## Contents

### MINIREVIEWS

1. Omicron variant (B.1.1.529) of SARS-CoV-2: Mutation, infectivity, transmission, and vaccine resistance  
   Ren SY, Wang WB, Gao RD, Zhou AM

12. Hepatitis B virus reactivation in rheumatoid arthritis  
   Wu YL, Ke J, Zhang BY, Zhao D

23. Paradoxical role of interleukin-33/suppressor of tumorigenicity 2 in colorectal carcinogenesis: Progress and therapeutic potential  
   Huang F, Chen WY, Ma J, He XL, Wang JW

### ORIGINAL ARTICLE

#### Case Control Study

35. Changes in rheumatoid arthritis under ultrasound before and after sinomenine injection  
   Huang YM, Zhuang Y, Tan ZM

43. Benefits of multidisciplinary collaborative care team-based nursing services in treating pressure injury wounds in cerebral infarction patients  
   Gu YH, Wang X, Sun SS

#### Retrospective Study

51. Outcomes and complications of open, laparoscopic, and hybrid giant ventral hernia repair  
   Yang S, Wang MG, Nie YS, Zhao XF, Liu J

62. Surgical resection of intradural extramedullary tumors in the atlantoaxial spine via a posterior approach  
   Meng DH, Wang JQ, Yang KX, Chen WY, Pan C, Jiang H

71. Vancomycin lavage for the incidence of acute surgical site infection following primary total hip arthroplasty and total knee arthroplasty  
   Duan MY, Zhang HZ

79. Distribution of transient receptor potential vanilloid-1 channels in gastrointestinal tract of patients with morbid obesity  
   Atas U, Erin N, Tazegul G, Elpek GO, Yildirim B

91. Value of neutrophil-lymphocyte ratio in evaluating response to percutaneous catheter drainage in patients with acute pancreatitis  
Influence of overweight and obesity on the mortality of hospitalized patients with community-acquired pneumonia

Wang N, Liu BW, Ma CM, Yan Y, Su QW, Yin FZ

Minimally invasive open reduction of greater tuberosity fractures by a modified suture bridge procedure

Kong LP, Yang JJ, Wang F, Liu FX, Yang YL

Increased levels of lactate dehydrogenase and hypertension are associated with severe illness of COVID-19


Age, alcohol, sex, and metabolic factors as risk factors for colonic diverticulosis

Yan Y, Wu JS, Pan S

Evaluation of right-to-left shunt on contrast-enhanced transcranial Doppler in patent foramen ovale-related cryptogenic stroke: Research based on imaging

Xiao L, Yan YH, Ding YF, Liu M, Kong LJ, Hu CH, Hui PJ

Characterization of focal hypermetabolic thyroid incidentaloma: An analysis with F-18 fluorodeoxyglucose positron emission tomography/computed tomography parameters

Lee H, Chung YS, Lee JH, Lee KY, Hwang KH

Clinical Trials Study

Low-dose intralesional injection of 5-fluorouracil and triamcinolone reduces tissue resident memory T cells in chronic eczema


Observational Study

Alterations in blink and masseter reflex latencies in older adults with neurocognitive disorder and/or diabetes mellitus


Predicting adolescent perfectionism: The role of socio-demographic traits, personal relationships, and media

Livazović G, Kuzmanović K

Novel m.4268T>C mutation in the mitochondrial tRNA\textsubscript{Leu} gene is associated with hearing loss in two Chinese families

Zhao LJ, Zhang ZL, Fu Y

Superior mesenteric venous thrombosis: Endovascular management and outcomes

Alnahhal K, Toskich BB, Nussbaum S, Li Z, Erben Y, Hakaim AG, Farres H

Randomized Controlled Trial

Zinc carnosine-based modified bismuth quadruple therapy vs standard triple therapy for Helicobacter pylori eradication: A randomized controlled study

Ibrahim N, El Said H, Choukair A
CASE REPORT

236  Acquired coagulation dysfunction resulting from vitamin K-dependent coagulation factor deficiency associated with rheumatoid arthritis: A case report  
Huang YJ, Han L, Li J, Chen C

242  Intraoperative thromboelastography-guided transfusion in a patient with factor XI deficiency: A case report  
Guo WJ, Chen WY, Yu X, Shen L, Huang YG

