## MINIREVIEWS

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>8808</td>
<td>Ear, nose, and throat manifestations of COVID-19 and its vaccines</td>
<td>Al-Ani RM</td>
</tr>
<tr>
<td>8816</td>
<td>Potential influences of religiosity and religious coping strategies on people with diabetes</td>
<td>Onyishi CN, Eseadi C, Ilechukwu LC, Okoro KN, Okolie CN, Egbule E, Asogwa E</td>
</tr>
</tbody>
</table>

## ORIGINAL ARTICLE

### Case Control Study

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
</table>

### Retrospective Study

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>8863</td>
<td>Evaluation of the prognostic nutritional index for the prognosis of Chinese patients with high/extremely high-risk prostate cancer after radical prostatectomy</td>
<td>Yang F, Pan M, Nie J, Xiao F, Zhang Y</td>
</tr>
</tbody>
</table>

### Observational Study

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>8872</td>
<td>Chlorine poisoning caused by improper mixing of household disinfectants during the COVID-19 pandemic: Case series</td>
<td>Lin GD, Wu JY, Peng XB, Lu XX, Liu ZY, Pan ZG, Qiu ZW, Dong JG</td>
</tr>
<tr>
<td>8880</td>
<td>Mental health of the Slovak population during COVID-19 pandemic: A cross-sectional survey</td>
<td>Kralova M, Brazinova A, Sivcova V, Izakova L</td>
</tr>
</tbody>
</table>
# Contents

**Prospective Study**

8893  Arthroscopic anatomical reconstruction of lateral collateral ligaments with ligament advanced reinforcement system artificial ligament for chronic ankle instability  
*Wang Y, Zhu JX*

**SYSTEMATIC REVIEWS**

8906  How to select the quantitative magnetic resonance technique for subjects with fatty liver: A systematic review  
*Li YW, Jiao Y, Chen N, Gao Q, Chen YK, Zhang YF, Wen QP, Zhang ZM*

8922  Lymphocytic choriomeningitis virus: An under-recognized congenital teratogen  
*Ferenc T, Vujica M, Mezljak A, Vilibic-Cavlek T*

**CASE REPORT**

8932  Alagille syndrome associated with total anomalous pulmonary venous connection and severe xanthomas: A case report  

8939  Colo-colonic intussusception with post-polypectomy electrocoagulation syndrome: A case report  
*Moon JY, Lee MR, Yim SK, Ha GW*

8945  Portal vein gas combined with pneumatosis intestinalis and emphysematous cystitis: A case report and literature review  
*Hu SF, Liu HB, Hao YY*

8954  Quadricuspid aortic valve and right ventricular type of myocardial bridging in an asymptomatic middle-aged woman: A case report  
*Sopek Merkaš I, Lakušić N, Paar MH*

8962  Treatment of gastric carcinoma with lymphoid stroma by immunotherapy: A case report  
*Cui YJ, Ren YY, Zhang HZ*

8968  Gallstone associated celiac trunk thromboembolisms complicated with splenic infarction: A case report  
*Wu CY, Su CC, Huang HH, Wang YT, Wang CC*

8974  Extracorporeal membrane oxygenation for lung cancer-related life-threatening hypoxia: A case report  
*Yoo SS, Lee SY, Choi SH*

8980  Multi-disciplinary treatment of maxillofacial skeletal deformities by orthognathic surgery combined with periodontal phenotype modification: A case report  
*Liu JY, Li GF, Tang Y, Yan FH, Tan BC*

8990  X-linked recessive Kallmann syndrome: A case report  
*Zhang P, Fu JY*

8998  Delayed complications of intradural cement leakage after percutaneous vertebroplasty: A case report  
*Ma QH, Liu GP, Sun Q, Li JG*
Coexistent Kaposi sarcoma and post-transplant lymphoproliferative disorder in the same lymph nodes after pediatric liver transplantation: A case report

Misdiagnosis of pancreatic metastasis from renal cell carcinoma: A case report
Liang XK, Li JI, He YM, Xu ZF

Discoid medial meniscus of both knees: A case report
Zheng ZR, Ma H, Yang F, Yuan L, Wang GD, Zhao XW, Ma LF

Simultaneous laparoscopic and arthroscopic excision of a huge juxta-articular ganglionic cyst compressing the sciatic nerve: A case report
Choi WK, Oh JS, Yoon SJ

One-stage revision arthroplasty in a patient with ochronotic arthropathy accompanied by joint infection: A case report
Wang XC, Zhang XM, Cai WL, Li Z, Ma C, Liu YH, He QL, Yan TS, Cao XW

Bladder paraganglioma after kidney transplantation: A case report
Wang L, Zhang YN, Chen GY

Total spinal anesthesia caused by lidocaine during unilateral percutaneous vertebroplasty performed under local anesthesia: A case report
Wang YF, Bian ZY, Li XX, Hu YX, Jiang L.

