SHORT COMMUNICATION

Vibrio cholera

Caroline Thomas

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ABSTRACT

The etiological agent of cholera, a severe diarrheal disease that can occur as an epidemic or sporadic disease, is Vibrio cholera. The V. cholera O1 and O139 serogroups that cause the cholera pandemic originated in the Indian subcontinent and have since spread around the world, killing millions of people each year, mostly in poor and disadvantaged nations. *V. cholera* O1 may generate biotype-specific cholera toxin and is further characterized as classical and El Tor biotypes. The present seventh pandemic El Tor strains have been replacing the sixth pandemic strains since 1961, resulting in the classical biotype strain that generates classical CT. The major issue is the continuous emergence of Atypical El Tor *V. cholera* srains encoding classical CT. The global threat of atypical El Tor V. cholera srains encoding classical CT is real. These Atypical El Tor strains have much more severe pathophysiology than El Tor or conventional strains. V. cholera pathogenesis is a complicated process that includes the coordinated expression of many virulence-associated genes to induce illness. We still don't know everything about V. cholera's virulence profile, including the direct and indirect expression of genes involved in the parasite's survival and stress adaption in the host. Whole genome sequencing has paved the way for a better understanding of the evolution and distribution of infectious pathogens, as well as outbreak detection and pathogen surveillance for the implementation of direct infection control measures in the clinic against many infectious pathogens, including V. *cholera*, in recent years.

Key Words: *Vibrio cholera; Bacteria; Infection*

INTRODUCTION

The causal agent of cholera is Vibrio cholera, a Gram- Negative Bacteria. Cholera can be sporadic, epidemic, or endemic, and is known for producing severe diarrhea and dehydration, which can lead to death in as little as 48 hours if not treated [1]. V. cholera is a small intestine extracellular pathogen that has been identified worldwide, primarily in poorer nations where transmission is exacerbated by inadequate sanitation and poverty, and is responsible for millions of fatalities each year. The World Health Organization (WHO) recorded 1,227,391 cholera cases and 5654 fatalities globally in 2017, according to the WHO's weekly epidemiological report (World Health Organization, 2019). Passage through the human host has been proven to have a crucial role in cholera outbreaks by producing a super infectious condition in cells, which are then expelled into the natural environment. V. cholera is a naturally occurring pathogen that can be found in the aquatic environment or in contaminated food, particularly shellfish and crustaceans. V. cholera, on the other hand, can be difficult to identify since it comes in two forms: viable and culturable (V & C) and Viable but Non-Culturable (VBNC). The Cholera Toxin (CT), which causes profuse rice-watery diarrhea, and the Toxin-Co Regulated Pilus (TCP), a type IV pilus that mediates adhesion, micro colony formation, and intestinal colonization, are two primary virulence factors linked to the development of cholera symptoms. Surprisingly, environmental influences have a big impact on the development of these virulence factors (CT and TCP).

Among the more than 200 serogroups identified, isolates designated as O1 and O139 generate cholera toxin and have been linked to cholera outbreaks. V. cholera isolates that do not agglutinate with O1 or O139 antiserum are known as non-O1 and non-O139 Vibrio's, respectively. These isolates are typically non-toxigenic, and they're thought to be the cause of rare but severe human gastroenteritis [2,3]. Based on different genetic, biochemical, and behavioral features, the V. cholera O1 serogroup is divided into two biotypes: Classical and El Tor. The hemolytic abilities, agglutination interactions with erythrocytes, and polymyxin B resistance phenotypes exhibited in El Tor strains are the main differences between these two biotypes [4,5]. TcpA, a gene that aids in the creation and control of a toxin co-regulated pilus, is also utilized to differentiate biotypes (El Tor or classical) based on sequence deletions in the classical allele. In 1992, a novel *V. cholera* strain dubbed O139 Bengal, a non-O1 Vibrio producing cholera epidemics, first arose in Asia, initially detected in Tamil Nadu, India. This strain is classed as a novel serogroup and is phenotypically and genetically similar to El Tor with some traits from classic strains. In August 1996, V. cholera, also known as O139 Calcutta, resurfaced as a cholera causative agent in Calcutta. These O139 Calcutta strains are genetically identical to O139 Bengal, but they have an additional CTX element from the El Tor type (CTX ET and CTX calc) and a distinct restriction-endonuclease map.

