J Cardiovasc Imaging. 2021 Jan;29(1):75-78
https://doi.org/10.4250/jcvi.2020.0024
pISSN 2586-7210·eISSN 2586-7296

Journal of Cardiovascular Imaging JCVI

Images in Cardiovascular Disease

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Successful Treatment of Classical Loeffler's Endocarditis

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OPEN ACCESS

Received: Feb 18, 2020
Revised: Apr 12, 2020
Accepted: Jun 4, 2020

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Conflict of Interest
The authors have no financial conflicts of interest.

A 56-year-old with a history of ulcerative colitis (UC) and rheumatoid arthritis (RA) was noted to have hypereosinophilia (2.19/μL) on a routine blood test. Our patient was asymptomatic with no signs of active RA or UC. Hypereosinophilia was initially thought secondary to sulfalazine, which our patient takes regularly for RA. However, the dose of sulfasalazine was reduced and the eosinophil count continued to rise, peaking at 19.23/uL. Blood tests were negative for auto-antibodies, parasites, vasculitis, and bone marrow biopsy were negative for myeloproliferative disorders. Computed tomography (CT) scan demonstrated a large mucocele of the appendix and abnormal attenuation in the left ventricle (LV). Due to risk of mucocele rupture and pseudomyxoma peritonei (PMP), our patient was admitted for a laparoscopic appendectomy. A routine electrocardiogram (ECG) post-operatively showed that the patient was in atrial fibrillation (AF). To evaluate the CT and ECG findings, our patient underwent a transthoracic echocardiogram (TTE). TTE revealed a thickened LV with small pericardial effusion (**Figure 1A, B**). A moderate mitral regurgitation (MR), and preserved LV systolic function were other notable findings on TTE. Cardiac magnetic resonance (CMR) with gadolinium contrast to further evaluate the echocardiographic changes showed

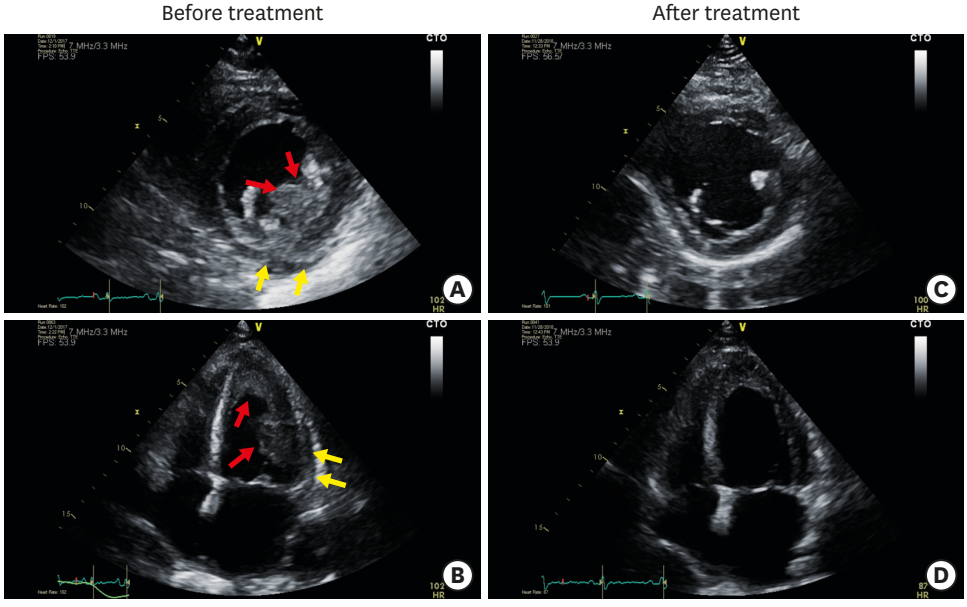

Figure 1. (A, B) Inferolateral and apical wall thickening in short-axis and four-chamber views respectively (red arrows). Mild pericardial effusion can also be noted in these views (yellow arrows). **(C, D)** Following treatment, there is significant reduction in inferolateral and apical wall thickness in the respective views. The pericardial effusion can no longer be seen.

