

Sasan Fallahi (MD)<sup>\*1,2</sup>  
Ahmad Reza Jamshidi (MD)<sup>2</sup>  
Farhad Gharibdoost (MD)<sup>2</sup>  
Mahdi Mahmoudi (PhD)<sup>2</sup>  
Pedram Paragomi (MD)<sup>2</sup>  
Mohammad Hossein Nicknam  
(MD, PhD)<sup>3</sup>  
Elham Farhadi (MSc)<sup>3</sup>  
Mostafa Qorbani (PhD)<sup>4</sup>

1- Department of Rheumatology,  
Kerman University of Medical  
Sciences, Kerman, Iran.

2- Rheumatology Research Center,  
Shariati Hospital, Tehran University  
of Medical Sciences, Tehran, Iran.

3- Molecular Immunology Research  
Center, Tehran University of  
Medical Sciences, Tehran, Iran.

4- Department of Epidemiology and  
Biostatistics, School of Public  
Health, Tehran University of  
Medical Sciences, Tehran, Iran.

**\* Correspondence:**

Sasan Fallahi, Rheumatology  
Research Center, Shariati Hospital,  
Kargar street, Tehran University of  
Medical Sciences, Tehran, Iran.  
Postal code: 1411713135

**E-mail:** drsfallahi@gmail.com

**Tel:** 0098 21 88026956

**Fax:** 0098 21 88026956

**Received:** 26 May 2012

**Revised:** 5 July 2012

**Accepted:** 25 July 2012

## Urolithiasis in ankylosing spondylitis: Correlation with Bath ankylosing spondylitis disease activity index (BASDAI), Bath ankylosing spondylitis functional index (BASFI) and Bath ankylosing spondylitis metrology index (BASMI)

### Abstract

**Background:** Increased incidence of renal stone has been reported in ankylosing spondylitis (AS), but unlike some well-known renal involvements, they have not been fully studied. The aim of this study was to investigate the association of AS with urolithiasis and also the relation between urinary stone and severity markers.

**Methods:** One hundred-sixty three AS patients were included in a cross-sectional study from Iranian AS association, Iran Rheumatology Center and Rheumatology Clinic of Shariati Hospital in Tehran. Prevalence of urolithiasis in AS patients was compared with results of a nationwide survey in Iran. Bath ankylosing spondylitis disease activity index (BASDAI), bath ankylosing spondylitis functional index (BASFI) and bath ankylosing spondylitis metrology index (BASMI) were determined for assessment of disease severity.

**Results:** Urolithiasis was observed in 11.7% of AS patients versus 5.7% of normal population ( $p=0.001$ ). After the elimination of corticosteroid effect, the prevalence of urolithiasis was still higher in AS patients than normal population but without maintaining significant difference. Significant higher values of BASFI, BASMI, BASDAI scores were observed in AS with urolithiasis than AS without urolithiasis.

**Conclusion:** The results confirmed the association of AS with urolithiasis. However, this may be partly due to the effect of other factors such as corticosteroid. Moreover, urolithiasis is accompanied with more severe diseases.

**Keywords:** Ankylosing spondylitis, Urolithiasis, BASDAI, BASFI

*Caspian J Intern Med 2012; 3(4): 508-513*

Ankylosing Spondylitis (AS) is the prototype of the widely interrelated group of Spondyloarthropathies with common genetic predisposing factors in particular HLA-B27. This chronic inflammatory disease primarily affects sacroiliac joints and at later stages imperils axial skeleton. Among Caucasians, the prevalence of AS has been reported from 68 per 100000 in the Netherlands to 210 per 100000 in the Norway (1-5). WHO-ILAR Community Oriented Program for Control of Rheumatic Diseases (COPCORD) study in Iran revealed the prevalence of AS from urban to rural areas are 0.12% and 1.1%, respectively (6, 7). Extra-articular disease can often be an associated feature in AS. Renal involvement which is one of the extra skeletal manifestations including IgA nephropathy, secondary amyloidosis and analgesic nephropathy (8). Urolithiasis is also a probable renal complication in AS but unlike some well-known renal involvements, this has not been fully studied. In some recent studies, an increased incidence of renal stone was reported in AS and AS has been proposed as an independent risk factor for nephrolithiasis (9, 10). The main aim of this study was to clarify the relationship between AS and symptomatic urolithiasis in Iranian patients with AS. We also intended to investigate the impact of urolithiasis on severity markers in AS patients.

