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Campylobacteriosis

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INTRODUCTION

Campylobacteriosis has been identified as an important food-borne zoonotic disease around the world, and it is caused by members of the genus *Campylobacter* which is ubiquitous bacteria (Iannino et al. 2019). The *Campylobacter* genus belongs to the family Campylobacteraceae and the genus consists of 39 species with 16 subspecies (Facciolà et al. 2017; Hlashwayo et al. 2020). The majority of infections in humans are caused by the thermophilic species *Campylobacter (C.) jejuni*, followed by *C. coli*, *C. lari*, and *C. upsaliensis*. (Leonard et al. 2011; Authority et al. 2013). The pathogen exists as normal flora in the intestinal tracts of many animal and bird species without causing any serious disease symptoms (Takamiya et al. 2011). There are several ways of disease transmission to humans, including consumption of undercooked meat or contaminated meat especially poultry, dairy product, contaminated water, as well as contact with diseased animals or feces (Zhang et al. 2018). In the modern society, *Campylobacter* has become the most common bacterial cause of human foodborne gastroenteritis worldwide (Acheson and Allos 2001). Along with gastrointestinal infections, it also causes various human systemic infections, including bloodstream infections, endocarditis, septic thrombophlebitis, pneumonia, neonatal sepsis, acute appendicitis and acute colitis of inflammatory bowel disease (Morishita et al. 2013; Alnimr 2014; Lagler et al. 2016). Other post-infection complications include severe demyelinating neuropathy, Guillain-Barre syndrome (GBS) and Miller-Fisher syndrome (MFS) (Scallan et al. 2015; Skarp 2016). *Campylobacter* infection are often common in particular age group, such as children under four years of age and people over 75 years (Levesque et al. 2013). Patients with hemoglobinopathy, inflammatory bowel illness and individual with impaired immune systems are at higher risk of getting *Campylobacter* infection (Kennedy et al. 2004).

Underdiagnoses and underreporting of the disease have been linked to difficulties in isolating and growing *Campylobacter* species, their high cost and the lack of a legal requirement for its monitoring in foods. As a result, information about outbreaks or the contamination of animal products does not accurately reflect the state of the nation (Mendonca et al. 2015).
Most of the time, *Campylobacter* infection is self-limiting and requires supportive therapy. However, antimicrobials may be needed in patients with severe, persistent and extraintestinal campylobacteriosis and in immune-compromised patients (Kaakoush et al. 2015). The presence of this pathogen in animal products and other foods that might contain these agents can pose a risk to human health (Facciolà et al. 2017).

History

Since 1909, it has been known that the pathogenic bacterium can result in animal abortion, especially in bovines and ovines. Two veterinary surgeons, McFadyean and Stockman (1913), studied epizootic abortion in sheep and discovered an unidentified bacterium that was frequently isolated from aborted fetuses and that resembles a *Vibrio (V.) fetus*. Initially, the classification of bacteria was based on morphology. Vincent (1947) isolated *V. fetus* from three pregnant women who were taken to a hospital with an unidentified fever. On inspection of the placenta, large necrotic and inflammatory regions were visible. Two of the three women had abortions, and the illness lasted for about four weeks. Sebald and Véron (1963) renamed this organism as *Campylobacter*. Only the blood of bacteremic individuals could be used for diagnosis of infection. Dekeyser et al. (1972) in collaboration with Butzler and his group at the St. Peter University Hospital, successfully isolated *Campylobacter* from feces. In 1980, Penner and Hennessy introduced serotyping methods that still serve as the foundation for strain typing today with the help of genotyping, phage typing, and biotyping. Until the middle of the 1980s, *C. jejuni* was known to be the most frequent cause of bacterial enterocolitis in humans. These days, *Campylobacter* has become a serious issue for both industrialized and developing nations' public health (Butzler 2004).

