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Research Article

Evaluation of Acute Enteritis Cases in Adults Presenting to the Emergency Department: A Single Tertiary Care Hospital Experience

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Introduction: Acute enteritis can occur due to numerous bacterial, viral, and parasitic infections. This clinical condition can have various spectra in vulnerable patient groups with immunodeficiency and inflammatory bowel disease. This study aimed to examine cases of acute enteritis in the emergency unit of a tertiary-care hospital.

Material and Methods: This cohort study was conducted with consecutive patients between September 2022 and November 2023. The clinical, laboratory, and microbiological data of the 194 patients were retrospectively examined. Patients with immunocompromised (IC) or inflammatory bowel disease (IBD) were analyzed in a healthy population.

Results: The mean age of patients was 47 ± 19 (19 - 91). One-third of the patients had IC, and in the stool analysis, leukocytes and blood were positive in one-third and one-twentieth of the patients, respectively. Only one-tenth of patients had positive stool culture results. IC patients presented with metabolic acidosis, increased creatinine levels, and acute-phase reactant levels. Despite the lower prevalence of leukocytes and blood in stool samples, culture positivity was higher in IC patients.

Conclusion: Stool culture positivity was low for the diagnosis of acute enteritis. Culture examination is important in IC patients, even in the absence of direct microscopic findings. Because patients with IC are more susceptible to complications, clinical assessment is important.

Keywords: Enteritis, Diarrhea, Culture, Immunocompromised

1. INTRODUCTION

Due to improvements in sanitation and medical management of diseases worldwide, diarrheal diseases are not as devastating and as feared as it was in the previous centuries.[1] However, they still remain an important cause of morbidity and mortality globally; in 2016 it was estimated to have caused more than 1.5 million deaths in the world, among which a third was children aged less than 5 years.[2] In terms of worldwide disability-adjusted life years (DALYs) lost, diarrhea was third in 2016, responsible for 74·4 million DALYs, more than half of which occurred among children younger than 5 years.2 In 2019 the number of diarrheal cases worldwide was estimated to be near seven billion.[3]

In developing nations, watery small intestinal or bloody diarrhea (dysentery) can be caused by diverse bacteria, viruses, or parasites. In such countries, diarrheal diseases occur all year round at a certain rate and are overlaid with spikes of increased cases due to epidemics.[4] Diarrhea may also occur during other viral and parasitic infections. Non-infectious etiologies of diarrhea, such as inflammatory bowel disease, drug side effects, endocrine diseases, and malabsorption, should also be considered in patients with repeated episodes of acute diarrhea.

Because diarrheal symptoms are mostly mild, self-limiting patients do not seek medical assistance. Even if they do, unless there is persistent fever, severe abdominal pain, bloody diarrhea, severe dehydration, immune suppression, inflammatory bowel disease, or significant cardiovascular disease, laboratory testing or imaging studies are not generally warranted.[4,5] This is because the yield of stool microscopy and cultures for the culprit pathogens is low (12.6%), even lower in

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diarrhea patients hospitalized for more than 72 hours (1.4%).[6] For most cases, the results reported are too late to be used in this self-limited disease. Therefore, this study aimed to examine the clinical characteristics of acute enteritis in the emergency unit of a tertiary care hospital.

2. MATERIAL AND METHODS

2.1. Methods of the study

This retrospective cohort study was conducted on 194 consecutive patients treated at a single tertiary care hospital between September 2022 and November 2023 with a diagnosis of acute enteritis. Demographic and clinical data of the patients were collected from the archives of paper patient files. Laboratory and microbiological data were collected by using an automated laboratory reporting system. The main etiologies of patients were classified as immunocompromised (IC), inflammatory bowel disease (IBD), or healthy. Patients with malignancy, immune deficiency, autoimmune disease, chronic renal and hepatic disease, or diseases requiring immunosuppressive drugs were considered IC. The presence of stool leukocytes and blood positive for stool culture was examined in the patient groups. Acute kidney injury, metabolic acidosis, liver enzyme elevation, and systemic inflammation markers were evaluated in the cohort.

