



Development of AA Amyloidosis in a Patient with Psoriasis: A Case Report

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ABSTRACT

Psoriasis is a chronic, recurrent, inflammatory and common skin disease of unknown aetiology. Amyloidosis is defined as a heterogeneous group of diseases in which generally soluble plasma proteins accumulate in the extracellular space as insoluble abnormal fibrils. Type AA amyloidosis is a late and severe complication of chronic inflammatory disorders and some chronic infections. Although psoriasis is a common inflammatory skin disease, the development of amyloidosis is rare. Herein, we presented a case of psoriasis who developed AA-type amyloidosis.

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Introduction

Amyloidosis is a heterogeneous group of diseases and is recognized by showing the accumulation of insoluble abnormal fibrillar forms of soluble proteins in the extracellular space in a tissue sample. The accumulation in primary amyloidosis is associated with a systemic or generalized type of monoclonal plasma cell proliferation. In secondary (reactive) amyloidosis, the accumulation is caused by an underlying chronic disease. Approximately 45% of amyloidoses are reactive amyloidosis. The leading causes of reactive amyloidosis are chronic infections, inflammatory diseases and malignancies. Although psoriasis is a common inflammatory skin disease, the development of amyloidosis is rare. Here, we presented a psoriatic case of secondary type amyloidosis.

Case Report

A 64-year-old female patient was followed for two years to diagnose plaque psoriasis on the scalp and joint extensor faces. There was no regular treatment for psoriasis. The patient had a history of swelling, pain and redness in the right ankle six weeks ago, and she used non-steroidal anti-inflammatory drugs and antibiotics. Her swelling and redness regressed, but the pain continued. She was admitted to the emergency service with a complaint of weakness and dry mouth. On the physical examination: there were no signs of swelling, hyperemia, temperature increase, or oedema in the right ankle. A lesion suggestive of psoriatic plaque was observed in the sacral region of the patient. Pretibial oedema was a bilateral positive. In the laboratory examination:



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leukocyte 16,300/mm³, hemoglobin 8.9 g/dL, platelet 372,000/mm³, CRP 204.6 mg/L, serum albumin 2.6 g/L, creatinine 6.4 mg/dL, urea 195 mg/dL, potassium 7.2 mmol/L. Her arterial blood gas analysis showed metabolic acidosis (pH 7.24 and HCO₃ 16.1 mmol/L). Urinalysis showed microscopic hematuria and protein positivity (+). There was no dilatation in bilateral pelvicalyceal structures in the urinary system ultrasonography (USG), and we excluded from post-renal kidney injury. We hospitalized her and started the patient dialysis treatment for acute renal dysfunction. On the 2nd day, the patient had macroscopic hematuria. We performed a kidney biopsy considering rapidly progressive glomerulonephritis due to restriction of urine output, detection of 3,360 mg/day proteinuria in 24-hour urine and macroscopic hematuria. After the biopsy, we applied 250 mg of methylprednisolone for three days and then 40 mg of methylprednisolone. A full immune evaluation revealed negative anti-nuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA), anti-double-stranded DNA (anti-dsDNA), anti-Sm, anti-Ro, anti-La, rheumatoid factor (RF), and anti-cyclic citrulline peptide antibody (anti-CCP). Protein electrophoresis, serum immunoglobulin, C3 and C4 serum levels were normal. The pathological result was compatible with AA-type amyloidosis.

Urinary system USG was performed in the patient whose hematuria continued to increase. In the cystoscopy of the patient with bladder floor irregularity, an organized hematoma filling the inside of the bladder was observed. The hematoma was partially cleared with cystoscopy. B-51 and B-13 allele genes were detected to determine the patient's HLA-B groups. Both sacroiliac joints were observed in normal width in the sacroiliac joint radiography, but periarticular sclerosis was noted on both sides (bilateral grade 2 according to New York criteria). No uveitis was detected in the eye examination. The patient did not need HD after six sessions. Her urine output increased. We planned to gradually decrease the corticosteroid treatment dose and discontinue the corticosteroid treatment, and started colchicine 0.5 mg twice a day.

Discussion

Causes of AA amyloidosis include rheumatic diseases (ankylosing spondylitis, rheumatoid arthritis, juvenile arthritis), idiopathic diseases (sarcoidosis, Crohn's disease, ulcerative colitis, Rosai-Dorfman disease), hereditary diseases (familial Mediterranean fever [FMF], tumour necrosis factor-associated periodic fever syndrome), infectious diseases (tuberculosis and leprosy), and malignant tumours (mesothelioma and Hodgkin's disease).¹ Few cases of coexistence of psoriasis and amyloidosis have been reported to date.² The first case of psoriasis-associated amyloidosis was described in 1965.²⁻⁴ Therefore, we tried to exclude other diagnoses accompanying amyloidosis in our patient, who did not have any other known comorbidities other than the diagnosis of psoriasis.

