Case report

Infective endocarditis caused by *Escherichia coli* of a native mitral valve

E. Benaisssa<sup>a,b</sup>,<sup>a</sup>, Ben Lahlou Yasssine<sup>a,b</sup>, M. Chadli<sup>b</sup>, A. Maleb<sup>c</sup>, M. Elouennass<sup>a,b</sup>

<sup>a</sup>Epidemiology and Bacterial Resistance Research Team/BIO-INOVA Centre, Faculty of Medicine and Pharmacy (University Mohammed V), Rabat, Morocco
<sup>b</sup>Department of Bacteriology, Mohammed V Military Teaching Hospital / Faculty of Medicine and Pharmacy (University Mohammed V), Rabat, Morocco
<sup>c</sup>Laboratory of Microbiology, Mohammed VI University Hospital / Faculty of Medicine and Pharmacy (University Mohammed the First), Oujda, Morocco

**ABSTRACT**

*Escherichia coli* is a rare cause of infectious endocarditis. We report a clinical case of *E. coli* endocarditis of a native mitral valve in a young 26-year-old woman with a recurrent urinary tract infection who had a high fever for one week despite probabilistic treatment with amoxicillin-clavulanic acid 3 g per day. The patient was successfully treated with antibiotics and recovered without surgery.

© 2021 Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

**Background**

Infectious endocarditis (IE) caused by *Escherichia coli* (*E.coli*) is rare, despite the frequency of septicemia due particularly to this bacterium. *E. coli* is the causative microorganism in approximately 0.51% of cases of IE [1]. Thirty-six cases of *E. coli* native valve IE that met the Duke criteria were reported in the literature from 1909 to 2002, and urinary tract infection was the most common cause of endocarditis due to *E. coli* [2]. The low incidence of *E. coli* IE has been attributed to the inability of this bacterium to adhere to the endocardium as well as the existence of antibodies to *E. coli* in normal serum [3]. Notably, however, the number of > 70-year-old patients with *E. coli* IE has recently increased, and about 70% of affected patients are older women [2]. In addition, the mortality rate of *E. coli* IE (21%) is higher than that of IE due to *Haemophilus* spp. *Aggregatibacter* spp, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* spp (HACEK group) [1,4].

We report a clinical case of *E. coli* endocarditis of a native mitral valve in a young 26-year-old woman with a recurrent urinary tract infection who had a high fever for one week despite probabilistic treatment with amoxicillin-clavulanic acid 3 g per day.

**Case report**

The patient, 26 years old woman, had been presenting for one week a high fever despite probabilistic treatment with amoxicillin-clavulanic acid 3 g per day, asthenia, chills, palpitations, general alteration of state, without any notion of chest pain and syncope. The anamnesis reported a notion of untreated stomatological affection and recurrent urinary tract infections. The initial clinical examination found the patient to be febrile at 39.9 °C, blood pressure at 10/6 mmHg, heart rate at 130 bat/min and respiratory rate at 24 cycles/min. Cardiovascular examination revealed stetho-acoustic semiology of mitral and aortic insufficiency. Transthoracic and transoesophageal echocardiography revealed the presence of a vegetation at the mitral valve (18 × 12 mm) (Fig. 1).

A chest radiograph showed no obvious signs suggesting pneumonia or pulmonary congestion. Electrocardiography showed sinus tachycardia and no other abnormalities. Laboratory tests showed a white blood cell count of 15,500/mm<sup>3</sup> with neutrophilic polynuclear predominance, C-reactive protein at 316 mg/dl, procalcitonin at 24 μg/L, hypochromic microcytic anemia with normal ferritin levels. Blood cultures were done by collecting three consecutive blood samples at intervals of one hour. The blood culture bottles were incubated at 37 °C for 18–24 h. The broth was subcultured on to 5% sheep blood agar and Bromocresol purple and incubated at 37 °C. After 18 h of incubation, Bromocresol purple agar medium showed lactose-fermenting colonies, about 2–3 mm in diameter. Colonies on blood agar were grey and non-haemolytic. They were found to be Gram-negative, motile, pleomorphic, cocobacilli, which were oxidase negative and catalase positive. An API 20E<sup>®</sup> gallery (Bio- Mérieux, Marcy l’étoile France) allowed the...
Identification of 98.9% E. coli with excellent identification (Code 5144572). Culture of the urine also grew E. coli with a colony count of >10^5 CFU/mL. The sputum culture showed no significant growth of pathogenic bacteria. The antibiogram was carried out by diffusion method in Mueller-Hinton agar medium in compliance with the recommendations of EUCAST 2020. The study of antibiotic sensitivity showed that this strain was resistant to ampicillin, ticarcillin and amoxicillin-clavulanic acid, sensitive to ceftriaxone, cefotaxime, imipenem, gentamicin, amikacin, ciprofloxacin, norfloxacin, Piperacillin + Tazobactam, cefoxitin and sulfamethoxazole-trimethoprim. Based on these findings, the patient was diagnosed with IE due to E. coli in accordance with the modified Duke criteria [5].