The institutional and/or national research committee's ethical guidelines were followed for all methods used in this study, including people. The study adhered to the principles outlined in the 1964 Declaration of Helsinki and its subsequent amendments or comparable ethical standards. Ethical approval for this study was obtained from the local research committee of the Istanbul University, Istanbul Faculty of Medicine [Approval No:2023-306]. Informed consent was obtained by opt-out from all patients or members of their families. Patient records and information were anonymized and de-identified before analysis.

2.2. Statistical analysis

Patient data were analyzed using IBM SPSS for Windows version 28.0 0.0(190) (Armonk, New York, U.S.A.). Numerical data are presented as mean ± standard deviation and categorical data as frequency and percentage. Two-group comparisons of numerical data with normal distribution were carried out using the independent samples Student's t-test. If the numerical data had a non-normal distribution, the Mann-Whitney U test was used. Categorical data comparisons were performed using the $\chi 2$ test. If the expected frequencies in cells were lower than five, groups were joined where appropriate until the expected cell count exceeded five. For 2 × 2 contingency tables, Yates correction was performed. If the assumptions were violated for 2 × 2 tables, Fisher's exact test was used.

3. RESULTS

3.1. Patient characteristics

Patient demographic data are presented in Table 1. The patients' ages ranged from 19 to 90 years old. IC patients were older (52 ± 17 vs. 45 ± 19 years, p=0.018), and IBD patients were younger (49 ± 18 vs. 40 ± 17, p=0.006) than the remaining patients. In the acute enteritis patient cohort, there was a slight predominance of female patients (47% vs %53), which was not apparent in IBD patients (49% vs. 51%), but was slightly more pronounced in IC patients (44% vs. 56%), but the difference was not statistically significant. Half of the patients were previously healthy before their index disease; one-sixth of the patients had IBD and malignancy. Clinical and biochemical data at admission are presented in Table 1. One-third of the patients were IC, and leukocytes and blood were positive in one-third and one-twentieth of patients, respectively. Only one-tenth of patients tested positive for stool culture. Stool culture results and antibiotic sensitivities of the isolates are shown in Table 2. The majority of the isolates were Salmonella spp. or Campylobacter spp. The only noteworthy observation of antibiotic resistance patterns was that Campylobacter spp. isolates were resistant to both ciprofloxacin and tetracycline, but sensitive to macrolides.

Table 1.

Demographic and biochemical data of the enteritis patients (n=194)

Parameters	n(%) or Mean ±SD*(Minimum – Maximum)
Age (years)	47 ± 19 (19 - 91)
IC	52 ± 17 *(p=0.018)**
IBD	49 ± 18 *(p=0.006)**
Gender	
Male	92 (47%)
Female	102 (53%)
Accompanying diseases	
None	96 (50%)
Inflammatory Bowel Disease	35 (18%)
Malignancy	35 (18%)
Hematologic	10 (5%)
Gastrointestinal	14 (7%)
Other	11 (6%)
Immune deficiency states	3 (1.5%)
Renal diseases	16 (8%)
Hepatic diseases	3 (1.5%)
Other chronic diseases	6 (3%)
Clinical parameters	
Immunocompromised	66 (34.0%)
Blood positive in stool	11 (5.7%)
Leukocytes present in stool	57 (29.4%)
Stool culture positive	20 (10.3%)
Blood chemistry	
Creatinine (mg/dl)	1.4 ± 1.4 (0.5-12.1)
Lactate (mmol/l)	1.6 ± 1.1 (0.4-8.1)
Ionized Ca^{+2}(mmol/l)	1.2 ± 0.1 (0.8-1.5)
CRP (mg/dl)	52 ± 73 (1-395)
ALT (IU/l)	23 ± 20 (2-123)
AST (IU/l)	25 ± 23 (6-225)
Blood gases	
pH	7.37 ± 0.07 (7.02-7.53)
Na^+ (meq/l)	137 ± 5 (118-148)
K^+ (meq/l)	4.0 ± 0.6 (1.1-6.4)
Cl^- (meq/l)	107 ± 10 (88-122)
$HCO3^-$ (meq/l)	22.2 ± 3.6 (11.2-36.0)
Anion gap (meq)	9 ± 5 (0 -25)
Blood count	
Hemoglobin (gr/dl)	12.2 ± 2.2 (6.2-16.2)
WBC (10^3 cells/ml)	9.1 ± 4.5 (0.3-28.3)
Neutrophils (10^3 cells/ml)	6.8 ± 4.1 (0.1-22.1)
Lymphocytes (10^3 cells/ml)	1.5 ± 0.9 (0.1-6.7)
Platelets (10^3 count/ml)	265 ± 123 (18-717)

