Incidence of Campylobacter and anaerobic bacteria among apparently healthy and diarrheic camel calves

By

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SUMMARY

A total of 100 fecal samples (60) from diarrheic and (40) from apparently healthy camel calves were bacteriologically examined for anaerobic bacteria and Campylobacter species.

Anaerobic bacteria, Clostridium perfringens, C. sordellii, C. sporogenes, Peptococcus species, Bacteroides fragilis and Eubacterium lentum were isolated in percentages of 61.67%, 3.33%, 3.33%, 18.75%, 8.33% and 5% respectively from the diarrheic camel calves. In case of apparently healthy animals the percentages were 12.5%, 0%, 0%, 22.5%, 10% and 2.5%, respectively.

Campylobacter organisms were identified as C. jejuni with percentage of 31.67% & 12.5% from diarrheic and apparently healthy while Campylobacter coli incidences were 3.33% and 5% respectively.

The Pathogenicity test was applied using C. sordellii, Campylobacter jejuni and Campylobacter coli strains in artificial infection (I/P) of Guinea pigs. However post mortem and histopathological examination of affected organs were studied. The results showed that pathogenic anaerobic bacteria and Campylobacter were shed through fecal matter in diarrheic and normal camel calves.

INTRODUCTION

The Camelidae is a comparatively small family of mammalian animals. There are two members of Old World camels living in Africa and Asia; the Arabian, the one-humped camel (Camelus dromedaries') and the Bactrian, the two-humped camel. Camels thrive in the harsh deserts of Africa and Asia and South American. They have been traditionally used for transport of people and things, to supply hides and fibres for clothing, other textile articles, and meat and milk products (Badran et al., 2008).

Camels have been used and bred for several thousand years, but
the efforts to understand their biology and diseases in greater depth has been done recently. Because camels are still such important animals in Africa, the Middle East and Asia, there have been more interest to understand their nutrition, health care needs, reproduction, behaviour, physiology, and its diseases (Wilson and Meyer, 1991).

Griffiths and Park, (1990) and Skirrow (1994) recorded that Campylobacter jejuni and Campylobacter coli are an important causes of diarrheic diseases of livestock, They suggested that the role of C. jejuni and C. coli in enteric disease is preponderant and only surpassed by Salmonella, and anaerobic bacteria.

Concerning to anaerobic bacteria causing diarrhea are Clostridium perfringens, C. sordelli, C. sporogenes and other anaerobic bacteria as Peptococcus Bacteroides fragilis, Micrococcus and Eubacterium species. were recorded by Allison et al., (1989).

The present study was aimed to apply more attention to camel as other domestic species. Much of the work so far has been carried out to study the role of campylobacters and anaerobic bacteria among camel calves which considered an important source of meat in Egypt.

**MATERIALS AND METHODS**

**Samples:**
A total of 100 fecal samples were collected from diarrheic (60 samples) and apparently healthy camel calves (40 samples) during the period of January, 2006 to March, 2006. Samples were collected in sterile labelled McCartney bottles from private farms at Giza Governorate.

**Bacteriological examination:**

**Anaerobic bacteria:**
All fecal samples were inoculated into cooked meat broth medium in duplicate. One of the two tubes was heated at 80°C for 10 min to eliminate vegetative organisms while the other inoculated medium was kept without heating and both were anaerobically incubated at 37°C for 48hr.

A loopful from inoculated broth was streaked onto 10% sheep blood agar plates while that from unheated medium was streaked the same medium containing 75 ug/ml neomycin sulphate blood agar and incubated for 48 hrs. The growing surface colonies which showed catalase negative reaction were picked up in pure form and re-inoculated into cooked meat broth for further identification. Identification and all isolates were done by cultural, morphological and chemical characters according to Koneman et al., (1988).
Typing of C. perfringens:
By Inoculation test in albino guinea pigs was done according to Stern and Batty (1975). as shown in Fig(7) and detection of toxins of C. perfringens by neutralization test was carried out on mice by intravenous injection of diluted fecal matter by saline in ratio of 1:5 and centrifugated 3000rp/m. (Okaley and Warrak 1953).

