SMJ- Sohag Medical Journal, Vol. 29 No (3) 2025

Print ISSN1687-8353 Online ISSN2682-4159

Original Article

Pattern Of Intestinal Parasites Among Cirrhotic Patients In Sohag University Hospitals

Reem M. Makbol[1], Asmaa Naser Mohamed[1], Nada Nadi Abd Elaal[1], Hesham Ebrahim Osman[2]

1. **Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, Sohag University, Sohag, Egypt**
2. **Department of Medical Parasitology, Faculty of Medicine, Sohag University, Sohag, Egypt**

Abstract

Background: Cirrhosis-associated immune dysfunction syndrome (CAIDS) is the final stage of chronic hepatic disease in which patients become more liable to various infections.

Aim: We aimed to determine the prevalence and pattern of intestinal parasitic infections in cirrhotic patients and to identify the risk factors associated with these infections.

Patients and methods: A case-control study was conducted among 100 patients who attended our Tropical Medicine and Gastroenterology outpatient clinic at Sohag University Hospital, classified into 50 patients with liver cirrhosis and gastrointestinal tract (GIT) complaints (case group) and 50 patients non-cirrhotic (control group). Participants were subjected to history taking, clinical examination, and investigations.

Results: 50% of cirrhotic cases had parasitic infection in comparison to 42 % of controls. Infection with Entamoeba histolytica / Entamoeba dispar, Cryptosporidium, Isospora, Hymenolepis nana, and Blastocystis hominis was more common in cirrhotic cases (12%, 10%, 10%, 8%, 6%), respectively, than in controls (6%, 4%, 6%, 2%, 2%), but this was statistically insignificant. The age, gender, diabetes, and hypertension were statistically significant in cirrhotic patients than in controls. Spontaneous bacterial peritonitis was more in cirrhotic parasitically infected cases than in cirrhotic non-infected cases, $P < 0.04$.

Conclusions: Entero-parasitic infection was more common among cirrhotic patients. Old age, male sex, diabetes mellitus (DM), and spontaneous bacterial peritonitis (SBP) increase the risk of parasitic infection in cirrhotic patients. Entamoeba histolytica / Entamoeba dispar, Cryptosporidium, Isospora, Hymenolepis nana, and Blastocystis hominis were more prevalent in cirrhotic parasitically infected cases than in controls.

Keywords: Cirrhosis, Enteroparasites, Peritonitis, Tropical, Parasitology

Abbreviations: CAIDS: Cirrhosis-associated immune dysfunction syndrome. **GIT:** Gastrointestinal tract, **CBC**: Complete blood count, **RBS**: Random blood sugar, **DM:** Diabetes millets, **SBP:** Spontaneous bacterial peritonitis, **STH**: Soil transmitted helminths, **IPS:** Intestinal parasitic infections, **PCR:** Polymerase Chain Reaction, **WASH:** Water Sanitation and Hygiene, **BMI:** Body mass index, **RBC:** Red blood cells, **WBC:** Wight blood cells.

Keywords: Cirrhosis, Enteroparasites, Peritonitis, Tropical, Parasitology
DOI : 10.21608/SMJ.2025.404739.1592 **Received:** June 17 , 2025 **Accepted:** August 03 , 2025
Published: September 01, 2025
Corresponding Author: Hesham Ebrahim Osman **E-mail** drhishame199@gmail.com
Citation: Hesham Ebrahim Osman. et al., Pattern Of Intestinal Parasites Among Cirrhotic Patients In Sohag University Hospitals SMJ,2025 Vol. 29 No (3) 2025 08 - 17

Introduction:

Liver cirrhosis is common in low, middle, and high-income countries; it is linked to excessive mortality and morbidity. [1] Liver cirrhosis is a consequence of long-standing hepatic inflammation, which is followed by diffuse hepatocellular fibrosis, in which regenerating hepatic nodules replace the normal hepatic architecture and ultimately result in hepatic failure. [2]

It is considered an immunocompromised condition that makes infections more likely and causes cirrhosis-associated immune dysfunction syndrome (CAID). [3]

Intestinal parasites are groups of worms that mainly affect the gastrointestinal tract. They contain flatworms (tapeworms and flukes) and roundworms (Ascariasis, Pinworm, and Hookworm infections). [4]

On the other hand, intestinal protozoa like Giardia lamblia (G. lamblia), Entameba histolytica (E histolytica), and Cryptosporidium species have a prevalence rate less than soil-transmitted helminths (STH). [5]