Etiology

Campylobacter species is gram negative, spiral rods or spirally curled in shape, ranging in size from 0.2–0.8 μm broad and 0.5–6.0 μm long and even cells can change to coccoid or spherical forms in reaction to stress or harmful conditions (Shane 1992; Kassem et al. 2017). They occur as

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normal inhabitants in the gastrointestinal tracts of a number of domestic animals and birds such as commercial broiler chickens (Aydin et al. 2001; Zimmer et al. 2003). There are at least 30 recognized species and subspecies in the genus *Campylobacter* (On 2013). They belong to the Campylobacteraceae family, a broad but phylogenetically separate group of Gram-negative bacteria that is classified in the Proteobacteria's epsilon division (Lastovica et al. 2014). Additionally, the family Campylobacteracea includes the genera *Arcobacter* and *Sulfurospirillum* (Fitzgerald and Nachamkin 2015). Thermophilic *Campylobacter* spp. (*C. jejuni*, *C. lari* and *C. coli*,) are recognized as being clinically significant as these are the primary causal agents of human campylobacteriosis (Hermans et al. 2012). All species lack spores and have a single bipolar flagellum that mediates a distinctive corkscrew-like or darting movement that can be seen in dark fields of illumination (Byrd et al. 2007). They require a microaerobic environment for optimal growth and are slow-growing, picky organisms that grow best at 37°C to 42°C on artificial media (Lastovica et al. 2014). Since the bacteria are often unable to ferment carbohydrates, the breakdown of amino acids serves as the main source of energy for the organism, and they are oxidase and catalase positive, indole negative and reduce selenite (Simon 1994; Kelly 2001).

Incidence and Distribution

The epidemiology of campylobacteriosis is considered complex. The rate of accurate incidences of *Campylobacter* epidemic is largely unknown. This is because campylobacteriosis outbreaks are rarely reported, and there are difficulties with diagnosis and variations in surveillance (Hansson et al. 2018). Until now, foodborne campylobactereiosis has been seen to increase in the health issue in both undeveloped and developing countries. Each year, there are more positive cases in North America, Europe, Australia, and New Zealand (Man 2011; Kaakoush et al. 2015). The frequency of *Campylobacter* infection is also high throughout Asia, Africa, and the Middle East despite the absence of adequate epidemiological data (Butzler 2004). Some studies report that in developed countries, the prevalence of *Campylobacter* causes gastroenteritis with a frequency of two to seven times higher than *Escherichia coli* O157:H7, *Shigella spp*., or even *Salmonella spp.* (Acheson and Allos 2001; Authority et al. 2014). Although most *Campylobacter* outbreaks are from waterborne or foodborne illness and most of these outbreaks are usually from animals (Frost et al. 2002), At least 80% of foodborne *Campylobacter* outbreaks were carried on by poultry products (Hsieh and Sulaiman 2018). Despite the fact that most *Campylobacter* occurrences in low-income countries originate from natural sources like rivers and streams where large number of people depend on these water bodies as their main source of drinking water (Clark et al. 2003; Platts-Mills and Kosek 2014). In addition, high-income nations including Norway (Jakopanec et al. 2008), Finland (Kuusi et al. 2004), New Zealand (Bartholomew et al. 2014), Canada (Clark et al. 2003), and Denmark have indicated that water supplies have been linked to *Campylobacter* epidemics (Kuhn et al. 2017). There was seasonal variation in the prevalence of *Campylobacter*, the number increases to its peak in warm weather; while with dropping temperatures, the numbers decrease (Hermans et al. 2012; Sahin et al. 2015). Although the specific cause of this seasonal change is undetermined, it is hypothesized that a rise in fly population and fly-mediated transmission increase the number of *Campylobacter* in warm months (Hald et al. 2004).

Transmission Routes of Campylobacter Infection

Most of the animals, including poultry, cattle, sheep, dogs, cats, ostriches, pigs, and European blackbirds carry several *Campylobacter* species in their intestinal tracts (Dearlove et al. 2016). These animals' feces contain these bacteria, that are spread into the surrounding (Goni et al. 2017). Under typical ambient conditions, *Campylobacters* typically cannot proliferate in feed, litter, or water because they are sensitive to oxygen and temperature. (Sahin et al. 2002; Newell and Fearnley 2003). Contaminated litter by *Campylobacter* may contribute to the persistence of the bacteria in the farm environments (Line 2002; Sahin et al. 2002). It can be spread mechanically by rodents and insects like houseflies (Sahin et al. 2015). Movement of people and equipment between farms may spread *Campylobacter*, which has been implicated in several studies as a potential risk factor (Hansson et al. 2007; Sahin et al. 2015).

