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A Case Report

# Reversibility of Dilated Cardiomyopathy in a child with Sepsis

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***ABSTRACT***

***D****ilated Cardiomyopathy is a diagnosis that usually carries a serious prognosis. However, among the numerous cases of dilated cardiomyopathy a few that are acute, fulminant and potentially reversible.*
*Sepsis induced cardiomyopathy is a reversible myocardial dysfunction that typically resolves in 7-10 days. It is characterized by left ventricular dilation and depressed ejection fraction.*

*Sepsis induced cardiomyopathy leads to a significant morbidity and mortality if not diagnosed early and treated judiciously with efficiency. Although the syndrome remains a dilemma along with conventional treatment of dilated cardiomyopathy, intensive fluid therapy, Ivabradine, trimetazidine dihydrochloride and above all aggressive source control and use of broad-spectrum antibiotics and maintenance of optimum hemodynamics with vasoactive agents. Our case study is reflecting that whenever an acute onset cardiomyopathy is found a reversible cause should be looked for.*

***Key words:*** *Reversible Dilated Cardiomyopathy, Sepsis, Ivabradine, Trimetazidine dihydrochloride.*

## Case History

### 1. Introduction

Abid , 9 years & 4 months old young boy weighing 41 kg, 1st issue of non-consanguineous parents presented with fever for 5 days, highest peak 105`F ,rashes for 4 days, eye congestion for 2 days associated with nausea, vomiting & weakness .On admission he was febrile with tachycardia and tachypnoea. His pulse volume was moderate, blood pressure was 80/50 mm of Hg, H.R 140 beat/min, RR 45 /min SPO2 92% in R/A. Skin survey revealed palpable erythematous rashes over legs, hands & trunk which blanch on pressure. Precordial examination revealed apex beat at left 6th ICS lateral to MCL with a prominent left ventricular heave, normal heart sounds without any added sound. He had fine crepts on both lung fields. His abdomen was soft with epigastric tenderness with no organomegaly. No signs of meningism, no significant lymphadenopathy was noted.

Electrocardiography showed that sinus tachycardia with left ventricular hypertrophy and strain. Heart rate was 140 b/min. PR interval was prolonged (170 msec, normal 150 msec) & his QRS duration was normal. His QTC was prolonged (655 msec, normal less than 440 msec). He had also T wave inversion in lateral leads and features of LVH. He had also T wave inversion in lateral leads and features of LVH. ST elevation and pathological q wave.

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Chest X-ray showed patchy opacities with air broncho-gram in mid and lower zones of both lung fields consolidation with pulmonary edema, pleural effusion, left dome of diaphragm is ill-delineated. pulmonary venous hypertension and interstitial pulmonary edema. Two- dimensional echocardiography revealed moderate left ventricular dilatation and hypertrophy, as well as severe global hypokinesis. The ejection fraction was severely depressed (35% [normally 60% plus or minus 6.25%]). The total creatinine kinase concentration and the myocardial isoenzyme level were within normal limits.

Investigation showed in CBC- neutrophilic leukocytosis with eosinophilia, DIC profile showed high fibrinogen level ( 407 mg/dl,[normal180-350]), high D-dimer (4360 microgram/L[normal<500]), high C-reactive protein(24.5mg/dl[normal<=0.33]),high Pro-calcitonin(127.18 ng/ml[normal <0.05]),high prothrombin time(15.5sec(control12sec])and INR 1.30,Triple antigen negative, urine profile – normal, S. electrolytes- hyponatremia, hypoalbuminemia, low S. calcium 6.9 mg/dl, negative Anti dsDNA Antibodies and Antinuclear antibody (ANA), s. Troponin I 120 pg/ml (125pg/ml) was normal and total CPK was mildly raised 490 U/L (22-198 U/L.),NT-pro BNP 1500 pg/ml ( 125 pg/ml),[normal]), chest X-ray shows patchy opacities with air broncho-gram are noted in mid and lower zones of both lung fields consolidation with pulmonary edema, pleural effusion, left dome of diaphragm is ill-delineated. So sepsis was diagnosed with the above investigation.

After admission, he was treated with Inj Ceftriaxone initially then Inj Meropenem, Inj Vancomycin & Inj Amikacin were added, Inj. Ondansetron, Inj. Pantoprazole, Paracetamol, Oxygen inhalation, IVIG ( 2 gm/kg) and I.V fluid . Shortly after admission, he gradually became dyspneic & restless, SPO2 remained 92-93% with 5L O2 , he had tachycardia (HR 150- 160/min), tachypnoea (RR 56-60/min), SPO2 95 % with 5L O2, BP 70/40 with low pulse volume & cool periphery . As management of shock, started normal saline I.V bolus followed by Inj Noradrenaline & Inj Dopamine in I.V drip.

