

World Journal of *Clinical Cases*

World J Clin Cases 2019 December 26; 7(24): 4172-4425



**REVIEW**

- 4172** Polyunsaturated fatty acids and DNA methylation in colorectal cancer
Moradi Sarabi M, Mohammadrezaei Khorramabadi R, Zare Z, Eftekhar E

ORIGINAL ARTICLE**Retrospective Study**

- 4186** Impact of resection margins on long-term survival after pancreaticoduodenectomy for pancreatic head carcinoma
Li CG, Zhou ZP, Tan XL, Gao YX, Wang ZZ, Liu Q, Zhao ZM
- 4196** Arthroscopy combined with unicondylar knee arthroplasty for treatment of isolated unicompartmental knee arthritis: A long-term comparison
Wang HR, Li ZL, Li J, Wang YX, Zhao ZD, Li W
- 4208** Intact, pie-crusting and repairing the posterior cruciate ligament in posterior cruciate ligament-retaining total knee arthroplasty: A 5-year follow-up
Ma DS, Wen L, Wang ZW, Zhang B, Ren SX, Lin Y
- 4218** Community-acquired pneumonia complicated by rhabdomyolysis: A clinical analysis of 11 cases
Zhao B, Zheng R

Clinical Trials Study

- 4226** Dissection and ligation of the lateral circumflex femoral artery is not necessary when using the direct anterior approach for total hip arthroplasty
Zhao GY, Wang YJ, Xu NW, Liu F

Observational Study

- 4234** Expression of interleukin-32 in bone marrow of patients with myeloma and its prognostic significance
Wang G, Ning FY, Wang JH, Yan HM, Kong HW, Zhang YT, Shen Q

Randomized Controlled Trial

- 4245** Effect of different types of laryngeal mask airway placement on the right internal jugular vein: A prospective randomized controlled trial
Zhang JJ, Qu ZY, Hua Z, Zuo MZ, Zhang HY

SYSTEMATIC REVIEW

- 4254** Chronic pain, posttraumatic stress disorder, and opioid intake: A systematic review
López-Martínez AE, Reyes-Pérez Á, Serrano-Ibáñez ER, Esteve R, Ramírez-Maestre C

CASE REPORT

- 4270 Acute appendicitis in a patient after a uterus transplant: A case report
Kristek J, Kudla M, Chlupac J, Novotny R, Mirejovsky T, Janousek L, Fronek J
- 4277 Pneumococcal infection transmission between family members with congenital asplenia: A case report
Shibata J, Hiramatsu K, Kenzaka T, Kato T
- 4285 Successful treatment of warfarin-induced skin necrosis using oral rivaroxaban: A case report
Kamada M, Kenzaka T
- 4292 Simultaneous *Paragonimus* infection involving the breast and lung: A case report
Oh MY, Chu A, Park JH, Lee JY, Roh EY, Chai YJ, Hwang KT
- 4299 Isolated peritoneal lymphomatosis defined as post-transplant lymphoproliferative disorder after a liver transplant: A case report
Kim HB, Hong R, Na YS, Choi WY, Park SG, Lee HJ
- 4307 Three-dimensional image simulation of primary diaphragmatic hemangioma: A case report
Chu PY, Lin KH, Kao HL, Peng YJ, Huang TW
- 4314 Natural orifice specimen extraction with laparoscopic radical gastrectomy for distal gastric cancer: A case report
Sun P, Wang XS, Liu Q, Luan YS, Tian YT
- 4321 Huge brown tumor of the rib in an unlocatable hyperparathyroidism patient with “self-recovered” serum calcium and parathyroid hormone: A case report
Wang WD, Zhang N, Qu Q, He XD
- 4327 Percutaneous management of atrium and lung perforation: A case report
Zhou X, Ze F, Li D, Li XB
- 4334 Epstein-Barr virus-positive post-transplant lymphoproliferative disorder presenting as hematochezia and enterobrosis in renal transplant recipients in China: A report of two cases
Sun ZJ, Hu XP, Fan BH, Wang W
- 4342 Postoperative multidrug-resistant *Acinetobacter baumannii* meningitis successfully treated with intravenous doxycycline and intraventricular gentamicin: A case report
Wu X, Wang L, Ye YZ, Yu H
- 4349 Reconstruction of massive skin avulsion of the scrota and penis by combined application of dermal regeneration template (Pelnac) and split-thickness skin graft with vacuum-assisted closure: A case report
Fang JJ, Li PF, Wu JJ, Zhou HY, Xie LP, Lu H

