Vascular Health and Risk Management Dovepress

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CASE REPORT

Fatal Acute Limb Ischemia Due to Catastrophic Late Endograft Infection and Adjacent Arterial Infection After Endovascular Aneurysm Repair – A Case Report

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Introduction: We present a case of late endograft infection that progressed to the left iliac and femoral arteries, leading to left lower extremity gangrene, and the patient's death.

Case: A 65-year-old male with a history of endovascular abdominal aortic aneurysm repair (EVAR) developed left acute limb ischemia (Rutherford category III) and abdominal pain. A CT scan showed significant gas formation around the endograft and complete occlusion of the left distal iliac artery to the femoral arteries. Despite undergoing hip disarticulation and wound care, aortic endograft removal was not possible due to a lack of replacement grafts. Microbiological cultures from arterial pus and urine identified multiple antibiotic-resistant extended-spectrum beta-lactamases (ESBL) producing Escherichia coli. Histopathological analysis of the common femoral artery specimen indicated chronic medium-sized arteritis characterized by endothelial erosion, fibrotic myocytes in the tunica media, and fibrosis of the adventitial layer with inflammatory cell infiltration. The patient succumbed in the ICU 6 days later due to uncontrolled sepsis.

Discussion: Although the incidence of endograft infection after EVAR is low (20–75% morbidity and mortality), it poses significant risks. Sources are often hematogenous, stemming from urinary or respiratory tract infections, and infections extending to subsequent arteries are very rare; they could cause chronic arterial inflammation and, in the long term, may lead to thrombosis and limb ischemia. This case highlights a low-grade infection that emerged 3 months post-procedure. Diagnosis typically involves CT angiography to detect periaortic gas or fluid. Management of high-grade infections necessitates complete endograft removal and graft replacement with infection-resistant options.

Conclusion: Endograft infections after EVAR, while rare, can have severe outcomes. Early diagnosis based on symptoms and CT-Scan. In high-grade infections, endograft removal is the gold-standard therapy, with ongoing follow-up post-EVAR being essential for prevention.

Plain Language Summary:

- Late stent-graft infection after minimal invasive surgery for aneurysm, which causes acute limb ischemia, is very rare and can spread to subsequent arteries (contiguous arterial infection).
- Infection of the stent-graft may cause blood vessel obstruction originated from infection.
- Marfen et al reported that the infected artery is a true vasculitis, with pathological findings showing chronic arteritis with antibiotic resistant Escherichia coli as the cause of stent-graft infection being very rare, and reports are scarce.

The stent-graft in a severe stent-graft infection MUST be removed to control the infection.

Keywords: arteritis, case report, endograft infection, ESBL, graft removal

Received: 13 July 2024
Accepted: 16 October 2024
Published: 1 November 2024

Introduction

With increasingly affordable endograft costs, endovascular abdominal aortic aneurysm repair (EVAR) is performed more favorably worldwide than open abdominal aortic surgical repair (OSR) because of its lower morbidity and initial mortality rate.[1,2] Even though it is considered a safer procedure than OSR, EVAR still poses the risk of various complications, with rates ranging from 16% to 30% for abdominal EVAR and up to 38% for thoracic EVAR. Major complications include endoleaks, device-related issues like graft migration or limb kinking, and systemic problems such as end-organ ischemia and cardiovascular events. Secondary interventions are required in 19–24% of cases to address these issues.[1,3] Endograft infection is a rare complication with an incidence range of 0.2–1%, and some centers have reported as high as 5%. It is the most feared complication, with a combined morbidity and mortality rate around 20–75%.[1,2,4,5] Spreading of the infection to the adjacent arteries from an endograft infection followed by acute limb ischemia is an even rarer complication that increases morbidity and mortality. We report a very rare case of severe catastrophic endograft and arterial infection 3 years after EVAR, causing fatal acute limb ischemia due to arterial thromboembolism. This case report is in accordance with CARE Case Report Guidelines.

