ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2021 September 26; 9(27): 7963-8279

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

Contents

Thrice Monthly Volume 9 Number 27 September 26, 2021

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Contents

World Journal of Clinical Cases

Thrice Monthly Volume 9 Number 27 September 26, 2021

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 9 Number 27 September 26, 2021

Contents

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Production Editor: *Ji-Hong Liu*; **Production Department Director:** *Xiang Li*; **Editorial Office Director:** *Jin-Lei Wang*.

NAME OF JOURNAL
World Journal of Clinical Cases

ISSN
ISSN 2307-8960 (online)

LAUNCH DATE
April 16, 2013

FREQUENCY
Thrice Monthly

EDITORS-IN-CHIEF
Dennis A Bloomfield, Sandro Vento, Bao-Gan Peng

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/2307-8960/editorialboard.htm

PUBLICATION DATE
September 26, 2021

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World J Clin Cases 2021 September 26; 9(27): 8232-8241

DOI: 10.12998/wjcc.v9.i27.8232

ISSN 2307-8960 (online)

CASE REPORT

Acute lower extremity arterial thrombosis after intraocular foreign body removal under general anesthesia: A case report and review of literature

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Author contributions: Jeon S and Hong JM designed the study; Jeon S, Hong JM and Ri HS drafted the manuscript; Lee H, Kim Y and Lee JJ collected data and pictures; Jeon S, Hong JM, Lee HJ, Kim E and Ri HS reviewed the literature and edited the manuscript; all authors agreed to be accountable for all aspects of the work; all authors issued final approval for the version to be submitted.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement: The authors declare that they have no conflicts of interest.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and

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Abstract

BACKGROUND

Surgery, which is a major risk factor for venous thrombosis, has rarely been considered a risk factor for arterial thrombosis. Recent studies have suggested that venous and arterial thromboses share common risk factors and have a bidirectional relationship. Accordingly, there is a growing interest in the risk of arterial thrombosis after surgery. We report a case of acute bilateral lower extremity arterial thromboses that developed after a prolonged surgery.

CASE SUMMARY

A 59-year-old man was hospitalized for intraocular foreign body removal surgery. He was a heavy-drinking smoker and had untreated hypertension and varicose veins in both legs. The operation was unexpectedly prolonged, lasting 4 h and 45 min. Immediately after emergence from general anesthesia, the patient complained of extreme pain in both legs. After the surgical drape was removed, cyanosis was evident in both feet of the patient. The pulse was not palpable, and continuous-wave Doppler signals were inaudible in the bilateral dorsalis pedis and posterior tibial arteries. Computed tomography angiography confirmed acute bilateral thrombotic occlusion of the popliteal arteries, proximal anterior tibial

revised according to the CARE Checklist (2016).

Manuscript source: Unsolicited manuscript

Specialty type: Medicine, research and experimental

Country/Territory of origin: South Korea

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B
Grade C (Good): C
Grade D (Fair): 0
Grade E (Poor): 0

Received: May 4, 2021
Peer-review started: May 4, 2021
First decision: June 24, 2021
Revised: July 8, 2021
Accepted: August 11, 2021
Article in press: August 11, 2021
Published online: September 26, 2021

P-Reviewer: Chauhan S, Chilimuri S
S-Editor: Gao CC
L-Editor: A
P-Editor: Yuan YY

arteries, and tibioperoneal trunks. Arterial pulse returned in both lower limbs after 6 h of heparin initiation. The patient was discharged on postoperative day 26 without any sequelae.

CONCLUSION

Acute lower extremity arterial thrombosis can occur after surgery. Anesthesiologists should pay particular attention to patients with risk factors for thrombosis.

