Srčani troponin I je najbolji izbor za otkrivanje oštećenja miokarda u bolesnika s upalnim miopatijama

Cardiac troponin I is the best choice in detecting myocardial damage in patients with inflammatory myopathies

Tatjana Zekić¹,
Luka Bastiančić^{1*},
Stanislav Laginja²,
Dragana Grohovac¹,
Tomislav Jakljević¹,
Srđan Novak¹,
Luka Zaputović¹

¹Medicinski fakultet Sveučilišta u Rijeci, Klinički bolnički centar Rijeka, Rijeka, Hrvatska

¹University of Rijeka School of Medicine, University Hospital Center Rijeka, Rijeka, Croatia

²Thalassotherapia Opatija – Klinika za liječenje, rehabilitaciju i prevenciju bolesti srca i krvnih žila, Opatija, Hrvatska

²Thalassotherapia Opatija – Clinic for treatment, rehabilitation and prevention of cardiovascular disease, Opatija, Croatia

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***ADDRESS FOR CORRESPONDENCE:** Klinički bolnički centar Rijeka, Kneževa 42, HR-51000 Rijeka, Croatia. / Phone: +385-91-7974-072 / E-mail: lbastiancic@hotmail.com

UVOD: Srčani troponin (cTnT) se smatra osjetljivim biljekom oštećenja kardiomiocita. Troponin je kontraktilni proteinski kompleks sastavljen od tri podjedinice (troponin C, I i T), primarno vezane za miofibrile i manje slobodno prisutne u citoplazmi. Izoforme T i I su specifične za miokard. Ekspresija cTnT je u manjoj mjeri prisutna i u skeletnom mišiću te je stoga moguća lažna pozitivnost u bolesnika s upalnim miopatijama i stanjima s nekrozom skeletnih mišića. Kod uporabe imunoeseja lažno pozitivni rezultati mogući su i u autoimunim bolestima radi križne reaktivnosti prisutnih protutijela.

PRIKAZ SLUČAJA: 53-godišnja prethodno zdrava, žena urednog fizikalnog nalaza, javila se u hitnu službu zbog umora i progresivne slabosti mišića. Inicijalni laboratorijski nalazi, krvna slika elektroliti, parametri bubrežne funkcije i analiza urina bili su u granicama normale, dok su cTnT, kreatin kinaza (CK) i jetreni enzimi bili znatno povišeni. Negirala je bolove u prsima i otežano disanje. Elektrokardiogram (EKG), radiogram prsnih organa, ultrazvuk abdomena i srca bili su uredni. Bolesnica je zbog sumnje na upalnu miopatiju hospitalizirana na Odjelu kliničke imunologije i reumatologije, gdje je potvrđena dijagnoza teškog oblika polimiozitisa s razvojem tetraplegije i slabim odgovorom na imunosupresivno liječenje. Visoka razina cTnT i CK perzistirala je tijekom šest tjedana. Srčani troponin I (cTnI) je bio normalan, kontrolni EKG i ultrazvuk srca također.

ZAKLJUČAK: Razina cTnI ima najveću specifičnost i stoga je najpouzdaniji biljeg oštećenja kardiomiocita u bolesnika s upalnim miopatijama.

INTRODUCTION: Cardiac troponin T (cTnT) is considered to be a highly sensitive marker of myocardial cell damage. Troponin is a contractile protein complex composed of three subunits (troponin C, I and T) primarily bound to myofibrils and less found free in the cytoplasm. Isoforms T and I are specific for the heart. Expression of the cTnT was observed to a lesser extent in skeletal muscle and therefore allows for false positivity in patients with inflammatory myopathies and conditions with skeletal muscle necrosis. False-positive results are also possible when using immunoassays in autoimmune diseases, because of the presence of antibodies and cross-reactivity.

CASE REPORT: A 53-year-old, previously healthy, Caucasian woman presented at the emergency department due to fatigue and muscle weakness, with a completely normal physical finding. Initial laboratory analysis found normal complete blood count, electrolytes, kidney function and urine tests but extreme elevation of cTnT, creatine-kinase (CK) and hepatic values. She had no chest pain or dyspnea. Electrocardiogram (ECG), chest X-ray, abdominal ultrasound and echocardiography were normal. Inflammatory myopathy was suspected and the patient was hospitalized at the Department of Clinical Immunology and Rheumatology, where the diagnosis of severe polymyositis with development of tetraplegia and poor response to immunosuppressive therapy was confirmed. Cardiac TnT and CK levels remained high during six weeks. Cardiac troponin I (cTnI) level was tested and found normal, as well as control ECG and echocardiography.

CONCLUSION: cTnI has the highest specificity and is the most reliable biomarker of myocardial cell damage in patients with inflammatory myopathies.

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