Distribution of haematological indices among subjects with *Blastocystis hominis* infection compared to controls

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Abstract

Introduction: Some studies suggest *Blastocystis hominis* is a potentially pathogenic protozoa. *Blastocystis hominis* contributed to anaemia in children aged 8–10 years old in one study.

Aim: To compare haematological indices in cases with *blastocystis hominis* infection with healthy controls.

Material and methods: From 2001 to 2012, 97600 stool examinations were done in 4 university hospitals. Parasites were observed in 46,200 specimens. Of these cases, subjects with complete laboratory investigation (complete blood count – CBC, ferritin, total iron binding capacity – TIBC, and serum) and blastocystis hominis infection were included in this study as the case group. Of these cases, 6851 cases had only *B. hominis* infection. In the control group, 3615 subjects without parasite infestation were included. Age, haemoglobin (Hb), serum iron, TIBC, white blood cell (WBC), platelet (PLT), mean corpuscular volume (MCV), haematocrit (HCT) and erythrocyte sedimentation rate (ESR) were recorded for cases and controls. SPSS software version 13.0 was used for analysis. Independent sample *t*-test and χ^2 tests were used for comparison.

Results: Erythrocyte sedimentation rate level was significantly higher in cases with *B. hominis* infection ($p < 0.05$). C-reactive protein level was positive in 1.46% of cases and 0.5% of controls, which was statistically significant ($p < 0.05$). Frequency of serum iron < 120 was significantly higher in cases with *B. hominis* infection compared to controls. Occult blood was positive in 0.93% of cases and in none of the controls ($p < 0.05$).

Conclusions: The ESR, CRP and occult blood was significantly higher in cases with *B. hominis* infection.

Introduction

Blastocystis hominis (*B. hominis*) is an obligate anaerobic protozoan found in the human large intestine and is the most common eukaryotic organism reported in human faecal samples [1]. The parasite is a zoonotic organism, found in birds and swine, and can be transmitted easily to humans via the oral-faecal route. The prevalence of *B. hominis* varies in different countries from 1.5% to 50% [2]. At first it was thought that the organism is a non-pathogenic yeast, but later *B. hominis* was classified as a protozoan of the stramenopiles line [3]. The pathogenesity of *B. hominis* is still in debate because this parasite is very common in the healthy population without causing any symptoms [4, 5]. However, some studies suggest that it is a potentially pathogenic protozoa and it was reported that *B. hominis* causes various problems such as watery diarrhoea, abdominal pain, bloating, fever, nausea and gastrointestinal problems [5, 6]. In addition, it was reported that *B. hominis* may contribute in colitis, terminal ileitis and rectal bleeding [7, 8]. *Blastocystis hominis* contributed to anaemia in children aged 8–10 years old as per one study [9].

Aim

The aim of the study was to compare haematological indices in cases with *blastocystis hominis* infection with healthy controls.

Material and methods

From 2001 to 2012, 97,600 stool examinations were done in 4 university hospitals. Parasites were observed in 46200 specimens. Of these cases, subjects with complete laboratory investigation (complete blood count – CBC, ferritin, total iron binding capacity – TIBC, and serum) were included in this study as the case. Of these cases, 6851 cases had only *B. hominis* infection. This retrospective study was carried out on 10786 subjects who visited the hospital for routine checkup. In this study, subjects who attended for routine checkup were included. Of 8761 cases with *B. hominis* infection, 6851 cases with complete laboratory examination were included. In this case control study, 6851 cases with *B. hominis* infection and 3615 controls were included. Age, haemoglobin (Hb), serum iron, TIBC, white blood cell count (WBC), red cell distribution width (RDW), platelet (PLT), mean corpuscular volume (MCV), haematocrit (HCT) and erythrocyte sedimentation rate (ESR) were recorded for cases and controls.

Statistical analysis

SPSS software version 13.0 (Chicago, IL, USA) was used for analysis. Independent sample *t*-test and χ^2 tests were used for comparison. A value of $p < 0.05$ was considered significant.

