

# World Journal of *Clinical Cases*

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## Contents

Thrice Monthly Volume 10 Number 29 October 16, 2022

## STANDARD AND CONSENSUS

- 10391** Baishideng's Reference Citation Analysis database announces the first Article Influence Index of multidisciplinary scholars

Wang JL, Ma YJ, Ma L, Ma N, Guo DM, Ma LS

## REVIEW

- 10399** Cholecystectomy for asymptomatic gallstones: Markov decision tree analysis

Lee BJH, Yap QV, Low JK, Chan YH, Shelat VG

- 10413** Liver transplantation for hepatocellular carcinoma: Historical evolution of transplantation criteria

Ince V, Sahin TT, Akbulut S, Yilmaz S

## MINIREVIEWS

- 10428** Prostate only radiotherapy using external beam radiotherapy: A clinician's perspective

Lee JW, Chung MJ

## ORIGINAL ARTICLE

## Retrospective Study

- 10435** Age-adjusted NT-proBNP could help in the early identification and follow-up of children at risk for severe multisystem inflammatory syndrome associated with COVID-19 (MIS-C)

Rodriguez-Gonzalez M, Castellano-Martinez A

- 10451** Clinicopathological characteristics and prognosis of gastric signet ring cell carcinoma

Tian HK, Zhang Z, Ning ZK, Liu J, Liu ZT, Huang HY, Zong Z, Li H

- 10467** Development and validation of a prognostic nomogram for decompensated liver cirrhosis

Zhang W, Zhang Y, Liu Q, Nie Y, Zhu X

## Observational Study

- 10478** Effect of medical care linkage-continuous management mode in patients with posterior circulation cerebral infarction undergoing endovascular interventional therapy

Zhu FX, Ye Q

- 10487** Effect of the COVID-19 pandemic on patients with presumed diagnosis of acute appendicitis

Akbulut S, Tuncer A, Ogut Z, Sahin TT, Koc C, Guldogan E, Karabulut E, Tanriverdi ES, Ozer A

**EVIDENCE-BASED MEDICINE**

- 10501** Delineation of a SMARCA4-specific competing endogenous RNA network and its function in hepatocellular carcinoma

*Zhang L, Sun T, Wu XY, Fei FM, Gao ZZ*

**SYSTEMATIC REVIEWS**

- 10516** Comparison of laboratory parameters, clinical symptoms and clinical outcomes of COVID-19 and influenza in pediatric patients: A systematic review and meta-analysis

*Yu B, Chen HH, Hu XF, Mai RZ, He HY*

**CASE REPORT**

- 10529** Surgical treatment of bipolar segmental clavicle fracture: A case report

*Liang L, Chen XL, Chen Y, Zhang NN*

- 10535** Multiple disciplinary team management of rare primary splenic malignancy: Two case reports

*Luo H, Wang T, Xiao L, Wang C, Yi H*

- 10543** Klippel-Trenaunay-Weber syndrome with ischemic stroke: A case report

*Lee G, Choi T*

- 10550** Vedolizumab in the treatment of immune checkpoint inhibitor-induced colitis: Two case reports

*Zhang Z, Zheng CQ*

- 10559** Novel way of patent foramen ovale detection and percutaneous closure by intracardiac echocardiography: A case report

*Han KN, Yang SW, Zhou YJ*

- 10565** Treatment failure in a patient infected with *Listeria* sepsis combined with latent meningitis: A case report

*Wu GX, Zhou JY, Hong WJ, Huang J, Yan SQ*

- 10575** Three-in-one incidence of hepatocellular carcinoma, cholangiocellular carcinoma, and neuroendocrine carcinoma: A case report

*Wu Y, Xie CB, He YH, Ke D, Huang Q, Zhao KF, Shi RS*

- 10583** Intestinal microbiome changes in an infant with right atrial isomerism and recurrent necrotizing enterocolitis: A case report and review of literature

*Kaplina A, Zaikova E, Ivanov A, Volkova Y, Alkhova T, Nikiforov V, Latypov A, Khavkina M, Fedoseeva T, Pervunina T, Skorobogatova Y, Volkova S, Ulyantsev V, Kalinina O, Sitkin S, Petrova N*

- 10600** *Serratia fonticola* and its role as a single pathogen causing emphysematous pyelonephritis in a non-diabetic patient: A case report

*Villasuso-Alcocer V, Flores-Tapia JP, Perez-Garfias F, Rochel-Perez A, Mendez-Dominguez N*

