Saudi Journal of Medicine (SJM)

Scholars Middle East Publishers Dubai, United Arab Emirates Website: www.saudijournals.com ISSN 2518-3389 (Print) ISSN 2518-3397 (Online)

Clinical Profile of Acute Viral Gastroenteritis in Adults: 1-Year Cross-Sectional Study

Dr. Prakash Babaliche MD^{1*}, Dr. Pallavi Goswami MBBS²

¹Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum, Karnataka, India

*Corresponding author Dr. Prakash Babaliche

Article History

Received: 15.03.2018 Accepted: 26.03.2018 Published: 30.03.2018

DOI:

10.36348/sjm.2018.v03i03.010



Abstract: Viral gastroenteritis is one of the most frequently occurring medical illnesses all over the world, especially in developing countries. Although, clinical features of viral gastroenteritis are generally limited to the intestine, they may also extend beyond the gastrointestinal tract, which could be life threatening and fatal. The etiology of viral gastroenteritis in adults usually remains unclear. The aim was to investigate the etiology and clinical profile among adult patients with acute viral gastroenteritis. Patients (100) underwent investigations including, hemoglobin test, urine examination, fasting blood sugar, liver function test, renal function test, and serum electrolytes. Stool specimens were tested for rotavirus and adenovirus using the rapid kit test. The categorical and continuous data were compared using chi-square test and student t-test, respectively. At 95% confidence interval, $p \le 0.05$ was considered as statistically significant. Of the 100 gastroenteritis patients, 26 patients were diagnosed positive for viral gastroenteritis. The leading cause of viral gastroenteritis was rotavirus (22) followed by adenovirus (4) and rotavirus-adenovirus co-infection (2). Diarrhea (100) was the major clinical feature. Incidence of gastroenteritis was mainly observed in patients (41) below 30 years of age. Acute viral gastroenteritis was significantly associated with the clinical features including vomiting, fever, dehydration, and reduced urine output; and biochemical parameters including renal and hepatic function abnormalities (p < 0.001). The study concluded that rotavirus, followed by adenovirus, can cause severe gastroenteritis and is an important etiologic agent in hospitalized adult cases of gastroenteritis.

Keywords: Acute viral gastroenteritis, adenovirus, adults, rotavirus, systemic infestation.

INTRODUCTION

Globally, diarrheal illnesses of viral etiology are reported to be the most common type of diarrheal illness [1]. Viruses such as rotavirus, adenovirus, and astrovirus are the most common causative agents of acute viral gastroenteritis. Although the new calicivirus is responsible for most of the outbreaks in industrialized nations, overall, rotavirus continues to be the leading source of gastroenteritis [1].

In developing countries, 440,000 deaths per year are reported due to rotavirus-induced viral gastroenteritis, especially in children. Rotavirus infection occurs in adults in cases of endemic disease, epidemics, travel-related disease, and child-to-adult transmission of infection [2]. Whereas, epidemics, endemics, and sporadic infections are more commonly observed with adenovirus infections [3]. The incidence of rotavirus induced gastrointestinal disease in adults is 2%-5% in western countries including the Netherlands, Michigan, Thailand, Sweden, Switzerland, and the United Kingdom [2]. Studies conducted in Japan, Australia, Indonesia and Mexico reported an increased

incidence of 11%-63% in adult patients with viral gastroenteritis [2].

Etiology of gastroenteritis includes various bacteria, parasites, and viral pathogens. The wide diversity of bacterial and viral infections that may cause diarrhea, complicates the accurate surveillance and diagnosis, especially in developing countries. In the developed countries, although gastroenteritis cases have decreased significantly due to improved sanitation, it has minimal effect on gastroenteritis of viral etiology [4].

The clinical presentation of rotavirus gastroenteritis is believed to be severe when compared to other types of viral gastroenteritis. The clinical course starts with a sudden onset of mild fever, vomiting and loose stools. On an average, vomiting is observed for 2-3 days and diarrhea lasts for 4-5 days. In contrast to rotaviruses, adenoviruses present with less frequent hyperthermia and dehydration. But the duration of infection is longer along with fever and vomiting. The clinical spectrum of rotavirus disease varies from asymptomatic infection to acute, severe,

²Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum, Karnataka, India

dehydrating diarrhea, including vomiting, which can be fatal [5]. Severe gastroenteritis leading to hospitalization and death is more frequently observed in adults and elderly patients [6].