Consumption of contaminated or undercooked meat (particularly poultry), contaminated raw milk, and other handling of various products of animal origin are the most typical ways that *Campylobacter* spp. spread to humans (Bronowski et al. 2014; Kaakoush et al. 2015). Additionally, direct interaction between people and animals, such as pets, was reported as a possible route of infection transmission (Westermarck 2016), and people who work with cattle are at a significant risk of contracting *Campylobacter* because these animals are frequently colonized by it (Hansson et al. 2018). Furthermore, drinking water has been implicated as a major reservoir for the existence of *Campylobacter*; the most typical *Campylobacter* species found in surface water is *C. jejuni* (Hokajärvi et al. 2013). Finally, raw vegetables were identified to be the second major risk factor after consuming contaminated chicken meat; it can become contaminated with *Campylobacter* directly at farms, or during transport, and processing in food manufacturers (Kwan et al. 2008; Bronowski et al. 2014). Finally, transmission of campylobacteriosis can occur between humans either through fecal-oral transmission or, in males, by homosexual contact (Kuhn et al. 2021). Congenital transmission has been described but is a rare neonatal issue (Shane 2019). Fig. 1 presents a graphic overview of the transmission of *Campylobacter* in animals and man.

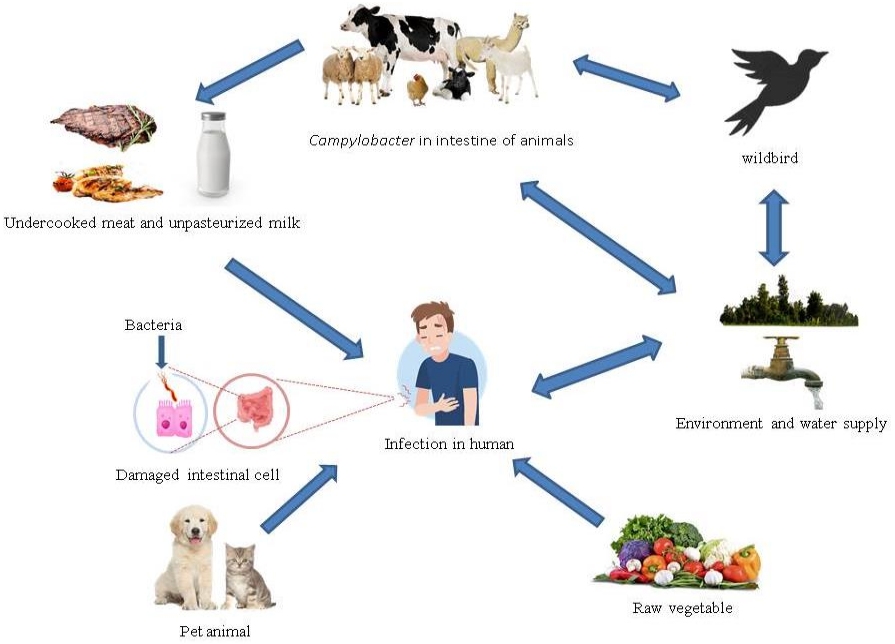

Fig. 1: Overview of pathways of *Campylobacter* transmission in both Human and animals.

Infections in Animals

Clinical Sings

Many different animal species can carry *Campylobacter* spp. asymptomatically (Komba et al. 2013). According to certain research, experimentally inoculating young chickens and turkey poults with *Campylobacter* might result in clinical illnesses such bloody or mucoid diarrhea, weight loss, or even mortality (Lam et al. 1992; Humphrey et al. 2014; Zhang and Sahin 2020). The severity of enterocolitis that results from infection depends on both the pathogenicity of the strain and the host's immune system (Sanyal et al. 1984; Soerjadi-Liem 1984). Infection by *Campylobacter* in dogs and cats presents as acute enterocolitis and is characterized by mild to watery diarrhea, dehydration, lethargy, anorexia, vomiting, pyrexia, and abdominal pain (Marks et al. 2011; Sykes and Mark 2013). Cattle, sheep, and swine are also colonized by *Campylobacter* spp., and clinical symptoms in young animals could be more severe than those in adults (Mendonca et al. 2015). *C. jejuni* can cause bovine mastitis and enteritis in calves, whereas *C. fetus* causes abortion and infertility in cattle (Stanley and Jones 2003). Hamsters, neonatal mice, rabbits, and ferrets are also susceptible to intestinal colonization with *C. jejuni*, which may result in persistent diarrhea (Bell and Manning 1991).