Intestinal parasitic infections (IPIs) remain a serious health problem in many parts of the world, mainly in tropical and subtropical areas. In underdeveloped nations, intestinal protozoan infections caused by G. lamblia, Blastocystis hominis (B. hominis), Cryptosporidium spp., and Entamoeba spp. are significant causes of diarrhea. IPIs are mostly self-limiting in immunocompetent individuals, but they may lead to serious side effects like chronic diarrhea and malabsorption in immunocompromised patients. [6] Their mode of transmission includes drinking contaminated water, as in the case of Giardiasis, or through skin penetration, as in the case of Strongyloides stercoralis (S. stercoralis), and faeco-oral routes, as intestinal protozoa. [[4] Many factors increase the risk of IPIs as overcrowding, poor hygiene, and ignorance of healthcare knowledge. [7] Additionally, in IPI-susceptible populations, solitary pit latrines, which collect human waste by digging a pit a few meters down into the ground, are typically shared by multiple households because most homes lack functional toilets. [8]

Intestinal parasitic infection in hepatic patients may present with severe diarrhea, electrolyte imbalance, and dehydration. [9]

They may be complicated with malnutrition, intestinal obstruction, growth retardation, and immunodeficiency. [10]

Conventional methods for diagnosing intestinal parasites include separating the parasite from the stool using faecal flotation or faecal sedimentation techniques. The kind and load of the specific parasite are then identified using microscopic techniques like Kato-Katz or McMaster. Other traditional techniques include the Harada Mori and Baermann methods, which involve cultivating parasites in faeces and looking for larvae under a microscope. However, direct smears have the lowest sensitivity since the parasites may be obscured by stool, but they don't need any initial isolation for microscopic investigation . [11]

Polymerase Chain Reaction test (PCR), a molecular technique, can typically identify the greatest variety of parasites with a comparatively high sensitivity. However, its applicability may be limited by the requisite technology and expense . [12]

There are different methods for protection against intestinal parasites. Water, Sanitation, and Hygiene (WASH) aims to enhance the quantity and quality of water while also facilitating sanitation and hygiene practices in low-income nations. [13] Fresh vegetables should be cleaned well before eating, as it is very important for preventing intestinal parasites. [14]

Albendazole is a highly efficient anthelmintic medication against A. lumbricoides and hookworms [15], while mebendazole or albendazole are more effective against Trichuriasis [13]. Some intestinal parasitic diseases, such as giardiasis, respond to antibiotics like metronidazole and tinidazole rather than anthelminthic agents . [16]

Patients and Methods

This case-control study was conducted on 100 patients who attended the Tropical Medicine and Gastroenterology Outpatient Clinic at Sohag University Hospital from April 2023 to April 2024. They were classified into 50 patients with liver cirrhosis and GIT complaints (cases) and 50 patients with GIT complaints but without any comorbid condition (controls). Patients with liver cirrhosis were included, but cirrhotic patients with renal failure or cardiac problems were excluded.

Patients were subjected to:

1- Complete history taking (Age, sex, body mass index (BMI), comorbid conditions). BMI =Weight (kg)/[Height (m)]2.

2- Clinical examination for vital signs, general examination, abdominal examination, and signs of hepatic encephalopathy.
3-Blood sample was collected from each patient under aseptic conditions for CBC, liver function, and kidney function.
4- Abdominal ultrasonography after an overnight fast with the patient in a supine position by a convex-type transducer of an ultrasonography device with a 3.5-5-MHz frequency (Mindray DP-2200, China) to assess liver size, surface, echo-pattern, focal lesion, and portal vein diameter. Liver size was measured, as span of right lobe in mid-clavicular line on oblique view and classified into: shrunken (<11cm), average (11-15cm), or enlarged (> 15cm) & spleen examined with comment on size, echo-pattern, focal lesion, and splenic veins diameter, and its length greater than 13cm was enlarged. [17]
5- Scanning of gall bladder, biliary channels, kidneys, and ascites was done under aseptic conditions in a sterile container under aseptic precautions according to the standard protocol for further analysis. Each sample was examined physically for aspect, color, and sediment, chemically for glucose and protein, and microscopically by a haem- hemocytometer for RBCs, WBCs, and differential count. Diagnosis of SBP was determined with a threshold neutrophil count of 250cells /mm^3 with or without positive bacterial culture. [18]
6- Stool examination: Three successive fresh morning stool samples collected in labeled, clean carton boxes and examined macroscopically for color, odor, special character, and consistency (watery, fatty, soft, mucoid, or bloody), and worms or gravid segments. Also, samples were examined microscopically for parasites: a Direct smear using normal saline and Lugol's iodine, b-Flotation concentration for eggs, larvae, or cysts by Modified Ziehl Neelsen stain.