249  Positron emission tomography and magnetic resonance imaging combined with computed tomography in tumor volume delineation: A case report  
Zhou QP, Zhao YH, Gao L

254  Successful response to camrelizumab in metastatic bladder cancer: A case report  
Xie C, Yuan X, Chen SH, Liu ZY, Lu DL, Xu F, Chen ZQ, Zhong XM

260  HER2 changes to positive after neoadjuvant chemotherapy in breast cancer: A case report and literature review  
Wang L, Jiang Q, He MY, Shen P

268  Hyper-accuracy three-dimensional reconstruction as a tool for better planning of retroperitoneal liposarcoma resection: A case report  
Ye MS, Wu HK, Qin XZ, Luo F, Li Z

275  Recurrent postmenopausal bleeding - just endometrial disease or ovarian sex cord-stromal tumor? A case report  
Wang J, Yang Q, Zhang NN, Wang DD

283  Complex proximal femoral fracture in a young patient followed up for 3 years: A case report  
Li ZY, Cheng WD, Qi L, Yu SS, Jing JH

289  Bilateral Hypertrophic Olivary Degeneration after Pontine Hemorrhage: A Case Report  
Zheng B, Wang J, Huang XQ, Chen Z, Gu GF, Luo XJ

296  Clinical characteristics and outcomes of primary intracranial alveolar soft-part sarcoma: A case report  
Chen JY, Cen B, Hu F, Qiu Y, Xiao GM, Zhou JG, Zhang FC

304  Removal of laparoscopic cerclage stitches via laparotomy and rivanol-induced labour: A case report and literature review  
Na XN, Cai BS

309  Cerebral venous sinus thrombosis in pregnancy: A case report  
Zhou B, Huang SS, Huang C, Liu SY

316  Eustachian tube teratoma: A case report  
Li JY, Sun LX, Hu N, Song GS, Dou WQ, Gong RZ, Li CT
<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>331</td>
<td>Lunate dislocation with avulsed triquetral fracture: A case report</td>
<td>Li LY, Lin CJ, Ko CY</td>
</tr>
<tr>
<td>361</td>
<td>Diagnostic and surgical challenges of progressive neck and upper back painless masses in Madelung’s disease: A case report and review of literature</td>
<td>Yan YJ, Zhou SQ, Li CQ, Ruan Y</td>
</tr>
<tr>
<td>371</td>
<td>Suspected cerebrovascular air embolism during endoscopic esophageal varices ligation under sedation with fatal outcome: A case report</td>
<td>Zhang CMJ, Wang X</td>
</tr>
<tr>
<td>381</td>
<td>An atypical primary malignant melanoma arising from the cervical nerve root: A case report and review of literature</td>
<td>Shi YF, Chen YQ, Chen HF, Hu X</td>
</tr>
<tr>
<td>388</td>
<td>Epidural blood patch for spontaneous intracranial hypotension with subdural hematoma: A case report and review of literature</td>
<td>Choi SH, Lee YY, Kim WJ</td>
</tr>
</tbody>
</table>
ABOUT COVER
Editorial Board Member of World Journal of Clinical Cases, Ravi Kant, MD, Associate Professor, Division of Endocrinology, Diabetes and Metabolism, Medical University of South Carolina/Anmed Campus, Anderson, SC 29621, United States. rkant82@hotmail.com

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 Science Citation Index Expanded (also known as SciSearch®), Journal Citation Reports/Science Edition, Scopus, PubMed, and PubMed Central. The 2021 Edition of Journal Citation Reports® cites the 2020 impact factor (IF) for WJCC as 1.337; IF without journal self cites: 1.301; 5-year IF: 1.742; Journal Citation Indicator: 0.33; Ranking: 119 among 169 journals in medicine, general and internal; and Quartile category: Q3. The WJCC's CiteScore for 2020 is 0.8 and Scopus CiteScore rank 2020: General Medicine is 493/793.