- 4355** Multisystem smooth muscle dysfunction syndrome in a Chinese girl: A case report and review of the literature
Chen SN, Wang YQ, Hao CL, Lu YH, Jiang WJ, Gao CY, Wu M
- 4366** Kidney inflammatory myofibroblastic tumor masquerading as metastatic malignancy: A case report and literature review
Zhang GH, Guo XY, Liang GZ, Wang Q
- 4377** Hydroxychloroquine-induced renal phospholipidosis resembling Fabry disease in undifferentiated connective tissue disease: A case report
Wu SZ, Liang X, Geng J, Zhang MB, Xie N, Su XY
- 4384** Spontaneous ovarian hyperstimulation syndrome: Report of two cases
Gui J, Zhang J, Xu WM, Ming L
- 4391** Castleman disease in the hepatic-gastric space: A case report
Xu XY, Liu XQ, Du HW, Liu JH
- 4398** KIT and platelet-derived growth factor receptor α wild-type gastrointestinal stromal tumor associated with neurofibromatosis type 1: Two case reports
Kou YW, Zhang Y, Fu YP, Wang Z
- 4414** Isolated elevated aspartate aminotransferase in an asymptomatic woman due to macro-aspartate aminotransferase: A case report
Zhan MR, Liu X, Zhang MY, Niu JQ
- 4420** Rehabilitation of anterior pituitary dysfunction combined with extrapontine myelinolysis: A case report
Yang MX, Chen XN

ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Ashu Seith Bhalla, MD, Professor, Department of Radiodiagnosis, All India Institute of Medical Sciences, New Delhi 110029, India

AIMS AND SCOPE

The primary aim of *World Journal of Clinical Cases* (WJCC, *World J Clin Cases*) is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

WJCC mainly publishes articles reporting research results and findings obtained in the field of clinical medicine and covering a wide range of topics, including case control studies, retrospective cohort studies, retrospective studies, clinical trials studies, observational studies, prospective studies, randomized controlled trials, randomized clinical trials, systematic reviews, meta-analysis, and case reports.

INDEXING/ABSTRACTING

The WJCC is now indexed in PubMed, PubMed Central, Science Citation Index Expanded (also known as SciSearch®), and Journal Citation Reports/Science Edition. The 2019 Edition of Journal Citation Reports cites the 2018 impact factor for WJCC as 1.153 (5-year impact factor: N/A), ranking WJCC as 99 among 160 journals in Medicine, General and Internal (quartile in category Q3).

RESPONSIBLE EDITORS FOR THIS ISSUE

Responsible Electronic Editor: Ji-Hong Liu

Proofing Production Department Director: Yun-Xiaojuan Wu

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Semimonthly

EDITORS-IN-CHIEF

Dennis A Bloomfield, Bao-Gan Peng, Sandro Vento

EDITORIAL BOARD MEMBERS

<https://www.wjnet.com/2307-8960/editorialboard.htm>

EDITORIAL OFFICE

Jin-Lei Wang, Director

PUBLICATION DATE

December 26, 2019

COPYRIGHT

© 2019 Baishideng Publishing Group Inc

INSTRUCTIONS TO AUTHORS

<https://www.wjnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjnet.com/bpg/GerInfo/287>

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

<https://www.wjnet.com/bpg/gerinfo/240>

PUBLICATION MISCONDUCT

<https://www.wjnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

Successful treatment of warfarin-induced skin necrosis using oral rivaroxaban: A case report

Momoka Kamada, Tsuneaki Kenzaka

ORCID number: Momoka Kamada (0000-0001-6396-9271); Tsuneaki Kenzaka (0000-0002-3120-6605).

Author contributions: Kamada M managed the case and redaction and correction of the manuscript; Kenzaka T assisted with redaction, correction, and reconstruction of the manuscript; all authors read and approved the final manuscript.

Informed consent statement:

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor of this journal.

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

CARE Checklist (2016) statement:

The authors have read the CARE Checklist statement, and the manuscript was prepared and revised according to the CARE Checklist statement.

