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A RARE FORM OF MYOCARDIAL INFLAMMATION – COMPLICATIONS WITH A VENTRICULAR ASSIST DEVICE AND SUCCESSFUL TRANSPLANTATION

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ABSTRACT

Giant cell myocarditis (GCM) is a rare, often fulminant inflammatory cardiomyopathy associated with rapid progression to advanced heart failure and high mortality. We report the case of a 52-year-old male with previously undiagnosed GCM who developed post-inflammatory cardiomyopathy leading to severe left ventricular dysfunction. Despite implantation of a cardiac resynchronization therapy defibrillator (CRT-D) and later a HeartMate III left ventricular assist device (LVAD) as a bridge to transplantation, the patient's condition gradually worsened. Orthotopic heart transplantation was performed 5.5 years after diagnosis, with histopathological analysis of the explanted heart confirming GCM. Post-transplant follow-up over 16 months demonstrated preserved graft function, absence of recurrence, and stable clinical status. This case highlights the importance of histopathological evaluation of explanted hearts, the diagnostic challenges of atypical GCM, and the role of LVAD and heart transplantation in ensuring long-term survival.

KEYWORDS

Giant Cell Myocarditis, Post-Inflammatory Cardiomyopathy, Heart Transplantation, Left Ventricular Assist Device, Cardiac Resynchronization Therapy, Endomyocardial Biopsy

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Introduction

Giant cell myocarditis (GCM) is a rare and rapidly progressive form of myocarditis, commonly leading to fulminant heart failure (HF), refractory ventricular arrhythmias or conduction system abnormalities. It carries a high mortality rate if left untreated[1]. Median survival is 3 months to death or transplant without appropriate therapy [2]. Echocardiographic images resemble dilated cardiomyopathy, which does not improve with guideline-based treatment. Ventricular tachycardia and heart block occur in a substantial number of patients. Recent studies suggest that the ventricular arrhythmias in GCM may be mediated by a cytokine-induced change in desmosomal protein expression. Diagnosis by endomyocardial biopsy can allow immunosuppressive therapy and timely use of mechanical circulatory support when indicated. Transplantation remains an effective therapy despite a 20–25% risk of GCM recurrence in the allograft [3], however recurrence in the native heart occurs up to 8 years after initial diagnosis.

Case presentation

A 52-year-old male with advanced heart failure due to post-inflammatory cardiomyopathy had a 5.5-year history of progressive disease. The patient experienced worsening heart failure, which led to the implantation of a cardiac resynchronization therapy defibrillator (CRT-D) for the primary prevention of sudden cardiac death (SCD).