Key Words: Thromboembolism; Thrombosis; Arterial thrombosis; Arterial occlusive diseases; Peripheral occlusive artery disease; Case report

Core Tip: The conventional literature emphasizes that surgery is a major risk factor for venous thrombosis rather than arterial thrombosis. However, recent studies have suggested that these two types of thromboses are closely related and share common risk factors. Accordingly, there has been a growing interest in the increased postoperative risk of arterial thrombosis. We report the case of a patient with multiple risk factors, who developed acute bilateral arterial thromboses of the lower limbs after an unexpectedly prolonged surgery. Although postoperative arterial thrombosis of the lower extremity is rare, anesthesiologists should pay particular attention to patients with risk factors for thrombosis.

Citation: Jeon S, Hong JM, Lee HJ, Kim E, Lee H, Kim Y, Ri HS, Lee JJ. Acute lower extremity arterial thrombosis after intraocular foreign body removal under general anesthesia: A case report and review of literature. *World J Clin Cases* 2021; 9(27): 8232-8241
URL: https://www.wjgnet.com/2307-8960/full/v9/i27/8232.htm
DOI: https://dx.doi.org/10.12998/wjcc.v9.i27.8232

INTRODUCTION

Thrombosis refers to the formation of a blood clot, which partially or fully blocks the blood flow, in a blood vessel[1]. The complications of thrombosis vary depending on the type and anatomical location of the blood vessel in which the clot is located. While venous thrombosis causes congestion in the upstream area, arterial thrombosis causes ischemia in the downstream area[2,3]. Traditionally, arterial thrombosis and venous thrombosis have been considered as distinct diseases, with different risk factors, underlying mechanisms, and treatments[4,5]. The well-established risk factors for venous thrombosis include trauma, surgery, and cancer, while the factors leading to arterial thrombosis include smoking, hypertension, and dyslipidemia[4].

Due to immobility and systemic hypercoagulability, surgery is a risk factor for venous thrombosis[4]. To reduce this preventable complication during surgery, patients scheduled for high-risk surgical procedures, such as major orthopedic, general, gynecological, urological, vascular, and neurological surgeries, are recommended to undergo thromboprophylaxis after a risk and benefit assessment[6,7]. In contrast, until recently, surgery has rarely been considered a risk factor for arterial thrombosis[4]. Recent studies have suggested that venous and arterial thromboses share many common risk factors and have a bidirectional relationship[4,5]. Therefore, there is a growing interest in the risk of arterial thrombosis after surgery. Herein, we report a case of acute bilateral lower extremity arterial thromboses that developed after intraocular foreign body removal under general anesthesia. A relevant literature review was also conducted.

CASE PRESENTATION

Chief complaints

A 59-year-old male patient (163 cm, 70 kg) complained of severe pain in both legs immediately after intraocular foreign body removal under general anesthesia.

History of present illness

After an accident at a construction site, in which a 3 mm iron particle entered the patient's left eye, the patient was hospitalized for foreign body removal surgery. Physical examination immediately after admission revealed no abnormal findings, except for the left eye injury. The patient did not complain of any discomfort in either leg. Preoperative electrocardiogram (ECG), chest radiography, and laboratory findings were unremarkable (Table 1). In the operating room, standard monitoring (ECG, pulse oximetry, noninvasive blood pressure, end-tidal CO_2 ($EtCO_2$), and esophageal stethoscope temperature measurement) was performed; the patient's initial (pre-induction) heart rate (HR), oxygen saturation (SpO_2), systolic blood pressure (SBP), diastolic blood pressure (DBP), $EtCO_2$, and respiratory rate (RR) were 60 beats/min, 100%, 179 mmHg, 78 mmHg, 30 mmHg, and 20 breaths/min, respectively. The vital signs and drugs used during the surgery are shown in Figure 1. The surgery lasted 4 h and 45 min and included phacoemulsification, vitrectomy, intraocular foreign body removal, endolaser photocoagulation, and fluid-air exchange. During surgery, the patient was in a supine position without restraints, and graduated compression stockings or intermittent pneumatic compression devices were not used. Immediately after emergence from general anesthesia, the patient complained of extreme pain in both legs.