RESPONSIBLE EDITORS FOR THIS ISSUE
Production Editor: Lin-YuTong Wang; Production Department Director: Xiang Li; Editorial Office Director: Jin-Lei Wang.
Acquired coagulation dysfunction resulting from vitamin K-dependent coagulation factor deficiency associated with rheumatoid arthritis: A case report

Yan-Jing Huang, Liang Han, Jing Li, Chao Chen

BACKGROUND
Rheumatoid arthritis (RA) is a common chronic inflammatory autoimmune disease with the main clinical feature of progressive joint synovial inflammation, which can lead to joint deformities as well as disability. RA often causes damage to multiple organs and systems within the body, including the blood hemostasis system. Few reports have focused on acquired coagulation dysfunction resulting from vitamin K-dependent coagulation factor deficiency associated with RA.

CASE SUMMARY
A 64-year-old woman with a history of RA presented to our hospital, complaining of painless gross hematuria for 2 wk. Blood coagulation function tests showed increased prothrombin time, international normalized ratio, and activated partial thromboplastin time. Abnormal blood coagulation factor (F) activity was detected (FII, 7.0%; FV, 122.0%; and FX, 6.0%), indicating vitamin K-dependent coagulation factor deficiency. Thromboelastography and an activated partial thromboplastin time mixed correction experiment also suggested decreased coagulation factor activity. Clinically, the patient was initially diagnosed with hematuria, RA, and vitamin K-dependent coagulation factor deficiency. The patient received daily intravenous administration of vitamin K1 20 mg, etamsylate 3 g, and vitamin C 3000 mg for 10 d. Concurrently, oral leflunomide tablets and prednisone were...
administered for treatment of RA. After the treatment, the patient's symptoms improved markedly and she was discharged on day 12. There were no hemorrhagic events during 18 mo of follow-up.

**CONCLUSION**

RA can result in vitamin K-dependent coagulation factor deficiency, which leads to acquired coagulation dysfunction. Vitamin K1 supplementation has an obvious effect on coagulation dysfunction under these circumstances.

**Key Words:** Acquired coagulation dysfunction; Rheumatoid arthritis; Coagulation factor deficiency; Vitamin K-dependent; Case report

©The Author(s) 2022. Published by Baishideng Publishing Group Inc. All rights reserved.

| Core Tip: Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune disease that frequently involves multiple organs and systems, potentially leading to coagulation dysfunction. In this paper, we report the rare case of a patient who was diagnosed with acquired coagulation dysfunction resulting from vitamin K-dependent coagulation factor deficiency associated with RA, and subsequently benefited from vitamin K1 supplementation treatment. This case report may provide some references for diagnosis and treatment of RA patients with coagulation dysfunction symptoms. |

**INTRODUCTION**

Rheumatoid arthritis (RA) is a systemic autoimmune disease with the main clinical manifestations of invasive arthritis[1]. The prevalence of RA is estimated to be 0.5%–1.0% globally[2]. The clinical features of blood system damage in patients with RA usually include anemia, neutropenia, thrombocytopenia, and hematological malignancies[3]. RA can also lead to acquired coagulation dysfunction, such as acquired hemophilia. Previous studies indicated that 4%–8% of acquired hemophilia cases were related to RA and that rituximab was effective for acquired FVIII inhibitors in RA patients[4,5]. Compared with healthy people, Dimitroulas et al[6] found that RA patients had higher levels of coagulation factors, such as tissue plasminogen activator, plasminogen activator inhibitor, fibrinogen (FBG), prothrombin fragments 1 and 2, and thrombomodulin, indicating that an imbalance of the coagulation and fibrinolysis systems was common in RA, although the underlying mechanism is not fully understood. Nevertheless, cases of acquired coagulation dysfunction caused by RA combined with vitamin K-dependent coagulation factors deficiency are rare. To further explore the possible etiology of coagulopathy in RA patients, we report a case of acquired coagulation dysfunction resulting from vitamin K-dependent coagulation factor deficiency associated with RA.

**CASE PRESENTATION**

**Chief complaints**

A 64-year-old female patient was admitted to the hospital on November 27, 2019, with a chief complaint of “painless gross hematuria for 2 wk”.

**History of present illness**

Two weeks previously, the patient had developed painless gross hematuria with no obvious cause. She presented with a whole course of hematuria, including blood clots,
infrquent urination, urgent urination, and urodynia, without pain in the waist or lower abdomen, nausea, or vomiting. At the beginning, the patient was treated at The Fifth People’s Hospital of Jingzhou City, Hubei Province, China. A computed tomography scan of the urinary system displayed a soft tissue density in her bladder. A cystoscopy was performed and intravesical blood clots were subsequently removed on November 19, 2019. However, the gross hematuria symptoms recurred after the treatment. Hence, the patient attended our hospital for medical treatment. She was admitted for hematuria of unknown etiology.

