Epidemiological study of Entamoeba histolytica in cancer patients

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Abstract

Amebiasis caused by Entamoeba histolytica is a third leading causes of death in worldwide. Little is known about its occurrence of parasite in subjects with cancer patients after receiving chemotherapy. This study aimed to determine, the frequency of E. histolytica in cancer patient admitted to oncology department in Kirkuk Teaching Hospital . Fresh stool specimens collected from 93 patients, detected by the E. histolytica / E. dispar olyclonal EILISA tests. Solid tumor form were the predominant type (65/93;69.8%)while the hematological cancer form were 30.1%(28/93, according to type of stool the high rate was demonstrate in semi –solid stool while according to color of stool was demonstrate in yellow colored stool. Cancer patients with E. histolytica / E. dispar were mostly from urban area then from rural area . E. histolytica / E. dispar positive cases occurred in patients whose their family were ≥6 individuals. And The high E. histolytica /E. dispar were demonstrated positive cases in illiterate individual

Keywords: E. histolytica , E. dispar ,Solid form tumor , Hematology form tumo

Residency & Educational state.

Introduction

Amebiasis is responsible for approximately 100,000 deaths per year, mainly in Central and South America, Africa, and India, as well as for considerable morbidity manifested as invasive intestinal or extra-intestinal clinical features. Amebiasis infections are endemic in most temperate and tropical climates in the developing world. In some tropical countries, antibody prevalence rates (reflecting past or recent infection) exceed 50 %. The prevalence of Amebiasis varies with the population of individuals affected, differing between countries and between areas with different socioeconomic conditions[1]. Sometimes up to 50 % of the population is affected in regions with poor sanitary conditions. It is thought that Amebiasis directly affects over 50 million people, causing loss of manpower and subsequent economic damage[2]. Transmission depends heavily on contaminated food and water, filth flies and cockroaches also are important mechanical vectors of cysts, their sticky, bristly appendages easily can carry cysts from a fresh stool to the dinner table, and the house fly habits of vomiting and defecating while feeding is an important mean of transmission. Carriers (cyst passers) handling food can infect the rest of their family or even other peoples if they work in restaurants, the uses of human feces as fertilizer in Asia, Europe and South America contribute to transmission. Reservoir host other than humans including dogs, pigs, and monkeys may play a role in the transmission of the disease[3].

Exposure to E. histolytica cysts can occur via Contaminated water and

food, spread by direct contact with an infected person's hands or with contaminated surfaces. Consuming food grown in feces-contaminated soil, fertilizer, or water. E. histolytica cysts can survive for weeks under moist conditions. Swimming pools are a possible source of contamination because E. histolytica survives chlorine levels sufficient to kill bacteria(3,4)

In up to 90% of E. histolytica infections, the symptoms are absent or very mild. These patients have normal rectosigmoidoscopic findings, without a history of blood in stool samples. Cysts and trophozoites lacking ingested RBCs may be visible on microscopy. Interestingly, most individuals infected with E. histolytica, but not E. dispar, develop serum antibody responses to the parasite even in the absence of invasive disease . So far, E. dispar has never been recognized as a cause of colitis or amebic liver abscess, although infection with these amebae is much more common than with E. histolytica, especially in developed countries(3). At the present, the diagnosis of intestinal amebiasis in many countries relies commonly on microscopic examination of stool samples for the presence or absence of E. histolytica/E. dispar. Unfortunately, it is not clear what percentage of patients infected with E. histolytica are asymptomatic . It was thought that asymptomatic infection by E. histolytica is common; signs and symptoms of invasive amebiasis develop in approximately 10% of the infected population . Estimation of the true prevalence of amebiasis is not easy, because many studies were done with just one microscopic examination of a stool sample . Asymptomatic E. dispar infections do not show evidence of disease or a serum anti-amebic antibody response, while symptomatic E. histolytica intestinal infection does show a systemic immune response (5).

As with other enteric organisms, antigen-specific secretory immunoglobulin A (IgA) antibodies have been found to mediate protection against intestinal infection by Entamoeba species(6,7). Colonic mucins are rich in galactose-containing carbohydrates and have been demonstrated to be the high-affinity receptor for the E. histolytica galactose-inhibitable surface lectin. In general, intestinal IgA antibodies prevent microbial binding to epithelial surfaces and promote clearance of pathogenic enteric organisms by agglutination and blocking of surface receptors essential for microbial pathogenesis and invasion[8].