Rapid antigen testing of the stool, either by EIA or latex agglutination tests, is used to aid in the diagnosis of rotavirus infection [7]. The rapid antigen detection tests have ensured rapid diagnosis with high sensitivity and specificity (> 98% sensitivity and specificity) in several infectious diseases, including viral gastroenteritis [7]. The antirotavirus antibodies (IgM and IgA) excreted in the stool after the first day of illness can remain positive for 10 days after primary infection and longer after reinfection; therefore, they are more accurate in the diagnosis of gastroenteritis of viral etiology [8].

Although gastroenteritis in adults implicates a major burden on the patients and the health care system, only a minority of the researchers have explored and investigated this study criterion so far [9]. Therefore, the present study was an attempt to investigate the etiology and clinical profile among adult patients with acute viral gastroenteritis.

MATERIALS AND METHODS Study Design

The study was a cross-sectional randomized study conducted for 1-year from January 2011 to December 2011. A total of 100 patients aged more than 18 years were included in the study. Patients with a history of passing more than three episodes of stools per day with a decrease in stool consistency and increase in stool urgency and/or vomiting and/or abdominal discomfort, and diarrhea for less than 2 weeks were included in the study. Patients with noninfectious causes of acute diarrhea, dysentery, and those proven positive for bacterial and parasitic infection by microscopy, stool hanging drop test, and stool culture were exempted from the study. Before the commencement of the study, ethical clearance was obtained from the Institutional Ethical Committee.

Data collection and clinical tests

After explaining the purpose of the study, written consent was obtained from the patients. The data were recorded in a predesigned and pretested proforma. Patients were subjected to routine investigations including hemoglobin test, chest X-ray, urine examination, serum creatinine, fasting blood sugar and complete biochemical profile including liver function tests (serum bilirubin-total and direct, serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), renal function test (blood urea and serum creatinine levels), and serum electrolytes test (serum sodium and potassium levels). The stool samples were collected in a sample collection tube. The diagnosis of viral etiology (rotavirus and adenovirus) was determined by SD Bioline Rota/Adeno rapid test

device (Standard Diagnostics, Inc., Republic of Korea). Based on the etiology, patients were divided into two groups: viral gastroenteritis positive and viral gastroenteritis negative.

Data Analysis

The data were coded and entered into a Microsoft Excel Worksheet. Student t-test, Chi-square test, and Fischer's exact test were used to compare the continuous and categorical data. At 95% confidence interval, $p \leq 0.05$ was considered as statistically significant.

RESULTS

Of the 100 acute gastroenteritis patients, viral gastroenteritis was observed in 26 patients. The leading cause of viral gastroenteritis was rotavirus (22) followed by adenovirus (4) and rotavirus-adenovirus coinfection (2). Among the clinical features, diarrhea was observed in all of the patients (100) as defined by the inclusion criteria, followed by vomiting (51), dehydration (37), fever (34), and reduced urine output (32). The severity of dehydration was determined by assessing the skin elasticity (37), tongue appearance (37), and patient's appearance (37). Female predominance (51) was observed in the incidence of acute gastroenteritis, and higher number of patients (41) were observed below 30 years of age.

Majority of the patients had liver (89) and renal function (73) abnormalities followed by electrolyte imbalance in few patients (12). Analysis of liver function tests revealed abnormal levels of total bilirubin (18), direct bilirubin (71), SGOT (55) and SGPT (40) in patients with acute gastroenteritis. Also, the levels of blood urea (66) and serum creatinine (50) were abnormal in the patients with renal function abnormalities. Among electrolytes, abnormal levels of serum sodium (8) and potassium (4) were observed in the patients.

Association of acute viral gastroenteritis with clinical features

In the present study, of the 100 acute gastroenteritis cases, vomiting was observed in 51 cases, wherein, 22 patients were of viral etiology and 29 were of non-viral etiology. A highly significant association was observed between vomiting and acute viral gastroenteritis (p < 0.001). The clinical presentation of fever was observed in 34 patients, in which 17 patients each were found in acute viral gastroenteritis and nonviral gastroenteritis groups. Fever was significantly associated with acute viral gastroenteritis (p < 0.001). Reduced urine output was observed in most of the acute viral gastroenteritis cases (21) as compared to non-viral gastroenteritis cases (11), establishing a highly significant association between reduced urine output and acute viral gastroenteritis (p < 0.001). Similarly, dehydration was observed in many of the acute viral gastroenteritis cases (21) when

compared to non-viral gastroenteritis cases (16), with a highly significant association between the presence of dehydration and acute viral gastroenteritis (p < 0.001). A significant association was observed between acute viral gastroenteritis and renal (p = 0.009) and liver

function (p < 0.001) abnormalities as well. Abnormal renal (24) and liver function (24) was observed in most of the acute viral gastroenteritis cases when compared to the acute non-viral gastroenteritis cases with renal (49) and liver (7) abnormalities (Table-1).

