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Severe Adult Rotavirus Gastroenteritis: A Rare Case with Multi-Organ Failure and Critical Management

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Patient: Male, 65-year-old
Final Diagnosis: Acute kidney injury • myocardial infarction • rotavirus infection
Symptoms: Diarrhea • vomiting
Clinical Procedure: Intravenous hydration • renal replacement therapy
Specialty: General and Internal Medicine

Objective: Rare coexistence of disease or pathology
Background: Infectious diarrheal illnesses such as rotavirus gastroenteritis are significant contributors to childhood morbidity and mortality, especially in low socio-demographic index regions. Major advances in addressing this issue include sanitation and clean water initiatives, as well as rotavirus immunization. In Australia, a robust vaccination program has significantly reduced childhood rotavirus infections, leading to decreased hospitalizations and mortality. However, cases of adult rotavirus still occur, and although these adult patients usually do not require interventional management, it is possible for them to present critically unwell and require resuscitation.
Case Report: A previously well 65-year-old man presented to the Emergency Department febrile and hypotensive with severe diarrhea attributed to rotavirus. Clinically, he presented with mixed hypovolemic and septic shock. Despite initial resuscitation, he had multiple severe acute end-organ complications, secondary to poor perfusion. He acquired an acute kidney injury, type-2 myocardial infarction, and ischemic hepatic injury. The mainstay of management was rapid fluid resuscitation, continuous renal replacement therapy, and monitoring in the Intensive Care Unit; however, it was crucial to empirically treat for other causes of shock.
Conclusions: To the best of our knowledge, there is a scarcity of reports documenting the management of severe rotavirus gastroenteritis in adults. We recommend advising elderly patients to avoid contact with individuals with diarrheal illnesses, especially rotavirus gastroenteritis. Clinicians should also promote awareness regarding the potential severity of a disease that is typically managed conservatively, and be aware that intervention can be required in severe gastroenteritis.

Keywords: Gastroenteritis • Hypovolemia • Rotavirus • Shock, Septic

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Background

Gastroenteritis is a commonplace disease that has global ramifications, with diarrheal illnesses being a significant contributor to annual childhood deaths [1]. Rotavirus was the leading cause of viral diarrheal death in children under the age of 5 years in 2015, with 146 500 deaths, with most of these deaths occurring in low socio-demographic index (SDI) regions [1]. Fortunately, with improved access to potable water and a robust vaccination schedule, hospitalizations and deaths from rotavirus gastroenteritis are decreasing in children [1-4]. In high, high-middle, middle, and low-middle SDI countries, rotavirus gastroenteritis proportionately causes more morbidity and mortality in the over 70-year-old population than in children [1,3,4]. The 2001-2002 Australian annual hospitalization rates for acute rotavirus gastroenteritis across all ages were 20.6 cases per 100 000 people [2]. Comparatively in 2008-2009, after the adoption of rotavirus vaccination into the Australian National Immunisation Program in 2007, hospitalization rates dropped to 6.2 per 100 000 people in the same category [2], and dropped further in 2016 to 1.8 per 100 000 people [3]. Interestingly, hospitalization rates in adults over 65 years increased slightly but significantly in the years immediately after the adoption of rotavirus vaccine in children [2,5,6]. This may have been attributable to increased recognition of rotavirus gastroenteritis in adults, as well as more frequent investigation of infectious diarrhea in adults [6]. While it is typically referred to as a self-limiting illness of children, rotavirus is known to affect adults who uncommonly have severe symptomatology [7] and rarely causes death [3]. Adults with rotavirus gastroenteritis are more likely to acquire the infection from a child that they care for or interact with than from any other source [8].

There is a dearth in the literature describing the extreme complications of infectious diarrhea from rotavirus in adults, and thus in this case we will describe an adult patient whose rotavirus gastroenteritis had devastating effects, leading to acute multi-organ injury. In this case, and in future cases, we would emphasize the importance of maintaining adequate end-organ perfusion to prevent or minimize ischemic injury. It is also imperative to be mindful of how rotavirus gastroenteritis, a disease that is considered self-limiting in relatively higher SDI countries, can result in severe complications. We also recommend advising elderly patients to avoid contact with younger populations with infectious diarrheal illness to minimize the risk of transmission.

Case Report

A previously well 65-year-old man presented to a tertiary hospital Emergency Department with diarrhea and was found to

be hypotensive, with a blood pressure of 70/40 mmHg, and febrile, with a temperature of 38.4 °C. The patient described having 2 days of diarrhea, with over 10 loose bowel motions over that period, and had vomited 5 times prior to presentation. He denied any pain and reported having had brown and green stools with no blood or mucous. His granddaughter, a young child attending kindergarten, had also had a similar diarrheal illness and the two had been in contact with each other 1 to 2 days prior to the patient's onset of symptoms. The patient had no significant past medical history, nor did he take any medication. Given the patient's age, he did not qualify for any previous rotavirus vaccination, and so was unvaccinated.

