POLISH HEART JOURNAL

Kardiologia Polska
The Official Peer-reviewed Journal of the Polish Cardiac Society since 1957

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ISSN 0022–9032 e-ISSN 1897–4279

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Authors: Piotr Jarosz, Barbara Zdzierak, Agata Wiktorowicz, Artur Dziewierz, Stanisław Bartuś

Article type: Clinical vignette

Received: January 13, 2025

Accepted: February 11, 2025

Early publication date: February 28, 2025

Rapid resolution of severe rheumatic mitral regurgitation following dapagliflozin and torasemide treatment

Piotr Jarosz[1], Barbara Zdzierak[1], Agata Wiktorowicz[1], Artur Dziewierz[1, 2], Stanisław Bartuś[1, 2]

[1]Clinical Department of Cardiology and Cardiovascular Interventions, University Hospital, Kraków, Poland
[2]2nd Department of Cardiology, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland

Correspondence to:
Piotr Jarosz, MD,
Clinical Department of Cardiology
and Cardiovascular Interventions,
University Hospital,
ul. Jakubowskiego 2,
30–688 Kraków, Poland,
phone: +48 12 400 22 62,
e-mail: pjarosz196@gmail.com

A 77-year-old woman with arterial hypertension, treated with ramipril, lercanidipine, and bisoprolol, and a history of gastroesophageal reflux disease presented to the hospital with a severe chest pain and elevated blood pressure (150/74 mm Hg). The electrocardiogram showed ST-segment depression in leads aVF and V5–V6. Laboratory tests revealed elevated high-sensitive troponin, peaking at 70.8 ng/l (reference: <14.0). Due to suspected acute coronary syndrome, emergency coronary angiography was performed and revealed normal coronary anatomy. Transthoracic echocardiography performed prior to invasive coronary artery angiography, demonstrated preserved left ventricular ejection fraction without wall motion abnormalities. However, severe mitral regurgitation (MR) was noted (vena contracta 9 mm, central large jet >50 % of left atrium area, MR volume 48 ml) (Figure 1A–C; Supplementary material, *Video S1*). The left ventricle was normal in size, and no mitral valve prolapse was evident (Supplementary material, *Video S2*). Additional findings included mildly elevated N-terminal pro-B-type natriuretic peptide at 849 pg/ml (reference: <125) and no signs of

pulmonary congestion on chest X-ray. Treatment was augmented simultaneously with torasemide (10 mg) and dapagliflozin (10 mg). Within 2 days of hospitalization patient's symptoms resolved, and blood pressure normalized. Follow-up transthoracic and transoesophageal echocardiography after 5 days showed improvement to mild/moderate MR (Figure 1C–D). The studies revealed thickened mitral valve leaflets with rheumatic changes (Figure 1E). She was discharged in stable condition with scheduled follow-up in the hospital's cardiology clinic.

Organic (primary) MR results from structural abnormalities of the mitral valve apparatus. Common causes include mitral valve prolapse, flail leaflet, rheumatic heart disease, coronary artery disease, infective endocarditis, and collagen vascular disorders. The aetiology determines management strategy [1]. Rheumatic heart disease, a severe sequela of rheumatic fever, can cause MR at any age [2]. It affects the mitral valve in up to 50% of cases, leading to insufficiency, mitral stenosis, or both. Rheumatic valves exhibit fibrous thickening, oedema, minimal calcification, non-fused commissures, annular dilatation, and anterior chordal elongation. In patients with overt heart failure (HF), pharmacotherapy should follow standard HF guidelines, as no specific adjustments are recommended for primary MR. While sodium-glucose co-transporter 2 inhibitors are cornerstone HF treatments, evidence for their efficacy in organic rheumatic MR remains limited, though studies demonstrate benefits with dapagliflozin in functional MR [3]. Although, the EFFORT study [3] examined functional MR over a 12-month follow-up period and used ertugliflozin rather than dapagliflozin. Although a drug class effect might be expected, potential differences in outcomes between specific medications cannot be ruled out. The rapid improvement in MR severity following hemodynamic optimization in our case suggests a potentially mixed mechanism, with an acute functional component superimposed on underlying rheumatic valve disease. Given the swift resolution of the valvar defect, we hypothesize there may be a synergistic effect of dapagliflozin and torasemide. However, further studies are needed to elucidate the role of sodium-glucose co-transporter 2 inhibitors in managing primary MR [4].

