Review Article

Campylobacter Species in the Middle East

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ABSTRACT

Campylobacter is a contributing factor in many pathogen conditions. The present study aimed to review the previous findings of studies on campylobacteriosis in the Middle East region. Acute gastroenteritis is caused by Campylobacter species, known as a widespread infectious disease with worldwide disbursement. There are various reports on the incidence of Campylobacter in the Middle East indicating infection rates of 1-1.4%. Campylobacter jejuni (C. jejuni) and C. coli are the main responsible campylobacteriosis for intestinal diseases. The occurrence of human campylobacteriosis is sporadic, transmission from one person to another person is too rare and the incidence is usually higher in warm seasons and tropical environments. The presence of Campylobacter species as normal flora in stool and its contamination in poultry meat and eggs can lead to infections. Lowering the number of Campylobacter in animal carcasses can greatly decrease the risk of infection in consumers. Due to the high Campylobacter spp. antibiotic resistance, the governments are required to devise controlling programs. People in the Middle East should buy meat from stores that are under sanitary monitoring.

1. Introduction

Gastrointestinal infections are correlated with morbidity and even cause mortality at different ages12,13. Recently, many outbreaks of gastrointestinal and diarrheal illnesses in the Middle East have been reported due to numerous microbial pathogens3-6. One of the most common and central causes of infectious diarrheal illness and gastroenteritis in adults and children worldwide is campylobacteriosis7.

The Campylobacter jejuni (C. jejuni) and C. coli are the main cause of campylobacteriosis resulting in a number of gastrointestinal diseases8,9. The occurrence of this disease is usually higher in eldersies, young children under 4 years of age, and young adults who are 20-40 years old8,10,11. The transmission of this organism from one person to another person rarely occurs12. Campylobacter infection rate is reported remarkably higher than those of shigellosis and salmonellosis13. This high rate of infection needs to be investigated with regard to the epidemiology of Campylobacter infection. Red blood cells and leukocytes have been found in most of the stool samples of infected patients with slightly elevated levels of peripheral White Blood Cell (WBC) counts14,15.

Campylobacteriosis is prevalent in third-world countries, and the main sources of human infections are food and environmental contamination16. In Egypt and Iraq, Campylobacter is the second most important cause of pediatric diarrhea17,18. The present review aimed to gather information on the prevalence of campylobacteriosis in the Middle East to educate researchers and policymakers to plan effective control strategies to overcome this overwhelming infection.

2. Incidence of disease

The prevalence rate of Campylobacter infection is associated with pathogen agents, geographical restrictions, and habitual conditions. Middle Eastern countries often do not have any national surveillance programs for controlling this infection. As can be seen in Table 1, there are different reports about the prevalence of Campylobacter in these countries indicating different infection rates3,19,20.
Investigation of *Campylobacter* prevalence in the Middle East has shown that this bacterium should be considered as one of the major pathogens in inflammatory diarrheal cases. However, during 2004-2005 in Iran, the prevalence of *Campylobacter* spp. was significantly lower than the *Shigella* spp., *Salmonella* spp., and *Escherichia coli*\(^21\).

The prevalence of *Campylobacter* species among 1010 stool samples collected from patients in the northwest of Iran during 2016 confirmed 6% of contamination\(^22\). There are some reports on infection rates from Iranian researchers, such as 4.6% in Babol during 2002\(^23\), 9.6% in Shiraz during 2004\(^24\), 5.4% in Tehran during 2004-2005\(^24\), 8% in Tehran during 2007\(^25\), 12.4% in Semnan during 2007\(^2\), 10% in Hamedan during 2013-2014\(^26\), and 6.4% in Zanjani during 2013-2015\(^27\).

The prevalence of *Campylobacter* species in Jeddah, Saudi Arabia, was identified as 4.5%\(^28\). Another researcher from Saudi Arabia during 2012 reported that the prevalence of *Campylobacter* spp. in stool samples of patients in Makkah and Jeddah hospitals was just 1.7%\(^29\). The prevalence of *Campylobacter* species among 2130 stool samples collected from Menia, Fayoum, Cairo, and Qaluobya of Egypt determined the fecal contamination rate of 35% in children\(^30\).

