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## LETTER TO EDITOR (VIEWERS CHOICE)

### UNUSUAL EXTRA-INTESTINAL MANIFESTATION OF THE ROTAVIRUS INFECTION

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A nine month-old male, previously healthy, and fully vaccinated except for rotavirus presented to the emergency department with history of fever, frequent vomiting, profuse non-bloody watery diarrhea and decreased activity for five days. On the last day he developed up-rolling of the eyes, and facial twitching for minutes. On examination he looked unwell, lethargic, and severely dehydrated. His growth parameters were normal. Vitals revealed a heart rate of 158 beats/min, respiratory rate of 50/min and blood pressure of 95/60mmHg. His capillary refill time was three seconds. He had weak spontaneous movements of the limbs with fair muscle tone. The deep tendon reflexes of the four limbs were not exaggerated. The initial laboratory work up showed white blood cell count (WBC) of 11,700/cumm with 62 percent being neutrophils, hemoglobin 12.4 g/dL, platelets 131000/cumm and C-reactive protein 5 mg/dL. Serum sodium was 172 mEq/L, potassium 3.6 mEq/L, chloride 146 mEq/L, bicarbonate 13.5 mEq/L, blood urea nitrogen (BUN) 8.6 mmol/L, serum creatinine 124 mol/L, alanine aminotransferase (ALT) 103 IU/L, aspartate aminotransferase (AST) 2142 IU/L, serum lactate 6.7 mmol/L and serum ammonia 54 mmol/L. His serum glucose, albumin and amylase were normal. The serology for cytomegalovirus (CMV), Epstein Barr virus (EBV), and other hepatitis viruses were negative. His stool was positive for rotavirus antigen (latex agglutination test), but was negative for adenovirus, and it was also negative for ova and parasite. His blood, urine and stool cultures revealed no bacterial growth. Urine was dark in color and was positive for blood (+++) and proteins (++) , its microscopy revealed no red blood cells (RBCs) but granulocytes. Brain computerized tomography was normal. The electroencephalogram showed mild degree of slow wave abnormalities over the left hemisphere, suggesting dysfunction involving this area. In addition, mild degree of diffuse nonspecific background slowing was present. Lumbar puncture was not done because of the instability of the patient's condition. Tandem mass spectroscopy and the urine gas chromatography were negative. On the second day of admission, he had muscle tenderness and stiffness and decrease in spontaneous movement. Serum creatine phosphokinase (CPK) was 41000 IU/L (normal range: 20-130 IU/L), lactate dehydrogenase was 754 IU/L. ALT peaked to 1,600 IU/L and AST to 3532 IU/L. The patient was treated with hydration and correction of electrolytes besides paracetamol and ibuprofen for analgesia. Over the following four weeks, the consciousness level was improving but signs of the upper motor neuron lesion

(spasticity and exaggerated deep tendon reflexes all over the limbs) were clearly evident. Brain MRI done after a month abnormal signal intensities were identified within the globus pallidus and the thalamus as well as in the anterior limb of the internal capsule bilaterally in asymmetrical fashion. This is an unusual presentation of rotavirus infection.

Among the first identified viral causes of gastroenteritis was rotavirus. It has been established that it represents the single most important viral cause of gastroenteritis among children worldwide. (1) The clinical picture may range from being asymptomatic to severe dehydration, seizures and even death. (2, 3) Although the primary site of infection is the small intestine, extra-intestinal sites like liver, kidney, central nervous system may be involved (4-8). The central nervous system manifestations may include seizures, encephalitis, cerebellitis, meningitis or encephalopathy with detection of rotavirus in their CSF. (5-7,9) Associated musculoskeletal complications like myositis and rhabdomyolysis has been reported. (10-12). According to a literature review done by Dickey et al in 2009, there were only 23 cases of central nervous system disease secondary to Rotavirus infection described in the literature. (9) Few cases of myositis and rhabdomyolysis due to rotavirus have been reported (10-12). In our case, rhabdomyolysis was evident by the clinical picture (weakness and tender passive movements), increase in the level of the serum CPK (much more than three-folds), and the myoglobinuria manifested by the positive blood and protein in the urine dipstick and the absence of red blood cells by microscopy. Other presentation seen in our patient was hepatitis and renal failure. Renal failure can be attributed to more than one factor mainly the dehydration, rhabdomyolysis and the direct rotavirus induced pathology on the renal tissue. Speculation about the rhabdomyolysis etiology in this case may include ischemia, direct viral invasion, hypercytokinemia e.g. tumor necrosis factor (TNF) alpha and increase in the free intracellular calcium which leads to the activation of the calcium-dependent proteases. (8,12) In the reported case, the serum calcium level was low.

Thus rotavirus should be considered in patients with diarrhea and extraintestinal manifestations such as renal failure, CNS manifestations and myositis.

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