Echo was done which revealed Dilated cardiomyopathy with EF 35 % , and Inj. Dobutamine , Inj. Frusemide in I.V drip, Tab. Digoxin, Tab. Ramipril, Tab Ivabradine, Tab. Trimetazidine , Tab. Spironolactone, Tab Thiamine was added. Abid was intubated and put on mechanical ventilator, as ABG revealed features of Type I respiratory failure.

As his BP was not improving even with inotrope support & PaO2/Fio2 revealed early ARDS so, Inj hydrocortisone was added as a management of catecholamine resistant shock with early ARDS. His dis-electrolytemias, hypocalcemia were corrected accordingly, Inj. 20% Albumin, 1 unit of PRBC & Inj. Vitamin D3 orally during ICU stay. After improvement of Echo & B.P, inotropes were stopped gradually, among the cardiac medications Tab. Digoxin, Tab. Trimetazidine dihydrochloride, Tab. Carvedilol & Tab. Ramipril were continued.

Tab. Fludrocortisone was added for raised spot urinary sodium. Pediatric nephrology consultation was taken for later developed diuresis.

After 4 days echo showed improvement of left ventricular function (LVEF 48%). After 7 days the patient was extubated, and oral feeding was started along with oral medications. After 10 days of shock cardiac function improved with LVEF 55 %. He was managed by the team of Pediatrician, Pediatric Cardiologist, ICU team, and Radiologist.

Course in the hospital: On day 19th the baby was discharged with oral medications, tab. carvedilol, tab ramipril, tab trimetazidine dihydrochloride, tab. fludrocortisone, tab. calcium, tab thiamine, syrup KCL, tab. pantoprazole.

After one month of follow up he was asymptomatic and normotensive with the medication. Two-dimensional echocardiography revealed normal left ventricular dimensions and contractility with ejection fraction was 68%. Gradually we could stop all cardiac medications.

**Methodology:**

**Discussion:**

Reversible cardiomyopathy have long fascinated cardiologists around the world (1-5).Reversible myocardial dysfunction (RMD) was first described in canine experimental model by Heyndrickx and coworkers at 1975 (6,7).The phenomenon of RCM generated after the induction of transient ischemia in coronary arteries without producing necrosis was called “stunned myocardium” in 1982.It was self- limited but could lead to decreased ventricular compliance(6,8).

The reversible cardiomyopathies can be classified into two groups, those due to metabolic causes and those due to hypocalcaemia (5). Reversible causes of dilated cardiomyopathy classified as

Metabolic:

a. Thyrotoxicosis
b. Cushing disease
c. Due to hypolcaemia.
    1. Due to D3 deficiency: Sepsis, Malnutrition, Kwashiorkor
    2. Due to repeated blood transfusions: Hypoparathyroidism

Hypocalcemia due to repeated blood transfusion in a Thalassemia patient has been reported to cause a dilated cardiomyopathy that improved with treatment. (9)

Hypoparathyroidism can cause severe hypocalcemia and cardiomyopathy and correction with recombinant parathormone has reversed the heart failure (10).

After thyroidectomy, parathyroid deficiency can occur and can cause a reversible cardiomyopathy (5)

Other correctable cardiomyopathies are Takotsubo myocarditis, tachy cardiomyopathy, cocaine cardiomyopathy, drug induced cardiomyopathy due to interferon alpha therapy, amphetamine

induced cardiomyopathy, thyrotoxic cardiomyopathy or sepsis (9-14).

Finally, in our patient's sepsis induced cardiomyopathy was diagnosed which was treated judiciously and successfully. Sepsis causes an acute cardiomyopathy that reverts to normal if the patients survives (5,13,14) have described the pathogenic mechanisms of the reversible cardiomyopathy that occurs during sepsis. In sepsis initially there is a "hyperdynamic shock" but the sepsis hearts have a lower stroke work index with a rightward shift of the Frank-Starling mechanism. In sepsis it is believed that cytokines mediate a cardiomyopathy. The cytoplasmic reticular Ca-ATPase is affected in sepsis (SERCA). The phosphorylation of SR proteins is affected in late sepsis. The phenomenon called "cytopathic hypoxia" occurs in sepsis. This is impaired mitochondrial oxygen content in spite of adequate oxygen supply (9). TNF (Tumour necrosis factor) alpha also triggers myocardial apoptosis.

Parker has described a "profound but reversible myocardial depression in septic shock "in a series of 20 patients (5,15,16). Myocardial depression is a major contributor to mortality and morbidity in septic patients. Contractile dysfunction in the septic heart is manifest as biventricular dilation, reversible decrease in ejection fraction, diminished blood pressure response to IV fluids and blunted ability to augment cardiac output despite increased levels of circulating catecholamines (16).