History of past illness

Although the patient was diagnosed with hypertension > 10 years earlier (baseline SBP/DBP, 160-180/100-78 mmHg), he had voluntarily not taken antihypertensive medication for years. He also had varicose veins in both legs.

Personal and family history

The patient was a heavy-drinking smoker[8]; he would drink more than 50 g of alcohol and smoke 18 cigarettes per day. The patient had no family history of hypercoagulable disorders.

Physical examination

After the surgical drape was removed, cyanosis was evident in both feet of the patient. The pulse was not palpable in the bilateral dorsalis pedis and posterior tibial arteries. For further evaluation and treatment, the patient was referred for a consultation to the vascular surgery department of our hospital. A hand-held continuous-wave Doppler examination revealed that Doppler signals of the bilateral dorsalis pedis and posterior tibial arteries were absent (*i.e.*, inaudible).

Laboratory examinations

Immediately after the surgery, a series of laboratory tests were performed. Routine postoperative laboratory test findings are presented in Table 1. Except for a decrease in protein S activity [22% (reference range[9], 65-160)] and an increase in fibrinogen degradation products [146.3 μg/mL (0.0-5.0)] and the D-dimer level [35.2 μg/mL (0.0-0.5)], the results of the hypercoagulability work-up were not specific [protein C activity, 102.6% (73.0-142.0); fibrinogen, 277.2% (170.0-380.0); and antithrombin III activity, 91.7% (80.0-120.0)]; factor V Leiden, lupus anticoagulant, anti-cardiolipin immunoglobulin (Ig) M, anti-cardiolipin IgG, anti-cardiolipin IgA, anti-phospholipid IgG, and prothrombin G20210A mutation findings were all negative. Blood cultures were also negative. Lipid profile was as follows: Low-density lipoprotein cholesterol level, 108 mg/dL (< 160); high-density lipoprotein cholesterol level, 69.0 mg/dL (35.0-72.0); and triglyceride level, 64 mg/dL (58-250 mg/dL). Cardiac markers were as follows: myoglobin level, 1192.8 ng/mL (15.2-91.2); creatine kinase (CK) level, 7081 U/L (5-217); CK-myocardial band level, 89.06 ng/mL (0.5-5.0); troponin I level, 0.02 ng/mL (0-0.05); and brain natriuretic peptide level, 28 pg/mL (0-100). Urinalysis results were as follows: color, yellow; clarity, clear; pH, 7.0 (5.0-6.5); urine occult blood, trace; urine RBC, 11-15/high power field (HPF; 0-2); urine WBC, 0-2 (0-2); urine glucose, negative. HbA1c and blood glucose levels were 5.9% and 99 mg/dL, respectively.

Table 1 Laboratory data

	Preoperative	After surgery (POD 0)	After hematochezia (POD 1)	POD 2	Reference range
Complete blood count					
WBC (10^3/μL)	6.80	13.11	10.00	8.4	4.0-11.0
RBC (10^6/μL)	4.44	4.6	3.89	3.25	4.5-6.0
Hb (g/dL)	14.7	15.1	12.6	10.4	14.0-17.0
Hct (%)	42.3	44.0	36.6	31.3	42.0-52.0
Plt (10^3/μL)	205	176	160	109	140-400
PCT (%)	0.2	0.18	0.16	0.11	
MPV (fL)	9.6	10.0	9.8	9.9	7-11
PDW (fL)	10.9	110.	10.2	10.3	11-16
Coagulation profile					
PT-INR	1.07		1.03	1.03	0.88-1.12
aPTT (s)	33.2		24.7	32.1	27-42
Liver and kidney function tests					
AST (U/L)	20		136	114	10-40
ALT (U/L)	18		62	59	6-40
ALP (U/L)	107		75	64	40-129
T bil (mg/dL)	0.75		0.51	0.88	0.1-1.2
Albumin (g/dL)	4.9		3.7	3.7	3.3-5.2
T chol (mg/dL)	213		169	156	175-210
BUN (mg/dL)	12.5		32.9	20.1	6-26
Creatinine (mg/dL)	0.76		0.98	0.77	0.4-1.2
GFR (mL/min/1.73 m^2)	105		78.3	103.4	
Uric acid (mg/dL)	4.1		6.1	3.3	2.5-8.0
Electrolyte					
Sodium (mmol/L)	142.2	144.2	139.4	139.7	138-148
Potassium (mmol/L)	4.08	3.70	4.25	4.01	3.5-5.3
Calcium (mg/dL)	9.2		7.7	7.8	8.5-10.3
Phosphorus (mg/dL)	3.4		3.2	2.3	2.0-4.6
Anion gap	11.7	20.4	14.3	10.4	
Myoglobin and muscle enzyme					
Myoglobin (ng/mL)		1192.8	295.0	96.7	15.2-91.2
Creatine kinase (U/L)		7081.0	6198.6	4697	5-217
CK-MB (ng/mL)		89.06	55.02		0.5-5.0

