Case Report

Post-measles Dilated Cardiomyopathy in an Eleven Months Old Infant: A case Report

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ABSTRACT

Cardiomyopathy is an acquired heart muscle condition that is considered to be rare among children but carries substantial risk of morbidity and mortality. Dilated cardiomyopathy has been identified as the commonest form of cardiomyopathy, accounting for about 50% of all cases, with acute myocarditis causing 10-25% of cases of dilated cardiomyopathy. Measles virus is a reported rare cause of acute myocarditis. We report a case of an eleven-month-old infant who presented with fever, cough and difficulty in breathing. Her symptoms developed following an episode of measles infection she had at about 7 months of age during an outbreak of measles infection in her community. Examination revealed displaced apex, 3rd heart sound with gallop rhythm and tender hepatomegaly. Chest X-ray showed cardiomegaly with left ventricular apex. Echocardiography confirmed dilated left atrial and left ventricular chambers, mitral regurgitation and severe left ventricular systolic dysfunction. She was placed on frusemide, captopril and digoxin with significant improvement, and was discharged home. She was seen once in the clinic, and was subsequently lost to follow-up, until 7 weeks later when she returned and succumbed to death.

Measles virus is a rare cause of myocarditis. A high index of suspicion is needed so as to minimize delay in diagnosis and increase chances of better treatment outcome.

Keywords: Dilated cardiomyopathy, Measles, Myocarditis, Systolic dysfunction

INTRODUCTION

Cardiomyopathy is an acquired heart disease of heterogeneous nature that is considered rare in children but with substantial risks of morbidity and mortality[1]. The incidence of cardiomyopathy in developed countries has been put at 1.13-1.24 per 100,000 children[1].A nationally reflective incidence is not available for Nigeria, however, an incidence of 9 per 100,000 children has been documented from a hospital study in South-western region of Nigeria[2].

Dilated cardiomyopathy (DCM) remains the most common form of cardiomyopathy recorded among children from different parts of the world and mostly among the under five age group[3, 4, 5]. It has varied aetiologies, ranging from familial (20-50%), acquired to idiopathic causes[3]. Myocarditis rank top among the acquired causes followed by ischaemic, metabolic, endocrine, haematologic, collagen vascular disorders and drugs. Measles virus is third from bottom among list of viruses that have been

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implicated as aetiologic agents of acute myocarditis[3]. There are few reported cases of measles virus as causes of acute myocarditis in literature[6-9].

CASE REPORT

ZA is an eleven months old female infant who was brought to the Emergency Paediatric Unit(EPU) of our facility with complaints of cough, fast breathing, and fever of 8/7. Cough was insidious in onset, present most part of the day and non-paroxysmal. It had progressively worsened and disturbed the child's sleep. Fast breathing was noticed following the onset of cough and had also worsened over time with associated frequent pauses while suckling. There was no associated darkening of the lips. Fever was noticed a day following onset of cough and described as low grade and was present throughout the day. It was relieved by administration of paracetamol. This was the fourth episode of similar symptoms and third hospital admission in the preceding four months. The first hospitalization was at the age of 7 months when she had fever, nasal discharge, cough and generalised body rashes. She was admitted at a private clinic and was managed for a measles infection for a duration of one week. About two weeks following discharge she again developed an episode of cough, fast breathing and fever. She was re-admitted and managed for pneumonia and discharged after 5 days. Within a week of discharge, she was taken back to the health facility on account of worsening cough and fast breathing. She was seen as an outpatient and placed on oral antibiotics. With no improvement in the child's condition, the mother sought care at the clinic in the internally displaced peoples'(IDP) camp from where she was referred to us. There was a history of poor appetite.

Pregnancy and delivery were supervised at a Primary Health Centre (PHC) and there was no immediate neonatal problem. She was exclusively breastfed and received all routine immunization on schedule prior to onset of ill-health. She was the second child of parents in a monogamous family setting, the older sibling is alive and well. Both parents are farmers with a secondary level of education.

On examination she was conscious, small for age, mildly pale, afebrile (36.7^0C), not cyanosed, well hydrated and no pedal oedema. She weighed 7 kg with a length of 87 cm and weight-for-length Z-score (WLZ) of<-3 SD. She was dyspneic, had flaring of alar nasi, intercostal and subcostal recessions with respiratory rate of 80 breaths/min. The breath sound was vesicular with no added sounds. She had an oxygen saturation in ambient air of 98% on pulse oximetry. Her pulse rate was 148 bpm, regular, full volume; and a slightly bulging precordium with displaced apex beat at 5LICS MCL. The heart sound was I, II & III gallop rhythm with no murmur. Her abdomen was full, soft, with smooth, tender hepatomegaly of 6 cm.