Lesions

Enterocolitis with varying degrees of severity is the hallmark of mammalian campylobacteriosis (Shane and Montrose 1985). Gross lesions include catarrhal to severe hemorrhagic enteritis. In newly hatched chicks, there is an accumulation of fluid, gas, or excess mucus, as well as intestinal distention and watery/foamy material in the ceca is apparent (Sanyal et al. 1984). Experimental research revealed that a novel *Campylobacter* species, *C. hepaticus*, which causes the spotted liver disease in chickens, is known for causing widespread, 1-2 mm grey-white lesions in the liver (Crawshaw et al. 2015). Microscopic findings include villous atrophy, mononuclear infiltration of the submucosa, and a mild edema of the lamina propria and submucosa of the intestines, mostly in ceca (Stern et al. 1988). In sheep, gross fetal lesions include fibrinous peritonitis, hepatomegaly, perforated liver with internal bleeding, and many dispersed, round-to-targetoid areas of hepatic necrosis between 1 mm and 2 cm in diameter (Jubb et al. 2012). In cattle, abortion can occur at any gestational age, with lesions characterized by endometritis, placentitis, fetal serositis, hepatitis, and pneumonia (Michi et al. 2016).

Diagnosis

The diagnosis is made using cultural isolation and detection of the causative agent, biochemical characterization, recent molecular techniques and tools (Sails et al. 2003). In domestic animals and wild species (crows, mice, and chimpanzees), samples were obtained from feces (rectal samples if possible). In the case of avian species, samples were taken using cloacal swabs, poultry house droppings, or the caecal contents of dead birds (Komba et al. 2013). After a pre-enrichment in a liquid medium, plate positive broth on selective media (blood-based and charcoal-based), serve to

remove toxic oxygen derivatives (Hsieh and Sulaiman 2018). Many media have been used for culturing *Campylobacter* species. Under laboratory circumstances, the bacteria are fastidious and slow-growing, needing a microaerobic environment (5% O_2, 10% CO_2, 85% N_2) at a higher temperature (42°C), with no growth being seen at temperatures below 31°C (Sahin et al. 2003). The most popular diagnostic methods for finding and identifying *Campylobacter* spp. in most laboratories are the enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction (PCR) (Lilja and Hänninen 2001).

Treatment

Fluid replacement and antispasmodics are typically sufficient. In the case of diarrhea, pets and other domestic animals should receive symptomatic treatment. In case of pyrexia or prolonged and serious circumstances, antibiotic medication may be necessary (George 1994). In cats and dogs, erythromycin (10 mg/kg in cats, 10–15 mg/kg in dogs orally every eight hours) or azithromycin (5–10 mg/kg in dogs and cats orally every twenty-four hours for five to twenty-one days) have been suggested as treatments (Marks et al. 2011). Cefoxitin, erythromycin, or enrofloxacin have all been effective treatments for dogs with cholangiohepatitis/cholecystitis and bacteremia associated with *C. jejuni* (Sykes and Mark 2013).

Prevention

Strict biosecurity measures are the sole action that has been shown to be successful in preventing the entry of *Campylobacter* spp. to farms and herds (Newell and Fearnley 2003). These include: cleaning and sanitizing the entire house, including every piece of equipment between successive cycles of livestock, which will prevent horizontal transmission (George 1994). Programs to control insect (e.g., flies and cockroaches) will reduce the probability of infection (Hald et al. 2007). A set of management established to reduce the entry of unauthorized people, birds, rodents, or other animals to farms and herds (Facciolà et al. 2017). There should be proper water purification (chlorination) as it prevents the introduction of viable *Campylobacter* spp. (Guerin et al. 2007). Besides biosecurity, there is various ways used on farms for control of *Campylobacter* colonization, such as immunization, feed additives, bacteriocins, bacteriophages, and competitive exclusion (Johnson et al. 2017).