Narrative

Patient Presentation

A 65-year-old man was admitted to the emergency department of our hospital, a top referral hospital in West Java Province, Indonesia, with severe pain and bluish spots on his left lower extremity, since 5 days before. His symptoms suddenly had a history of trauma, which was preceded by abdominal pain for 5 months, which worsened over time.

This patient was known to have undergone the EVAR procedure (Medtronic EndurantTM) in April 2020, 3 years and 9 months ago during the COVID-19 pandemic in our hospital, but had never shown up for routine follow-up. The EVAR procedure was an aorto-bi-iliac endograft because of its large-diameter (10.6 cm) infrarenal abdominal aortic aneurysm with a thick thrombus. The main body (ETBF2516C166EE) and one extension limb (ETLW1616C156EE) were inserted via the right femoral approach, and two left extension limbs (ETLW1620C156EE and ETLW2020C82EE) were inserted via the left femoral approach using the open surgery/cutdown method (Figure 1) and performed in the cardiology suite cathlab. Despite the patient suffering from chronic obstructive pulmonary disease (COPD), arterial hypertension, and obesity, the procedure was uneventful, with no endoleaks or palpable pedal pulses, and he was discharged from the hospital on the third day with oral antibiotics (ciprofloxacin 2 × 500 mg) and 80 mg acetylsalicylic acid once daily. He only showed up once a week after the procedure to check the cutdown wound and then never showed up for further follow-up.

Diagnostic Workup

On admission, he was alert with moderate abdominal and left hip pain and skin mottling over his left limb from the foot to the inguinal region. His heart rate was approximately 120×/min with a temperature of approximately 38°C. We found a non-pulsating mass on his abdomen, with normal bowel sounds and no signs of peritonitis. His left limb was cold with no sensory or motor function, no capillary refill time, and no pulse in any of the left lower extremity arteries, which was consistent with Rutherford's classification of acute limb ischemia category III (unsalvageable). The patient's right lower extremity function was normal. Initial laboratory findings revealed signs of sepsis, with a hemoglobin (Hb) level of 11.5 g/dL, white blood cell count (WBC) of 26,430/μL, and platelet count (Plt) of 177,000/μL. The Random blood glucose level was 172 mg/dL, creatinine level was 0.75 mg/dL, with a low albumin level of 2.61 g/dL. The procalcitonin level was 73.57 ng/mL (<0.25 ng/mL) and C-reactive protein was 21.6 mg/dL (<1 mg/dL). The patient was immediately rehydrated and administered heparin IV, meropenem 3 × 1 g IV and vancomycin 2 × 1 g IV. Computed tomography (CT) angiography of the abdomen revealed a large aneurysm sac filled with an old thrombus, with intravenous contrast inside the intact graft with no sign of endoleaks; there was a massive distribution of gas surrounding the endograft, and no sign of thrombosis inside the main body or both extension limbs of the endograft. The distal part of the left iliac artery was not visible, most likely because of thrombosis.