The isolation rate of *Campylobacter* species from the stool samples of children in Baghdad, Iraq, was reported as 10% during 1991\(^31\). Another report from Basrah, Iraq, showed that *C. jejuni* was detected in 13.86% of all diarrhea cases and 31.03% of all bloody diarrhea cases\(^17\). As mentioned, *Campylobacter* was the second most frequent bacterial isolate in children with diarrhea, and the second most common cause of bloody diarrhea in Basrah, Iraq\(^17\). The *C. jejuni* was found to be the second most common isolate with a rate of 8.3% in Turkey during 1997\(^32\). Other Turkish researchers reported the prevalence of campylobacteriosis in their studies as 1.43% in Kayseri during 2002-2003\(^33\) and 4.2% in Istanbul during 2013-2015\(^34\).

### Table 1. Prevalence of *Campylobacter* species in meats of animals and human samples in the Middle East

<table>
<thead>
<tr>
<th>Country</th>
<th>City or state</th>
<th>Year</th>
<th>H-A*</th>
<th>Age</th>
<th>Incidence rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iran</td>
<td>Shiraz</td>
<td>2004</td>
<td>H</td>
<td>All ages</td>
<td>9.6%</td>
</tr>
<tr>
<td></td>
<td>Zanjan</td>
<td>2013-2014</td>
<td>H</td>
<td>Adult</td>
<td>6.4%</td>
</tr>
<tr>
<td></td>
<td>Tehran</td>
<td>2004-2005</td>
<td>H</td>
<td>All ages</td>
<td>5.4%</td>
</tr>
<tr>
<td></td>
<td>Hamedan</td>
<td>2013-2014</td>
<td>H</td>
<td>Child</td>
<td>10%</td>
</tr>
<tr>
<td></td>
<td>Ardabil</td>
<td>2016</td>
<td>H</td>
<td>Adult</td>
<td>6%</td>
</tr>
<tr>
<td></td>
<td>Babol</td>
<td>2002</td>
<td>H</td>
<td>Child</td>
<td>4.6%</td>
</tr>
<tr>
<td></td>
<td>Semnan</td>
<td>2007</td>
<td>H</td>
<td>Child</td>
<td>12.4%</td>
</tr>
<tr>
<td></td>
<td>Mashhad</td>
<td>2005</td>
<td>A</td>
<td>-</td>
<td>76%</td>
</tr>
<tr>
<td></td>
<td>Esfahan</td>
<td>2006-2008</td>
<td>A</td>
<td>-</td>
<td>47.1%</td>
</tr>
<tr>
<td>Egypt</td>
<td>Cairo</td>
<td>1993</td>
<td>H</td>
<td>Child</td>
<td>8%</td>
</tr>
<tr>
<td></td>
<td>Menia, Fayoum, Cairo, Qaluobya</td>
<td>2015</td>
<td>H</td>
<td>Child</td>
<td>35%</td>
</tr>
<tr>
<td></td>
<td>Qena</td>
<td>2014-2015</td>
<td>A</td>
<td>-</td>
<td>24.6%</td>
</tr>
<tr>
<td></td>
<td>Beheira</td>
<td>1995-1998</td>
<td>H</td>
<td>Child</td>
<td>13%</td>
</tr>
<tr>
<td>Turkey</td>
<td>Istanbul</td>
<td>2013-2015</td>
<td>H</td>
<td>All ages</td>
<td>4.2%</td>
</tr>
<tr>
<td></td>
<td>Ankara</td>
<td>1993-1994</td>
<td>H</td>
<td>Child</td>
<td>8.3%</td>
</tr>
<tr>
<td></td>
<td>Kayseri</td>
<td>2002-2003</td>
<td>H</td>
<td>All ages</td>
<td>1.43%</td>
</tr>
<tr>
<td>Iraq</td>
<td>Basrah</td>
<td>2000-2001</td>
<td>H</td>
<td>Child</td>
<td>13.86%</td>
</tr>
<tr>
<td></td>
<td>Baghdad</td>
<td>1991</td>
<td>H</td>
<td>Child</td>
<td>10%</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>Makkah, Jeddah</td>
<td>2012</td>
<td>H</td>
<td>All ages</td>
<td>1.7%</td>
</tr>
<tr>
<td></td>
<td>Jeddah</td>
<td>1989-1990</td>
<td>H</td>
<td>All ages</td>
<td>4.5%</td>
</tr>
</tbody>
</table>