- 10606** Cardiac myxoma shedding leads to lower extremity arterial embolism: A case report

*Meng XH, Xie LS, Xie XP, Liu YC, Huang CP, Wang LJ, Zhang GH, Xu D, Cai XC, Fang X*

- 10614** Extracorporeal membrane oxygenation in curing a young man after modified Fontan operation: A case report  
*Guo HB, Tan JB, Cui YC, Xiong HF, Li CS, Liu YF, Sun Y, Pu L, Xiang P, Zhang M, Hao JJ, Yin NN, Hou XT, Liu JY*
- 10622** Wandering small intestinal stromal tumor: A case report  
*Su JZ, Fan SF, Song X, Cao LJ, Su DY*
- 10629** Acute mesenteric ischemia secondary to oral contraceptive-induced portomesenteric and splenic vein thrombosis: A case report  
*Zhao JW, Cui XH, Zhao WY, Wang L, Xing L, Jiang XY, Gong X, Yu L*
- 10638** Perioperative anesthesia management in pediatric liver transplant recipient with atrial septal defect: A case report  
*Liu L, Chen P, Fang LL, Yu LN*
- 10647** Multiple tophi deposits in the spine: A case report  
*Chen HJ, Chen DY, Zhou SZ, Chi KD, Wu JZ, Huang FL*
- 10655** Myeloproliferative neoplasms complicated with  $\beta$ -thalassemia: Two case report  
*Xu NW, Li LJ*
- 10663** Synchronous renal pelvis carcinoma associated with small lymphocytic lymphoma: A case report  
*Yang HJ, Huang X*
- 10670** *Leclercia adecarboxylata* infective endocarditis in a man with mitral stenosis: A case report and review of the literature  
*Tan R, Yu JQ, Wang J, Zheng RQ*
- 10681** Progressive ataxia of cerebrotendinous xanthomatosis with a rare c.255+1G>T splice site mutation: A case report  
*Chang YY, Yu CQ, Zhu L*
- 10689** Intravesical explosion during transurethral resection of bladder tumor: A case report  
*Xu CB, Jia DS, Pan ZS*
- 10695** Submucosal esophageal abscess evolving into intramural submucosal dissection: A case report  
*Jiao Y, Sikong YH, Zhang AJ, Zuo XL, Gao PY, Ren QG, Li RY*
- 10701** Immune checkpoint inhibitor-associated arthritis in advanced pulmonary adenocarcinoma: A case report  
*Yang Y, Huang XJ*
- 10708** Chondroid syringoma of the lower back simulating lipoma: A case report  
*Huang QF, Shao Y, Yu B, Hu XP*
- 10713** Tension-reduced closure of large abdominal wall defect caused by shotgun wound: A case report  
*Li Y, Xing JH, Yang Z, Xu YJ, Yin XY, Chi Y, Xu YC, Han YD, Chen YB, Han Y*

- 10721** Myocardial bridging phenomenon is not invariable: A case report  
*Li HH, Liu MW, Zhang YF, Song BC, Zhu ZC, Zhao FH*
- 10728** Recurrent atypical leiomyoma in bladder trigone, confused with uterine fibroids: A case report  
*Song J, Song H, Kim YW*
- 10735** Eczema herpeticum *vs* dermatitis herpetiformis as a clue of dedicator of cytokinesis 8 deficiency diagnosis: A case report  
*Alshengeti A*
- 10742** Cutaneous allergic reaction to subcutaneous vitamin K<sub>1</sub>: A case report and review of literature  
*Zhang M, Chen J, Wang CX, Lin NX, Li X*
- 10755** Perithyroidal hemorrhage caused by hydrodissection during radiofrequency ablation for benign thyroid nodules: Two case reports  
*Zheng BW, Wu T, Yao ZC, Ma YP, Ren J*
- 10763** Malignant giant cell tumors of the tendon sheath of the right hip: A case report  
*Huang WP, Gao G, Yang Q, Chen Z, Qiu YK, Gao JB, Kang L*
- 10772** Atypical Takotsubo cardiomyopathy presenting as acute coronary syndrome: A case report  
*Wang ZH, Fan JR, Zhang GY, Li XL, Li L*
- 10779** Secondary light chain amyloidosis with Waldenström's macroglobulinemia and internodal marginal zone lymphoma: A case report  
*Zhao ZY, Tang N, Fu XJ, Lin LE*
- 10787** Bilateral occurrence of sperm granulomas in the left spermatic cord and on the right epididymis: A case report  
*Ly DY, Xie HJ, Cui F, Zhou HY, Shuang WB*
- 10794** Glucocorticoids combined with tofacitinib in the treatment of Castleman's disease: A case report  
*Liu XR, Tian M*
- 10803** Giant bilateral scrotal lipoma with abnormal somatic fat distribution: A case report  
*Chen Y, Li XN, Yi XL, Tang Y*
- 10811** Elevated procalcitonin levels in the absence of infection in procalcitonin-secreting hepatocellular carcinoma: A case report  
*Zeng JT, Wang Y, Wang Y, Luo ZH, Qing Z, Zhang Y, Zhang YL, Zhang JF, Li DW, Luo XZ*