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

Momoka Kamada, Department of Internal Medicine, Hyogo Prefectural Tamba Medical Center, Tamba 669-3395, Japan

Tsuneaki Kenzaka, Division of Community Medicine and Career Development, Kobe University Graduate School of Medicine, Kobe 652-0032, Japan

Corresponding author: Tsuneaki Kenzaka, MD, PhD, Professor, Division of Community Medicine and Career Development, Kobe University Graduate School of Medicine, 2-1-5, Arata-cho, Hyogo-ku, Kobe 652-0032, Japan. smile.kenzaka@jichi.ac.jp

Telephone: +81-78-3826732

Fax: +81-78-3826283

Abstract

BACKGROUND

Heparin is commonly recommended for warfarin-induced skin necrosis; however, there is currently no established therapy for this disease. We present a serious case of warfarin-induced skin necrosis that was successfully treated with oral rivaroxaban, a factor Xa inhibitor.

CASE SUMMARY

A 48-year-old woman was admitted to the hospital for cellulitis of the right lower extremity. After antibiotic treatment, she developed pain and swelling of the left lower extremity, and deep vein thrombosis of both lower extremities was diagnosed. She was treated with a continuous heparin injection; subsequently, oral warfarin was concomitantly administered. Heparin was terminated after the therapeutic range was reached. On the following day, the patient had swelling and pain in the left lower extremity. In addition to decrease in protein S activity due to systemic lupus erythematosus, warfarin also reduced protein C activity, resulting in further hypercoagulation and skin necrosis. Warfarin was discontinued, and continuous heparin injection was resumed. Although the patient had to undergo amputation of the distal end of her left foot, continuous heparin injection was switched to oral rivaroxaban, and she was eventually discharged from the hospital in remission.

CONCLUSION

Administration of direct oral anticoagulants instead of warfarin is important in patients with decreased protein S and C activity.

Key words: Skin necrosis; Warfarin; Heparin; Rivaroxaban; Systemic lupus erythematosus; Case report

ses/by-nc/4.0/

Manuscript source: Unsolicited manuscript

Received: September 2, 2019

Peer-review started: September 2, 2019

First decision: November 13, 2019

Revised: November 17, 2019

Accepted: November 30, 2019

Article in press: November 30, 2019

Published online: December 26, 2019

P-Reviewer: Ciccone MM, De Ponti F

S-Editor: Ma YJ

L-Editor: A

E-Editor: Liu JH



©The Author(s) 2019. Published by Baishideng Publishing Group Inc. All rights reserved.

Core tip: We present a serious case of warfarin-induced skin necrosis that was successfully treated with oral rivaroxaban, a factor Xa inhibitor. Administration of direct oral anticoagulants instead of warfarin is important in patients with decreased protein S and C activity.

Citation: Kamada M, Kenzaka T. Successful treatment of warfarin-induced skin necrosis using oral rivaroxaban: A case report. *World J Clin Cases* 2019; 7(24): 4285-4291

URL: <https://www.wjgnet.com/2307-8960/full/v7/i24/4285.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v7.i24.4285>

INTRODUCTION

In warfarin-induced skin necrosis, the production of protein C and S is inhibited in an early stage after warfarin administration, which increases coagulability and thrombosis formation in the capillaries and venules of the dermis or subcutaneous tissue, leading to skin ischemia or necrosis^[1]. Although there is currently no established treatment for warfarin-induced skin necrosis, heparin is commonly recommended^[1]. However, the use of non-vitamin K antagonist anticoagulants is recommended in some case reports^[2-6].

We report the case of a patient who developed serious warfarin-induced skin necrosis as well as protein S deficiency caused by systemic lupus erythematosus (SLE), who was then successfully treated with oral rivaroxaban.

CASE PRESENTATION

Chief complaints

A 48-year-old woman presented to the emergency room with chief complaints of swelling of the right lower extremity and pyrexia.

History of present illness

Regarding her present illness, pyrexia and redness, and swelling of the right lower extremity developed 10 and 5 d before hospitalization, respectively. She visited our hospital because the pyrexia was unresolved, and the symptoms of the lower extremity worsened.

History of past illness

Her medical history included paronychia of the right big toe. She was gravida 3 and para 3, with no history of abortion.

Personal and family history

Her father had a history of cerebral infarction.

