sup>[35]</sup>. IE is a risk factor for stroke<sup>[36]</sup>. Intravenous drug use, HIV, chronic liver disease, elevated CRP, microorganisms such as *Staphylococcus aureus*, the presence of vegetations on echocardiography, multiple movable mitral valve vegetations, vegetation > 10 mm in size, and prosthetic materials are risk factors for a clinically significant increased risk of embolism<sup>[37-40]</sup>.

6 Antibiotics are the basis of treatment for IE and appropriate antibiotic therapy is important to control local and systemic infection, eradicate microorganisms from the vegetation and reduce the risk of complications such as septic embolism<sup>[41]</sup>. There is no standardisation in the use of antibiotics<sup>[42]</sup>. Most infections are caused by streptococci, staphylococci or enterococci, and the microbial profile of pathogenic microorganisms has changed over time. *Staphylococcus* species now account for an increasing proportion of IE cases<sup>[43]</sup>. The most common of these pathogens is *Staphylococcus aureus*<sup>[43]</sup>. Successful treatment of IE relies on the eradication of microorganisms by antimicrobial drugs. Many antimicrobial drugs are used alone or in combination to treat IE, including beta-lactams ( $\beta$ -lactams), aminoglycosides, glycopeptides, oxazolidinones, complex macrocyclic compounds and quinolones<sup>[44]</sup>. 1 Antibiotics are administered immediately after blood cultures are performed. The choice of antibiotic should be specific to the organism isolated from the blood culture and will depend primarily on

the antibiotic sensitivity of the pathogen and whether the valve is natural or prosthetic<sup>[29]</sup>.

Medical treatment of IE has a mortality rate of 60% ~ 90% and a high proportion of patients with active IE are at high risk of death without cardiovascular surgery<sup>[45]</sup>. Effective surgical treatment can reduce mortality by 8% ~ 16%<sup>[46, 47]</sup>. Heart failure, persistent or uncontrolled infection, IE caused by fungal or highly drug-resistant strains, heart block, and circumferential or aortic root abscesses caused by IE are indications for Class I surgery<sup>[26]</sup>. Of these, heart failure is the most common indication for surgery in patients with IE, accounting for 60% of patients undergoing surgery, followed by uncontrolled infection (48%) and embolic episodes (18%)<sup>[48]</sup>. Surgery is usually recommended for this group of patients early in the disease, during hospitalisation and before the end of antibiotic therapy<sup>[41]</sup>. Patients with refractory pulmonary oedema or cardiogenic shock due to IE should undergo emergency surgery within 24 h<sup>[26]</sup>.

In IE, mitral valve is often affected<sup>[49]</sup>. The prognosis for IE with mitral valve lesions is often poor, and extensive excision of the infected focus and mitral valve replacement (MVR) are considered to be the better surgical approach. However, the graft can easily become another source of infection, and mitral valve repair (MVP) can overcome this disadvantage. A meta-analysis has shown that the in-hospital mortality rate of MVP is lower than that of MVR in the surgical treatment of active native mitral IE. The efficacy and follow-up of MVP is comparable to that of MVR, with a lower mortality rate at 5 years after surgery, suggesting a lower risk of reoperation or recurrence of infection after long-term follow-up MVP<sup>[50]</sup>. A systematic review found that in cases of mitral valve surgery, valve repair resulted in lower in-hospital and long-term mortality, lower recurrence rates, fewer repeat surgeries after mitral valve surgery and fewer cerebrovascular events compared with valve replacement<sup>[51]</sup>.

Gram-positive bacteria account for more than 70% of IE cases, and are the main pathogen of community-acquired and hospital-acquired IE cases<sup>[52]</sup>. Gram negative bacteria usually do not cause IE<sup>[53]</sup>. Gram-negative IE accounts for less than 5% of IE<sup>[54]</sup>.

In the past 50 years, the incidence rate of gram-negative endocarditis has been increasing<sup>[55, 56]</sup>. In addition to the traditional risk factors of structural heart disease (congenital or acquired)<sup>[57, 58]</sup>, most gram-negative endocarditis is mainly caused by medical related events<sup>[59]</sup>. Patients with indwelling catheters, invasive lines, and intracardiac devices are at higher risk<sup>[60, 61]</sup>. Among patients with gram-negative endocarditis, 60% had renal insufficiency<sup>[17]</sup>, and about 52% had embolic symptoms<sup>[60]</sup>. Embolic symptoms can be divided into the presence or absence of central nervous system involvement, including neurological complications such as stroke or transient ischemic attack, and other complications such as metastatic infection, mesenteric ischemia and peripheral terminal artery occlusion<sup>[41, 62]</sup>. A large prospective study found that the incidence of new murmurs was 48% ~ 60%, and 30% ~ 40% of patients lost weight<sup>[17, 59]</sup>. Studies have reported that about 11% ~ 33% of patients have splenomegaly<sup>[59, 63]</sup>.

