## Systemic, secondary amyloidosis in a patient with psoriatic arthritis

Sirs,

Before the use of antibiotics, systemic secondary amyloidosis (AA amyloidosis) was most often associated with chronic infectious diseases. Since the advent of modern antibiotic therapy, the spectrum has shifted to chronic, long-standing rheumatic disorders (1).

Our patient, a Caucasian female, was referred at the age of 54 years to the National Institute of Rheumatology and Physiotherapy for the treatment of very active symmetric, seronegative polyarthritis which had started only 2 months earlier on the MCP and PIP joints of the hands, and on the knees and ankles. She was placed on low dose prednisone treatment; hydroxychloroquine and later parental gold therapy were discontinued after a few months due to poor compliance.

At the age of 57 years the patient developed corona psoriatica, and her nails became pitted. Her psoriasis required only topical tar applications. From the age of 57 a mild proteinuria with microscopic haematuria was noted. Microscopy and culture of the urine proved sterile. An intravenous pyelogram showed ureterolithiasis in the left urether. After surgical removal of the ureterolith (ureterotomia) her haematuria ceased, but the proteinuria remained. According to the Moll and Wright classification her initially rheumatoid-like psoriatic arthritis seemed moderatly active between the age of 57 to 60, synovitis was observed only in a few small joints of the hand, but her knees continued to show flexion deformities, and she suffered from stiffness in the spine. On X-rays no mutilation, but periostitis on the hands and bilateral sacroiliitis were observed.

Laboratory test results indicated unusually active disease: Westergreen was frequently over 100 mm/h, CRP titer was over 2048, and chronic anaemia was detected. Serum protein electrophoresis revealed no monoclonal peak, only low serum albulmin and an increase in the gamma globulin level. From the age of 60 her general condition gradually deteriorated, she became bedridden and lost 44 kg. Her bucca biopsy revealed amyloid deposits. The quantity of proteinuria in her last 3 years ranged between 5-6 gm/24 hr. Blood urea and the se kreatinin level was normal. She died at the age of 63 years in cardiogen shock, 10 days after an acute extensive anterior myocardial infarction.

Autopsy proved coronaria thrombosis to be the cause of death. Amyloidosis was also observed in the kidneys, pancreas, gastrointestinal tract, thyroid gland, heart, liver, lungs, and synovial membrane, in addition to the skin. In every organ the severity of amyloidosis in the small, medium and large arteries, venules, and in the collagen and reticular fibres was estimated to be from 0-3 points. We found the most severe amyloid deposition in the skin, adrenal glands, pancreas and kidneys (Table I). Comparing different tissue structures, the most severe amyloid deposits were located in the small and medium size arteries and reticular fibres.

The first report of a relationship between amyloidosis and psoriatic arthritis was published by Ferguson *et al*. in 1969 (2). Reviewing the literature, we found only a few psoriatic arthritis patients with secondary amyloidosis reported (3-6).

In our case the AA is thought to be the complication of a longstanding, severe, active psoriatic arthritis,since no chronic bacterial inflammation or other clinical or histological evidence of chronic illness was found. Whereas in primary cutaneous amyloidosis amyloid deposits are usually confined to the upper proportion of the dermis, in secondary systemic amyloidosis amyloid deposition can be found in the deeper areas of the reticular dermis, around the appendages, in the blood vessel walls and in the subcutis (4, 7).

There is no effective treatment for the removal of amyloid deposits. Most therapeutic regimens are therefore directed towards suppressing the inflammatory response in order to control the underlying disease and achieve a possible clinical recovery from reactive systemic amyloidosis.

**Table I.** Extent of amyloid deposition in various organs and organ structures.

| | a | A | AA | v | V | VV | coll | ret | n | Average | Extent |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Skin | 3 | 3 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 1.8889 | 17 |
| Adrenal gland | 3 | 3 | 3 | 0 | 2 | 3 | 0 | 0 | 0 | 1.5556 | 14 |
| Pancreas | 3 | 2 | 1 | 1 | 2 | 3 | 1 | 1 | 0 | 1.5556 | 14 |
| Kidneys | 3 | 3 | 1 | 0 | 2 | 1 | 1 | 1 | 1 | 1.4444 | 13 |
| Intestines | 3 | 1.5 | 0 | 2 | 1 | 1 | 1 | 3 | 0 | 1.3889 | 12.5 |
| Heart | 3 | 2 | 1 | 1 | 1 | 0 | 2 | 2 | 0 | 1.3333 | 12 |
| Spleen | 3 | 3 | 0 | 3 | 0 | 0 | 0 | 2 | 0 | 1.2222 | 11 |
| Thyroid gland | 3 | 2 | 1 | 0 | 2 | 2 | 0 | 0 | 0 | 1.1111 | 10 |
| Liver | 2 | 2 | 0 | 0 | 0 | 1 | 0 | 2 | 0 | 0.7778 | 7 |
| Muscles | 2 | 1 | 0 | 0 | 0 | 1 | 1 | 2 | 0 | 0.7778 | 7 |
| Femoral head | 2 | 1 | 0 | 0 | 0 | 0 | 1.5 | 1.5 | 0 | 0.6667 | 6 |
| Synovial membrane | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0.2222 | 2 |
| Acetabulum | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.2222 | 2 |
| Lungs | 1 | 0.5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.1667 | 1.5 |
| Peripheral nerve | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.1111 | 1 |
| Brain | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

a: small arteries, A: medium arteries, AA: large arteries, v: small veins, V: medium veins, VV: large veins, coll: collagene fibres, ret: reticular fibres n: nerves.

I. UJFALUSSY[1]
M. BÉLY[2]
É. KOÓ[1]
M. SESZTÁK[3]

*[1]Department "D", [2]Pathological Department, and [3]Department "A" of the National Institute of Rheumatology and Physiotherapy, Budapest, Hungary.*

*Please address correspondence and reprint requests to: Ilona Ujfalussy, ORFI, Department D, Budapest, Árpád f. u 7, Hungary, H-1023.*

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