Physical examination upon admission

The patient's physical examination findings during examination were as follows: Body temperature, 39.4 °C; blood pressure, 107/65 mmHg; pulse rate, 81 beats/min and regular; respiratory rate, 13 breaths/min; and oxygen saturation, 97% (room air). Physical findings included swelling, warmth, redness, and pain in the right lower extremity as well as tinea unguis in the right foot.

Laboratory examinations

Blood test findings on admission were as follows: White blood cell count, 4800 cells/ μ L; C-reactive protein, 9.51 mg/dL; prothrombin time (PT), 13.5 seconds; activated partial thromboplastin time, 34.5 s; and D-dimer, 6.9 μ g/mL (Table 1).

The patient was admitted to the hospital for cellulitis of the right lower extremity. Cefazolin (1 g) was administered every 8 h; subsequently, pyrexia declined. However, she redeveloped pyrexia (temperature, 39 °C). Considering the possibility of drug fever, we switched the antibiotic to clindamycin (600 mg) and administered it every 8 h from day 4 of hospitalization. As skin findings improved, the treatment of cellulitis was completed after 8 d.

Table 1 Laboratory data upon admission

Parameter	Recorded value	Standard value
White blood cell count	4800/ μ L	4500-7500/ μ L
Lymphocyte count	1300/ μ L	
Red blood cell count	327×10^3 / μ L	$380-480 \times 10^3$ / μ L
Hemoglobin	9.4 g/dL	11.3-15.2 g/dL
Hematocrit	31.3%	36%-45%
Platelet count	10.6×10^3 / μ L	$13-35 \times 10^3$ / μ L
International normalized ratio	1.05	0.80-1.20
Activated partial thromboplastin time	34.5 s	26.9-38.1 s
Fibrinogen	374 mg/L	150-400 mg/dL
D-dimer	6.9 μ g/mL	≤ 1.0 μ g/mL
C-reactive protein	9.51 mg/L	≤ 1.0 mg/L
Total protein	7.5 g/dL	6.9-8.4 g/dL
Albumin	3.6 g/dL	3.9-5.1 g/dL
Total bilirubin	0.6 mg/dL	0.2-1.2 mg/dL
Aspartate aminotransferase	22 U/L	11-30 U/L
Alanine aminotransferase	24 U/L	4-30 U/L
Lactate dehydrogenase	262 U/L	109-216 U/L
Creatine phosphokinase	75 U/L	40-150 U/L
Blood urea nitrogen	7.6 mg/dL	8-20 mg/dL
Creatinine	0.43 mg/dL	0.63-1.03 mg/dL
Sodium	137 mEq/L	136-148 mEq/L
Potassium	3.6 mEq/L	3.6-5.0 mEq/L
Chloride	103 mEq/L	98-108 mEq/L
Glucose	146 mg/dL	70-109 mg/dL
Hemoglobin A1c	5.7%	$\leq 5.8\%$

Imaging examinations

On day 8 of hospitalization, the patient developed swelling of the left lower extremity. Deep vein thrombosis was suspected because she was on bed rest for the treatment of cellulitis. Contrast-enhanced computed tomography revealed deep vein thrombi in both femoral veins (Figure 1); she was diagnosed with bilateral deep vein thrombosis of the lower extremities. Blood tests for the evaluation of thrombophilia as a risk factor for the development of deep vein thrombosis revealed decreased protein S activity (Table 2); therefore, the patient was diagnosed with deep vein thrombosis caused by protein S deficiency.

Continuous intravenous infusion of heparin was initiated for deep vein thrombosis on day 8 of hospitalization, and oral warfarin was concomitantly administered from day 14 of hospitalization. Continuous heparin injection was completed on day 19 of hospitalization when the PT-international normalized ratio (INR) reached the therapeutic target range (1.6-2.5). Immediately after heparin cessation, the patient developed pain, swelling, redness, and blisters in her left lower extremity, as well as cold peripheries and skin necrosis (Figure 2). The activities of protein S and C at this time were both $< 10\%$. Warfarin administration during the period of low protein S activity subsequently led to decreased protein C activity, and discontinuation of the heparin injection caused hypercoagulation, leading to the onset of skin necrosis. The patient had a score of 7 points on the Naranjo adverse drug reaction probability scale^[7]. Accordingly, we diagnosed her with warfarin-induced skin necrosis.

