

Homoeopathic Management of Dilated Cardiomyopathy Progressing to Congestive Heart Failure in A 64-Year-Old Diabetic Woman

Dr. Kirthana L.¹, Dr. Prasobh M. P.²

¹Post Graduate Scholar, Department of Materia Medica, White Memorial Homoeopathic Medical College, Kanyakumari 629177.
Corresponding Author Email: [kirthuteddy17\[at\]gmail.com](mailto:kirthuteddy17[at]gmail.com)

²H. O. D and P. G Guide, Department of Materia Medica, White Memorial Homoeopathic Medical College, Kanyakumari 629177

Abstract: Dilated cardiomyopathy (DCM) is a condition characterized by the enlargement and weakening of the heart's ventricles, leading to impaired contraction and reduced blood pumping efficiency. This typically begins in the left ventricle and can progress to affect the right ventricle and atria. As the heart chambers dilate, the heart muscle doesn't contract normally and can't pump blood effectively, which can lead to heart failure [1]. **Case Summary:** This case report outlines the clinical course of a 64-year-old female patient with a history of type 2 diabetes mellitus and dilated cardiomyopathy (DCM), who developed congestive cardiac failure (CCF) managed with Homoeopathic treatment. The prescription was based on repertorisation using Phatak repertory and NAJA TRIPUDIANS were prescribed. Initial echocardiogram showed severe left ventricular (LV) dysfunction (Ejection Fraction 30%) with chamber dilation. An ECHO report taken after approximately 1 year showed significant improvement in LV systolic function (Ejection Fraction 57.7%) and reduction in LV dimensions. The report discusses the patient's clinical presentation, diagnostic evaluation, progression of disease, and clinical implications for managing such complex comorbidities with Homoeopathy.

Keywords: Dilated cardiomyopathy, congestive cardiac failure, Homeopathy, Echocardiography, Naja Tripudians

1. Introduction

Dilated forms of cardiomyopathy are characterized by ventricular chamber enlargement and systolic dysfunction with normal LV wall thickness; usually diagnosis is made with 2-dimensional echocardiography. DCM leads to progressive heart failure and a decline in LV contractile function, ventricular and supraventricular arrhythmias, conduction system abnormalities, thromboembolism, and sudden or heart failure-related death. About 20% to 35% of DCM cases have been reported as familial, although with incomplete and age-dependent penetrance, and linked to a diverse group of >20 loci and genes [2].

Beyond genetic predispositions, DCM encompasses a wide range of etiologies. A significant proportion of cases are classified as idiopathic, meaning the exact cause remains unidentified despite thorough investigation. Other known causes include viral myocarditis (often enteroviruses or adenovirus), toxic exposures (such as alcohol abuse or certain chemotherapeutic agents like doxorubicin), autoimmune diseases, nutritional deficiencies (like thiamine), and peripartum cardiomyopathy occurring during or after pregnancy. While ischemic heart disease can cause similar ventricular dilation and dysfunction, DCM is typically defined by these features arising from non-ischemic causes, though ischemia must often be ruled out. Regardless of the underlying cause, the common pathway involves damage to the myocardium, leading to impaired contractility, ventricular remodeling (dilation), and eventually, the clinical syndrome of heart failure [3, 4]. Dilated cardiomyopathy (DCM) is a significant contributor to emergency room visits in our country. It is a relatively common cause of heart failure, with a prevalence of approximately 0.04% and an annual incidence of 5 to 8 cases per 100, 000 people, showing a steady increase

over time [5, 6]. One study reported a prevalence of DCM in adults as 1 in 2, 500 [7]. This report highlights the interplay of diabetes and cardiomyopathy in the progression to end-stage heart failure.

Patient Information

- **Age:** 64 years
- **Gender:** Female
- **Chief Complaint:** Progressive shortness of breath and fatigue over the past 3 months. Loss of appetite with nausea for 10 days.

History of Present Illness

The patient has loss of appetite with nausea along with worsening dyspnea on exertion and difficulty carrying out her daily activities. She also has dyspnea on lying down at night requiring her to prop up with multiple pillows at night. Over the last month, she has noticed bilateral lower extremity swelling and ascites. No chest pain or palpitations were reported. The patient was hospitalized and was under supervision of a cardiologist.