POD: Postoperative day; WBC: White blood cell; RBC: Red blood cell; Hb: Hemoglobin; Hct: Hematocrit; Plt: Platelet; PCT: Plateletcrit; MPV: Mean platelet volume; PDW: Platelet distribution width; PT-INR: Prothrombin time international normalized ratio; aPTT: Activated partial thromboplastin time; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; T bil: Total bilirubin; T chol: Total cholesterol; BUN: Blood urea nitrogen; GFR: Glomerular filtration rate; CK-MB: Creatine kinase myocardial band.

Imaging examinations

On computed tomography (CT) angiography, filling defects in the bilateral popliteal arteries, bilateral proximal anterior tibial artery, and bilateral tibioperoneal trunk were visible, which confirmed the Doppler findings (Figure 2). Concomitant venous thrombosis was not observed.

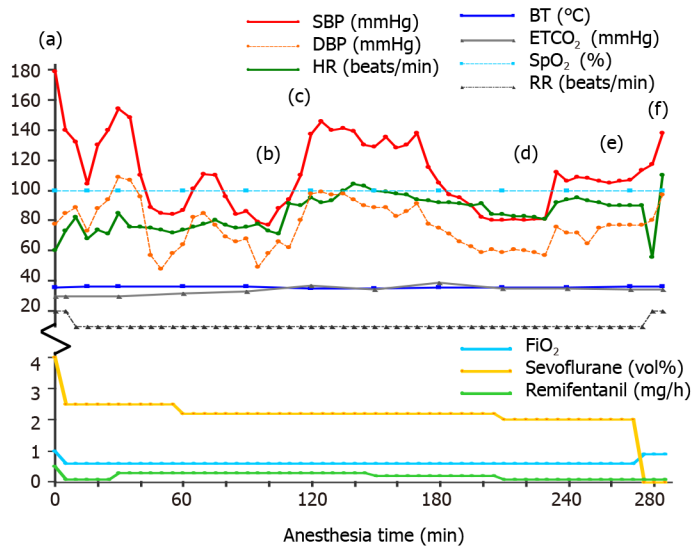

Figure 1 The vital signs and drugs used during the operation. Intravenous drugs: (a) Propofol 70 mg and rocuronium 50 mg; (b) Ephedrine 10 mg; (c) Rocuronium 10 mg; (d) Ephedrine 5 mg; (e) Ramosetron 0.3 mg and ketorolac 30 mg; and (f) Pyridostigmine 10 mg and glycopyrrolate 0.4 mg. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; BT: Body temperature; $ETCO_2$: End-tidal CO_2; SpO_2: Oxygen saturation; RR: Respiratory rate; FiO_2: Fraction of inspired oxygen.