When evaluating the cause of decrease in protein S activity in this patient, we observed that she developed pleurisy approximately at the same time as the onset of bilateral deep vein thrombosis of the lower extremities. Along with lymphopenia, the patient fulfilled 2 clinical and 3 immunologic criteria of the Systemic Lupus International Collaborating Clinics classification^[7]: Antinuclear antibody-positive (640-fold, homogeneous); low complement C4 (10 mg/dL; reference value, 17-45 mg/dL), C3 (56 mg/dL; reference value, 86-160 mg/dL) and CH50 (25 U/mL; reference value, 30-45 U/mL) levels; and direct Coombs test-positive. Therefore, the patient was diagnosed with SLE.

The activities of protein S and C over time are shown in Figure 3. Protein S activity

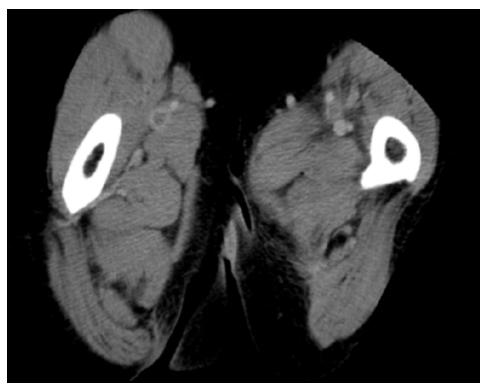


Figure 1 Contrast-enhanced computed tomography on hospitalization day 8. Deep vein thrombi in both femoral veins were observed.

was normalized with 30 mg of prednisolone (PSL). Based on these results, the decrease in protein S activity in this patient was attributed to SLE. Warfarin was discontinued on the day 22 of hospitalization, and continuous heparin injection was resumed for warfarin-induced skin necrosis, and protein C activity was normalized. Necrotic tissue debridement was performed on day 35 of hospitalization (Figure 4), and left side toe amputation and free flap surgery were performed on day 67 of hospitalization (Figure 5). Subsequently, heparin was discontinued, and the patient was switched to oral rivaroxaban (15 mg/d) on day 68 of hospitalization. She was admitted to another hospital for rehabilitation on day 86 of hospitalization. At the 2-year follow-up, no aggravations of skin necrosis were observed, and the activities of both proteins S and C remained normal.

FINAL DIAGNOSIS

Decreased protein S activity caused by systemic lupus erythematosus, and warfarin-induced skin necrosis decreased with protein C activity.

TREATMENT

Prednisolone and rivaroxaban.

OUTCOME AND FOLLOW-UP

At the 2-year follow-up, no aggravations of skin necrosis were observed, and the activities of both proteins S and C remained normal. Activity of systemic lupus erythematosus was stable.

DISCUSSION

In this report, the patient developed warfarin-induced skin necrosis as well as decreased protein S activity secondary to SLE. Warfarin-induced skin necrosis occurred after discontinuation of heparin; however, the skin necrosis was not aggravated by the SLE treatment or oral administration of rivaroxaban, a direct factor Xa inhibitor. Only three other cases showing response to rivaroxaban for warfarin-induced skin necrosis have been previously reported^[4-6].

Our patient developed bilateral deep vein thrombosis of the lower extremities during the treatment for cellulitis. A detailed examination for thrombophilia revealed markedly decreased protein S activity. Protein S deficiency can occur due to the following: Congenital factors, pregnancy, oral hormonal contraceptive use, disseminated intravascular coagulation, acute thromboembolism, human immunodeficiency virus infection, nephrotic syndrome, liver disease, L-asparaginase chemotherapy, varicella recovery, antiphospholipid antibody syndrome, oral steroid use, and vitamin K deficiency (*i.e.*, decreased food intake, biliary obstruction, and oral warfarin administration)^[8]. Protein S deficiency has been rarely reported in patients

Table 2 Tests of thrombophilia

Parameter	Recorded value	Standard value
Anti-cardiolipin β 2-glycoprotein I complex antibody	2.0 U/mL	< 3.5 U/mL
Lupus anticoagulant	1.3	< 1.3
Protein S activity	< 10%	56%-126%
Protein C activity	83%	64%-146%
Antithrombin III	79%	79%-121%

with SLE^[9,10]. Our patient developed pleurisy during the clinical course. She was diagnosed with SLE after she fulfilled the Systemic Lupus International Collaborating Clinics classification criteria (*i.e.*, clinical criteria of pleurisy and lymphopenia as well as the immunologic criteria of positive antinuclear antibody, low complement, and positive direct Coombs-test)^[7]. She had no family history, history of abortion, hepatic dysfunction, or renal dysfunction suggestive of congenital diseases. After initiating PSL treatment, protein S activity returned to normal as SLE improved, indicating that decreased protein S activity was caused by SLE.