In 1984, Parker et al. reported decreased ejection fraction and increased end-diastolic volume in septic shock survivors. These changes in left ventricular function were of rapid onset and reversed over 7–10 days in survivors; however, they were less profound in those who died (16, 17,18)

Ryota Sato, Michitaka Nasu et al described in" Sepsis-induced cardiomyopathy that despite the lack of diagnostic criteria for sepsis-induced cardiomyopathy to date, it is known to have three characteristics. The first is left ventricular dilatation with normal or low filling pressure. This probably occurs due to an increase in the left ventricular compliance, which was first described in patients with septic shock in 1984 (16,17,18). Later, another study of the left ventricle response to volume loading showed that there was an abnormal increase in left ventricular end-diastolic volume in sepsis survivors, implying increased ventricular compliance (19). The second characteristic is depressed ejection fraction. Parker et al. reported that end-diastolic and systolic ventricular volumes were increased but with normal or elevated stroke volume and cardiac index in septic shock survivors. In this study, although the number of patients are less, these results suggest that decreased ejection fraction may be caused by ventricular dilatation and not by decreased stroke volume. Because ejection fraction is defined as the stroke volume divided by the end-diastolic ventricular volume, the denominator increases as the ejection fraction decreases (18). Vincent et al. demonstrated that the left and right ventricular ejection fractions are depressed in patients with septic shock (20,21). Other studies support this result and have shown the development of right ventricular dilatation (17.22,23). The third characteristic of sepsis-induced cardiomyopathy is that it should normalize within 7–10 days (16, 24,25). In sepsis-induced cardiomyopathy diagnosis, the first and second characteristics are particularly important and easy to detect using echocardiography. Thus, echocardiography in sepsis management is the most important thing for diagnosing sepsis-induced cardiomyopathy.

In his study "Profound but reversible myocardial depression in patients with septic shock" by Parker MM et al described that to characterize the role of cardiac function in septic shock, serial radionuclide cine angiographic and hemodynamic evaluations were done on 20 patients with documented septic shock. Although all patients had a normal or elevated cardiac index, 10 patients had moderate to severe depression of their ejection fraction with values below 0.40. Thirteen of twenty patients survived their episode. Paradoxically, 10 of 13 survivors, but none of the 7 non survivors, had an initial ejection fraction less than 0.40 (p less than 0.005). The mean initial ejection fraction for the survivors was 0.32 +/- 0.04, and their mean end systolic and end diastolic ventricular volumes were substantially increased with a normal stroke volume. The survivors' serial scans showed a gradual return to normal ejection fraction and ventricular volume by 10 days after the onset of shock (16).

Immediately after admission we evaluated the cardiac function of our patient. His cardiac index was near to normal but left ventricular EF was 0.38 with LV E/E' ratio 16 and TAPSE was 17mm with global hypokinesia with biventricular dilation. In a descriptive study of regarding the analysis of ventricular contractility by echocardiography, it is known that some patients experience segmental contractility alterations, with hypokinesis mainly at the apex and basal LV segments (6,17,18 26,27).

The LV diastolic function (compliance) can be studied using transmitral and pulmonary veins Doppler-pulsed and Doppler tissue imaging echocardiography (6).In the study "Sepsis-induced cardiomyopathy" by Francisco J, Manuel R et al described that noninvasive assessment of diastolic filling by Doppler echocardiography provides important information about LV status. The ratio of mitral velocity to early diastolic velocity of the mitral annulus (E/E') by tissue Doppler imaging that combines the influence of transmitral driving pressure and myocardial relaxation are associated with invasive measures of diastolic LV performance, and had shown to predict the mean left ventricle diastolic pressure (M-LVDP). In shock patients preferences in the measurement site of Doppler tissue imaging E 'maximal velocity (at lateral or septal mitral annulus) had not been shown (28).A ratio E/E '<8 accurately predicted normal M-LVDP, and E/E'> 15 identified very high M-LVDP. Wide variability was present in those with E/E' from 8 to 15 (29).

Electrocardiography showed that sinus tachycardia with left ventricular hypertrophy and strain. Heart rate was 140 b/min. PR interval was prolonged (170 msec, normal 150 msec) & his QRS duration was normal. His QTc was prolonged (655 msec, normal less than 440 msec). He had also T wave inversion in lateral leads and features of LVH with ST elevation and pathological q wave.

Gardner et al. have described the ECG changes in both hypocalcaemia and Hypercalcemia. Hypocalcaemia causes on lengthening of the QT segment or QT prolongation they also report that those with a corrected QTc of more than 500 m secs have an increased chance of arrhythmias. Various authors have also described hypocalcaemic cardiomyopathy in different cases that reversed with treatment (30,31,32). However, our child did not have any arrhythmia.

