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INDEXING/ABSTRACTING

The WICC is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Journal Citation Reports/Science Edition, Current Contents®/Clinical Medicine, PubMed, PubMed Central, Scopus, Reference Citation Analysis, China National Knowledge Infrastructure, China Science and Technology Journal Database, and Superstar Journals Database. The 2022 Edition of Journal Citation Reports® cites the 2021 impact factor (IF) for WJCC as 1.534; IF without journal self cites: 1.491; 5-year IF: 1.599; Journal Citation Indicator: 0.28; Ranking: 135 among 172 journals in medicine, general and internal; and Quartile category: Q4. The WJCC's CiteScore for 2021 is 1.2 and Scopus CiteScore rank 2021: General Medicine is 443/826.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: Hua-Ge Yn, Production Department Director: Xu Guo; Editorial Office Director: Jin-Lei Wang.

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Thrice Monthly

EDITORS-IN-CHIEF

Bao-Gan Peng, Jerzy Tadeusz Chudek, George Kontogeorgos, Maurizio Serati, Ja Hveon Ku

EDITORIAL BOARD MEMBERS

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

PUBLICATION DATE

September 16, 2022

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INSTRUCTIONS TO AUTHORS

https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS

https://www.wignet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS

https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT

https://www.wignet.com/bpg/gerinfo/208

ARTICLE PROCESSING CHARGE

https://www.wignet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS

https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION

https://www.f6publishing.com

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World J Clin Cases 2022 September 16; 10(26): 9404-9410

DOI: 10.12998/wjcc.v10.i26.9404

CASE REPORT

ISSN 2307-8960 (online)

Antiphospholipid syndrome with renal and splenic infarction after blunt trauma: A case report

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Specialty type: Emergency medicine

Provenance and peer review:

Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

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

P-Reviewer: Sandoval C, Chile; Yi XL, China

Received: May 8, 2022 Peer-review started: May 8, 2022 First decision: May 30, 2022 **Revised:** June 12, 2022 Accepted: August 6, 2022 Article in press: August 6, 2022 Published online: September 16,



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Abstract

BACKGROUND

In trauma patients, bleeding is an immediate major concern. At the same time, there are few cases of acute vascular occlusion after blunt trauma, and it is unclear what assessment and diagnosis should be considered for these cases. Herein, we describe a patient diagnosed with antiphospholipid syndrome after a hypercoagulable workup for acute renal and splenic vascular occlusion due to blunt trauma.

CASE SUMMARY

A 20-year-old man was admitted to the emergency department with abdominal pain after hitting a tree while riding a sled 10 h ago. He had no medical history. Radiological investigations revealed occlusion of the left renal artery with global infarction of the left kidney and occlusion of branches of the splenic artery with infarction of the central portion of the spleen. Attempted revascularization of the left renal artery occlusion through percutaneous transluminal angioplasty failed due to difficulty in passing the wire through the total occlusion. Considering the presence of acute multivascular occlusions in a young man with low cardiovascular risk, additional laboratory tests were performed to evaluate hypercoagulability. The results suggested a high possibility of antiphospholipid syndrome. Treatment with a subcutaneous injection of enoxaparin was started and changed to oral warfarin after two weeks. The diagnosis was confirmed, and he continued to visit the rheumatology outpatient clinic while taking warfarin.

CONCLUSION

A hypercoagulable workup can be considered in trauma patients with acute multivascular occlusion, especially in young patients with low cardiovascular risk.

Key Words: Wounds and injuries; Wounds, Nonpenetrating; Antiphospholipid syndrome; Renal artery obstruction; Splenic infarction; Case reports

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Core Tip: Acute vascular occlusion following blunt trauma has been reported in a few trauma patients. We describe a case diagnosed with antiphospholipid syndrome after a hypercoagulable workup for acute multivascular occlusion due to blunt trauma. In trauma patients with acute multivascular occlusion or a hypercoagulable state, especially in young patients with low cardiovascular risk, a hypercoagulable workup may help evaluate other risk factors.