Warfarin induced skin necrosis develops when proteins C and S production is inhibited in the early stage after the administration of warfarin, a vitamin K antagonist, resulting in increased coagulability and hypercoagulation^[11]. Our case demonstrated that serious skin necrosis can occur in the early stage of treatment despite achieving a PT-INR within the therapeutic range. Onset of skin necrosis can occur within a few hours or several weeks after warfarin administration^[11]. There are no currently established anticoagulant therapies for patients with decreased proteins S and C activities. Heparin, which is not a vitamin K antagonist, is commonly used for the treatment of warfarin-induced necrosis^[11]. However, direct oral anticoagulants, such as dabigatran and rivaroxaban, have been found to be more effective for our patient based on previous findings^[2-6]. We could find only three studies reporting that oral rivaroxaban was effective^[7-9]. Heparin requires continuous intravenous injection, and its administration is not suitable for patients with deep vein thrombosis who require long-term anticoagulant therapy, such as our patient, because of the route of administration and necessity of hospitalization.

CONCLUSION

We report the case of a patient with warfarin-induced skin necrosis who was successfully treated with oral rivaroxaban, a factor Xa inhibitor. It is important to administer DOACs instead of warfarin in patients with decreased activities of protein S and C, which are vitamin K-dependent coagulation inhibitors. During treatment of bilateral deep vein thrombosis of the lower extremities, it is necessary to closely examine the patient for the presence of thrombophilia and avoid warfarin administration until confirmation of the examination results. Furthermore, continuous heparin injections should be carefully discontinued when warfarin is administered.

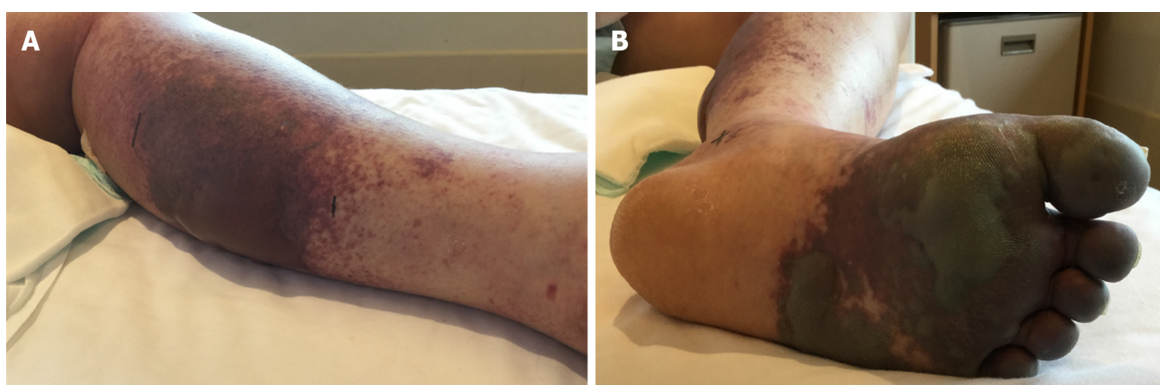


Figure 2 Skin necrosis observed in the left lower extremity on hospitalization day 19. A: Left calf; B: Left foot.