Results

Demographic features are shown in Table I. There were significant differences between the two groups regarding distribution of iron levels, with the exception of iron level range 120–140 μg/dl (Table I). For iron level < 80 μg/dl, the relative frequency was significantly higher in the case group. For iron level > 120 μg/dl, the relative frequency was significantly higher in the control group ($p < 0.05$).

Frequency of TIBC = 300–350 μg/dl and 400–450 μg/dl was higher among the cases, and the frequency of TIBC = 350–400 μg/dl was significantly higher among the controls (Table II). There was a significant difference between the two groups regarding platelet count, with the exception of PLT < 150,000/μl and PLT > 450,000/μl (Table II). Subjects with an eosinophil count of 1% were significantly more frequently in the case group than in the control group. The reverse was true for subjects with an eosinophil count of 2%. There was no significant difference between the two groups in eosinophil count in other ranges (Table II).

Discussion

Occult blood was more common in the group with *B. hominis* infection compared to the control group. This may be due to colitis from the *B. hominis* infection. Leder *et al.* studied over 2800 healthy people over 15 months [10]. They did not find significant correlation between clinical symptoms and the presence of *B. hominis*. In the study by Zuckerman *et al.*, colonsocopy did not show any significant abnormality [11]. In the study by Wakid on 1238 workers in the western region of Saudi Arabia, no significant correlation was found between blastocystis infection and occult blood [12]. Our samples were collected from hospitals. These differences may be due to selection bias.

In our study the frequency of iron level < 80 was significantly higher in subjects with *B. hominis* infection. In the study by El Deeb *et al.* on pregnant woman with iron deficiency anaemia compared to those without iron

Table I. Demographic features among cases and controls

Parameter		Cases (n = 6851)	Controls (n = 3615)	Value of p
Gender		M = 4134, F = 3037	M = 1459, F = 2156	
Age range [years]	0–10	5741 (83.80)	2977 (82.4%)	0.069
	11–20	322 (4.70%)	265 (7.3%)	< 0.001
	21–30	292 (4.27%)	193 (5.3%)	0.015
	31–40	240 (3.50%)	75 (2.1%)	< 0.001
	41–50	125 (1.82%)	46 (1.3%)	0.040
	51–60	80 (1.17%)	34 (0.9%)	0.256
	61–70	35 (0.52%)	17 (0.5%)	0.977
	71–80	14 (0.2%)	8 (0.2%)	0.907
	81–90	2 (0.02%)	0 (0.0%)	NA

Table II. Comparison between the two groups regarding haematological indices

Parameter		Case (n = 6851)	Control (n = 3615)	Value of p
ESR, mean ± SD		11.16 ±8.67	10.32 ±5.01	< 0.01
Occult blood	Pos	34 (0.49%)	0	< 0.001
Hb (NL = 11–18 g/dl)	< 10	12 (0.16%)	11 (0.3%)	0.120
	10	98 (1.43%)	70 (1.9%)	0.063
	11	427 (6.23%)	325 (9.0%)	< 0.001
	12	1120 (16.35%)	830 (23.0%)	< 0.001
	13	1122 (16.37%)	585 (16.2%)	0.826
	14	1143 (16.70%)	323 (8.9%)	< 0.001
	15	2671 (39.00%)	1411 (39.0%)	0.986
	16	194 (2.83%)	48 (1.3%)	< 0.001
	17	45 (0.65%)	6 (0.2%)	0.001
	18	15 (0.22%)	5 (0.1%)	0.215
	19	4 (0.06%)	1 (0.0%)	0.353
Fe [μg/dl]	20–40	19 (0.27%)	0 (0%)	0.002
	40–60	206 (3.00%)	8 (0.2%)	< 0.001
	60–80	5888 (85.94%)	2702 (74.7%)	< 0.001
	80–100	674 (9.83%)	761 (21.1%)	< 0.001
	100–120	58 (0.84%)	136 (3.8%)	< 0.001
	120–140	6 (0.08%)	8 (0.2%)	0.152
TIBC (NL = 240–450 μg/dl)	< 240	11 (0.16%)	8 (0.2%)	0.697
	240–300	693 (10.11%)	374 (10.3%)	0.778
	300–350	3031 (44.24%)	1492 (41.3%)	0.003
	350–400	2003 (29.23%)	1486 (41.1%)	< 0.001
	400–450	1099 (16.04%)	250 (6.9%)	< 0.001
	> 450	14 (0.21)	5 (0.1%)	0.271
PLT (NL = 150000–450000/μl)	< 150000	134 (1.95%)	75 (2.1%)	0.611
	150000–200000	468 (6.83%)	328 (9.1%)	< 0.001
	200000–250000	751 (10.97%)	263 (7.3%)	< 0.001
	250000–300000	4127 (60.23%)	2336 (64.6%)	< 0.001
	300000–350000	905 (13.20%)	251 (6.9%)	< 0.001
	350000–400000	199 (2.90%)	168 (4.6%)	< 0.001
	400000–450000	151 (2.20%)	114 (3.2%)	< 0.001
	> 450000	118 (1.72%)	80 (2.2%)	0.079