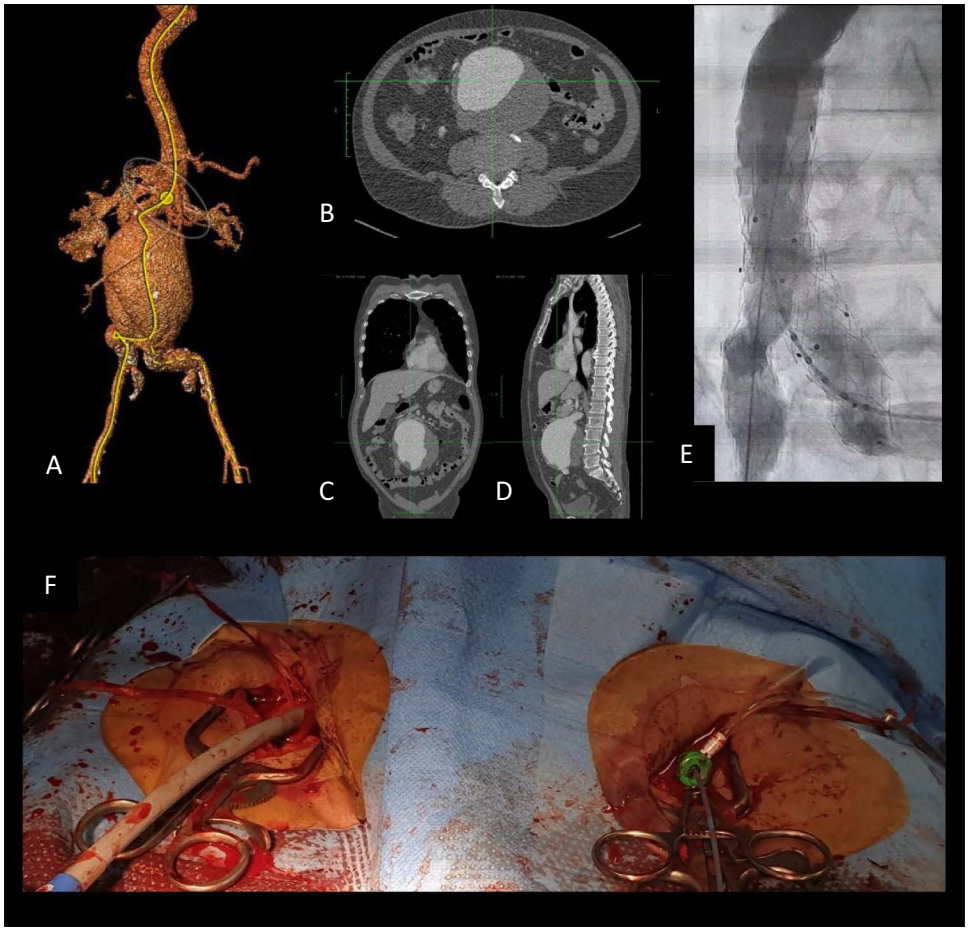

Figure 1 Computed tomography angiography (CTA) before EVAR procedure, showing a very large (10.6cm) infrarenal abdominal aortic aneurysm (**A-D**); Post EVAR final angiogram, showing result with no endoleak (**E**); and the bilateral cutdown procedure (**F**).

Management

We planned an immediate laparotomy to remove the whole infected graft and ligate the aorta infrarenally, followed by a right axillofemoral extra-anatomical bypass and hip-disarticulation amputation for infection control. Unfortunately, the desired prosthesis graft was not available at that time, and ordering a rarely used prosthesis graft took approximately a month; therefore, we planned to perform only hip disarticulation to control the infection. We were only able to perform hip disarticulation on the eighth day after admission, waiting for patient and family consent (at first, the patient and family refused amputation). Intraoperatively, we attempted to identify the left common femoral artery first, but it was necrotic and severely infected. We proceeded with hip disarticulation and found pus and smelly odor originating from the inside of the iliac artery, with necrotic tissue surrounding it (Figure 2). We managed to perform hip disarticulation and kept the wound open (open wound care with normal saline-impregnated gauze, changed twice daily).

Outcome

On post-operative day 1 (POD 1) in intensive care unit (ICU), the patient was on norepinephrine 3.5 mcg/min to maintain his blood pressure. The lab was pancytopenia, with a Hb level of 6.8 g/dL, WBC of 3890/µL, and platelet count (Plt) of 105,000/µL. The patient's albumin level decreased to 1.32 g/dL. The patient was transfused with packed red blood cells and albumin. The antibiotics administered remained the same until microbiological culture. On POD 3, the Hb increased to 9.3 g/dL, Plt to 168,000/µL, and the albumin level to 1.75g/dL; but the WBC increased to 15,800/µL (Table 1). A chest radiograph revealed bronchopneumonia. However, the amputation wound started to improve with granulation, and no pus was found (Figure 3). Unfortunately, the patient died on POD 6 due to uncontrolled sepsis, hypoalbuminemia, and bronchopneumonia.

