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*Case Report*

# Burns Cardiomyopathy in a Nigerian Child

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**ABSTRACT**

DCM following thermal injury is rare, it has been reported in nearly 7% of children who sustained more than 70% burn injuries. The cardiomyopathy following burns often presents weeks to months after the injury.The cause is unknown although inflammatory mediators may play an aetiologic role. The cardiomyopathy resulting from this is reversible with appropriate medical management. To the best of the authors' knowledge, there are no reported cases in African children hence we present an eight year boy in apparent sound state of health before the burns he suffered. He developed features of congestive cardiac failure due to onset of dilated cardiomyopathy secondary to the injuries suffered from burns. He was commenced on tablet enalapril, digoxin, multivitamins, aspirin and calcium tablets. He improved clinically, his ejection fraction gradually improved on echocardiography was discharged after six weeks on admission. His became symptom free remarkably and he resumed school. He has been stable on follow, the last ejection fraction on follow up one year after diagnosis was 56%.

**Key words:** Burns, Cardiomyopathy, Nigeria, Child.

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## INTRODUCTION

Dilated cardiomyopathy (DCM) is a heart muscle disease characterised by the presence of a dilated and poorly functioning left or both ventricles. It is the most the most common cardiomyopathy worldwide and a common cause of congestive cardiac failure in children.

The aetiology of DCM may be the result of multiple genetic or a result of infection, inflammation, endocrine or metabolic disorders or malnutrition. Approximately 30% to 35% of patients have a genetic form of DCM.[1-3] Infants and children have a wider spectrum of causes[4-7] and identifying the aetiology may be challenging especially in resource poor and developing countries. Other causes in children includes long standing supraventricular tachycardia, burns cardiomyopathy, anomalous origin of the left coronary artery arising from the pulmonary artery and critical coarctation of the aorta.[8-11]

DCM following thermal injury is rare, it has been reported in nearly 7% of children who sustained more than 70% burn injuries.[10,12] The cardiomyopathy following burns often presents weeks to months after the injury.[10,13] The cause is unknown although inflammatory mediators may play an aetiologic role.[14-16] The cardiomyopathy resulting from this is reversible with appropriate medical management.[10,13] To the best of the authors' knowledge, there are no reported cases in African children. The case report below is that of a boy in apparent sound state of health before the burns he suffered. He developed features of congestive cardiac failure due to onset of dilated cardiomyopathy secondary to the injuries suffered from burns.

## CASE REPORT

An 8 years old boy who presented with a three month old history of progressively worsening breathlessness and a two month history each of easy fatigability on mild exertion, diaphoresis, palpitations, orthopnea and paroxysmal nocturnal dyspnea. He also had a history of facial swelling, chest pain and passage of blood stained frothy urine three weeks prior to presentation.

The breathlessness progressed over three months and became worse three days prior to presentation necessitating referral from the referring hospital. The chest pain was dull in nature and centrally located. There was neither a prior history of the use of a cosmetic or bathing soap with mercurial content, nor a history of oliguria, insect stings, abdominal and leg swellings.

Prior to the development of the above symptoms, he had varying degrees of partial and full thickness burns to his lower limbs, abdomen and fingers involving 38% of his total body surface area (TBSA) three months earlier (Figure 1). He received treatment for the burns injury at a burns unit in a tertiary hospital where he was referred from and he had two sessions of skin graft done with blood transfusions. The presenting symptoms were noticed after the skin graft. On account of the presenting symptoms, a diagnosis of post-burns nephropathy in anemic heart failure was entertained at the referring hospital. He was commenced on analgesics, antibiotics, multivitamins and vitamin C and referred to LASUTH for further management.

Ante-natal, birth and post-natal periods were uneventful. He achieved motor [gross and fine] and speech milestones appropriate for age. He is a basic two pupil with past results showing good academic performance. He had received all vaccines according to the National program on immunization schedule. He is the last of two children in a monogamous family.