**LETTER TO THE EDITOR**

- 10817** "Helicobacter pylori treatment guideline: An Indian perspective": Letter to the editor  
*Swarnakar R, Yadav SL*
- 10820** Effect of gender on the reliability of COVID-19 rapid antigen test among elderly  
*Nori W, Akram W*

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

Rui Tan, Jiang-Quan Yu, Jing Wang, Rui-Qiang Zheng

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### **Abstract**

#### **BACKGROUND**

Infective endocarditis (IE) is a rare disease with a high mortality rate. *Leclercia adecarboxylata* (*L. adecarboxylata*) is a movable Gram-negative bacillus of enterobacteriaceae, and it can rarely be a pathogen which often affects 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. Surgical treatment was decided to postpone. On the ninth day of admission, the patient developed a sudden high fever and shock and was transferred to the Cardiac Intensive Care Unit, 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*. 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:** *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 infective endocarditis caused by *L. adecarboxylata* that occurred in an immunocompetent male patient. He presented with clinical symptoms after diarrhoea. *L. adecarboxylata* was identified to be the only pathogen and the 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.

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## INTRODUCTION

*Leclercia adecarboxylata* (*L. adecarboxylata*) is a movable Gram-negative rod-shaped bacterium in the *Enterobacteriaceae* family, most commonly found in water and soil[1,2] and rarely isolated from clinical specimens, with most of the *Enterobacteriaceae* characteristics, such as partly anaerobic, oxidase-negative, mesophilic, peripheral flagellate bacilli[3]. Infective endocarditis (IE) caused by *L. adecarboxylata* is a very rare disease in the clinic. Only three cases of *L. adecarboxylata* endocarditis have been reported in the existing literature[4-6]. Herein 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-wk 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 a 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 (MVR).

### History of past illness

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

### Personal and family history

The patient had no personal and family history.

### Physical examination

The patient had a 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

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

### Imaging examinations

The echocardiogram of the patient at 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 (CT) scan 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, we discussed and decided to postpone the surgical treatment.

### Recurrence of disease changes

On the 9<sup>th</sup> day of hospitalization, the patient developed a sudden high fever with a temperature of 38.6°C. The examination showed a 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 1).

### Preliminary diagnosis

The initial diagnosis 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 performed to determine the etiology and susceptibility to antibiotics in this patient with acute IE. Gram staining, antacid bacillus staining, and culture for aerobic and anaerobic bacteria were performed on blood and intraoperatively resected valve vegetation. 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 *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.

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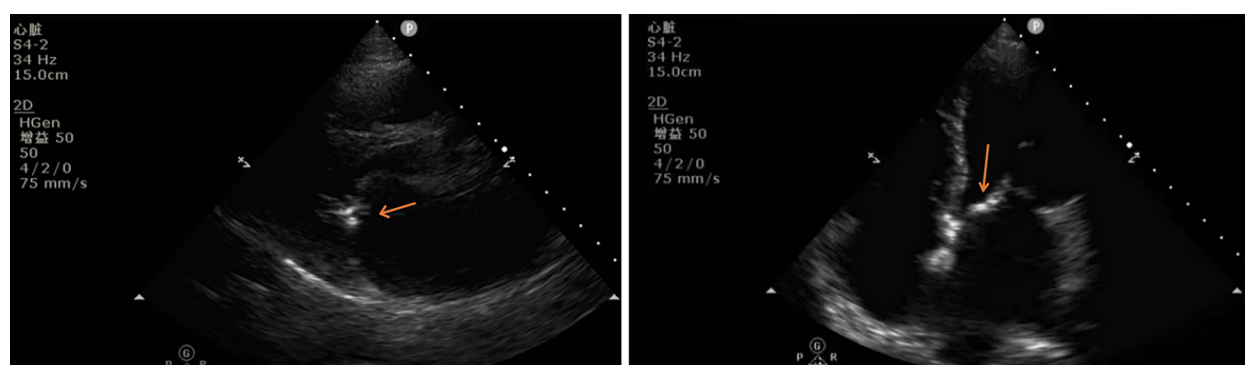
## FINAL DIAGNOSIS

The final diagnosis 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.