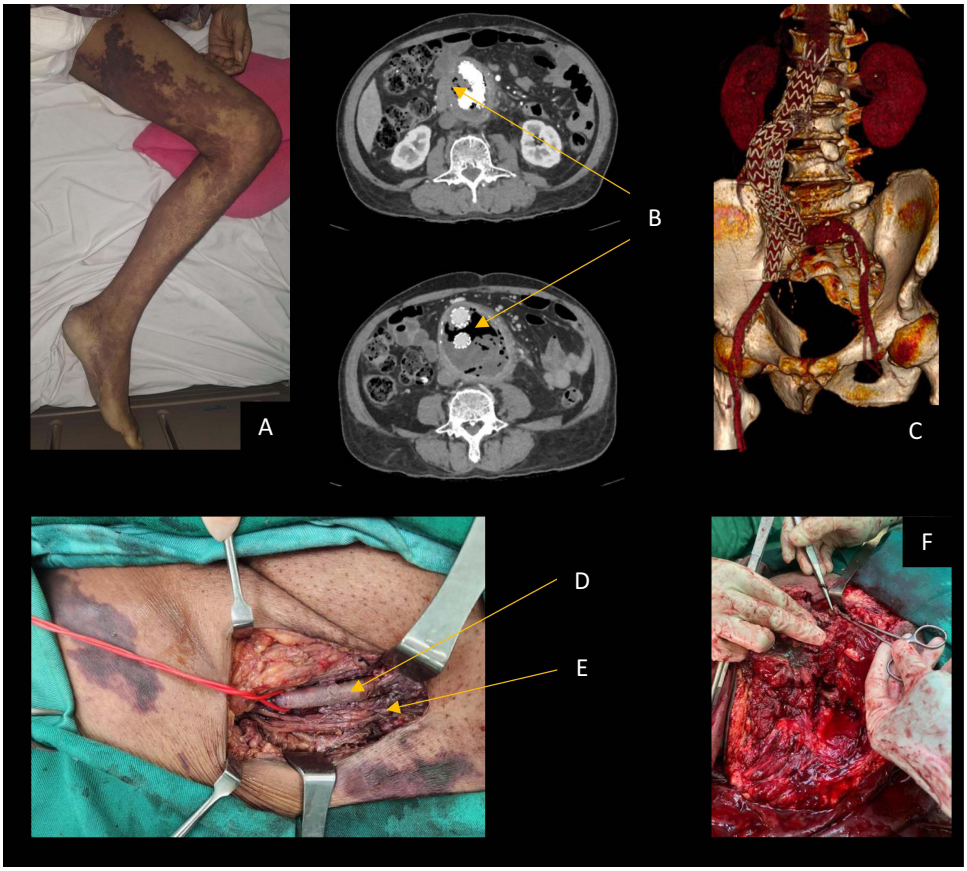

Figure 2 Skin mottling of the left lower-extremity (**A**); CTA of abdominal aorta, showing gas distribution surrounding the endograft (**B**); thrombosis of distal left external iliac artery, with intact endograft with no endoleak (**C**); infected and necrosis of common femoral artery and surrounding tissue (**D-E**); infected and necrosis of external iliac artery (**F**) with necrotic of adjacent tissue.

Histopathological examination of the infected common femoral artery specimen revealed chronic medium-sized arteritis with endothelial erosion, fibrotic myocytes in the tunica media, and fibrosis of the adventitial layer with inflammatory cell infiltration.

Table 1 The Laboratory Result in ICU

	Admission	POD 1	POD 2	POD 3	POD 5
Hb (g/dL, 13–17)	11.5	6.8	7.2	9.3	7.3
Platelets (/µL, 150.00–450.000)	177.000	105.000	143.000	168.000	176.000
WBC (/µL, 4.000–11.000)	26.430	3.890	13.800	15.080	17.160
Hematocrit (%, 42–54)	35.9	21	21.7	28	22.4
Random Blood Glucose (mg/dL, < 200)	172	116	121	–	–
BUN (mg/dL, 20–40)	37.7	15.5	21	–	16.6
Creatinine (mg/dL, 0.6–1.2)	0.75	0.44	0.44	–	0.43
Albumin (g/dL, 3.8–5.1)	2.61	1.32	1.97	1.75	–
CRP (mg/dL, < 1)	21.6	–	–	–	–
Procalcitonin (ng/mL, < 0.25)	73.57	–	–	–	–

