The Dutch Cholera Epidemic of 1832

as Seen through 19th Century Medical Publications

Antoinette van der Kuyl

Eburon
Utrecht 2021

ISBN 978-94-6301-378-9 (paperback)
ISBN 978-94-6301-388-8 (ebook)

Eburon Academic Publishers, Utrecht, The Netherlands
www.eburon.nl

Cover image: Nicolaas Johannes Roosenboom (1805-1880), *Several people near fishing boats on the beach at Scheveningen*. (Museum am Herrenberg, Bad Bentheim, Germany)
Cover design: Textcetera, The Hague

Contents

CHAPTER I

Why Write about Cholera?

The second Asiatic cholera pandemic, which formally lasted from 1829-1849, shocked the West, as it was generally believed that the era of deadly epidemic diseases was long over in Europe. At first, it was assumed that the disease would be limited to Asia, as it had been during the first cholera pandemic (1817-1823). But when the *cholera morbus* reached Russia in 1830, European countries started to prepare themselves for the inevitable, a disease with, apparently, a high rate of fatalities, known to the English-speaking world as either Asiatic, spasmodic, epidemic, Indian, malignant, or blue cholera. The cholera disaster of the 1830s came as a severe blow to a continent that was trying to emerge from the revolutionary era of the late 18th century that ended in the Napoleonic wars. Governments were fighting poverty, disease, perceived debauchery, and political instability, by attempting to return to the values of the past. In hindsight, it can be said that the cholera debacle, ultimately, became the beginning of a new era, where advances in science, medicine, public health, and human rights laid the foundations for modern western civilisation.

In the Netherlands, the first cholera outbreak appears to have started in the summer of 1832, in the little fishing port of Scheveningen. But questions such as, whether the disease came from ports in Britain, whether it started through illegal fishing practices which avoided state control, or, indeed, who were the first patients, remained in doubt. Whether the first patients were two sailors, Leendert Knoester and Cornelis Harteveld,

suffering with diarrhoea in late June 1832, or 60 year-old Arie Mooiman[1], who died, unmistakably, of Asiatic cholera in early July 1832, sparked a fierce discussion at the time which those involved did not succeed in getting a definitive answer.

This second cholera pandemic did not, however, go unrecorded. It was preceded, and succeeded, by an explosion of professional, and not so professional, publications. There was an abundance of historical eyewitness reports on the pestilence in all major European languages. These, in turn, led to a large number of modern studies on the subject. According to Christopher Hamlin, a North American historian who specialises in the history of science and medicine, studies on cholera can be divided into three categories: 19th century accounts, cholera histories, and modern cholera science.[2] There are numerous examples of contemporary English-language volumes on local outbreaks in European countries. These include François Delaporte's work on the 1832 cholera outbreak in Paris and Richard Ross's publication on the cholera epidemic in Prussia in 1831. There were also smaller studies in scientific journals, such as Sean Burrell and Geoffrey Gill on the 1832 cholera in Liverpool, Catherine Kudlick on the 1832 Parisian cholera, Margaret Barnet on the 1832 outbreak in York, and many others.[3]

A wealth of written reports on the cholera outbreak of 1832 appeared in the Netherlands at the time. These publications contained everything from early preparations, to epidemiological data, and patient case descriptions, as well as the various treatments that were tried. In addition, a debate raged on the contagiousness of the disease, a highly controversial issue at that time. This book is an investigation of the original Dutch medical publications on that Asiatic cholera pandemic, describing the different aspects from a historical perspective with an emphasis on the medical insights and their development. It is, primarily, meant to fill a gap in the scientific literature regarding the first cholera pandemic in the Netherlands by describing, for an international audience, the Dutch responses to the novel infectious threat. According to Hamlin's scheme, the research for this book should be classified as a cholera history, based on 19th century accounts supplemented with modern science. As Hamlin further points out, cholera studies, from the 1960s onwards, first evolved from describing the cultural

and social history of cholera to descriptions of the cholera pandemics, and, after 1985, reverted back to descriptions of local outbreaks similar to that which had been popular in previous times. This study is consistent with the evolutionary timeline by returning to a detailed description of a local outbreak, namely the first Dutch cholera pandemic of 1832.