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

Article information

Conflict of interest: None declared.

Funding: None.

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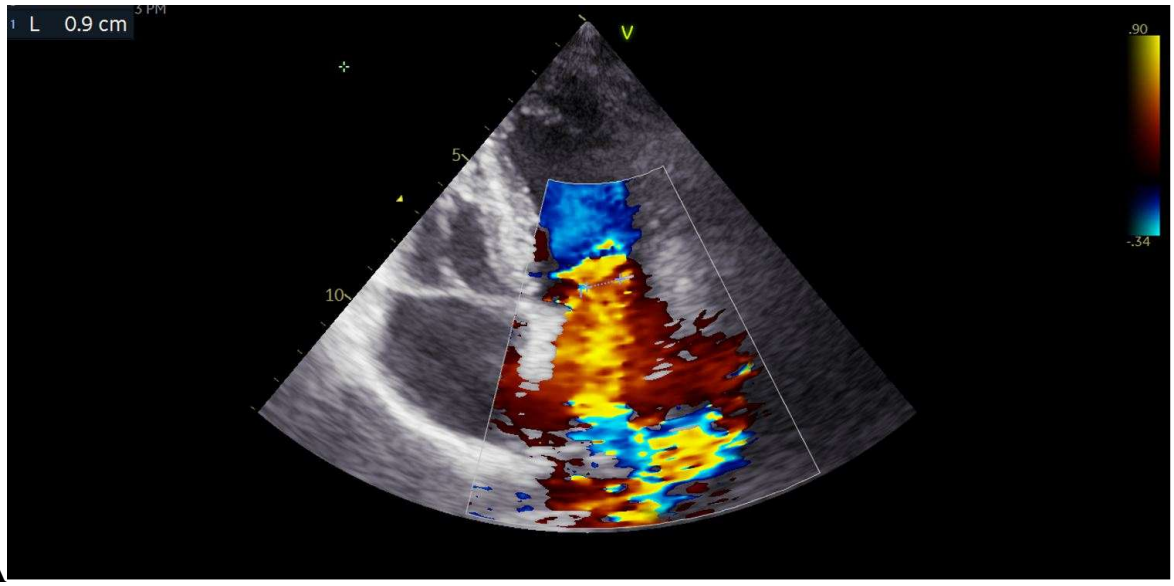
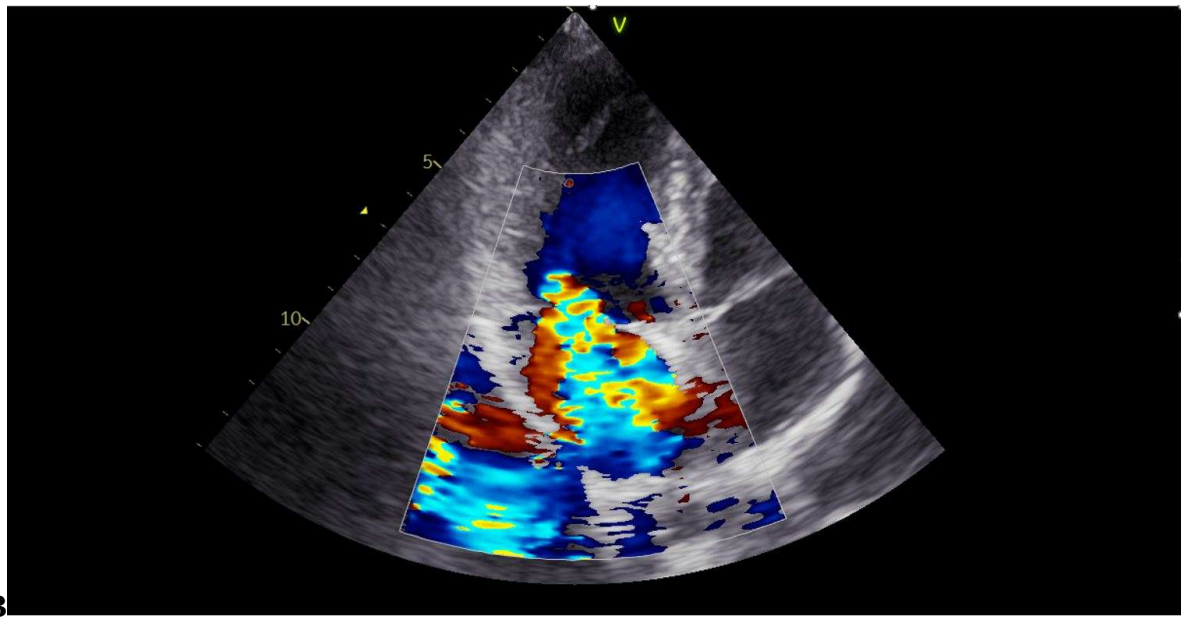