On examination, the patient had a distended, non-tender abdomen, which was resonant to percussion, and bowel sounds were audible. His chest was clear and he had equal air entry to both lungs on auscultation. Peripheral limb pulses were not palpable and he had cool peripheries.

The serum hemoglobin level was 160 g/L (reference range: 125-175), white cell count $18.1 \times 10^9/L$ (4.0-11.0) and platelet count $128 \times 10^9/L$ (150-450). He had hypokalemia, with a potassium level of 2.7 mmol/L (3.5-5.2). The patient had raised inflammatory markers, with a C-reactive protein level of 144 mg/L (04), procalcitonin 235 µg/L (0.00-0.07), and ferritin 2708 µg/L (30410). The patient had a creatinine level of 286 µmol/L (45-90), which was raised from a baseline of 83 µmol/L, and a raised lactate level of 6.9 mmol/L (0.5-2.0).

Blood and urine cultures were sterile, and respiratory swabs were negative for a complete panel of viral pathogens, including severe acute respiratory syndrome coronavirus 2. A sample of unformed feces detected rotavirus on multiplex tandem polymerase chain reaction assay. No typing or titer for rotavirus was able to be ascertained. The assay was negative for other viruses such as norovirus, sapovirus, astrovirus, and adenovirus. No ova, cysts, and parasites were identified, and no bacterial pathogens such as *Clostridium difficile* were detected on microscopy. Cerebrospinal fluid testing was negative for tuberculosis and cryptococcal antigen, and cultures were negative.

The initial high sensitivity troponin level was 834 ng/L (0-20), with a serial repeat test recording 1062 ng/L, consistent with a type-2 acute myocardial infarction. Electrocardiogram showed new anteroinferior T-wave inversion, and echocardiogram showed no ventricular or valvular dysfunction. The patient's liver function tests were raised, with an alanine transaminase of 1124 U/L (5-40) and aspartate transaminase of 1101 U/L (5-35), which was consistent with ischemic liver injury. A hepatitis screen was negative, as was serology testing for hepatitis A, B, and C, and HIV 1 and 2. The patient had a urinary albumin/creatinine ratio of 112.7 mg/mmol (<2.5), raising concerns for a glomerulonephritis; however, a glomerulonephritis

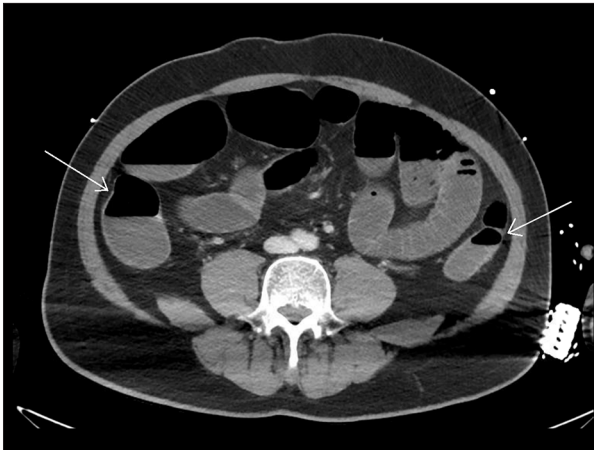


Figure 1. Abdominal computed tomography angiography showing small and large bowel dilatation, with fluid and gas levels throughout (large bowel indicated with arrows).

screen was negative and consistent with an acute tubular necrosis, secondary to the poor perfusion state that occurred when the patient was shocked. On immune screening, the patient had an atypical antineutrophil cytoplasmic antibodies (ANCA) pattern that resembled neither a perinuclear nor cytoplasmic ANCA pattern. Furthermore, the patient had negative myeloperoxidase and proteinase-3 immunofluorescence.

A range of radiographic images were done in the acute resuscitation phase. A computed tomography (CT) angiogram of the abdomen and pelvis revealed moderate dilatation of almost all of the small bowel, as well as entire dilatation of the large bowel; however, there was no identified transition point and no clinical evidence of bowel obstruction (**Figure 1**). CT angiogram was performed to rule out aortic aneurysm and dissection, given the patient clinically presented with shock.