But why choose cholera as the subject of study? What is its significance? The recurring cholera pandemics during the whole of the 19th century helped shape both medicine and politics, awaken social awareness, and reform public health. These are the topic of further publications, such as Christopher Hamlin's own comprehensive biography of cholera, Peter Baldwin's study on how historical contagious diseases influenced public health policies in Britain, France, Germany, and Sweden, Rosenberg's 'The cholera years' on how the cholera pandemics influenced society in the USA, and Kotar & Gessler's more general cholera history.

In addition, I chose cholera because, as Eddy Houwaart, who has described the effect of cholera epidemics on the emergence of sanitary reforms in the Netherlands has stated that, in the first place: 'No other disease evoked so many emotions as the cholera'; and secondly, 'because cholera has been, until the end of the 19th century, the subject of fierce scientific and political controversies over the cause of the disease and its control, respectively.' Furthermore, over the years, Houwaart wrote in 1991: 'the number of cholera deaths became a measure of the effectiveness of health policy and of the validity of any scientific theory of communicable diseases.'[4] And the former statement still holds true almost three decades later, as in 2017, forty-seven countries were listed as 'affected by cholera', with nine countries experiencing large outbreaks.[5] So, roughly estimated, two centuries after the first global cholera alarm, the infection remains, according to the Global Task Force on Cholera Control: 'a highly sensitive, highly specific indicator for extreme poverty and harsh living conditions', as 'the presence of cholera indicates that a population does not have access to even basic water or basic sanitation'. The Task Force, which aims to reduce, considerably, cholera's morbidity and mortality by 2030 stresses the importance of indicating cholera control once again as the 'pathway to improved equity', because 'the continued existence of cholera outbreaks is a violation of human rights and dignity'.[6] Very little has

changed over time in this respect, despite being aware of cholera's causes and despite being able to take effective measures.

In addition to the authors of the original publications, I am indebted to earlier studies on the cholera epidemics in the Netherlands, which were published in Dutch, such as Martinus van Andel's 1938/1939 papers on the cholera in Gorinchem, Pieter 't Hart's description of cholera epidemics in Utrecht, and Har Meijer's 2005 PhD thesis on the cholera outbreaks in Leiden. Eddy Houwaart wrote an insightful and elaborate study on the reformation of the 19th century public health system in the Netherlands as a consequence of the cholera pandemics, which was a great help when describing the medical situation in the Netherlands at that time.

Epidemiologists, and others, define an epidemic as an infectious disease that affects many persons at the same time and spreads in a region or community where the disease is normally not prevalent. A 'pandemic' is an epidemic disease that has travelled the world, affecting people on most, or all, continents. An increase of cases of a disease that is already locally present, a so-called endemic disease, is commonly termed an 'outbreak', although the World Health Organization also uses 'outbreak' – at least one case with evidence of local transmission – for epidemic diseases. At first glance, however, these terms appear to simply describe various levels of disease spread, namely local ('outbreak'), regional or national ('epidemic'), or worldwide ('pandemic'). Since it seems illogical to write about, for instance, the cholera pandemic in Amsterdam, I will here use the terms outbreak and epidemic to indicate the first cases (outbreak) and subsequent increase in the prevalence of cholera in a town, city, province, or country (epidemic), and use the term pandemic only when referring to the global situation. Indeed, 19th century authors likewise used 'epidemic' to describe the cholera dispersal and prevalence at the local level.