A

B



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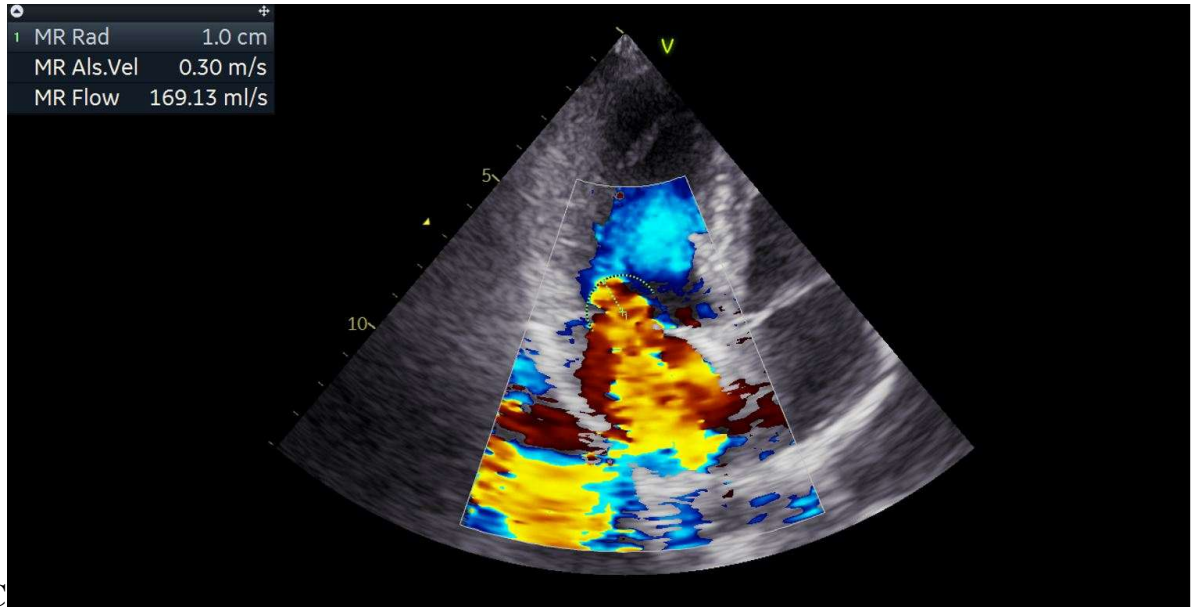