*H-A: Human-Animal*

### 2.1. Seasonal distribution

The incidence of campylobacteriosis is higher in warm seasons and hot weather. The prevalence of this organism in the south of Iran (9.6% in Shiraz) in warm weather was higher than in the north of this country with cold weather (6.4% in Zanjani and 6% in Ardabil)\(^22,24,27\). The obtained result of a survey on diarrheal cases in Cairo, Egypt, was indicative of a higher rate of prevalence in young children during the rainy seasons\(^35\). Regardless of these reports, a study conducted in Turkey did not support the relationship between the distribution of *Campylobacter* spp. and weather changes\(^36\).

### 3. Detection of *Campylobacter* species

There have been successfully developed laboratory methods for examining the diversity of *Campylobacter* enteritis, such as Polymerase Chain Reaction (PCR), Genotyping methods (ribotyping and pulsed-field gel electrophoresis), ELISA for detecting DNA, antigens in stool samples, and also specific culturing of the organism from fecal specimens\(^37-42\). Rapid detection of *Campylobacter* species is extremely crucial in ensuring food and water safety. The ability of PCR to amplify minute amounts of specific microbial DNA sequences has made it a powerful molecular tool\(^43,44\). It has been reported that multiplex PCR diagnostic tools are fast, inexpensive, and sensitive for *Campylobacter* species\(^45\). Multiplex PCR is one of the possible and trustworthy molecular methods for indicating the prevalence of bacterial diseases, especially *Campylobacter* infection in a single sample\(^44,45\). The PCR assay offers an effective alternative to traditional biochemical typing methods for the identification and differentiation of *C. jejuni*, *C. coli*, *C. lari*, *C. upsaliensis*, and *C. fetus* subspecies fetus\(^46\). There are other possible methods for the detection of *Campylobacter* infection, such as specific culture under specific
conditions\textsuperscript{20,47}. Currently, fecal lactoferrin has become one of the important factors for investigating the fecal samples suspected of bacterial infections\textsuperscript{27}. Acute bacterial infections of the gastrointestinal tract or high severity of bowel diseases can induce increased levels of lactoferrin\textsuperscript{48}. It has been reported that the fecal lactoferrin values were higher in patients with Campylobacter (10.32 μg/g) and Salmonella (11.17 μg/g)\textsuperscript{49}.

4. Distribution of Campylobacter species

Most recent reports have determined C. jejuni and C. coli are the main causes of campylobacteriosis in intestinal diseases\textsuperscript{9}. In addition, C. fetus is known as an opportunistic pathogen in humans\textsuperscript{30}. A study conducted on food and stool samples collected from different areas of Tehran, Iran, indicated that 44% of all isolates were C. jejuni, 22% C. coli, and 33% as other Campylobacter strains\textsuperscript{51}. In another study in Iran, the prevalence rate of C. jejuni was estimated at 75%, compared to 25% C. Coli\textsuperscript{9}. During 2006 in Ankara, Turkey, the distribution of Campylobacter strains was reported as 70.1% C. jejuni, 21.1% C. coli, and 8.6% C. lari\textsuperscript{52}. In a study conducted in Istanbul, Turkey, in 2009, the distribution of Campylobacter strains was indicated as 56.5% C. jejuni, 33.9% C. coli, and 9.6% C. lari\textsuperscript{53}. The highest isolation of Campylobacter spp. was found to be 81% C. jejuni, and 18% C. coli in the Abu Homos district of Beheira Governorate in Egypt\textsuperscript{18}.

The C. jejuni account for most human Campylobacter isolates. There are two antigenic typing schemes for C. jejuni; the first one is Lior scheme with 108 serotypes\textsuperscript{54} and the second one is Penner scheme with >60 serotypes\textsuperscript{55}. The antigen in Lior typing scheme is heat-labile that is not truly known. The antigens in the Penner typing scheme are capsule contained lipopolysaccharide (LPS) and lipooligosaccharide (LOS)\textsuperscript{56}. However, protective antigens are not properly determined and defined.

