sciendo

10.2478/AMB-2022-0029

CASE SERIES

SURGICAL TREATMENT OF INFECTIVE ENDOCARDITIS, COMPLICATED WITH SPLENIC ABSCESSES

A. Keltchev[1], S. Kazakov[1], M. Alexieva[2], E. Mekov[3], G. Yankov[2]

[1]Department of Cardiac Surgery, University Hospital "Acibadem City Clinic" – Sofia, Bulgaria
[2]Department of Respiratory Diseases, Medical University – Sofia, Bulgaria
[3]Department of Occupational Diseases, Medical University – Sofia, Bulgaria

Abstract. *Infectious endocarditis (IE) represents an inflammation of the heart endothelium usually as a result of bacterial colonization after trauma. Splenic abscess is a rare and dangerous extracardiac manifestation of IE. An expert multidisciplinary collaboration is necessary for the diagnosis and treatment strategy of this high-risk and challenging disease. Valvuloplasty and splenectomy are the mainstay of radical treatment and offer excellent long-term results. We present two cases with infectious endocarditis, complicated with splenic abscesses, which were successfully operated in the Department of Cardiac Surgery.*

Key words: *infective endocarditis, splenic abscess, valvuloplasty, splenectomy*

Corresponding author: *Assoc. Prof. Evgeni Mekov, MD, PhD, UMHAT „Sv. Ivan Rilski", 15, „Acad. Ivan Geshov" blvd, 1431 Sofia, Bulgaria, e-mail: evgeni.mekov@gmail.com*

Received: *11 July 2022* – ***Accepted:*** *6 August 2022*

INTRODUCTION

Infectious endocarditis (IE) represents an inflammation of the heart endothelium, usually as a result of bacterial colonization after trauma. Splenic abscess is a rare and dangerous extracardiac manifestation of IE. We present two cases of patients with infectious endocarditis, complicated with splenic abscesses, who were successfully operated in the Department of Cardiac Surgery at University Hospital "Acibadem City Clinic" in Sofia.

Case 1

A 49-year-old woman was admitted to the Department of Cardiac Surgery with complaints of sore throat, fever, and diffuse body pain. As a concomitant disease, she reported an ischemic stroke. The patient was intoxicated and in severely impaired overall condition. Mild tachycardia, decreased heart sounds, diastolic murmur 2/6 in the second right intercostal space and irradiating to the heart apex, and mild tenderness in the left hypochondrium were present on physical exam. There were petechiae in both upper and lower extremities, small erythematous lesions of palms and under nails bilaterally, Janeway lesions, and Osler nodules. Laboratory values revealed mild anemia, hyperglycemia, and hypoproteinemia. Chest x-ray showed bilateral reticular shadows, congestive hila, and bilateral small pleural effusions. CT scan revealed bilateral pleural and pericardial effusions and splenomegaly up to 140 x 48 x 115 mm (Fig. 1). In splenic parenchyma hypodense, fluid-equivalent zones (liquefication) were presented with irregular shape and density around 25HE, indicative of abscesses, most prominent in the upper pole with dimensions in the axial

plane of 80 x 40 mm. The perisplenic fat planes were compacted. There was thickened mesenteric fat tissue (mesenteric panniculitis), mild enlarged single mesenteric lymph nodes, and a small amount of liquid in the minor pelvis. The electrocardiogram showed sinus tachycardia. Echocardiography revealed a tricuspid aortic valve with vegetation in noncoronal cusp with dimensions of 12/9 mm, a perforation of noncoronal cusp, severe aortic insufficiency, which occupies the whole left ventricle outflow tract (LVOT) (Fig. 2). There was a small pericardial effusion (100 ml), and small bilateral pleural effusions (right – 400 ml, left – 300 ml). Abdominal echography revealed splenomegaly, 45 mm in the hilum, two cystic structures in the middle, and a mild perisplenic effusion.

A patch plasty of paravalvular abscess of the aortic valve, aortic valve replacement with mechanical prosthesis ATS No 20, mitral valvuloplasty with a ring Profile No 28, and splenectomy was performed.