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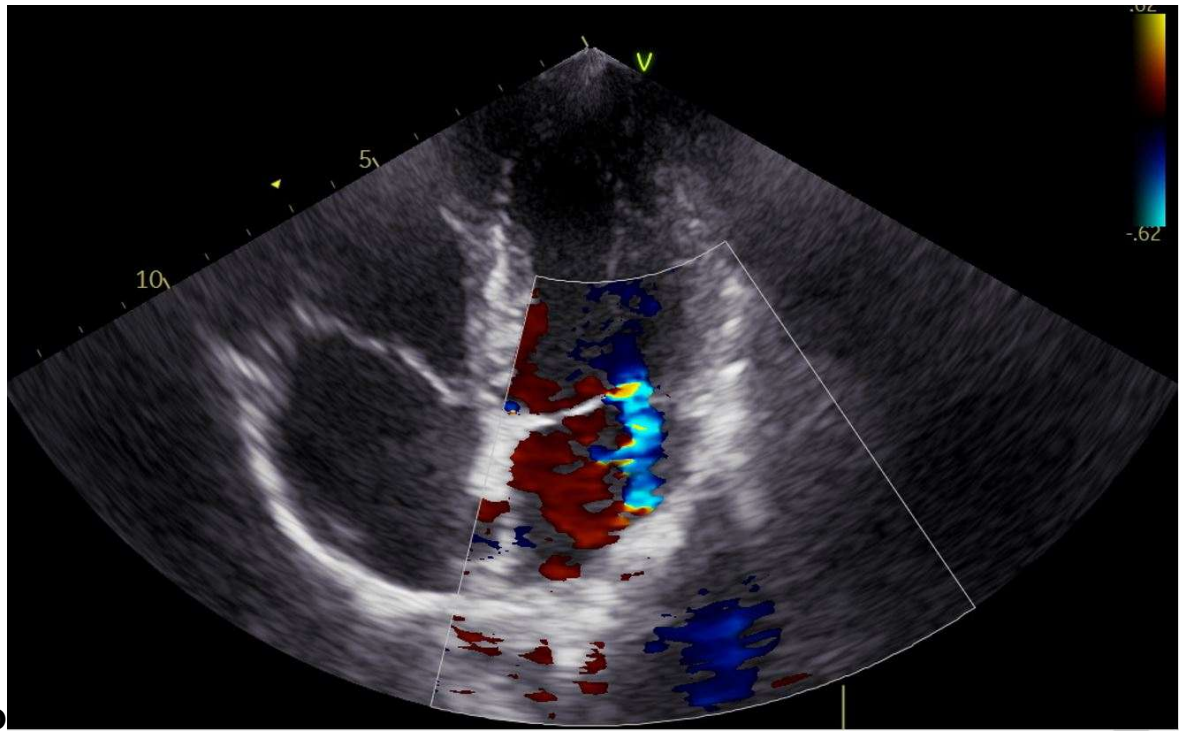

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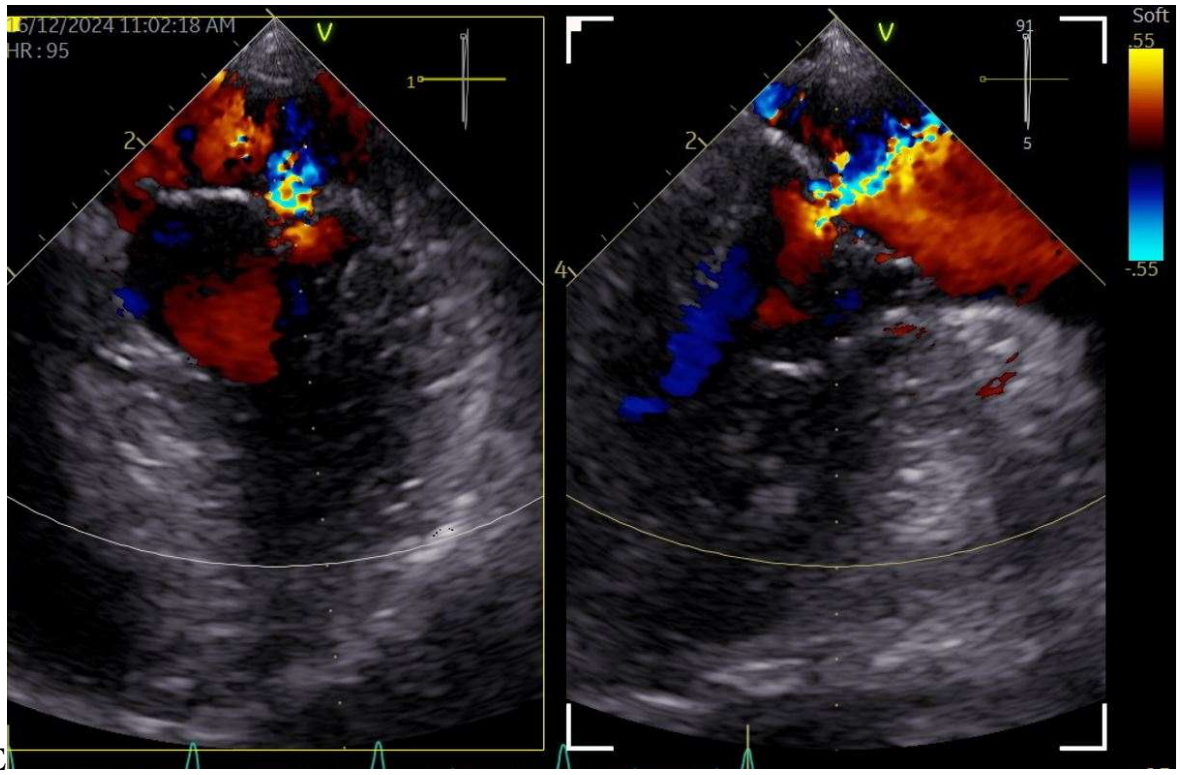