Pathogenicity test using Clostridium sordellii:
A total of ten albino Guinea pigs 300-400 grams of weigh were used to study the pathogenicity of the isolated strain of Clostridium sordellii, after Guinea pigs were isolated in hygienic cages and kept under observation. The Guinea pigs were experimentally infected by intra peritoneum route with dose of 1ml whole culture (WC) containing 10^5 toxigenic C. sordellii in cooked meat broth according to TE-Wen Chang et al. (1978) and Uzal and Kelly (1998).

Campylobacter:
All the samples were subjected to bacteriological examination according to Skirrow and Benjamin (1980). as follow: One gram of faecal material was triturated in one ml of sterile saline solution (0.9%) and then centrifugated at 3000r.p.m. for 5 minutes. Few drops of the supernatants fluid were immediately cultivated onto thioglycolate media,. The inoculated tubes were incubated at 25°C-37°C, and 42°C for 24 hours under reduced O2 (5%), CO2 (10%) and N2 (85%).

Biochemical tests:
Campylobacter isolates were tested for biochemical testes according to Holt et al., (1994).

Pathogenicity test of Campylobacter bacteria:
Campylobacters isolates were tested for its pathogenicity according to Coid et al. (1987). A total of 10 albino Guinea pigs 300-400 grams of weigh were used to study the pathogenicity of the isolated strains of C. jejuni and C. coli. The Guinea pigs were experimentally infected by intra peritoneal route with dose of 5x10^9 of viable organisms/ml. The mortality and the morbidity rates were recorded.

Histopathological study:
The histopathological study was investigated among the internal organs of the inoculated albino Guinea pig Liver, small intestine, cecum, and colon were fixed in 10% buffered neutral formalin solution, processed by slandered paraffin method, sectioned at 4-5 μ and finally stained with Hematoxylin and Eosin (Bancroft et al., 1996).

RESULTS AND DISCUSSION
From results achieved in Table (1) it is noted that Clostridium perfringens isolates were 42%, C. perfrin-
gens type B was 18.33% and 10% followed by type C 8.33% and 2.5% from diarrheic and apparently healthy, but types D were isolated from diarrheic camel calves only in percentages of 35%. These results were similar to Mohamed (1996) and Greco (2005).

In case of other Clostridium bacteria C. sordellii and C. sporogenes (2) strains for each in 3.33% from the diarrheic cases only but in case of Peptococcus (20) 20% strains (11) 18.75% from diarrheic and (9) 22.5% from apparently healthy camel calves, Bacteroides fragilis strains (10)10% 5 strains isolated from each diarrheic and apparently healthy camel calves in percentage of (8.33%) and 10% respectively and Eubacterium lentum 3 (5%) strains implicated from diarrheic and 1 (2.5%) strain isolated from apparently normal camel calves which agree with Allison et al. (1989) with differ in some anaerobic bacteria and its percentages.

The bacteria were discerned in mixed infection camel calves (C. perfringens and peptococcus), (C. perfringens, Eubacterium lentum), (Bacteroides fragilis, peptococcus), (C. perfringens, C. sporogenes) lastly (C. perfringens and C. sordellii).

The obligatory anaerobic bacteria prevalence in apparently healthy and diarrheic camel calves it is noted that the highest percentage of C. perfringens, Bacteroides fragilis, Peptococcus species, and Eubacterium lentum, respectively. Clostridium sporogenes and Eubacterium lentum are capable of proliferating, invading and destroying intestinal epithelium, cause enteritis in calves with cytotoxic and diarrheic effect (Hara-Kudo 1997). Clostridium sporogenes often inhibited other bacterial species and usual mixed with C. perfringens. Many other species of C. sporogenes produce toxins similar to tetanus toxin as well as many other toxins. (Craig Baumgartne 2004). C. sordellii is a respective anaerobic virulent bacterium strains cause lethal infections in several animal species, such as enteritis enterotoxaemia in lambs and calves leading to sudden death. The role of C. Sordellii toxins in pathogenesis produce up to 7 identified exotoxins. Of these, lethal toxin (LT) and hemorrhagic toxin (HT) are regarded as the major virulence factors. Other exotoxins include an oxygen-labile hemolysin, neuraminidase, DNase, collagenase, and lysolecithinase. Al-dape et al. (2006).

