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Systemic Lupus Erythematosus with Obstructive Uropathy
— Case report and review —

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We report a case of patient with documented SLE who displayed dysuria, gastrointestinal (GI) symptoms and renal insufficiency associated with the unusual occurrence of bilateral hydroureteronephrosis due to ureterovesical junction stricture (obstructive uropathy). Pathologic investigations disclosed chronic interstitial cystitis (IC) with evidence of focal immune complex deposition in the blood vessel walls of the bladder. The GI symptoms and dysuria regressed with initial therapy for SLE with steroids. However, the persistent obstructive uropathy (OU) and renal insufficiency required bilateral nephrostomy followed by steroids plus intravenous pulse injection of cyclophosphamide. The obstructive uropathy was relieved even after removing the nephrostomy tube and renal function remained stable. Including this case, nineteen SLE patients associated with clinical and radiographic findings of OU were found in the world literature and reviewed to find any consistent pattern of clinical features. Most of the patients with OU in SLE were female (mean age, 31.7 yr) and orientals (63 %), and had interstitial cystitis (89 %) as a common underlying cause with concomitant involvement of the GI tract (89 %) and WHO class IV or V advanced glomerulonephritis (67 %). Despite the remarkable response (68 %) to steroids in majority of OU patients associated with SLE, certain patients still required surgical correction (32 %) and some even died (32 %). OU, potentially reversible, was not an exception in patients with SLE, which might be overshadowed by other major organ involvement of SLE.

Key Words : *Obstructive uropathy, Systemic lupus erythematosus, Chronic interstitial cystitis*

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease entity with multiorgan involvement. The involvement of the genitourinary tract has been traditionally represented by glomerulonephritis, and extra-renal involvement of the genitourinary tract was considered an exception to the rule that SLE can affect any organ system. Indeed, there has been little mention of lower urinary tract involvement in SLE from major clinical reviews of the topic (Harvey et al., 1954 ; Dubois, 1974 ; Fries and Holman, 1976 ; Ropes, 1976).

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However, since bladder involvement with interstitial cystitis (IC) in a patient with SLE was first reported by Shipton (1965), subsequently, many similar scattered reports followed and obstructive uropathy (OU) such as hydroureteronephrosis was shown on radiographic findings in the patients with contracted bladder and IC in SLE as a primary manifestation of SLE (De La Serna et al., 1981 ; Weisman et al, 1981).

Two recently reported large series regarding the association between bladder involvement due to IC and SLE revealed the lack of description of the nature or clear incidence of frank OU such as hydronephrosis in patients with SLE (Moriuchi et al., 1989 ; Meulders et al, 1992). Moreover, the occurrence of OU with extra-bladder involvement would not be a particular event in view of the multisystem-involving nature of SLE.

These considerations prompted us to review all the reported cases in the literature with special reference to the clinical features of OU associated with SLE. Also, the present study included our experience of OU due to IC in a middle aged female patient with a documented history of SLE.

CASE REPORT

A 35 year old Korean woman was first evaluated at Hanyang University Hospital, Seoul, Korea in September 1991 because of arthralgia, recurrent fever and anemia of 1 month duration. Laboratory studies revealed a hemoglobin level of 10.2 g/dl and an ESR of 65 mm/hr. Urinalysis showed numerous red blood cells, white blood cells under high power field and 2 (+) proteinuria. BUN 18 mg/dL, creatinine 1.0 mg/dL, creatinine clearance (Ccr) 90ml/min and a 24-hour urinary protein of 1100 mg were shown. Immunologic studies revealed a (+) ANA (titer 1 : 1240) with a speckled pattern, a positive direct Coomb's test, high levels of antibody to double-stranded DNA (20 unit/ml), depressed total complement (CH50) of 63 U/ml with low levels of C_4 (23 mg/dL) and C_3 (38 mg/dL), a negative reaction to both VDRL and rheumatoid factor. SLE was diagnosed according to the revised ARA criteria for the classification of SLE (Tan et al., 1982). A percutaneous renal biopsy revealed a membranous glomerulonephritis, class V by WHO classification of lupus nephritis. Prednisolone, 50 mg daily was initiated with rapidly resolving systemic symptoms of SLE but a further treatment plan could not be performed due to loss of follow-up in the out patient clinic with very poor compliance to medications.