**History of past illness**

The patient had a history of RA for 40 years, with regular treatment of oral indomethacin and prednisone tablets. At the time of admission, the patient had suffered joint pain and stiffness symptoms for several months.

**Physical examination**

The patient had suffered joint pain and stiffness symptoms.

**Laboratory examinations**

After admission, laboratory tests revealed the following results: Prothrombin time (PT), 38.6 s; international normalized ratio (INR), 3.97; FBG, 4.85 g/L; activated partial thromboplastin time (APTT), 109.8 s; thrombin time, 16.9 s; and D-dimer, 0.89 µg/mL FEU. The results for blood coagulation factor (F) activity were as follows: FII, 7.0%; FV, 122.0%; and FX, 6.0%. Thus, vitamin K-dependent coagulation factor deficiency was considered. Thromboelastography produced the following findings: R, 10.2 min; K, 3.6 min; angle, 57.4; MA, 76.1 mm; CI, -2.1; and LY30, 0.0%. Routine urine examination showed red blood cells (occult blood), 3+; white blood cells (granular), 3+; nitrite-positive urinary protein, 3+; specific gravity > 1.030; urine glucose, ±; ketonuria, 2+; and urinary bilirubin, 3+. An APTT mixed correction experiment was performed on December 4. The patient’s APTT was 55.7 s. Her blood was then mixed with blood from a normal patient in a 1:1 ratio, and the APTT was determined immediately after mixing. The results showed that the mixed blood APTT was 41.4 s (normal control APTT, 37.3 s), and the Rosner index was 7.4 KUA/L, indicating a lack of coagulation factors. The level of rheumatoid factor (RF) was 35.4 IU/mL, RF IgG type was 25.87 RU/mL, RF IgM type was 103.81 RU/mL, and anti-cyclic citrulline polypeptide antibody was 48.4 U/mL. Antinuclear antibodies showed nuclear homogeneous type (1:100), suggesting the presence of trace amounts of antinuclear antibodies. Anti-neutrophil cytoplasmic antibody was positive for perinuclear type. C-reactive protein was 34.2 mg/L, indicating the possibility of infection or inflammation. The disease activity score in 28 joints was 3.7, indicating moderate disease activity.

Routine blood tests showed the following results: Red blood cells, 3.35 × 10¹²/L; hemoglobin, 86.0 g/L; white blood cells, 5.6 × 10⁹/L; neutrophils, 3.42 × 10⁹/L; lymphocytes 1.49 × 10¹²/L; and platelets, 343.0 × 10⁹/L. Liver function tests were: Alanine aminotransferase, < 5 U/L; glutamic oxaloacetic transaminase, 19 U/L; total protein, 62.1 g/L; albumin, 28.8 g/L; globulin, 33.3 g/L; total bilirubin, 5.5 µmol/L; direct bilirubin, 2.9 µmol/L; indirect bilirubin, 2.6 µmol/L; alkaline phosphatase, 96 U/L; γ-glutamyl transpeptidase, 13 U/L; total cholesterol, 3.69 mmol/L; and lactic dehydrogenase, 244 U/L. Other examinations did not show any obviously abnormal values, suggesting that no other disease condition was present.

**FINAL DIAGNOSIS**

The final diagnosis was hematuria, acquired coagulation dysfunction, RA, and vitamin K-dependent coagulation factor deficiency.

**TREATMENT**

The patient was given intramuscular injection of vitamin K1 (10 mg) on day 1 after admission. From November 29 to December 8, she received daily intravenous administration of 20 mg of vitamin K1, 5 g of etamsylate, and 3000 mg of vitamin C. Oral leflunomide tablets and prednisone were administered for treatment of RA.
Table 1 Blood coagulation function test results