Fig, 1. Case 1. Abdominal CT scan, showing bilateral pleural effusions, splenomegaly, and splenic abscesses

We proceeded with upper midline laparotomy and a very enlarged and septic spleen was revealed, closely adhered to the diaphragmatic peritoneum. A ruptured subcapsular abscess was found, covered and limited by the diaphragmatic peritoneum. Gastrosplenic, sple-

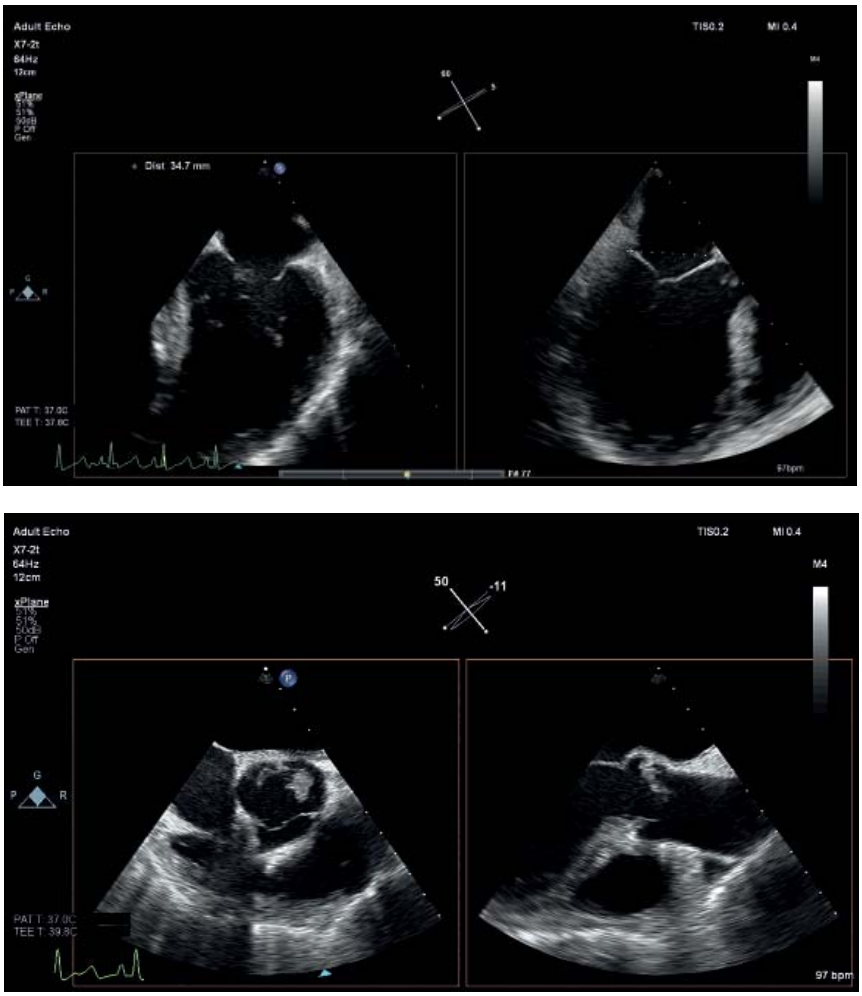


Fig. 2. Echocardiographic images, demonstrating infective vegetations

nocolic, lienorenal, and pancreaticosplenic ligaments were transected using Ligasure. Splenic artery and vein were ligated and transected and splenectomy was performed. The splenic bed was lavaged with diluted Braunol solution. Longitudinal sternotomy and pericardiotomy were performed in extracorporeal circulation settings and a Custodiol cardioplegic arrest of the heart. A tricuspid aortic valve with cusps vegetations was revealed with perforation in the base of the left coronary cusp (LCC) and two paravalvular abscesses in the region of commissures L/N and L/R (Fig. 3). The aortic valve was excised and the paravalvular abscess cavities were closed with a pericardial patch and continuous running suture 5/0 Prolene. The left atrium was enlarged and a mitral valve with a dilated ring was found. Ring annuloplasty was performed with Medtronic Profile ring No 28 through 13 interrupted mattress sutures Ethibond 2/0. The left atrium was closed with two opposite running Prolene sutures 3/0. At the aortic position, mechanical valve prosthesis Medtronic No 20 was implanted with 12 interrupted mattress Ethibond sutures 2/0 with pledgets. Aortic closure a Modo De Bakey was performed through two opposite continuous 4/0 Prolene sutures. In addition, implantation of an atrium-ventricular pacemaker due to bradycardia was placed. Two drains were positioned retrosternally and retrocardially.

Macroscopically a septic spleen with multiple parenchymal and subcapsular abscesses was found (Fig. 4).