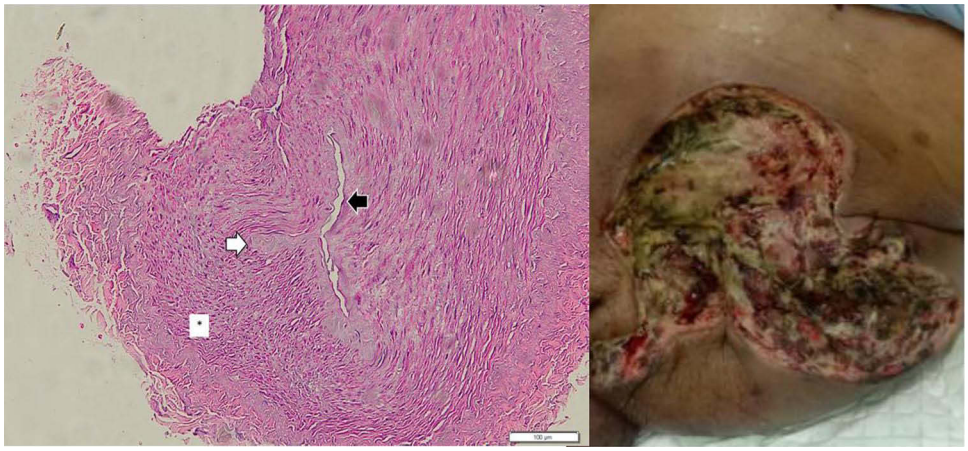

Figure 3 Right: wound condition on POD 5; the wound granulation started to appeared with slough but no pus. Left: Hematoxylin-Eosin stain of common femoral artery specimen (100X): Chronic medium-sized arteritis; Black Arrow: Narrowed lumen, tunica intima with endothelial erosion. White Arrow: Fibrotic myocytes in tunica media. Asterisk: Fibrotic tunica adventitia with inflammatory cells infiltration.

Microbiological culture of the iliac artery pus and urine showed extended-spectrum beta-lactamases (ESBL) producing Escherichia coli, which is resistant to multiple antibiotics, and sensitive only to gentamicin, amikacin, imipenem, and meropenem (Table 2).

Patient Perspective

Patient's daughter gives the consent for the publication of this case. We confirm that informed consent was obtained from the patient's daughter, which explicitly included permission for the publication of the case details and associated images. She is very thankful for all the doctors involved in giving treatment for her father. She hopes all surgeons around the

Table 2 Culture and Sensitivity Results; Samples Were Taken During Surgery

Sample: Pus		**Sample**	
Organism: Escherichia coli		**Organism**	
Name of Antibiotics	**Interpretation**	**Name of Antibiotics**	**Interpretation**
Group A (Access)		**Group A (Access)**	
• Ampicillin	R	• Ampicillin	R
• Amoxycillin-Clavulanate	S	• Amoxycillin-Clavulanate	R
• Gentamicin	S	• Gentamicin	S
• Chloramphenicol	S	• Chloramphenicol	R
• Tetracycline	S	• Tetracycline	R
• Trimethoprim-Sulfamethoxazole	S	• Trimethoprim-Sulfamethoxazole	R
Group B (Watch)		**Group B (Watch)**	
• Ciprofloxacin	R	• Ciprofloxacin	R
• Levofloxacin	R	• Levofloxacin	R
• Ceftazidime	R	• Ceftazidime	R
• Cefotaxime	R	• Cefotaxime	R
• Moxifloxacin	R	• Moxifloxacin	R
• Ampicillin-Sulbactam	R	Ampicillin-Sulbactam	R

(*Continued*)

Table 2 (Continued).