<table>
<thead>
<tr>
<th>Hospitalization days</th>
<th>PT</th>
<th>INR</th>
<th>FBG</th>
<th>APTT</th>
<th>TT</th>
<th>D-D</th>
<th>FDPs</th>
<th>AT</th>
<th>PTA</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>38.6</td>
<td>3.97</td>
<td>4.85</td>
<td>109.8</td>
<td>16.9</td>
<td>0.89</td>
<td>-</td>
<td>-</td>
<td>19</td>
</tr>
<tr>
<td>4</td>
<td>18.4</td>
<td>1.53</td>
<td>5.09</td>
<td>53.9</td>
<td>16.4</td>
<td>0.94</td>
<td>&gt; 4.0</td>
<td>80</td>
<td>54</td>
</tr>
<tr>
<td>8</td>
<td>16.9</td>
<td>1.37</td>
<td>4.21</td>
<td>55.7</td>
<td>17.3</td>
<td>0.81</td>
<td>&gt; 4.0</td>
<td>67</td>
<td>62</td>
</tr>
<tr>
<td>11</td>
<td>16.4</td>
<td>1.31</td>
<td>4.82</td>
<td>55.5</td>
<td>16.7</td>
<td>1.28</td>
<td>4.2</td>
<td>75</td>
<td>65</td>
</tr>
</tbody>
</table>

Normal reference range: Prothrombin time: 11.5-14.5 s; international normalized ratio: 0.5-1.2; fibrinogen: 2.0-4.0 g/L; activated partial thromboplastin time: 29-42 s; thrombin time: 14-19 s; D-Dimer: < 0.5 μg/mL FEU; FDPs: < 0.5 μg/mL; AT: 80%-120%; PTA: 75%-125%. Hospitalization day 1 was November 27, 2019; day 2: November 28, 2019; day 4: November 30, 2019; day 8: December 4, 2019; day 11: December 7, 2019. PT: Prothrombin time; INR: International normalized ratio; FBG: Fibrinogen; APTT: Activated partial thromboplastin time; TT: Thrombin time; D-D: D-dimer; FDPs: Fibrin degradation products; AT: Antithrombin; PTA: Prothrombin activity.

OUTCOME AND FOLLOW-UP

During the treatment period, the results of blood coagulation function tests (Table 1), blood coagulation factor activity test (Table 2) and routine urine tests (Table 3) were reviewed. PT, INR, FBG, and particularly APTT were significantly decreased (Table 1), and a trend toward gradual recovery of coagulation function was observed (Figure 1). The results for blood coagulation factor activity are shown in Table 2; FV activity returned to normal, while FII and FX activities did not return to their normal ranges, but did show significant increases compared with the previous results. A routine urine test on December 5 showed: Red blood cells (occult blood), 3+; white blood cells (granular), 1+; urinary protein, 1+; and specific gravity, 1.009. Other indicators were all normal, and the patient’s hematuria symptoms had improved considerably. On December 7, the patient’s urine color returned to normal. The marked improvement in the patient’s symptoms continued, and she was discharged from hospital. Besides anti-inflammatory therapy, she continued receiving vitamin K1 orally for 3 mo after discharge. The patient was followed for 18 mo, without any hemorrhagic events.

DISCUSSION

Acquired coagulation dysfunction has a complicated etiology, and can arise secondary to liver diseases, vitamin K-dependent coagulation factor deficiency, pregnancy, neoplastic diseases, autoimmune diseases, and use of certain drugs. The present patient had no family history of hemophilia or severe bleeding tendency during the previous 60 years, or of serious bleeding during pregnancy and delivery. In the previous 2 years, in addition to oral indomethacin and prednisone tablets for treatment of RA, she was prescribed nifedipine sustained-release tablets and insulin to control blood pressure and blood glucose, respectively, with no other suspicious drug usages or toxic exposures. After combining the medical history and other laboratory examinations, the possibility of liver disease or neoplastic disease was ruled out.

The diagnosis was made on the basis of the following clinical features. First, hematuria was the main clinical symptom. Second, increased PT, APTT, and INR indicated coagulation dysfunction. Third, prolongation of the R parameter on thromboelastography suggested that coagulation factor activity was decreased. Analyses revealed that FII was 7.0% and FX was 6.0%, indicating possible vitamin K-dependent coagulation factor deficiency. The Rosner index of 7.4 (< 11) in the APTT mixed correction experiment also suggested a lack of coagulation factors. Finally, after treatment with vitamin K1, the results of routine urine analysis, coagulation function, and coagulation factor activity were significantly improved, and the urine color returned to normal. Therefore, the coagulation dysfunction in this patient may have been due to vitamin K-dependent coagulation factor deficiency, which is usually caused by a lack of vitamin K.