Bacteroides fragilis is the most common anaerobe recovered from various infections, such as intra-abdominal infection, foot ulcers and sepsis. Resistance to β-lactam antibiotics in Gram-
negative anaerobic bacteria is an important problem in the treatment of the infectious diseases caused by these micro-organisms. (Arpin et al., 2002). Campylobacter Jejunii incidence was 24% from total samples and these results verified were simulated to what reported by Minihan et al. (2004) and Inglis et al. (2005) who isolated Campylobacter Jejunii (13%) from fecal matter of camels; While Campylobacter coli incidence was 14.0%.

The incidence of Campylobacter jejuni from apparently healthy camel calves was (5) 12.5% but from diarrheic cases was (19) 31.67%, in spite of C. coli of healthy group was (2) 5% and of diarrhoeic ones was 2(3.33% (Table2). The shedding of Campylobacter bacteria from apparently healthy and diseased camel calves cleared that there is significant variation in incidence of isolation of C. jejuni and C. coli between apparently healthy and diarrhoeic camel calves Snodoggrass et al., (1986) and Adesiyum et al., (1992).

The Pathogenicity test which was applied we noted that C. Jejunii is more virulence than C. coli and this result was appeared in the time of death after injection the first elapse one day and the second elapse three days and for conformation P.M. and the histopathological studies were done.

In case of C. sordellii experimental infection to G. pig death occurred after 24 hrs from injection.

Concerning to the single infected cases The Clostridium perfringens were 28 in percentage of 46.6 and 4 cases in percentage 0f 10 in diarrhoeic and apparently healthy camel calves respectively. In state of Bacterioides fragilis 5 (12.5%) and 5 (8.33%), Peptococcus species 8 (13.33%) and 8 (20%) in addition to Eubacterium lentum 1 (1.66%) and 1 (2.5%) the single cases of Campylobacter jejuni were 5(6.66%) and 3 (10%) in diarrhoeic and apparently healthy respectively; As cleared in Table (3).

In this study improved that there is mixed infection between the identified Campylobacter and anaerobic bacteria as achieved in Table (3). From results revealed that there is coaggregation between Campylobacter jejuni and C. perfringens in percentages of 13.33 in diarrheic cases however C. coli in percentage of 1.66% in apparently healthy ones ,In case of C. sordellii and C. jejuni only of diarrhoeic ones in 3.33%, C. sporogens implicated in diarrheic cases with C. coli only in 3.33% Peptococcus was coaggregated with both C. jejuni and C. coli in per-

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percentage of 3.33, 1.66 and 5 in diarrheic and apparently healthy group respectively this deals with that is a synergistic action in inducing enteritis in calf camels and *Eubacterium* was only with *C. jejuni* in percentage of 3.33 and this agree with obtained by Jenkison & Dymock (2004).

**Histopathological results and discussion:**

1-Macroscopic examination

Post mortem of Guinea pig Injected by *Clostridium sordellii microorganism* died 24 hrs post infection showing congestion in small intestine, colon and liver Fig (1).

Post mortem lesions of experimentally infected guinea pigs with *Campylobacter coli* died 5days post infection showing multiple focal necrotic areas in the liver together with small abscess formation while the intestine are pale, (Fig. 8 ).

Post mortem lesions of experimentally infected guinea pigs with *Campylobacter Jejuni* died 24 hrs post infection showing congested liver and sever hemorrhagic small intestine, (Fig.9). The cecum showing erythematic and distended with fluid, this picture agrees with that observed with Humphrey et al. (1985).

2-Microscopic examination

A-Histopathological changes in experimentally infected guinea pigs with *Clostridium sordellii microorganisms*:

Microscopic examination of small intestine and colon showed destructed and necrotic mucosa with diffused mononuclear cells infiltration in the submucosa (Figs. 2 & 3, respectively) and histological changes of hepatic tissues revealed mononuclear cells infiltration in the portal areas, (Fig. 4). Together with sporadic necrobiotics changes through out the hepatic tissues (Fig. 5)

B-Histopathological changes in experimentally infected guinea pigs with *Campylobacter-coli*:

Hepatic tissue revealed different pathological alterations including both the portal tract and hepatic tissue. For the portal area; there were hyperplasia in the bile duct, vacuities, and mononuclear cells infiltrations, (Fig. 10). Among the hepatic tissue, there were multiple focal areas of coagulative necroses hepatocytes infiltrated with mononuclear cells, (Fig. 11). Abscess formation was also performed some liver samples and consisted of central suppuration surrounded with pyogenic membrane which consisted of; dilated vessels, leucocytic infiltrations, and fibroblastic prolifera-
tions, (Fig. 12 & 12a).