4.1. Polymicrobial infections involving Campylobacter

It has been reported that C. jejuni antibodies have cross-reactions with Shigella sonnei, Neisseria meningitides, Shigella flexneri, and Escherichia coli plus C. rectus and C. coli\textsuperscript{57}. The flagellar proteins of C. jejuni could show signs of cross-reaction with antibodies produced against C. coli, Helicobacter pylori, and C. rectus\textsuperscript{58}. However, the occurrence of campylobacteriosis usually has a simultaneous infection with multiple pathogens, such as E. coli, Listeria monocytogenes, Staphylococcus aureus, Cronobacter sakazakii, Salmonella enterica, Vibrio cholera, Shigella, and Yersinia species\textsuperscript{61-65}.

5. Transmission of disease and clinical symptoms

The occurrence of human campylobacteriosis is sporadic making it difficult to trace the reasons and routes of transmission\textsuperscript{56}. The clinical symptoms of enteritis caused by Campylobacter species consist of watery, non-bloody, and non-inflammatory diarrhea which may give way to severe inflammatory diarrhea with abdominal pain and fever. However, C. jejuni typically leads to acute and self-limited signs\textsuperscript{23,57,60}. The diagnosis of Campylobacter infection is impossible with routine clinical signs and it usually has a co-infection with multiple pathogens, such as E. coli, Listeria monocytogenes, Staphylococcus aureus, Cronobacter sakazakii, Salmonella enterica, Vibrio cholera, Shigella, and Yersinia species\textsuperscript{61-65}.

From another point of view, individuals living in developed countries with exposure to Campylobacter species usually suffer from severe and bloody diarrhea\textsuperscript{66}. Another common digestive tract symptom of Campylobacter infection is abdominal pain, however, vomiting is not the usual sign. Fever, headache, asthenia, and anorexia can also be observed\textsuperscript{67-70}. The risk of developing inflammatory bowel diseases has already increased since diseases, such as ulcerative colitis and Crohn’s disease, are followed by Campylobacter diarrhea\textsuperscript{71}.

Campylobacter infections may lead to the further development of autoimmune diseases, including reactive arthritis\textsuperscript{72} and neurological illness (Guillain-Barre syndrome and its variant, Miller Fisher syndrome). Guillain-Barre’ Syndrome is an acute, symmetric, and ascending paralysis that is estimated to occur 30 times for every 100,000 Campylobacter cases and the fatality ratio is almost 10%\textsuperscript{73,74}.

5.1. Age and gender of patients

A current study on 1010 stool samples of adult patients of different ages and both genders concluded that age and gender did not have a significant effect on the prevalence of campylobacteriosis\textsuperscript{52}. However, the occurrence of this disease was usually higher in elderly individuals above 75 years old, young children under 4 years of age, and young adults who are 20-40 years old\textsuperscript{2,10,11}.

Children experience a progressive increase in all isotypes of Campylobacter-specific serum antibodies in the first two years of life, followed by a continued increase in IgA titers, indicative of frequent exposure to the organism, and improved mucosal immunity\textsuperscript{75}. The highest isolation of Campylobacter was reported in children of 6-12 months with diarrhea in the Abu Homos district of Beheira Governorate in Egypt\textsuperscript{10}.

During 2010-2011, Girgis et al. reported that gender is not an effective factor in the prevalence of this infection in Egypt but younger patients had more related infections to Campylobacter species\textsuperscript{76}. In a study in the south of Iran, it was determined that the prevalence of C. jejuni in females was significantly higher than males, and also the highest contamination rate was observed in younger patients aged 11-15 years old\textsuperscript{24}. The highest isolation rate of Campylobacter spp. in Turkey during 1997 was indicated in the range of 6-14 years\textsuperscript{32}. In a study in Basrah, Iraq, the frequency of C. jejuni infection was
6. Animal sources of Campylobacter

Environmental contamination of Campylobacter species can be caused by domestic animals and poultry (as natural reservoirs of Campylobacter species)78. In another study, the prevalence of Campylobacter species was investigated in different animal species during 2002-2012. The findings indicated that chickens were the most common reservoir/source of Campylobacter infection with an average contamination rate of 70.9%, whereas cattle were determined as the second most common source with a rate of 19.3%, dogs and pigs were other sources with the rates of 8.6% and 1.2%, respectively.79 Furthermore, some reports have found this pathogen as normal flora of other animals, such as sheep carcases at abattoir80,81. The isolation of thermophilic Campylobacter spp. was presented as 11.1%, 21.6%, and 50.4% for beef, mutton, and chicken samples, respectively, during 2009 in Istanbul, Turkey. In another study in Kayseri, Turkey, the isolation of C. jejuni among 6667 samples collected from humans, dogs, cattle, and chickens was reported as 1.43%, 43.50%, 31.16%, and 56%, respectively.82 The presence of Campylobacter species in stool samples and eggshells of poultry as a normal flora has also been investigated83.