In addition, the duration of the human intestinal anti-lectin IgA antibody response has been reported to be as short as a mean of 17 days to as long as 36 months in 53% of amebic liver abscess (ALA) subjects. Numerous groups are working on a galactose-inhibitable lectin-based amebiasis subunit vaccine designed to induce a protective mucosal or cellular immune response[9]. Without further characterization of the human intestinal immune response and a better understanding of the dynamics (intensity and duration over time) of this response, it will be impossible to rationally develop and design a lectin-based amebiasis subunit vaccine for study in animal models and humans[10].

Materials and Methods

A total of 93 stool samples were collected from patients complaining from malignant cancer with abdominal disorder (abdominal pain and diarrhea) were used in the present study. The samples were collected from patients with cancer during their attendance to Oncology Department at AL-Azadi Teaching Hospital from

period February 2013 to February 2014. Patients age were ranged from 1-10 to 81-90 years.

2.1 Sample collection:-

2.1.1.Stool collection:-

A fresh stool sample was collected from each patient using disposable plastic container .Then examined using double wet preparation as see with light microscopy using. Small amount (0.5 ml - 3 ml) of stool specimens were stored in sterile screw cap containers and kept at -20°C using deep freez until being examined by ELISA.

Examination of stool specimens

Direct (Wet Mount) Examination

Stool samples were examined by wet mount preparation to detect the trophozoites and/or cysts of E. histolytica / E. dispar. Two slides were prepared for each sample, using a clean grease free slides, a small drop of normal saline was placed on the slide and mixed with a small pea size after well mixing of the sample using a wooden stick, a clean cover slid then placed and the specimen examined using light microscopy under low and high power magnification. The identification of the parasite was done by direct wet mount using normal saline 0.9 %, buffered methylene blue, and lugol's iodine 1 % (WHO, 1991)(11).

ELISA for E. histolytica / E. dispar Stool Antigen[11].

(Diagnostic Automation Co. Germany)

The Diagnostic Automation Inc. ELISA stool antigen assay was performed on 93 of stool samples from patients with cancer and 10 control stool specimens (microscopy negative for E. histolytica / E. dispar trophozoite and/or cyst) and the test performed according to manufacturing company.

Results

A total of 93 patients with malignant cancer complaining from abdominal disorder (abdominal pain and diarrhea) were enrolled in the present study and healthy group 10 non infected with amebiasis and without cancer of different age groups nearly match to infected group of cancer patients. Table 1

According to the clinical investigation, and histopathological examination the patients divided into solid group of cancer which involved that of digestive system: (colon cancer, pancreatic cancer and gastric cancer) and hematology form cancer which included leukemia and lymphoma. Solid tumor form the predominant type (65/93; 69.8%) while the hematological cancer form 30.1% (28/93), of the solid tumor, colon cancer was the common (29/65:44%) followed by gastric cancer (21/65:32.3%) and pancreatic cancer (15/93:23%). While in the hematological cancer form leukemia was (15/28: 53.57%) and lymphoma was (13/28: 46.42%).Table2

The higher rate of E. histolytica /E. dispar was demonstrate in semi –solid stool (45.98%,17/37), followed by solid stool(30.4%, 7/23) and then liquid stool (27.27%, 9/33). However, the differences were not statistically significant (X2=2.993, P>0.05), Table 3.

The higher rate of E.histolytica / E.dispar positivty was demonstrated in yellow colored (64.2%) stool, while the lowest was in black colored stool (0%), with significant difference (X2 = 14.252 ; P= 0.003), Table 3.

Regarding residency, Table 4 shows that 48 (51.6%) cancer patients were from urban area while 45(48.4%) cancer patients were from rural area. Thus there was no significant difference in distribution of cases between rural and

urban areas (X2= P>0.05). The infection rate with E. histolytica in urban areas was 23 (24.73%), while in rural area was 10(10.75%), with significant difference (X2= 6.698 and P=0.01).

Table 5) shows that 66.7% (22/33 cases) of E. histolytica / E. dispar positive cases occurred in patients whose their family were ≥6 individuals. In addition, large size families (≥ 6 idividual) accounted for 23.65%. E. histolytica infection from the total cases (22/93 cases). However, there was non significant (X2=2.381, P>0.05) differences in positivity rate in relation to family size.

The high E. histolytica /E. dispar were demonstrated positive cases in illiterate individual (33.3%), followed by primary school educational level (30.3%:10/33) and secondary school level (24.2, 8/33 cases). While the lowest rate of infection with E. histolytica /E. dispar was lower (6.1, 2/33) in the subject with higher educational level, (Table 6).

