

Left Ventricular Outflow Tract Thrombus in a Child with Dilated Cardiomyopathy: An Atypical Location

Mohammed Baba Abdulkadir^{1,2}, Olayinka Rasheed Ibrahim², Folake Moriliat Afolayan², Olanrewaju Timothy Adedoyin^{1,2}

¹Department of Paediatrics and Child Health, University of Ilorin, ²Department of Paediatrics and Child Health, University of Ilorin Teaching Hospital, Ilorin, Kwara State, Nigeria

Abstract

Dilated cardiomyopathy (DCM) may be associated with formation of intracardiac thrombi which may embolize and result in life-threatening complications. We present a 19-month-old female child with DCM who presented with a right hemispheric cerebrovascular accident. Urgent echocardiography revealed poor left ventricular systolic function and a thrombus attached to the interventricular septum along the left ventricular outflow tract. There was resolution of the thrombus following the use of warfarin.

Keywords: Cerebrovascular accident, dilated cardiomyopathy, left ventricle, thrombus

INTRODUCTION

Intracardiac thrombi are a complication of dilated cardiomyopathy (DCM).^[1] Factors implicated in the formation of thrombi in DCM include low-velocity swirling of blood, abnormal endocardial surfaces, atrial fibrillation, and a hypercoagulable state.^[2] Thrombi complicate between 14% and 23% of DCM among children.^[1,3,4] The most common site for thrombi in DCM is the apex where blood flow is generally slowest.^[5] The major risk of the left ventricular thrombi is embolization to critical organs, particularly the brain where they may result in cerebrovascular accidents.^[6]

We present a case of the right hemispheric cerebrovascular accident in a 19-month-old Nigerian child with DCM.

CASE REPORT

AH is a 19-month-old female child seen first at 3 months of age on account of recurrent difficulty in breathing, cough, easy fatigability, and poor weight gain. She was small for age with small volume pulses, tachycardia, and hypotension with no cardiac murmurs. An echocardiogram confirmed a diagnosis of DCM, and she was commenced on oral hydrochlorothiazide and spironolactone. She was lost to follow-up 3 months later and presented again at 19 months of age with a 3 days history of sudden weakness of the left arm and leg. She had

no similar symptoms in the past. There was no fever, seizures, loss of consciousness, speech difficulty, cough, or difficulty in breathing. On examination, she was conscious, afebrile (axillary temperature -36.0°C), mildly pale, with no cyanosis, jaundice, or pedal edema. Her pulse rate was 116 beats per minute, and blood pressure was 80/40 mmHg with a raised jugular venous pressure. Apex beat was displaced and on auscultation had distant first and second heart sounds. She had nontender hepatomegaly. Pupillary light reflexes were normal. There was left upper motor neuron facial nerve palsy. Power was 1/5 in all muscle groups in the left upper and lower limbs with exaggerated deep tendon reflexes.

She had a sinus tachycardia with increased R amplitude in V5 and V6 on an electrocardiogram. Transthoracic echocardiography revealed a dilated globally hypokinetic left ventricle (LV) with a left ventricular internal diameter in diastole of 5.16 cm and fractional shortening of 10.0%. Other LV parameters are shown in Table 1. There was a mild pericardial effusion with maximal depth of 0.8 cm. On

Address for correspondence: Dr. Mohammed Baba Abdulkadir, Department of Paediatrics and Child Health, University of Ilorin, P.M.B. 1515, Ilorin, Kwara State, Nigeria.
E-mail: docmohng@gmail.com

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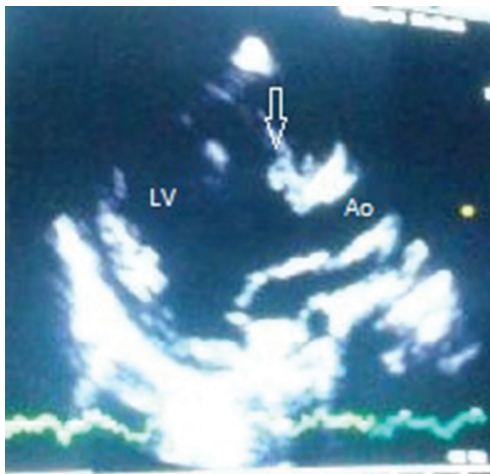