Citation: Lee NA, Jeong ES, Jang HS, Park YC, Kang JH, Kim JC, Jo YG. Antiphospholipid syndrome with renal and splenic infarction after blunt trauma: A case report. World J Clin Cases 2022; 10(26): 9404-9410

URL: https://www.wjgnet.com/2307-8960/full/v10/i26/9404.htm

DOI: https://dx.doi.org/10.12998/wjcc.v10.i26.9404

INTRODUCTION

Except for head injuries, bleeding is the immediate major concern for most patients experiencing trauma [1,2]. In contrast, there are few cases of acute vascular occlusion and consequent infarction after trauma. In blunt trauma, blood vessels can be compressed or stretched. Intimal injury from vascular stretching can lead to vessel dissection or intramural thrombosis, resulting in vessel stenosis or total occlusion[3]. This can progress to organ infarction.

Usually, splenic and renal infarctions are related to cardiac thromboembolism[4], other systemic pathologies[5], sickle cell trait carriers[6], and infection[7]. The incidence of renal or spleen infarction in patients with blunt trauma is unknown. However, there are reports that 16.7%-30.8% of patients with renal infarction have renal artery injury [8,9], 20% of patients with spleen infarction have splenic artery injury, and 20% of patients with spleen and renal infarctions have trauma[9].

This case report describes a patient with acute infarction of the left kidney and the central portion of the spleen due to vascular occlusion after blunt trauma. The patient was diagnosed with antiphospholipid syndrome (APS) after a hypercoagulable workup. Through this case report, we suggest that additional evaluation of hypercoagulability is needed in patients with acute multivascular occlusion after blunt trauma.

CASE PRESENTATION

Chief complaints

A 20-year-old man was admitted to the emergency department with abdominal pain after hitting a tree while riding a sled 10 h ago.

History of present illness

The patient's symptoms started after the trauma, and the abdominal pain worsened over time.

History of past illness

The patient had no previous medical history.

Personal and family history

The patient did not smoke and had no relevant family history.

Physical examination

At the time of admission, the patient's Glasgow Coma scale was 15/15, blood pressure was 120/70 mmHg, body temperature was 36.3 °C, heart rate was 94 bpm, respiratory rate was 20 breaths per minute, and oxygen saturation in room air was 98%. A physical examination revealed tenderness without rebound pain over the left upper quadrant.

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Laboratory examinations

The results of the laboratory investigations were as follows: white blood cell count, $21.8 \times 10^3 / \mu L$ (normal range, 4.8-10.8); neutrophils, 86.8% (50-75); hemoglobin, 13.1 g/dL (12-18); platelet count, $172 \times$ 10³/μL (130-450); fibrin degradation products, 16.4 μg/mL (0-5); D-dimer, 6.19 mg/L (0-0.55); creatinine, 1.37 mg/dL (0.5-1.3); modification of diet in renal disease estimated glomerular filtration rate (eGFR), 66.938 mL/min/1.73 m²(71.11-214.2); and chronic kidney disease epidemiology collaboration eGFR, 74.24 mL/min/1.73 m² (79.1-157.0). Lactate, fibrinogen assay, activated partial thromboplastin time, and prothrombin time (PT) values were within normal limits.

Imaging examinations

Radiological investigations revealed occlusion of the left renal artery with global infarction of the left kidney (Figure 1), occlusion of the branches of the splenic artery with infarction of the central portion of the spleen (Figure 2), a small amount of hemoperitoneum, and left adrenal hematoma (Figure 3).

Further diagnostic work-up

The percutaneous transluminal angioplasty attempted to revascularize the left renal artery occlusion failed due to difficulty in passing the wire through the total occlusion. In addition, considering the acute splenic and renal infarctions with multivascular occlusion in this young male patient without underlying disease, a laboratory evaluation of hypercoagulability was performed to exclude the possibility of other diseases. Lupus anticoagulant (LA), antinuclear antibody (ANA) titer, antineutrophil cytoplasmic antibody (ANCA), anti-cardiolipin (aCL) immunoglobulin M (IgM) and immunoglobulin G (IgG), and anti-β2-glycoprotein I (GPI) IgM and IgG tests were performed.

