



Giardiasis in cattle: clinical assessment of giardia lamblia in cattle at Basrah, Iraq

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Abstract

The aim of this study is for assessment cattle giardiasis clinically. It is first time study in type done in Iraq. It was conducted from September 2012 until May 2013 throughout it collected 1049 fecal samples from cattle of various ages and of both sexes, collected randomly from different areas of the northern Basrah. About 322 positive cases have been recorded in cattle and the infection rate was 30.6%. 241 cases out of 322 showed clinical signs while 81 infected cows did not show clinical signs which are asymptomatic. The clinical examination data showed variety of presentation among symptoms and signs. The percent of symptomatic type was higher than the percentage of asymptomatic giardiasis in cattle. The study reported symptoms such as diarrhea, foul smelling stool, and flatulence, loss of appetite, elevated temperature, constipation and emaciation in different proportions.

Keywords: *Giardia, Diarrhea, Asymptomatic, Cattle.*

1. Introduction

The genus *Giardia* falls within the phylum Sarcocystophora, class Zoomastigophora, order Diplomonadida and family Hexamitidae [1]. Giardiasis was the most frequently diagnosed water borne disease and the major public health concern of water utilities in the developed and developing nations, water is an important vehicle for the transmission of *Giardia* to human and livestock [2]. *Giardia* has been implicated as an etiological agent alone and in combination with other enteric pathogens in calf diarrhea [3], [4], [5]. The genus currently comprises six species, namely: *G. agilis*, *G. ardeae*, *G. duodenalis*, *G. microti*, *G. muris*, and *G. psittaci*, which are distinguished on the basis of the morphology and ultrastructure of their trophozoites [6]. The World Health Organization added *Giardia* to the list of parasitic diseases in 1981 [7]. The specific mechanisms of *Giardia* pathogenesis leading to diarrhea and intestinal mal-absorption are not completely understood and no specific virulence factors have been identified. The attachment of trophozoites to the brush border of intestine will produce a mechanical irritation or mucosal injury [8], [9]. According to the studies, giardiasis is a complex of pathophysiological alterations; one of them is the changed permeability of enterocytes resulting from the cyto-pathological effect of parasite's metabolites [10]. The pathogenesis has been studied in human epithelial cell lines [8], in laboratory animals [8], [10], [11], in goat kids [12], and in calves [13]. Cattle giardiasis is clinically and economically important because of the occurring reduction of productivity [14], [15].

2. Materials and methods

2.1. Clinical examination of cattle

Cattle were examined clinically and data were collected concerning external shape, body temperature, which observed and recorded.

2.2. Faecal samples

Through this study collect 1049 fecal samples. All fecal samples were collected freshly, directly from rectum or as soon as after defecation, collected samples stored in sterilized cups were used for each animal and all these cups were labeled by information with number, date of collection, animal condition, nature of feces, age of animal, address and name of owner. After collecting samples, transported to laboratory of Parasitology / Veterinary Medicine Collage for confirmed diagnosis.

a) Direct smear with normal saline

According to [16] and [17] direct smear done by comminute 5-6 stool or faecal balls or 2-4 gm with pestle and mortar (TOKYO / Japan). Then transfer a loop-fall of the material to a slide matchstick, and a drop of diluted fluid (normal saline) were placed on glass microscopic slide (ATACO / China) to form a uniform suspension. Spread it on the slide and apply cover slip (ATACO / China). Finally examine the slide microscopically under low power (10X) and high power (40X).

b) Direct smear with Lugol's iodine

After prepare the direct smear, used Lugol's iodine to kill and staining the trophozoites and cysts. Technique of this method by adding of one drop of Lugol's iodine (Himedia / India) to prepare slide and examination by light microscope (Olympus / Japan) under low (10X) and high power (40X) [16] and [17].