Ethical consideration: The study protocol was approved by the Ethical Committee of Research (Registration No.: Soh-Med-23-04 -13MS), and written informed consent was obtained from each participant.

Statistical analysis: Data were analyzed by using the Statistical Package for the Social Sciences version 12.1 (SPSS 12.1) for Windows. Quantitative data were presented as M±SD or median and interquartile range (IQR). Student t-test compared the means of two groups. Qualitative data were presented as numbers and % and compared by Chi-square (X^2) test or one-way ANOVA. P-value was considered significant if less than 0.05.

Results

This case-control study was conducted on 50 cirrhotic cases and 50 non-cirrhotic controls attending the Tropical Medicine and Gastroenterology outpatient clinic at Sohag University Hospital. The mean age of cirrhotic cases was 58±8.98, 66% of them were males. While the mean age of non-cirrhotic cases was 34±9.46, 36% of them were males.
The demographic characteristic of the studied participant showed that age, gender, history of diabetes, and history of hypertension were higher statistically significant in cirrhotic patient than non-cirrhotic, P value (<0.001, 0.003, 0.02, 0.01) respectively, but BMI was lower statistically significant in the cirrhotic than the non-cirrhotic patients, P value (<0.001).
Laboratory examination showed cirrhotic cases with lower significant HB level and platelet count than non-cirrhotic cases (P <0.001 & <0.001, respectively), but serum creatinine was higher in cirrhotic cases than in controls (P < 0.001).
Abdominal U/S showed shrunken liver in 50% of cirrhotic patients compared to 0% of controls, and irregular liver surface in 100% of cirrhotic patients compared to none in controls (P < 0.001, & < 0.001 respectively). Cirrhotic patients showed a significant increase in splenic size compared to controls (P< 0.001).
Cirrhotic cases were classified into parasitically infected and parasite-free. Diarrhoea and dysentery were significantly more common in cirrhotic infected patients than non-infected cirrhotic ones (P 0.04, 0.02, respectively).
Ultrasonography showed that ascites was more in parasitically infected cirrhotic cases than in non-infected cirrhotic ones (P <0.005). Ascitic fluid among cirrhotic patients showed high significantly neutrophilic count in parasitically infected cirrhotic patients than in non-infected cases (P=0.006). Spontaneous bacterial peritonitis was significant in parasitically infected cirrhotic cases than in cirrhotic non-infected ones (P 0.04). No significant relation was found between cirrhotic parasitically infected and non-infected ones as to CBC, renal, and liver functions.

High parasite rates were in cirrhotic cases, 25 (50%), than in non-cirrhotic 21 (42 %), without a significant difference (P 0.42). These were Entamoeba histolytica/disbar, Cryptosporidium, Isospora, Hymenolepis nana and Blastocystis *hominis* more in cirrhotic parasitic infected cases 6(12%), 5(10%), 5(10%), 4(8%), & 3(6%), than in controls 3(6%), 2(4%), 3(6%), 1(2%) & 1(2%) respectively, without significant. *G. lamblia* was more in controls than in cases 10(20%), & 8(16%), respectively, but without significant (P=0.6).

In univariate logistic regression, detected ascites, high protein, and increased neutrophil count in ascitic fluid as the main risk factors predicting intestinal parasites in cirrhotic patients, but these risk factors were not detected in multivariate analysis.

Details were given in tables (1, 2, 3, 4, 5, 6, 7, 8, 9 & 10).