Three years later in October 1994, she was admitted with complaints of suprapubic pain, dysuria, frequency of small volume (20—30 times a day), urgency, and nocturia of 1 month duration. Simultaneously she developed diarrhea, abdominal pain and general malaise. At the time of admission the patient's Ccr and 24-hr protein excretion were 12 ml/min and 1500 mg, respectively. Serum creatinine was 4.2 mg/dL with BUN of 68 mg/dL. ESR was accelerated to 87 mm/hr. The hemoglobin was 8.6 g/dL with a postive Coomb's test. Urinalysis revealed many WBC/HPF, 5—10 RBC/HPF, 2 (+) proteinuria and no bacteria or casts. Repeated urine cultures were negative.

Abdominal X-ray films demonstrated dilatations and fluid accumulations in the small and large bowels which were compatible with paralytic ileus. An excretory urogram showed no nephrogram. An ultrasonogram revealed grade 4 hydronephrosis and hydroureter (Fig. 1A). Antegrade pyelography after nephrostomy tube insertion was performed 14 days after admission and showed bilateral hydroureteronephrosis with non-visualized bladder (Fig. 1B). Cystoscopy revealed a bladder capacity of 25 ml with a contracted thickened bladder wall, diffuse hyperemia,

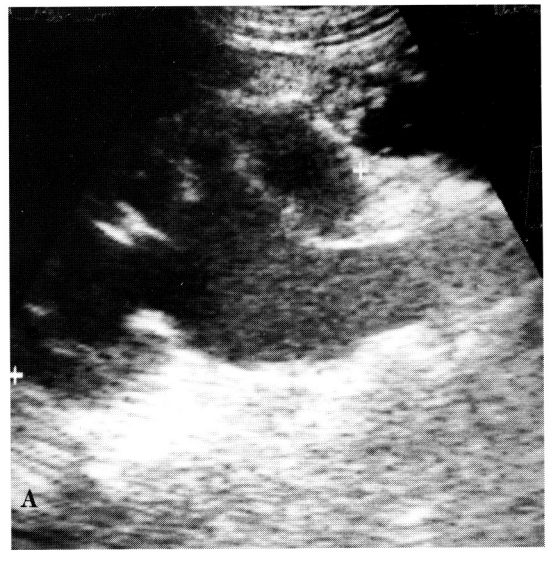

Fig. 1A. Ultrasonogram showed hydroureteronephrosis (grade 4) of the right kidney. A similar finding was seen in the left kidney (not shown) showing bilateral involvement of obstructive uropathy.

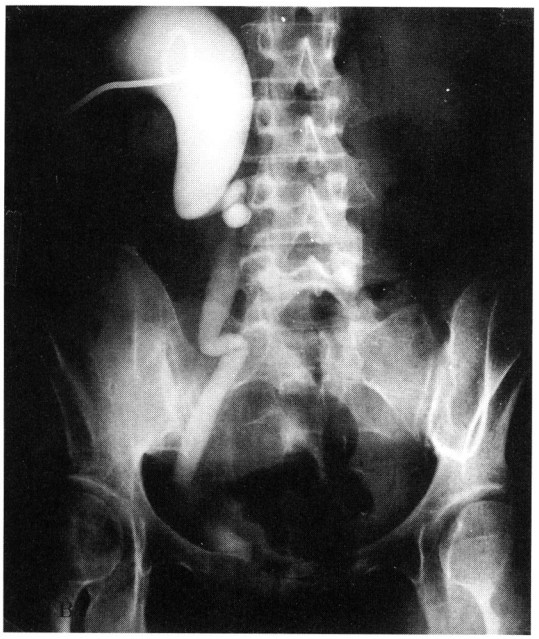

Fig. 1B. Antegrade pyelography through right percutaneous nephrostomy revealedhydroureteronephrosis with obstruction at the ureterovesical junction. Left antegrade pyelography showed similar findings (not shown).

edema, and friability of mucosa without visualization of both ureteral orifices. Voiding cystourethrogram showed a small and contracted bladder with thickened and irregular walls and diverticula of variable sizes, but no vesicoureteral reflux was demonstrated (Fig. 2). The biopsy of the bladder wall revealed changes of interstitial cystitis disclosing diffuse edema of the submucosal tissue, hypervascularity and focal perivascular infiltrations of lymphocytes, plasma cells and some neutrophils (Fig. 3A and B). Immunofluorescent studies revealed focal deposits of IgG, IgM, IgA, C3, C1 and fibrinogen in a granular pattern on small blood vessel wall or the lamina propria of bladder (Fig. 4). No staining in the basement membrane zone was present.