## Methods

This study was conducted from May 2010 to March 2011. One hundred-sixty three patients with AS were recruited from three sources via convenience sampling method: Iranian AS Association, Iran Rheumatology Center (a center dedicated to rheumatologic patients) and rheumatology clinic of Shariati hospital (Tehran, Iran). AS was defined according to the 1984 modified New York criteria (11). The patients with recurrent urinary tract infections and hyperparathyroidism were not included in study. All patients gave informed consent in accordance with principles of the 1964 declaration of Helsinki prior to the inclusion in the study. A structural questionnaire was used to assess the presence of urolithiasis (ultrasound was performed only for the patients who had symptoms suspicious to urinary stone and the stones with any size were considered as positive result), peripheral arthritis, inflammatory bowel disease (diagnosed by colonoscopy only when the patients had symptoms suspicious to this co-morbidity) and to define the age, sex, disease duration, body mass index (BMI) (weight/height<sup>2</sup>), disease severity indices scores including Bath ankylosing spondylitis disease activity index (BASDAI) (12), Bath ankylosing spondylitis functional index (BASFI) (13), Bath ankylosing spondylitis metrology index (BASMI) (14, 15), physical activity, calcium supplementation, drug treatments such as corticosteroid (regular systemic corticosteroid equivalent to at least 5 mg prednisolone). Validity and reliability of the Persian version of BASDAI and BASFI questionnaires were assessed prior to the present study with adequate values (16). The prevalence of urolithiasis in AS patients was compared with the result of a population-based survey which had been performed on 7649 individuals in 2007 at all regions of Iran (17). This population was considered as control group. The data analysis was performed using SPSS version 18.

We used chi square and/or Fisher's exact test for comparing the categorical variables such as urolthiasis between the AS patients and normal population. Independent two-samples t-test was used to compare continuous variables such as BASFI, BASMI, BASDAI scores between the AS patients with urolithiasis and without urolithiasis. In each case,  $p < 0.05$  was considered significant.

## Results

The mean age for AS and normal population groups were

$37.7 \pm 9.87$  and  $40 \pm 4.7$  years, respectively. The distribution of baseline characteristics, including age, sex, obesity (BMI  $\geq 30$ ) in AS patients and normal population are summarized in table 1.

**Table 1. Comparison of age, sex and body mass index in AS patients versus normal population**

Variable	Ankylosing Spondylitis NO (%)	Normal Population NO (%)
Age Group	15-29	36 (22.1)
	30-39	64 (39.3)
	40-49	44 (27)
	$\geq 50$	19 (11.7)
Sex	male	129 (79.1)
	female	34 (20.9)
BMI <sup>a</sup>	<30	129 (79.1)
	$\geq 30$	33 (20.24)

a. One missing value among patients' data

**Comparison of AS patients with normal population:** Symptomatic urolithiasis was reported by 19 (11.7%) of AS patients compared with 436 (5.7%) of normal population ( $p=0.001$ , OR=2.18, 95% CI, 1.34-3.56). The ethnic distribution of patients and control group was approximately the same. With the exclusion of patients that received calcium supplementation, the difference between AS patients and normal population was maintained significantly: 13 out of 122 (10.7%) vs. 5.7%, respectively ( $p=0.02$ , OR=1.97, 95% CI, 1.1-3.53).

Similarly, after excluding the patients with corticosteroid treatment, the results still revealed higher prevalence of urolithiasis in AS patients versus normal population but significant difference was not maintained: 9 out of 100 patients (9%) versus 5.7% in normal population ( $P = 0.16$ ). Moreover, the prevalence of urinary stone was separately evaluated among males and females. In men, renal stone was reported in 16 out of 129 AS patients (12.4%) as compared with the 229 out of 3748 normal population (6.11%) with significant difference between groups ( $p=0.04$ , OR=2.18, 95% CI, 1.27-3.74). In women, the renal stone was reported in 3 out of 34 AS patients (8.82%) compared with 207 out of 3901 normal population (5.31%) without significant difference between them ( $p=0.425$ , OR=1.73, 95% CI, 0.52-5.70). After adjustment for age, the more frequent history of

uroolithiasis was seen in AS patients compared with normal population in all age groups; but this difference was significant only in age group 30-39, 40-49 years ( $p < 0.0001$ ,  $p = 0.038$ , respectively) (table 2).