Ruptured splenic artery aneurysms in pregnancy and usefulness of endovascular treatment in selective patients: A case report and review of literature
Lee SH, Yang S, Park I, Im YC, Kim GY

Gastrointestinal metastasis secondary to invasive lobular carcinoma of the breast: A case report
Li LX, Zhang D, Ma F

Post-bulbar duodenal ulcer with anterior perforation with kissing ulcer and duodenocaval fistula: A case report and review of literature
Alzerwi N

Modified orthodontic treatment of substitution of canines by first premolars: A case report
Li FF, Li M, Li M, Yang X

Renal cell carcinoma presented with a rare case of icteric Stauffer syndrome: A case report
Popov DR, Antonov KA, Atanasova EG, Pentschev CP, Milatchkov LM, Petkova MD, Neykov KG, Nikolov RK

Successful resection of a huge retroperitoneal venous hemangioma: A case report
Qin Y, Qiao P, Guan X, Zeng S, Hu XP, Wang B

Malignant transformation of biliary adenofibroma combined with benign lymphadenopathy mimicking advanced liver carcinoma: A case report
<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>9112</td>
<td>Congenital hepatic cyst: Eleven case reports</td>
<td>Du CX, Lu CG, Li W, Tang WB</td>
</tr>
<tr>
<td>9121</td>
<td>Endovascular treatment of a ruptured pseudoaneurysm of the internal carotid artery in a patient with nasopharyngeal cancer: A case report</td>
<td>Park JS, Jang HG</td>
</tr>
<tr>
<td>9127</td>
<td>Varicella-zoster virus meningitis after spinal anesthesia: A case report</td>
<td>Lee YW, Yoo B, Lim YH</td>
</tr>
<tr>
<td>9132</td>
<td>Chondrosarcoma of the toe: A case report and literature review</td>
<td>Zhou LB, Zhang HC, Dong ZG, Wang CC</td>
</tr>
<tr>
<td>9142</td>
<td>Tamsulosin-induced life-threatening hypotension in a patient with spinal cord injury: A case report</td>
<td>Lee JY, Lee HS, Park SB, Lee KH</td>
</tr>
<tr>
<td>9148</td>
<td>CCNO mutation as a cause of primary ciliary dyskinesia: A case report</td>
<td>Zhang YY, Lou Y, Yan H, Tang H</td>
</tr>
<tr>
<td>9156</td>
<td>Repeated bacteremia and hepatic cyst infection lasting 3 years following pancreatoduodenectomy: A case report</td>
<td>Zhang K, Zhang HL, Guo JQ, Tu CY, Lv XL, Zhu JD</td>
</tr>
<tr>
<td>9162</td>
<td>Idiopathic cholesterol crystal embolism with atheroembolic renal disease and blue toes syndrome: A case report</td>
<td>Cheng DJ, Li L, Zheng XY, Tang SF</td>
</tr>
<tr>
<td>9168</td>
<td>Systemic lupus erythematosus with visceral varicella: A case report</td>
<td>Zhao J, Tian M</td>
</tr>
</tbody>
</table>

**LETTER TO THE EDITOR**

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>9176</td>
<td>Imaging of fibroadenoma: Be careful with imaging follow-up</td>
<td>Ece B, Aydin S</td>
</tr>
</tbody>
</table>
ABOUT COVER
Editorial Board Member of *World Journal of Clinical Cases*, Mohsen Khosravi, MD, Assistant Professor, Department of Psychiatry and Clinical Psychology, Zahedan University of Medical Sciences, Zahedan 9819713955, Iran. m.khosravi@zaums.ac.ir

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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, prospective 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 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.

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CASE REPORT

Idiopathic cholesterol crystal embolism with atheroembolic renal disease and blue toes syndrome: A case report

De-Jin Cheng, Lin Li, Xiang-Yue Zheng, Shui-Fu Tang

Abstract

BACKGROUND
Cholesterol crystal embolization (CCE) is a multisystemic and fatal disease with multiple clinical manifestations; however, there are few cases of idiopathic CCE. Here we report a patient with idiopathic CCE accompanied by atheroembolic renal disease and blue toes who had a relatively good prognosis in the short-term due to early treatment with corticosteroids and statins.