3. Main results

322 cases out of 1049 cattle examined were infected with giardiasis. The percent was 31.6%. Cattle showed clinical sings which are diarrhea, bloating, fever, anorexia, constipation; emaciation and foul smelling greasy faecal present and some cattle appear as an asymptomatic, table (1). There was a highly significant difference ($P \leq 0.01$) between giardiasis types and percentage of infection among symptomatology among cattle, the percent of symptomatic type was 74.8%, while the percentage of asymptomatic giardiasis was 25.2% table (2).

Table 1: Clinical Features of Giardiasis in 322 Cows with Percentage

Clinical features	Number of cases	%
Diarrhea	283	87.8
Foul smelling greasy faecal	241	74.8
Bloating	228	70.8
anorexia	209	64.9
Fever	42	13
Constipation	42	13
Emaciation	212	65.8
Asymptomatic	81	25.2

Table 2: Giardiasis Types and Percentage of Infection in Cows

Giardiasis types	Number of infected animals	%
Symptomatic cases	241	74.8
Asymptomatic cases	81	25.2
Total	322	30.6
$X^2=64.797, df= 1, P<0.01$		

4. Conclusion

This study was recorded two types of giardiasis, symptomatic and asymptomatic. The percentage of asymptomatic cases of giardiasis was 25.2%, on the other hand the symptomatic giardiasis contributed to 74.8%. There was a highly significant differences ($P < 0.01$) between two giardiasis types. This agree with most studies as study of [19] who mentioned the infection with *G. lamblia* ranges from asymptomatic passage of cysts, to acute diarrhea, to a syndrome of chronic diarrhea and mal-absorption. There are two types of giardiasis: the asymptomatic and symptomatic infections [20]. The clinical picture of giardiasis varies ranging from asymptomatic infection or acute self-limiting to severe, chronic one [21]. Many factors such as variation in parasite virulence and host immune defense may contribute to asymptomatic giardiasis [22]. The parasite adaptations promoting cyst survival in the external environment and trophozoite infectiveness and persistence in the mammalian small intestine each contribute to being key virulence properties for this parasite to cause symptomatic disease [23]. In this study eight common symptoms recorded in cattle were diarrhea 87.8%, foul smelling greasy stool 74.8%, bloating 70.8%, anorexia 64.9%, fever 13%, constipation 13% and emaciation 65.8%. These were agreeing with [24] the most important clinical symptoms are diarrhea and mal-absorption. The intestinal troubles followed by nausea and anorexia, other signs could be low-degree fever, flatulence and abdominal distension with cramps occurred in acute stage, later symptoms include profuse, watery and foul-

smelling diarrhea [7] and [25]. During the chronic stage lethargy, headache and muscle pain with progressive weight loss, loss of appetite and mal-absorption could be present [25]. Impaired fat absorption and steatorrhea are common in symptomatic giardiasis; stools are usually greasy and malodorous and float in water [26]. It only cause diarrhea in adult calves, chronic giardiasis in calves result in weight loss, lower feed conversion ratio and reduced slaughter weight [25]. In a study of calves naturally infected with *Giardia*, all infected animals were noted to have intermittent diarrhea, and mucus was seen in many fecal samples [27]. Typically, the symptoms accompanying infection include diarrhea, flatulence, upper intestinal cramps, abdominal distension, nausea, weight loss and mal-absorption [28]. Host factors such as immune status, nutritional status and age, as well as differences in virulence and pathogenicity of *G. duodenalis* isolates are recognized as important determinants for the severity of infection [29], [30] and [31]. The mechanism of all these manifestations were due to attachment of trophozoites to the brush border of intestine will produce a mechanical irritation or mucosal injury [8], [9]. The colonization of trophozoites in the small intestine results in a reduction in the height of the microvilli and therefore a loss of absorptive surface area [29]. This loss of absorptive surface leads to mal-absorption of glucose, electrolytes and water, and reduces disaccharidases activity [32]. This results in the small intestine filling with mucous and fluid, and ultimately mal-digestion and hyper-motility, all responsible for the clinical manifestation of diarrhea [33]. As a result, enterocytic brush border is damaged, the increased epithelial permeability leads to an inflammatory response, digestive and absorptive changes, that correlate with the brush border injury and disaccharidases deficiencies [11].

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