Case Report
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대한영상의학회지

Bilateral Ureteral Calcifications Associated with Systemic Lupus Erythematosus: A Case Report[1]

전신 홍반성 루푸스에서 발생한 양측 요관 석회화: 증례 보고[1]

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A 23-year-old woman with systematic lupus erythematosus (SLE) presented with left flank pain and hematuria, after seven months of hospital treatment with prednisolone. Computed tomography (CT) of abdomen showed multifocal calcifications of bilateral ureteral walls and hydronephrosis by left ureteral stenosis. These calcifications may be associated with lupus-induced inflammatory reaction of ureteral wall. In this study, we also present review of the literature associated with the similar cases.

Index terms
Systemic Lupus Erythematosus
Ureter
Calcification

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease which can involve any organs and tissues, resulting in inflammation and tissue damage. Calcifications in SLE can occasionally occur in various parts of body, but calcifications of bilateral ureters are rarely found in literature (1-4). Here, we report a case of a 23-year-old woman with calcifications in both ureteral walls confirmed by computed tomography (CT). Although ureteral calcification in SLE is a very rare event, the clinician should be aware of this manifestation.

CASE REPORT

A 23-year-old female patient visited our hospital due to left flank pain and hematuria which she had for one week. Two years ago, she was admitted to our hospital with a history of painful swelling of both proximal interphalangeal joints. She was diagnosed as SLE and was treated as an outpatient with prednisolone daily. Afterward, she was referred to obstetric department for pregnancy, where she successfully delivered a baby. On the admission, laboratory examinations showed that the peripheral blood white cell count was 5.83 × 10^9/L (66.6% for neutrophils and 25.2% for lymphocytes) and red blood cell count was 3.12 × 10^{12}/L. Also, a large amount of red blood cells were found on a routine urinalysis. Subsequently, CT was performed for further evaluation, and multifocal calcifications with central lucency along both ureteral walls accompanied by dilatation of left renal pelvis were shown on a non-contrast image (Fig. 1A-C). On blood laboratory testing, parathyroid hormone was slightly high (77.8 pg/mL), but serum calcium, phosphate, and creatinine levels were normal. After the examinations, we excluded other causes of ureteral wall calcifications, so we considered that these calcifications may be related to lupus-induced inflammation. The ureteroscopy and biopsy were performed, and they confirmed the same diagnosis (Fig. 1D). She underwent ureteroscopic lithotripsy at the same time, and this could not completely remove calcifications of both ureteral walls. Then, double J

catheters were inserted at both ureters, and left flank pain was improved. The follow-up CT scan was performed 6 months later, after double J catheter removal and standard medical treatment, but multifocal calcifications of the left ureteral wall were slightly aggravated (Fig. 1E). Finally, she underwent left percutaneous nephrostomy in our hospital. During one year follow-up, her condition was stable without any urinary symptoms.

DISCUSSION

The common causes of ureteral calcifications are inflammation by tuberculosis and schistosomiasis. Rarely, amyloid infiltration or ureter tumors can also cause ureteral calcification (5-8).

In our case, there is central lucency in the both ureteral calcifications. It is quite evident that the location of calcification was ureteral wall. She had an upper normal range of parathyroid hormone, but normal serum levels of calcium and phosphorus and other causes of ureteral calcification were also excluded.

SLE is an autoimmune disease that is characterized by inflammation, immune complex deposition, vasculitis, and vasculopathy. Ectopic calcification in SLE patient is an unusual complication. The calcification in SLE patients mostly involves the soft tissue, which is mostly shown as calcification in the subcutaneous tissue (2). Calcification in SLE can also be occasionally found in various parts of body, such as joint, aortic valve, cardiac valves, spleen, parotid gland, and pancreas. The pathogenesis of these ectopic calcifications has not yet been confirmed, but inflammation is thought to be one of causes of the development.