Chest radiograph showed globular cardiac silhouette (CTR 0.66) with LV apex and increased pulmonary vascular marking [Fig 1]. An assessment of congestive cardiac failure 2^0 DCM in a severely malnourished child was made.

Echocardiography showed dilated left cardiac chambers (atrium and ventricle) with poor contractility, normal coronary artery and left sided aortic arch in 2D mode. Doppler showed mitral and tricuspid valve regurgitations. On M-mode, there was severe reduction in ejection fraction (EF 22%), and fractional shortening (FS: 10%). The left ventricular internal diameter in diastole (LVIDd) was 4.6 cm (+8 Z-score)[Fig 2]. With the echocardiographic findings, the diagnosis of dilated cardiomyopathy was confirmed. She was placed on diuretics, ACEI and digitalis for heart failure and nutritional rehabilitation was instituted. She responded to treatment and was discharged after the mother was counselled on the importance of treatment compliance. She was subsequently seen at follow-up clinic 2/52 later and the mother counselled again. However, patient subsequently defaulted follow-up for about 2/12 and was later brought in dead.

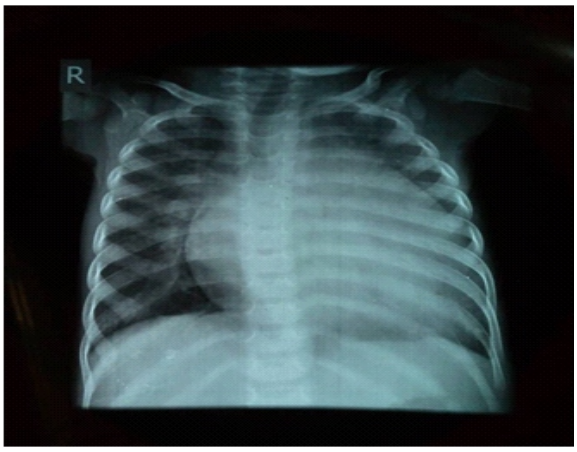

Fig 1: Chest Radiograph showing cardiomegaly

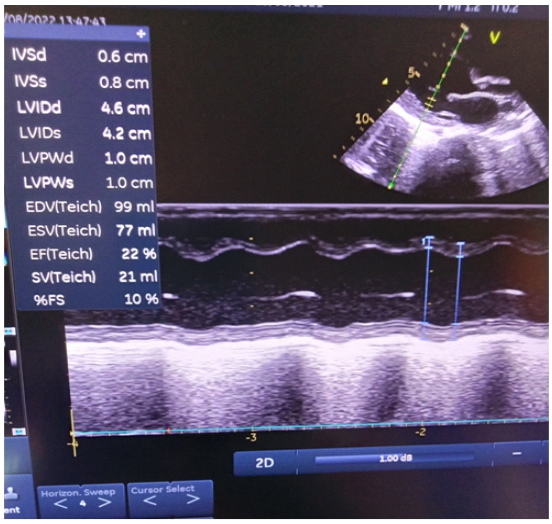

Fig 2: M-mode Echocardiography image

DISCUSSION

Dilated cardiomyopathy has a guarded prognosis; with about 1/3 of patients dying within the first two months of diagnosis, 1/3 remaining same and the other third improving or recovering completely[4]. It has been observed that those who improved usually do so within the first 6 months. Of the documented cases of measles myocarditis in children cited, only the 6 months old infant recovered without sequel[9]. This was because the diagnosis was made early and appropriate therapy was given. Our patient went in and out of hospitals for four months after the initial measles infection before presenting at our facility.

The presence of co-morbidities, in this case the severe malnutrition, significantly contributes to the morbidity and mortality associated with DCM. It is difficult to ascertain if the malnutrition is a cause or consequence of the DCM. Although, DCM of viral aetiology has better prognosis than familial/genetic DCM, outcome is however dependent on how early treatment was instituted as well as compliance. Our patient probably would have done better if the condition was recognised early, appropriate treatment instituted and if there was adequate social support to have prevented the early default.

CONCLUSION

Measles virus is a rare cause of myocarditis and requires a high index of suspicion to minimize delay in diagnosis and increase chances of better treatment outcome.

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