On examination at presentation, he was in respiratory distress (flaring alae nasi, subcostal and intercostal recessions), afebrile, moderately pale, acyanosed in room air, with facial puffiness and pedal oedema up to mid-shin. He was tachypnoeic with respiratory rate of 66 cpm, and had coarse basal crepitations bilaterally on auscultation of the lungs. He was tachycardic with a pulse rate of 140 bpm which was regular, normal volume and synchronous with other peripheral pulses. He had normal blood pressure, 100/70 mmHg, hyperactive precordium, displaced diffuse apex beat located at the 6$^{th}$ left intercostal space mid-axillary line and a first, second and third heart sounds were present. Abdominal examination revealed a soft, smooth and tender hepatomegaly with a palpable edge 10cm below the right costal margin. Musculoskeletal system examination revealed healed scars on the thighs and legs, suprapubic region of the abdomen and index, middle, ring and little fingers of both hands. There were areas of hypertrophic scars and keloids formed at the back which were skin graft donor sites (Figure 2 and 3).

A diagnosis of congestive cardiac failure secondary to severe anaemia in a post burn patient with nephropathy was made. He was admitted,

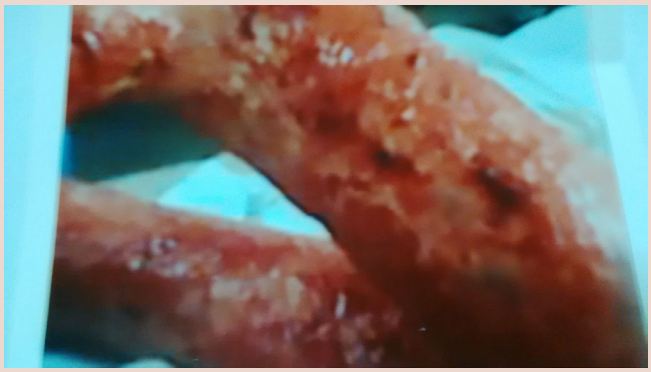

**Figure 1:** Part of the burns wound involving the lower limb.

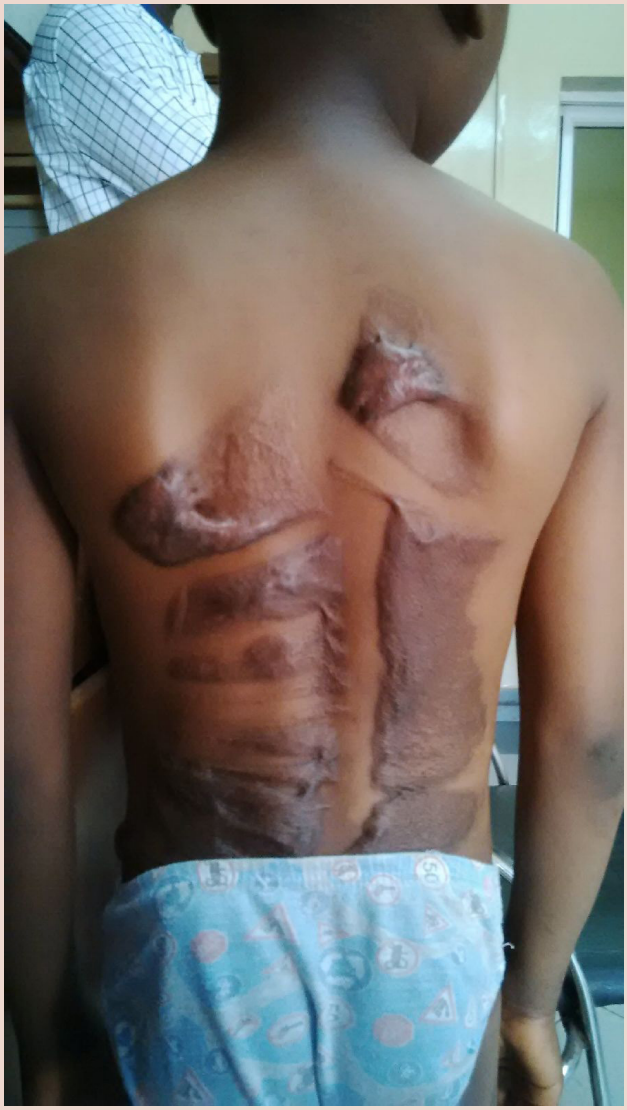

**Figure 2:** Donor site for wound grafting.

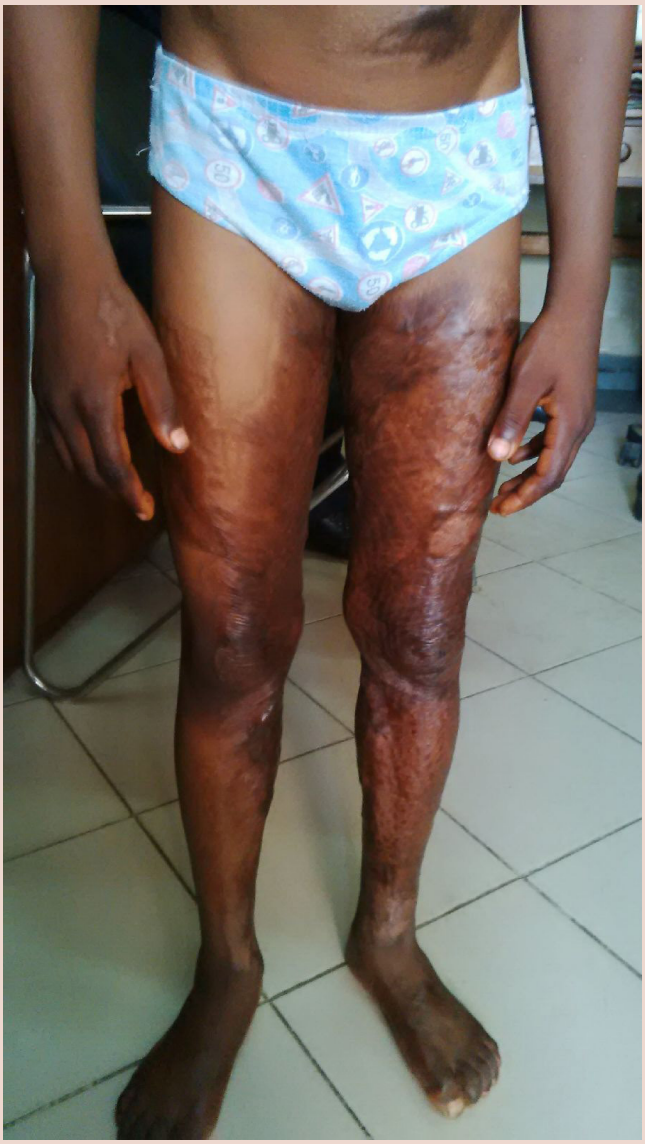

**Figure 3:** Healed burns wound with scar.

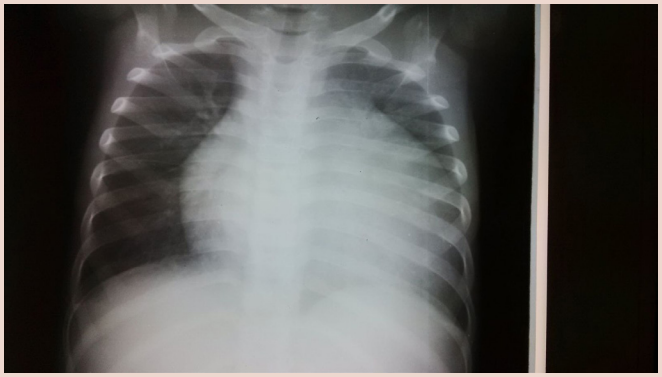

**Figure 4:** Chest radiography at admission showing cardiomegaly.

commenced on intranasal oxygen, intravenous furosemide and cefuroxime. Strict monitoring of fluid input and output, daily weighing and blood pressure monitoring were done.

Chest radiograph showed in Figure 4 showed cardiomegaly.