Sample: Pus		**Sample**	
Organism: Escherichia coli		**Organism**	
Name of Antibiotics	**Interpretation**	**Name of Antibiotics**	**Interpretation**
Group C (Reserved)		**Group C (Reserved)**	
• Amikacin	S	• Amikacin	S
• Cefazolin	R	• Cefazolin	R
• Cefepime	R	• Cefepime	R
• Imipenem	S	• Imipenem	S
• Meropenem	S	• Meropenem	S
• Piperacillin-Tazobactam	S	• Piperacillin-Tazobactam	R
• Aztreonam	R	Aztreonam	R
Impression: Extended Spectrum Beta Lactamase (ESBL)		Impression: Extended Spectrum Beta Lactamase (ESBL)	

Notes: S, Sensitive; R, Resistant.

world are learning from this case. She also stated that hopefully in the future nobody needs to go through what her father experienced. Also, permission for publication has been given by Dr Hasan Sadikin Hospital Ethics Committee.

Discussion

Endograft infection is a rare but serious complication. Unlike open surgery, EVAR is considered to have a lower graft infection rate owing to its relatively closed procedure during endograft delivery.[1,6] The risk factors for endograft infection are multifactorial, and the likelihood of endograft infection increases in patients with altered host defenses such as immune disorders, corticosteroid administration, malnutrition, and diabetes. External factors may have also contributed to this finding. Factors such as infection at remote or adjacent sites, emergency procedures, groin incision, breach in aseptic technique, endograft implantation in the radiology suite, or prolonged operation time are considered possible contributors to graft infection.[7,8]

The onset of infection varies, with some reporting between 1 and 128 months, and an average time of occurrence of 22–25 months. It is categorized as early (<3 months) or late (>3 months). Others have reported that aortic graft infections have occurred as late as >4 months with a mean time of more than 40 months.[6,7,9,10] Late endograft infection is mostly low-grade in presentation, with nonspecific symptoms such as weakness, weight loss, and malaise, and early endograft infection usually presents with high-grade signs of infection such as fever, sepsis, abdominal pain, lumbar pain, hemorrhagic shock, and graft thrombosis.[1,2,8] CT angiography provides a sensitivity of 94% and specificity of 85–100% for a severe infection,[2] and it is recommended as a first line diagnostic modality.[8] The radiologic signs of endograft infection vary, such as ectopic or periaortic gas, perigraft fluid, abscess, soft tissue enhancement, pseudoaneurysm, or focal bowel thickening,[8,11] which were very obvious in our case (Figure 2).

Endograft infections causing adjacent arterial infections and thrombosis are rarely observed, and reports in the literature are scarce. A literature search through PubMed® and Cochrane Library did not reveal many cases, except for infected prosthetic grafts, pseudoaneurysms, or some old sources. Arteries are relatively resistant to infection because of their ability to bind to and cause infection of the endothelial surface.[12] Arterial endothelial cells act as a vascular barrier to infection and injury;[13] thus, if bacteria finally break this barrier and cause endothelial cell inflammation, it may induce autophagy-mediated vascular leakage and cytokine overproduction, leading to dreadful consequences.[12,14] Diagnosis of arterial infection is difficult and often very late until it has complications such as arterial rupture or another catastrophic event.[12] Spreading of the infection to the artery from the adjacent abscess (infection source) is known as a *contiguous arterial infection*, and emboli from an infected vascular prosthesis that lodge in the peripheral artery are known as septic (infected) arterial emboli, a classification introduced by Wilson et al in 1978.[15]

This is the first case of endograft infection reported since the start of the routine EVAR procedure in 2019. To date, we have performed 34 EVAR cases, and femoral access was the cutdown method in the Cardiology Suite Cathlab. The infection, in our case, started from an aortic endograft that spread to the arteries of the left lower extremity. Since the patient developed symptoms approximately 5 months ago and no treatment was administered, the time duration was sufficient for such a disastrous infection. The left acute limb ischemia was possibly caused by an embolus that developed inside the infected aortic endograft.

The best treatment for severe endograft infection is the complete removal of the infected endograft and debridement of the surgical field, followed by revascularization and long-term antibiotic treatment. The method of revascularization varies from extra-anatomical bypass (axillo-bifemoral) and cryopreserved allograft to impregnated prosthetic graft,[1,2,8,16] which was not available in our hospital at that time.