In addition to antispasmodics and antidiarrheal agents, prednisolone was started with a daily dose of 60 mg from the first day of admission. Within 7 days the diarrhea stopped, and abdominal pain disappeared but renal insufficiency and urinary frequency persisted despite medications. Renal insufficiency was rapidly and completely relieved by bilateral percutaneous nephrostomy. Cyclophosphamide was then

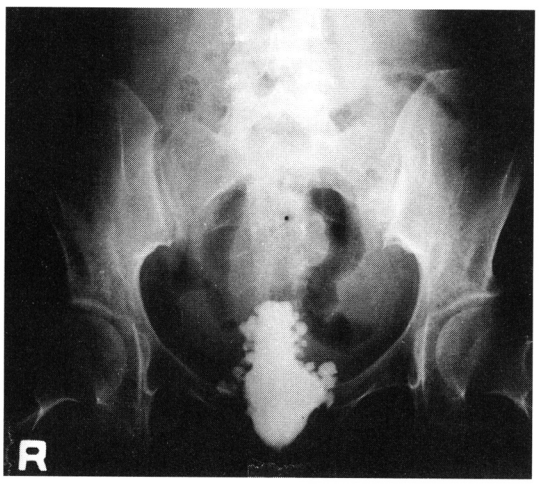

Fig. 2. A voiding cystourethrogram revealed a small and contracted bladder with thickened and irregular walls and diverticula of variable size and no evidence of vesicoureteral reflux on either side.

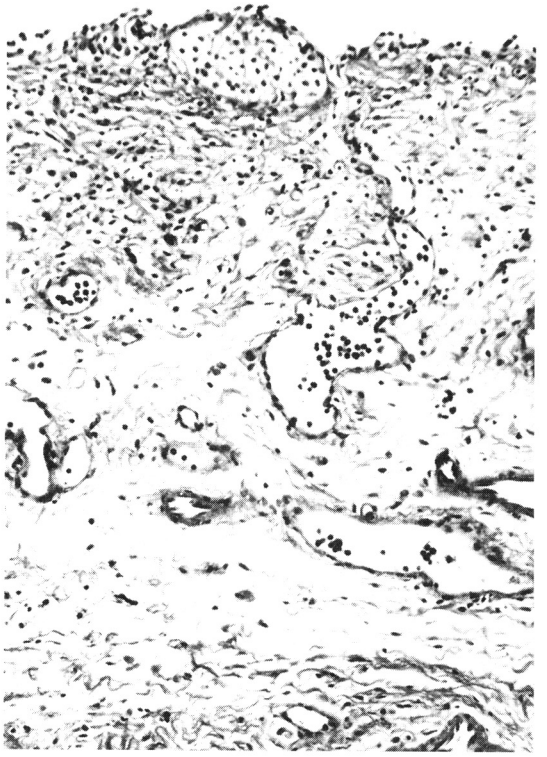

Fig. 3A. The bladder mucosa showed relatively intact uroepithelium with focal denuded mucosa, diffuse edema, hypervascularity and focal perivascular infiltration of lymphocytes, plasma cells and some neutrophils (Masson's trichrome, × 200).

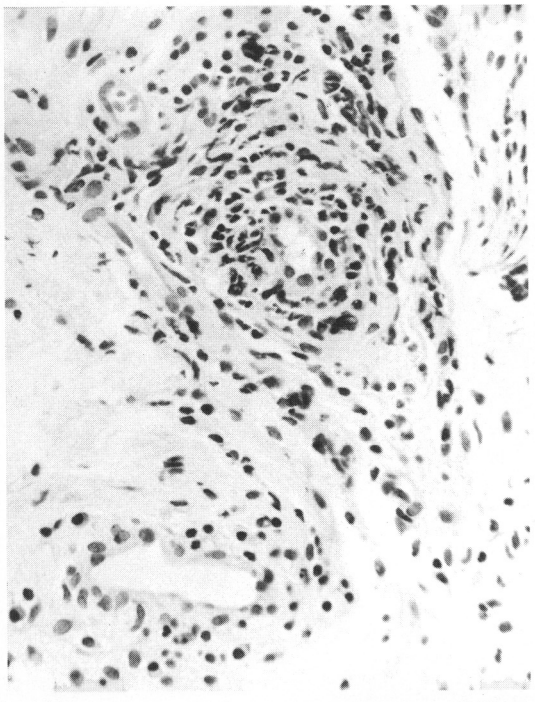

Fig. 3B. There were foci of transmural inflammatory infiltrates in some segments of venules (Hematoxylin and eosin, × 300).