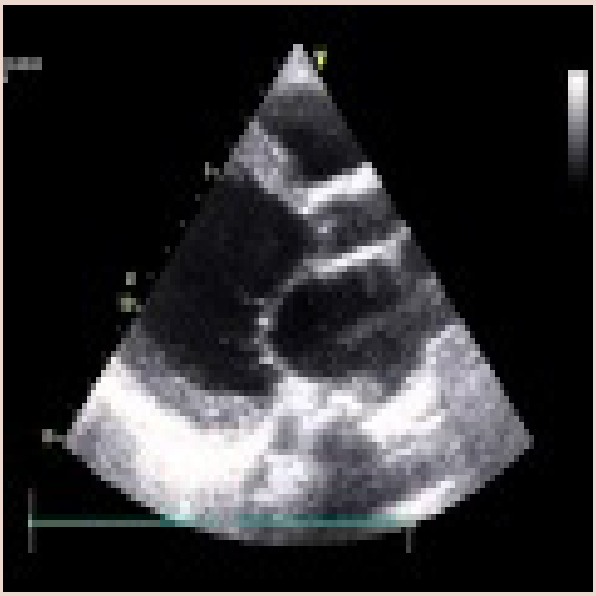

**Figure 5:** 2-Dimensional echocardiography at presentation showing poor contractility.

Click the link below for video https://youtu.be/Li8q435e0Lg

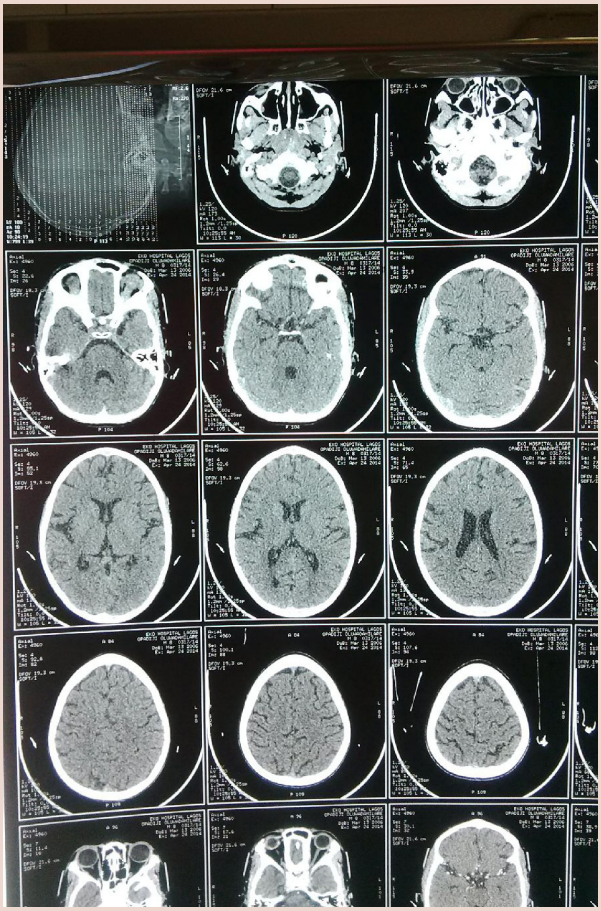

**Figure 6:** Magnetic resonance imaging of the brain.

Electrocardiography (ECG showed sinus tachycardia, left atrial abnormality, leftward axis deviation, and low voltage on limb lead, left ventricular hypertrophy with polarization. Abdominal ultrasound scan revealed an enlarged liver.

Echocardiography (Figure 5) revealed dilatation of all the cardiac chambers with ejection fraction of 25% with minimal circumferential pericardial effusion suggesting a cardiomyopathy. Urea was elevated but creatinine was normal. The full blood count results were normal, urinalysis revealed hematuria and proteinuria, a 24 hours urine protein was elevated but clotting profile was normal. The dark urine cleared and a repeat electrolyte and urea were normal.

A diagnosis of DCM following burns was made following the results of investigations. He was commenced on enalapril, digoxin, multivitamins and calcium tablets. While on admission, he developed a transient ischemic attack which affected his right arm and leg and was aphasic, aspirin was added and a MRI scan of the brain was done which revealed a deepening of the sulci and widening of the fissures as well as ventricles suggesting brain atrophy. No focal lesion was seen in the cerebellum, brainstem and cerebral hemispheres (Figure 6). The event resolved with 24 hours and both motor and speech functions restored. The ejection fraction gradually improved on echocardiography and he improved clinically and was discharged after six weeks on admission.