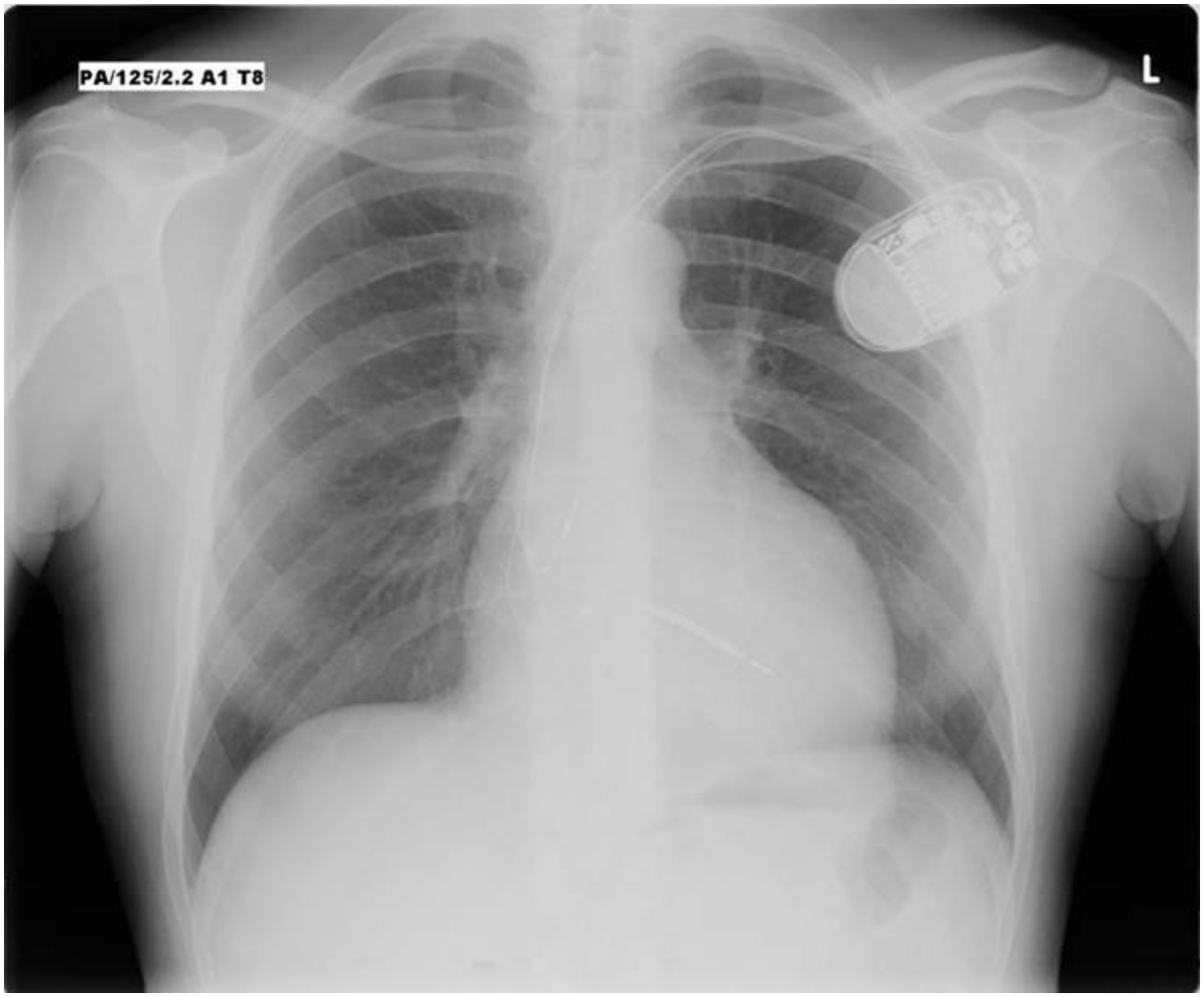

Fig. 1. *Chest X-ray in posteroanterior (PA) projection. Visible implanted CRT-D and cardiomegaly.*

At age 47, nine months after implantation of a CRT-D, he was hospitalized in our transplant unit. Then echocardiography demonstrated a left ventricular ejection fraction (LVEF) of 12% and tricuspid annular plane systolic excursion (TAPSE) of 18 mm.