**Table 2. Prevalence of urolithiasis in AS patients and normal population in different age groups**

Age Group	AS NO (%)	Normal Population NO (%)	Pvalue
15-29	1 (2.8)	12 (0.9)	0.294
30-39	10 (15.6)	44 (3.4)	<0.001
40-49	6 (13.6)	74 (5.6)	0.024
≥50	2 (10.5)	306 (8.3)	0.667
Total	19 (11.7)	436 (5.7)	0.001

**AS patients:** In AS patients, urolithiasis was more frequent among the males than the females but the difference was not statistically significant (12.4% vs. 8.8%,  $p = 0.77$ ). BMI in AS patients with urolithiasis was higher than AS patients without urolithiasis ( $p = 0.01$ ). The tendency to be sedentary was higher in patients with urolithiasis (31.6%) versus those without urolithiasis (28.1%), although statistically the significant difference was not observed ( $p = 0.75$ ,  $OR = 1.183$ ,

95% CI, 0.42-3.33). About 15.8% of AS patients with urolithiasis had IBD versus 5.6% of patients without urolithiasis ( $p = 0.121$ ,  $OR = 3.19$ , 95% CI, 0.8-13.3) (table 3). Nearly, 25.2% of total AS patients received Calcium supplements. Among AS cases with urolithiasis, 31.6% had history of calcium consumption versus 24.3% in patients without urolithiasis ( $p = 0.33$ ,  $OR = 0.70$ , 95% CI, 0.25-1.97). But, in the age category (30-39 years old), calcium consumption was less frequent in patients with urolithiasis in comparison with patients without urolithiasis (10.0% vs. 27.8%,  $p = 0.429$ ). Ten out of 19 (52.6%) AS patients with urolithiasis had received corticosteroid treatment in comparison with 53 out of 144 (36.8%) patients without urolithiasis ( $p = 0.18$ ) (table 2).

**Urolithiasis and AS: severity markers:** The higher values of BASFI, BASMI, BASDAI scores were observed in AS patients with urolithiasis than patients without urolithiasis ( $p = 0.003$ ,  $p = 0.037$ ,  $p = 0.055$ ). Peripheral arthritis was seen in 12 patients with urolithiasis (63%) and 71 patients without urolithiasis (49.3%); although this association was not statistically significant ( $p = 0.256$ ,  $OR = 1.76$ , 95% CI, 0.66-4.73). The disease duration difference between AS patients with urolithiasis and without urolithiasis was not statistically significant ( $p = 0.12$ ) (table 3).

**Table 3. Comparison of severity markers and other characteristics in AS with and without urolithiasis**

	Urolithiasis + (N=19)	Urolithiasis - (N=144)	P-value
BASFI	57.737±26.049	38.44±26.829	0.004
BASDAI	32.526±15.079	26.06±13.63	0.06
BASMI	4.826±1.949	3.83±1.85	0.03
Arthritis NO (%)	12 (63.2)	71 (49.3)	0.26
IBD NO (%)	3 (15.8)	8 (5.6)	0.12
Corticosteroid NO (%)	10 (52.6)	53 (36.8)	0.2
Calcium supplement NO (%)	6 (31.6)	35 (24.3)	0.33
Disease duration	17.53±9.008	14.09±8.346	0.09
Sedentary NO (%)	6 (31.6)	39 (28.1) <sup>a</sup>	0.79
BMI <sup>b</sup>	29.522±5.595	26.25±5.15	0.01

Values are mean ± SD, unless otherwise stated

a. Five missing data were considered in the analysis

b. One missing data was considered in the analysis

## Discussion

Our results revealed significantly the higher prevalence of urolithiasis in AS patients versus normal population. The

exact etiology of urolithiasis in AS is unknown but some hypotheses for hypercalciuria and resulting nephrolithiasis

have been proposed including the elevated circulating cytokines such as TNF- $\alpha$ , IL-6 and IL-17, disruption of vitamin D and Calcium metabolism due to sub-clinical bowel disease (IBD) which is common in AS, coexistent gout, prolonged immobilization caused by arthralgia, anti-inflammatory drugs such as corticosteroid, nonsteroidal anti-inflammatory drugs (NSAIDs), calcium supplementation and genetic polymorphisms such as ANKH mutation (18-30).