The patient was treated with intravenous infusion of ceftriaxone 2 g twice daily for six weeks and amikacin 160 mg once daily for ten days. He responded well to treatment and was afebrile within 72 h after initiation of therapy. Antibiotic treatment was continued for sex weeks. Repeat blood cultures were sterile.

Discussion

E. coli has emerged in recent years as an increasingly important cause of morbidity and mortality in both immunocompetent and immunosuppressed persons [6]. Nevertheless it remains an extremely uncommon cause of IE [7]. The increase in the numbers of immunocompromised patients has led to a change in the spectrum of organisms causing native valve endocarditis. E. coli is a common cause of urinary tract infections [6]. The low incidence of endocarditis caused by this organism has been attributed to its inability to adhere to endocardium, and also to the fact that normal serum often has antibodies to E. coli [8]. Gram-negative bacteria are less sensitive to complement-mediated lysis and other humoral innate immune defences; they lack surface proteins that specifically bind host matrix molecules and prosthetic material which make them rare causative agents of IE [6]. However, they possess virulence factors such as adhesins, iron acquisition systems, and toxins which make them serious pathogens once they gain entry into a normally sterile extra intestinal site [9]. IE caused by Gram-negative organisms is associated with high mortality and significant morbidity and necessitates aggressive medical management and early surgical intervention. This patient had no history of cardiac disease and the source of the infection was likely to be the urinary tract. He responded well to antibiotic therapy and did not require surgical intervention.

Conclusion

Urinary tract infection appears to be an important predisposing factor in the development of E. coli endocarditis in this young woman who had no specific cardiac risk factors. Persistent fever even in an immunocompetent individual with a urinary tract infection despite specific antibiotic treatment should be investigated to rule out serious infections such as endocarditis.

Author statement

All authors have seen and agreed to the submitted version of the paper, and bear responsibility for it.

Ethical approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the ethics committee of the Mohammed V Military University Hospital in Rabat.

Author contributions

BE, YB have been involved in drafting in the manuscript, CM, MA ha revising the manuscript and ELM have given final approval of the version to be published.

Declaration of Competing Interest

The authors report no declarations of interest.

References

[1] Morpeth S, Murdoch D, Cabell CH, Karchmer AW, Pappas P, Levine D, et al. International collaboration on endocarditis prospective cohort study (ICE-PCS) investigators. Non-HACEK gram-negative bacillus endocarditis. Ann Intern Med 2007;147:829–35, doi:http://dx.doi.org/10.7326/0003-4819-147-12-200712180-00002.
[2] Micol R, Lorbolay O, Jaureguy F, Bonacors S, Bingen E, Lefort A, et al. Escherichia coli native valve endocarditis. Clin Microbiol Infect 2006;12:401–3, doi:http://dx.doi.org/10.1111/j.1469-0691.2006.01375.x.
[3] Watanakunakorn C, Burkett T. Infective endocarditis in a large community teaching hospital, 1980–1990. A review of 210 episodes. Medicine 2013;92:100, doi:http://dx.doi.org/10.1097/MDR.0000000000000003.
[4] Chambers ST, Murdoch D, Morris A, Holland D, Pappas P, Almela M, et al. International collaboration on endocarditis prospective cohort study investigators. HACEK infective endocarditis: characteristics and outcomes from a large, multinational cohort. PLoS One 2013e63181 8.
[5] Baddour LM, Wilson WR, Bayer AS, Fower VG, Tleyjeh IM, Rybak MJ, et al. Infective endocarditis in adults: diagnosis, antimicrobial therapy, and management of complications. Circulation 2015;132:1435–86, doi:http://dx.doi.org/10.1161/CIR.0000000000002296.
[6] Menon T, Balakrishnan N, Somasundaram S, Dhandapani P. Native valve endocarditis caused by Escherichia coli. J Clin Diagn Res 2017;11(6):DD05–6, doi: http://dx.doi.org/10.7860/JCDR/2017/27201.10046.
[7] Johannes S. Escherichia coli endocarditis of the aortic valve with formation of a paravalvular abscess cavity. Echocardiography 2005;22:126.
[8] Watanakunakorn C, Burkett T. Infective endocarditis in a large community teaching hospital 1980–1990. A review of 210 episodes. Medicine 2013;72:120.
[9] Russo TA, Johnson JR. Proposal for a new inclusive definition for extraintestinal pathogenic isolates of Escherichia coli: ExPEC. J Infect Dis 2000;181:1753–4.