In the early postoperative period, the patient developed right-sided pneumonia (Fig. 5). The patient was discharged on the 13th postoperative day. Two months later she was in an excellent overall condition.

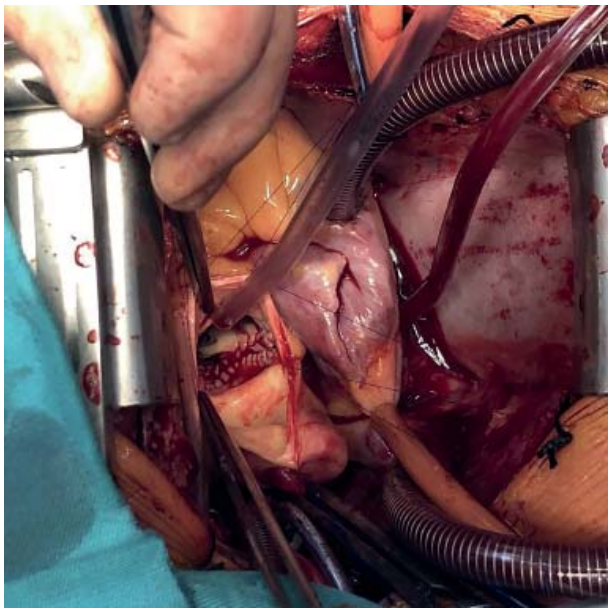


Fig. 3. Case 1. Intraoperative view of the aortic valve

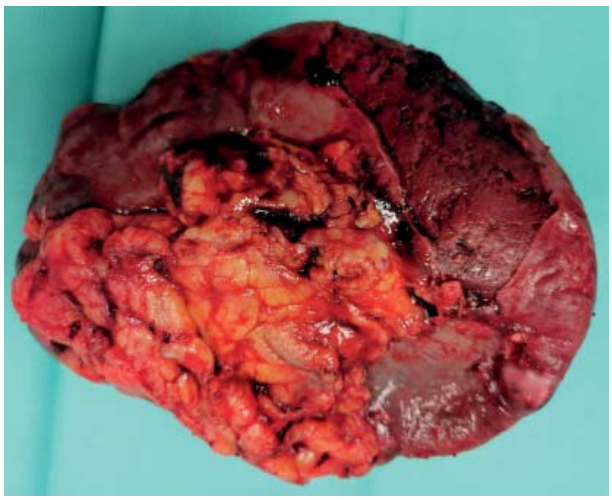


Fig. 4. Case 1. Postoperative specimen of the septic spleen, covered by diaphragmatic peritoneum



Fig. 5. Case 1. CT scan, performed on the 3rd postoperative day, showing a massive right lung infiltration

Case 2

A 48-year-old man presented to the Department of Cardiac Surgery at University Hospital "Acibadem City Clinic" in Sofia with complaints of fatigue and weakness for 6 months. He reported of concomitant and past history of iron-deficiency anemia, mandibular surgery with a prolonged tracheostomy, and right inguinal hernioplasty. He was treated for SARS CoV-2 (positive) pneumonia 6 months ago. He was in impaired overall condition. Tachycardia, hypotonia, and holosystolic murmur 3/6 on the mitral position were present. Mild anemia, CRP 28.2, and hypoalbuminemia were noted on the lab tests. CT scan showed small left pleural and pericardial effusions (Fig. 6). Hypodense abnormalities were revealed in the left ventricle myocardium apically, as well as in the posterior papillary muscles. There was splenomegaly up to 134 x 123 x 62 mm with several homogenous hypodense zones with irregular shape and fluid-equivalent density around 20 HE, without enhancement after intravenous contrast administration, interpreted as infarcts with