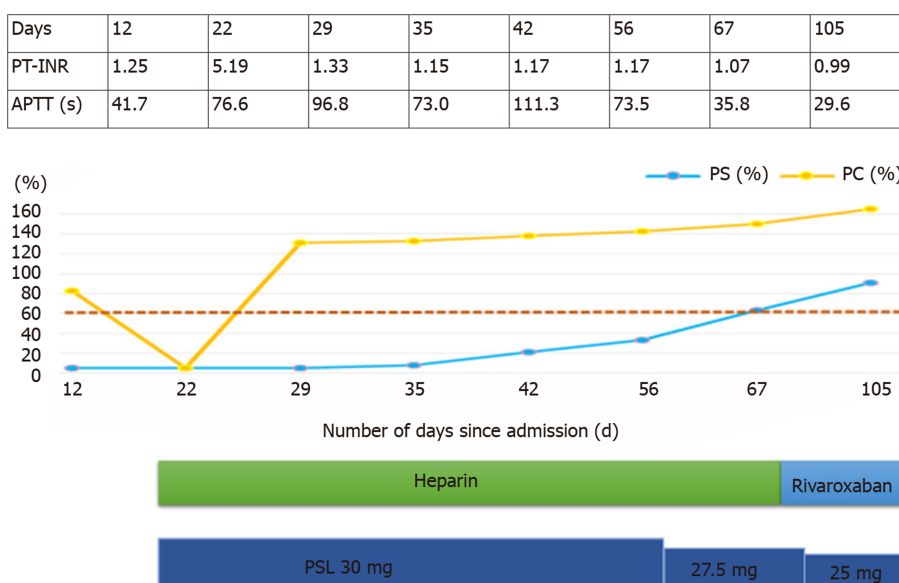


Figure 3 Clinical course of the patient. The course of treatment with prednisolone and anticoagulants as well as the activities of proteins C and S are shown. PSL: Prednisolone.

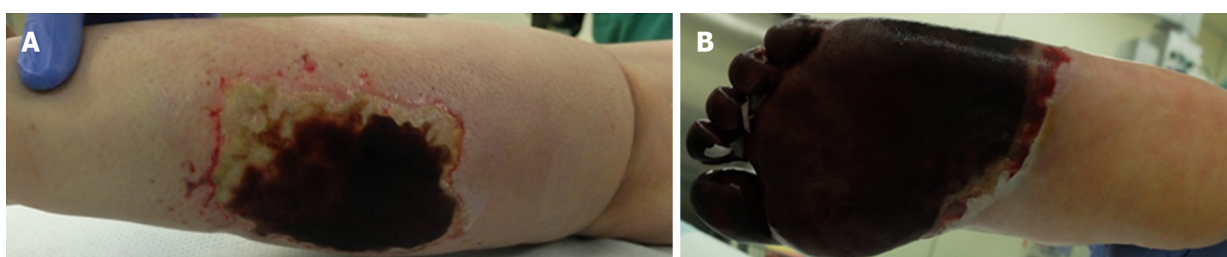


Figure 4 Debridement of necrotic tissue in the left lower extremity (hospitalization day 35). A: Left calf; B: Left foot.

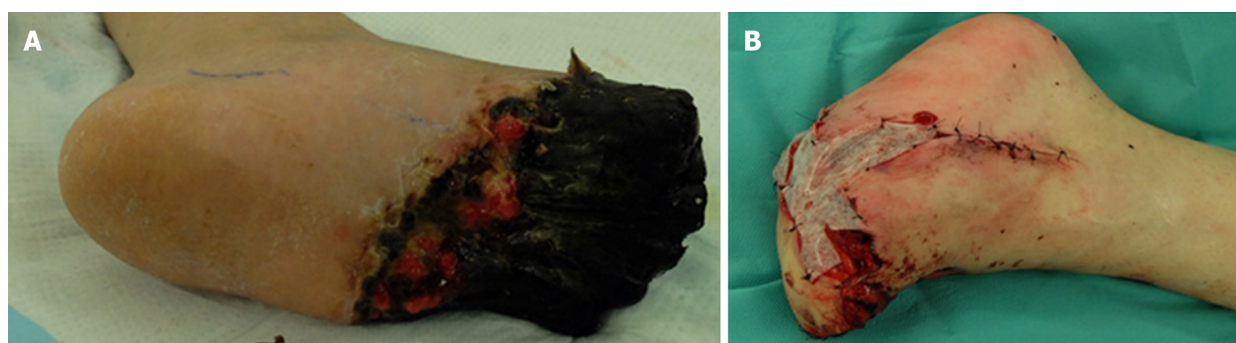


Figure 5 Left toe amputation and free flap surgery were performed on hospitalization day 67. A: Pre-surgery of left foot; B: Post-surgery of left foot.