This picture agree with that described by Acik, and Cetinkaya (2005).

The microscopic picture of small intestine showed mucosal necrosis, (Fig.13). And sub mucosal congestion with mononuclear cells infiltrations, (Fig. 13a). The histopathological changes in colon were confined to degeneration in the mucosal layer with submucosal congestion (Fig. 14), And leukocytic infiltrations, (Fig. 14a). Russel, et al (1989).

C- Histopathological changes in experimentally infected guinea pigs with Campylobacter-Jejuni

Liver tissue contributed congestion in the central veins, together with cellular alterations appeared as; swelling, inculsion, and polymorphism, (Fig. 15), (Khalil, 2002). The microscopic picture of small intestine revealed duodenal hemorrhagic mucosa, degenerated glands, ( Fig. 16), and cellular infiltration in both mucosa and submucosal, (Fig. 16-a ). The same alterations reported in the C.jejuni with submucosal depletion in the intestinal tonsils, (Fig.17&17-a). The same lesions described by Welkos (1984).

The Cecal histopathology included severely dilated cecal glands with luminal desquamated and destructed epithelial lining, (Fig. 18). Also there was depletion in the pyres patches, (Fig. 18 -a). This picture was matched with that of Humphrey, et al (1985) who confirmed that C. jejuni is a major cause of diarrhea and enterocolitis in humans and animals.

CONCLUSION

From the current study, it can be concluded that the importance of the anaerobic intestinal flora as C.sordellii ,C.sporogenes, peptococcus species ,Bacterioes fragilis and Eubacterium lentum in addition to C.perfringens in the induction of diarrhea which some times leads to do synergy in inducing enteritis with Campylobacters in calf camels. And attention should be paid for raising calf camels in farms and of even of small handlers under control measures of management, nutrition and medical cares by vaccination agents the anaerobic bacterial diseases.
Table (1): Incidence of anaerobic bacteria isolates from apparently healthy and diarrheic camel calves.

<table>
<thead>
<tr>
<th>Anaerobic microorganisms</th>
<th>Diarrheic camel calves (60)</th>
<th>Apparently normal camel calves (40)</th>
<th>Total (100)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td><em>C. perfringens:</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type B</td>
<td>37</td>
<td>61.67</td>
<td>5</td>
</tr>
<tr>
<td>Type C</td>
<td>11</td>
<td>18.33</td>
<td>4</td>
</tr>
<tr>
<td>Type D</td>
<td>5</td>
<td>8.33</td>
<td>1</td>
</tr>
<tr>
<td><em>C. sordellii</em></td>
<td>21</td>
<td>35</td>
<td>0</td>
</tr>
<tr>
<td><em>C. sporogenes</em></td>
<td>2</td>
<td>3.33</td>
<td>0</td>
</tr>
<tr>
<td><em>Peptococcus species.</em></td>
<td>11</td>
<td>18.75</td>
<td>9</td>
</tr>
<tr>
<td><em>Bacteroides fragilis</em></td>
<td>5</td>
<td>8.33</td>
<td>5</td>
</tr>
<tr>
<td><em>Eubacterium lentum</em></td>
<td>3</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

Table (2): Incidence of *Campylobacter* Species from apparently healthy and diarrheic camel calves.

<table>
<thead>
<tr>
<th>Campylobacter species</th>
<th>Apparently healthy camel calves (40)</th>
<th>Diarrheic camel calves (60)</th>
<th>Total (100)</th>
</tr>
</thead>
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<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td><em>C. jejuni</em></td>
<td>5</td>
<td>12.5</td>
<td>19</td>
</tr>
<tr>
<td><em>C. coli</em></td>
<td>2</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>17.5</td>
<td>21</td>
</tr>
</tbody>
</table>
Table (3) Single and Mixed infection cases in and between Campylobacters and anaerobic bacteria.