In Turkey, a Mediterranean country, the most common cause of reactive amyloidosis is FMF disease, a hereditary familial disease.⁵ Our patient was born in Azerbaijan. We avoided the diagnosis of FMF because she did not describe abdominal pain and fever attacks in his childhood, and she did not have a family history of FMF. Since rheumatoid arthritis is one of the most common causes of reactive amyloidosis, RF and anti-CCP were requested for diagnosis. The results were negative, and the diagnosis of rheumatoid arthritis was excluded because the patient did not have a history of morning stiffness or involvement in small joints.

The patient, who was evaluated for another cause, ankylosing spondylitis (AS), had no symptoms that would meet the diagnostic criteria and no findings suggestive of inflammatory bowel disease. She has no enthesopathy. No uveitis was detected in the eye examination. In the sacroiliac joint X-ray, both sacroiliac joints were found to be of normal width, but periarticular sclerosis was noted on both sides (bilateral grade 2 according to New York criteria). For the presence of sacroiliitis or axial spondyloarthritis or AS, the presence of bilateral stage 2 or unilateral at least stage 3 sacroiliitis is required.⁶ However, considering the clinical symptoms, the diagnosis of AS was dismissed. Distal interphalangeal involvement, asymmetric sacroiliitis or spondylitis, dactylitis

and enthesitis can be seen in psoriatic arthritis.⁷ In a patient diagnosed with psoriasis, this involvement may be associated with psoriatic spondyloarthritis.

Systemic lupus erythematosus (SLE), Behçet's disease and Takayasu's arteritis are rare causes of reactive amyloidosis.⁵ In our patient, malar rash, photosensitivity; Since ANA, anti-dsDNA, Anti-Sm, Anti-Ro, Anti-La antibodies were not positive, the diagnosis of SLE was ruled out. Our patient had good peripheral pulses. She had no findings to suspect Takayasu's arteritis. HLA B51 alone was not found to be significant in a patient with female gender, but HLA B51 single allele positivity, who did not describe oral aphthae, genital ulcer, pustular rash, or uveitis attack. Although the HLA-B51 antigen differs between ethnic groups, it is present in approximately 20% healthy individuals.⁸

Gregory et al.⁴ reported psoriasis-associated amyloidosis in 18 patients in their 1950-1992 Mayo Clinic screening. Twelve of them had psoriatic arthritis, and four had pustular psoriasis.⁴ Psoriatic arthritis is chronic inflammatory arthritis associated with psoriasis involving the peripheral joint, spine, and entheses area.⁷ It can be thought that this situation facilitates the development of amyloidosis in psoriatic patients.² It can be considered a psoriatic arthritis attack in our patient who had arthritis symptoms six weeks before hospitalization. In amyloidosis accompanying psoriasis, amyloid deposition is frequently seen in the kidneys, gastrointestinal tract, or both systems. When amyloidosis is detected in most cases, the diagnosis of psoriasis is an average of 14.4 years.² In our case, the diagnosis of psoriasis was made two years ago, which is a very early period for the detection of amyloidosis, according to the literature.

Considering the patient's recent high-dose NSAID, the nephrotic syndrome may be thought to be due to NSAID use. The association of NSAIDs and the nephrotic syndrome has been recognized for a long time, and minimal change disease and membranous nephropathy were the most common findings in kidney biopsies of these patients.⁹ Considering that minimal change disease does not show any results on light microscopy, a biopsy may have enabled us to diagnose amyloidosis incidentally. The patient's current nephrotic

syndrome may be due to NSAID use, amyloid deposition, or both. Arthropathy is also present in 85% of patients with amyloidosis accompanying psoriasis.^{2,3} In the study of Gregory et al.⁴, most of the patients had psoriatic arthritis or pustular psoriasis. In other words, it is seen that amyloidosis can develop in patients with more severe psoriasis. Amyloid deposition accompanying psoriasis is a rare complication detected later than other diseases accompanied by amyloidosis. However, in our case, it was observed that this situation was not always late, but amyloid deposition could be observed earlier.

Conclusion

Colchicine treatment is mandatory to prevent AA amyloidosis in FMF, which is one of the important diseases that cause amyloid deposition.¹⁰ Suppose the amyloid deposition is detected in such a short time in psoriasis. Would it be a correct approach to give colchicine prophylaxis to every patient, as in FMF, to prevent amyloidosis? Other similar observations are needed to answer this question.

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Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contribution

Study Conception: MS; Study Design: MS; Supervision: AY, AO, MY, MG, KD; Materials: MS, SEGB; Data Collection and/or Processing: MS, SEGB; Statistical Analysis and/or Data Interpretation: MS, SEGB; Literature Review: MS; Manuscript Preparation: MS, AE; Critical Review: AE.

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