6.1. Poultry as a source of Campylobacter spp.

Poultry and specifically broiler chickens have a metabolic temperature of 42°C leading to an optimal environment for Campylobacter growth84. Poultry carcases in stores that are not under sanitary monitoring and the consumption of undercooked poultry meat are important reasons for such outbreaks85. However, the transmission of infection from chickens to humans is greater owing to the high levels of chicken meat consumption86,87. In developing countries, close contact with animals, including chickens, was found to be an important risk factor for acquiring the infection88,89. A diarrheal disease survey in Cairo, Egypt, in young children indicated that the prevalence of Campylobacter spp. is highly associated with keeping fowls at home.85 The presence of Campylobacter species as normal flora in stool samples and eggs of poultry is one of the major causes that might affect the prevalence of campylobacteriosis in patients90,93. Reducing the number of Campylobacter in poultry carcases can significantly decrease the risk of infection in consumers90,91. One of the suggested approaches for controlling the Campylobacter species as normal flora in poultry is using feed additives, such as probiotics and symbiotics. It is reported that a mixture of fructooligosaccharide and a galactooligosaccharide + Bifidobacterium strain (B. longum subsp. longum PCB133) can significantly reduce the C. jejuni concentration in poultry feces92. The other common way is the use of antibiotics as growth promoters in the poultry diet; however, this pathogen is highly resistant to many antibiotics. Previously from 35 layer farms of Northern Jordan, the high resistance of this organism to ciprofloxacin (100%), tetracycline (100%), gentamicin, and amoxicillin (41%) was reported while the low resistance to nalidixic acid (21%), erythromycin (14%), and florfenicol was observed93.

A comparative study in Esfahan, Iran, reported Campylobacter persistence in different poultry meat samples. The highest rate observed in quail meat (68.4%) and 56.1%, 27.4%, and 11.7% of contamination was respectively recorded for chicken, turkey, and ostrich meats. The overall prevalence of Campylobacter spp. in the investigated samples was 47.1% (377 from 800) which contained 76.4% C. jejuni and 23.6% as C. coli.94. The prevalence of Campylobacter species among samples collected from different cities of Egypt determined that the contamination rate was 40.4% for the intestine of chicken, 37.5% for the liver of chicken. A study performed in Saudi Arabia during 2012, the 81.7% prevalence of campylobacteriosis in carcass samples of chicken was presented.95 This report was close to the report of Turkish researchers who had indicated the presence of Campylobacter species in 83% of chicken meat samples.96

6.2. Other foods and water sources, and infectious dose

Campylobacteriosis can result from the consumption of suspected food sources and daily products of animals.95 Recent studies indicated that the consumption of unpasteurized milk and ingestion of undercooked chicken was significantly associated with acquiring infection but there was no possible effect of poultry on the incidence of Campylobacter disease95,76. Gent et al. reported the outbreak of Campylobacter infection due to poor food handling in a fast food outlet located in a university that infected over 100 students96. Moreover, the obtained results of this outbreak led to the identification of 6 different Campylobacter subtypes isolated from 11 patients. In a study focused on food samples collected from different areas of Tehran, Iran, the prevalence rates of C. jejuni, C. coli, and other Campylobacter strains were reported.25 In the cities of Menia, Fayoum, Cairo, and Qalalubya, Egypt, the contamination rates of Campylobacter species in water and foods were estimated as 30%, 4.44%, 6.6%, and 13.33% in tap water, raw milk, Karish cheese, and yogurt, respectively. During 2014-2015, samples of raw milk, Karish cheese, and yogurt were collected from local markets in Qena City, Egypt, to evaluate the prevalence of Campylobacter species. The results suggested that 24.6% of samples were contaminated with Campylobacter spp. and the highest contamination rate belonged to raw milk while the lowest was observed in.
yogurt samples. Wagenaar et al. found that 23% of infected human cases with campylobacteriosis were associated with the consumption of unpasteurized milk products in Egypt.

