Case Report

Heart Failure with Recovered Ejection Fraction: Report of a Case in Jos, Nigeria

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Abstract

Some patients in heart failure (HF) are able to withstand treatment, recover ejection fraction (EF) enough to require little or no further treatment. They belong to the distinct entity now called HF with recovered EF where patients start as HF with reduced EF and with treatment end up as HF with mid-range EF or even HF with preserved EF. This case report is on one such patient who presented in HF with features of dilated cardiomyopathy. With treatment, he promptly came out of HF, and myocardium remodeled toward recovery of function, which also reflected on electrocardiographic voltages. He remained out of failure despite deescalation of anti-failure regimen. Characterizing this group well will permit a paradigm shift in the management of HF; with the understanding that the myocardium can recover function or go into remission depending on underlying pathology.

Keywords: Heart failure, Nigeria, recovery, remodeling, systolic function

Introduction

Many years ago in 1975, Ikeme *et al*.[1] writing on the clinical features of idiopathic cardiomegaly in the tropics highlighted a group of such patients who as they recovered with treatment showed elevated blood pressure. Since ignoring the blood pressure, though they had been admitted as heart failure (HF) secondary to dilated cardiomyopathy (DCM) led to worse myocardial function and relapse of more severe HF; clinicians sought to tell this category of patients early. At the time of these studies, there was no way of telling which HF patient assumed to be consequent on DCM could manifest elevated blood pressure with treatment and relapse into worse HF. An attempt was made later by Falase and Kolawole in a clinical and radiological study.[2] In 1991, Okeahialam[3] posited that using seven echocardiographic indices, namely end-diastolic diameter (EDD), shortening fraction, ejection fraction (EF), diastolic septal wall thickness, diastolic left ventricular posterior wall thickness (LVPWThd), amplitude of septal motion, velocity of septal motion, and diastolic LVPWThd/EDD ratio, it was possible to tell which HF patients secondary to DCM had significant background hypertension and would be expected to manifest elevated blood pressure with HF therapy. To test the validity of this method, a small group of HF secondary to DCM patients suspected significantly hypertensive by this method was followed up. The sensitivity and specificity turned out to be 73% and 36%, respectively.[4] In 2001, Punnoose *et al*.[5] reported the existence of a distinct clinical entity – HF with recovered EF (HFRecEF), recommending that the group should be better characterized and targeted for more studies. We hereby report this case which we opportunistically encountered to contribute our local data to this now better defined entity – HFRecEF. Reports of such cases in sub-Saharan Africa are scarce.

Case Report

A 46-year-old man was referred to second author in 2015 as a case of hypertensive HF from a peripheral health facility for expert management. He presented with shortness of breath and palpitations of 3 months duration. He gave a history of hypertension which he did not treat. There was a history

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Submitted: 29-Nov-2016 **Revised:** 05-Sep-2019
Accepted: 17-Oct-2019 **Published:** 02-Mar-2020

How to cite this article: Okeahialam BN, Ikeme AC. Heart failure with recovered ejection fraction: Report of a case in Jos, Nigeria. Niger Med J 2020;61:48-50.

Access this article online

Quick Response Code:

Website: www.nigeriamedj.com

DOI: 10.4103/nmj.NMJ_294_16

suggestive of airway hyperreactivity-cough, dyspnea, and wheezing whenever he inhaled smoke, oil fumes, or dust in the past 4 years. He admitted to chronic alcohol abuse.

On entry, he looked fairly well. Blood pressure was normal (110/70 mmHg). Heart sounds were normal but for few ectopics and a Grade 2/6 apical pansystolic murmur. He was advised to moderate alcohol intake and put on anti-HF regimen consisting of lisinopril 5 mg daily, furosemide 80 mg daily, spironolactone 25 mg daily, digitalis 0.125 mg daily, and acetylsalicylic acid 75 mg daily. Liver function test was normal as well as packed cell volume, urea/creatinine, uric acid, fasting blood sugar, and cholesterol. Electrocardiogram (ECG) revealed left ventricular hypertrophy and occasional ventricular ectopic. Echocardiography in May 2015 revealed left ventricular eccentric hypertrophy, global hypokinesia, and normal valves. Systolic function was impaired [Table 1]. He responded promptly, following up without fail and maintained on treatment. One month later, the apical pansystolic murmur disappeared. Early in 2016, digitalis was replaced with carvedilol 1.56 mg daily which was later increased to 3.125 mg daily. In July of that year when he came for one of his reviews, he reported tolerating more physical activity and rarely got dyspneic. A repeat ECG showed an increase in QRS voltage [Figure 1], prompting the suspicion of hypertrophic cardiomyopathy and request for repeat echocardiography. In August 2016, echocardiography was repeated by the same operator, in the person of the first author, and it now showed left ventricular concentric hypertrophy, reduction in chamber sizes, and return of systolic function toward normal [Table 1]. Side by side it was obvious, he had remodeled; left ventricular size returned to normal, its walls had thickened, and systolic function had recovered.