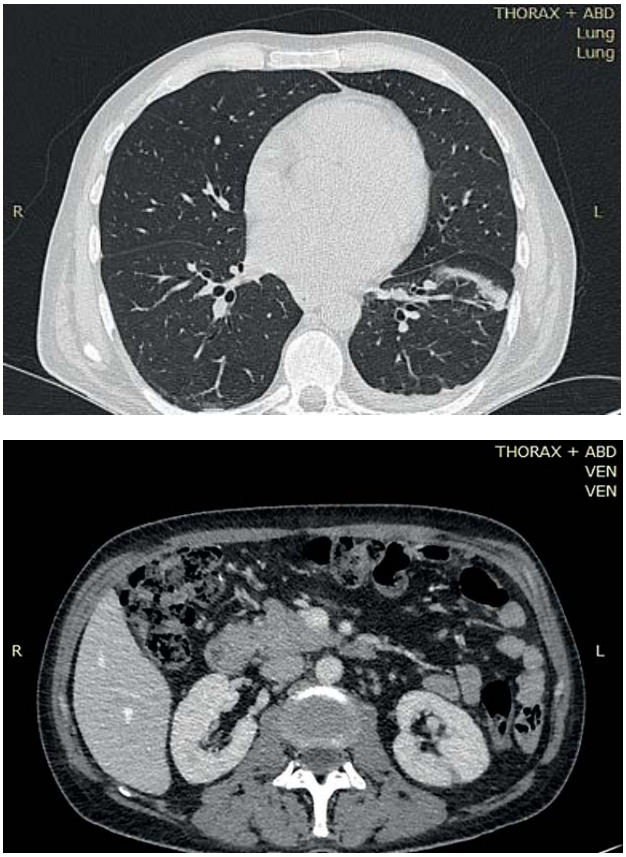


Fig. 6. Case 2. CT scans demonstrated a left pleural effusion and atelectasis, splenomegaly, splenic abscesses, and renal infarcts

septic embologenous genesis. Small fluid perilienally was present. Hypodense small triangle zones in kidneys – single in the lower pole of the right kidney and several in the left kidney, were suspicious for renal infarcts. Echocardiography revealed aortic annulus – 24 mm; sinus Valsava – 30 mm; aortic sinotubular junction – 28 mm; ascending aorta – 29 mm, left atrium 36 mm. IVS/LVPW 13/13 mm; EDD/ESD – 54/43 mm; EDV/ESV – 160/60 ml; EF 61%; aortic valve – aortic insufficiency I grade; mitral valve – annulus – 38 mm, flail of P2 segment of PML (posterior mitral leaflet), from atrial side of the anterior mitral leaflet (AML) additional echogenic structure was visualized with dimensions of 11/7 mm, hardly mobile, the most probably vegetation or disrupted chordae, severe mitral insufficiency with a jet in the whole left atrium; tricuspid valve – 32 mm, tricuspid insufficiency I grade, TAPSE 22 mm; indirectly measured systolic pressure in right ventricle 50 mmHg. Right ventricle RV-D1-30 mm, VCI 18 mm with preserved respiratory collapse; left pleural effusion 300 ml.

We started antibiotic treatment for 14 days, but there was an echographic progression of the disease – EDV/ESV – 198/140 ml, EF 50%, hyperkinetic left ventricle; aortic valve with mobile vegetation of LCC – 12/14 mm, aortic insufficiency 3 grade, severe mitral regurgitation, mild tricuspid regurgitation; left pleural effusion 450 ml. Abdominal echography showed splenomegaly. In the middle third a hypo-/anechoic zone 27/28 mm with a triangle shape, heterogeneous parenchyma, and hypoechoic inclusions, was suspected of splenic infarct.

A mitral valve replacement with mechanical prosthesis SJM Masters 33 mm, aortic valve replacement with mechanical prosthesis Medtronic ATS 24 mm, splenectomy, and tracheostomy was performed. We decided to perform an intraoperative tracheotomy for early ambulation of the patient.

Initially, an upper midline laparotomy was performed. A significantly enlarged and septic spleen with dense adhesions to the diaphragmatic peritoneum was found. Gastrosplenic, splenocolic, lienorenal, and pancreaticosplenic ligaments were transected using Ligasure. The splenic artery and vein were ligated and transected and a splenectomy was performed. The spleen bed was lavaged with diluted Braunol solution. A longitudinal sternotomy and pericardiotomy were performed. Pericardial adhesions were dissected and left ventricle hypertrophy was found. The operation was in extracorporeal circulation settings and a cardioplegic arrest of the heart. The aortic valve during the inspection was with fresh vegetation on the left coronary cusp with dimensions of 10/5 mm and the valve was excised (Fig. 7). Mitral valve was with vegetations of both leaflets and P2 prolapse of PML. The mitral valve was excised

