



# Rotavirus Gastroenteritis Associated with Encephalopathy, Myositis, Transaminitis and Hypoalbuminemia

Maharshi Trivedi<sup>1</sup> · Abhishek Jain<sup>1</sup> · Dheeraj Shah<sup>1</sup> · Piyush Gupta<sup>1</sup>

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## Abstract

Rotavirus is a common cause of acute gastroenteritis in children. Manifestations of rotavirus gastroenteritis beyond gastrointestinal tract are rare. Rotavirus has been reported to be associated with encephalopathy, myositis and elevated liver enzymes; but simultaneous presentation of all these conditions in the same child is extremely rare. The authors report a case of 17-mo-old girl who presented with acute rotavirus gastroenteritis with G3 + G9P[8] strain associated with hypernatremia, encephalopathy, myositis, transaminitis and hypoalbuminemia. Child had complete recovery with no neurological sequelae on follow-up, and liver enzymes and albumin returned to normal. The authors suggest that rotavirus infection should be considered in the differential diagnosis of a child with encephalopathy or myositis, particularly if associated with acute diarrhea.

**Keywords** Rotavirus infection · Encephalopathy · Myositis · Transaminitis

## Introduction

Rotavirus gastroenteritis has been rarely reported to cause extra-intestinal symptoms [1, 2] and are usually restricted to one site. The authors report a child with rotavirus gastroenteritis with multisystem involvement in form of encephalopathy, myositis, transaminitis and hypoalbuminemia.

## Case Report

A 17-mo-old developmentally normal girl, without any significant past history, reported with complaints of loose stools, vomiting and fever for 7 d along with lethargy and swelling of feet for last 3 d, and an episode of convulsion. Loose stools were watery, 6–8 times per day, and without any associated blood or mucus. The child had received intravenous fluids and medications, including phenytoin. There was no history of abdominal distension, decreased urine output, or any history suggestive of shock or hemodynamic instability during treatment outside the hospital. She had not received rotavirus vaccine.

On examination, child was lethargic but there was no sign of dehydration or shock. Her weight-for-age, length-for-age and weight-for-height were all between –1 and +1 SD. She had thickened feel of skin, pallor, and periorbital and pedal edema, without any rash or petechiae. There were no signs of meningeal irritation or any cranial nerve involvement. There was hypotonia, decreased power in all limbs, absent deep tendon reflexes, and flexor plantar response. Child exhibited excessive irritability on palpation of limbs suggestive of muscle tenderness and restriction of range of motion due to stiffness of skin and muscle. Liver was palpable 3 cm below the costal margin and spleen tip was palpable. Rest of the systemic examination was normal. Laboratory investigations revealed microcytic hypochromic anemia, thrombocytopenia, hypernatremia, hypokalemia, raised serum transaminases, raised creatine phosphokinase (CPK) (both total and CPK-MB fraction) and hypoalbuminemia (Table 1). Arterial blood gas (ABG) showed normal pH (7.44) and normal bicarbonate level (25.6 mmol/L). Stool for rotavirus antigen was positive by rapid immunochromatographic test (EpiTuub). CSF was acellular with protein levels of 12 mg/dl sugar of 104 g/dl and sterile culture. As there was no evidence of acidosis or deranged renal function, and mother was giving oral rehydration fluid in inappropriate dilution before being referred to authors' hospital, hypernatremia was considered to be iatrogenic. Acute rotavirus gastroenteritis with hypernatremia, myositis, transaminitis and encephalopathy was considered. Hypernatremia and hypokalemia were corrected and

✉ Dheeraj Shah  
shahdheeraj@hotmail.com

<sup>1</sup> Department of Pediatrics, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi 110095, India