Table 1: Demographic characteristics of participants:

Variable	Cirrhotic cases (N=50)		Non-cirrhotic cases (N=50)		P-value
Age (years): M ± SD Median (IQR)	58 ± 8.98 59 (54-65)		34 ± 9.46 33 (30.5-36.75)		<0.001
	Number	Percentage	Number	Percentage	
Male Female	33 17	66% 34%	18 32	36% 64%	0.003
DM	13	26%	4	8%	0.02
Hypertension	12	24%	3	6%	0.01
BMI: Underweight Normal Over weight Obese	0 7 24 19	0 14% 48% 38%	1 17 4 28	2% 34% 8% 56%	<0.001

DM: Diabetes mellitus, **BMI:** Body Mass Index

Table 2: CBC and renal function among participants:

Variable	Cirrhotic cases (N=50)		Non-cirrhotic cases (N=50)		P-value
	Number	Percentage	Number	Percentage	
Hb; Normal : Low	19 31	38% 62%	44 6	88% 12%	< 0.001
WBCs; Normal : Leukocytosis	34 16	68% 32%	40 10	80% 20%	0.17
Eosinophil: Normal : Eosinophilia	48 2	96% 4%	48 2	96% 4%	1 1
Platelets: Normal : Thrombocytopenia	25 25	50% 50%	50 0	100% 0%	< 0.001
Creatinine (mg/dl): M± SD Median (IQR)	1.57±0.78 1.8 (0.9- 2.05)		1.04 ±0.33 1.05 (0.7-1.27)		< 0.001

WBC: White blood cells

Table 3: Abdominal U/S findings among participants:

Variable	Cirrhotic cases (N=50)		Non-cirrhotic cases (N=50)		P value
Liver size: Average : Shrunken : Enlarged	17 25 8	34% 50% 16%	44 0 6	82% 0% 12%	< 0.001
Liver surface: Smooth : Irregular	0 50	0% 100%	50 0	100% 0%	< 0.001
Spleen size: Normal : Mildly enlarged : Moderate : Huge	1 7 31 11	2% 14% 62% 22%	48 1 1 0	96% 2% 2% 0%	< 0.001

Table 4: Clinical manifestations among parasitically infected and non-infected cirrhotic cases:

Variable	Cirrhosis with parasites (N=25)		Cirrhosis without parasites (N=25)		P value
	Number	Percentage	Number	Percentage	
Abdominal pain	18	72%	14	56%	0.24
Nausea & vomiting	12	48%	9	36%	0.39
Diarrhoea	15	60%	8	32%	0.04
Dysentery	5	20%	0	0	0.02
Worms in stool	0	0	0	0	----
Anus & vulva itching	1	4%	0	0	0.31
Weight loss	0	0	0	0	----
Abdominal tenderness	1	4%	5	20%	0.08

Table 5: Ascites and ascetic fluid analysis among parasitically infected and non-infected cirrhosis t

variable	Infected cirrhotic cases (N=25)		Non-infected cirrhotic cases (N=25)		P value
	Number	Percentage	Number	Percentage	
Ascites: Absent : Present	3 22	12% 88%	12 13	48% 52%	0.005
Ascetic fluid Protein: Mean ± SD Median (IQR)	2.65±1.15 2.8 (2.5-3.5)		1.74±1.77 2 (0- 3.5)		0.15
Cells: Normal : High neutrophil : Increased lymphocytes	8 15 2	32% 60% 8%	19 6 0	76% 24% 0	0.006
Spontaneous bacterial peritonitis (SBP): Yes : No	13 12	52% 48	6 19	24% 76%	0.04

Table 6: CBC, liver, and renal function among parasitically infected (n=25) and non-infected cirrhotic patients (n=25):

Variable	Parasitically infected cirrhotic cases		Parasite-free Cirrhotic cases		P-value
	Number	Percentage	Number	Percentage	
Hb: Normal : Low	7 18	28% 72%	12 13	48% 52%	0.15
WBCs: Normal : Leukocytosis	14 11	54% 44%	20 5	80% 20%	0.07
Eosinophil: Normal : Eosinophilia	24 1	96% 4%	24 1	96% 4%	1
Platelets: Normal : Thrombocytopenia	11 14	44% 56%	14 11	56% 44%	0.4
Total bilirubin: Normal : Increased	17 8	68% 32%	14 11	56% 44%	0.38
Albumin: Normal : Hypoalbuminemia	4 21	16% 84%	6 19	24% 76%	0.48
PTP: Normal : Impaired	2 21	16% 84%	2 23	8% 92	0.38
Creatinine (mg/dl): M± SD Median (IQR)	1.26 ± 0.7 2.8 (0.8-1.75)		1.11 ± 0.64 0.9 (0.6-1.45)		0.39

WBC: White blood cells, PT: Prothrombin time

Table 7: Prevalence of parasites among participants:

