myositis. *Arthritis Rheum* 1976; 19: 243-8.
9. QUEIRO-SILVA R, BANEGIL I, DE DIOS-JIMENEZ DE ABERASTURI JR, BELZUNEGUI-OTANO J, GONZALEZ-BENEITEZ C, FIGUER-OA-PEDROSA M: Periarticular calcinosis associated with the anti-Jo-1 antibodies sine myositis. Expanding the clinical spectrum of the anti-synthetase syndrome. *J Rheumatol* 2001; 28: 1401-4.
10. LOVE LA,LEFF RL,FRASERDD *et al.*: A new approach to the classification of idiopathic inflammatory myopathy: Myositis-specific autoantibodies define useful homogeneous patient groups. *Medicine (Baltimore)* 1991; 70: 360-74.

Pulmonary and renal involvement in ankylosing spondylitis

Sirs,

Ankylosing spondylitis (AS), which is primarily a disease of the musculoskeletal system, can be accompanied by extraskeletal manifestations, but renal and pulmonary involvement are rare (1).

A 57-year-old man diagnosed with AS 30 years previously was admitted with the symptoms of dyspnea, back pain, polyuria, and swelling of the face. He had a spinal deformity and hip joint involvement. A systemic examination was normal apart from an increased expiration phase on auscultation. Laboratory investigations revealed: serum creatinine 1.6 mg/dl (normal 0.6-1.3), BUN 28 mg/dl (normal 6-20), total protein 5.8 gr/dl (normal 6.4-8.3), albumin 2.7 gr/dl (normal 3.4-5.0), erythrocyte sedimentation rate 75 mm/hr, and creatinine clearance 45 ml/min. Urinary analyses showed a density of 1008, 500 mg proteinuria, 8-10 leukocytes/hpf and 3 gr proteinuria/24 hr. Arterial blood gases were as follows: pH 7.46, PO_2 64 mm Hg, PCO_2 30 mm Hg, and oxygen saturation 94%. In pulmonary function tests (PFT) FVC was 3.16 (66%),FEV1 2.58 (68%),FEV1/FVC 81%, and there was a restrictive type ventilatory pattern. On chest x-ray there were bilateral interstitial infiltrative lesions in the middle and lower zones of the lung parenchyma. A Ppd test was done and found to be negative, as was sputum cytology repeated 3 times. High resolution computed tomography (HRCT) revealed a bilateral ground glass appearance in the lung parenchyma (Fig. 1). On renal ultrasound the dimensions of the left kidney were 144 x 64 mm,the thickness of the parenchyma was 15 mm,and the parenchymal echo was interpreted to be grade 2. In flow patterns obtained from the interlober arteries, RI was 0.69 in the spectral analysis, which was compatible with a renal parenchymal disease. A biopsy was scheduled but refused by the patient. Therefore a rectal biopsy was performed for possible amyloidosis but was negative. Serum IgA was 2.23 g/l (normal 0.7-4.0). The patient was started on pulse steroid therapy and sulphasalazine 2 gr/day.

Lung involvement occurs in 1% of AS patients approximately 20 years after onset of the disease. Although upper lobe fibrotic and infiltrative lesions are characteristic, in our patient chest x-rays showed middle and lower lobe infiltration (2, 3). There was no other explanation other than AS (such as methotrexate administration or occupational lung disease) for this finding.

There was a slight to moderate decrease in the patient's PFT, specifically the vital capacity and total lung capacity denoting a restrictive type ventilatory defect. Despite diminished thoracic mobility, a significant deterioration in PFTs is not usual in AS due to diaphragmatic compensation, as in our patient. We detected a ground glass appearance which is a strong evidence of interstitial lung disease in HRCT.

In AS, possible causes of renal dysfunction include; secondary amyloidosis, IgA and analgesic nephropathy (4). Regarding the absence of gross hematuria, a normal IgA level and proteinuria of 3 gr/24 hr, the possibility of IgA nephropathy was ruled out in our patient. In cases with analgesic nephropathy, there is usually colic type pain due to papillary necrosis, a sterile pyuria and proteinuria < 1 gr/24 hr, so we also ruled out analgesic nephropathy.