CHAPTER 2

Cholera, the Bacterium and the Disease: an Introduction

The root cause of the severe gastroenteritis known to us as cholera is *Vibrio cholerae* 1854 Pacini[7], a curved, rod-like bacterium that prefers salty water as its habitat. Because of its comma-like shape, the bacterium has sometimes alternatively been referred to as *Vibrio comma*, after the comma bacillus observed by Robert Koch in 1883. The *Vibrio cholerae* bacterium possesses, at one end, a single powerful flagellum, a tail-like structure, enabling it to move at a surprising speed. However, environmental *Vibrio cholerae* are commonly not pathogenic for humans. They do not encode a toxin called cholera toxin, which is essential for inducing the massive outflow of water and electrolytes from the intestinal epithelium, a characteristic of severe cholera.[8] The capacity to encode toxins, however, is often carried by a bacteriophage, a virus that infects bacteria and, after doing so, commonly adds its genes to the bacterial genome. So, toxigenic *Vibrio cholerae* are infected by a toxin encoding virus, in this case the CTX (Cholera ToXin) phage.

During the devastating cholera pandemic of 1854, an Italian anatomist from Florence with a passion for microscopy, Filippo Pacini (1812-1883), took an interest in the disease. He analysed stools, vomit, blood, as well as stomach and intestinal samples from cholera victims, using an advanced microscope manufactured in the workshop of Giovanni Battista Amici of the University of Bologna.[9] Pacini was the first to see millions of trapped

objects in mucosal filaments (*'miriadi di vibrioni'*). When he broke up the filaments, the objects easily spread into the surrounding fluid. These so-called 'vibrions' were repeatedly observed by Pacini in the intestinal samples and faeces of those who succumbed to cholera, and so he postulated that they were the likely cause of the disease.[10] However, as he did not try to culture the vibrions in a laboratory setting, he could not confirm a causal relationship between these apparent 'living, parasitic contagions' and the diarrhoeal disease, a flaw in his research he dutifully acknowledged.[11] Filippo Pacini published his research on cholera in Italian in multiple papers during 1854-1880. His memoirs, which include his original observations, are kept at the Central National Library in Florence. However, Pacini's research received hardly any notice and was soon forgotten, while at approximately the same time a similar concept of a living contagion was being proposed, albeit without laboratory evidence, by the now famous British doctor, John Snow.[12] In his second edition of, 'On the Mode of Transmission of Cholera', published in 1855, Snow suggested that cholera is transmitted from person to person, not in the form of effluvia, invisible emanations such as vapours arising from patients which can get inhaled by bystanders, but as a 'morbid matter or poison' that 'enters the alimentary canal' and has the 'property of reproducing its own kind'. As such it 'must necessarily have some sort of structure, most likely that of a cell'.[13] Snow was also the first to show that cholera is transmitted via water contaminated with sufferers' excretions.[14]

Eventually, however, Pacini did receive recognition for his discovery of the cholera bacterium. In 1965, the Judicial Commission of the International Committee on Bacteriological Nomenclature decided that the correct name for the microbe was to be *Vibrio cholerae* 1854 Pacini and not *Vibrio* Müller 1773. The first description of *Vibrio*-like microbes, apparently, had been attributed to the Danish zoologist Otto Friedrich Müller (1730-1784).[15]

It was not until the fifth cholera pandemic was tormenting the world during 1883-1894 that genuine scientific interest was brought to bear in discovering the cause of cholera. The German scientist, and later Nobel prize winner, Dr Robert Koch (1843-1910), discoverer of numerous pathogenic bacterial species, was sent to Alexandria in 1883 by the State Secretary of the Interior for the German Imperial Health Service, Karl Heinrich