<table>
<thead>
<tr>
<th>Case of camel calves</th>
<th>Diarrheic</th>
<th>Apparently healthy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Single infection</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>C. perfringens</em></td>
<td>28(46.6%)</td>
<td>4(10%)</td>
</tr>
<tr>
<td><em>Bacteriodes fragilis</em></td>
<td>5(12.5%)</td>
<td>5(8.33%)</td>
</tr>
<tr>
<td><em>Peptococcus</em> species</td>
<td>8(13.3%)</td>
<td>8(20%)</td>
</tr>
<tr>
<td><em>Eubacterium lentum</em></td>
<td>1(1.66%)</td>
<td>1(2.5%)</td>
</tr>
<tr>
<td><em>C. jejuni</em></td>
<td>5(6.66%)</td>
<td>3(10%)</td>
</tr>
<tr>
<td><em>C. coli</em></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Mixed infection</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>C. jejuni</em> +<em>C. perfringens</em></td>
<td>8(13.33%)</td>
<td>1(2.5%)</td>
</tr>
<tr>
<td><em>C. coli</em> +<em>C. perfringens</em></td>
<td>1(1.66%)</td>
<td>0</td>
</tr>
<tr>
<td><em>C. jejuni</em> +<em>C. sordellii</em></td>
<td>2(3.33%)</td>
<td>0</td>
</tr>
<tr>
<td><em>C. jejuni</em> +<em>Peptococcus</em> species</td>
<td>2(3.33%)</td>
<td>0</td>
</tr>
<tr>
<td><em>C. jejuni</em> +<em>Eubacterium</em> lentum</td>
<td>2(3.33%)</td>
<td>0</td>
</tr>
<tr>
<td><em>C. coli</em> +<em>C. sporogenes</em></td>
<td>2(3.33%)</td>
<td>0</td>
</tr>
<tr>
<td><em>C. coli</em> +<em>Peptococcus</em> species</td>
<td>1(1.66%)</td>
<td>1(2.5%)</td>
</tr>
</tbody>
</table>
Legends of Figures

Fig.1: Post mortem of Guinea pig Injected by *Clostridium sordellii* showing congestion in small intestine colon and liver.

Fig.2: Small intestine of GP infected by *Clostridium sordellii* showing Diffused mononuclear cell infiltration and mucosa appeared destructed and necrotic. (H&E X 40).

Fig. 3: Colon of GP infected by *clostridium sordellii* showing diffused mononuclear cells infiltration with destructed and necrotic mucosa (H&E X 400).

Fig.4: Livers of guinea pig infected by *clostridium sordellii* showing mononuclear cells infiltration scattered throughout portal areas. (H&E X 40)

Fig.5: Livers of guinea pig infected by *clostridium sordellii* showing sporadic necrobiotics changes.(H&E X 400)

Fig.6: The double zone of heamolysis of *Clostridium Perfringens* micro-organism colonies on blood agar plate.

Fig.7: The dermonecrotic reaction of *Clostridium Perfringens* toxins and typing on guinea pig showing the pathogenic action of the different toxins of the agent.

Fig.8: Post mortem examination of guinea pigs dead after 5-days from inoculation by *Campylobacter coli* showing liver abscesses and moderate intestinal congestion.

Fig.9: Post mortem examination of guinea pigs dead after 48-hours from inoculation by *Campylobacter Jejuni* showing severs intestinal congestion and hepatitis.

Fig.10: Liver of Guinea pigs inoculated with *Campylobacter coli* dead After 5-days showing hyperplasia in the bile duct (arrow) together with mononuclear cells infiltration (m) and vasculitis (v) in the portal area. [H&E X 200].

Fig.11: Liver of Guinea pigs inoculated with *Campylobacter coli* dead After 5-day showing focal areas of coagulative necrosed hepatocytes replaced by mononuclear cells in filtration (arrows). [H&E X 100].

Fig.12: Liver of Guinea pigs inoculated with *Campylobacter coli* showing abscess formation with central suppuration (s) surrounded with pyogenic membrane that consisting of dilated blood vessel, leukocytic infiltration and fibroblastic proliferation(arrows) [H&E X 4].

Fig.12a: Liver of Guinea pigs inoculated with *Campylobacter coli* showing higher power of the abscess. [H & E x 100].
**Fig.13:** Small intestine of Guinea pigs inoculated with *Campylobacter coli* showing necrosed mucosa (arrow) [H&E X 200].