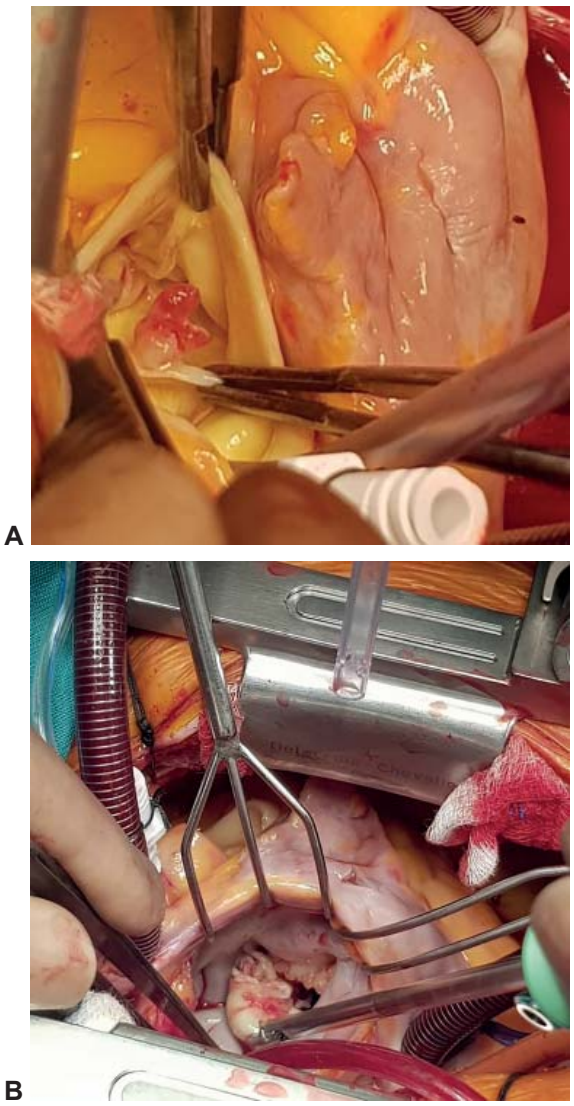


Fig. 7. Case 2. Intraoperative views of aortic and mitral vegetations

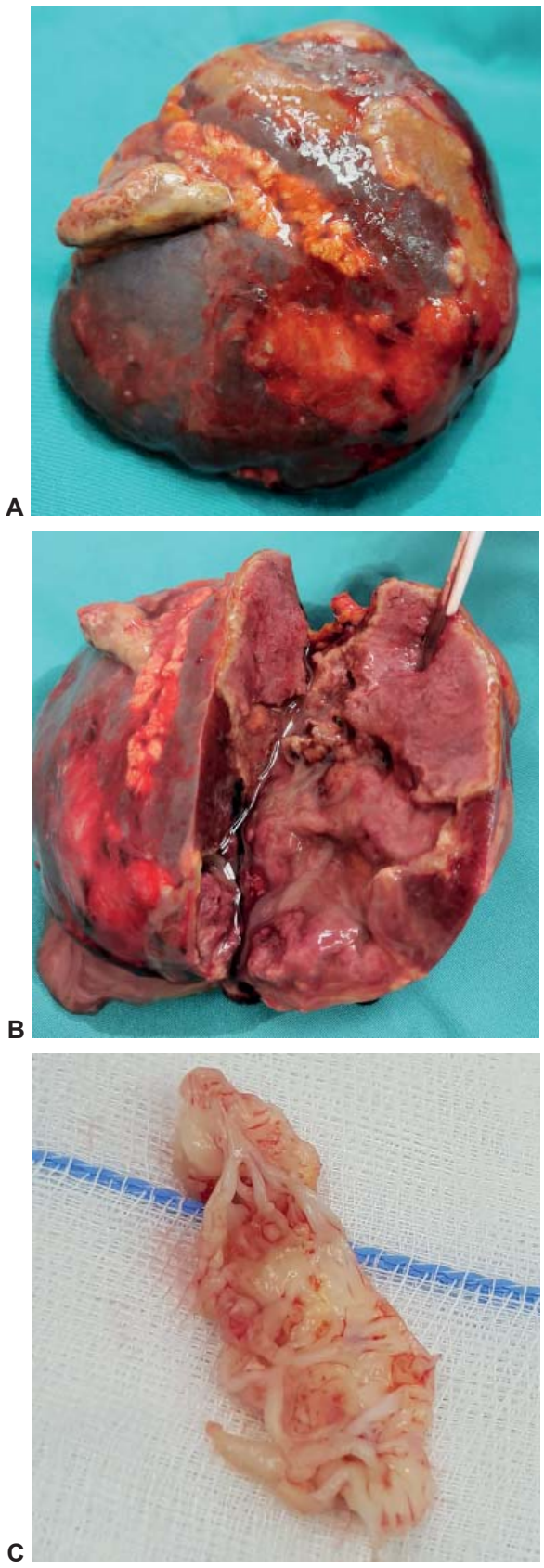


Fig. 8. Case 2. **A, B**: Postoperative specimens of the spleen with abscesses; **C** – infective vegetations