von Boetticher.[16] In 1884, Koch was working on, and would soon, in 1890, finalise, the famous Koch's postulates, the four criteria which need to be fulfilled for a causal relationship to be established between a pathogen and a disease.[17] Robert Koch also led the *Deutschen Cholera-Commission*, the German Cholera Commission, which was to investigate the extant outbreak in Egypt.[18] Comprising of, besides Koch, medical doctors Georg Gaffky, Bernhard Fischer, and chemist Hermann Treskow, the commission rediscovered the comma bacillus in microscopic preparations of the intestinal mucosa of cholera victims and in the watery stools of advanced cholera patients. They were, most likely, completely unaware of the earlier work of Pacini.[19] At the same time, a French team, consisting of two co-workers of the notable Dr Louis Pasteur, namely Drs Emile Roux and Louis Thuillier (who died of cholera during the investigations), medical Professor Dr Isidore Strauß, and veterinary Professor Dr Edmond Nocard, detected, independently, the curved bacillus, again exclusively in cholera patient samples. The French drew a similar conclusion about this bacillus being, in all probability, associated with the disease.[20] In 1884, Koch, who had relocated to India as the cholera epidemic in Egypt had subsided, reported that he had been able to grow the microbe in pure culture, one of the requirements he himself had devised. Nonetheless, the comma bacillus could not be reliably linked to disease due to the lack of an animal model for cholera. One of the postulates formulated by Koch, namely, 'to reproduce the disease in healthy organisms', could, thus, not be fulfilled. Attempts to infect mice, dogs, monkeys, and chickens with the cholera germ had proved fruitless as no signs of disease were seen. These negative results were a major obstacle to the acceptance of the comma bacillus as the causative agent of cholera. It was not just Koch's critics who believed this, but also Koch himself who stated that he: 'wasn't sure whether [the presence of the bacillus in patients] was cause or consequence.'[21] Likewise, French scientists had also been unable to find an animal species suitable as a model for cholera. They had also tried to infect rodents such as mice and guinea pigs, birds, such as chickens, pigeons, turkeys and quails, and also rabbits, pigs and again a monkey.[22] Disappointed, they concluded that the microbe observed in abundance in the majority of cases could not be attributed at present to a specific action in the disease process.[23] After their

return, they pointed out to the *Société de Biologie* in Paris that they, indeed, could not associate the 'abundant microbes' to the disease, but that they had seen some remarkable 'small bodies' in patients' blood samples. Koch, however, dismissed these objects as blood platelets.[24]

Nevertheless, Koch and his team finally returned to Berlin, and were welcomed as heroes, although the medical profession remained divided on the importance of Koch's findings for the aetiology of cholera.[25] In July 1884, a congress to discuss the cholera problem, the *Erste Konferenz zur Erörterung der Cholerafrage,* was held in Berlin with Robert Koch as the principal speaker.[26] However, a notable absentee was the leading cholera expert at that time, hygienist Dr Max Joseph von Pettenkofer (1818-1901), founder of the *Hygienische Institut* in Munich in 1879. He was a major opponent of Koch's bacterial origin theory about cholera. Instead, von Pettenkofer, after having studied the 1854 epidemic in Munich, described cholera as a complex multi-faceted disease, with influences from both time and place in collaboration with an individual disposition, combining contagious and miasmic ('bad air') concepts of the disease.[27] This *Boden* (soil) theory stated that cholera germs were changed into a miasma by a specific type of soil. People would then inhale that bad air and become ill. The quality of the soil was important. For the transformation to occur, it needed to be low, wet, and permeable, not high, dry, and stony. The theory, therefore, explained why some regions were not affected by cholera.[28] Furthermore, the famous pathologist and public health advocate, Dr Rudolf Virchow (1821-1902), speaking at the First Conference in 1884, more or less agreed with the aetiology of von Pettenkofer's *Boden* theory for cholera. He had also fought the germ theory of Louis Pasteur, as Virchow attributed all diseases to the degradation of the state of cells.[29] Of course, this is true, but the cause of cellular pathology could be the invasion by an external organism, a possibility Virchow first rejected.[30] However, by the end of the conference, Virchow had to admit that he was now much more convinced that the bacillus was the organism which caused the disease in cholera cases.[31] Von Pettenkofer's theories meanwhile had disastrous results. As he had claimed that the groundwater level was equally important in the generation of the miasma, he thus did not advocate water filtration. This led to a devastating epidemic of cholera in Hamburg in 1892 where 1.3%

of the population died of the disease.[32] By contrast, in the neighbouring city of Altona, where water filtered through sand had been made available, there were only a few cholera deaths registered. The Prussian Government then summoned Koch to Hamburg, where he introduced disinfection and quarantine of patients. In an attempt to save his theory that cholera was not caused by a solitary contagion, 74-year-old von Pettenkofer agreed to swallow a culture of the comma bacillus which would fulfil the third Koch postulate. This postulate implied that a similar disease should be induced in a healthy organism after infection with the microbe. Fortunately for him, he only developed some diarrhoea after drinking the poisonous concoction, although the comma bacterium could be found in his stools. Thus, the action did not meet the third postulate, but nevertheless, von Pettenkofer's fate was sealed.[33] Koch's ideas had already been incorporated in a new set of laws for epidemic control in the German Empire.