Urolithiasis unlike some well-known renal involvements has not been fully studied in AS. Among the few reported studies, a group of studies detected higher prevalence of urinary stone in AS but some others did not present similar reports (9, 10, 22). Our results were compatible with the two following studies:

Korkmaz et al. compared 80 AS patients with 72 Behcet's patients and 98 healthy individuals as controls. Renal stone prevalence was found to increase in AS patients (25%) versus Behcet's disease (5.5%) and healthy controls (3.3%). Also, they found disease duration was longer in AS patients with renal stone compared with patients without urolithiasis (9).

Canales et al. reported kidney stones in 29% of the 79 spondyloarthropathies (SpA) patients compared with 12.5% of the 64 rheumatoid arthritis (RA) patients as controls. Populations were similar in all features except that RA patients were more likely to have used corticosteroid, bisphosphonate, and calcium supplementation. Despite adjusting for medication use and matching two similar populations, the patients with SpA had a higher incidence of kidney stones than those with RA and finally they suggest that SpA may be an independent risk factor for urinary stone formation (22). The results of these two above studies are similar to the findings of our study.

In the current study after adjusting sex and age groups, more frequent history of urolithiasis persisted in AS patients compared with normal population, but this difference was maintained significantly only in age group 30-39, 40-49 years and male gender (table 1). Therefore, the association of AS with urolithiasis seems to be more significant in males.

One of the influential parameters in renal stone formation is the history of calcium supplementation which has a controversial role. Some recent studies have challenged the notion that calcium supplementation inhibits urinary stone formations (31). With the exclusion of patients that received calcium supplementation in present study, the difference between the prevalence of urolithiasis in AS patients and

normal population decreased but was maintained significantly. Consequently, the calcium supplementation is unlikely to play role as inhibitory stone former in our AS patients. The other predisposing factor for nephrolithiasis in AS is receiving prednisone treatment which is expected to be more frequent in AS patients (24, 25). Although after excluding the patients with corticosteroid treatment, the results still revealed higher prevalence of urolithiasis in AS patients as compared with normal population but the difference was not significant. Hence, the association of AS with urolithiasis may be somewhat due to the effect of corticosteroid treatment. Obesity seems to be a notable nephrolithiasis risk factor considering the significant association between higher BMI and urolithiasis in our AS patients which is similar to the results observed in Iranian normal population (17).

Interestingly, urolithiasis accompanied some different clinical and therapeutic patterns in AS. Overall, a line of evidence linked coexistent renal stone with more intense progression of AS (32). Lui et al. showed in their cohort (performed on 38 AS patients with urolithiasis and 76 patients without urolithiasis matched for age, sex, and ethnicity) that patients with urolithiasis had more functional disability, based on BASFI. Trends were noted in the urolithiasis group toward higher BASDAI and more peripheral joint involvement. No significant difference was detected in BASMI (32). Also, Cansu et al. reported renal stone history in 27.5% of Turkish patients with AS, introducing a correlation between nephrolithiasis and higher radiological scores in AS. It is believed that renal stone accompanies AS cases with more severe radiographic damages and presumably poor prognosis (33). In accordance with these studies, we found a significantly higher BASFI, BASMI and also borderline significantly higher BASDAI in AS patients with urolithiasis.

Furthermore, Lui et al. revealed a significant association of AS with Crohn's disease in AS patients with urolithiasis (32). A higher prevalence of IBD was also detected in our AS patients with urolithiasis than AS without urolithiasis; but the difference was not statistically significant. A causative correlation between Crohn's disease and urolithiasis has been explained and different pathologic pathways may cause stone formation in Crohn's patients (34, 35). Thus, higher prevalence of urolithiasis in AS patients to some extent may be due to more frequent IBD in these patients. One of the limitations of our study was the

inaccessibility of complete drug history in our control group, most importantly calcium supplementation and corticosteroid treatment. Moreover, some other risk factors including IBD, hyperparathyroidism or prolonged immobilization was not recorded in healthy controls. Despite the consideration of age, sex, corticosteroid and calcium supplementation in analysis, adjustment for some other urinary stone risk factors between normal population and AS patients was not completely possible. For example, the majority of AS patients received NSAIDs as treatment and we were not able to adjust it with control population.

Only the symptomatic urolithiasis was considered in current study. Therefore, the exact prevalence of urolithiasis could have been underestimated among the AS patients and consequently the prevalence of urolithiasis (symptomatic and asymptomatic totally) in our AS patients is probably higher than the values reported in this survey.