*L. adecarboxylata* was first described by H. Leclerc<sup>[64]</sup> in 1962 and was previously known as "Enteric group 410 or *Escherichia adecarboxylata*". The name *L. adecarboxylata* was officially given by Tamura *et al*<sup>[65, 66]</sup> in 1987. The true frequency of infections caused by *L. adecarboxylata* has been underestimated and underreported for decades. The underestimation of *L. adecarboxylata* infections is mainly due to the fact that several strains of the bacterium are often misidentified as *Escherichia coli*, as the two species share a number of morphological and metabolic characteristics<sup>[67]</sup>.

*L. adecarboxylata* is a ubiquitous microorganism, most commonly found in water and soil<sup>[1, 2]</sup>. Cases of *L. adecarboxylata* infection in patients with corneal abscesses combined with keratitis have been reported with a history of exposure to the aquatic environment<sup>[68]</sup>. *L. adecarboxylata* also exists in the intestinal symbiotic flora of some animals<sup>[69]</sup>, which is an active gram-negative rod-shaped bacterium in the Enterobacteriaceae family, with most of the Enterobacteriaceae characteristics, such as partly anaerobic, oxidase-negative, mesophilic, peripheral flagellate bacilli<sup>[3]</sup>.

*L. adecarboxylata* is a rare emerging pathogen and immunosuppression is the most significant risk factor for infection in *L. adecarboxylata*, often affecting immunodeficient



patients, including those with immunomodulators, haematological malignancies or receiving chemotherapy<sup>[70]</sup>, and is the only isolate cultured from blood, sputum, urine and peritoneal fluid in immunosuppressed individuals<sup>[71-73]</sup>. There are currently about three cases of immunocompetent patients with monomicrobial *L. adecarboxylata* infection<sup>[74-76]</sup>. The remaining cases are all immunosuppressed patients<sup>[70]</sup>. *L. adecarboxylata* is usually isolated as part of a polymicrobial infection in immunocompetent patients<sup>[66, 67, 70]</sup>. Because of its low virulence, *L. adecarboxylata* is often overlooked in the context of polymicrobial infections and is asymptomatic carriage or colonisation. Among the polymicrobial infections, the most common co-infecting pathogen of *L. adecarboxylata* is Enterococcus. The others are Acinetobacter, Fusarium, Staphylococcus epidermidis, Klebsiella and Pseudomonas aeruginosa<sup>[77, 78]</sup>. *L. adecarboxylata* has been isolated mainly from food, water and environmental sources or from animal specimens. Knowledge of the route of transmission of *L. adecarboxylata* is limited, and the possible sources of infection are unknown. In the available case reports, *L. adecarboxylata* can be isolated from specimens including blood, wound pus, faeces, urine, gallbladder, peri-ciliary and ciliary abscesses, synovial fluid, peritoneal fluid from peritoneal dialysis, sputum, cerebrospinal fluid, catheters<sup>[79, 80]</sup>. A few cases with diabetes as the only risk factor have *L. adecarboxylata* infection of the skin and soft tissues<sup>[81, 82]</sup>. *L. adecarboxylata* has been implicated in endocarditis<sup>[6]</sup>, catheter-associated bacteremia<sup>[78]</sup>, bacteremia, sepsis<sup>[3, 67]</sup>, septic arthritis, meningitis<sup>[83]</sup>, cellulitis<sup>[2, 84]</sup>, urinary tract infections<sup>[85]</sup>, pneumonia<sup>[70]</sup> and bacterial peritonitis, especially in patients on peritoneal dialysis<sup>[86]</sup>. Of these, the most common clinically are catheter-associated male urinary tract infections (translocation through the genitourinary tract), followed by ventilator-associated pneumonia, peritoneal dialysis peritonitis, corneal abscesses, vascular graft infections (entry into the infected host *via* catheter or wound)<sup>[87]</sup> and intestinal translocation (translocation of bacteria through the mucosal barrier of the gastrointestinal tract, presumably gastrointestinal bacteremia)<sup>[2, 70]</sup>. Bacteremia may be due to bacterial translocation across the intestinal mucosal barrier as a result of colonic megacolon disease, antibiotic use, and altered dietary habits. To