Table-1: Association between acute viral gastroenteritis and clinical characteristics

Variable		Viral gastroenteritis		m volue
		Positive, n (%)	Negative, n (%)	<i>p</i> -value
Vomiting	Present	22 (43.14)	29 (56.86)	< 0.001*
	Absent	4 (8.16)	45 (91.84)	
Fever	< 37.5	9 (13.64)	57 (86.36)	< 0.001*
	> 37.5	17 (50)	17 (50)	
Urine output	Normal	5 (7.35)	63 (92.65)	< 0.001*
	Reduced	21 (65.63)	11 (34.38)	
Abdominal pain	Present	6 (42.86)	8 (57.14)	0.121
	Absent	20 (23.26)	66 (76.74)	
Dehydration	Present	21 (56.76)	16 (43.24)	< 0.001*
	Absent	5 (7.94)	92.06 (63)	
Renal function abnormality	Normal	2 (7.41)	25 (92.59)	0.009
	Abnormal	24 (32.88)	49 (67.12)	0.009
Liver function abnormality	Normal	2 (2.90)	67 (97.10)	< 0.001*
	Abnormal	24 (77.42)	7 (22.58)	\\ 0.001
Electrolyte imbalance	Normal	21 (23.86)	67 (76.14)	0.187
	Abnormal	5 (41.67)	7 (58.33)	

*statistically significant

In the renal function tests, there was significant increase in levels of blood urea (63.31 \pm 16.14 vs. 42.30 \pm 23.93 mg/dL; $p{<}0.001)$ and serum creatinine (1.54 \pm 0.45 vs. 1.16 \pm 0.42 mg/dL; $p{<}0.001)$ in patients with positive viral etiology as compared to patients with non-viral etiology of acute gastroenteritis (Table-2).

In this study, a significant increase was also observed in the levels of direct bilirubin (1.00 \pm 0.48 vs. 0.75 \pm 0.34 mg/dL; p = 0.005) and total bilirubin (0.63 \pm

0.38 vs. 0.44 ± 0.29 mg/dL; p = 0.014) in patients with viral gastroenteritis when compared to patients with non-viral gastroenteritis. A significant increase was observed in the liver transaminases including SGOT (80.38 \pm 50.38 vs. 35.78 \pm 33.57 mg/dL; p<0.001) and SGPT (90.96 \pm 48.63 vs. 50.41 \pm 49.62 mg/dL; p<0.001) in patients with positive viral etiology for gastroenteritis as compared to nonviral gastroenteritis (Table-2).

Table-2: Comparison of mean values of renal and liver profile in viral gastroenteritis

Variable		Viral gastroenteritis		m volue
		Positive (Mean ± SD)	Negative (Mean \pm SD)	<i>p</i> -value
Renal profile	Blood urea (mg/dL)	63.31 ± 16.14	42.30 ± 23.93	< 0.001*
	Serum creatinine (mg/dL)	1.54 ± 0.45	1.16 ± 0.42	< 0.001*
Liver profile	Serum bilirubin-Direct (mg/dL)	1.00 ± 0.48	0.75 ± 0.34	0.005^{*}
	Serum bilirubin-Total (mg/dL)	0.63 ± 0.38	0.44 ± 0.29	0.014^{*}
	SGOT (mg/dL)	80.38 ± 50.38	35.78 ± 33.57	< 0.001*
	SGPT (mg/dL)	90.96 ± 48.63	50.41 ± 49.62	< 0.001*

*statistically significant; SGOT, Serum aspartate aminotransferase; SGPT, Serum alanine aminotransferase

DISCUSSION

The present study aimed at evaluating the clinical profile of acute viral gastroenteritis in adult patients and to assess its systemic manifestations through the evaluation of the biochemical parameters. Rotavirus infection followed by adenovirus infection were the leading causes of acute viral gastroenteritis. Due to the high incidence, morbidity, and mortality, studies are usually carried out involving the pediatric

age-group, whereas the trials in adults are limited. In a study conducted by Huh *et al.*, in 10,028 samples of different age-groups, 29% were of viral etiology with 19.3% of rotavirus infection and 0.007% of adenovirus infection [10]. Similarly, Akan *et al.*, conducted a study in 672 patients reported 18.7% cases with rotavirus positivity, 8.9% cases with adenovirus positivity and 4.4% cases with rotavirus-adenovirus coinfection [4]. These findings are in accordance with the rotavirus and

adenovirus positivity results observed in the present study.