When the comparison preformed between those with educational level of secondary and below and those with higher educational level the rate of infection with E. histolytica was 38.2 (29/76 cases) in secondary education and below, while it was 3.5%in those with higher educational level, (Table 7).

Disscussion

The present study indicated that E. histolytica / E. dispar infection was detected in 35.5% of cancer patients with developed infection with E. histolytica / E. dispar.

In literature, the prevalence of E. histolytica infection varies globally[12], and with a range of 1% in developed countries and 80% in developing countries[13]. In Iraq, the reported studies suggest a incidence rate of 20.7% to 29.5%. But in a restudy preformed in Basrahm suggest a incidence of 27.7% in adult[14].

This the higher prevelance rate of E. histolytica /E. dispar in our subject patients could be attributed to immunosuppressive state in patients with cancer. Subjects with some type of immuncompromised status and those receiving immunosuppressive chemotherapy have an increased incidence of parasitic infection including E. histolytica [15-18].

The present study E. histolytica incidence rate was higher to that reported by Botero et al.[19], as they found incidence rate of 9.91% in immunocompromised patients. However, the above incidence rates were including E. histolytica/E. dispar, but in this study Ent.histolytica incidence rate was 10.7% and thus E. dispar forms 24.8% which was non pathogenic. However the comparison is difficult since different studies were with different study designs.

Cancer type influenced the infection with E. histolytica /E. dispar as this study indicated. Patients with solid cancer show a rate of infection with E. histolytica / E. dispar as compared to hematogenous cancer . In addition, within the solid tumor the infection rate with E. histolytica / E. dispar was higher in patients with colonic and pancreatic cancer than in those with gastric cancer. However, the difference was not significant. Furthermore, E. histolytica /E. dispar infection was significantly higher in patients with leukemia as compared to those with lymphoma.

A study preformed in Saudia Arabia[20], reported incidence rate of 5.2% for E. histolytica in immunocompromised patients. In addition, Mohanad et al.[21] reported a rate of 1.7% for E. histolytica in Northen in India. However, the present study E. histolytica infection rate was relatively higher than that previously reported for western Nepal

(27.7%)[22], Colombia (25.2%) Florez et al.[23], Ethiopia (24.8%)[24].

In a recently reported study, E. histolytica infection was reported in 5.7% among HIV/AIDS, patients in Nigria . However E. histolytica /E. dispar infection rate in HIV/AIDS patients was influenced by sexual behavior[25]. Furthermore, in a pediatric immunocompromised population, the infection rate was 4.7% [26].

Although infected host with E. histolytica deploys a strong immune response, the parasite has developed a remarkable number of mechanisms to evade these attacks[27]. The diversity in E. histolytica infection rate in immunocompromised individual as this study indicated and as reported in literature are due to different factors.

The factors that influence variation in infection rate with E. histolytica in immunocompromised subjects include personal difference in innate immunity, ability of parasite strains to evade host immune response, variability of host inflammatory response that contribute to tissue damage, and host genetics[28, 29]. Parasitic infection severity, natural course and manifestation was modified by the compromise in host immune response[30].

Cancer was associated with immune deficiency and this subsequently enhances the emergence of infection. Since the suppression is not presented as entity, thus there is a differences in infection rates with E. histolytica in different reported studies settings[31].

A recently reported study[32], suggest that in animal model, the presence of commensal Clostiridia- related bacteria in gut is protective during E. histolytica infection. This finding may add explanation for host response differences to E. histolytica infection and geographical variation in infection rate in immunocomptent and immunocompromised subjects.

Amoebiasis or unknown factors related to infection with E. histolytica may stimulate the proliferation of Lymphoma cells[33]. Although present study finding indicated that tumor type may influence E. histolytica infection rate, other study[33], not reported a statistical significant differences between the different type of immunocompromised condition. In previous studies on adults immunocompromised population who are with cancer, the prevalence rate varied from 2%to 50% for parasitic infection[34]. About similar prevalence rates (42%) were demonstrated in immunocompromised children with malignant disease [35].

Parasitic infections also reported in 84.3%of subjects with HIV infection. The present study incidence rate was lower to that reported for Indonesia in immunocompromised children[36] and Malysia[37].