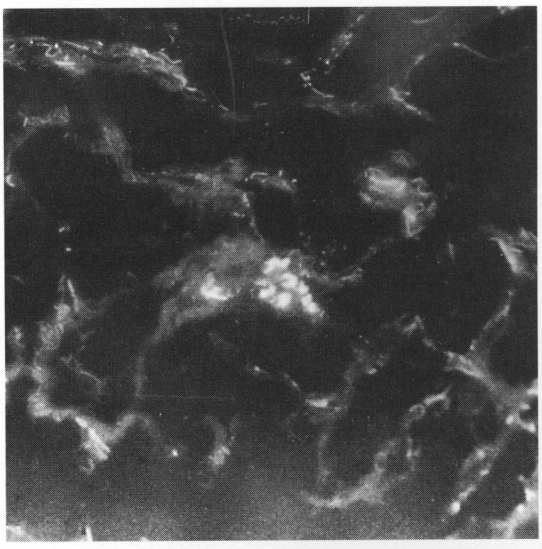

Fig. 4. Focal positive IF staining for IgM, in a granular pattern was observed in small blood vessels in the lamina propria of the bladder biopsy (× 200).

given (1 g i.v./month). In January, 1995, a follow-up cystoscopy revealed increased bladder capacity to 200 ml and visualization of bilateral ureteral orifices. Bilateral nephrostomy tubes were removed and renal function remained stable with serum creatinine, 1.2 mg/dL and no further surgical procedure was needed.

DISCUSSION

With early relief of obstruction in OU, the defects in function including renal insufficiency usually disappear completely. However, chronic obstruction may produce permanent loss of renal parenchymal mass and excretory function. Therefore, early diagnosis and prompt therapy are essential to minimize the irreversible damage caused by obstruction on kidney structure and function (Seifter and Brenner, 1992). Likewise, we consider the recognition of obstructive uropathy in SLE important because it is one of the reversible causes of renal failure in patients with SLE. As described in our patient, severe renal function deterioration was not caused by lupus glomerulonephritis but by OU with bilateral hydroureteronephrosis, which was relieved immediately with percutaneous nephrostomy and renal function remained stable with medical treatment even after removal of the nephrostomy tube.

As shown in Table 1, since the first report of OU with bilateral hydroureteronephrosis due to retroperitoneal fibrosis in a 17 year old oriental female with SLE in 1974 (Lloyd et al, 1974), the association of well-documented SLE and typical OU have been reported in 19 patients (Table 1). In the review of these patients, female patients (84 %) predominated and mean age was 31.7 years. These were consistent with the general sex and age distribution of the patients with SLE. In contrast, oriental patients comprised 12 (63 %) of 19 patients with SLE associated OU, which is quite different from racial prevalence of blacks in patients with SLE in general (Hahn, 1992). However, this review is too small to allow for speculation of this racial difference in the different pathogenesis of SLE with OU compared to SLE in general.

Out of 19 patients except for 2 patients-one with the first case of OU in SLE with the underlying cause of retroperitoneal fibrosis (patient 1) and another one with ureteral vasculitis (patient 13), 17 (89 %) patients

Table 1. Summary of reported cases of obstructed uropathy associated with SLE in the literature.