Variable	Cirrhotic cases (N=50)		Non-cirrhotic cases (N=50)		P-value
Parasitic infection	Number	Percentage	Number	Percentage	
Positive	25	50%	21	42%	0.42
Negative	25	50%	29	58%	

Table 8: Stool analysis of participants:

Variable	Cirrhotic cases (N=50)		Non-cirrhotic cases (N=50)		P-value
Reaction: Alkaline	26	52%	31	62%	
: Acidic	24	48%	19	38%	0.3
Entamoeba histolytica/dispar	6	12%	3	6%	0.29
Giardia lamblia	8	16%	10	20%	0.6
Blastocystis hominis	3	6%	1	2%	0.32
Cryptosporidium	5	10%	2	4%	0.24
Cyclospora	2	4%	2	4%	1
Isospora	5	10%	3	6%	0.46
Enterobius vermicularis	0	0%	1	2%	0.32
Hymenolepis nana	4	8%	1	2%	0.17

Table 9: Univariate logistic regression to predict risk factors of intestinal parasites in cirrhotic patients:

Independent factors	OR	Univariate 95% CI		P-value
		Lower	Upper	
Age	0.99	0.95	1.04	0.84
gender	2.48	0.74	8.35	0.14
Diabetic	2.95	0.76	11.34	0.12
Hypertension:	1	0.27	3.66	1
Over weight	0.75	0.14	4.09	0.74
Obese	0.67	0.12	3.87	0.65
Anaemia	2.37	0.73	7.67	0.15
Leucocytosis	3.14	0.89	11.06	0.08
Eosinophilia	1	0.05	16.92	1
Thrombocytopenia	1.62	0.53	4.94	0.39
Creatinine	1.43	0.6	3.39	0.46
Bilirubin	0.59	0.18	1.89	0.38
Hypoalbuminemia	1.65	0.4	6.78	0.48
PT	0.45	0.07	2.75	0.39
Liver size: Shrunken	0.5	0.02	11.08	0.66
: Enlargement	1.23	0.07	21.24	0.88
Spleen size: Mild enlargement	0	0	0	0
: Moderate enlargement	0.15	0.02	1.23	0.07
: Huge splenomegaly	0.35	0.08	1.57	0.17
Ascites	6.76	1.6	28.54	0.009
Protein levels in ascitic fluid	1.5	1.01	2.22	0.04
WBCs in ascitic fluid: High neutrophil count	5.93	1.69	20.85	0.005
: Increased lymphocytes	3.83	0	0	0.99
Reaction	0.4	0.13	1.26	0.12
SBP	0.29	0.08	0.97	0.045

OR: Odds ratio (it measures the association between the exposure to the factor and the outcome)
OR=1 means the exposure does not affect the outcome
OR>1 means the exposure is associated with an increase in the probability of disease occurrence
OR<1 means the exposure is associated with a decrease in the probability of disease occurrence
BMI: body mass index, WBC: white blood cells, SBP: Spontaneous bacterial peritonitis, PT: prothrombin time

Table 10: Multivariate logistic regression to predict risk factors of parasites in cirrhotic patients:

Independent factors	OR	Multivariate 95% CI		P-value
		Lower	Upper	
Ascites	0.2	0.01	4.35	0.34
Proteins in ascetic fluid	0.87	0.38	1.98	0.75
WBCs in ascitic Fluid: High neutrophil : Increased lymphocytes	0 1.14	0 0	0 0	1 0.99
SBP	7.6	0	0	0.99

WBC: white blood cells, SBP: Spontaneous bacterial peritonitis,

Discussion

During this case-control study, the prevalence and pattern of intestinal parasitic infection in cirrhotic patients, and the most common risk factors of intestinal parasitic infection in cirrhotic patients were evaluated.

This work was conducted on 100 patients, 50 patients with liver cirrhosis and GIT complaints (case group), and 50 patients with GIT symptoms but without any comorbidities. In the present study, the demographic characteristics of the studied participants showed that age, gender, history of diabetes, and history of hypertension were statistically significant higher in cirrhotic patients than in non-cirrhotic patients, but BMI was significantly lower in cirrhotic patients than in non-cirrhotic patients. This result was in agreement with Ibrahim et al. [19] who conducted a study on 90 patients complaining of liver cirrhosis with gastrointestinal symptoms (mainly abdominal pain and diarrhea) and 45 patients complaining of gastrointestinal symptoms without liver cirrhosis (control group), they reported that diabetes mellitus and hypertension were statistically significantly higher among the cases group than control group. However, the age, sex did not show significant differences. Also, Quintana et al. [20] reported that diabetic patients had a significantly higher frequency of cirrhosis.