* Abbreviations: SD; Standard deviation, n (%) or Mean ±SD*(Minimum – Maximum), CRP: C-reactive protein, ALT: Alanine transaminase, AST: Aspartate transaminase, WBC: White blood cell, * Mann-Whitney u test

Table 2.

Stool culture and antibiotic sensitivity of isolates

Pathogen	Count n (%)	Antibiotic resistance n (%)				
		Ampicillin	Ciprofloxacin	Trimethoprim	Macrolides	Tetracycline
Aeromonas spp.	1(5%)	-	0 (0%)§	0 (0%)	-	-
Salmonella spp.+	8(40%)	0 (0%)	0 (0%)	0 (0%)	-	-
Campylobacter spp.*	8(40%)	-	8 (100%)	-	0 (0%)	8 (100%)
Shigella spp.†	1(5%)	0 (0%)	0 (0%)	1 (100%)	-	-
Vibrio spp.‡	2(10%)	2 (100%)€	0 (0%)	0 (0%)	-	-

+; *C type Salmonella 2, S.enteritidis 5, S.typhimurium 1,*; C.jejuni 7, other 1,†;Sh. sonnei 1,‡;V. vulnificus 1, V. parahemolyticus 1,* §; Levofloxacin sensitive, €; Ampicillin-clavulanate sensitive

3.2. Clinical and laboratory characteristics of acute enteritis with immunocompromised patients

The frequency of positive stool leukocyte and blood in stool was lower in IC patients (40% vs. 19%, p=0.005, OR: 0.36 (95% CI 0.17-0.75)) and (36% vs. 0%, p=0.017, OR: 0.64 (95% CI 0.57-0.71)) respectively. However, there was no significant difference between culture positivity in immunocompetent and IC patients (10% vs. 11%). On total blood count, IC patients had lower hemoglobin (12.5 ± 2.1 vs. 11.7 ± 2.3 g/dl, p=0.029) and platelet levels (298 ± 120 vs. 210 ± 109 *103/ml, p<0.001). On biochemical data, serum creatinine (1.1 ± 1.0 vs. 1.8 ± 1.9 mg/dl, p=0.012), CRP (42 ± 54 vs. 69 ± 95 mg/dl, p=0.05) and lactate (1.4 ± 0.7 vs. 1.9 ± 1.4 mmol/l, p=0.016) levels were higher and HCO3- levels (23.0 ± 3.3 vs 20.8 ± 3.6 mmol/l, p<0.001) lower in IC patients. (Table 3)

Table 3.