This organism is occasionally isolated from streams, lakes, and ponds. The survival of *Campylobacter* in surface water of the Mediterranean area has been reported. A diarrheal disease survey in Cairo, Egypt, in young children indicated that the prevalence of *Campylobacter* spp. was highly associated with having an outdoor source of drinking water.

The current report has indicated the overall prevalence of *Campylobacter* in beef livers in Egypt was 26.66% in 2013, while this isolation rate was higher than the previous study conducted in Japan which reported only 5% *Campylobacter* spp. in beef livers. Ghafir et al. suggested that the high level of hepatic *Campylobacter* resistance is probably due to the moisture of the liver surface, which might protect this foodborne pathogen. Fecal carriage of *Campylobacter* by the slaughtered cows is another possible route of contaminating beef livers in an abattoir that may cause campylobacteriosis using undercooked beef livers.

Reducing the number of *Campylobacter* in poultry carcass can greatly reduce the risk of foodborne disease. Few available studies reveal the exact number of cells inducing human infections. In a kindergarten, contamination of foods at the level of 10 cells of *C. jejuni* per 100 ml caused infections.

7. The role of immunity in *Campylobacter* infection

The presence of a specific serum immunoglobulin A (IgA), IgM, and IgG in acute campylobacteriosis has been reported. IgA remained elevated for almost 1 month whereas IgM and IgG persisted for almost 3 months. Healthy individuals with occupational exposure to *C. jejuni* produced IgM without clinical symptoms.

The results of a study on antibody titers (log_{10}) of diarrhea patients in Cairo, Egypt, indicated that 28 patients with diarrhea had *C. jejuni*-reactive antibodies. Ten of these cases were culture-positive for this organism with significant titers of IgA and IgG. Among asymptomatic culture-positive patients (10 cases), 6 subjects had reactive IgM, and 7 subjects had IgG. However, IgA was not detected in asymptomatic patients. Another study indicated that the chronic carriers of *C. jejuni* could increase the levels of IgG and IgM but not IgA. The presence of antibodies in healthy populations living in areas of hyperendemicity or developing countries has also been reported.

8. Prevention and control

Various animal models have been tested with different vaccine formulations and each has shown varying levels of success. Most of the investigated vaccines are against *C. jejuni*. The limited literature concerning virulence determinants of *C. jejuni* makes it difficult to prepare effective live attenuated vaccines. Oral immunization of mice with an attenuated *Salmonella enterica* serovar Typhimurium vectoring *Campylobacter* PEB1 antigen or CjaA protein failed to protect the animals against intestinal colonization with the challenge *Campylobacter* strain even though specific serological responses were seen.

It is proposed that vaccination may be a possible control approach, as infected people mount a strong immune response. In addition, possible immunity against *Campylobacter* in the absence of acute infection in workers of slaughterhouses was reported after initial exposure. There have also been attempts to develop *Campylobacter* vaccines, whole-cell oral vaccine formulations having been tested with good results in primates and a vaccine based on whole-cell formulations and purified flagellin giving some protective immunity in the mouse model. A recombinant truncated flagellin protein (rFla-MBP) based on the conserved region of flagellin of *C. coli* VC167 strain was used as a vaccine-induced antibody response and 60% protection against *C. jejuni* in a ferret model of diarrhea.

An alternative way to decrease food contamination is reducing the load of the pathogen in its main reservoir, the broiler chickens. The control programs, including increasing biosecurity and vaccination of broiler chickens, are being explored.

9. Conclusion

The prevalence of *C. jejuni* is much more common than other *Campylobacter* spp. in the Middle East leading to the incidence of *Campylobacter* infection rated between 1 and 14%. The people who live with animals should check the health of animals regularly and not consume suspected sources of food. Individuals are recommended to buy meat, particularly chicken meat, from stores that are under sanitary monitoring. Ultimately, further investigations can show all the possible pathogens responsible for polymicrobial acute diarrheal infections in addition to *Campylobacter* species and also elucidate their mechanisms of pathogenesis.

Declarations

Competing interests

Authors declare no conflicts of interest.

Authors’ contribution

Authors participated equally in preparing and writing the manuscript.

Availability of data and materials

All findings and related data of the present study are prepared for publishing in the submitted journal.

References