The present study indicated that E. histolytica infection was more predominant (45.9%) in semi-solid stool type. However, E. histolytica infection rate differences between stool types were statistically not significant. This finding not agreed to that reported by others in immunocompetent children and adults as they reported high infection rate in liquid stool[38-40]. This variation may be explained on the basis of that immune deficiency/suppression lead to change in the ecology of microbial flora of the gut. Such changes may influence the presence or absence of certain microbes including parasites such as E. histolytica /E. dispar. E. histolytica infection rate differ significantly between different stool color and the rate of infection was predominant in yellow colored stool.

Residence not influence the distribution of cancer cases, however, E. histolytica / E. dispar infection rate was

significantly higher in urban than in rural areas (X2 =6.698, P=0.01). This finding agreed to that reported recently[40], in immunocompetent subjects in Kirkuk and to that reported by Kurt et al[41]. However, the present study finding is not consistent to that reported for Samara as there is no difference in prevalence between rural and urban areas[42]. In addition, this study findings disagree with finding of Kadir and Salomon studyin Al-Tameem prorvince[43], that showed a high infection rate in rural than in urban areas[. Moreover, the study differ from AL-Samarray[38], who recorded a higher infection rate in rural than in urban.

The higher infection rate in urban than in rural may be due to increasing density of immigrants in Kirkuk city especially after the occupation of Iraq by the coalition forces in 2003. In addition, personal hygiene and low socioeconomic status in urban areas and may affect the infection rate in urban area.

Family individual of ≤ 6 was with higher infection (66.7%) rate as compared to other family size. However, the difference was not statistically significant. This finding was consistent to that reported recently (48.05%) for Kirkuk in normal individuals[40]. In addition, other studies reported a higher incidence of infection in large size as compared to small size families[44,45].

The present study indicated a decreased in E. histolytica infection rate with increase in education level. However, the differences not reach a significant level. This finding was agreed to that reported recently for Kirkuk[40], in immunocompetent subjects and that reported in other geographical areas[46].

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Table 1: Study population

Group		Number
Infected(Cancer patient with amebiasis)	Detected with Tak labI	33
Non –infected cancer patient without amebiasis.		66
Control group		10

Table 2:- Frequency distribution of cancer type in study population.

Type of cancer		No. of Patients	%
Solid cancer	Colon cancer	29	31.1%
	Gastric cancer	21	22.6%
	Pancreatic cancer	15	16.1%
	Total	65	69.9%
Hematological cancer	Leukemia	15	16.1%
	Lymphoma	13	14%
	Total	28	30.1%
Total		93	

Table 3: Macroscopic examination of stool specimens from cancer patients

Characteristics		Tested cases. (93)		E. histolytica / E.dispar			
				Positive		Negative	
		No.	%	No.	%	No.	No.
Consistency	Liquid (diarrhea)	33	35.4%	9	27.27%	24	72.72%
	Semi-solid	37	39.78%	17	45.9%	20	54.05%
	Solid	23	24.73%	7	30.4%	16	69.56%
	X2 = 2.993		P= 0.223				
Color	Yellow	14	15.05%	9	64.2%	5	35.71%
	Greenish	31	33.33%	15	4.38%	16	51.61%
	Dark brown	42	45.1%	9	21.4%	33	87.57%
	Black	6	6.45%2	0	0	6	100%
	X2 = 14.251		P=0.003				

Table 4: Frequency of E. histolytica / E. dispar according to residency

Site of residency	No. Examined	No. Positive	Positive %	Positive % from total
Urban	48(51.6%)	23	51.11%	24.73% %
Rural	45(48.4%)	10	22.22%	10.7% %
Total	93(100%)	33	35.5%	25.66 %

X2 =6.698 P=0.010

Table 5:- Frequency of E. histolytica / E. dispar according to family size.

Family size	No. Examined	No. Positive	Positive % from total positive	Positive % Per group	Positive % from total
≤3	13	3	9.1	23.07%	3.22%
4-5	16	8	24.2	50%	8.6%
6≥	64	22	66.7	34.375%	23.65%
Total	93	33	100		35.48%

X2= 2.381 p>0.05

Table 6:- Distribution of E. histolytica according education.

Educational State	No. of Examined	No. of positive	Positive %
Illiterate	25	11	33.3%
Primary	35	10	30.3%
Secondary	16	8	24.2%
Preparatory	6	2	6.1%
Institution and Bachelor	11	2	6.1%
Total	93	33	
X2= 4.446	P=0.349		

Table 7:- Comparison of E. histolytica positivity rate between subjects with ≤ secondary and higher educational level.

Educational level	Total	Positive	%
≤ Secondary	76	29	38.2%
Higher	17	4	23.5%
X2= 1.3	P>0.05		