The source of late endograft infection is the hematogenous spread of bacteremia originating from the urinary or respiratory tract.[8,17] Urinary tract infections can be one of the causes of mycotic aneurysm formation.[18] Other possible sources of infection are the transmission of bacteria from the gut due to the connection between the graft and the gut or groin, or the graft can erode to the 3rd or 4th portion of the duodenum.[11,16]

The finding of extended-spectrum beta-lactamases producing Escherichia coli (ESBL-EC) in iliac artery swabs and urine cultures indicated that the possible source of infection in this case was a hematogenous spread of a urinary tract infection, even though this patient had no previous symptoms of urinary tract infection. This ESBL-EC is a type of pathogen that is often found in around 70–90% of community urinary tract infections and is resistant to beta-lactam antibiotics, penicillins, and third-generation cephalosporins.[19] This bacteria, which probably migrates from the anus to the meatus of the urinary tract, has become a serious concern for nosocomial infections around the world, including Indonesia, due to its high level of antimicrobial multidrug-resistance.[20,21] In some regions of Indonesia, the prevalence of ESBL-EC infection could be as high as 62.2% and 85%.[21,22] The fatality rate of ESBL-EC infection alone is high, approximately 12.8% in 30 days and even higher (40.5%) in bacteremic pneumonia.[23,24]

The histopathological findings of chronic medium-sized arteritis suggested that the involved arteries had been infected for a long period of time. The mechanism by which these arteries can be infected may involve direct pathogen invasion of the endothelial cells due to the extension of focal infection affecting arteries or as a result of a blood-borne septic embolization, which is characterized by the accumulation of smooth muscle cells, expression of reactive oxygen species, cytokines, chemokines, cellular adhesion molecules, endothelial dysfunction, and damage to the vessel wall.[25,26] In our case, damage to the vessel wall was demonstrated by the presence of endothelial cell erosion, myocyte fibrosis in the tunica media, and fibrosis of the adventitia layer. Thus, this sign of arterial wall inflammation caused by infection is considered true vasculitis, which makes this case rare.[26]

Our limitation was that not all types of prosthesis graft replacements for revascularization were available at that time. Instead of just amputation and open wound care, a complete endograft removal will control the septic condition more effectively.

This is the main reason why we kept the endograft in situ, which became a dilemma because endograft removal followed by ligation of the infrarenal aorta without revascularization would cause more catastrophic damage due to bilateral lower extremity gangrene. Another limitation is that this case could have been prevented earlier. We understand that our patient did not come for follow-up, possibly because of the COVID-19 pandemic, when most hospitals in our country were overwhelmed by COVID-19 patients. However, we must keep in contact with and remind the patient for follow-up despite a pandemic situation. Finally, we did not check the COVID-19 vaccine status or perform nasal swabs upon admission. Although COVID-19 infection is no longer an issue, there are many reports regarding the incidence of vasculitis and thrombosis due to COVID-19 infection.[25,26]

Conclusion

Aortic endograft infection is a very serious complication with high mortality rate. The infection can rapidly spread to the contiguous arteries, causing arterial necrosis leading to thrombosis, or occluded by septic embolism from the infected graft. In severe condition, the best treatment should be removal of the entire endograft followed by revascularization

either by impregnated prosthesis graft replacement or extra-anatomical bypass. In our case, patient mortality is due to uncontrolled sepsis, most likely because the infected endograft remain in situ, as well as very late action due to delayed in patient and family consent. This case emphasize, that continuous follow-up after EVAR is mandatory, and resources availability, ie certain type of prosthesis vascular graft is essential in centers performing EVAR, as preparation if another case of aortic graft infection arises.

Acknowledgments

We thank our vascular and endovascular surgeons, Indra Prasetya Yarman and Hafidh Seno Radi Utomo, Hendro, Jonathan Mark Yobel and all the residents involved in this study.

Disclosure

The authors report no conflicts of interest in this work.

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