Between 1899 and 1923, the classical biotype of V. cholera was responsible for the sixth pandemic. There were no further recorded cholera pandemics until 1960, with the exception of a few isolated outbreaks. The El Tor strain produced the seventh cholera pandemic, which began in Sulawesi, Indonesia in 1961, spread to Africa in 1970, and then to Latin America in 1991, with huge outbreaks in Lima, Peru, the first Latin American country to record cholera. A tiny epidemic of cholera was recorded for the first time in Brazil's history at Paranagua Bay, Parana State (south of the country) in 1999. In August 2000, South Africa witnessed one of the deadliest cholera epidemics in its history, with 114,000 illnesses and 260 recorded deaths by the end of January 2002. (KNZ Department of Health media release, 7 February 2002).

The 71st World Health Assembly passed 'Ending Cholera: A Global Roadmap to 2030' in May 2018. By 2030, the Global Task Force on Cholera Control will have devised a new global roadmap (plan) to cut cholera mortality by 90% and eliminate the disease in as many as 20 nations. Early identification and prompt action to control epidemics, a focused multi-sectoral strategy to avoid cholera recurrence, and an efficient coordination framework for technical assistance, advocacy, resource mobilization, and cooperation at the local and global levels are among the methods. The risk of imported cases or occasional outbreaks in industrialized nations, as well as the rapid spread of epidemics in underdeveloped countries, highlight the necessity of knowing the virulence and pathology of current V. cholera strains.

CONCLUSION

Infections with bacteria and the formation of new strains in any species have always been difficult. V. cholera, like other bacteria, may be found all over the world and continues to evolve over time. Understanding the pathophysiology of V. cholera is difficult due to its dual distinctive trait of being able to be sustained in both a less infectious and infectious form, as well as modifying or gaining numerous virulence genes or mobile genetic elements. Increased poverty, poor water quality, overuse of antibiotics, and climatic changes are all encouraging more virulence and antibiotic resistance genetic exchange,

Editorial Office, Journal of Clinical Microbiology and Infectious Disease, Windsor, United Kingdom

Correspondence: Caroline Thomas, Editorial Office, Journal of Clinical Biology and Infectious Disease, Windsor, United Kingdom, e-mail clinicalmicro@scienceresearchpub.org

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which has previously been linked to the evolution of new variant strains that originated and spread among underdeveloped and developing Asian and African countries. However, with modernized worldwide travel and Asian and African nations serving as global commercial hubs, developed countries are at a greater danger of cholera spread if preventative steps are not taken. There has been no clear plan to cholera elimination since the early outbreaks in 1861. Various vaccinations, medicines, and intervention methods (hand washing, safe toileting, and a variety of other measures) have been developed and implemented across the world, but they are insufficient to completely eliminate the illness. Despite several studies showing the relationship between environmental factors and the organism or the acquisition of virulent phages and antimicrobial resistant mobile genetic elements in environmental strains, the emergence of new pathogenic Atypical El Tor strains and factors involved in natural selection of V. cholera strains remain unknown. It's worth noting that little is known internationally regarding the relationship between environmental Atypical El Tor V. cholera strains, their genetic virulence traits, and antibiotic resistance capabilities. To further understand the distribution, development, and Amr profile of these toxigenic and non-toxigenic environmental Atypical variations of V. cholera strains, more research is needed.

The development of molecular tools such as WGS and large-scale comparative genomics has certainly aided in the identification, categorization, and understanding of the epidemiology of disease-causing pathogenic microorganisms. As a result, WGS must be used to better understand present variation and follow genomic evolution of V. Cholera Atypical El Tor variants. This would allow public health officials to detect and prevent the spread of closely related pathogenic isolates internationally by identifying patterns of disease dissemination within an area and tracking their origins while comparing strains throughout the world. In the end, this might offer enough detail to allow for the creation of effective cholera treatment and prevention throughout the world.

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