Infection in Human

Clinical Signs

The foremost symptom in foodborne campylobacteriosis is gastrointestinal illness, which manifests as an acute diarrheal disease with clinical symptoms, such as abdominal pain and fever. The diarrheal disease is usual 12-24 hours before the onset of clinical symptoms that presents a period of myalgia, pyrexia, and malaise (Mendonca et al. 2015). The symptoms are not distinct enough for the doctor to distinguish them from other bacterial infections induced on by food poisoning (Edward and David 2004). The incubation period may range from 1 to 5 days, reaching up to 10 days, based on the amount of infection and the strain of organism (George 1994; García and Cravioto 2007). Although the condition is typically self-limited and symptoms may disappear in a week, the consequences of dehydration, hepatitis, and neurological abnormalities may be seen (Akitoye et al. 2002). In addition to gastroenteritis, *Campylobacter* infections also result in reactive arthritis, irritable bowel syndrome, Guillain-Barré Syndrome (GBS), Miller Fisher Syndrome, bacteremia, septicemia, meningitis, cardiovascular complications, and abscesses (Fitzgerald and Nachamkin 2015; Kaakoush et al. 2015). Death as a result of campylobacteriosis is uncommon, but it can occur when the condition is widespread in people who are immunocompromised or in old or very young patients (Mendonca et al. 2015).

Lesions

The infection is most commonly occurred in ileum, jejunum, and colon. These lesions might include mild hyperemia, congestion, edema and the hemorrhages that ultimately leads to ulcerative and necrotic changes (George 1994). Histologically, edematous and bloody Jejunal, ileal, and colonic mucosa are present, and enlarged mesenteric lymph nodes may be seen with lymphocytic infiltration in acute cases, but polymorphonuclear leukocytes and eosinophils may accompany ulceration and abcessation (Blaser et al. 1980). Microscopic analysis of rectal biopsy samples from people who had severe *C. jejuni* enteritis revealed a mixed collection of inflammatory cells in the lamina propria, mucosal epithelial ulceration, loss of mucus, and crypt abscess in the epithelial glands (Cover and Blaser 1989).

Diagnosis

Fecal samples have been identified as preferred specimens for the detection of *Campylobacter* spp. in people who have digestive problems (Senok and Botta 2009). Isolation of *Campylobacter* spp. from the blood is confirmatory. Serological techniques have been established for the direct detection of *Campylobacter* spp. based on antigen-antibody interaction (Regnath and Ignatius 2014). For the molecular subtyping of *Campylobacter* isolates, numerous DNA-based techniques have been created. These include polymerase chain reaction (PCR), pulsed-field gel electrophoresis (PFGE), Multilocus sequence typing (MLST), random amplified polymorphic DNA (RAPD), and amplified fragment length polymorphism (Fitzgerald et al. 2001).

Treatment

Many cases of campylobacteriosis require only supportive therapy. Sometimes antibiotics are given, especially when the symptoms are severe or prolonged and in immunocompromised patients (Same and Tamma 2018). Erythromycin, clarithromycin, and azithromycin are the antimicrobial agents of choice (Mendonca et al. 2015). Antibiotic-resistant diseases, particularly those resistant to fluoroquinolones, are on the rise as a result of the overuse of antibiotic in both human and animal populations (Iovine 2013). The use of a probiotic (Lactobacillus acidophilus) along with a prebiotic (fructooligosaccharides, lactosucrose) showed positive effects on the balance of intestinal microflora, as well as an improvement in recovery from infection and immune status during the investigation of alternative antibiotics sources for the treatment of campylobacteriosis (Rastall 2004).

Prevention

Foodborne campylobacteriosis can be minimized by taking the proper precautions while handling and preparing meals from an animal origin (Butzler 2004). It's important to properly cook raw meat and poultry because *Campylobacter* is sensitive to heat and cooking the food at 70 °C will reduce the chance of getting an infection (Facciolà et al. 2017). When preparing food in the kitchen, wash hands with warm water and soap, keep work area, kitchen sink, cutting boards and tools clean (Biabana 2004), and animal products and other foods should be prepared using different cutting boards and cutlery. Untreated water and unpasteurized milk products should not be consumed (FDA 2012). Diseases prevalence can be decreased by keeping appropriate refrigeration from the time of processing chicken and red meat to the moment of preparation (George 1994). Children, pregnant women, and immunocompromised people should be kept away from sick animals and animal feces (Campagnolo et al. 2018). Those who have campylobacteriosis should practice good hygiene to avoid the illness from spreading (Hemsworth and Pizer 2006).

Conclusion

Campylobacter enteritis in humans has become increasingly well-known during the past three decades, with current isolation rates exceeding those of *Salmonella* and *Shigella* spp.; the two common gastrointestinal infections. There is a possible relationship between *Campylobacter*-related infection in both humans and domestic animals. Strategic plans for campylobacteriosis prevention and control are also essential. Veterinary and medical professionals should need to work together in order to reduce the rate of infection transmission to human from animal sources.

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