In the present study, patients were most commonly observed below 30 years of age, followed by 30–45 years of age. In a study conducted by Akan *et al.*, 63.3% patients were observed below 14 years of age followed by 35% patients observed among age-group of 15–64 years and 11% in the geriatric age-group [8]. A female predominance was observed in the present study, in contrast to the findings observed in the study conducted by Akan *et al.*, wherein a male predominance was observed (52.7%) [8].

In the present study, vomiting, dehydration, and reduced urine output were observed more frequently in viral gastroenteritis cases when compared to non-viral gastroenteritis cases. Also, in the study by Coffin et al. rotavirus induced gastroenteritis was more related to vomiting, fever, and diarrheavomiting coexistence [11]. The present study suggests a high association between vomiting and acute viral gastroenteritis. Present study findings are in accordance with the study conducted by Akan et al. in which a significant difference was observed in the incidence of vomiting between the rotavirus cases and non-rotavirus cases (p = 0.01) [4]. According to studies conducted by Kapikian et al., and Ward et al., fever was reported in 17% and 19% of the viral diarrhea cases, respectively [12, 13]. Also, in the current study a significant association was established between acute viral gastroenteritis and fever.

Gastroenteritis of viral etiology, especially that of rotavirus infection has a more severe clinical outcome [4, 14]. According to a study by Ackaboy et al. rotavirus induced viral gastroenteritis can result in an elevation in the hepatic transaminases suggestive of extra-intestinal manifestations of viral gastroenteritis [15].

Similarly, in a study conducted Antonopoulos et al, a considerable increase was observed in the levels of serum creatinine (1.3 ± 0.1) mg/dL vs. 1.0 ± 0.1 mg/dL, p = 0.17) and blood urea nitrogen (68.7 \pm 15.2 mg/dL vs. 38.7 \pm 4.4 mg/dL, p =0.06) in dehydrated patients as compared to controls [16]. Also, Celik et al. reported a slight increase in the levels of serum creatinine (0.55 \pm 0.2 mg/dL vs. 0.53 \pm 0.2 mg/dL, p = 0.609) and blood urea nitrogen (15.8 \pm 11.3 mg/dL vs. 11.0 \pm 3.2 mg/dL, p = 0.047) in acute rotavirus gastroenteritis patients than in the controls [17]. The current study results are in conjunction with these findings, which indicate a significant association between renal function abnormalities and acute viral gastroenteritis with abnormal renal function in patients with positive viral etiology when compared to patients with non-viral etiology.

Teitelbaum *et al.*, in their study observed that 20% patients with rotavirus gastroenteritis had increased levels of liver transaminases [18]. The present study results are similar to these results, wherein elevated liver enzymes were more observed in viral gastroenteritis cases when compared to non-viral gastroenteritis cases.

The present study revealed an overall prevalence of viral gastroenteritis as 26%, wherein rotavirus followed by adenovirus were the important causative organisms in hospitalised adult patients. Viral gastroenteritis usually extends beyond the intestine in to the blood and has the potential to be widely distributed and cause systemic manifestations, as observed in the present study. Therefore, it indicates that acute viral gastroenteritis causes significant morbidity in adults. The cross-sectional study design possesses as a limitation in the inference of the study findings to a large population.

CONCLUSION

The current study indicates that acute gastroenteritis tends to occur in a severe form in adults with occurrence outside the gastrointestinal system. Further, the findings of this study have to be confirmed involving large sample size. In addition, a follow-up is required to determine the incidence of rotavirus and adenovirus gastrointestinal infections in adults and to evaluate the potentially serious extraintestinal manifestations.