As regards complete blood count (CBC), this research found that cirrhotic cases show statistically significantly lower Hb level and platelet count than non-cirrhotic cases, but cirrhotic cases show statistically significantly higher creatinine level than non-cirrhotic cases. These results agreed with Bentley et al. [21] who found that anemia is a common complication of cirrhosis, with an incidence of up to 75%, Manrai et al. [22] who detected that the prevalence of anemia may reach 66%-75% in cirrhotic patients, and Singh et al. [23]who revealed that low hemoglobin levels was associated with increasing severity of hepatic disease.

Also, this work agrees with Lim and Cuker[24] who reported that low platelet count is the most frequent hematological complication seen in hepatic disease. The results in this work agreed with Klavan and Fortune. [25] who reported that increased serum creatinine is a frequent laboratory finding for patients with cirrhosis. This may be due to the use of diuretics as a medication in the treatment of liver cirrhosis.

Regarding abdominal U/S findings among the studied participants, we found that cirrhotic cases show a significant increase in the size of the spleen in comparison to non-cirrhotic cases. This was agreed with the study of Li et al. [26], who showed that splenomegaly is the most common abnormality associated with liver cirrhosis. This is mostly due to portal hypertension.

When cirrhotic cases were classified into parasitically infected and non-infected cases, it was found that diarrhea and dysentery were the most significant clinical manifestations in cirrhotic infected patients compared to non-infected cases. Our results were in contrast with Ibrahim et al. [19]. who found that the most statistically significant clinical symptom in the cirrhotic cases was abdominal pain. Also, clinical manifestations like nausea, anorexia, and diarrhea have higher levels among the cirrhotic patients than the control group, without significant differences. Our results may be supported by that; the parasitic infections are the most infectious agents leading to diarrhea that persists for more than 2 weeks . [27]

Regarding ultrasonographic findings among cirrhotic cases, the presence of ascites was statistically significant in parasitically infected cirrhotic cases compared to non-infected cirrhotic cases. This was in contrast to Vardar et al. [28], who

found that there were no significant differences between parasitically infected and non-infected cirrhotic patients regarding the presence of ascites.

This study showed that spontaneous bacterial peritonitis was statistically significant in cirrhotic infected cases (52%) than in non-infected cases (24%). This agrees with Cornick et al. (2015), who reported that intestinal parasites lead to breakdown in the mucosal barriers, allowing bacteria to translocate into the peritoneal cavity. They also reported that the resultant neutrophilic ascites can complicate the clinical picture, as it represents the immune system's reaction to bacteria rather than the parasite.

This study revealed that there was a statistically insignificant difference between cirrhotic and non-cirrhotic patients regarding parasitic distribution, whereas half of the cirrhotic cases had parasitic infection (50%) in comparison to 42% of non-cirrhotic cases. This was in agreement with Ibrahim et al. [19], who found that the prevalence of parasitic infection was significantly higher in the cirrhotic cases than in non-cirrhotic cases. This may be due to the immunocompromised state of cirrhotic cases. Our results were in contrast to Vardar et al. [28], who conducted a study on 154 patients with Child-Pugh C grade liver cirrhosis and detected that the prevalence of intestinal parasites was 13.6% in patients with cirrhosis. This prevalence was significantly less than control group.

In this study, the infection with Entamoeba histolytica / Entamoeba disbar, Cryptosporidium, Isospora, Hymenolepis nana and Blastocystis hominis was more in cirrhotic parasitic infected cases (6 (12%), 5 (10%), 5 (10%), 4 (8%), 3(6%)), respectively, than in control which was (3 (6%), 2 (4%), 3 (6%), 1 (2%), 1 (2%)) respectively, but this was statistically insignificant. This agreed with Vardar et al. (2011), who found that there was no statistically significant relationship between cirrhotic and non-cirrhotic patients regarding parasitic distribution. They found that *B. hominis* was the most frequent parasite among their studied groups. This difference may be due to environmental and demographic changes.