Past Medical History

Type 2 Diabetes Mellitus (diagnosed 15 years ago) with medication of Metformin 500 mg twice daily along with mother tincture of *Syzygium jambolanum*, suboptimal glycemic control with HbA1c 8.9 %.

Physical Generals

- Appetite reduced in the past 1 week.
- Thirst reduced with reduced frequency of urination.
- Constipation was present.

Family History

- H/O diabetes in her father
- No known family history of sudden cardiac death.

Volume 14 Issue 5, May 2025

Fully Refereed | Open Access | Double Blind Peer Reviewed Journal

www.ijsr.net

Physical Examination (Initial Presentation)

- a) **General Appearance:** Alert, fatigued, and dyspnoeic at rest.
- b) **Vital Signs:** BP 140/90 mmHg, HR 95 bpm (regular), RR 20, SpO₂ 94% on room air.
- c) **Cardiovascular:**
 - Jugular venous distension (JVD) noted at 8 cm above the sternal angle.
 - Apical impulse displaced laterally and inferiorly.
 - S3 gallop heard over the apex.
 - No murmurs or pericardial rubs.
- d) **Respiratory:** Bibasilar crackles present.
- e) **Abdomen:** Mild ascites. Puddle sign positive.
- f) **Extremities:** Bilateral pitting edema up to the mid-calf.

- **Valves:** Mild Mitral Regurgitation (MR), Severe Tricuspid Regurgitation (TR). Thickened mitral valve.
- **Other:** Moderate Pulmonary Hypertension. Mild to moderate pericardial effusion. No clot.
- **Impression:** Dilated Cardiomyopathy with Severe LV dysfunction.

Diagnosis

Dilated cardiomyopathy with decompensated congestive cardiac failure in the context of poorly controlled diabetes mellitus.

Repertorization and Medicine Selection

Clinical repertory was chosen based on the situation and echo report. Repertorization was done using PHATAK repertory and referred to materia medica [8]. Naja is indicated when "Inability to speak, with choking, nervous, chronic palpitation; chronic hypertrophy and valvular disease of heart; shortness of breath; palpitation of heart, < walking and lying on side with anxiety and fear of death; attacks occur frequently during night [9].

Diagnostic Workup**Initial Echocardiogram (15.08.2023):**

- **Findings:** Dilated Left Atrium (LA: 42mm) and Left Ventricle (LV). Left Ventricular Internal Dimension diastolic (LVIDD): 59mm, systolic (LVIDS): 50mm.
- **Function:** Severely reduced Left Ventricular Ejection Fraction (LVEF): 30%. Global hypokinesia of LV.

Remedy Name	Spong	Cact	Naja	Kali-c	Puls	Lach	Tarent	Laur
Totally	6	5	5	5	5	4	4	3
Symptoms Covered	4	4	4	3	2	2	2	3
Kingdom	1	1	1	1	1	1	1	1
[Phatak] [Phatak A-Z]Heart:Hypertrophy:	1	1	1	1	1	1	1	1
[Phatak] [Phatak A-Z]Heart:Valves:	1	1	1	2	2	1	2	1
[Phatak] [Phatak A-Z]Heart:Dilatation:	1	2	1	1	1	1	1	1
[Phatak] [Phatak A-Z]Respiration:Difficult, suffocating, choking:	3	1	2	2	3	3	2	1
[Phatak] [Phatak A-Z]Asthma:Cardiac (See Respiration difficult):	1	1	1	1	1	1	1	1

Prescription

NAJA TRIPUDIANS 200 twice daily was prescribed initially.

- JVP and pedal edema reduced.
- *Prescription:* NAJA 200 WAS CONTINUED.

Follow-Up**a) Follow-up 1 (16/08/2023):**

- Patient reported improvement in shortness of breath and fatigue.
- Swelling in legs reduced.
- Anxiety decreased; the patient was discharged from the hospital.
- *Prescription:* NAJA 200 WAS CONTINUED.

b) Follow-up 2 (2/09/2023):

- Patient reported further improvement in symptoms.

c) Follow-up 3 (26/09/2023):

- Patient reported further improvement in symptoms.
- Ascites reduced.
- *Prescription:* PLACEBO WAS GIVEN.

d) Follow-up 4 (2/11/2023):

- Shortness of breath were present only during exertion.

