Dear Sir,

Takayasu’s arteritis (TA) is a chronic arteriopathy of unknown etiology, which involves large and medium-sized arteries. It predominantly occurs in young women, and affects many organs. Renal artery involvement [1] and some glomerular lesions [2] have been reported in this disease, but it is very rarely complicated with amyloidosis. We report here a case of TA which is complicated by systemic amyloidosis presented with nephrotic syndrome.

A 22-year-old female patient complained of gradually increasing edema in her legs which had first been noticed 3 months before. On physical examination, radial and brachial pulses were absent bilaterally, and diminution of the right carotid pulse was noted. Blood pressure was not measurable on the right arm, and it was 100/60 mm Hg on the left arm, and 200/120 mm Hg on the lower extremities. Pretibial edema was also noted. On laboratory evaluation, 24-hour proteinuria was 6 g, Bence-Jones protein was negative. Erythrocyte sedimentation rate was 121 mm/h. Blood urea nitrogen was 9 mg/dl, serum creatinine 0.7 mg/dl, total cholesterol 370 mg/dl and serum albumin 1.2 g/dl. Serum IgG, A, M and complement levels were normal. Rheumatoid factor, anti-nuclear antibody and anti-dsDNA were negative. Sputum and urine examination showed no Mycobacterium tuberculosis on smear. Chest X-ray, electrocardiogram and echocardiogram were normal. Renal biopsy revealed extensive amyloid deposits in the glomeruli by Congo red. In intra-arterial digital subtracting angiography (DSA), occlusion of both subclavian arteries beyond the origin and collateral channels with evidence of vessel filling distal to the block were detected; abdominal arteries were normal (fig. 1 and 2). She was discharged with prednisolone and colchicine treatment. There was no significant improvement in her findings 2 months later, and she did not report back for follow-up. Albuminuria is not an uncommon finding in TA [1]. In addition to ischemic or hypertensive renal disease, some primary glomerular lesions associated with TA may be responsible for albuminuria seen in patients with TA [2], but there are only few articles reporting patients with TA presenting with nephrotic syndrome [3, 4]. Interestingly all of these patients have
amyloidosis. On the other hand, it has long been recognized that some patients with chronic inflammatory disease develop amyloidosis, and therefore amyloidosis secondary to TA may be responsible for albuminuria in some patients. As far as we know, there are not more than 10 such cases in the world literature [4-6]. In our patient, even after careful evaluation, we found no evidence of any chronic disease which could have caused systemic amyloidosis, and we conclude that amyloidosis in this patient is secondary to TA. We would recommend repeated urinalysis for searching proteinuria in all cases of TA, and if necessary renal biopsy should be performed. We think primary glomerular lesions with TA, and amyloidosis complicating TA will not be rare conditions in the future.

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Fig. 1. Intra-arterial DSA shows occlusion of both subclavian arteries beyond the origin.
Fig. 2. Collateral channels with evidence of vessel filling distal to the block of right subclavian artery.

References


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