STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Polymyositis induced by atorvastatin

Zapalenie wielomięśniowe wywołane przez atorwastatynę

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We present the case of a 61-year-old female who developed polymyositis after atorvastatin treatment. She was admitted to hospital in July 2014 with fever, weight loss, myalgia, and limb weakness. Additionally in anamnesis, the following are seen: diabetes type 2, hypertension, and hypercholesterolaemia (treated with atorvastatin 20 mg daily for 6 months). In physical examination muscle weakness was observed (upper limbs 4° and lower limbs 3° in Lovett scale). Laboratory tests revealed increased levels of creatine kinase (CK) — 3977 U/L, alanine aminotransferase (ALT) — 176 U/L and aspartate aminotransferase (AST) — 128 U/l. In electromyography myogenic-dominant damage was described. Atorvastatin was withdrawn. Methylprednisolone pulse therapy was started (3 × 500 mg). Then the patient, with a diagnosis of myopathy, was sent (in August 2014) to the Department of Rheumatology for continuation of treatment. The muscle weakness was still observed (upper limbs 4° and lower limbs 3° in Lovett scale) and laboratory results were as follows: CK 1299.7 U/L, ALT 133.2 U/L, AST 50.5 U/L, aldolase 15.6 U/L, lactate dehydrogenase (LDH) — 742 U/L. Antinuclear antibodies were negative. The presence of anti-SS-A antibodies was observed (Fig. 1). The myositis profile was negative (Fig. 2). In spite of such results the clinical diagnosis of atorvastatin-induced polymyositis was made. Treatment with methylprednisolone pulse (3 × 500 mg) was continued with subsequent oral prednisone therapy (25 mg daily). In control after 1 month a slight improvement of lower limbs muscle strength was observed (4° in Lovett scale, upper limbs remain constant). Laboratory tests revealed the following: CK 873.5 U/L, ALT 77.6 U/L, AST 39 U/L, LDH 704 U/L, aldolase 9.7 U/L. We continued treatment with methylprednisolone pulse (3 × 500 mg). Subsequently the oral prednisone dose was reduced to 20 mg daily. The latest observation was made in November 2014. The upper limbs muscle strength was normal and lower limbs remained constant (4° in Lovett scale). Laboratory results were as follows: CK 259.3 U/L, ALT 40.5 U/L, AST 23 U/L, LDH 401 U/L. The treatment with methylprednisolone pulse was continued, but the dose was reduced (3 × 250 mg). Oral prednisone dose was also decreased to 17.5 mg. Because of permanent hypercholesterolaemia ezetimibe was prescribed. Now patient is under our observation with planned control. Our case describes patients who develop polymyositis after atorvastatin therapy. Because of the non-autoimmune origin of the disease the patient did not reveal a characteristic immunological pattern. Withdrawing the drug did not improve the symptoms. Immunosuppressive therapy with corticosteroids was necessary. Statins may be a cause of the whole spectrum of muscle damage, but full-blown polymyositis or dermatomyositis are rather rare. It is crucial to establish any association with a drug as soon as possible because a key role in the treatment is played by the elimination of any agent causing the disease.

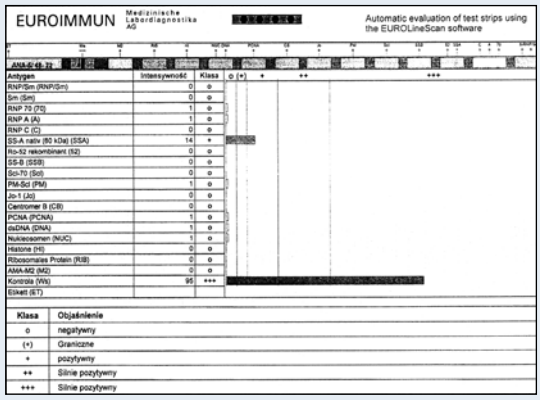

Figure 1. Antinuclear antibodies profile

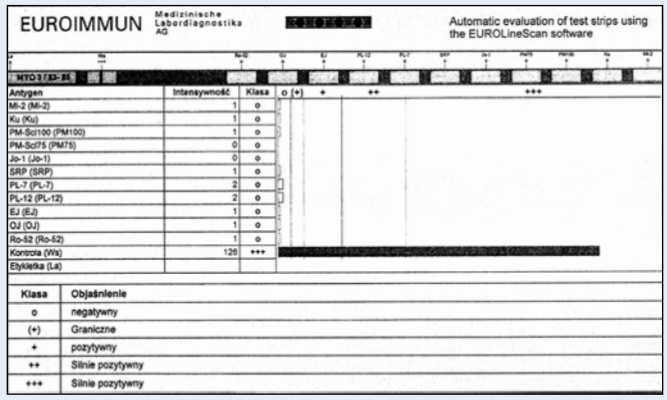

Figure 2. Myositis profile

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