SUBMITTED 29 SEP 20
REVISION REQ. 23 DEC 20; REVISION 18 JAN 21
ACCEPTED 7 APR 21
ONLINE-FIRST: MAY 2021
DOI: https://doi.org/10.18295/squmj.5.2021.076

A rare case of emphysematous endocarditis caused by Escherichia coli

***Ahmed S. Al-Dhahli,[1] Rashid Al-Umairi,[2] Osama Elkadi[1]**

[1]Department of Radiology, Rustaq Hospital, Ministry of Health, Rustaq, Oman; [2]Department of Radiology, Royal Hospital, Muscat, Oman
*****Corresponding Author's e-mail: asdhahli@gmail.com*

Abstract

Infective endocarditis (IE) is an infection of the heart endocardium with significant morbidity and mortality. Gram negative infection particularly emphysematous type of IE is an extremely rare and life-threatening disease. We report a 59-year-old diabetic woman who was admitted to the Rustaq Hospital, Rustaq, Oman in 2017 with the diagnosis of pneumonia for which she was started on antibiotics. Shortly afterwards, she developed facial and mouth deviation and became more tachypneic. Computed tomography of the brain demonstrated bilateral multiple small infarcts. Pulmonary angiography computed tomography (CTPA) was performed which ruled out pulmonary embolism. Nonetheless, it revealed an air containing lesion around the mitral valve. Transthoracic echocardiography demonstrated a hyperechoic mobile lesion related to the mitral valve. Blood culture grew *Escherichia Coli* (*E. coli*). Diagnosis of *E. coli* Emphysematous IE was made based on modified Duke criteria. Recommended treatment for non-HACEK IE includes extended antibiotic course and surgery for selected patients.

Keywords: Endocarditis; Escherichia coli; Mitral Valve; Echocardiography; Embolism, Intracranial; Emphysema; Case Reports; Oman.

Introduction

Infective endocarditis (IE) is a life-threatening condition which is defined as an infection of the cardiac endocardium. The valves are the most common cardiac structure affected by the

disease. IE is the most prevalent infection of the cardiovascular system.[1] Patients can present with constitutional symptoms including fever, chills and weight loss. Other clinical presentations of the disease are related to cardiac involvement including heart failure, vasculitis and systemic embolic phenomena.[2]

IE is caused by gram positive organisms in majority of the patients. *Staphylococci*, *streptococci* and *enterococci* are responsible for more than 80% of IE cases.[3] Rarely, IE can be caused by a group of gram-negative organisms other than *Haemophilus species, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens*, or *Kingella species* known as non-HACEK gram-negative bacteria.[4, 5] In older publications, IE caused by non-HACEK gram-negative organisms was predominantly seen among intravenous (IV) drug users. However, more recent studies have reported that the majority of non-HACEK IE was among patients with implanted endovascular devices, including prosthetic valves, permanent pacemakers and implantable cardioverter-defibrillators.[6]

Likewise, *E. coli* IE is rare with limited number of reported cases in the literature and it is usually associated with higher mortality rate compared to HACEK gram-negative IE.[4-6] The reported inpatient mortality rate is 24% for non-HACEK IE in comparison to 4% for the HACEK IE .[7,8] Al Abri S et al has reported 4.1% incidence of *E.coli* IE in Oman.[9] Emphysematous type of IE is extremely rare with very limited reported cases in the literature.[3, 10, 11,12] In this paper we report a 59-year old woman with an emphysematous IE caused by *E. coli*.

Case Report

A 59-year-old woman presented to the emergency department in the Rustaq Hospital, Oman in 2017 with 5 days history of cough, shortness of breath, fever and chest discomfort. The patient had a medical history of uncontrolled diabetes, hypertension and ischemic heart disease. Her echocardiography 10 years prior to admission demonstrated left ventricular hypertrophy with a normal left ventricle ejection fraction. There was no mitral or tricuspid valve regurgitation.

On physical examination, she was conscious, alert and oriented. Her vital signs were as follows: blood pressure 102/60 mm Hg, body temperature 38°C, heart rate 109 beat per minute, O2 saturation 90% and random blood sugar 19.3 mmol/L. Chest auscultation

revealed bilateral fine crepitation more on the right. The remainder of the systemic examination was unremarkable.