assess the relationship between gastrointestinal and bacteraemia and *L. adecarboxylata*, four cases of *L. adecarboxylata* associated with gastrointestinal pathology were reviewed. Three patients of these reported cases presented with only intestinal mucosal disturbances with *L. adecarboxylata* as a possible source of infection<sup>[74]</sup>. These patients did not undergo any invasive intervention prior to isolation of the bacteria from their blood. In the fourth reported case, the bacterial translocation could have been the result of mucosal alterations due to invasive interventions to the gastrointestinal tract<sup>[88]</sup>. It can therefore be hypothesised that the gastrointestinal pathology causing the alteration of the mucosal barrier leads to a higher risk of this *L. adecarboxylata* bacteraemia<sup>[76]</sup>. Our case presented with clinical signs associated with IE following diarrhoea, and we hypothesize that the intestine may have been the entry point for this *L. adecarboxylata* infection. Our patient had severe mitral stenosis and other underlying organic heart disease, with sudden acute cerebral infarction, high fever, high white blood cell count and shock during the course of the disease. Echocardiography showed formation of vegetations in the anterior mitral leaflet, and cultures of blood and vegetations showed *L. adecarboxylata* infection. Of the reported cases of endocarditis in the world, only three were caused by *L. adecarboxylata*<sup>[4-6]</sup>. To our knowledge, this is the first case of *L. adecarboxylata* endocarditis in China. In our case, the diagnosis was based on one main criterion for IE (echocardiographic finding of a vegetation) and three minor criteria (positive cultures of blood and vegetation, fever of 38.6°C and embolic infarction)<sup>[25]</sup>. After mitral valve replacement and 4-week piperacillin-tazobactam anti-infection treatment, the patient's cardiac function returned to normal. The optimal regimen and duration of antibiotic therapy for *L. adecarboxylata* endocarditis remains imperfect due to the low number of case reports. As an emerging pathogen, further research into the pathogenesis and risk factors of *L. adecarboxylata* is needed.

## CONCLUSION

There are few cases of *L. adecarboxylata* isolated from environmental and clinical specimens, and few cases of *L. adecarboxylata* infection have been reported in the

literature. In previous cases, it was most commonly isolated as part of a mixture of microorganisms that could be the only pathogen cultured in immunosuppressed patients. In our case, *L. adecarboxylata* was the only pathogen isolated from bacterial cultures in an immunocompetent patient, and there are only three existing case reports of monomicrobial *L. adecarboxylata* infection in immunocompetent patients<sup>[74]</sup>. Of the case reports of endocarditis found worldwide, only three cases of endocarditis were caused by *L. adecarboxylata*<sup>[4-6]</sup>. We report the first case of IE caused by *L. adecarboxylata* in China. In this case, symptoms of IE developed after diarrhoea, and we speculate that the intestine may be the portal of entry for *L. adecarboxylata* infection. Clinicians should be aware of the gastrointestinal translocation of this pathogen and the possibility of causing IE.



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PRIMARY SOURCES

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- 1

Amer Harky, Sevim Zaim, Apeksha Mallya, Joel Jacob George. "Optimizing outcomes in infective endocarditis: A comprehensive literature review", Journal of Cardiac Surgery, 2020

Crossref

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---
- 2

[www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov)

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64 words — 2%

---
- 3

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Internet

46 words — 1%

---
- 4

Aref A. Bin Abdulhak, Imad M. Tleyjeh. "Indications of Surgery in Infective Endocarditis", Current Infectious Disease Reports, 2017

Crossref

40 words — 1%

---
- 5

Bora Lee, Jung-Ju Sir, Sung-Won Park, Cheol-Hoon Kwak et al. "A Case of Leclercia adecarboxylata Endocarditis in a Woman With Endometrial Cancer", The American Journal of the Medical Sciences, 2009

Crossref

36 words — 1%

---
- 6

[onlinelibrary.wiley.com](http://onlinelibrary.wiley.com)

Internet

34 words — 1%

---
- 7

Xiao-Yan Yang, Yi Tang, Hong-Xia Yuan. "Electrocardiogram Findings Worthy of Vigilance

18 words — < 1%

8 Martí-Carvajal, Arturo J, Lucieni O Conterno, Ricardo Hidalgo, Joey SW Kwong, Petros Georgoulas, Georgia Salanti, and Arturo J Martí-Carvajal. "Antibiotic therapy for treatment of infective endocarditis", Cochrane Database of Systematic Reviews Protocols, 2012.

16 words — < 1%

Crossref

9 Gomes, Anna, Andor W J M Glaudemans, Daan J Touw, Joost P van Melle, Tineke P Willems, Alexander H Maass, Ehsan Natour, Niek H J Prakken, Ronald J H Borra, Peter Paul van Geel, Riemer H J A Slart, Sander van Assen, and Bhanu Sinha. "Diagnostic value of imaging in infective endocarditis: a systematic review", The Lancet Infectious Diseases, 2016.

15 words — < 1%

Crossref

10 Guo-Ying Feng, Yu Cheng, Xiu Xiong, Zheng-Rong Shi. "Conversion therapy of hepatic artery ligation combined with transcatheter arterial chemoembolization for treating liver cancer: A case report", World Journal of Clinical Cases, 2021

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