Ruptura korde mitralnog zaliska i akutni glomerulonefritis – slučaj ili povezani događaj

Rupture of mitral valve chordae and acute glomerulonephritis – case or related event?

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Uvod: Jedan od najčešćih uzroka rupture kordi mitralnog zaliska (MZ) je miksotomozno promijenjena struktura zaliska^{1,2}, što dovodi do akutnog volumnog opterećenja i progresivnog srčanog popuštanja. Predstavljamo bolesnika s teškim volumnim opterećenjem uslijed teške mitralne regurgitacije i akutnog glomerulonefritisa. Ključno pitanje bilo je odrediti što je bio precipitirajući događaj te što je potrebno prvo liječiti.

Opis slučaja: Šezdesetogodišnji bolesnik s od ranije poznatim prolapsom mitralnog zaliska i blagom mitralnom regurgitacijom hospitaliziran je u lokalnoj bolnici u slici akutnog srčanog i bubrežnog popuštanja. Dva mjeseca prije hospitalizacije bolesnik se žalio na intoleranciju napora, ortopneju, noćno prenožavanje i subfebrilitet (do 37,5 °C), a zbog akutnog tonzilofaringitisa liječen je antibioticima. U trenutku prijema bolesnik je izrazito narušenog funkcijskog statusa (NYHA IV. stupanj) s akutnim nefritičkim sindromom što je zahtijevalo intermitentne hemodijalize. Nakon tjedan dana bolesnik je premješten Klinički bolnički centar Zagreb u slici plućnog edema, hemodinamski nestabilan, pod sumnjom na endokarditis ali uz sterilne hemokulture. Trodimenzionalnim transtorakalnim i transezofagusnim ultrazvukom srca ustanovljena je ekscentrična hipertrofija lijeve klijetke s blago narušenom sistoličkom funkcijom, uvećana lijeva pretklijetka (100 ml), teška mitralna regurgitacija (volumen regurgitacije 65 ml) uslijed rupture korde s potpunim izvrtnjem P1 (uz prolaps ostalih segmenata). Endokarditične vegetacije mitralnog zaliska nisu nađene. Akutno bubrežno popuštanje dodatno je volumno opteretilo lijevu klijetku što je izazvalo naprezanje miksotomoznog mitralnog aparata prvenstveno kordi (slika 1). Pucanje korde izazvalo je potom masivnu mitralnu regurgitaciju i akutno srčano popuštanje. Nakon početne stabilizacije nastojali smo ustanoviti uzrok akutnog nefritičkog sindroma. Ultrazvučnim pregledom bubrega utvrđeno je difuzno obostrano hiperehogeno zadebljanje bubrežnog parenhima. Nakon seroloških ispitivanja i biopsije bubrega ustanovljen je brzoprogresivni glomerulonefritis (pauci imuni c-ANCA pozitivan). Liječenje je uključivalo boluse kortikosteroida, ciklofosfamid i plazmaferezu uz povremene hemodijalize. Nakon oporavka bubrežne funkci-

Introduction: One of the most common causes of mitral chordae rupture is a myxomatous valve disease^{1,2} that causes acute volume overload and progressive heart failure. We present a patient with severe volume overload due to massive mitral regurgitation and acute glomerulonephritis. The key issue was to determine the precipitating event and which condition should be treated first.

Case report: 60-year-old patient with known mitral valve prolapse (MVP) and mild mitral regurgitation (MR) presented himself in a local hospital as acute heart and renal failure. Two months prior hospitalization he had experienced intolerance to physical exertion, orthopnea, night sweats and fever up to 37.5 °C and has received antibiotics due to tonsillopharyngitis. At the time of admission, he was in functional NYHA IV status with acute nephritic syndrome that required intermittent hemodialysis. One week after, the patient was transferred to University Hospital Centre Zagreb in pulmonary edema, hemodynamically unstable, referred as a suspected endocarditis, although blood cultures were sterile. Three-dimensional transthoracic and transesophageal echocardiography revealed eccentric hypertrophy of the left ventricle with mild reduction of ejection fraction, left atrial enlargement (100ml), severe MR (regurgitant volume 65ml) from a ruptured chordae with complete inversion of the P1 (and prolapse of other segments). Vegetation as sign of endocarditis of the mitral were not found. Acute renal failure contributed to the volume overload of the left ventricle which caused a strain on the myxomatous mitral apparatus that primarily affected the chordae (Figure 1). Chordae rupture led to massive mitral regurgitation and acute heart failure. After initial stabilization, we tried to establish the cause of acute nephritic syndrome. Ultrasound examination of the kidneys found diffuse hyperechogenic thickening of the parenchyma. After serologic testing and renal biopsy, a rapidly progressive glomerulonephritis (GN) (pauci – immune cANCA positive) was diagnosed. Treatment included corticosteroid boluses, cyclophosphamide and plasmapheresis along with intermittent hemodialysis which significantly improved renal function so the patient was prepared for cardiac

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je učinjen je kardiokirurški zahvat zamjene mitralnog zaliska mehaničkom protezom. Postoperativni tijek protiče uredno osim epizode fibrilacije atrijsa koja je uspješno konvertirana elektrokardioverzijom. Patohistološkom analizom ustanovljena je miksotomatozna degeneracija valvule bez bakterijske infiltracije. Narednih šest mjeseci prati se uredan oporavak bolesnika.

Zaključak: Akutni glomerulonefritis progredirao je u bubrežno popuštanje, potom uvjeti teškog volumnog opterećenja izazivaju pucanje korde ranije miksotomatozno promjenjenog mitralnog zaliska s posljedičnim razvojem "circulus vitiosus". U cilju očuvanja i bubrežne i srčane funkcije najprije je započeto liječenje glomerulonefritisa, a nakon stabilizacije proveden je kardiokirurški zahvat uz odlični oporavak bolesnika.

surgery. The patient underwent a mitral valve replacement with a mechanical valve. Postoperative period went uneventful, except paroxysmal atrial fibrillation successfully treated with electrocardioversion. Pathohistological analysis of the resected valve showed myxomatous degeneration without bacterial infiltration. A six months follow up revealed adequate patient recovery.

Conclusion: Acute glomerulonephritis progressed into renal failure, severe volume overload caused chordae rupture of the previously myxomatously altered mitral valve with a consequent development of the "circulus vitiosus". In order to preserve the renal and cardiac function, glomerulonephritis was treated first and after stabilization, cardiac surgery was performed with an excellent recovery of the patient.

FIGURE 1. Upper glomerul showing fibrocellular crescent formation, lower glomerul with cellular crescent formation, signs of acute tubular damage and interstitial inflammation. Three-dimensional ultrasound revealed rupture of mitral valve chordae with complete inversion of the P1.

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