CASE SUMMARY
A 76-year-old man complained of coldness, numbness and purple color change in his left foot for 7 d. He had a feeling of fatigue, constipation, foamy urine, poor appetite and sleep. He had a lacunar infarction for 5 years and hypertension for 9 mo. Laboratory results showed elevated eosinophils, cholesterol, uric acid, serum creatinine, urea and 24 h urine analysis revealed proteinuria. A renal biopsy revealed atheroembolic renal disease. Taken together, these findings strongly supported the diagnosis of idiopathic CCE and atheroembolic renal disease.

CONCLUSION
Atheroembolic renal disease and blue toes syndrome can be caused by idiopathic CCE, and early treatment with corticosteroids is effective but requires further investigation.

Key Words: Idiopathic cholesterol crystal embolism; Atheroembolic renal disease; Blue toes syndrome; Corticosteroids; Case report; Prognosis
INTRODUCTION

Cholesterol crystal embolization (CCE) is caused by diffusion of cholesterol atheroma debris and can be triggered by intraarterial interventions and anticoagulant therapy, leading to both ischemic and inflammatory damage to the target organ[1]. However, there are only a few reports of patients with idiopathic CCE who did not receive medication or arterial intervention[2,3]. As cholesterol atheroma can occlude all types of small arteries, the disease has various clinical manifestations involving organs such as the brain, eye, kidney, gastrointestinal system and skin, which makes CCE a fatal disease with poor prognosis[4]. In a review of 221 cases of histologically proven CCE, the mortality rate was as high as 80%[5]. Here we report a patient with idiopathic CCE accompanied by atheroembolic renal disease and blue toes who had a relatively good prognosis in the short-term due to early treatment with corticosteroids and statins.

CASE PRESENTATION

Chief complaints
A 76-year-old man complained of coldness, numbness and purple color change (Figure 1A) in his left foot for 7 d. He had a feeling of fatigue, constipation, foamy urine, poor appetite and sleep, but no fever, headache or abdominal pain. His urine output was normal.

History of present illness
A 76-year-old man complained of coldness, numbness and purple color change in his left foot for 7 d. And he came to our hospital for treatments.

History of past illness
The patient had a lacunar infarction for five years and took three Fufang Xueshuantong capsules three times daily. He also had hypertension (maximum blood pressure: 189/102 mmHg) for 9 mo and took one tablet of amlodipine, atorvastatin calcium and fosinopril sodium tablets 10 mg once daily. There was no history of surgery.

Physical examination
Physical examination upon admission showed no abnormalities.

Laboratory examinations
Laboratory findings (Table 1) showed elevated eosinophils, triglycerides, serum creatinine and urea. The 24 h urine analysis showed increased proteinuria.

Imaging examinations
Doppler ultrasonography showed carotid atherosclerosis with carotid plaque formation (Figure 2A) and both kidneys were of normal size; however, kidney parenchymal thickness (approximately 13 mm) was slightly thinner (Figure 2B). Liver, spleen and pancreas were normal. A renal biopsy (Figure 3) showed glomerulus ischemic globular sclerosis in all 41 glomerulus; and mesangial cells and stroma showed slight hyperplasia in the rest of the glomerulus, one small cell fibrous crescent, and some of the glomerular capillaries were ischemic and wrinkled. With regard to the renal tubules, epithelial cells showed cavitation and granular degeneration, crystal nucleation within some renal tubules, a few renal tubules demonstrated cavity expansion, disappearance of brush border, multifocal and atrophy (shrinkage area of approximately 60%); diffuse and patchy infiltration of the kidney interstitium by lymphocytes, plasma cells and a few eosinophils; small artery wall thickening with a narrow lumen,
Table 1 Laboratory results

<table>
<thead>
<tr>
<th>Items</th>
<th>Reference value</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Eosinophil count (x 10^9/L)</td>
<td>0.05-0.3</td>
<td>3.46</td>
</tr>
<tr>
<td>Serum creatinine (mmol/L)</td>
<td>57-97</td>
<td>239</td>
</tr>
<tr>
<td>Serum urea (mmol/L)</td>
<td>3.6-9.5</td>
<td>23.32</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>2.6-5.2</td>
<td>3.90</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>0.34-1.60</td>
<td>1.63</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>&gt; 1.04</td>
<td>1.43</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>≤ 3.37</td>
<td>2.19</td>
</tr>
<tr>
<td>Urinary protein (g/24 h)</td>
<td>≤ 0.15</td>
<td>0.89</td>
</tr>
</tbody>
</table>

HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol.