Our results disagreed with Al-Ghandour et al. [29] who reported that *Cryptosporidium* and *Giardia* was more in cases (28 %, 15 %) respectively than control (14 %, 12 %) and agreed with them in *Blastocystis hominis* as they reported a little percentage of this parasite in cases and control, (3%, 1%), respectively.

Also, this work disagreed with Ibrahim et al. [19], who found that the most common parasites in both cases and control groups were *B. hominis* (26.7%), and *Cryptosporidium spp* was found in 21.1% of the controls. But this study also agreed with them in the higher percentage of *E. histolytica* in the cases than in the controls (5.6% vs 2.2%, respectively).

In univariate logistic regression, this study showed that the presence of ascites, high protein, and increased neutrophil count in ascitic fluid were the main risk factors that predicted intestinal parasitic infection in cirrhotic patients, but these risk factors were not detected in multivariate analysis. This agreed with Halliez and Buret. [30] , who reported that high ascitic fluid protein levels among parasitically infected patients were often associated with vascular permeability and inflammatory changes caused by parasitic invasion. Parasitic infection causes granulomatous inflammation or cyst formation, damaged vascular integrity [30], causing plasma proteins to exude into the peritoneal cavity. [31]

Conclusion

Diarrhea and dysentery were significantly more common in cirrhotic infected patients than in non-infected cirrhotic ones. The spontaneous bacterial peritonitis was higher in cirrhotic infected cases than in non-infected cirrhotic ones. There was no significant difference between cirrhotic cases and non-cirrhotic cases according to stool analysis. Entamoeba histolytica/dispar, Cryptosporidium, Isospora, Hymenolepis nana, and Blastocystis hominis were more prevalent in cirrhotic parasitically infected cases than in controls.

References

1- Edelson, P.K., et al., Maternal anemia is associated with adverse maternal and neonatal outcomes in Mbarara, Uganda. J Matern Fetal Neonatal Med, 2023. 36(1): p. 2190834.

2- **Sepanlou, SG, Safiri, S, Bisignano, C, *et al.*** The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol. Hepatol,.**2020:** 5(3):245-266.

3- **Pellicoro, A., Ramachandran, P., Iredale, J.P., *et al.*** Liver fibrosis and repair: Immune regulation of wound healing in a solid organ. Nature Rev. Immunol, **2014,** 14 (3):181-94.

4- **Rodríguez-Negrete, EV, Gálvez-Martínez M, Sánchez-Reyes K, *et al.*** Liver cirrhosis: The Immunocompromised state. J. Clin. Med, **2024,**.13(18):1-17.

5- **Feleke B E, Beyene M B, Feleke T E, et al.** Intestinal parasitic infection among household contacts of primary cases: a comparative cross-sectional study. *PLoS ONE*, **2019,** 14(10):1–11.

6- **Kirk M D, Pires S M, Black R E, et al.** World Health Organization Estimates of the Global and Regional Disease Burden of 22 Foodborne Bacterial, Protozoal, and Viral Diseases. 2010: A Data Synthesis. PLoS Medicine, **2015,** 12(12):1–21.

7- **Rasti, S, Hassanzadeh, M, Hooshyar, H, *et al.*** Intestinal parasitic infections in different groups of immunocompromised patients in Kashan and Qom cities, central Iran. Scand. J. Gastroenterol. **2017,** 52(6-7):738–41.

8- **Pazmiño, FA, Mora-Salamanca, AF, Mahecha, BSP, *et al.*** Prevalence of intestinal parasitism in preschool and school children in Colombia: Systematic review and meta-analysis- is. Trop. Med. Inter. Health, **2022,** 27, (9):781-94.

9- **Ngui, R, Aziz, S, Chua, KH, *et al.*** Patterns and risk factors of soil-transmitted helminthiasis among orang asli subgroups in peninsular Malaysia. Am. J. Trop. Med. Hyg, **2015,** 93(2): 361-70.

10- **Tuli, L, Singh, DK, Gulati, A K, *et al.*** A multiattribute utility evaluation of different methods for the detection of enteric protozoa causing diarrhea in AIDS patients. Biomed. (BMC) Microbiol. J, **2010,** 10:11-66.

11- **Chifunda K and Kelly P.** Parasitic infections of the gut in children, *Paediatrics and International Child Health*, **2019,** 39(1): 65–72.

12- **Requena-Méndez A, Chiodini P, Bisoffi Z, *et al.*** The Laboratory Diagnosis and Follow Up of Strongyloidiasis: A Systematic Review. PLoS Neglect. Trop. Dis, **2013,** 7(1):1-10.