together with a part of the subvalvular apparatus, affected by endocarditis. Mechanical valve prosthesis was implanted with 15 interrupted mattress sutures Ethibond 2/0 with pledgets. The left atrium was closed with two opposite running Prolene sutures 3/0. On the aortic position mechanical valve prosthesis with 15 interrupted mattress sutures Ethibond 2/0 with pledgets was implanted. Closure of the aorta a Modo De Bakey through two opposite running 4/0 Prolene sutures was performed. An atrium-ventricular pacemaker was implanted due to bradycardia. Two drainages (retrosternal and retrocardiac) were positioned. At the end of the operation ARDS developed and due to the expected prolonged postoperative mechanical ventilation, a tracheotomy was performed in the operating room during the same procedure.

A septic spleen with multiple parenchymal and subcapsular abscesses and infective vegetations was found (Fig. 8).

The patient was discharged on the 12th postoperative day. The prolonged postoperative period was due to

postoperative ARDS, so a tracheotomy and invasive mechanical ventilation were needed for 5 days.

DISCUSSION

The incidence of IE is approximately 6/100,000 placing them in fourth place among the most life-threatening infectious diseases after sepsis, pneumonia, and intraabdominal infections [1]. The mean age of patients is 64 years and the most common clinical symptoms and signs are shortness of breath, cardiac murmur, and fever [2]. Our patients were rather young (49 and 48 years, respectively) and with rather atypical clinical manifestations.

The development of IE requires the simultaneous occurrence of several independent factors: alteration of the cardiac valve surface to produce a suitable site for bacterial attachment and colonization; bacteremia with an organism capable of attaching to and colonizing valve tissue; and creation of the infected mass or 'vegetation' by 'burying' of the proliferating organism within a protective matrix of serum molecules (fibrin) and platelets [3]. Staphylococcus aureus, Streptococcus species, and Enterococcus faecalis are the most common microorganisms. Blood cultures are negative in less than 5% of patients with IE [4]. Prior antibiotic therapy, inadequate microbiological techniques, or infection with highly fastidious bacteria or fungi could be associated with culture-negative endocarditis [5].

Risk factors for the development of IE are cardiac (rheumatic heart disease, prosthetic valves, permanent pacemakers, mitral valve prolapse, bicuspid aortic valve) and extracardiac (injection drug use, long-term intravenous lines, healthcare-associated IE, HIV infection, immunosuppressive drugs, chronic kidney disease. It is interesting to note, that our patients didn't have any predisposing factors.

Of note, 20-50% of patients with IE have embolic complications. The biggest risk factor is the size (> 10 mm) and mobility of vegetations [1].

The aortic valve is the most commonly affected (30.6%), alone or combined with mitral and tricuspid valves (35%) [6]. More than 50% of patients with endocarditis will need valve surgery [7]. Homograft implantation for aortic valve treatment is associated with lower complication rates, including a lower incidence of recurrent infection and in the case of the mitral valve, a biological or mechanical prosthetic valve appears appropriate [8].

The conservative treatment strategy is a first-line strategy. Surgical treatment should be considered in patients with signs of heart failure, severe valve dysfunction, prosthetic valve endocarditis, invasion with paravalvular abscess or cardiac fistulas, recurrent systemic embolization, and large mobile vegetations, and persistent sepsis despite adequate antibiotic therapy for more than 5 to 7 days [9].

One study reported 16% in-hospital mortality, and 78.57% overall survival [6]. In our cases, pneumonia and ARDS were significant postoperative complications.

Splenic abscess is a potentially life-threatening clinical entity with an incidence of 0.14% to 0.70% [10]. IE is associated with about 10% of splenic abscesses [11]. Symptoms of splenic abscess usually include persistent or recurrent fever and sepsis, left hypochondrium abdominal pain, abdominal distension, splenomegaly, nausea, vomiting, hiccups, and anorexia.