Laboratory investigations showed high white blood cell counts 17.8 x 10^3 /uL (normal range: 2.4-9.5 10^3/uL) with 81% neutrophil predominance, high glycosylated hemoglobin 9.6% (normal range: 4.8 – 6%) and high troponin T 153.1 pg/mL (normal range:0-14 pg/mL). Platelet count, hemoglobin, creatinine and urea levels were normal. Her chest radiograph showed a patchy opacity which was interpreted as pneumonic consolidation for which the patient was started on ceftriaxone.

On the second day of admission, the patient's Glasgow Coma Scale deteriorated; as she was responsive only to pain stimuli. Subsequently, she developed facial and mouth deviation so that a non-contrast enhanced Computed Tomography of the brain was performed using 64-slice Philips CT machine that demonstrated multiple small subtle hypodense foci in both centrum semiovali (Figure 1) and corpus callosum. The distribution and characteristics of the hypodensities were suggestive of acute ischemic infarctions due to thromboembolic cause.

On the third day of admission, the patient was persistently febrile despite antibiotic administration and blood pressure dropped to 87/50 mmHG. Pulmonary embolism (PE) was suspected based on high D-dimer and worsening tachypnea. Subsequently, a Pulmonary angiography computed tomography (CTPA) was performed on a 64-slice Philips CT machine. The procedure was performed craniocaudally with the following parameters: 0.625 mm detectors, auto-mA (average of 224 mA), 120 kVp, and a 512×512 matrix. The study was done with IV contrast material utilizing pulmonary embolism protocol. Coronal and sagittal reformats were reconstructed. The study ruled out PE, but it demonstrated an air containing lesion around the mitral valve (Figure 2). There was mitral valve annulus calcification. Transthoracic echocardiogram was performed which revealed mild mitral regurgitation with a mobile hyperechoic mass related to the anterior mitral valve leaflet associated with posterior acoustic shadow (Figure 3). The left ventricle ejection fraction was 25%. Blood culture from a single blood sample yielded growth of gram negative bacilli *Escherichia Coli* (*E. coli*) and consequently the antibiotic was changed based on sensitivity profile to Piperacillin/tazobactam 4.5 grams TID on third day of admission. Diagnosis of infective endocarditis was established based on modified Duke clinical criteria.[8]

Nevertheless, the patient's clinical condition deteriorated on fourth day of admission and eventually she arrested with ventricular arrhythmia then into asystole leading to her demise after a short period of restoration of normal sinus rhythm.

Discussion

In our case report, we describe a patient with uncontrolled diabetes who was admitted with an impression of pneumonia and who was later on found to have emphysematous infective endocarditis (IE) that ultimately led to patient's death. Although, we were not able to obtain tissue sample to confirm the diagnosis of IE, we established the diagnosis of possible IE based on modified Duke criteria[8]: One major criterion (endocardial involvement as evident in echocardiogram as a lesion attached to the mitral leaflet), and two minor criteria (persistent fever and vascular phenomenon of multiple small embolic infarcts in the brain). The positive blood culture was yielded from a single blood sample which revealed growth of *E. coli* which is an atypical microorganism for IE. No significant bacterial growth could be obtained from urine culture, and no other source of infection could be identified.

E. coli is the most common non-HACEK gram-negative organism that cause IE, accounting for approximately one third of the cases.[10] IE secondary to *E. coli* is usually associated with higher mortality and morbidity in comparison to IE due to HACEK gram-negative organisms. The source of IE secondary to *E. coli* is usually urinary tract infection.[4, 5] Diabetes, malignancy, excessive alcohol consumption, and hemodialysis are considered to be risk factors for developing IE secondary to E. *coli*.[6]

In our case, air containing lesion was seen around the mitral valve on CTPA indicating emphysematous type of IE (CT is the radiological gold standard tool for the diagnosis of emphysema). Literature review revealed two reported cases of *E. coli* related emphysematous IE.[3,12] There are two additional Emphysematous IE cases reported in the literature caused by *Finegoldia magna* and *Citrobacter koseri.* [10, 11] The case reports share similar radiological finding with our case of air density vegetation around the mitral valve. Furthermore, all patients in these few case reports had diabetes mellitus including our patient.[3,10,11,12]