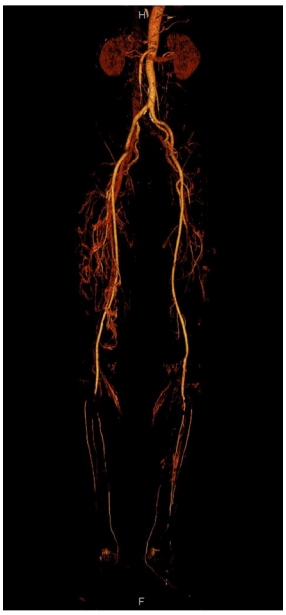

Figure 2 Computed tomography angiography findings. Filling defects are seen in the bilateral popliteal arteries, bilateral proximal anterior tibial arteries, and bilateral tibioperoneal trunks.

Further diagnostic work-up

Transesophageal echocardiography revealed no structural or functional abnormalities, and there was no evidence of a cardiac embolic source. Postoperative ECG showed a normal sinus rhythm.

FINAL DIAGNOSIS

The patient was diagnosed with acute thrombotic occlusion of the bilateral popliteal arteries, proximal anterior tibial arteries, and tibioperoneal trunk.

TREATMENT

After surgery, the patient was administered oxygen at the rate of 5 L/min using a nasal cannula, and the patient's vital signs were stable, except for tachycardia caused by pain (HR, 119-125 beats/min; SpO_2, 97%-100%; SBP, 110-120 mmHg; DBP, 55-80 mmHg; and RR, 15-20 breaths/min). For pain control, intravenous fentanyl 100 mcg and pethidine 25 mcg were administered immediately and 30 min after surgery, respectively. Immediately after the diagnosis was confirmed, intravenous unfractionated heparin (UFH) was administered for anticoagulation, with a bolus loading dose of 5000 units, followed by a maintenance dose of 800 units/h. After 2 h, heparin infusion was stopped, and surgical thrombectomy was planned. However, upon arrival in the operating room, that is 4 h after heparin cessation, the arterial pulse had returned in both lower limbs. Therefore, the surgery was canceled, and heparin therapy was reinitiated. Lipo-prostaglandin E1, a potent vasodilator and platelet aggregation inhibitor, was administered as an adjuvant treatment[10]. After 10 h of heparin reinitiation, the patient had hematochezia with a total volume of approximately 500 mL. Heparin infusion was immediately stopped, and the patient was closely monitored. The patient's vital signs remained stable (HR: 74-92 beats/min; SpO_2: 97%-99%; SBP: 120-140 mmHg; DBP: 80-82 mmHg; and RR: 20-21 breaths/min). Due to the repeated heparin infusion and discontinuation, activated partial thromboplastin time (aPTT) monitoring was not performed. Emergency sigmoidoscopy and esophagogastroduodenoscopy revealed no ischemic lesions or obvious sources of bleeding. Eight hours after the discontinuation of heparin, the aPTT level normalized, and hematochezia disappeared. The results of the laboratory tests after hematochezia are summarized in Table 1. No definite bleeding focus was noted on follow-up abdominal CT, gastroduodenoscopy, and sigmoidoscopy performed on postoperative days (PODs) 3, 5, and 7, respectively.

Although myoglobinuria was absent, the patient's history, symptoms, and markedly elevated myoglobin and CK levels strongly suggested rhabdomyolysis. Hydration was performed for kidney protection, and serial ECG monitoring and laboratory tests were performed. No specific ECG abnormalities were found immediately after surgery and on PODs 1-3. The serial laboratory results are summarized in Table 1. Myoglobin and CK levels normalized at POD 3 and 11 (37.3 ng/mL and 159 U/L), respectively, and the patient recovered completely from rhabdomyolysis without any sequelae.