Laboratory tests were reported on day 7 after the injury and were as follows: LA, weak positive (1.41) (0-1.30, negative; 1.31-1.50, weak positive; 1.51-2.0, moderate positive; > 2.0, strong positive; LA1 screening reagent, LA2 confirmation reagent; Siemens Healthcare, Marburg, Germany); ANA titer, negative (< 1:80, negative; ANA HEp-2 standard kit; Aesku Diagnostics, Wendelsheim, Germany); ANCA, negative (< 1:20, negative; ANCA Ethanol; Aesku Diagnostics, Wendelsheim, Germany); aCL IgM, negative (< 7 U/mL, negative); aCL IgG, negative (< 10 U/mL, negative; aCL IgM, IgG; Orgentec Diagnostika, Mainz, Germany); anti-β2-GPI IgM, negative (< 7 U/mL, negative); and anti-β2-GPI IgG, negative (< 7 U/mL, negative; Phadia EliA; Thermofisher, Freiburg, Germany).

FINAL DIAGNOSIS

APS is diagnosed when the patient meets at least one clinical criterion and one laboratory criterion. Clinical criteria include clinical episodes of arterial, venous, or small vessel thrombosis or pregnancy morbidity. Vascular thrombosis of clinical criteria can be diagnosed with pathologic confirmation or appropriate imaging studies. Laboratory criteria include LA, aCL IgG and/or IgM, or anti-β2-GPI IgG and/or IgM detected twice or more within an interval of at least 12 wk[10]. This patient had acute splenic and renal infarctions with complete vessel occlusion confirmed by arteriography. This event meets the clinical criteria. For the laboratory criteria, the LA test showed weak positivity (1.41). Although the presence of LA must be reconfirmed after 12 wk, LA is an antibody with high specificity [11]. Therefore, it was judged that the possibility of APS was high, and inpatient treatment for APS was performed by a rheumatologist.

TREATMENT

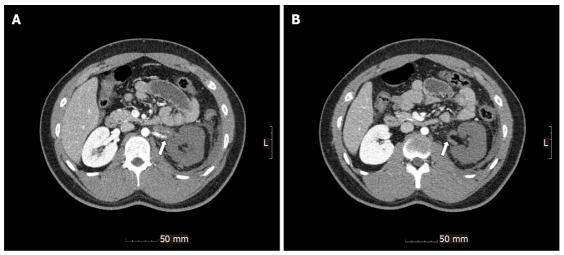
Treatment with a subcutaneous injection of enoxaparin [low-molecular-weight heparin (LMWH)] was started. In addition, the department of infectious diseases considered the patient to have functional asplenia due to the near-total splenic infarction; thus, asplenia vaccination was performed.

After 2 wk, the LMWH was changed to warfarin. Thereafter, if the follow-up LA test is positive, lifelong warfarin therapy should be considered. The patient was discharged 3 wk after the injury.

OUTCOME AND FOLLOW-UP

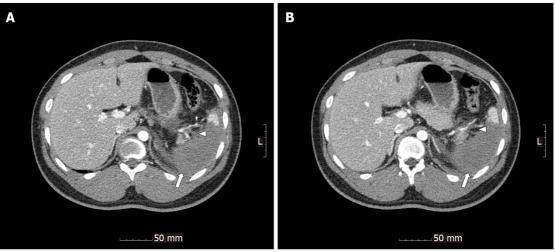
The result of the LA test performed at the rheumatology outpatient clinic was 1.51, moderate positive (0-1.30, negative; 1.31-1.50, weak positive; 1.51-2.0, moderate; > 2.0, strong). The patient was diagnosed with APS. He continues to visit the rheumatology outpatient clinic and is taking warfarin. Follow-up ultrasonography revealed atrophic changes in the left kidney, resolved splenic infarction in the superior pole, and resolution of the fluid collection in the perisplenic space without abscess. In addition, creatinine and eGFR were within the normal range.