Infectious emboli, trauma, recent surgery, diabetes, malignancies, and immunosuppression are risk factors. Staphylococcus aureus is the primary organism in 50% of cases with splenic embolization/abscess [12]. Splenic infarction typically appears as a peripheral, well-defined, and wedge-shaped low attenuation area on a CT scan, and the typical appearance of splenic abscess on a CT scan is as a focal low attenuation area with peripheral enhancement [13]. In the presented cases IE was the primary source of septic emboli, which induced splenic infarcts and consequently complicated as abscesses.

The therapeutic strategies include intravenous antibiotics, percutaneous drainage, and splenectomy. Splenic abscess in the IE settings creates treatment challenges. Performing a splenectomy eliminates the potential for prosthetic valve infection, but creates immune-compromised condition and a tendency for bleeding [14]. Some authors advise doing valvular surgery first if there is no heart failure. The third alternative as in the presented cases is the simultaneous procedures.

CONCLUSION

IE, complicated with splenic abscesses is a potentially life-threatening condition. Valvuloplasty and splenectomy is the first treatment line and offers excellent long-term results. The presented cases are rare and successfully operated as a one-stage procedure.

Authors' contribution

AK, SK, MA, and GY performed the experiments. EM and GY conducted the follow-up. All authors read and approved the final version of the manuscript.

Disclosure Summary: *The authors have nothing to disclose.*

REFERENCES

1. Şimşek-Yavuz S, Akar AR, Aydoğdu S et al. Consensus Report on Diagnosis, Treatment and Prevention of Infective Endocarditis by Turkish Society of Cardiovascular Surgery (TSCVS), Turkish Society of Clinical Microbiology and Infectious Diseases (KLIMIK), Turkish Society of Cardiology (TSC), Turkish Society of Nuclear Medicine (TSNM), Turkish Society of Radiology (TSR), Turkish Dental Association (TDA) and Federation of Turkish Pathology Societies (TURKPATH) Cardiovascular System Study Group. Turk Gogus Kalp Dama 2020;28(1):2-42.
2. Babes EE, Lucuta DA, Petchesi CD et al. Clinical features and outcome of infective endocarditis in a university hospital in Romania. Medicina 2021;57:158.
3. Holland TL, Baddour LM, Bayer AS et al. Infective endocarditis. Nat Rev Dis Primers 2016;2:16059.
4. Atilla OD, Temizyurek Z, Kirman E. Infective endocarditis complicated with cerebral and splenic infarction in a hemodialysis patient. World J Emerg Med 2013;4(3):229-31.
5. Medani S, O'Callaghan P. Rare manifestations of infective endocarditis: the long known, never to be forgotten diagnosis. BMJ Case Rep 2015;2015:bcr2015211276.
6. Rendón-Elías FG, Abadiano JADC, Ortega LSA et al. Surgical management of infective endocarditis: 10 years experience. Medicina Universitaria 2012;14(57):196-204.
7. Cuervo G, Escrihuela-Vidal F, Gudiol C, Carratalà J. Current challenges in the management of infective endocarditis. Front Med 2021;8:641243.
8. Antunes MJ. The role of surgery in infective endocarditis revisited. Rev Port Cardiol 2020;39(3):151-3.
9. Pettersson GB, Hussain ST. Current AATS guidelines on surgical treatment of infective endocarditis. Ann Cardiothorac Surg 2019;8(6):630-44.
10. Basukala S, Pathak BD, Mainali G et al. An uncommon case of splenic abscess with gangrenous gas necrosis. Hindawi Case Reports in Surgery, Volume 2022, Article ID 9279418.
11. McOwat L, Wong KF, Varughese GI, Nayak AU. Splenic abscess as a potential initial manifestation of quiescent infective endocarditis in a patient with bronchopneumonia. BMJ Case Rep 2015;2015:bcr2014206794.
12. Takeuchi N, Emori K, Yoshitani M et al. A case of splenic abscess complicated by infective endocarditis. Surgical Infections Case Reports 2016;1.1:139-41.
13. Naito R, Mitani H, Ishiwata S et al. Infective endocarditis complicated with splenic abscess successfully treated with splenectomy followed by double valve replacement. Journal of Cardiology Cases 2010;2:e20-e22.
14. Aljadayel HAB, Kahwaji A, Nataj SZ et al. Simultaneous surgery is effective treatment and could be the management of choice in patients with infective endocarditis and splenic abscess. J Cardio Case Rep 2020;3:1-3.