Choosing appropriate antibiotic treatment for Non-HACEK gram negative related IE is based on sensitivity profile of the responsible pathogen.[13] Treatment by a combination of antibiotics with β-lactams (penicillins, cephalosporins, or carbapenems) and either an

aminoglycoside or fluoroquinolone for 6 weeks is considered appropritate.[8] An infectious disease expert opinion in IE should be obtained due to known antibiotic resistance in Non-HACEK organisms.[8] Some patients in this group may require surgical intervention especially for *Pseudomonas* related left-sided IE. [8,13]

Conclusion

Emphysematous infective endocarditis caused by *E. coli* is an extremely rare disease that is associated with increased mortality and morbidity. Therefore, clinicians should be aware of this entity and pay close attention to the clinical course of patients with *E. coli* IE during hospitalization in order to reduce mortality and morbidity related to the disease.

References

1. Yusuf, S., Sharma, J., Durand, J. and Banchs, J., 2014. Endocarditis and myocarditis: a brief review. Expert Review of Cardiovascular Therapy. 2014;10(9), pp1153-64. https://doi.org/10.1586/erc.12.107
2. 2. Ferro J, Fonseca A. Infective endocarditis. Handbook of Clinical Neurology. 2014;75-91. https://doi.org/10.1016/B978-0-7020-4086-3.00007-2.
3. Kim C-J, Yi J-E, Kim Y, Choi HJ. Emphysematous endocarditis caused by AmpC beta-lactamase-producing Escherichia coli: A case report. Medicine. 2018;97(6):e9620-e DOI: 10.1097/MD.0000000000009620.
4. Chambers ST, Murdoch D, Morris A, Holland D, Pappas P, Almela M, et al. HACEK infective endocarditis: characteristics and outcomes from a large, multi-national cohort. PLoS One. 2013;8(5):e63181-e DOI: 10.1371/journal.pone.0063181.
5. Morpeth S, Murdoch D, Cabell CH, Karchmer AW, Pappas P, Levine D, et al. Non-HACEK gram-negative bacillus endocarditis. Ann Intern Med. 2007;147(12):829-35 DOI: 10.7326/0003-4819-147-12-200712180-00002.
6. Akuzawa N, Kurabayashi M. Native valve endocarditis due to Escherichia coli infection: a case report and review of the literature. BMC Cardiovasc Disord. 2018;18(1):195- DOI: 10.1186/s12872-018-0929-7.
7. Chambers S, Murdoch D, Morris A, Holland D. et al HACEK Infective Endocarditis: Characteristics and Outcomes from a Large, Multi-National Cohort. PLoS ONE. 2013;8(5):e63181. DOI: 10.1371/journal.pone.0063181.
8. Baddour LM, Wilson WR, Bayer AS, Fowler VG, Jr., Tleyjeh IM, Rybak MJ, et al. Infective Endocarditis in Adults: Diagnosis, Antimicrobial Therapy, and Management of Complications: A Scientific Statement for Healthcare Professionals From the

American Heart Association. Circulation. 2015;132(15):1435-86 DOI: 10.1161/cir.0000000000000296.

9. Al Abri S, Zahedi F, Kurup P, Al-Jardani A, Beeching N. The epidemiology and outcomes of infective endocarditis in a tertiary care hospital in Oman. Journal of Infection and Public Health. 2014;7(5):400-406. https://doi.org/10.1016/j.jiph.2014.04.004
10. Cohen M, Stephens EH, Garshick M, Wang S-H, Frager SZ, Mautner L, et al. Native Valve Emphysematous Endocarditis Caused by Finegoldia magna in a Novel Pathogenic Role. Infectious Diseases in Clinical Practice. 2016;24(1).
11. Kesler S, Kim H, Perlman D, Dincer HE, Thenappan T, Tomic R. Air in the Left Ventricle. An Unusual Case of Endocarditis. Am J Respir Crit Care Med. 2016;193(10):1176-7 DOI: 10.1164/rccm.201506-1156IM.
12. Law D, Thomas M. Escherichia coli emphysematous endocarditis. The Lancet Infectious Diseases. 2020;20(3):381. https://doi.org/10.1016/S1473-3099(19)30700-5.
13. Sexton D, Chu V. Antimicrobial therapy of left-sided native valve endocarditis. From https://www.uptodate.com/contents/antimicrobial-therapy-of-left-sided-native-valve-endocarditis. Accessd: January 2021.