Table II. Continued

Parameter		Case (n = 6851)	Control (n = 3615)	Value of p
Eosinophil (%)	1	719 (10.49%)	440 (12.2%)	0.008
	2	5387 (78.63%)	2751 (76.1%)	0.003
	3	316 (4.61%)	189 (5.2%)	181
	4	150 (2.19%)	94 (2.6%)	0.185
	5	98 (1.43%)	58 (1.6%)	0.484
	6	49 (0.71%)	20 (0.6%)	0.527
	7	58 (0.84%)	35 (1.0%)	0.440
	8	21 (0.30%)	6 (0.2%)	0.287
	9	19 (0.27%)	10 (0.3%)	0.700
	10	19 (0.27%)	6 (0.2%)	0.491
	> 10	15 (0.22%)	6 (0.2%)	0.788
WBC count (NL = 4000–11000 cells/μl)	2000–2999	58 (0.84%)	66 (1.82%)	< 0.001
	3000–3999	447 (6.52%)	238 (6.59%)	0.907
	4000–4999	878 (12.81%)	352 (9.73%)	< 0.001
	5000–5999	1491 (21.76%)	823 (22.77%)	0.239
	6000–6999	1487 (21.70%)	806 (22.30%)	0.486
	7000–7999	903 (13.18%)	553 (15.30%)	0.002
	8000–8999	864 (12.61%)	516 (14.27%)	0.016
	9000–9999	550 (8.02%)	210 (5.80%)	< 0.001
	10000–10999	96 (1.40%)	33 (0.92%)	0.031
	11000–11999	29 (0.42%)	5 (0.14%)	0.042
	12000–12999	27 (0.39%)	8 (0.23%)	0.140
	13000–13999	21 (0.30%)	5 (0.13%)	0.100
CRP		Pos: 100 (1.46%)	18 (0.5%)	< 0.001

deficiency, the frequency of *B. hominis* was significantly higher in the first group [13]. In another study by Yavasoglu *et al.*, the rate of *B. hominis* infection was significantly higher among cases with iron deficiency anaemia [14]. In the study by Cheng *et al.* haemoglobin, neutrophil count and haematocrit were decreased in subjects with *B. hominis* infection [15]. In the recent study by El Deeb and Khodeer, the frequency of parasites was significantly higher in cases with iron deficiency (anaemia) compared to non-anaemic patients [16].

Although the exact mechanism of iron deficiency in subjects with *B. hominis* was not understood, some studies showed invasion of *B. hominis* in the gastrointestinal tract and subsequent blood loss [8]. In the literature, *B. hominis* was reported as the possible cause of acute abdomen in children [17]. There is evidence regarding the presence of inflammation when *B. hominis* is present [18]. Yavasoglu *et al*. did not find evidence that supported the invasion of mucosa [14].

Another mechanism that may lead to the development of anaemia in blastocystis is that infected subjects may return to the molecular structure of *B. hominis*. Stenzel *et al.* showed that in the endocytic pathway of *B. hominis* cationic ferritin is essential [19].