His posterior anterior chest x-ray film showed cardiomegaly with pulmonary edema.

Immediately after admission we corrected the dehydration of the child with judicious fluid and started dopamine (10 microgram/kg/min) and noradrenaline (0.2 microgram/kg/min) to maintain the MBP at 50$^{th}$ centile. Sympathetic overstimulation in critically ill patients (catecholaminergic storm) affects the heart and can lead to impaired diastolic function, tachycardia and tachyarrhythmias, myocardial ischemia, stunning, apoptosis and necrosis (33). In our patient dopamine caused tachycardia (>180b/min) and we had to reduce the dose (5 microgram/kg/min) (6). we started inf. dobutamine judiciously after few hours as afterload reducing agent because the child had persistently low cardiac output despite adequate LV filling pressure and clinically after getting proper fluid resuscitation (6). Dobutamine is the preferred treatment recommended by current clinical guideline.

Ryota Sato, Michitaka Nasu et al described in"Sepsis-induced cardiomyopathy" is characterized by left ventricular dilatation and depressed ejection fraction that typically normalize within 7–10 days. Our current understanding is that sepsis-induced cardiomyopathy is induced by endotoxins and cytokines and that the initial management should be the same as for septic shock without cardiomyopathy. However, the lack of quality evidence that dobutamine improves survival and the concerning reports that it may adversely affect outcomes in patients with sepsis imply that the routine use of dobutamine should no longer be recommended. In the near future, levosimendan or mechanical support with ECMO may be developed as a therapeutic option, but further study is needed to confirm whether it is truly effective in sepsis-induced cardiomyopathy. Levosimendan has beneficial effects on both ventricles, independent of the beta-adrenergic signaling or changes in intracellular calcium concentration, by increasing contractile myofilament sensitivity to calcium (6). Although we did not start levosimendan.

We treated our child having sepsis induced dilated cardiomyopathy by preload reducing agent, afterload reducing agent and inotrope. We started beta blocker (carvedilol). Over 50 years of experimentation with beta-blockers in sepsis, since Berk studies in 60s and 70s, have been insufficient for its inclusion in universal guidance of sepsis patients with heart failure, demonstrating that beta blockers are effective preventing ischemia, decreasing oxygen demand (reducing cardiac output up to 20% without worsening of oxygen utilization or increase lactate levels, allowing for greater preserving cardiac function. As the evidence suggest that beta adrenergic stress is a major factor in the pathogenesis SIMD, the use of beta-blocking agents could be beneficial (6,34).

Day after starting we had to stop tab carvedilol to maintain blood pressure as his blood pressure was continuously below 50$^{th}$ centile. We started tab Ivabradine by nasogastric tube. Ivabradine (acts on the $I_f$ (*f* is for "funny", which is highly expressed in the sinoatrial node. $I_f$ is a mixed $Na^+$–$K^+$ inward current activated by hyperpolarization and modulated by the autonomic nervous system, selectively inhibits the pacemaker $I_f$ current in a dose-dependent manner. Blocking this channel reduces cardiac pacemaker activity, selectively slowing the heart rate and allowing more time for blood to flow to the myocardium (35,36).At the same time we also started, Tab Trimetazidine (inhibits β-oxidation of fatty acids through inhibition of long-chain 3-ketoacyl-CoA thiolase, which enhances glucose oxidation. It ensures proper functioning of ionic pumps and transmembrane Na-K flow by preventing decrease in intracellular ATP levels which were essential for recovery of cardiac function in sepsis induced dilated cardiomyopathy (37,38)

An integral part of medical treatment for underlying heart failure we kept the baby on diuretics (frusemide), digoxin and ACE inhibitor (enalapril) after extubating. But the child's serum creatinine was at higher limit and we changed it to tab ramipril.

Our child also treated with tab calcium and inj vit D. As he had hypocalcemia. Hypocalcemia was due to sepsis. As his other causes of hypocalcemia (hypoparathyroidism, repeated blood transfusion, malnutrition (10,39) were excluded.

Prabha et al described a patient with reversible cardiomyopathy due to symptomatic hypocalcemia with hypoparathyroidism and hypothyroidism (post thyroid surgery) who was treated accordingly and responded well (5).

**Conclusion:**

Sepsis-induced cardiomyopathy is characterized by left ventricular dilatation and depressed ejection fraction that typically normalize within 7–10 days. Our current understanding is that sepsis-induced cardiomyopathy is induced by endotoxins and cytokines and that the initial management should be the same as for septic shock without cardiomyopathy. As demonstrated in the case described here the use of appropriate medical therapy is important for reversing severe sepsis induced dilated cardiomyopathy and for timing the intubation so that the patient recovered early with severely compromised left ventricular function

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