OUTCOME AND FOLLOW-UP

Immediately after surgery, the patient complained of motor weakness in both lower extremities, and the muscle strength parameters according to the expanded Medical Research Council of Great Britain grading scale[11] were as follows, right/Left: hip flexion (2/5-), hip extension (2/5-), hip abduction (2/5-), hip adduction (2/5-), knee flexion (2/5-), knee extension (2/5-), ankle dorsiflexion (3/5-), ankle plantar flexion (3/5-), great toe extension (3/5-), and great toe flexion (3/5-). To evaluate the cause of motor weakness, the ankle brachial index (ABI) was measured; the right and left ABIs were within the normal range (1.26 and 1.21, respectively). Electromyogram and nerve conduction examinations showed non-specific findings. On POD 12, lipo-prostaglandin E1 was discontinued, and beraprost (0.12 mg/d), aspirin (100 mg/d), and physical therapy were initiated. The motor function of both lower extremities gradually improved and returned to normal, and the patient was discharged on POD 26 without any sequelae. The timeline of this case is shown in Figure 3. This study was approved by the Institutional Review Board of Pusan National University Hospital, Republic of Korea (ID 2104-014-101).

DISCUSSION

The conventional literature emphasizes the difference between arterial and venous thromboses[4]. The pathophysiology of venous thrombosis has been described as Virchow's triad, that is, stasis, hypercoagulability, and alterations in the endothelium [1,3,4]. In contrast, the pathophysiology of acute arterial thrombosis includes rupture of an atherosclerotic plaque associated with high shear rates and disruption of the endothelium[1,3,4]. Moreover, it is still recommended to treat arterial thrombosis with drugs that target platelets and venous thrombosis with drugs that target proteins of