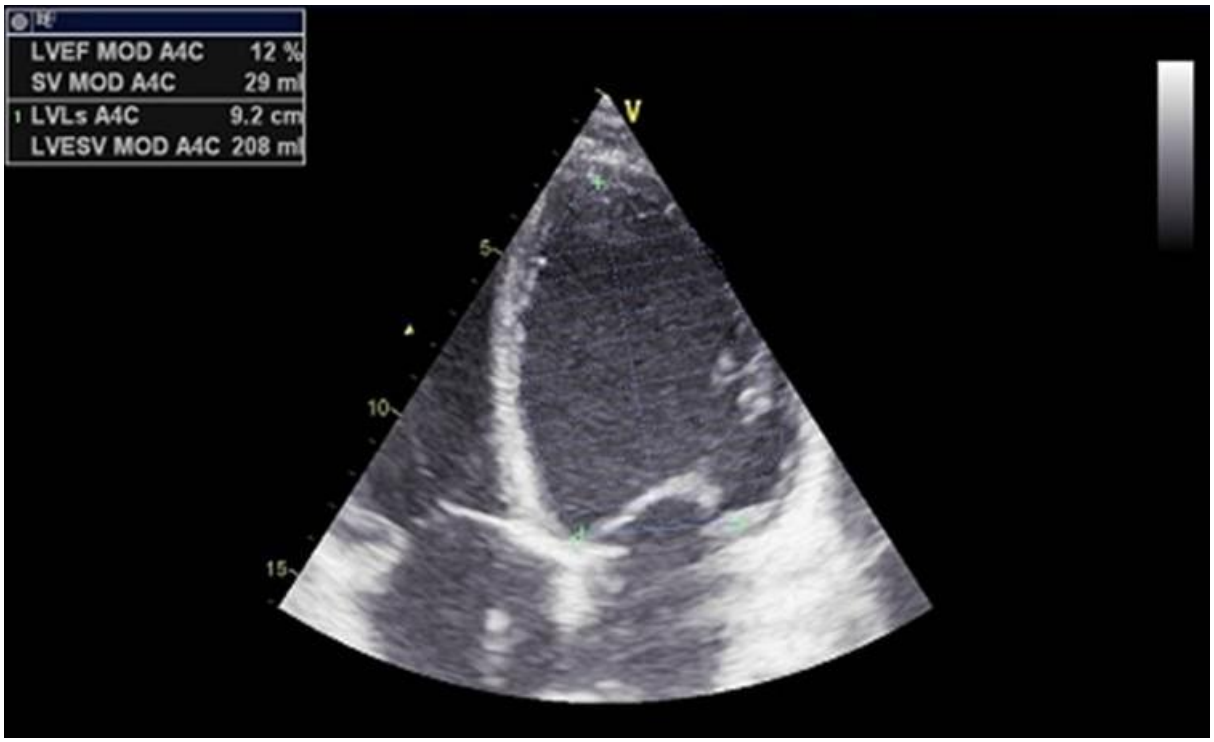

Fig. 2. *Transthoracic echocardiography showing severely impaired left ventricular contractility with a left ventricular ejection fraction (LVEF) of 12%, [AS8] [MS9] as well as a significantly enlarged left ventricle with a left ventricular end-diastolic volume (LVEDV) of 208 ml. Right ventricle is smaller, with dimensions relatively within normal limits compared to the enlarged left ventricle.*

Video 1. Transthoracic echocardiography showing severely impaired left ventricular contractility with a left ventricular ejection fraction

Three months later, due to severe heart failure, a HeartMate III left ventricular assist device (LVAD) was implanted. He remained LVAD-dependent for over four years, developing recurrent driveline infections and worsening heart failure, which became the indication for transplantation.

Orthotopic heart transplantation was performed after 5.5 years from the diagnosis of severe left ventricular systolic dysfunction (5.5 years after CRT-D implantation and 4.5 years after LVAD implantation). Bicaval anastomosis technique was used, with simultaneous LVAD and CRT-D explantation. The surgery was uncomplicated. Microbiological evaluation revealed no evidence of infection, and early endomyocardial biopsies showed no cellular rejection. Postoperatively, the patient developed persistent sinus bradycardia of the cardiac graft, which resolved with oral theophylline. Echocardiography confirmed preserved biventricular systolic function without pericardial or pleural effusion.

From the explanted heart several tissue samples were taken – from three localizations of left ventricle (anterior, lateral and posteroinferior wall of the left ventricle), from the right ventricle and interventricular septum. Histopathological examination of the left ventricle revealed an inflammatory granuloma containing multinucleated giant cells without associated fibrosis, located in the anterior wall—findings characteristic of giant cell myocarditis.