13- **Mationg M.L.S., Gordon C A, Tallo V L, *et al.*** Status of soil-transmitted helminth infections in schoolchildren in Laguna Province, the Philippines: Determined by parasitological and molecular diagnostic techniques. *PLoS Neglected Tropical Diseases;* **2017,** 11(11): 1–16.

14- **Yates, T., Allen, J, Leandre, J.M., *et al.*** Wash Interventions in Disease Outbreak Response. Humanitarian Evidence Programme, Oxford, **2017**

15- **Anuar T S, Salleh F M, Moktar N, *et al*. Soil**-Transmitted helminth infections and associated risk factors in three orang asli tribes in peninsular Malaysia. Scientific Reports, **2014, (**4):1–7.

16- **Moser W, Labhardt N D, Cheleboi M, *et al.*** Unexpected low soil-transmitted helminth prevalence in the Butha-Buthe district in Lesotho, results from a cross-sectional survey. *Parasites and Vectors*; **2017,** 10(1):1–5.

17- **Benchimol M, Gadelha A P, Souza W D, *et al.*** Ultrastructural Alterations of the Human Pathogen Giardia intestinalis after Drug Treatment. *Pathogens*; **2023,** 12(6): 1-25.

18- **Pozo, AL, Godfrey, EM, Bowles, KM, *et al.*** Splenomegaly: Investigation, diagnosis, & ma-management. Blood Rev, **2009,** 23(3):105-11.

19- **Angeli P, Bernardi M, Villanueva C, *et al.*** EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. *J Hepatology*; **2018,** 69(2):406–60.

20- **Ibrahim S.S., Imam N, Ismail M.A., *et al.*** Prevalence of opportunistic parasites among liver cirrhosis patients in Beni Suef. *Egypt Acad J Biol Sci E Med Entomol Parasitology,* **2022,** 14:133-42.

21- **Quintana, JO, García-Compean, D, González, JA, *et al.*** The impact of diabetes mellitus on the mortality of patients with compensated liver cirrhosis prospective study. Ann. Hepatol, **2011,** 10(1):56-62.

22- **Bentley B.H., Ventura F, Lippert W.C.** Hemolytic anemia in cirrhosis: A case of spur-cell anemia. *Ann Intern Med Clin Cases;* **2024,** 3:1-4.

23- **Manrai M, Dawra S, Kapoor R, *et al*.** Anemia in cirrhosis: An underestimated entity. *World J Clin Cases;* 2022, 10(3):777-89.

24- **Singh S, Manrai M, Kumar D, *et al.*** Association of liver cirrhosis severity with anemia: Does it matter? Ann. Gastroenterol. *2020,* 33(3):272-76.

25- **Lim H.I., Cuker A.** Thrombocytopenia and liver disease: Pathophysiology and periprocedural management. *Hematology;* 2022, 2022(1):296-302.

26- **Klavan H.L, Fortune B.E.** Elevated creatinine in a patient with cirrhosis. *Clin Liver Dis (Hoboken);* 2016, 7(3): 48-52.

27- **LI L, Duan M, Chen W, *et al*.** The spleen in liver cirrhosis: revisiting an old enemy with novel targets. *J Transl Med;* 2017, 15(1):1-10.

28- **Khurana S, Gur R, Gupta N.** Chronic diarrhea and parasitic infections: Diagnostic challenges. *Indian Journal of Medical Microbiology; 2021,* 39(4):413-416.

29- **Vardar, R., Ersöz, G., Dağci, H, *et al.*** The prevalence of intestinal parasites is lower than expected in patients with decompensated cirrhosis. Turk. J. Gastroenterol, 2011, 18(1): 9-13.

30- Al-Ghandour *et al*. Intestinal Parasitic Infections among Egyptian Patients with Chronic Liver Diseases at Zagazig University Hospital *Afro-Egypt J Infect Endem Dis*; 2021, 11(1):27-38**.**

31- **Halliez M.C., Buret A.G.** Gastrointestinal parasites and the neural control of gut functions. *Front Cell Neurosci;* 2015, 9:1-20.

32- **Ramachandran NP.** Non-microbial etiology: Foreign body reaction maintaining post-treatment apical periodontitis. Endodontic Top, 2003, 6(1): 114-134.