Figure 1 Blue toes on the left foot. A: On admission; B: 8 mo after discharge.

Figure 2 Doppler ultrasonography. A: The red arrow shows carotid plaque formation; B: Kidney parenchymal thickness was approximately 13.3 mm.

endometrial hyperplasia, and cholesterol embolism filling several small arteries. Congo and oxidized Congo red were negative. Immunofluorescence showed no deposition of immune complexes.

**FINAL DIAGNOSIS**

The patient was diagnosed with idiopathic cholesterol crystal embolization and atheroembolic renal disease.
TREATMENT

He was treated with 30 mg of prednisone acetate tablets once daily during hospitalization and the dosage was gradually tapered after discharge. The patient also received a 20 mg tablet of atorvastatin calcium, 50 mg tablet of clopidogrel bisulfate, 10 mg tablet of fosinopril sodium and 10 mg of amlodipine once a day, respectively.

OUTCOME AND FOLLOW-UP

During the 12-mo follow-up period, the color of his left foot gradually returned to normal (Figure 1B). The gradual reduction in prednisone acetate is shown in Figure 4. Following treatment with prednisone, eosinophils and serum creatine gradually decreased after discharge (Figure 4).

DISCUSSION

CCE is a multisystemic disease with various clinical manifestations induced by atherosclerotic plaques, and these plaques are composed of platelets, fibrin, necrotic cell debris, and cholesterol crystals (CCs) [6]. Hemodynamic changes, intraplaque hemorrhage, and inflammation, which may occur spontaneously (namely idiopathic CCE) or due to invasive procedures, can induce plaque erosion and rupture that expose the plaque components to the systemic circulation. Initially, CCs only cause ischemic injury; but the subsequent inflammatory reaction aggravates end-organ injury. Endothelial injury, oxidative stress, activation of the renin-angiotensin-aldosterone system, leukocyte aggregation, complement activation, and release of leukocyte enzymes are all considered responsible for end-organ injury [1,7]. Approximately 80% of cases of CCE have been reported to be due to anticoagulation therapy and catheter manipulation, and there are few cases of idiopathic CCE [5]. The patient with definite pathological findings in this report had no history of surgery or anticoagulation therapy, which supported the diagnosis of idiopathic CCE. Male and older patients with atherosclerotic cardiovascular risk factors are more susceptible to CCE than other patients, and the overall in-hospital mortality among patients with CCE was 11% [9]. Older age, hypertension and lacunar infarction in this patient were high risk factors.
With regard to treatment, secondary prevention of cardiovascular disease is of utmost importance in these patients, such as statins, antiplatelet therapy, cessation of smoking, and control of blood pressure, weight, and glycemia. Furthermore, anti-inflammatory treatment such as corticosteroids and cyclophosphamide are alternative choices but these drugs have not been evaluated in randomized controlled trials [7]. Some studies demonstrated improved renal function with high-dose corticosteroid treatment in patients with CCE[10,11]. One patient with leg ulceration caused by CCE was reported to improve with colchicine and corticosteroids[12]. However, several studies have shown that corticosteroid therapy results in good renal outcome in CCE patients in the short-term, but does not have a favorable effect on long-term renal outcome[13]. Statins, prednisone, clopidogrel and antihypertensive agents were administered to our patient. In the 12 mo follow-up period, his renal function gradually improved and the level of eosinophils gradually decreased, which demonstrated that prednisone treatment was effective. However, the long-term effect of corticosteroid treatment and the dose reduction regimen require further investigation in randomized controlled trials.

CONCLUSION

Atheroembolic renal disease and blue toes syndrome can be caused by idiopathic CCE, and early treatment with corticosteroids is effective but requires further study.

FOOTNOTES

Author contributions: Tang SF decided the patient’s treatment plan; Cheng DJ wrote the paper; and Li L and Zheng XY collected the clinical data.

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Conflict-of-interest statement: All authors report no relevant conflicts of interest for this article.

CARE Checklist (2016) statement: The authors wrote the manuscript according to the requirements of the CARE Checklist.

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Country/Territory of origin: China

ORCID number: De-Jin Cheng 0000-0002-7407-0197; Xiang-Yue Zheng 0000-0001-8190-3022; Shui-Fu Tang 0000-0002-3622-663X.
REFERENCES