The incidence of renal amiloidosis in AS is reported to be 1-3% (5, 6). Proteinuria or nephrotic syndrome is the most common clinical manifestation where a biopsy should be performed (7), although unfortunately we could not do so as our patient refused. We were able to evaluate the rectal biopsy specimens, which were amyloid negative. However rectal biopsy has been proven to be positive in only 65% of systemic amyloidosis cases.

Here we present a case of AS with both renal and pulmonary involvement, which are rare clinical presentations. Extra-skeletal manifestations should be investigated in patients with longstanding AS in the presence of new symptoms.

I. NADIR E. KAPTANOGLU
S. TOPCU S. ICAGASIOGLU

Address correspondence to: Dr Isilay Nadir, Cumhuriyet Üniversitesi, Tip Fakültesi Hastanesi, Ic Hastaliklari Ana Bilim Dali, Sivas, Turkey. E-mail: isilay_nadir@hotmail.com

References

1. VAN DER LINDEN S, VAN DER HEIJDE D: Ankylosing spondylitis. In RUDDY S, HARRIS ED, SLEDGE CB (Eds.): *Kelly's Textbook of Rheumatology*, vol. 2, 8th ed., Philadelphia, W.B. Saunders Co. 2001; 1039-53.
2. TURETSCHEK K, EBNER W, FLEISCHMANN D *et al.*: Early pulmonary involvement in ankylosing spondylitis: Assessment with thin-section CT. *Clin Radiol* 2000; 55: 632-636.
3. FELTELIUS N, HEDENSTROM H,HILLERDAL G *et al.*: Pulmonary involvement in ankylosing spondylitis. *Ann Rheum Dis* 1986; 45: 736-40.
4. JONES DW, MANSELL MA, SAMUELL CT *et al.*: Renal abnormalities in ankylosing spondylitis. *Br J Rheumatol* 1987; 26:341-5.
5. GRATACOS J, COLLADO A, SANMARTI R *et al.*:Coincidental amyloid nephropathy and Ig A glomerulonephritis in a patient with ankylosing spondylitis. *J Rheumatol* 1993; 20(9): 1613-5.
6. KOVACSOVICS-BANKOWSKY M, ZUFFERY B, SO AK *et al.*: Secondary amyloidosis: A severe complication of ankylosing spondylitis. Two case-reports. *Joint Bone Spine* 2000; 67: 129-33.
7. VILAR MJ, CURRY SE, FERRAZ MB *et al.*: Renal abnormalities in ankylosing spondylitis. *Scand J Rheumatol* 1997: 26: 19-23.

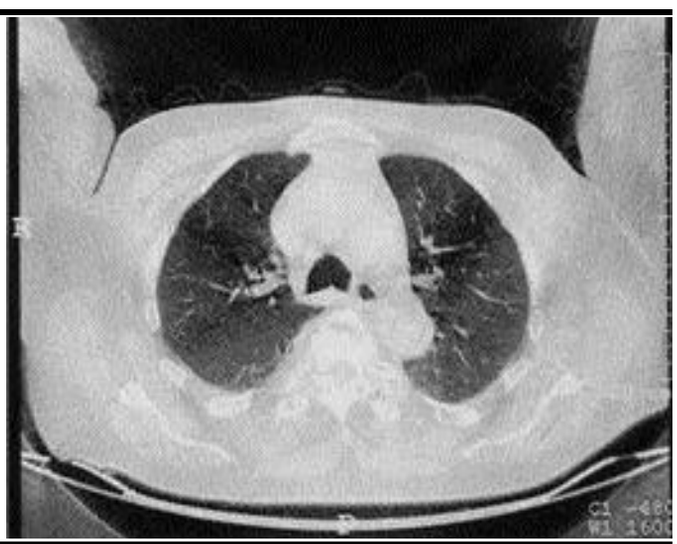

Fig. 1. Ground glass appearance on HRCT.