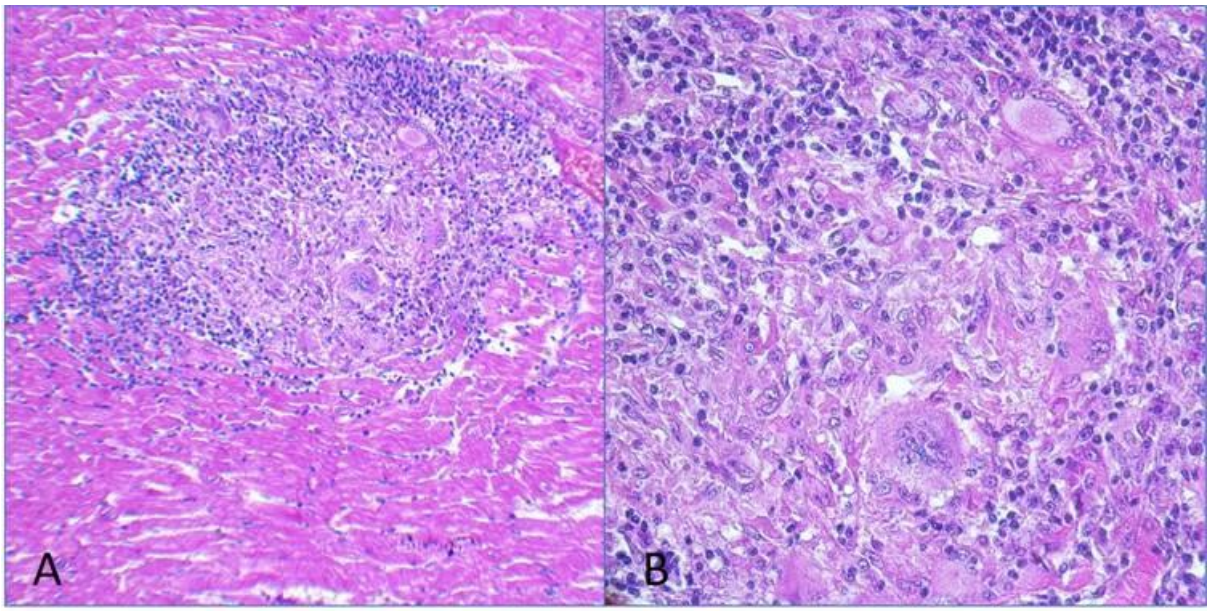

Fig. 3. *Inflammatory granuloma within the anterior wall of the left ventricle, H&E, magnification x200 (A), multinucleated giant cells within the granuloma, H&E, magnification x400 (B).*

Similar patterns of subendocardial, intramuscular, and perivascular fibrosis with variable sarcoplasmic alterations were also observed throughout the left ventricular walls and interventricular septum, the latter demonstrating an extensive collagenous scar. In addition, multifocal stromal, perivascular, and subendocardial fibrosis was present, accompanied by degenerative fatty changes in the right ventricle. Sarcoplasmic alterations included myofibrillolysis and premiocytolysis. Importantly, no cardiomyopathic muscle fibers were identified. Overall, the findings are consistent with post-inflammatory cardiomyopathy secondary to giant cell myocarditis. The patient was discharged in stable condition. A six-week biopsy showed no acute rejection (ISHLT grade 0), and graft function remained normal. Comorbidities included chronic kidney disease stage G3a (solitary kidney, eGFR 45 ml/min/1.73 m²) and mild iron-deficiency anemia.

Over the next 16 months, the patient remained clinically stable until a routine biopsy revealed acute graft rejection (ISHLT grade 3a). High-dose glucocorticoid therapy resulted in improvement to ISHLT grade 1a.

Discussion

We present the case of a patient in whom previously undiagnosed GCM led to dilated cardiomyopathy (DCM), followed by heart failure (HF) requiring CRT-D implantation, then LVAD support, and ultimately heart transplantation. Histopathological examination of the explanted heart confirmed the diagnosis of GCM.

Our patient's clinical course illustrates a chronic, slowly progressive form of giant cell myocarditis (GCM), yet it differs substantially from other reported long-term presentations, such as the case described by Fallon et al.[8] In that report, the patient remained in a remarkably stable condition for nearly a decade with minimal clinical deterioration until late progression to advanced heart failure. By contrast, our patient's 5.5-year disease trajectory, although prolonged, was characterized by persistent severe left ventricular systolic dysfunction, early dependence on mechanical circulatory support (HeartMate III LVAD), and recurrent driveline infections.

Moreover, in our case, the diagnosis of GCM was established only after orthotopic heart transplantation, with histopathology showing advanced, "burnt-out" myocarditis marked by diffuse fibrosis and limited residual inflammatory activity.

These differences highlight the heterogeneity of GCM, where some chronic forms can remain hemodynamically stable for many years, while others progress inexorably despite prolonged mechanical support, ultimately requiring transplantation.

