The coexistence of SLE and beta-thalassemia is extremely rare. To date, only two cases of sickle cell/beta-thalassemia with SLE have been described (2, 3). In addition, SLE patients with beta-thalassemia trait have been reported (5), while osteonecrosis in a patient with hemoglobin E beta-thalassemia has been recently published (6). At the best of our knowledge this is the first report of the coexistence of SLE and beta-thalassemia major.

The relationship between the two diseases remains unclear. Genetic factors may contribute to their pathogenesis. Our patient had gene mutation resulting in abnormal beta-chain synthesis and HLA typing: B18, DR10 that have been associated with lupus. Furthermore, multiple transfusions may alter the immunological response of thalassemia patients. Experimental transfusion of plasma containing autoantibodies has been shown to lead to autoantibody formation. Therefore, the immune status of the patient as well as the effect of multiple allo- and autoantigenic blood transfusions can induce antibody formation. The perpetuation of antibody formation may result from the continuous stimulation of the immune system. Autoantibody production has been described in thalassemia patients (7) with variable clinical significance. They may exhibit the characteristics of natural autoantibodies or, under unclear circumstances, they may become the pathogenic autoantibodies that are found in SLE. It is known that T cell subsets play a pivotal role in the pathogenesis of SLE. On the other hand, T lymphocytes from thalassemia patients are activated in vivo and they present several abnormalities (2). Thus, the lymphocyte background in thalassemia patients may under the influence of a triggering factor contribute to the development of SLE. Our patient had a low CD4<sub>+</sub>/CD8<sub>+</sub> ratio, which has been described in lupus nephritis patients (8). In addition, desferoxamine, which modifies T-cell-mediated immune responses, may also play a role in SLE expression (9, 10).

In conclusion, we described a patient with beta-thalassemia major and SLE. Further studies will clarify whether the association of these two diseases is real or if it constitutes an occasional coexistence.

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revealed that her left kidney had become nonfunctional. Left nephrectomy was carried out, after which her hypertension was controlled. However, after 6 months her hypertension returned and radiological examination showed multiple stenoses throughout a long segment from the origin of the right renal artery. Based on these findings the diagnosis of “unclassified vasculitis” was made and therapy with prednisolone and cyclophosphamide was started. When the patient was admitted to our clinic, she had a history of low back pain that was worse in the early morning hours and after prolonged rest. The pain diminished with physical activity. On physical examination, she showed growth retardation (height and weight), and an abnormal Schober test. A complete blood count, urinalysis and kidney function tests were normal. She showed growth retardation (height and weight), and an abnormal Schober test. A complete blood count, urinalysis and kidney function tests were normal. A murmur was heard over the carotid regions bilaterally. Articular examination revealed severe low back pain that was not specific for diseases including rheumatoid arthritis, Still’s disease, polymyositis, polymyalgia rheumatica and ankylosing spondylitis. She was negative for HLA B27 and RF. When the patient was admitted to our clinic, she had a history of low back pain that was worse in the early morning hours and after prolonged rest. The pain diminished with physical activity. On physical examination, she showed growth retardation (height and weight), and an abnormal Schober test. Articular examination revealed severe low back pain that was not specific for diseases including rheumatoid arthritis, Still’s disease, polymyositis, polymyalgia rheumatica and ankylosing spondylitis. She was negative for HLA B27 and RF. One month after the beginning of treatment ESR and CRP values returned to normal and control imaging studies are scheduled for 6 months later.

The case presented here shows that TA remains a challenge for clinicians. It has been reported to occur in association with certain diseases including rheumatoid arthritis, Still’s disease, polymyositis, polymyalgia rheumatica and ankylosing spondylitis (3). Sacroiliac MRI study to the etiology of our patient’s inflammatory low back pain revealed bilateral sacroilitis. The absence of a family history, uveitis and HLA B27 are consistent with the diagnosis of seronegative juvenile onset spondyloarthropathy. Review of the published work revealed only a few patients with seronegative spondyloarthropathy and a small number of these were in the pediatric age (4-10). The cause of the occurrence of two such rare diseases together is unknown, but a common immunological mechanism or undefined genetic susceptibilities have been proposed in the pathogenesis of TA and ankylosing spondylitis (1, 4). Our patient’s history and clinical course led us to believe that the two diseases had a simultaneous onset. However, the predominance of symptoms such as leg and low back pain that are not specific for TA resulted in the delay of the diagnosis and the progression of arterial involvement.

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Fig. 1. Angiography showing stenosis of right renal artery and the segmental occlusion of superior mesenteric artery (a), high grade stenosis of the right common carotid artery (b), and stenosis in the superficial femoral artery (c). Contrast-enhanced axial T1-weighted MR image reveals bilateral sacroilitis (d).

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