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CLINICAL CASE

COMPLICATIONS CAUSED BY THE ACTION OF STATINS

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Statins are a group of hypolipidemic agents. The mechanism of action of all statins is the competitive inhibition of the enzyme 3-hydroxyl-3methyl-glutaryl coenzyme A-reductase, which catalyzes the initial and intermediate stages of biosynthesis of cholesterol (transformation of HMG-CoA into the mevolat - precursor of sterols) in the liver. Inhibition of cholesterol synthesis in the liver leads to an increase in the density of low density lipoprotein receptors in hepatocytes and in accordance with the increase in the cholesterol catabolism of low density lipoprotein. In addition, there is a decrease in the formation of low density lipoprotein as a result of inhibition of the synthesis of their precursors - very low density lipoproteins in the liver. All this leads to a decrease in blood plasma levels of total cholesterol, low density lipoprotein cholesterol and very low density lipoprotein, and to a lesser extent triglycerides; an increase in the level of anti-atherogenic lipoproteins of high density was found. When using statins, side effects from different organs and systems of the body are possible. Severe side effects of statins are side effects of the bone and muscle system. Often among them are statin myopathy and rhabdomyolysis. These phenomena are associated with the fact that statins accelerate apoptosis of muscle cells, may cause damage to the membrane ion channels, inhibit the synthesis of selenoproteins. Light forms of myopathy can only manifest themselves by increasing the concentration of blood creatine phosphokinase. Rhabdomyolysis, which is manifested by pain in the calf muscles, in the lumbar region or throughout the body, nausea, vomiting, changes in the color of urine to red or brown (due to the release of myoglobin in urine) are observed in approximately 4 cases per million patients on year in the use of modern statins. Despite criticism, the drugs of the statin group are among the leaders in pharmaceutical sales in the world. So in 2004, expenditures on hypolipidemic drugs in the world amounted to a total of $ 30 billion. Over the years and the development of technology, this financial law is not decreasing.

In this regard, we consider it necessary to lead a clinical case that illustrates this problem.

Patient K. (Dec. 18, 1983) admited to the central ambulance department, complaining of an increase in muscle pain in the thigh during the last week. Acute pain and swelling in the ankles. Fever, shortness of breath, abdominal discomfort, headache and dizziness were dismissed during the initial inspection.

It is known from the anamnesis that the patient regularly uses statins for the treatment of hypercholesterolemia and Duodarf (active ingredient: dutasteride, tamsulosin hydrochloride) due to prostate hyperplasia. During examination: a patient of moderate severity, BMI within the age range corresponds to normal nutrition. No jaundice. No cyanosis. There is no shortness of breath at rest and during activity. Turgor of skin is slightly lowered but good for age standards. There are no pathological abnormalities in the head / neck areas. In the lungs, vesicular respiration, wheezing or pathological noise was not detected, Eupnoe. The heart sounds pure, rhythmic, no noise. Pulse normal. Unperformable peripheral pulse status. Abdomen is soft, there are no symptoms of peritonitis or muscle resistance. The liver and spleen are not palpable. Mr. Pasternatsky's negative. There is no pain in the spine. The upper and lower extremities are actively and passively free-moving. In the area of the foot between the middle bone and thumb, the peripheral light edema, palpable and characterized by minimal redness and a painful symptom. No neurological deficiencies, clear language. No paresis. Good coordination. The patient focuses in time and space.

Diagnoses:

1. Rhabdomyolysis from statins therapy (pravastatin)
2. A severe gout attack between metartatic bones and thumb
3. Arterial hypertension
4. Hyperplasia of the prostate gland Epicrisis:

In laboratory chemistry, we see a increasing of creatinine kinase 429 U / l (N = up to 190 od / l), an increase in liver tests GOT 102 (N = less than 50), GPT 79 (N = less than 50), and an increase in bilirubin to

1.3 mg / dl (N = 1.1). LDH 335 (N = 250), c-reactive protein 11.3 (N = 0.5 mg / dcl). Instrumentally: ultrasound scan, chest X-ray. - pathology is not detected. There was no systemic inflammation, trauma or other pathological processes during clinical examinations.

In the context of the clinical picture, acute rhabdomyolysis was discussed during statin therapy. In this regard, pravastatin has been discontinued. With repeated biochemical examination, we see a marked decrease in liver tests, as well as levels of creatinine kinase.

The patient receives symptomatic therapy against pain, electrolyte infusion therapy and physical decreasing of physical activity. Since edema was seen by us as a manifestation of acute gout attack, initial therapy with prednisolone for 3 days was prescribed, as well as allopurinol in a simple dosage. All of the above clinical actions have led to a reduction in complaints.

Pathogenetic rationale: The patient is more likely to have statins begin to destroy muscle cells, which could lead to renal and hepatic insufficiency without emergency intervention. The withdrawal of statins and symptomatic treatment corrected the patient's condition.