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Figure 1 Renal infarction seen in the initial computed tomography scan. A: Occlusion of the left renal artery with global infarction of the left kidney; B: Left renal artery without blood flow.



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Figure 2 Splenic infarction seen in the initial computed tomography scan. A and B: Multiple occlusions of the branches of the splenic artery (arrowhead) with infarction of the central portion of the spleen (arrow).

DISCUSSION

Antiphospholipid syndrome is an autoimmune syndrome characterized by vascular or pregnancy morbidity in the presence of persistent laboratory evidence of antiphospholipid antibodies (aPL)[12]. In the absence of a thromboembolic event or pregnancy morbidity, the presence of aPL alone is not sufficient for a diagnosis of APS. Furthermore, aPL can be a temporary finding due to other acute diseases, such as systemic lupus erythematosus, bacterial infection (syphilis, Lyme disease), viral infection (human immunodeficiency virus, hepatitis), or the use of medications such as chlorpromazine, phenytoin, quinidine, hydralazine, or amoxicillin[13-17]. Therefore, the diagnosis of APS is based on the presence of aPL on two or more occasions at least 12 wk apart in addition to vascular thrombosis or pregnancy morbidity[10,18].

In this case, characteristically, the patient was young, had no medical and family history, had low cardiovascular risk, had thromboembolic events rather than hemorrhage, and progressed to renal and splenic infarction. As a result, hypercoagulability was evaluated and APS was diagnosed. Given that this patient had not experienced a previous thromboembolic event, the trauma may have triggered the formation of aPL and thromboembolism[19]. Acute multivascular occlusion is triggered by traumainduced intimal injury of blood vessels and consequent thrombosis. This suggests that it is necessary to consider the possibility of a hypercoagulable state and evaluate its causes, not only in cases of nontraumatic vascular thrombosis but also in cases of acute vascular thrombosis related to trauma.



DOI: 10.12998/wjcc.v10.i26.9404 Copyright ©The Author(s) 2022.

Figure 3 Left adrenal hematoma seen in the initial computed tomography scan.

For the initial management of patients with suspected APS, the use of LMWH and conversion to warfarin is preferred rather than the use of direct oral anticoagulants (DOACs). This is because DOACs are less effective than warfarin for recurrent thrombosis prevention in arterial (rather than venous) thrombosis, particularly among younger patients [20,21]. Most patients with APS have a high probability of experiencing recurrent thromboembolic events. Therefore, lifelong anticoagulation therapy is recommended. Standard-intensity warfarin (target international normalized ratio range of PT, 2-3) anticoagulation is recommended for patients with venous thrombosis, while standard-intensity warfarin plus aspirin is recommended for patients with arterial thrombosis. However, standard-intensity warfarin alone can be considered for patients with arterial thrombosis that have low cardiovascular risk factors[22]. In this case, the patient had arterial thrombosis with APS, but he was a young patient with low cardiovascular risk. Therefore, he was prescribed only warfarin for his anticoagulant therapy.

This case is significant in that it suggests the importance of a hypercoagulable workup for patients with acute thromboembolism after trauma.

CONCLUSION

A hypercoagulable workup can be considered for trauma patients with acute multivascular occlusion or a hypercoagulable state, particularly for young patients with low cardiovascular risk.

FOOTNOTES

Author contributions: Lee NA, Jeong ES reviewed the literature and contributed to manuscript drafting; Jang HS analyzed and interpreted the imaging findings; Kang JH analyzed and interpreted the rheumatologic findings; Park YC, Kim JH, Jo YG were responsible for important intellectual content; all authors have read and approve the final manuscript.

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

Conflict-of-interest statement: None of the authors had conflicts of interest.

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

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Country/Territory of origin: South Korea



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S-Editor: Zhang H L-Editor: A P-Editor: Zhang H

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