One of the advantages of the current study was that the control group included individuals from a population-based survey and not from patients with other diseases such as Behçet's disease or rheumatoid arthritis. In addition, the higher number of AS patients studied in current survey compared with other studies is another advantage of our study. Although, based on earlier studies in the control group, the sample size of patients is still small and a larger sample size may be required. Our results showed the association of AS with urolithiasis. However, this association may be in part due to other factors like corticosteroids consumption or associated IBD. Furthermore, urolithiasis is accompanied with more severe diseases. Therefore, concerning the urolithiasis in AS patients seems to be useful and we suggest the evaluation of nephrolithiasis in follow up and management of AS patients especially those that receive corticosteroid for any reason. Further studies about genetic polymorphisms such as ANKH mutation that probably involve urolithiasis of AS is also suggested.

### Acknowledgments

The authors would like to thank the coordinator of Iranian AS Association for cooperating in the recruitment of the AS patients.

**Funding:** This study was a part of fellowship thesis and research project (code: 89-03-41-11076) which was

supported and funded by Tehran University of Medical Sciences.

**Conflict of interest:** None.

### References

1. Kaipiainen-Seppänen O, Aho K, Heliövaara M. Incidence and prevalence of ankylosing spondylitis in Finland. *J Rheumatol* 1997; 24: 496-9.
2. van der Linden SM, Valkenburg HA, de Jongh BM, Cats A. The risk of developing ankylosing spondylitis in HLA-B27 positive individuals. A comparison of relatives of spondylitis patients with the general population. *Arthritis Rheum* 1984; 27: 241-9.
3. Ahearn JM, Hochberg MC. Epidemiology and genetics of ankylosing spondylitis. *J Rheumatol* 1988; 16: 22-8.
4. Bakland G, Nossent HC, Gran JT. Incidence and prevalence of ankylosing spondylitis in Northern Norway. *Arthritis Rheum* 2005; 53: 850-5.
5. Saraux A, Guillemin F, Guggenbuhl P, et al. Prevalence of spondyloarthropathies in France: 2001. *Ann Rheum Dis* 2005; 64: 1431-5.
6. Davatchi F, Tehrani Banihashemi A, Gholami J, et al. The prevalence of musculoskeletal complaints in a rural area in Iran: a WHO-ILAR COPCORD study (stage 1, rural study) in Iran. *Clin Rheumatol* 2009; 28: 1267-74.
7. Davatchi F, Jamshidi AR, Banihashemi AT, et al. WHO-ILAR COPCORD Study (Stage 1, Urban Study) in Iran. *J Rheumatol* 2008; 35: 1384-90.
8. Vilar MJ, Cury SE, Ferraz MB, Sesso R, Atra E. Renal abnormalities in ankylosing spondylitis. *Scand J Rheumatol* 1997; 26: 19-23.
9. Korkmaz C, Ozcan A, Akcar N. Increased frequency of ultrasonographic findings suggestive of renal stones in patients with ankylosing spondylitis. *Clin Exp Rheumatol* 2005; 23: 389-2.
10. Incel NA, Gokoglu F, Nacir B, Incel N. Bone and stone in ankylosing spondylitis: osteoporosis and urolithiasis. *Clin Rheumatol* 2006; 25: 667-70.
11. van der Linden S, Valkenburg HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the New York criteria. *Arthritis Rheum* 1984; 27: 361-8.
12. Garrett S, Jenkinson T, Kennedy LG, et al. A new approach to defining disease status in ankylosing