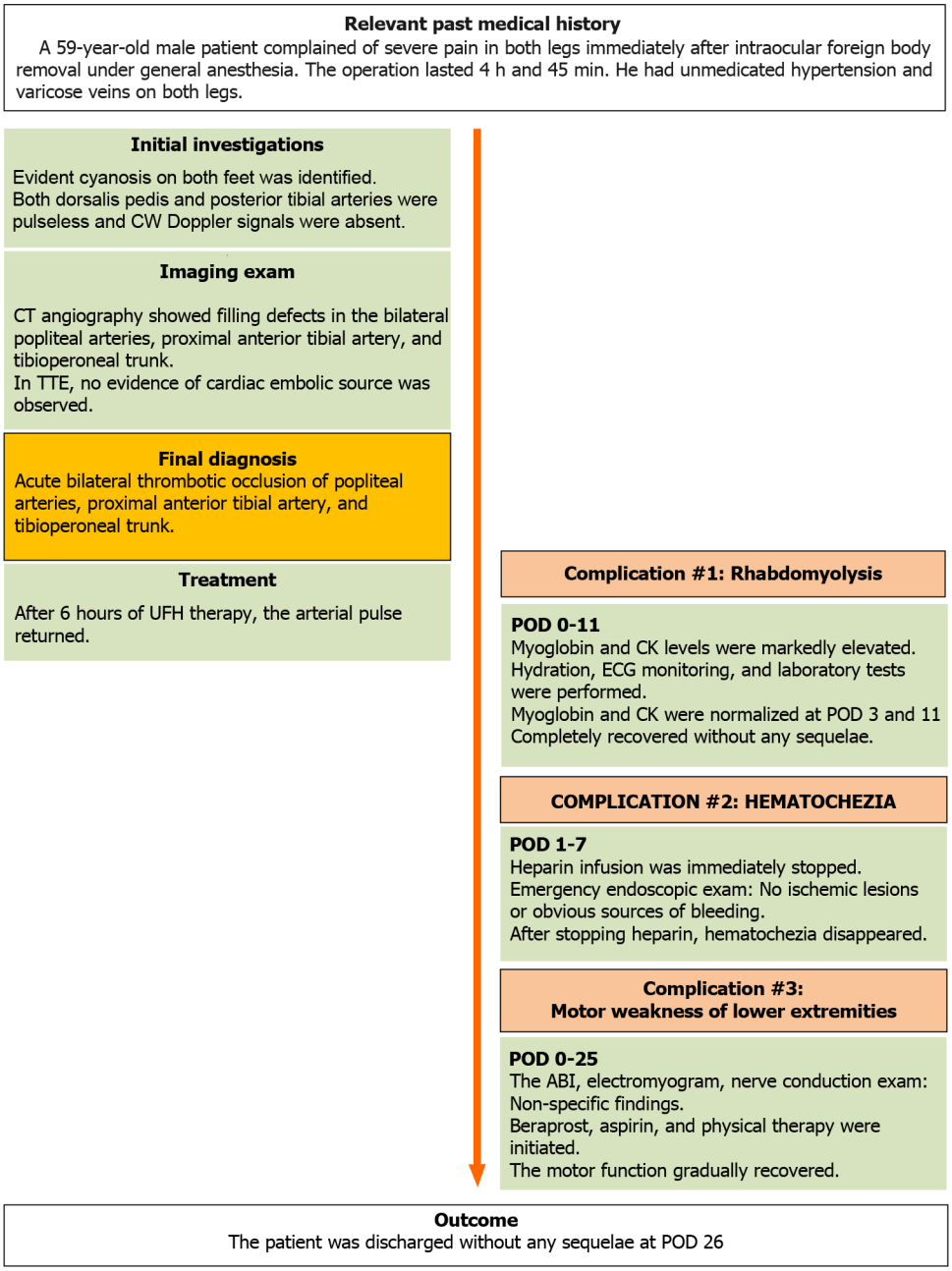

Figure 3 Case report timeline. CW: Continuous wave; TTE: Transthoracic echocardiogram; UFH: Unfractionated heparin; ABI: Ankle brachial index; POD: Postoperative day.

the coagulation cascade[1,4].

However, recent epidemiological studies have suggested that venous thrombosis and arterial thrombosis are closely related[4,5]. The most probable biological explanation for the link between these two types of thrombosis is that they share common cardiovascular risk factors, such as advanced age, immobility, obesity, smoking, hypertension, cancer, hormone replacement therapy, infection, major trauma, thrombophilia, and surgery[4,12,13].

In the present case, the patient had multiple risk factors (advanced age, smoking, hypertension, varicose veins, and protein S deficiency) and developed acute arterial thromboses in the lower limbs during an unexpectedly prolonged operation.

The aging process involves degeneration of vessel walls, activation of the coagulation system, and a decrease in physical activity[3,4], which exponentially increase the incidence of venous and arterial thromboses[4,14]. Specifically, compared with young adults, patients aged > 40 and > 50 years are at a significantly higher risk of venous and arterial thrombosis, respectively[15,16].

Cigarette smoking generates a prothrombotic environment by increasing the arterial intima-media thickness, promoting endothelial dysfunction, and increasing platelet

activation and prothrombic biomarkers[17,18]. Smoking is a particularly strong risk factor for arterial thrombosis[17,18]. However, evidence on the effect of smoking on venous thrombosis remains controversial[13]. According to a recent large-scale, population-based survey, smoking is a potential risk factor for venous thrombosis if additional risk factors are present[19].

In patients with hypertension, despite the continuous exposure of the vessel wall to high pressure, complications of hypertension are paradoxically more strongly associated with thrombosis than with hemorrhage[20,21]. In this context, hypertension has been considered the classical leading cause of arterial diseases of the heart, brain, and leg[4,22,23]. In addition, a recent meta-analysis reported that patients with hypertension are at a high risk of venous thromboembolism [odds ratio, 1.51; 95% confidence interval (CI): 1.23-1.85][13].

For venous thrombosis, varicose vein and protein S deficiency are well-documented risk factors; however, with regard to arterial thrombosis, the effects of varicose veins and protein S deficiency remain unclear[24,25]. In a retrospective cohort study using national health insurance data, Chang *et al*[24] found that varicose veins were significantly associated with peripheral arterial disease (adjusted hazard ratio, 1.76; 95%CI: 1.72-1.79). However, this study did not fully consider the possible confounding factors due to the inherent limitation of claims data, which necessitates further evaluation of associations between varicose veins and arterial thrombosis[24].