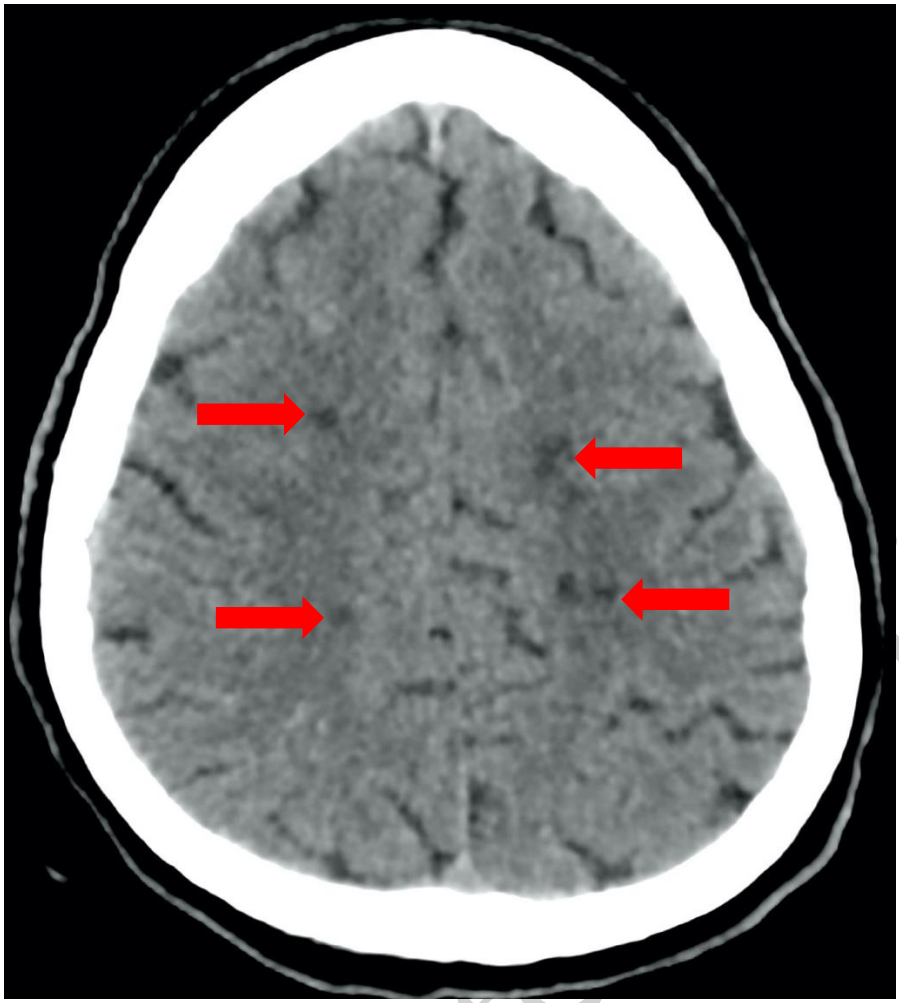

Figure 1. Non-Contrast enhanced Computed Tomography of the brain showing bilateral ill-defined small hypodense foci in bilateral centrum semiovale and corpus callosum (not shown) suggestive of acute infarction (Red arrows).