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## 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 (1 g q12h) was administered empirically after blood cultures were retained for anti-infective treatment. Considering



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**Figure 1** Bedside echocardiogram showed a vegetation attaching to the anterior leaflet of mitral valve (as shown by arrows).

that the patient had acute IE with shock, which is 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 (24 µg/min) to maintain blood pressure and circulatory stability, vancomycin (1 g q12h) for experienced anti-infection, and warfarin (2.5 mg 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. Two tubes of blood were obtained for pre-operative blood cultures, suggesting *L. adecarboxylata* infection, and anti-infective therapy with piperacillin tazobactam (4.5 g q8h) in combination with vancomycin (1 g 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 3 d after surgery, and negative on the fifth and seventh postoperative days. On the 17<sup>th</sup> day of hospitalization, the patient returned to the general ward of the Cardiacvascular Surgery Department and continued anti-infective treatment with vancomycin (1g q12h) and piperacillin tazobactam (4.5 g 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 the follow-up visit 1 mo after surgery, the patient was asymptomatic, and a new echocardiography showed that the patient had normal biologic valve function after MVR, and no abnormal echogenicity was detected around the valve.

## DISCUSSION

The first case of aortic valve disease combined with endocarditis was reported by Lazare Rivière[7] in 1646, and in 1806, Jean-Nicolas Corvisart[8] introduced the term 'vegetation'. Jean Baptiste Bouillaud[9] called the inner layer of the heart the "endocardium", and the term "endocarditis" came into use in 1835 [10]. IE is a rare disease with a high mortality rate, ranging from 1.5 to 11.6 per 100000 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% [11] and 5-year mortality rates as high as 45% [12]. In the real clinic, these figures may be underestimated [13].

Although the incidence varies little numerically between countries, there are significant differences in patterns. 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 [14], 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 [15,16]. Currently, the most common susceptibility disease in IE is valvular heart disease [17], and with an ageing population, there is an increasing incidence of degenerative valve disease such as mitral and aortic regurgitation [18]. Of these, mitral valve prolapse is the predominant structural abnormality in native valve endocarditis. Nosocomial infections are also an important factor in the disease, with

hospital-acquired staphylococci being the main source of infection in high-income countries, mainly affecting the elderly[11].

As a typical lesion of IE, 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[19]. Endothelial damage triggers thrombosis caused by fibrin and platelet deposits [20]. When bacteraemia occurs, bacteria can reach and colonise these damaged areas. 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[21].

IE includes both natural valve endocarditis and IE caused by intracardiac prosthetic materials. The latter includes prosthetic valve endocarditis[22] and IE associated with pacemakers, implantable cardiac defibrillators, and ventricular assist devices. Early and accurate diagnosis of IE is essential, as delayed treatment can affect the prognosis of the disease[23,24]. The diagnosis of IE is widely made using microbiological techniques and imaging techniques (echocardiography, CT, and nuclear imaging). 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[22]. The 2000 modified Duke criteria classify the diagnosis into 'major' and 'minor' criteria and classify cases as "definite", "possible", or "rejected"[25]. 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 echocardiography.

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

The clinical features of IE are highly variable and depend largely on the infecting organism and the medical history of heart disease or not[27,28]. The clinical presentation may be acute, subacute, or chronic[29]. Approximately 90% of infected patients present with fever, and IE is also an important cause of unexplained fever[30-32]. It is accompanied by various systemic symptoms such as appetite loss and weight loss[33]. About 85% of patients have heart murmurs[34]. Clinical features also include neurological signs and symptoms[35]. IE is a risk factor for stroke[36]. 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[37-40].

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[41]. There is no standardisation in the use of antibiotics[42]. 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[43]. The most common of these pathogens is *Staphylococcus aureus*[43]. Successful treatment of IE relies on the eradication of microorganisms with 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[44]. Antibiotics are administered immediately after blood cultures are performed. The choice of antibiotics 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[29].

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[45]. Effective surgical treatment can reduce mortality by 8%-16%[46,47]. 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[26]. 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%)[48]. Surgery is usually recommended for this group of patients early in the disease, during hospitalization, and before the end of antibiotic therapy[41]. Patients with refractory pulmonary oedema or cardiogenic shock due to IE should undergo emergency surgery within 24 h[26].