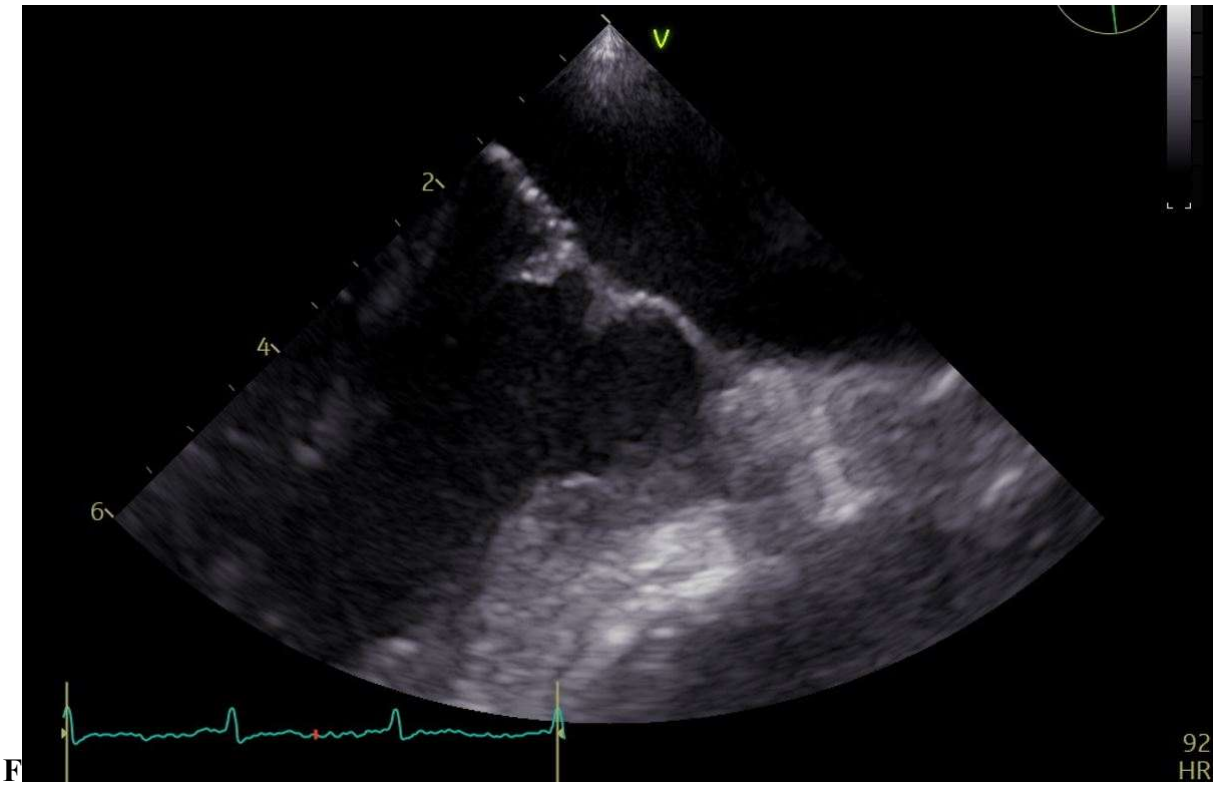
F 

Figure 1. A. Severe mitral regurgitation (MR) in the 4-chamber view on transthoracic echocardiography (TTE); vena contracta measures 9 mm. **B.** Central large jet >50 % of the left atrium area. **C.** Severe MR in the 3-chamber view on TTE; proximal isovelocity surface area measures 10 mm. **D.** Mild to moderate MR in the 4-chamber view on TTE after 5 days of treatment **E.** Mild to moderate MR in transesophageal echocardiography (TOE) after 5 days of treatment. **F.** Rheumatic changes of the mitral valve observed on TOE