Clinical and laboratory characteristics of acute enteritis with immunocompromised patients

	Immunocompromised Patients	Immunocompotent Patients	P-*value*
Stool leukocyte (%)	19	40	**0.005**
Stool blood (%)	0	36	**0.017**
Culture positivity (%)	11	10	0.785
Hemoglobin (g/dL)	11.7 ± 2.3	12.5 ± 2.1	**0.029**
PLT (10^3/mL)	210 ± 109	298 ± 120	**<0.001**
Creatinine (mg/dL)	1.8 ± 1.9	1.1 ± 1.0	**0.012**
CRP (mg/dL)	69 ± 95	42 ± 54	**0.05**
Lactate (mmol/l)	1.9 ± 1.4	1.4 ± 0.7	**0.016**
HCO3 (mmol/l)	20.8 ± 3.6	23.0 ± 3.3	**<0.001**

Abbreviations: PLT: Platelet count, CRP: C-reactive protein, HCO3: Bicarbonate,
*Mann-Whitney u test, Student's t-test, $\chi 2$ test

3.3. Clinical and laboratory characteristics of acute enteritis with inflammatory bowel disease patients

The frequency of detection of blood and leukocytes in stool was significantly higher in IBD patients than in other members of the cohort (20% vs. 2.5 %, p<0.001, OR: 9.7 (95% CI 2.7-35.3) for blood and 63% vs. 22 %, p = 0.005, OR: 6.0 (95% CI 2.7-13.1) for leukocytes, respectively). There was no significant difference in stool culture positivity between patients with and without IBD (3% vs. 12%, p=0.134). On comparison of biochemistry parameters of patients with or without IBD, serum creatinine (0.8 ± 0.2 mg/dl vs. 1.6 ± 1.6 mg/dl, p<0.001), AST (18 ± 5 IU/l vs. 28 ± 26 IU/l, p=0.031) and ALT (16 ± 13 IU/l vs. 25 ± 22 IU/l, p=0.008) levels were lower, and serum bicarbonate levels (24.6 ± 2.0 mmol/l vs. 21.7 ± 3.6 mmol/l, p<0.001) were higher in IBD group. On blood count parameters white blood cell count (10.5 ± 4.5 *1000 cells/ml vs. 8.7 ± 4.4 *1000 cells/ml, p=0.021), lymphocyte count (1.9 ± 0.7 *1000 cells/ml vs. 1.3 ± 1.0 *1000 cells/ml, p=0.004) and platelet counts (364 ± 136 *1000 cells/ml vs. 236 ± 103 *1000 cells/ml, p<0.001) were higher in the IBD group. (Table 4)

Table 4.

Clinical and laboratory characteristics of acute enteritis with inflammatory bowel disease patients

	IBD Patients	Immunocompotent Patients	*P-value*
Stool leukocyte (%)	63	22	**0.005**
Stool blood (%)	20	2.5	**<0.001**
Culture positivity (%)	3	12	0.134
Creatinine (mg/dL)	0.8 ± 0.2	1.6 ± 1.6	**<0.001**
AST (IU/L)	18 ± 5	28 ± 26	**0.031**
ALT (IU/L)	16 ± 13	25 ± 22	**0.008**
HCO3 (mmol/L)	24.6 ± 2.0	21.7 ± 3.6	**<0.001**
WBC (10^3/mL)	10.5 ± 4.5	8.7 ± 4.4	**0.021**
LYMPH (10^3/mL)	1.9 ± 0.7	1.3 ± 1.0	**0.004**
PLT (10^3/mL)	364 ± 136	236 ± 103	**<0.001**

Abbreviations: IBD: Inflammatory bowel disease, AST: Aspartate transaminase, ALT: Alanin transaminase, HCO3: Bicarbonate, WBC: White blood cell, LYMPH: lymphocyte, PLT: Platelet count,
*Mann-Whitney u test, Student's t-test, $\chi 2$ test

3.4. Laboratory characteristics of patients with positive stool leukocytes

Stool culture positivity and blood in stool were higher in patients with positive stool leukocytes (6% vs. 21%, p=0.003, OR: 4.3 (95% CI 1.65-11.19)) and (3% vs. 12%, p=0.016, OR: 4.66 (95% CI 1.31-16.59)), respectively. In terms of biochemistry and blood counts, only a significant difference was observed in the platelet count, which was higher in the positive stool leukocyte group (248 ± 116 vs. 306 ± 131, p=0.009). (Table 5)

Table 5.