When comparing our patient with those previously described in the literature - Ren et al. describe in their study three patients who survived for a long time — one patient with complete heart block who lived as long as 10 years, another who underwent heart transplantation 5 years after endomyocarditis, and a third who

received a heart transplant 2 years after developing heart failure[4]. Ekström et al. estimate that, in GCM, the probability of transplant-free survival is 42% at 5 years from symptom onset[5].

Endomyocardial biopsy is considered the gold standard for the diagnosis of myocarditis[6]. However, a biopsy performed prior to heart transplantation might not be diagnostically helpful, as the pathological changes in GCM are often located in the free wall of the left ventricle. Standard myocardial biopsy is usually performed by obtaining tissue samples from the right ventricle, owing to its more accessible anatomical location. Therefore, studies indicate that in patients with suspected myocarditis, biventricular endomyocardial biopsy provides better diagnostic accuracy compared to selective biopsy of either ventricle. Bleble et al. show that myocarditis was confirmed in 89 out of 127 examined patients (70.1%) by biventricular biopsy. They indicate that in 75.3% of cases, the diagnostic criteria were met in both ventricles, in 18% in the left ventricle alone, and in 6.7% in the right ventricle alone[7]. Therefore, in such cases biventricular biopsy should be mandatory.

Due to diagnostic challenges in vivo, the disease in our patient was diagnosed only after histopathological examination of the explanted organ. This case demonstrates the value of performing routine histopathological analysis of explanted hearts in patients with end-stage heart failure requiring transplantation. Such examinations can offer significant benefits for treatment, including increased vigilance for disease recurrence, without placing additional burden on the patient. During follow-up after transplantation, no recurrence of GCM was observed, although this complication is considered relatively common. The patient remained stable, which suggests that heart transplantation may provide lasting benefit in carefully selected cases.

The heart transplantation procedure using the bicaval anastomosis technique was completed without complications. The absence of signs of graft rejection for 1.5 years in subsequent endomyocardial biopsies indicates well-managed immunosuppressive therapy and stability of the transplanted organ.

LVAD can effectively support GCM treatment as a bridge to transplantation, though it increases septic shock risk from catheter-related infections. This case—over four years from LVAD implantation to heart transplant without major complications—is rarely reported.

Conclusions

Giant cell myocarditis (GCM) is a rare, rapidly progressive inflammatory cardiac disease associated with high mortality if left untreated. In most cases, the diagnosis is made by endomyocardial biopsy (≈52%), while fewer are identified at autopsy (17%) or through histopathological analysis of explanted hearts (22%). This underlines the importance of actively searching for GCM in patients with progressive heart failure of unexplained etiology. Orthotopic heart transplantation (OHT) remains a viable and effective therapeutic option in advanced stages of GCM[9,10].

In the presented case, a 52-year-old male developed post-inflammatory cardiomyopathy due to previously unrecognized GCM, which persisted for 5.5 years until heart transplantation. The patient first required implantation of a left ventricular assist device (LVAD) as a bridge to transplantation, followed by successful OHT. In our case, GCM was diagnosed only on histopathological examination of the explanted heart.

Throughout the entire disease course—including advanced heart failure, LVAD therapy, heart transplantation, and follow-up—the patient remained clinically stable, with no decline in exercise tolerance reported during outpatient visits. Repeated endomyocardial biopsies confirmed the absence of acute cellular rejection, and echocardiography demonstrated preserved graft function 1.5 years post-transplant. Importantly, no recurrence of GCM was observed.

This case raises awareness of atypical and latent forms of GCM, highlights the value of histopathological examination of the explanted heart, and supports consideration of GCM in patients with long-standing cardiomyopathy of unclear etiology. Moreover, it illustrates that early initiation of LVAD therapy as a bridge to transplantation, followed by careful post-transplant management and immunosuppressive therapy, can ensure a favourable and stable clinical course. Further research should aim to improve early diagnosis, optimize immunosuppressive regimens, and develop strategies for managing exacerbations, recurrences, and arrhythmias in patients with GCM.

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