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a hypertrophic LV with widespread subendocardial late gadolinium enhancement (LGE), implicating endomyocardial fibrosis (EMF) (**Figure 2A-C**). CMR also revealed an area of LGE in the right ventricle (RV) and a mural thrombus in the LV apex (**Figure 3A-C**). During admission, our patient developed ataxia and left-sided visual inattention for which brain magnetic resonance imaging was performed. This showed areas of hypoperfusion in the watershed distribution; this was thought to be secondary to local thrombus formation from a hyperviscous state and distant micro-thromboemboli (**Figure 4**).

Findings from all the described imaging modalities were highly suggestive of Loeffler endocarditis. Therefore, following the appendectomy, our patient was commenced on corticosteroids and anticoagulation.

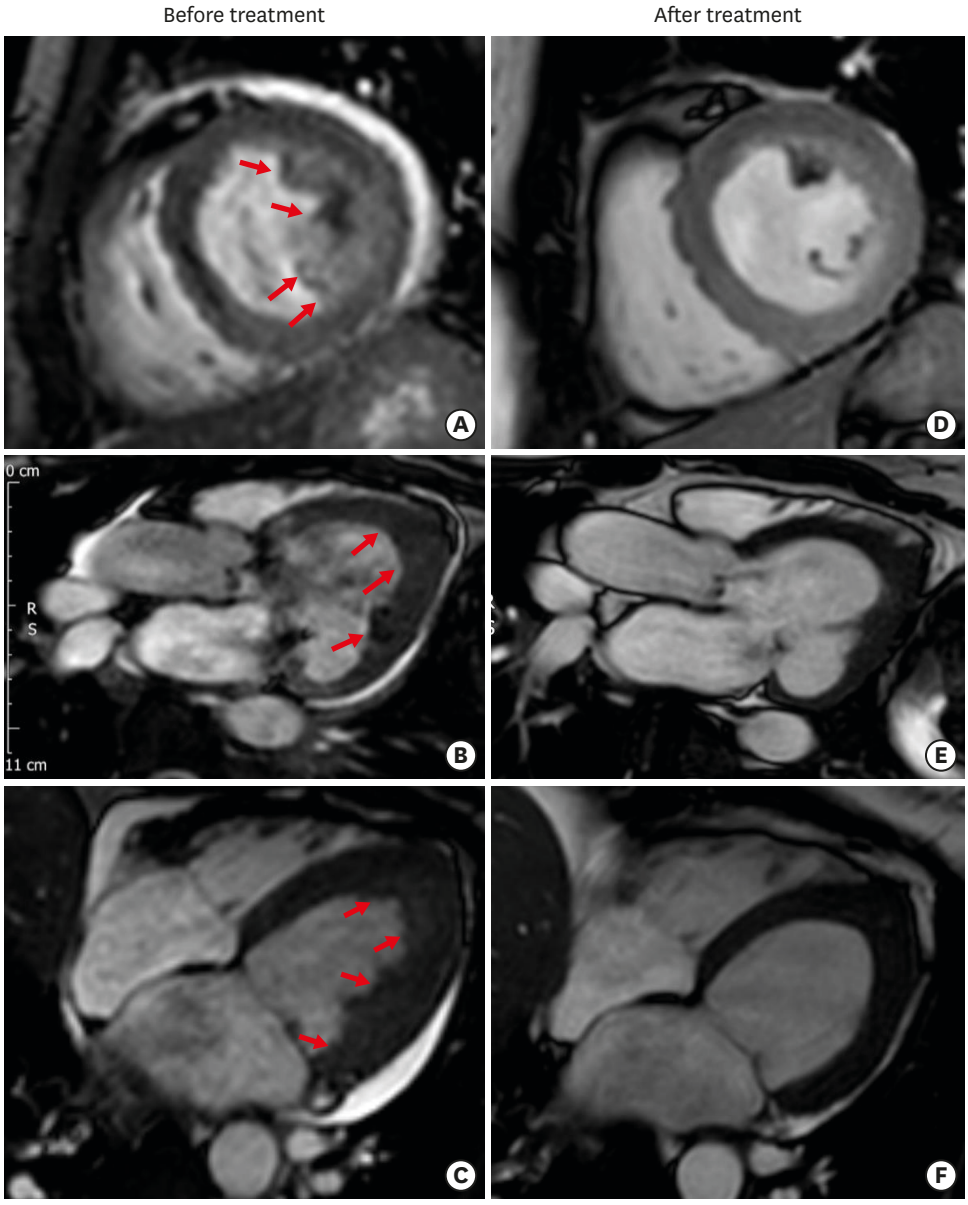

Figure 2. (A, B, C) Steady state free precession cine MRI showing increased LV wall thickness from mid to the apex, particularly at the antero-lateral and inferior walls, in the short axis, LVOT and four-chamber views (red arrows). **(D, E, F)** Significant reduction in wall thickness noted on short-axis, LVOT and four-chamber views respectively. LV: left ventricle, LVOT: LV outflow, MRI: magnetic resonance imaging.

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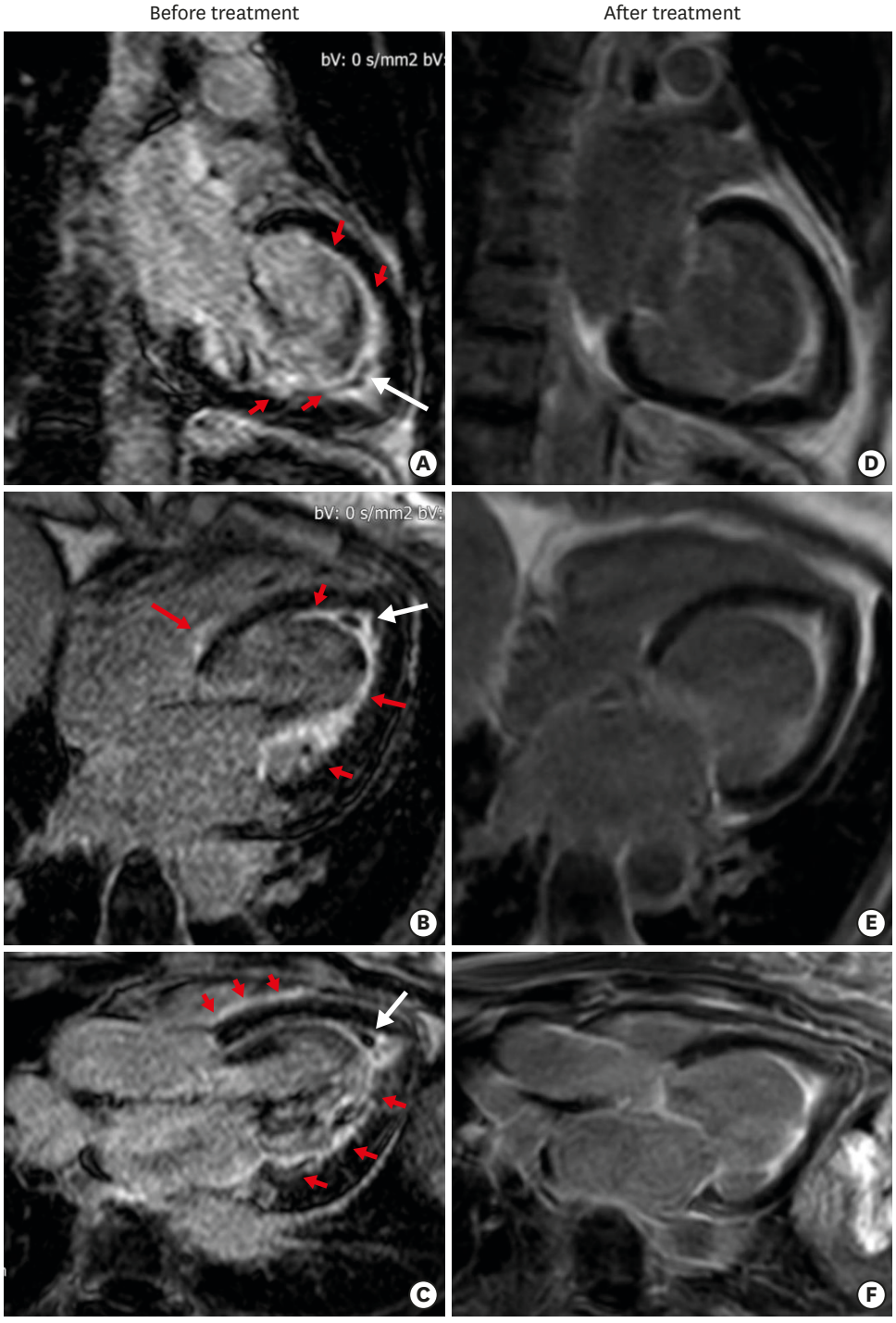