The follow-ups were done every 2 weeks and there were 3 aggravations of complaint and NAJA 200 was repeated during the 1-year period. An ECHO report was taken after that. All through the treatment, the patient's diabetic control was taken into account.

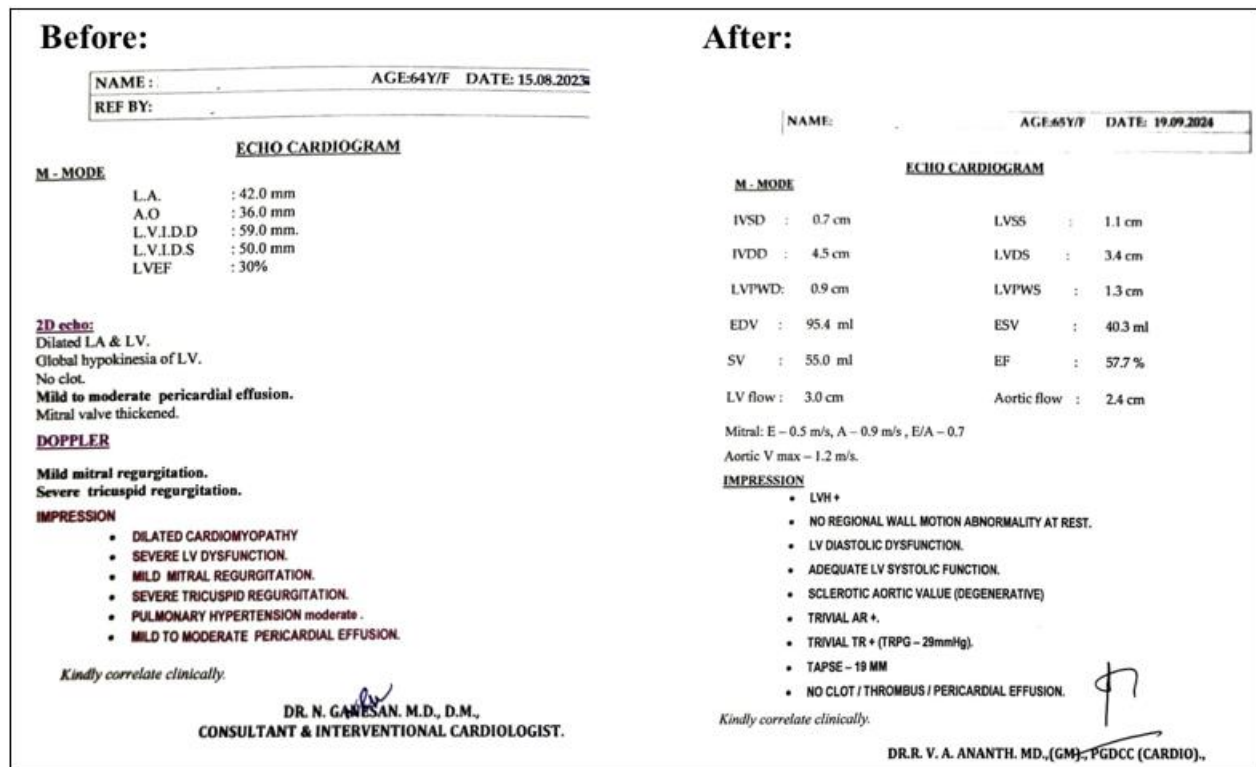


Figure: Echo cardiogram report of patient before and after treatment.

2. Result and Discussion

During the one-year period of homeopathic treatment alongside undisclosed conventional management, the patient demonstrated significant improvement in her clinical symptoms and overall well-being. Symptoms such as fatigue, breathlessness, and exercise intolerance showed marked resolution. The patient reported better quality of life, with improved physical activity levels.