- spondylitis: the Bath Ankylosing Spondylitis Disease Activity Index. *J Rheumatol* 1994; 21: 2286-91.
13. Van Weely SF, Van Denderen CJ, Van der Horst-Bruinsma IE, et al. Reproducibility of performance measures of physical function based on the BASFI, in ankylosing spondylitis. *Rheumatology (Oxford)* 2009; 48: 1254-60.
  14. Jenkinson TR, Mallorie PA, Whitelock HC, et al. Defining spinal mobility in ankylosing spondylitis (AS). The Bath AS Metrology Index. *J Rheumatol* 1994; 21:1694-8.
  15. Van Der Heijde D, Landewe R, Feldtkeller E. Proposal of a linear definition of the Bath Ankylosing Spondylitis Metrology Index (BASMI) and comparison with the 2-step and 10-step definitions. *Ann Rheum Dis* 2008; 67: 489-93.
  16. Bidad K, Fallahi S, Mahmoudi M, et al. Evaluation of the Iranian versions of the bath ankylosing spondylitis disease activity index (BASDAI), the bath ankylosing spondylitis functional index (BASFI) and the patient acceptable symptom state (PASS) in patients with ankylosing spondylitis. *Rheumatol Int* 2011; November 20 [E pub ahead of print].
  17. Safarinejad MR. Adult urolithiasis in a population-based study in Iran: prevalence, incidence and associated risk factors. *Urol Res* 2007; 35: 73-82.
  18. Carter S, Lories RJ. Osteoporosis: a paradox in ankylosing spondylitis. *Curr Osteoporos Rep* 2011; 9: 112-5.
  19. Pacifici R. Idiopathic hypercalciuria and osteoporosis--distinct clinical manifestations of increased cytokine-induced bone resorption? *J Clin Endocrinol Metab* 1997; 82: 29-31.
  20. Ghazali A, Fuentes V, Desaint C, et al. Low bone mineral density and peripheral blood monocyte activation profile in calcium stone formers with idiopathic hypercalciuria. *J Clin Endocrinol Metab* 1997; 82: 32-8.
  21. Mielants H, De Keyser F, Baeten D, Van den Bosch F. Gut inflammation in the spondyloarthropathies. *Curr Rheumatol Rep* 2005; 7: 188-94.
  22. Canales BK, Leonard SM, Singh JA, et al. Spondyloarthropathy: an independent risk factor for kidney stones. *J Endourol* 2006; 20: 542-6.
  23. Stewart AF, Adler M, Byers CM, Segre GV, Broadus AE. Calcium homeostasis in immobilization: an example of resorptive hypercalciuria. *N Engl J Med* 1982; 306: 1136-40.
  24. Kukreja SC, Bowser EN, Hargis GK, et al. Mechanisms of glucocorticoid-induced osteopenia: role of parathyroid glands. *Proc Soc Exp Biol Med* 1976; 152: 358-61.
  25. Suzuki Y, Ichikawa Y, Saito E, Homma M. Importance of increased urinary calcium excretion in the development of secondary hyperparathyroidism of patients under glucocorticoid therapy. *Metabolism* 1983; 32: 151-6.
  26. Domrongkitchaiporn S, Ongphiphadhanakul B, Stitchantrakul W, et al. Risk of calcium oxalate nephrolithiasis after calcium or combined calcium and calcitriol supplementation in postmenopausal women. *Osteoporos Int* 2000; 11: 486-92.
  27. Khan SR, Canales BK. Genetic basis of renal cellular dysfunction and the formation of kidney stones. *Urol Res* 2009; 37: 169-80.
  28. Tsui HW, Inman RD, Paterson AD, Reveille JD, Tsui FW. ANKH variants associated with ankylosing spondylitis: gender differences. *Arthritis Res Ther* 2005; 7: 513-25.
  29. Sayer JA. The genetics of nephrolithiasis. *Nephron Exp Nephrol* 2008; 110: e37-43.
  30. Timms AE, Zhang Y, Bradbury L, Wordsworth BP, Brown MA. Investigation of the role of ANKH in ankylosing spondylitis. *Arthritis Rheum* 2003; 48: 2898-902.
  31. Jackson RD, LaCroix AZ, Gass M, et al. Calcium plus vitamin D supplementation and the risk of fractures. *N Engl J Med* 2006; 354: 669-83.
  32. Lui NL, Carty A, Haroon N, et al. Clinical correlates of urolithiasis in ankylosing spondylitis. *J Rheumatol* 2011; 38: 1953-6.
  33. Cansu DU, Calisir C, Savas Yavas U, Kasifoğlu T, Korkmaz C. Predictors of radiographic severity and functional disability in Turkish patients with ankylosing spondylitis. *Clin Rheumatol* 2011; 30: 557-62.
  34. Manganiotis AN, Banner MP, Malkowicz SB. Urologic complications of Crohn's disease. *Surg Clin North Am* 2001; 81: 197-215.
  35. McConnell N, Campbell S, Gillanders I, Rolton H, Danesh B. Risk factors for developing renal stones in inflammatory bowel disease. *BJU Int* 2002; 89: 835-41.