Figure 3. (A, B, C) Steady-state free precession cine CMR showing extensive subendocardial LGE of the LV on two chamber, four-chamber and LVOT views (red arrows). There is mural thrombus in the thickened LV apex (white arrow). LGE of the basal septal RV is noted in four-chamber and LVOT views. **(D, E, F)** Reduction in LGE of the LV and RV with improvement in size of apical thrombus following treatment is noted in the respective views. CMR: cardiac magnetic resonance, LGE: late gadolinium enhancement, LV: left ventricle, LVOT: LV outflow, RV: right ventricle.

Two weeks following mucocele resection surgery and steroids, the eosinophil count dropped to 0.07/μL. Five months later, TTE and CMR showed a reduction in LV wall thickness (**Figure 1C, D**

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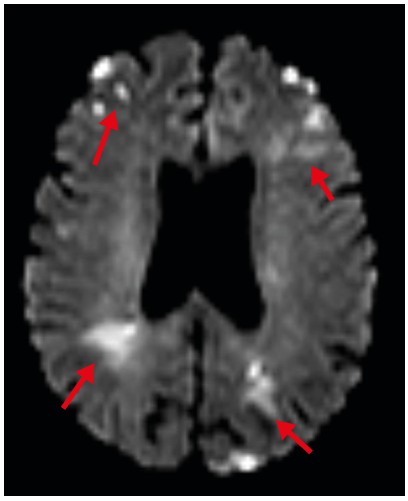

Figure 4. Diffusion weighted brain MRI series with 120 images and b-value of 1000 seconds/mm^2 showing multiple areas of restricted diffusion bilaterally (red arrows). MRI: magnetic resonance imaging.

and **Figure 2D-F** respectively). MR had reduced from moderate to mild on TTE. The extent of LGE in the LV and RV had reduced, leaving only a thin layer of EMF; the LV apical thrombus had also nearly resolved (**Figure 3D-F**).

One month later, our patient once again had a raised eosinophil count peaking at 1.2/ μL. Corticosteroids were restarted to prevent any further end organ damage. Surveillance CT scans have shown findings suggestive of PMP. Our patient's eosinophil count had normalised whilst on a weaning corticosteroid regimen and now awaits specialist input with regards to PMP. Follow-up echocardiograms have remained largely unchanged.

Cardiac involvement as a result of hypereosinophilia is commonly referred to as Loeffler endocarditis. Stages of eosinophilic infiltration of the endo-myocardium range from acute necrosis, to a thrombo-embolic phase, and finally, EMF.[1] Hypereosinophilia can be caused by hypersensitivity, vasculitis, medications, parasitic and fungal infections, and myeloproliferative conditions[1]; UC and RA have also been known to cause hypereosinophilia.[2][3] No specific cause for hypereosinophilia was identified in our patient. At this time, it is unclear whether the mucinous appendix was simply an incidental finding or a contributing factor for hypereosinophilia. Our case demonstrates classical features of Loeffler endocarditis and that treatment is possible with early medical therapy.

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