In IE, the mitral valve is often affected[49]. The prognosis for IE with mitral valve lesions is often poor, and extensive excision of the infected focus and 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 are comparable to those 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[50]. 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[51].

Gram-positive bacteria account for more than 70% of IE cases, and are the main pathogen of community-acquired and hospital-acquired IE cases[52]. Gram-negative bacteria usually do not cause IE [53]. Gram-negative IE accounts for less than 5% of IE cases[54]. In the past 50 years, the incidence of Gram-negative endocarditis has been increasing[55,56]. In addition to the traditional risk factors of structural heart disease (congenital or acquired)[57,58], most cases of Gram-negative endocarditis are mainly caused by medical related events[59]. Patients with indwelling catheters, invasive lines, and intracardiac devices are at higher risk[60,61]. Among patients with Gram-negative endocarditis, 60% had renal insufficiency[17], and about 52% had embolic symptoms[60]. 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[41,62]. A large prospective study found that the incidence of new murmurs was 48%-60%, and 30%-40% of patients lost weight[17, 59]. Studies have reported that about 11%-33% of patients have splenomegaly[59,63].

*L. adecarboxylata* was first described by H. Leclerc[64] in 1962 and was previously known as "Enteric group 410 or *Escherichia adecarboxylata*". The name *L. adecarboxylata* was officially given by Tamura et al [65,66] 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[67].

*L. adecarboxylata* is a ubiquitous microorganism, most commonly found in water and soil[1,2]. 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[68]. *L. adecarboxylata* also exists in the intestinal symbiotic flora of some animals[69], 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[3].

*L. adecarboxylata* is a rare emerging pathogen and immunosuppression is the most significant risk factor for infection with *L. adecarboxylata*, which often affects immunodeficient patients, including those with immunomodulators and haematological malignancies or receiving chemotherapy[70], and is the only isolate cultured from blood, sputum, urine, and peritoneal fluid in immunosuppressed individuals [71-73]. There are currently about three reported cases of immunocompetent patients with monomicrobial *L. adecarboxylata* infection[74-76]. The remaining cases are all immunosuppressed patients[70]. *L. adecarboxylata* is usually isolated as part of a polymicrobial infection in immunocompetent patients[66, 67,70]. Because of its low virulence, *L. adecarboxylata* is often overlooked in the context of polymicrobial infections, and asymptomatic carriage or colonization is common. 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*[77,78].

*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, and catheters[79,80]. A few cases with diabetes as the only risk factor have *L. adecarboxylata* infection of the skin and soft tissues[81,82]. *L. adecarboxylata* has been implicated in endocarditis[6], catheter-associated bacteremia[78], bacteremia, sepsis[3,67], septic arthritis, meningitis[83], cellulitis[2,84], urinary tract infections[85], pneumonia[70], and bacterial peritonitis, especially in patients on peritoneal dialysis[86]. 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)[87], and intestinal translocation (translocation of bacteria through the mucosal barrier of the gastrointestinal tract, presumably gastrointestinal bacteremia)[2,70].

Bacteremia may be due to bacterial translocation across the intestinal mucosal barrier as a result of megacolon, antibiotic use, and altered dietary habits. To assess the relationship between gastrointestinal pathology and *L. adecarboxylata* bacteraemia, four cases of *L. adecarboxylata* associated with gastrointestinal pathology were reviewed. Three of these reported cases presented with only intestinal mucosal disturbances with *L. adecarboxylata* as a possible source of infection[74]. 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[88]. 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[76]. 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 a vegetation in the anterior mitral leaflet, and cultures of blood and vegetation showed *L. adecarboxylata* infection. Of the reported cases of

endocarditis in the world, only three were caused by *L. adecarboxylata*[4-6]. 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)[25]. After mitral valve replacement and 4-wk 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 remain 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[74]. Of the case reports of endocarditis found worldwide, only three cases of endocarditis were caused by *L. adecarboxylata*[4-6]. 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.

## FOOTNOTES

**Author contributions:** Tan R and Yu JQ were the patient's Cardiac Intensive Care Unit doctors, reviewed the literature, and contributed to the drafting of the manuscript; Wang J reviewed the literature and contributed to the drafting of the manuscript; Zheng RQ was responsible for revising the manuscript for important academic content; all authors issued final approval of the version to be submitted.

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