

Case Reports

Dilated Cardiomyopathy with Entamoeba Histolytica Associated Hemolytic Uremic Syndrome in a Child: A Case Report with Review of the Literature

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Abstract

This research aims at reporting an unusual 16 month old female who developed dilated cardiomyopathy and type IV renal tubular acidosis during the course of diarrhea positive Hemolytic Uremic Syndrome (D+ HUS) following Entamoeba histolytica infection.

D+ HUS with cardiomyopathy and Entamoeba histolytica is rare. Associated Type IV RTA has not been described in the literature.

Keywords: Cardiomyopathy, Hemolytic Uremic Syndrome, Entamoeba Histolytica, Hyperkalemic Metabolic Acidosis.

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Introduction

Diarrhea positive Hemolytic Uremic Syndrome (D+HUS) is associated with multiorgan involvement. However, involvement of the heart is rare. Herein, we report a case of a young female with heart failure secondary to dilated cardiomyopathy, during the course of D+HUS, following Entamoeba histolytica infection.

Case Report

A previously well 16 month old girl was admitted with bloody diarrhea, vomiting, and fever. Physical examination revealed a borderline

blood pressure for age, moderate dehydration, and pallor.

On admission, the patient had leukocytosis with 88% segmented polymorphonuclears, with normal platelet count, hemoglobin, creatinine, urea, and serum electrolytes. Stool analysis showed red and white cells, in addition to Entamoeba histolytica cysts and trophozoites. Urine culture was positive for Escherichia Coli (non OH7: 157), and negative for shigella. The patient was put on ceftriaxone and metronidazole. On the third day at hospital, the patient developed edema. Blood pressure was 110/75 (> 95th percentile for age and gender).

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Blood Urea Nitrogen (BUN) was 18 mmol/L, creatinine 255 umol/L, sodium 128 mmol/L, Potassium 4.6 mmol/L, blood sugar 5.6 mmol/L, hemoglobin 10.3 gm/dL, white blood cell count $25.2 \times 10^9/L$, platelet count $31.0 \times 10^9/L$, schistocytes in peripheral smear. The patient was put on insensible water losses. On the fifth day, hemoglobin was 8.1 gm/dL, platelets $100.0 \times 10^9/L$, albumin 2.3 gm/dL, creatinine 546 umol/L, BUN 35 mmol/L, uric acid 1.0 mmol/L, and Lactic dehydrogenase 6480 U/L.

Hemoglobin dropped to 6.4 gm/dL. The patient needed two packed red cell transfusions. Peritoneal dialysis was initiated on the seventh day at hospital, and discontinued on the eleventh day. On the sixteenth day, urine output increased, serum creatinine and urea started to drop, hemoglobin and platelets increased.

On the 22nd day at hospital, the patient developed tachypnea, an enlarged liver, and a gallop rhythm. Chest film showed an enlarged heart. Echocardiography disclosed an ejection fraction of 59%, mild left ventricular dysfunction, moderate mitral regurgitation, moderate left atrial dilatation, and mild tricuspid regurgitation. The patient had metabolic acidosis. Cardiac enzymes were normal. The patient was put on insensible fluid losses, furosemide, and dobutamine. Tachypnea improved. On the 26th day at hospital, hydralazine was started for hypertension. Serum creatinine was 132 umol/L, BUN 15 mmol/L, uric acid 0.29 mmol/L, and potassium 6.7 mmol/L, in association with a normal anion gap metabolic acidosis and a positive urine anion gap. The patient was discharged from hospital on the 38th day on sodium polystyrene sulfonate, sodium bicarbonate, and hydralazine.

The patient kept having hypertension, metabolic acidosis, hyperkalemia. Captopril was added. A 24 hour urine collection revealed > 1 gram proteinuria. A renal biopsy showed severe focal and segmental glomerulosclerosis by light and electron microscopy. Immunofluorescence was negative. The arterioles were severely narrowed.

Eight months after the acute illness, the patient still had hypertension, and hyperkalemia with

metabolic acidosis. The serum creatinine dropped to 53 umol/L. A recent echocardiograph showed an ejection fraction of 57%, with left ventricular end diastolic diameter 3 cm, and no mitral regurgitation. Plasma aldosterone and rennin were normal.

Discussion

The purpose of this paper is to report an unusual 16 month old female who developed dilated cardiomyopathy during the course of D+ HUS with entamoeba histolytica infection, a non E coli O157:H7 UTI, and an associated type IV renal tubular acidosis.

The D+HUS extrarenal manifestations include the heart.

Cardiac involvement is very rare in D+HUS. It may be restricted to the myocardium,¹⁻⁴ or the pericardium.⁵

Cardiomyopathy may occur during the acute illness⁶⁻⁸ as in our patient, or manifest as a late complication of the disease.¹ Bhimma et al. from South Africa, reported cardiomyopathy in 3.7% of HUS cases during an epidemic of post Shigella dysentery HUS.⁷

Volume overload, electrolyte disturbances, and hypertension are associated with cardiovascular dysfunction in D+HUS.⁹

Poulton et al. reported two children with dilated cardiomyopathy associated with D+HUS in the absence of hypertension, fluid, or electrolyte imbalance.²

In our patient, the cause of the cardiomyopathy is multifactorial. In addition to the microangiopathy, hypertension, and hyperkalemia probably played a role.

Our patient had a non-Escherichia coli O157:H7 UTI. Although there are reports of D+HUS following non E coli O157:H7 UTI,¹⁰ we cannot be certain that it was etiologic in our case.

Entamoeba histolytica has been associated with D+HUS.¹¹

To the best of our knowledge, there is no mention in the literature of cardiomyopathy complicating *Entamoeba histolytica* HUS.

The most common extraintestinal manifestation of amebiasis is liver abscesses followed by pleuropulmonary involvement.^{12, 13}

In addition, after conducting a Medline search, we could find one mention of hyperkalemia (attributed to medication) during recovery from HUS.¹⁴

In conclusion, a case of *Entamoeba histolytica* associated HUS is described, with cardiomyopathy and type IV renal tubular acidosis. It is important to look for cardiomyopathy if a patient with HUS develops tachypnea after an apparent recovery.

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اعتلال عضلة القلب في التنادرة اليوريمية الانحلالية بعد الإصابة بالأميبيا في طفلة: تقرير حالة مرضية ومراجعة الأدبيات

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الملخص:

نورد في هذا التقرير حالة غير عادية لطفلة اصيبت بالأميبيا ومن ثم التنادرة اليوريمية الانحلالية المصحوبة باعتلال عضلة القلب وحمضاض انيبيبات الكلبي من النوع الرابع. الالتهاب المعوي بالاميبيا واعتلال عضلة القلب في حالات التنادرة اليوريمية الانحلالية هي نادرة الحدوث. أما حمضاض انيبيبات الكلبي فلم توصف من قبل.

الكلمات الدالة: اعتلال عضلة القلب، التنادرة اليوريمية الانحلالية، الأميبيا، حمضاض انيبيبات الكلبي.