Patient (Reference)	Age/Sex	Race	Cause	Type of OU	Interval*	GI symptoms	Renal biopsy	Cutaneous vasculitis	CNS symptoms	ANA	DNA	C_3	Treatment (steroid/I)	Operation	Outcome (cause of death)
1. Llyoid et al.(1974)	17/F	Ori	RF	HUN	3Y 6M	ns	ns	ns	ns	1 : 80	+	ns	S(+++)		
2. Kataoka et al.(1980)	37/F	Ori	IC	HUN	7Y 5M	P,D	ns	+	ns	1 : 320	1 : 640	ns	S(+)	C	
3. Weisman et al.(1981)	34/M	Whi	IC	HUN	0	D,M	Class V	+	−	1 : 640	high	40	S(++)		
4. DeLa Serna et al.(1981)	20/F	ns	IC	UD	0	D	Class IV	+	ns	+	+	>30	S(+), Im(+)		died (infection)
5. Orth et al.(1983)	30/F	ns	IC	HUN	ns	P,D,I	ns	−	−	ns	+	+	S(++)		
6. Orth et al.(1983)	22/F	ns	IC	HUN	ns	D,M	Class II	−	+	ns	−	−	S(+++)		died (SLE)
7. Isaka et al.(1983)	42/F	Ori	IC	HUN	0	V,D,A	ns	−	ns	ns	ns	ns	S(−)	N	
8. Takabayashi et al.(1985)	38/F	Ori	IC	HUN	1Y	V,D	ns	−	−	ns	ns	ns	S(?)		died (cerebral bleeding)
9. Takabayashi et al.(1985)	47/F	Ori	IC	HUN	0	V,D,I,A	Class V	ns	ns	1 : 64	21	40	S(−)	N	died (uremia, GI bleeding)
10. Takabayashi et al.(1985)	38/F	Ori	IC	HUN	1Y	V	Class II	ns	ns	1 : 10	37	20	S(+++)		
11. Takabayashi et al.(1985)	42/F	Ori	IC	HUN	0	V,D	Class III	−	−	1 : 160	140	27	S(+++)		
12. Kato et al.(1985)	30/F	Ori	IC	HUN	9Y	P,V,D,M	ns	ns	ns	ns	ns	ns	S(+++), Im(++)		
13. Baskin et al.(1989)	18/F	Ori	UV	UD	ns	ns	ns	−	+	ns	ns	ns	ns, ns	F	
14. Moriuchi et al.(1989)	37/F	Ori	IC	HUN	14Y	P,D,I,A	Class V	+	−	1 : 10	−	44	S(−)	F	died (perforation of ileum)
15. Vincencio et al.(1989)	33/F	ns	IC	HUN	3Y	P,D	ns	ns	ns	1 : 160	1 : 40	24	S(+++)		
16. Kunimi et al.(1989)	23/M	Ori	IC	HUN	4Y	V,D	ns	ns	ns	1 : 1280	26.8	43	S(++)		
17. Meulder et al.(1992)	33/F	Whi	IC	HUN	9Y	D,I,M,A	Class V	+	+	1 : 20	−	45	S(−)	N,C	died (infection)
18. Arriba et al.(1993)	26/M	ns	IC	HUN	4Y	P	ns	ns	ns	+	+	96	S(++), Im(+)		
19. Present case(1994)	36/F	Ori	IC	HUN	3Y	P,D,I	Class V	ns	ns	1 : 1280	15	38	S(+), Im(+)	N	

*Interval between onset of SLE and onset of obstructive uropathy(OU)

OU=obstructive uropathy ; Ori=oriental ; ns=not stated ; Whi=white ;

RF=retroperitoneal fibrosis ; IC=interstitial cystitis ; UV=ureteral vasculitis ;

HUN=hydroureteronephrosis ; UD=ureteral dilatation or ectasis ;

P=abdominal pain ; V=vomiting ; D=diarrhea ; I=ileus ; M=malabsorption ; A=ascites

Im=immunosuppressives (azathioprine or chlorambucil or cyclophosphamide) ;

ns=not stated ; +=present (positive) ; −=absent (negative) ;

S(+), (++), (+++)=mild, moderate, remarkable response to steroid treatment ;

I(−), (+), (++)=no, mild, moderate response to other immunosuppressives ;

C=cystoplasty ; N=nephrostomy ; F=skin-ureter fistula

developed obstructive uropathy with bladder involvement, of whom radiological imaging with IVP or ultrasonogram showed bilateral hydroureteronephrosis in 16 patients and only bilateral ureteral dilatation without hydronephrosis in 1 patient. Therefore, there is no doubt that the bladder is a leading site than any other part of the genitourinary system causing OU in patients with SLE. Furthermore, it was suggested that bladder involvement regardless of the development of obstructive uropathy may have been underestimated in SLE based on autopsy findings (Alarcon-Segovia et al, 1984) : 16 out of 35 necropsies from SLE patients were found to have primary histologic changes in the bladder including interstitial cystitis in 11 patients among them, but none of them had symptoms referred to the bladder. On bladder biopsy and cystographic findings, all the 17 lupus patients with obstructive uropathy with bladder involvement (Table 1) were found to have small contracted bladders with thickened walls and submucosal edema, fibrosis and infiltration of inflammatory cells such as mononuclear cells and neutrophils consistent with chronic interstitial cystitis (IC).