Vitamin K is a coenzyme for many γ-glutamyl carboxylase enzymes[7,8]. When vitamin K is lacking, the γ-glutamyl carboxylases lose their biological activity and ability to synthesize vitamin K-dependent coagulation factors, leading to the disorder of the coagulation function. A previous study found that the serum levels of vitamin K1, menaquinone-4, and menaquinone-7 in patients with RA were significantly lower than those in healthy people[9]. Therefore, RA may be the direct cause of the vitamin...
K-dependent coagulation factor deficiency, which in turn caused the coagulation dysfunction in the present patient.

Vitamin K supplementation is the main therapeutic measure for treatment of vitamin K-dependent coagulation factor deficiency[10]. Under such circumstances, patients showed improved coagulation factor activity, PT, APTT, and bleeding symptoms after treatment with vitamin K1[7]. In the present case, the patient’s symptoms improved markedly after intravenous administration of vitamin K1, etamsylate, and vitamin C.

The relationship between RA and vitamin K-dependent coagulation factor deficiency has rarely been reported. The present case report provides some references for diagnosis and treatment of RA patients with coagulation dysfunction symptoms. Clinicians should consider investigating vitamin K deficiency in such RA cases. It remains unclear whether the vitamin K-dependent coagulation factor deficiency caused by RA was accidental, or whether there was an internal relationship with autoimmunity. The mechanism for how RA can cause vitamin K deficiency requires further elucidation.

**Table 2 Blood coagulation factor activity test results**

<table>
<thead>
<tr>
<th>Hospitalization days</th>
<th>II</th>
<th>V</th>
<th>VII</th>
<th>VIII</th>
<th>IX</th>
<th>X</th>
<th>XI</th>
<th>XII</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>7</td>
<td>122</td>
<td>-</td>
<td>-</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>36</td>
<td>96</td>
<td>95</td>
<td>168</td>
<td>71</td>
<td>25</td>
<td>86</td>
<td>40</td>
</tr>
</tbody>
</table>

Normal reference range: II: 70%-120%; V: 70%-120%; VII: 55%-170%; VIII: 60%-150%; IX: 60%-150%; X: 70%-120%; XI: 60%-150%; XII: 50%-150%.

**Hospitalization day 1: November 27, 2019; day 2: November 28, 2019; day 9: December 5, 2019.**

**Table 3 Routine urine test results**

<table>
<thead>
<tr>
<th>Hospitalization days</th>
<th>RBCs</th>
<th>WBCs</th>
<th>Urinary protein</th>
<th>Nitrite</th>
<th>Specific gravity</th>
<th>Ketonuria</th>
<th>Urinary bilirubin</th>
<th>Urine glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>3+</td>
<td>3+</td>
<td>3+</td>
<td>Positive</td>
<td>&gt; 1.030</td>
<td>2+</td>
<td>3+</td>
<td>±</td>
</tr>
<tr>
<td>9</td>
<td>3+</td>
<td>1+</td>
<td>1+</td>
<td>Negative</td>
<td>&lt; 1.009</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Normal reference range: Red blood cells: Negative; white blood cells: Negative; urinary protein: Negative; nitrite: Negative; specific gravity: 1.015-1.025; ketonuria: Negative; urinary bilirubin: Negative; urine glucose: Negative. Hospitalization day 1: November 27, 2019; day 2: November 29, 2019; day 9: December 5, 2019. RBCs: Red blood cells; WBCs: White blood cells.

**Figure 1 Results for prothrombin time, activated partial thromboplastin time, fibrinogen, and international normalized ratio.** The results showed that the blood coagulation function was gradually recovering. The hospitalization days were: Day 1, November 27, 2019; day 2: November 28, 2019; day 4: November 30, 2019; day 8: December 4, 2019; and day 11: December 7, 2019. PT: Prothrombin time; APTT: Activated partial thromboplastin time; INR: International normalized ratio; FBG: Fibrinogen.
CONCLUSION

RA can result in vitamin K-dependent coagulation factor deficiency, which leads to acquired coagulation dysfunction. Vitamin K1 supplementation has an obvious effect on coagulation dysfunction under these circumstances.

REFERENCES
