Akutni infarkt miokarda u hipereozinofilnom sindromu

Acute myocardial infarction associated with hypereosinophilic syndrome

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Hipereozinofilni sindrom (HES) je poremećaj pretjerane produkcije eozinofila s multiorganskom infiltracijom. Kardiovaskularne komplikacije se javljaju u oko 40% bolesnika, a očituju se srčanim popuštanjem (75%), mitralnom regurgitacijom (42%), miokarditisom, endokardijalnom fibrozom i muralnim trombom (57%).¹ Infarkt miokarda je rijetko zabilježena komplikacija HES-a, a u literaturi je opisan kao posljedica embolijskih zbiljanja uslijed endokardijalne fibroze i tromba u lijevoj klijetki.²

Prikazani bolesnik ima 38 godina i postavljenu dijagnozu HES-a. U ranom stupnju bolesti razvija akutni koronarni sindrom (ACS) s jasnom kliničkom slikom i porastom kardioselektivnih enzima (TnI 28,99 g/L). U elektrokardiografskom zapisu je prisutan sinusni ritam, blok desne grane te ishemijska anterolateralna. Ehokardiografski sistolička funkcija bila je reducirana (EF 30%) s hipokinezijom apikoanterolateralnog segmenta lijeve klijetke. Po terapiji s kortikosteroidima i hidroksiurejom, koja je standardna početna terapija HES-a, uz terapiju srčanog popuštanja i ACS, postigne se kratkotrajni oporavak bolesnika. No, ehokardiografski nalaz je ostao nepromijenjen, uz progresiju dijasoličke disfunkcije po tipu restrikcije, a u kasnijem tijeku se formira muralni tromb u hipokinetičnom segmentu lijeve klijetke. U akutnoj fazi infarkta miokarda, bolesnik je reanimiran uslijed ventrikularne fibrilacije. U daljnjem tijeku se razvijaju gastrointestinalne komplikacije, multiple infarkte slezene i apscese u trbušnoj šupljini koji su liječeni kirurškim pristupom. Postoperativni tijek bolesti je kompliciran sepsom s multiorganskim zatajivanjem.

AKZ je rijetka kardijalna komplikacija HES-a. Upućuje na endokardijalnu fibrozu i uznapredovalu srčanu bolest koja zahtijeva agresivniji terapijski pristup, a često i kirurško liječenje. Bolje razumijevanje patogeneze bolesti bi trebalo pomoći u boljem liječenju i boljoj prognozi ovih bolesnika. Novija terapija s monoklonalnim protutijelom daje obećavajuće rezultate.

Hypereosinophilic syndrome (HES) is a disorder of excessive production of eosinophils with multiple organ infiltration. Cardiovascular complications occur in about 40% of patients and are reflected in heart failure (75%), mitral regurgitation (42%), myocarditis, endocardial fibrosis and mural thrombus (57%).¹ Myocardial infarction (MI) is a rare complication of HES, and it is described as a result of embolic events due to endocardial fibrosis and thrombus formation in the left ventricle.²

We present a case of 38-years old patient diagnosed with HES. In the early stage of disease he developed acute coronary syndrome (ACS) with a typical clinical manifestation and increase of cardioselective enzyme (TnI 28.99 g/L). The ECG showed a record of sinus rhythm, right bundle branch block and anterolateral ischemia. Transthoracic echocardiography showed reduced left ventricular systolic function (EF 30%) with hypokinesia apicoanterolateral segment. After treatment with corticosteroids and hydroxyurea, the first-line therapy for HES, and treatment of heart failure and ACS, a short-term recovery of patient was reached. Echocardiographic findings remained unchanged, with the progression of diastolic dysfunction (restriction type), forming of the mural thrombus in hypokinetic segment of the left ventricle. In the acute phase of MI, the patient was resuscitated due to ventricular fibrillation. In the further course gastrointestinal complications developed, with multiple splenic infarctions and abdominal abscesses which were treated surgically. The postoperative period was complicated with sepsis and multiorgan failure.

ACS is a rare cardiac complication of HES. It refers to the endocardial fibrosis and advanced heart disease that requires aggressive therapeutic approach and often surgical treatment. A better understanding of the pathogenesis of the HES should help in better treatment and prognosis of these patients. Newer therapy with monoclonal antibodies gives promising results.

LITERATURE

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