Laboratory characteristics of patients with positive stool leukocytes

	Positive Stool Leukocyte	*P-value*
Stool culture positivity (%)	21/16	**0.003**
Stool blood (%)	12/3	**0.016**
PLT (10^3/mL)	306 ± 131/248 ± 116	**0.009**

Abbreviations: PLT: Platelet count
*Mann-Whitney u test, Student's t-test, $\chi 2$ test

4. DISCUSSION

In this study, half of the patients in the cohort were previously healthy, one-sixth had IBD, and one-third were IC. Only one-tenth of patients had positive stool culture results. Stool culture positivity and etiologic agents were similar to a hospital-based retrospective study reported previously.[6] The most noteworthy finding was the stool culture yield; the presence of stool leukocytes or blood increased the positivity of stool cultures fourfold. However, positive leukocyte or blood in stool was significantly lower in IC patients, but no significant difference in culture positivity was observed compared to immunocompetent patients. Detection of blood and leukocytes in stool was significantly higher in IBD patients with lower stool culture positivity without reaching significance compared to patients without IBD.

It should also be noted that stool culture positivity was unchanged in IC (10% vs. 11%), although stool leukocyte positivity was low, reflecting the fact that the leukocyte count was low in these patients. In IBD patients, blood and leukocyte positivity in stool was high, reflecting IBD disease activity rather than dysentery, and stool culture yields were very low without statistical significance (3% vs. 12%, p=0.134). In contrast, cases of IBD in which stool blood and leukocyte positivity reflected IBD disease activity and the yields of stool cultures were low.

White blood cell, lymphocyte, and platelet counts were higher in the IBD group than in the non-IBD group. The findings in patients were as expected and reflected the characteristics of this disease, such as higher rates of leukocytes and blood in stool, which were caused by IBD disease activation rather than infection. Higher white blood cell count and platelet counts may also reflect increased inflammation in this group of patients.[7]

IC patients had lower hemoglobin and platelet levels and higher serum creatinine, CRP, and lactate levels. Significant findings in IC patients compared to immunocompetent patients include lower hemoglobin and platelet levels, reflecting the suppression of erythroid and megakaryocytic series. Innate and adaptive immunity play distinct roles in host defense. Owing to the presence of malignancy, chronic kidney disease, or immune deficiency disorders, IC patients are more likely to develop opportunistic infections that could cause acute enteritis.[8] Both immune deficiency and infection caused bone marrow suppression in this patient group. Higher CRP levels reflect greater circulatory compromise and inflammation in IC patients.

Acute enteritis is characterized by vomiting and nausea, with dehydration mostly developing in the mild-to-moderate range. Severe dehydration may result in acute kidney injury via pre-renal hypoperfusion. Because of impaired compensation for acute enteritis and excessive fluid loss with diarrhea, these patient groups presented with higher lactate and creatinine levels. In addition, metabolic acidosis was increased in patients with IC. In addition to contributing to kidney disease, bicarbonate loss in the gastrointestinal tract along with diarrhea also plays a role in the development of metabolic acidosis. Adequately restored fluid loss is essential for controlling the kidney injury and acid-base balance.[9]

This study has some limitations. The number of patients was relatively small, and only Turkish patients were included in the study. Despite this limitation, the patient cohort revealed significant findings, including an adequate number of patients in special groups.

In conclusion, gastroenteritis was found to be numerically less frequent in stool culture positivity. However, the caveat was in IC cases, in which the positivity of stool leukocytes and blood decreased without a change in stool culture positivity. Early diagnosis and management of enteritis in IC patients are important to prevent the development of systemic complications.

Article Information Form

The Declaration of Conflict of Interest/ Common Interest

No conflict of interest or common interest has been declared by authors.

The Declaration of Research and Publication Ethics

The research followed the ethical guidelines established by the Helsinki Declaration and its later revisions, or those of an equivalent kind. The study was approved by the local research committee at Istanbul University, Istanbul Faculty of Medicine. (Acceptance No. 2023-306.)

Copyright Statement

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