REFERENCES

- 1. Clark, B., & McKendrick, M. (2004). A review of viral gastroenteritis. *Current opinion in infectious diseases*, 17(5), 461-469.
- 2. Anderson, E. J., & Weber, S. G. (2004). Rotavirus infection in adults. *The Lancet infectious diseases*, 4(2), 91-99.
- 3. Parashar, U. D., Hummelman, E. G., Bresee, J. S., Miller, M. A., & Glass, R. I. (2003). Global illness and deaths caused by rotavirus disease in children. *Emerging infectious diseases*, 9(5), 565-572.
- Akan, H., İzbırak, G., Gürol, Y., Sarıkaya, S., Gündüz, T. S., Yılmaz, G., Hayran, O., & Vitrinel, A. (2009). Rotavirus and adenovirus frequency among patients with acute gastroenteritis and their relationship to clinical parameters: a retrospective study in Turkey. Asia Pacific family medicine, 8(1), 8
- Fischer, T. K., Ashley, D., Kerin, T., Reynolds-Hedmann, E., Gentsch, J., Widdowson, M. A., ... & Glass, R. I. (2005). Rotavirus antigenemia in patients with acute gastroenteritis. *The Journal of infectious diseases*, 192(5), 913-919.
- Gangarosa, R. E., Glass, R. I., Lew, J. F., & Boring, J. R. (1992). Hospitalizations involving gastroenteritis in the United States, 1985: the special burden of the disease among the

- elderly. American journal of epidemiology, 135(3), 281-290.
- 7. Kumar, M. L., Super, D. M., Lembo, R. M., Thomas, F. C., & Prokay, S. L. (1987). Diagnostic efficacy of two rapid tests for detection of respiratory syncytial virus antigen. *Journal of clinical microbiology*, 25(5), 873-875.
- Akan, H., İzbırak, G., Gürol, Y., Sarıkaya, S., Gündüz, T. S., Yılmaz, G., Hayran, O., & Vitrinel, A. (2009). Rotavirus and adenovirus frequency among patients with acute gastroenteritis and their relationship to clinical parameters: a retrospective study in Turkey. Asia Pacific family medicine, 8(1), 1.
- Svenungsson, B., Lagergren, Å., Ekwall, E., Evengård, B., Hedlund, K. O., Kärnell, A., Löfdahl, S., Svensson, L., & Weintraub, A. (2000). Enteropathogens in adult patients with diarrhea and healthy control subjects: a 1-year prospective study in a Swedish clinic for infectious diseases. Clinical infectious diseases, 30(5), 770-778.
- Huh, J. W., Kim, W. H., Moon, S. G., Lee, J. B., & Lim, Y. H. (2009). Viral etiology and incidence associated with acute gastroenteritis in a 5-year survey in Gyeonggi province, South Korea. *Journal* of Clinical Virology, 44(2), 152-156.
- 11. Coffin, S. E., Elser, J., Marchant, C., Sawyer, M., Pollara, B., Fayorsey, R., Nelson, L., Lawley, D., Goveia, M., Stek, J., & Hille, D. (2006). Impact of acute rotavirus gastroenteritis on pediatric outpatient practices in the United States. *The Pediatric infectious disease journal*, 25(7), 584-589.
- 12. Kapikian, A. Z., Wyatt, R. G., Levine, M. M., Yolken, R. H., VanKirk, D. H., Dolin, R., Greenberg, H. B., & Chanock, R. M. (1983). Oral administration of human rotavirus to volunteers: induction of illness and correlates of resistance. *Journal of infectious diseases*, 147(1), 95-106.
- Ward, R. L., Bernstein, D. I., Young, E. C., Sherwood, J. R., Knowlton, D. R., & Schiff, G. M. (1986). Human rotavirus studies in volunteers: determination of infectious dose and serological response to infection. *Journal of Infectious Diseases*, 154(5), 871-880.
- 14. Girard, M. P., Steele, D., Chaignat, C. L., & Kieny, M. P. (2006). A review of vaccine research and development: human enteric infections. *Vaccine*, 24(15), 2732-2750.
- Akcaboy, M., Oguz, M. M., Acoglu, E. A., Acar, M., Zorlu, P., Hosnut, F. O., & Senel, S. (2016). Systemic Manifestation of Rotavirus Infection in Children: A Report of Three Cases. *Iranian Red Crescent Medical Journal*, 18(8).
- Antonopoulos, C. N., Kalkanis, A., Georgakopoulos, G., Sergentanis, T. N., & Rigopoulos, D. N. (2011). Neutrophil gelatinaseassociated lipocalin in dehydrated patients: a preliminary report. *BMC research notes*, 4(1), 435.

- 17. Çelik, T., Altekin, E., İşgüder, R., Kenesari, Y., Duman, M., & Arslan, N. (2013). Evaluation of neutrophil gelatinase-associated lipocalin in pediatric patients with acute rotavirus gastroenteritis and dehydration. *Italian journal of pediatrics*, 39(1), 52.
- 18. Teitelbaum, J. E., & Daghistani, R. (2007). Rotavirus causes hepatic transaminase elevation. *Digestive diseases and sciences*, 52(12), 3396-3398.