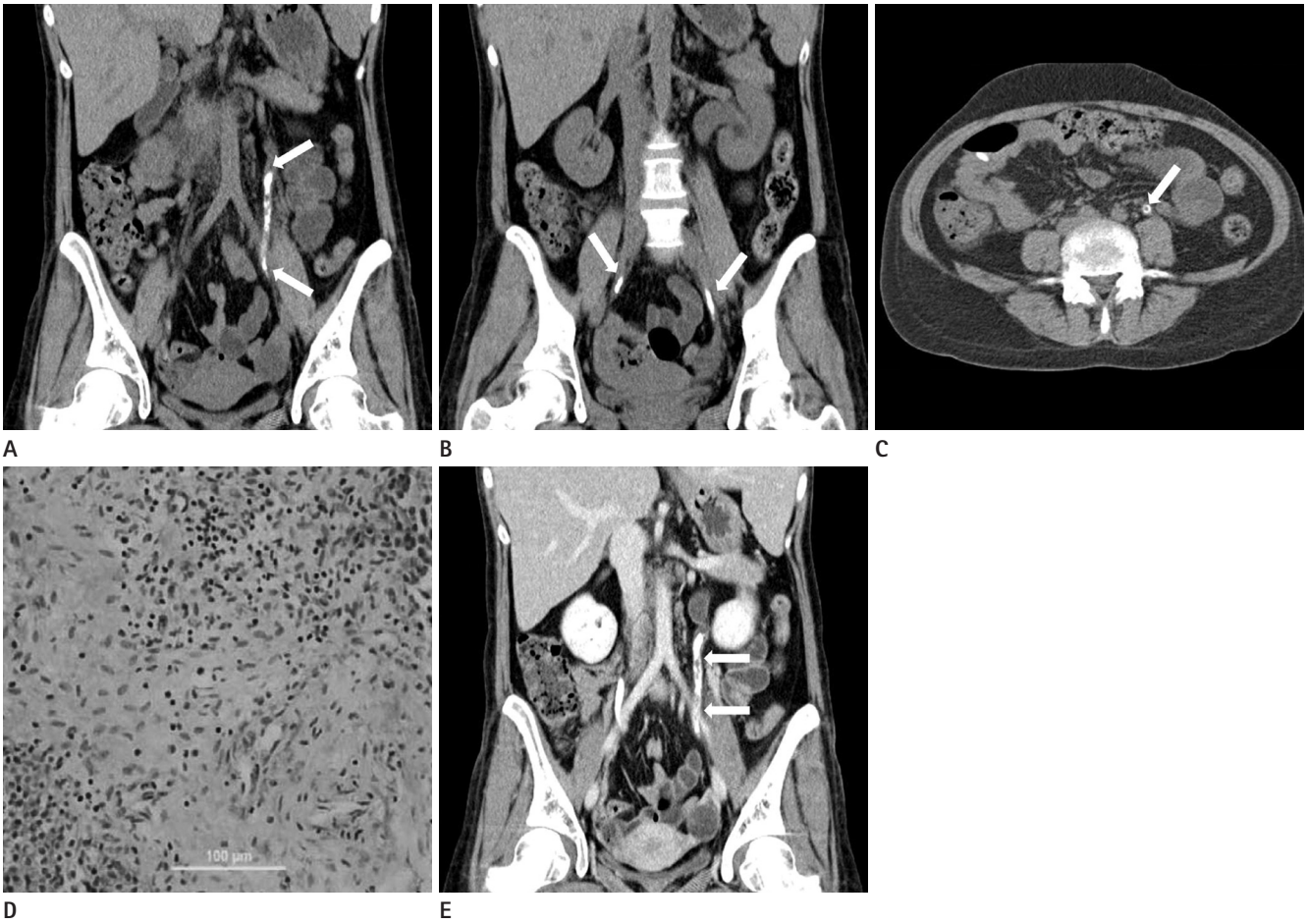

Fig. 1. A 23-year-old female patient with systematic lupus erythematosus.
A–C. Non-contrast coronal CT scans **(A, B)** show multifocal calcifications (arrows) in both ureters. Non-contrast axial CT scan **(C)** shows calcifications with central lucency (arrow) in left ureter, consistent with calcifications of ureteral wall.
D. Microscopy shows edema and mixed inflammatory cells infiltration of the lamina propria mainly consisting of lymphocytes (H&E, × 100 original magnification). The calcifications are also noted at lamina propria of ureteral wall (not shown).
E. Follow-up coronal CT scan after 6 months later shows slight aggravation of multifocal calcifications of left ureteral wall (arrows).

Jiang et al. (1) reported the case of bilateral ureteral calcifications in SLE patient, which was similar to our case. They speculated that the calcifications of both ureteral walls may be associated with lupus-induced inflammatory involvement of ureteral tissue. Brain parenchyma calcification has also been reported, and the possible mechanism has been suggested that the immunological vascular injury and thrombosis, which are mediated by antibodies or immune complexes, cause multiple infarcts with subsequent vascular calcification in vulnerable regions. Masuda et al. (9) observed symmetrical progressive intracranial calcifications in SLE and speculated that it was caused by hypoxic vulnerability in specific areas due to microvascular impairment for vasculitis and thrombosis. Okada et al. (10) suggested that the active vitamin D3 therapy could increase the risk of ectopic calcification in SLE, especially in patients with lupus nephritis or low total protein levels. Even in patients with low-grade or moderate renal dysfunction, subclinical hyperparathyroidism has been observed, and these can cause abnormal ectopic calcification in SLE.

In our case, the most probable mechanism for calcifications of both ureteral walls was SLE related calcification. Several case reports have documented ureteral involvement in SLE, usually in the form of strictures, and they have postulated that vasculitic involvement of periureteral vessels lead to ischemia and necrosis (4). The soft tissue dystrophic calcification occurs in the areas of tissue necrosis. Additionally, the vitamin D therapy could contribute to both ureteral calcifications in SLE patients, especially in patients with lupus nephritis or low total protein levels (10). In our case, the patient had a history of receiving multivitamin therapy before and after delivery, which included vitamin D. Her parathyroid hormone was in the upper normal level, and her blood total protein was slightly low. This blood laboratory test showed probability of subclinical hyperparathyroidism due to low grade renal dysfunction.

In conclusion, if symptoms of flank pain or renal colic are present in patients of SLE, ureteral involvement of SLE can be one of causes of ureteral calcifications, in addition to tuberculosis and schistosomiasis. More researches are needed on the cause of ureteral calcification in SLE patients.

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전신 홍반성 루푸스에서 발생한 양측 요관 석회화: 증례 보고[1]

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전신 홍반성 루푸스를 앓고 있는 23세 여자 환자가 옆구리 통증과 혈뇨를 주소로 내원하였다. 그녀는 전신 홍반성 루푸스로 인해 7개월간 프래드니솔론 치료를 받은 기왕력이 있었다. 복부 전산화단층촬영 소견상 양측 요관 벽에 다원성 석회화가 관찰되었고, 좌측 요관 협착으로 인한 수신증이 확인되었다. 이 석회화는 요관 내막의 루푸스로 인한 염증반응과 연관된 석회화로 생각된다. 이에 문헌 고찰과 함께 증례를 보고하는 바이다.

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