REFERENCES

- 1 **Kakagia DD**, Papanas N, Karadimas E, Polychronidis A. Warfarin-induced skin necrosis. *Ann Dermatol* 2014; **26**: 96-98 [PMID: 24648693 DOI: 10.1016/j.jaad.2008.12.039]
- 2 **Tripodi A**, Martinelli I, Chantarangkul V, Clerici M, Artoni A, Passamonti S, Peyvandi F. Thrombin generation and other coagulation parameters in a patient with homozygous congenital protein S deficiency on treatment with rivaroxaban. *Int J Hematol* 2016; **103**: 165-172 [PMID: 26586461 DOI: 10.1007/s12185-015-1898-6]
- 3 **Bakoyiannis C**, Karaolani G, Patelis N, Maskanakis A, Tsaples G, Klonaris C, Georgopoulos S, Liakakos T. Dabigatran in the Treatment of Warfarin-Induced Skin Necrosis: A New Hope. *Case Rep Dermatol Med* 2016; **2016**: 3121469 [PMID: 27110410 DOI: 10.1155/2016/3121469]
- 4 **Martinelli I**, Bucciarelli P, Artoni A, Fossali EF, Passamonti SM, Tripodi A, Peyvandi F. Anticoagulant treatment with rivaroxaban in severe protein S deficiency. *Pediatrics* 2013; **132**: e1435-e1439 [PMID: 24144709 DOI: 10.1542/peds.2013-1156]
- 5 **Lai J**, Ramai D, Alchi R, Bloomfield D. Anticoagulation therapy for thromboembolism prevention: a case of warfarin-induced skin necrosis in the setting of protein C deficiency. *BMJ Case Rep* 2017; 2017 [PMID: 28500260 DOI: 10.1136/bcr-2016-218015]
- 6 **Menon N**, Sarode R, Zia A. Rivaroxaban dose adjustment using thrombin generation in severe congenital protein C deficiency and warfarin-induced skin necrosis. *Blood Adv* 2018; **2**: 142-145 [PMID: 29365322 DOI: 10.1182/bloodadvances.2017012047]
- 7 **Petri M**, Orbai AM, Alarcón GS, Gordon C, Merrill JT, Fortin PR, Bruce IN, Isenberg D, Wallace DJ, Nived O, Sturfelt G, Ramsey-Goldman R, Bae SC, Hanly JG, Sánchez-Guerrero J, Clarke A, Aranow C, Manzi S, Urowitz M, Gladman D, Kalunian K, Costner M, Werth VP, Zoma A, Bernatsky S, Ruiz-Irastorza G, Khamashta MA, Jacobsen S, Buyon JP, Maddison P, Dooley MA, van Vollenhoven RF, Ginzler E, Stoll T, Peschken C, Jorizzo JL, Callen JP, Lim SS, Fessler BJ, Inanc M, Kamen DL, Rahman A, Steinsson K, Franks AG, Sigler L, Hameed S, Fang H, Pham N, Brey R, Weisman MH, McGwin G, Magder LS. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. *Arthritis Rheum* 2012; **64**: 2677-2686 [PMID: 22553077 DOI: 10.1002/art.34473]
- 8 **Bauer KA**. Protein S deficiency. 2017. Available from: https://www.uptodate.com/contents/protein-s-deficiency?search=Protein%20S%20deficiency&source=search_result&selectedTitle=1~76&usage_type=defaultdisplay_rank=1
- 9 **Lertnawapan R**, Sakonlaya D. Lupus protein-losing enteropathy patient with protein C and protein S deficiency-induced thrombosis: A case report with review of the literature. *Acta Reumatol Port* 2017; **42**: 265-268 [PMID: 28375198]
- 10 **Ginsberg JS**, Demers C, Brill-Edwards P, Bona R, Johnston M, Wong A, Denburg JA. Acquired free protein S deficiency is associated with antiphospholipid antibodies and increased thrombin generation in patients with systemic lupus erythematosus. *Am J Med* 1995; **98**: 379-383 [PMID: 7709951 DOI: 10.1016/S0002-9343(99)80317-9]
- 11 **Naranjo CA**, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, Janecek E, Domecq C, Greenblatt DJ. A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther* 1981; **30**: 239-245 [PMID: 7249508 DOI: 10.1038/clpt.1981.154]



Published By Baishideng Publishing Group Inc
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-2238242
E-mail: bpgoffice@wjgnet.com
Help Desk: <https://www.f6publishing.com/helpdesk>
<https://www.wjgnet.com>