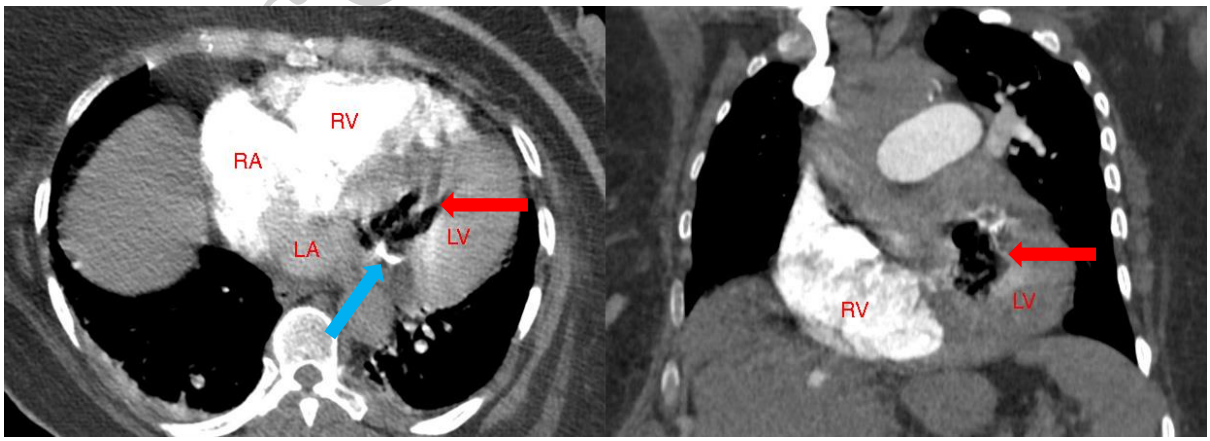

Figure 2. Right: Axial Pulmonary Angiography Computed Tomography (CTPA) showing an air containing lesion around the mitral valve (Red arrow). Note is made of mitral annual calcification (Blue arrow). Left: Coronal reformat demonstrating the air density lesion in the mitral valve (red arrow). RA: Right atrium, RV: Right ventricle, LV: left ventricle, LA: Left atrium

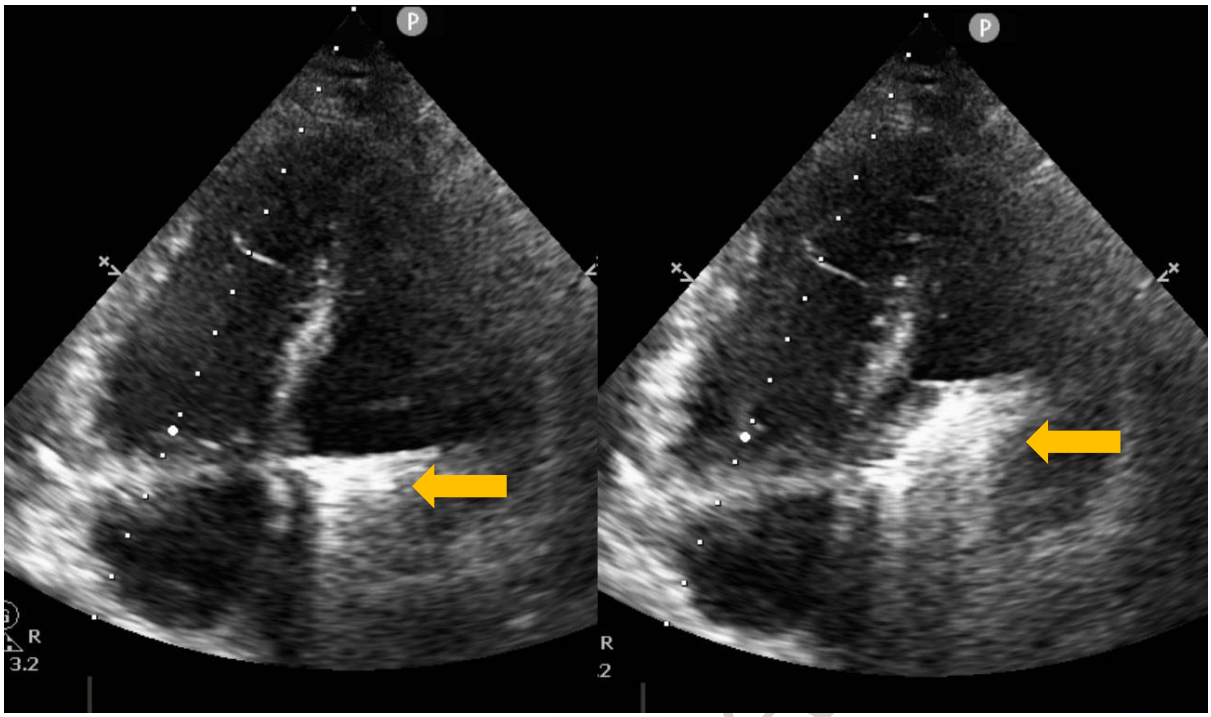

Figure 3: Right. Four chamber view echocardiogram in systole showing a hyperechoic lesion related to anterior leaflet of the mitral valve (arrow). Left: Redemonstration of the mobile hyperechoic lesion related to the mitral valve in diastole.