Discussion

The earlier observations by researchers in our environment[1,2] highlighted HF patients who recovered myocardial function and could then sustain elevated blood pressures, leading to the speculation that though branded as DCM they actually had "burnt-out" hypertensive heart disease. This it would appear is now the group referred to as HFRecEF.[5] The patient here reported though with a history of hypertension which he treated with levity was normotensive when he came in with HF. Had he not known that he was previously diagnosed with hypertension, he would have been branded a case of HF secondary to DCM. When hypertension overlaps a myocardium that is primarily diseased, the result is early failure with large hypofunctioning heart.[6] Following the HF treatment (renin-angiotensin-aldosterone system blockade, digitalis, diuretics, and anti-platelets), he promptly came out of failure and remained so all through follow-up. Comparing the two echocardiographic results [Table 1], it becomes obvious that remodeling occurred – left atrial and ventricular sizes reduced, left ventricular walls thickened, and systolic function recovered. The disappearance of the functional mitral incompetence murmur depicts normalization of the mitral valve ring size. This is also supported by the increase in the ECG-QRS voltage amplitude [Figure 1]. Clinically, he tolerated more exercise and withdrawal of digitalis did not worsen his clinical status.

His improvement probably was a consequence of reverse remodeling with hypertrophy of the left ventricular walls. It may also result from natural history recovery or response to therapy.[7] It occurs in a wide spectrum of clinical cases where background myocardial disease runs a self-limiting cause or a factor toxic to the myocardium is removed.[8] The patient was abusing alcohol but discontinued when counseled to moderate intake. The renin angiotensin aldosterone system blocker lisinopril also kept his blood pressure in check.

This phenomenon presents a challenge to the clinician that of telling when the new status is consequent upon complete myocardial recovery or merely going into remission.[9] The likelihood of relapse if antifailure drugs are discontinued[10] should compel clinicians to follow-up and maintain them on treatment although tailored to clinical status.

Table 1: Echocardiographic data of patient

Index	May 2015	August 2016	Remarks
Ao (mm)	29	31	
LA (mm)	41	32	Decrease
mVexc (mm)	18	20	Increase
EF Slope (mm/sec)	110	110	
EPSS (mm)	13	12	Decrease
SWThd (mm)	10	12	Increase
SWThs (mm)	10	13	Increase
EDD (mm)	62	48	Decrease
LVPWThd (mm)	11	12	Increase
LVPWThs (mm)	15	15	
EF (%)	36	46	Increase
SF (%)	18	23	Increase

Ao – Aortic root, LA – Left atrium, mVexc – Mitral valve excursion, EF Slope – E/F points slope, EPSS – E point septal separation, SWThd – Diastolic septal wall thickness, SWThs – Systolic septal wall thickness, EDD – End diastolic diameter, LVPWThd – Left ventricular posterior wall thickness in diastole, LVPWThs – Left ventricular posterior wall thickness in systole, EF – Ejection fraction, SF – Shortening fraction

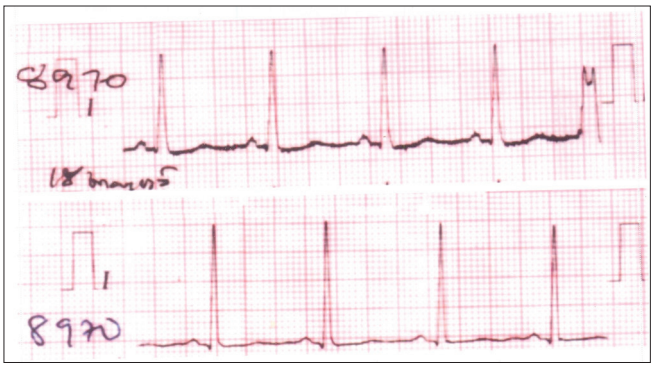

Figure 1: Two electrocardiogram tracings of the patient. The top electrocardiogram is the tracing when the patient first presented in May 2015; showing a QRS voltage of 16 mm. The bottom electrocardiogram is the same patient's tracing in August 2016 when repeat Echocardiography showed thicker walls and reduced cavity size of the left ventricle. The QRS voltage had increased to 21 mm

Conclusion

It is possible depending on the etiology and background myocardial condition for some HF cases to recover EF with treatment. The burden on the clinician now is that of determining if, how and when treatment could be discontinued. At the moment, the consensus seems to be on continuing follow-up and tailoring management to the clinical status of the patient.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgment

The authors would like to thank Mr. Patrick Agbonkhese of the Medical Instruction and Technology Unit of Faculty of Medical Sciences, University of Jos, Nigeria, for preparing the ECG image used as Figure 1.

Financial support and sponsorship

Nil.

Conflict of interest

There are no conflicts of interest.

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