During his cholera studies, Dr Koch speculated that the comma bacillus generated some kind of toxin. In his view, cholera symptoms resembled intoxication.[34] In fact the idea that the cause of cholera was a type of poison had also been postulated during the 1831 Berlin epidemic.[35] In addition, Dr Horn had stated that during autopsies of cholera deaths: 'little or no inflammation is seen. The most important and specific nature of this disease, that should be attributed to an organic poison, is that the blood has been changed into a tar-like, greasy substance like that which is found after intoxication with prussic acid (hydrogen cyanide), arsenic, or carbon monoxide, and which is never seen in epidemic-miasmic diseases.' These speculations turned out to be correct, for in 1959 the toxin produced by *Vibrio cholerae* was isolated by the Indian scientist Dr Sambhu Nath De working at the Medical College of Calcutta (now Kolkata).[36] After culturing *Vibrio cholerae* bacteria, Dr De discarded the bacterial cells and filtered the remaining liquid. Subsequently, he introduced this liquid containing the presumed toxin into loops artificially created in the small intestine of rabbits. After 24 hours, the inoculated intestinal loops were filled with rice water-like fluid and the villi (small structures shaped like fingers in the small intestine) had, in some places, completely disappeared, both of which are hallmarks of cholera. Dr De, thus, showed that *Vibrio cholerae* bacteria do indeed produce a toxic substance that is essential for inducing

the peculiar choleric changes in the intestinal lining. In addition, the rabbit model, used by Dr De at the same time, delivered the missing evidence for Robert Koch's third postulate, requiring the induction of the symptoms of the particular disease in a healthy organism.

While the ancient Greeks had already used the word 'cholera' to describe a severely unsettled stomach, it was not until the beginning of the 19th century that cholera, caused by toxin-expressing *Vibrio cholerae* bacteria, became a familiar disease in the West. In 1817, the first epidemic of 'Asiatic cholera' was recognised in India after an outbreak in the vicinity of Calcutta. It subsequently spread to that city and then throughout the country. Most likely, cholera had already been present in India before 1817, as Dr John Snow mentioned in his 1855 publication:

> The existence of Asiatic Cholera cannot be distinctly traced back further than the year 1769, …… (when it) was prevalent at Madras and that it carried off many thousands of persons in the peninsula of India from that time to 1790'. From 1790 till 1814, 'we have very little account of the disease', 'although, of course, it might exist in many parts of Asia without coming under the notice of Europeans'. In June 1814, the cholera appeared with great severity in the 1st battalion of the 9th Native Infantry regiment, on its march from Jaulnah to Trichinopoly; while another battalion, which accompanied it, did not suffer, although it had been exposed to exactly the same circumstances, with one exception. In 1817, the cholera prevailed with unusual virulence at several places in the Delta of the Ganges.[37]

From 1820 onwards, cholera became a pandemic after travelling to South-East Asia, Central Asia, the Middle East, Eastern Africa, and along the Mediterranean coast. After a second cholera outbreak in 1829, again in India, the disease journeyed north into Russia (August 1830) and then onward to Hungary (June 1831), Poland (February 1831), Finland (August 1831), and Prussia (August 1831). Cholera reached the British Isles in October 1831 and then migrated with Irish immigrants to Canada (1832), and subsequently to the United States of America. In 1832, it was introduced into other European countries such as the Netherlands, possibly via England. Here the disease was named *Cholera Asiatica,* or occasionally *Cholera*

Morbus -cholera disease-, or simply *Cholera,* especially in the newspapers.[38] Sometimes, the name *Cholera morbus* was used to indicate the endemic, sporadic disease that resembled its Asian counterpart, *Cholera Epidemica,* in symptoms but not in severity.[39] The French and English mostly referred to the disease as plain *cholera morbus,* without capitals.[40] From the Netherlands, the disease threatened the western borders of what is now Germany.[41] The second cholera pandemic, meanwhile, also extended to the Norwegian city of Drammen and some heavily populated areas around the Drammen fjord, arriving there in autumn 1832.[42] However, relatively few people were affected in Norway, resulting in only 80 deaths.