Although there have been several published studies regarding *B. hominis* infection, there are few publications on its association with haematological indices.

Limitations – the method of this study was retrospective, and this is the main limitation.

Conclusions

Blastocystis hominis is a possible factor in haematological abnormalities. Another cohort study on a healthy population is recommended for determination of the cause and effect relationship.

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References

1. Tan KS. Blastocystis in humans and animals: new insights using modern methodologies. Vet Parasitol 2004; 126: 121-44.
2. Su FH, Chu FY, Li CY, et al. Blastocystis hominis infection in long-term care facilities in Taiwan: prevalence and associated clinical factors. Parasitol Res 2009; 105: 1007-13.
3. Yoshikawa H, Nagano I, Wu Z, et al. Genomic polymorphism among Blastocystis hominis strains and development of subtype-specific diagnostic primers. Mol Cell Probes 1998; 12: 153-9.
4. Dogruman-Al F, Kustimur S, Yoshikawa H, et al. Blastocystis subtypes in irritable bowel syndrome and inflammatory bowel disease in Ankara, Turkey. Mem Inst Oswaldo Cruz 2009; 104: 724-7.
5. Eroglu F, Genc A, Elgun G, et al. Identification of Blastocystis hominis isolates from asymptomatic and symptomatic patients by PCR. Parasitol Res 2009; 105: 1589-92.
6. Jones MS, Whipps CM, Ganac RD, et al. Association of Blastocystis subtype 3 and 1 with patients from an Oregon community presenting with chronic gastrointestinal illness. Parasitol Res 2009; 104: 341-5.
7. Russo AR, Stone SL, Taplin ME, et al. Presumptive evidence for Blastocystis hominis as a cause of colitis. Arch Intern Med 1988; 148: 1064.
8. Tsang T, Levin B, Morse S. Terminal ileitis associated with Blastocystis hominis infection. Am J Gastroenterol 1989; 84: 798.
9. Pegelow K, Gross R, Pietrzik K, et al. Parasitological and nutritional situation of schoolchildren in the Sukaraja District, West Java, Indonesia, Southeast Asian. J Trop Med Public Health 1997; 28: 173-90.
10. Leder K, Hellard ME, Sinclair MI, et al. No correlation between clinical symptoms and Blastocystis hominis in immunocompetent individuals. J Gastroenterol Hepatol 2005; 20: 1390-4.
11. Zuckerman MJ, Watts MT, Ho H, et al. Blastocystis hominis infection and intestinal injury. Am J Med Sci 1994; 308: 96-101.
12. Wakid MH. Fecal occult blood test and gastrointestinal parasitic infection. J Parasitol Res 2010; 2010. pii: 434801. doi: 10.1155/2010/434801.
13. El Deeb HK, Salah-Eldin H, Khodeer S. Blastocystis hominis as a contributing risk factor for development of iron deficiency anemia in pregnant women. Parasitol Res 2012; 110: 2167-74.
14. Yavasoglu I, Kadikoylu G, Uysal H, et al. Is Blastocystis hominis a new etiologic factor or a coincidence in iron deficiency anemia? Euro J Haematol 2008; 81: 47-50.
15. Cheng H, Guo Y, Shin JW. Hematological effects of Blastocystis hominis infection in male foreign workers in Taiwan. Parasitol Res 2003; 90: 48-51.
16. El Deeb H, Khodeer S. Blastocystis spp.: frequency and subtype distribution in iron deficiency anemic versus non-anemic subjects from Egypt. J Parasitol 2013; 99: 599-602.
17. Andiran N, Acikgoz ZC, Turkay S, et al. Blastocystis hominis – an emerging and imitating cause of acute abdomen in children. J Pediatr Surg 2006; 41: 1489-91.
18. Stenzel D, Boreham P. Blastocystis hominis revisited. Clin Microbiol Rev 1996; 9: 563-84.
19. Stenzel D, Dunn L, Boreham P. Endocytosis in cultures of Blastocystis hominis. Int J Parasitol 1989; 19: 787-91.

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