Follow-up Echocardiogram (19.09.2024) revealed significant objective improvements:

- LV Function:** Left Ventricular Ejection Fraction (LVEF) improved markedly from 30% to 57.7%, indicating adequate LV systolic function.
- LV Dimensions:** Reduction in LV size noted, with Left Ventricular Internal Dimension diastolic (reported as IVDD) decreasing from 59mm to 4.5cm (45mm) and systolic (reported as LVDS) decreasing from 50mm to 3.4cm (34mm).
- Valvular Regurgitation:** Tricuspid Regurgitation improved from Severe to Trivial (TRPG 29mmHg). Aortic Regurgitation noted as Trivial. Mitral Regurgitation was not mentioned, possibly resolved or trivial.
- Pericardial Effusion:** Resolved (No effusion noted).
- Other Findings:** Evidence of Left Ventricular Hypertrophy (LVH) and LV Diastolic Dysfunction (E/A ratio 0.7) noted. Sclerotic aortic valve (degenerative). Normal Tricuspid Annular Plane Systolic Excursion (TAPSE 19mm). No regional wall motion abnormalities, clot, or thrombus.

These objective findings correlate with the patient's reported symptomatic improvement. The treatment is continued for further improvement.

3. Conclusion

This case demonstrates significant clinical and echocardiographic improvement in a patient with dilated cardiomyopathy, congestive heart failure, and type 2 diabetes over a one-year period during which homeopathic treatment (Naja Tripudians) was administered. Notably, LVEF normalized, LV dimensions reduced, TR severity decreased, and pericardial effusion resolved.

While the presented outcome is positive, attributing it solely to the homeopathic intervention is challenging without detailed information on concurrent conventional cardiac medications and changes in glycemic control. The case highlights the potential for improvement in complex cardiac patients and suggests that individualized Homeopathic remedies, as part of a broader management strategy, may contribute to better health outcomes. However, the limitations regarding confounding factors mean that current evidence from this single case is insufficient to establish confirmatory evidence of homeopathy's efficacy. Rigorous trials controlling for conventional treatments and other variables are necessary to validate the effectiveness of Homeopathic interventions in managing dilated cardiomyopathy with diabetes mellitus.

Declaration Of Patient Consent

The authors certify that they have obtained patient consent, and that the patient has given her consent for her clinical information and her reports to be reported in the journal. In addition, the patient was made to understand that her name and initials would not be published, and efforts would be made to conceal her identity.

References

- [1] Dilated Cardiomyopathy (DCM) | American Heart Association, (n. d.). <https://www.heart.org/en/health-topics/cardiomyopathy/what-is-cardiomyopathy-in-adults/dilated-cardiomyopathy-dcm> (accessed May 6, 2025).
- [2] B. J. Maron, J. A. Towbin, G. Thiene, C. Antzelevitch, D. Corrado, D. Arnett, A. J. Moss, C. E. Seidman, J. B. Young, American Heart Association, Council on Clinical Cardiology, Heart Failure and Transplantation Committee, Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups, Council on Epidemiology and Prevention, Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention, *Circulation* 113 (2006) 1807–1816. <https://doi.org/10.1161/CIRCULATIONAHA.106.174287>.
- [3] D. I. Keller, L. Carrier, K. Schwartz, Genetics of familial cardiomyopathies and arrhythmias, *Swiss Med Wkly* 132 (2002) 401–407. <https://doi.org/10.4414/smw.2002.10037>.
- [4] Heart Disease: A textbook of Cardiovascular Medicine, 5/E, edited by Eugene Braunwald, W. B. Saunders, Philadelphia (1997) 2143 pages, illustrated, \$125.00 ISBN: 9-7216-5666-8-PMC, (n. d.). <https://pmc.ncbi.nlm.nih.gov/articles/PMC6655936/> (accessed May 6, 2025).
- [5] Keller DI, Carrier L, Schwartz K. Genetics of familial cardiomyopathies and arrhythmias. *Swiss Med Wkly*.2002; 132 (29–30): 401–7.
- [6] Manolio TA, Baughman KL, Rodeheffer R. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute Workshop). *Am J Cardiol*.1992; 69: 1458–66.
- [7] Taylor MR, Carniel E, Mestroni L. Cardiomyopathy, familial dilated. *Orphanet J Rare Dis*.2006; 1: 27.
- [8] Phatak Repertory. Zomeo Pro Lan. Mumbai: Hompath; [cited 2025 May 6].
- [9] C. Hering, The guiding symptoms of our materia medica, Philadelphia: American Homoeopathic Publishing Society; J. M. Stoddart & Co., 1879. <http://archive.org/details/guidingsymptoms00heri> (accessed May 6, 2025).