Protein S, a cofactor of protein C, inactivates coagulation factors Va and VIIIa and inhibits thrombin generation[25]. In a retrospective family cohort study, Mahmoodi *et al*[26] reported that protein S deficiency increases arterial thromboembolic risk in patients below 55 years of age (adjusted hazard ratio, 4.6; 95%CI: 1.1-18.3). Furthermore, Cho *et al*[27] suggested that protein S deficiency could be an independent risk factor for peripheral arterial occlusion. The authors also reported that patients with arterial occlusion with protein S deficiency demonstrated characteristic angiographic findings, such as long segment thrombotic occlusion of a main peripheral artery without atherosclerosis. Moreover, in the present case, protein S deficiency could be considered a possible trigger for arterial thrombosis. Therefore, further well-designed research is needed to investigate the effect of protein S deficiency on the development of arterial thrombosis.

Surgery is an independent risk factor for venous thrombosis[28]. Surgery itself induces blood stasis, release of tissue factors, and a generalized hypercoagulable environment[3,29]. With prolonged surgical time, patients are more likely to be exposed to a prothrombic state. In a large retrospective cohort study, Kim *et al*[28] demonstrated that in all types of surgery, surgical duration is directly correlated with an increased likelihood of the development of venous thromboembolism. Specifically, in Kim *et al*[28]'s study, the longest operation duration demonstrated a 1.27-fold increase in the odds of developing venous thromboembolism (95%CI: 1.21-1.34) as compared with the average operation duration; similarly, the shortest operation showed an odds ratio of 0.86 (95%CI: 0.83-0.88). Surgical procedures could also lead to arterial thrombosis-related complications, such as stroke and myocardial infarction[3,30,31], and there has been a growing interest in the increased risk of postoperative arterial thrombotic disease[4].

Prevention is the most effective strategy for limiting the adverse consequences of thromboembolism in surgical patients[29,32]. Thromboprophylaxis includes mechanical methods, such as the use of graded compression stockings, intermittent pneumatic compression devices, and pharmacologic methods using UFH and low-molecular-weight heparin[29,32]. These thromboprophylaxis strategies were designed for venous thromboembolism; however, recent studies have demonstrated that some of these strategies, including the use of intermittent pneumatic compression devices, UFH, and low-molecular-weight heparin, are also effective against arterial thrombotic diseases [33,34].

As ophthalmic surgery is considered as a low-risk procedure, routine thromboprophylaxis is often overlooked, and relevant guidelines for thromboprophylaxis during ophthalmic surgery are scarce[35,36]. In a previous survey-based study of anesthesiologists involved in the management of ophthalmic surgeries, 45% of respondents reported experiencing thromboembolism after ophthalmic surgery; however, only 40% stated that there were routine assessments for indications and contraindications of thromboprophylaxis in preanesthetic clinics[36]. In this case too, the preoperative thromboembolism risk assessment was overlooked. Moreover, while it was planned for < 2 h, the surgery was unexpectedly prolonged. As prevention is the best policy, this case highlights the importance of preoperative thromboembolic risk assessment, intraoperative communication between the surgeon and anesthesiologist (particularly in the context of unexpectedly prolonged surgery), and the need for consensus guide-

lines for the prevention of thromboembolism during ophthalmic surgery.

CONCLUSION

In summary, acute bilateral lower extremity arterial thromboses can occur unexpectedly after surgery. Our results suggest that anesthesiologists should pay particular attention to patients with multiple risk factors for thrombosis, especially those undergoing lengthy or high-risk surgical procedures. Although acute arterial thrombosis of the lower limb following surgery is rare, in cases with suggestive manifestations, additional evaluation for accurate diagnosis should be performed as soon as possible to prevent complications and improve outcomes.

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