Up to the present, twenty one patients with IC associated with SLE have been reported in the world literature, of whom 17 patients were accompanied with OU on radiological imaging (Table 1) and 4 patients were without OU (Boye et al, 1979 ; Orth et al, 1983 ; Sotolongo et al, 1984). Therefore, the presence of IC in SLE does not necessarily mean the development of OU, but OU development may depend on the severity or chronicity of inflammatory activity during the course of IC due to SLE. In the typical case of IC in SLE, the patient suffers from urinary frequency associated with suprapubic discomfort and dysuria ; no infecting organisms can be isolated from the urine and at cystoscopy there is a small bladder that may show ulcers and stellate scars. The etiology of IC in SLE is unknown but infection (Staples et al., 1974), blockage of small vessels or lymphatics, nervous conditions-transverse myelopathy (Adrianakos et al., 1975), immunosuppressive drug toxicity (Aptekar et al., 1973) or autoimmune mechanisms have been suggested.

Of the various postulated etiological mechanisms for IC in SLE, the autoimmunemechanism is the most popular. Deposits denatured DNA, IgG, IgM, IgA, and C3 by IF in the bladder was shown in a female patient with documented SLE who developed IC as her main manifestation of the disease (Boye et al, 1979). These deposits represent immune complexes in the bladder. Immune complex-mediated vasculitis has been further suggested in patients with IC in SLE, as blood vessels of the bladder as well as the other organs showed deposits of immune complexes (De La Serna and Alarcon-Segovia, 1981 ; Weisman et al, 1981). Along with the immunofluorescent studies of our case, these previous findings led us to the conclusion that OU with bladder involvement due to IC in patients with SLE has been the consequence of immune complex deposition.

In contrast to idiopathic IC, lupus related IC is frequently associated with hydroureteronephrosis which is either due to detrusor muscle spasm resulting in vesicoureteral reflux or usually due to fibrosis of the ureterovesical junction (Orth et al., 1983). In view of the transient nature of detrusor muscle spasm and somewhat chronicity of fibrosis, the interval between onset of OU and onset of other clinical manifestations of SLE in 17 patients with IC in the present review showed variable range from simultaneous onset to maximum 14 years (Table 1).

As expected, each patient in the present review had moderately severe multisystem disease and evidence of SLE activity along with the development of symptoms with obstructive uropathy. Of particular interest is the strong association between OU and severe gastrointestinal manifestations noted in patients with SLE. As shown in Table 1, a variety of GI manifestations such as vomiting, diarrhea, abdominal pain, paralytic ileus, malabsorption and even ascites were found alone or in combination of two, three or four of them, which can be classified as so called lupus enteropathy. Lupus enteropathy is thought to be due to venulitis or lymphangiectasia (Weisner et al., 1981 ; Chase et al., 1982). However, there is no known causative relationship between enteropathy and obstructive uropathy in SLE, so far.

Another major organ involvement in patients with OU and SLE is concomitant glomerulonephritis of advanced degree. Present review showed WHO morphologic lupus classification in 9 patients ; class V in 5, class IV in 1, class III in 1 and class II in 2. Otherwise, simultaneous involvement of other widespread multisystem such as skin (vasculitis) and CNS were not prominent as in the GI tract and kidney involvement. Moreover, the serologic activity of ANA, DNA antibody and complement were not increased uniformly in all 19 cases of OU associated with SLE.

The natural history of OU due to bladder or

extra-bladder involvement in SLE is unknown at present and the role for specific therapy is undefined. But, corticosteroids, a well known therapeutic agent in major organ involvement of SLE would be chosen as an initial therapeutic agent for OU associated with SLE. Present review (Table 1) suggested that early adequate medical treatment might avoid irreversible changes in the bladder as well as the kidneys, since more than two-thirds (68 %) of these patients showed symptomatic improvement with avoidance of eventual surgical intervention alone or both steroids plus other immunosuppressive drugs. However, despite medical treatment, surgical intervention such as cystoplasty or urinary diversion was needed to relieve obstruction and the long term prognosis of patients with OU in SLE was not favorable. Six (32 %) out of 19 patients died of various causes.

In summary, OU is associated with SLE and is more common than previously recognized. A typical case of OU associated with SLE was presented. Oriental middle-aged female with the concomitant gastrointestinal manifestation and symptoms of cystitis was described. These clinical characteristics of OU associated with SLE was presented through review of the literature. This review might enable clinicians to pinpoint this clinical entity in patients with SLE and could prevent further irreversible complications of OU with early recognition and interventions.

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