Of course, the opinion had already been voiced that 'plagues could come from afar'. Describing the diseases prevalent in the Netherlands around 1824, Dr Henricus Thijssen, a former student of Dr Gerardus Vrolik (1775-1859), stated that many pestilences such as plague and leprosy had been imported from the East. He attributed most illnesses to the wet climate, the marshy land, the bad dietary habits, and the moral and physical weakening of the Dutch population. However, he also raised concerns about the importing of much-appreciated 'invigorating spices' used for cooking. In his opinion, the 'early and extensive trade' had opened a Pandora's box from which diseases flowed into Europe.[43]

The two early cholera pandemics were not the last to trouble the world. A total of seven pandemics, ranging from 1817-1823, 1826-1837, 1846-1862, 1864-1875, 1883-1894, 1899-1923, and from 1961 to the present day, the seventh is still ongoing, are recognised today. The closely-related seventh pandemic strains left the Bay of Bengal in three waves since their putative origin in the 1950s. Named El Tor, they belong to a different bacterial biotype than the strains which caused earlier epidemics.[44] These early strains are referred to as the classical biotype. Both classical and El Tor biotypes are variants of the O1 serogroup of *Vibrio cholerae*. This was confirmed by the analysis of old bacterial genomes preserved in an intestinal sample collected by Dr John Neill from a patient who died in the 1849 cholera outbreak in Philadelphia.[45]

Comparing the 1849 *Vibrio cholerae* genome with modern isolates, evolutionary biologists Alison Devault and her co-workers from the Anthropology Department of Canada's McMaster University in Ontario

calculated that the common ancestor of all pathogenic cholera strains probably arose only 430 to 440 years ago, when assuming a constant rate of evolution. However, others, who had been analysing much larger data sets showed that the evolutionary trajectory of cholera bacteria has not been that straightforward.[46] The Canadians accepted the new evidence and that the real origin of cholera likely lies much further back in time, well before all historically described pandemics.[47] *Vibrio cholerae* comprises a very diverse species with around 200 recognised serotypes. The marine bacterium normally inhabits the brackish water of river deltas, and is especially prevalent in the Ganges delta in India. It can be found there either as free-living bacteria or in association with organisms such as algae, plankton, oysters, crustaceans, and fish. Most serotypes of this bacterium cannot colonise the human gut, but a transition was probably made by members of the O1 serogroup at some time in the past.[48] As river deltas are commonly densely populated, it is likely that a substantial number of people eating either fish or shellfish from these waters, or drinking water contaminated with inflowing seawater, ingested these bacteria so often as to enable the species to adapt to the human body.

Although *Vibrio* strains may differ in virulence and an awareness now exists of the options to avoid or treat the infection, cholera is still a major killer in our time with an estimated 1.4 to 4.3 million cases each year, resulting in 28,000 to 142,000 deaths worldwide.[49]

Today, cholera is diagnosed by culturing *Vibrio cholerae* on selective growth media from suspect stool samples. Suspicions of the disease are aroused in particular when individuals present with acute watery diarrhoea. Dark-field microscopy may help in identifying the microbe. In addition, solid surface Rapid Diagnostic Tests, often in the form of dipsticks, which rely on modern biochemical techniques to recognise parts of the bacterium, are available.[50] However, diagnosing cholera from symptoms alone, as was required in the past, is difficult. Especially mild cases, which comprise up to 80% of those infected, may resemble other diarrhoeal maladies.[51] What is unique to cholera, however, is the abundant rice water-like diarrhoea smelling of fish, a watery stool with flecks of whitish materials originating from mucus and discarded intestinal epithelial cells. Other symptoms such as vomiting and abdominal cramps may