**Fig.13a:** Small intestine of Guinea pigs inoculated with *Campylobacter coli* showing submucosal congestion (C) and mononuclear cells infiltration (m). (H&E X 200).

**Fig.14:** Colon of Guinea pigs inoculated with *Campylobacter coli* showing mucosal degeneration (arrow) with submucosal congestion (C) [H&E X 200].

**Fig.14a:** Colon of Guinea pigs inoculated with *Campylobacter coli* showing submucosal leucocytic infiltration (L). (H&E X 200).

**Fig.15:** Liver of Guinea pigs inoculated with *Campylobacter Jejuni* showing different hepatocytic changes in the form of; swelling, binuculeation, polymorphisms, and clear cytoplasm of some (arrow). (H&E X 400).

**Fig.16:** Duodenum of Guinea pigs inoculated with *Campylobacter Jejuni* showing mucosal hemorrhage (H), mononuclear cells infiltration (m), and degenerated glands (arrows). (H&E X 200).

**Fig.16a:** Duodenum of Guinea pigs inoculated with *Campylobacter Jejuni* showing submucosal congestion (C), edema, and mononuclear cells infiltration (arrows). [H&E X 100].

**Fig.17:** Jejunum of Guinea pigs inoculated with *Campylobacter Jejuni* showing mucosal glandular degeneration (arrow) and mononuclear cells infiltration (m). (H&E X 200).

**Fig.17a:** Jejunum of Guinea pigs inoculated with *Campylobacter Jejuni* showing submucosal depletion in the intestinal tonsils (arrow). [H&E X 100].

**Fig.18:** Cecum of Guinea pigs inoculated with *Campylobacter Jejuni* severely dilated cecal gland with destruction of its wall and desquamation of It's lining epithelium in the lumen (arrow) [H&E X 100]

**Fig.18a:** Cecum of Guinea pigs inoculated with *Campylobacter Jejuni* showing congestion (arrows) and depletion in the pyre's patches (d) (H&E X 100).
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Wen Chang, S.; Gorbach, L.; and John, B. (1978): “Neut-
المصلح العربي

تم فحص عدد 100 عينة براز جمال صغيرة تم تجميعها من بعض المزارع
الجمال بالجبيزة بتكتولوجيا لعزل وتوصيف الميكروبات اللاهوائية والكامبينوباكتر
منها عدد 60 جمال صغيرة مصابة بالاسهل و 40 طبيعية ظاهرا وكان مدى تواجد
البكتيريا اللاهوائية المسببة الاسهل للكلسترودوم بيرفريجيزي نوع B ونوع
D والكلسترودوم سوداني والكلسترودوم سوداني دايتي ولاح كوكاس والبكتيريا
فراجيلز وايفياكتريمي بنسوب 18.33% و3.33% و0.75% و5% من عقول الجمال المصابه بالأسهل على التوالي. يكون
البكتيريا اللاهوائية ظاهرة في حالة السلمية ظاهرة كانت النسب كانت 10% و4% و8% و2% و4% و2% و4%.

الكلمينوباكتر جيوجنائي 31.5% و12.5% من عقول الجمال المصابة
بالاسهل والسلمية ظاهرا وکالمينوباكتر كولبي 35% و 5% من عقول الجمال
المصابه بالإسهال والسلمية ظاهرا وتم تطبيق اختبار بعض الميكروبات المعزولة
الباحية البكلولوجية بالحقن في أربعة نحدي في البروتين وبعد الحقن أجزاء الصفة
التشريحية وفحص شرائح الوستوباكترولي الخاص الداخلي للمصابة.

تم اكتشاف رابطة بين الميكروبات اللاهوائية المعزولة والكلمينوباكتر في أحداث
الأسهل بدليل تناج الميكروبيين في بعض العينات التي تم فحصها.
ومن النتائج البكلولوجية تم أثبات أن الميكروبات اللاهوائية المعزولة و
الكلمينوباكتر متواجدة مع نتائج الفحص الوستوباكترولي وننيل بالإضافة أن
ميكروبات الكلمينوباكتر والميكروبات اللاهوائية تخرج مع براز الجمال الصغيره
السلمية والسلامية ظاهرة.

المحكمون:

أ.د. محمد أسامة الشاذلي
أ.د. جاكي عبد الحليم الجاكي