The patient was resuscitated with intravenous (i.v.) crystalloid rehydration, with poor response. He was initiated on an adrenaline infusion of 10 µg/min, requiring several bolus doses to increase the blood pressure to a range safe enough for transfer for urgent CT imaging. Later, the adrenaline infusion was switched to noradrenaline (typically the first line vasopressor for septic shock). A potassium infusion was initiated at a rate of 20 mmol/h. For undifferentiated sepsis empirical antibiotic cover, both i.v. vancomycin 1.5 g and i.v. piperacillin-tazobactam 4.5 g were administered. Because the patient had poor oxygen saturations even when on high-flow nasal prongs, a decision was made to intubate the patient using a rapid sequence intubation technique and transfer to the Intensive Care Unit (ICU). Continuous renal replacement therapy (CRRT) was commenced in the ICU for 5 days, and subsequently the patient was transferred to the ward for ongoing care. The patient had 3 sessions of intermittent inpatient hemodialysis

due to prolonged acute kidney injury, which was also eventually ceased. Renal function gradually improved, with creatinine levels lowering to 201 µmol/L after a month in hospital. Liver function tests also improved, with alanine transaminase and aspartate transaminase decreasing to 91 U/L and 82 U/L, respectively. The patient did not require any urgent cardiac catheterization and was started on amlodipine 10 mg daily and aspirin 100 mg daily to manage the type-2 myocardial infarction.

During the prolonged hospital stay of 5 weeks, the patient developed a nosocomial urinary tract infection and pneumonia. Urine culture grew *Escherichia coli*, producing extended spectrum beta lactamase. Both infections were successfully treated with i.v. meropenem 1 g daily.

The patient later required transfer to a subacute facility for inpatient rehabilitation and, after 14 days, he was discharged home with ongoing outpatient follow-up with the nephrology team.

Discussion

This patient had rotavirus gastroenteritis severe enough to cause hypovolemic and septic shock. Typically, dehydration after significant fluid losses is the cause of death in children with gastroenteritis globally [7], which parallels this patient's complex pathophysiology. In most cases, gastroenteritis can be managed conservatively, or if severe dehydration is clinically suspected, i.v. fluids can improve a patient's condition or prevent deterioration [8]. There is no indication to commence curative anti-viral treatment for viral gastroenteritis, given the complications associated with severe gastroenteritis are as a result of inadequate hydration leading to poor peripheral perfusion.

CT imaging of the abdomen and pelvis revealed that almost the entire bowel wall was inflamed, and bowel loops were distended with copious amounts of fluid and gas, suggestive of significant losses (**Figure 1**). With output greatly outweighing input, the patient's hypovolemic state eventuated in decreased end-organ perfusion, indicated by an elevated serum lactate level. Notably, the patient satisfied systemic inflammatory response syndrome, with his fever, hypotension, and raised white cell count. This mixed hypovolemic and septic shock resulted in extensive ischemic damage, reflected through the type-2 myocardial infarction, acute kidney injury, and ischemic liver injury.

To the best of our knowledge, despite being a common infection, there are only a few case studies describing ischemic injury secondary to viral gastroenteritis and no specific reports on adults with rotavirus presenting with type-2 myocardial infarction and ischemic hepatitis, as our patient did. This could be explained by elderly patients commonly either having full

recovery due to improved health care systems [3] or dying from rotavirus gastroenteritis, with cases of significant morbidity where the patient recovers being rare [5]. Landa et al described a case of sapovirus gastroenteritis causing septic shock in an adult patient [9]. Similar to our case, the patient was diagnosed with a typically benign pathogen causing profound illness and raised inflammatory markers and required ICU admission. This patient had some end-organ damage also, with a mild transaminitis. Unfortunately, our patient had a more severe ischemic liver injury, as well as the other ischemic complications of the heart and kidneys, requiring continuous renal replacement therapy and hemodialysis. There are, however, plenty of case reports of children with severe rotavirus leading to complications or even death [10-13]; however, none of these cases describe adults with the same extremity of disease.

Rotavirus immunization aims to mimic the natural immunity that an individual gains when they are infected by the ubiquitous wild-type rotavirus [14]. Neither the rotavirus pathogen nor the vaccine provides life-long immunity; however, in the case of children, they both decrease the severity of subsequent rotavirus infection as well as the chances of hospitalization and death [14,15]. The rotavirus immunization brand used in Australia is Rotarix, and has a maximum age limit for administration of 24 weeks of age [15,16]. There are no trials or reports suggesting any benefits from administration of rotavirus immunization in older infants, children, and adults. Given his age, the patient never qualified for rotavirus vaccination and thus was unimmunized [16], and it was unfortunately impossible to assess whether he had previously been

infected by rotavirus. In addition to his age, his unvaccinated status meant that he was at higher risk for developing severe rotavirus gastroenteritis than an individual with partial protection or immunization [5,7]. Another pertinent risk factor for the patient to acquire rotavirus was his recent exposure to children [8].

Conclusions

Despite rotavirus gastroenteritis classically being treated as a self-limiting disease in higher SDI settings, it can progress to a fulminant and life-threatening state in both children and adults, particularly in low SDI countries. Clinicians need to be aware of the potential severity of rotavirus gastroenteritis and recommend avoiding individuals with infectious diarrhea to minimize transmission, especially in those individuals who remain unvaccinated for rotavirus and are at increased risk of severe disease.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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