Figure 1: Parasternal long axis view demonstrating dilated left ventricle and thrombus (arrow) at left ventricular outflow tract. AO=Aorta

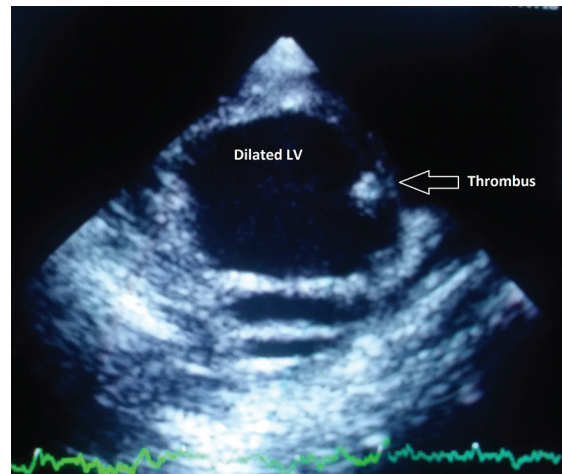


Figure 2: Modified parasternal view demonstrating thrombus. LV=Left ventricle

Table 1: Selected echocardiographic parameters at presentation and 6 weeks later

Parameter	At presentation	6 weeks later
Right ventricular diameter (diastole) (cm)	0.72	0.96
Left ventricular diameter (diastole) (cm)	5.16	5.08
Left ventricular diameter (systole) (cm)	4.62	4.28
Ejection fraction (%)	23.0	33.0
Fractional shortening (%)	10.0	16.0
Left ventricular mass (g)	96	98
Left ventricular mass index (g/m ²)	240	249

parasternal long axis and apical views, an echogenic mass was seen along the interventricular septum 1.8 cm from the aortic valve annulus within the LV [Figure 1]. A modification of the parasternal axis view made the mass more visible [Figure 2]. The mass was immobile and measured 0.86 by 0.49 cm in cross-section with an area of 0.34 cm². The mass was suspected to be a thrombus. There was functional mitral regurgitation with a vena contracta width of 0.4 cm, and the mitral valve was not thickened or deformed. Urinalysis was normal, and serial blood cultures revealed no growth. A diagnosis of DCM with right hemispheric cerebrovascular accident and a solitary left ventricular outflow tract (LVOT) thrombus was made. Baseline clotting profile revealed an international normalized ratio (INR) of 0.92. She was commenced on warfarin with close monitoring of INR to achieve a value of between 1.5 and 2.0. Subsequent INR achieved were between 1.11 and 1.96. She gradually recovered neurologic function over a 4-week period and became more ambulant with full power in the upper limbs and power of three in the left lower limb. A repeat echocardiogram done 6 weeks after presentation documented disappearance of the cardiac mass. The left ventricular systolic function still remained poor with a fractional shortening of 16.0%. Other parameters are shown in Table 1. She is currently on aspirin, hydrochlorothiazide, spironolactone, and captopril.

DISCUSSION

Cerebrovascular accident is not an uncommon complication in individuals with DCM often resulting from embolization of cardiac thrombi.^[6] However, it is rare for the thrombus to be located along the LVOT where flow, albeit diminished is not sluggish as compared to the apex or lateral wall.^[6] A plausible reason for this abnormal location is the geometry of the LV in this patient [Figure 1] with ballooning of the septum which may have created abnormal forces damaging the endocardium at the site. This mechanism is similar to the formation of nonbacterial thrombotic endocarditis in infective endocarditis following endocardial damage.^[7] Another plausible reason for the thrombus is an apical thrombus dislodged and “trapped” at the LVOT. Few authors have described the occurrence of a thrombus in the LVOT.^[8,9] Most of these cases were associated with LVOT obstruction and syncope. This did not occur in this case possibly because of its size and location.

In conclusion, thrombi can arise along the LVOT in children with DCM and may carry a higher risk of embolization.

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Conflicts of interest

There are no conflicts of interest.

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