His appointments were kept regularly and echocardiography was done on each visits. His symptoms have improved remarkably and he is back in school. The last repeat electrocardiography done one year after his initial presentation with DCM revealed sinus tachycardia, right and left atrial enlargement, normal axis, evidence of biventricular hypertrophy and he is still on his medications.

## DISCUSSION

Severe thermal injury is known to cause DCM. Cardiomyopathy following burns, have been reported in both children and adults.[10,13] The cause is not fully understood, however several factors that include plasma volume loss, hypoxia, release of hormones and complex interplay of inflammatory cytokines have been implicated.[14]

The initial response to severe burn injury is characterized by a decrease in cardiac output and metabolic rate.[17] Other haemodynamic features of burn shock includes a decrease in stroke volume, venous return, coronary blood flow, peak systolic blood pressure, mean arterial pressure, estimated myocardial work, stroke work, myocardial oxygen consumption, myocardial oxygenation, myocardial contractility and myocardial compliance.[14] These initial response will result in both right and left heart failure and depression in cardiac contractility and it is thought to be mediated by circulating vasoconstrictors.[18,19]

Extra-cardiac changes noted following severe burns includes elevated plasma levels of catecholamines, vasopressin, angiotensin-11 and neuropeptide-Y which despite adequate resuscitation may have deleterious effects on the cardiovascular system.[14] Vasopressin plays a role in the initial decrease in myocardial contraction and it may be partly because of coronary constriction.[20] With severe burns there is a 10-20 fold surge of catecholamines that mediate profound hyper-metabolic response which results in cardiac deficiency, local myocardial hypoxia and cardiac death.[21-25] The plasma catecholamines are elevated months to years post burns and the derangement in cardiac physiology can last up to 2 years.[21,22,24] Prolonged exposure of circulating catecholamines have been implicated in cardiomyopathies, myocarditis and myocardial lesions.[26,27]

Inflammatory sequel to burn including cytokine release, activation of the complement cascade, neutrophil adherence and activation, release of free radicals and an increase in intracellular calcium[28-30] may serve to incite and propagate cardiac dysfunction.[14] Inflammatory mediators

or cytokines directly implicated in this cardiotoxicity includes TNF-α (tumor necrosis factor), IL-1β, IL-2, IL-6 and IFN-gamma.[31,32] TNF-α is a multifunctional cytokine implicated in several cardiac related conditions including congestive cardiac failure, septic cardiomyopathy and cardiac dysfunction following burns.[33] Both systemic and local production (from the myocytes) of high levels of TNF-α can be found in serum following major burns and this may contribute to myocyte apoptosis and subsequent cardiac dysfunction.[14] The interaction of the hemodynamic changes, hormonal changes and inflammatory mediators results in cardiotoxicity and cardiomyopathy.[14,34]

The cardiotoxicity may not be clinically evident until a few weeks to months after the burns injury. Patients with turbulent clinical course are more likely to develop cardiomyopathy.[10] The subject in the case write up developed congestive cardiac failure three months after the thermal injury. Mak et al[10] also reported burns in boys with full thickness burns and the symptoms developed more than 30 days after the burn injury. Similarly, Chen et al reported DCM in three adults six months after the burns injury.

Other notable finding in the index case was the development of a transient ischemic attack which resolved spontaneously without a recurrence. The cause of that event is not known and there no previous documentation of a similar findings in other authors who have reported burns cardiomyopathy.

The cardiomyopathy following burns is treatable and reversible. Our patient received digoxin, Angiotensin converting enzyme inhibitor (ACEI), and calcium supplements with remarkable improvement which was evident both clinically and with echocardiography within one year of treatment. Mak et al administered digoxin, diuretics, ACEI and beta-blockers to their burns and within 2-11 years of follow up, all the subjects recovered. Chen et al also documented recovery on all their subjects with burns cardiomyopathy.

## CONCLUSION

We documented a case of post-burn cardiomyopathy. Although it is a rare cause of DCM, it has been reported previously in advanced countries on patients who had severe burn injuries. It presents with congestive cardiac failure weeks to months after the initial burn injury. The cause is poorly understood, but a combination of inflammatory mediators, hormonal and hemodynamic changes after the burns injury has been implicated. The myocardial damage is treatable and reversible.

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