**Table 1** Laboratory parameters of the index child at the time of admission

Laboratory parameters	Values
<b>Complete blood counts</b>	
Hemoglobin	6.9 g/dl
Total leukocyte counts	$5.6 \times 10^9/L$
Platelet count	$75 \times 10^9/L$
Packed cell volume	26.4%
<b>Biochemical parameters</b>	
Blood urea	55 mg/dl
Serum creatinine	0.3 mg/dl
Serum sodium	165 mEq/L
Serum potassium	2.5 mEq/L
Serum bilirubin	0.2 mg/dl
SGOT	232 IU/L
SGPT	528 IU/L
Serum albumin	2.2 g/dl
Total protein	4.1 g/dl
Serum calcium	8.9 mg/dl
Creatine phosphokinase total	>5000 IU/L
Creatine phosphokinase MB	968 IU/L
<b>Others</b>	
C-reactive protein	0.6 mg/dl
Serum Widal	Non reactive
Rapid malaria antigen test	Negative
Blood culture	Sterile
Urine for fungal hyphae	Negative
Urine culture	Sterile
Stool for bacteria and protozoa	Negative
Stool for rotavirus antigen	Positive

MB Muscle/brain; SGOT Serum glutamate oxaloacetic transaminase; SGPT Serum glutamate pyruvate transaminase

phenytoin was continued. Over next three days, fever and loose stools subsided but child was lethargic, and developed increasing stiffness of all four limbs. Power was reduced in all four limbs and reflexes were not elicitable. Thickened feeling of skin and muscles persisted and sensorium did not improve over next 7 d. Repeat serum CPK level was 3540 U/L, and CPK-MB was 367 U/L suggesting falling trend; blood and urine cultures were sterile. Contrast-enhanced computed tomography (CECT) of head was normal. Stool for rotavirus typing revealed G3 + G9P[8] strain of rotavirus. CSF for rotavirus could not be processed because of drying of sample. Child was managed symptomatically and showed improvement. At the time of discharge, child was conscious, oriented, and had normal tone with near-normal (4/5) power in all limbs. During follow-up after six weeks, child had normal tone and deep tendon reflexes, and flexor plantar response. CPK total was 268 U/L and CPK MB was

77 U/L; SGOT was 35 IU/L, SGPT was 18 IU/L, total protein was 6.7 g/dl and serum albumin was 4.2 g/dl.

## Discussion

Rotavirus gastroenteritis has been reported to cause extra-intestinal symptoms involving various systems; but multisystem involvement is rare. The index child presented with neurological symptoms following acute gastroenteritis, which suggested dyselectrolytemia or acute viral encephalopathy. Persistence of neurological symptoms even after gastroenteritis settled, and serum electrolytes normalized, made authors' suspect acute viral encephalopathy. Elevation of CPK was consistent with myositis. Finally a diagnosis of rotavirus acute gastroenteritis with encephalopathy with myositis with hepatic involvement (transaminitis with hypoalbuminemia) was made. A concomitant infection such as dengue causing multisystem involvement can cause similar presentation; however, the child had no rash or hemoconcentration, and she presented mainly with acute gastroenteritis during winter season (December) when there was no seasonal outbreak of Dengue in the city. However, as dengue or any other infection causing multisystem involvement was not ruled out by investigations, there remains a possibility of such co-occurrence.

Possible mechanisms of neurological manifestations in rotavirus infection are direct viral invasion, increased nitrites and nitrates in serum and CSF or, change in homeostasis of  $Ca^{++}$  or  $Cl^{-}$ , dysregulation of the genes related to immune or stress response [3] or overproduction of cytokines by activated T cells and macrophages [4]. In the present case G3 + G9P[8] serotype was isolated from the stool of patient. To date G1, G3, G4, G5, G9 VP7 antigen and P4, P6, P8 VP4 antigen viruses have been isolated from serum, CSF or stool of patient with rotavirus encephalopathy [5, 6].

Myositis is considered a consequence of direct virus invasion into muscle or an immunological reaction [7]. There are reports showing elevated AST and ALT levels in rotavirus gastroenteritis [8]. However there are no reports showing hypoalbuminaemia in patients with biochemical hepatitis, which was present in index case.

## Conclusions

The authors documented a rare presentation of rotavirus diarrhea associated with multisystem involvement in form of encephalopathy, myositis, transaminitis and hypoalbuminemia. Rotavirus should be considered in the differential diagnosis of a child with gastroenteritis presenting with encephalopathy or myositis.

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### Compliance with Ethical Standards

**Conflict of Interest** None.

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