**Clostridium botulinum, A FOODBORNE PATHOGEN AND ITS IMPACT ON PUBLIC HEALTH**

Manal Hadi Ghaffoori Kanaan¹, Ahmad M. Tarek²

¹ Department of Agriculture, Technical Institute of Suwaria, Middle Technical University, Baghdad, Iraq.
² Department of Crime evidence, Institute of Medical Technology Al-Mansour, Middle Technical University, Baghdad, Iraq.

*Corresponding author E-mail: manalhadi73@yahoo.com (Kanaan)

**ABSTRACT**

Clostridium botulinum (C. botulinum) is an important cause of human disease, especially in hospitalized patients, as well as in animals. Botulism is a debilitating sickness that disturbs the body's nervous system and is initiated by incorporation of powerful neurotoxins produced by C. botulinum bacteria. Botulism typically associated with the ingestion of botulinum toxin (BoNT), which is already involved in food, and therefore, toxins arrive the human body through the gastrointestinal tract to produce the standard form of botulism are called consumption botulism. The outbreak of the food industry contaminated by BoNT is a public health crisis. The bioterrorism threat regarding the deliberate distribution of BoNT food and / or the aerosolization of BoNT raises public health and safety due to high mortality and potential morbidity. The aim of this review was, therefore to open up the idea of the disease by C. botulinum and its impact on public health to protect consumers from diseases and toxins transmitted by food. Meeting these criteria, the issue of C. botulinum with the net programs of modern foods satisfies food poisoning, reducing the transmission of these pathogens and their spores and / or toxins from food and feed. There are many unanswered questions about the epidemiology of this pathogen, and it would be prudent to monitor the ongoing research on this organism to determine whether it represents a risk as a foodborne pathogen.

**Keywords:** Botulism, C. botulinum, food, public health

**How to cite this article:** Kanaan MHG, Tarek AM(2020): Clostridium botulinum, a foodborne pathogen and its impact on public health, Ann Trop Med & Public Health; 23 (5): 346-359. DOI: http://doi.org/10.36295/ASRO.2020.2357

**INTRODUCTION**

Food-borne botulism is a lethal sickness triggered via the absorption of a powerful neurotoxin that is created through the growth of this pathogen in foods that cause undecorated food poisoning (1,2). Three major factors have increased the likelihood of this pathogen being the cause of a food-borne disease: the increasing recognition of community-induced C. botulinum infection, recent studies to recognize this pathogen in animals and food and the similarities between animal and botulinum isolates and humans. Initial diagnosis and handling of botulism is important to reduce the risk of death (3,4). A speedy epidemiologic study is crucial to forestall additional cases wherever even dangerous foods area unit accessible. Public Health Services and
Centers for sickness management and sickness management (CDC) offer 24x7 diagnostic recommendation, epidemiologic support and diagnostic laboratory services. The Center for Disease Control and Prevention offers powerfulness anti-poisonous substance (for varieties A, B and E of C. true bacteria) and bivalent botulinum anti-poisonous substance (for varieties A and B). Care professionals suspecting a patient with food poisoning ought to contact the Department of medical specialty of the National Health Service (3,4). While food native botulism is generally connected with canned vegetables, meat, fish and dairy products (1). Many unknown facts about the nature of botulism, its related to different food environments and the current impact on public health of this disease. Therefore, the current review is intended to provide information about this disease in order to

i. Knowledge of the food-borne diseases caused by C. botulinum and their impact on public health.

ii. Sensitize individuals to the risks and safety of food and feed to care for consumers from food borne diseases and toxins.

iii. Meet the needs of modern food safety databases around the world to provide safe and inexpensive food for all buyers, which can help in the microbiological control of food produces.

**Background information on C. botulinum and botulism.**

**Background to C. botulinum.**

*Clostridium botulinum* could be a cluster of artistically distinctive pathogens that are similar in this they're clostridia and yield natively numerous neurotoxins perparallel pharmacologic activity. This organismis straight, slightly hooklike, gram-positive (in young cultures), rod-shaped, anaerobic, 0.5-2.0 μm wide and 1.6-2.2.0 μm long, with oval and last spores (5). The seven species of pathogen (A - G) are illustrious by the matter properties of the neurotoxins they yield. Types A, B, E plus, in occasional suitcases, F produce sickness in humans, sorts C and D produce diseases in fowls and mammals. The sort G known in 1970 has not however been established as a reason behind sickness in humans or animals. Necessary epidemiologic and medical topographies discriminate the categories of food poisoning that produce human diseases. Sporadicsuitcases of food poisoning in infants and adults are confirmed once settlement of the gut by non-botulinum bacterial species that have created botulinum neurolysin (6). The mechanism of action and structure of all isomers of neurotoxins are analogous. Every toxigenic clostridia yields a peptide that's excited by proteases once microorganism lysis (6). The capability of this bacteria to cause sickness in humans is directly associated with the assembly of heat-resistant spores that survive maintenance approaches and kill non-sporulating organisms (7). The warmth resistance of spores fluctuates from kind to kind and additionally from strain to strain. though some strains don't survive at 80°C, the spores of the many strains would like temperatures on top of the boiling purpose to confirm their destruction (8, 9). In addition, the spores vacuum flask tolerance multiplied with the high pH scale and reduces the salinity of the soil during which the spores aredeferred(10).

In several suitcases, it's neither sensible nor fascinating to treat a product to eradicate all spores of this pathogen. As a result, most management approaches specialize in inhibiting the expansion and production of poisons. The most restriction factors for the event of this pathogen in foods are: a) temperature, b) pH, c)
water activity, d) reaction potential, e) preservative, and (f) competitive microorganisms. All of those factors are mutually beneficial and also the development of alone facet, therefore provides the impact of different factors (Figure 1). The communication of aspects perhaps has a positive or a negative outcome on the reserve of this pathogen. Overall, chemical action strains grow optimally at 40°C; The lower limit is 10°C, the higher limit is 45-50°C. Non-proteolytic strains, together with sort E, could still grow at 3°C. The minimum pH scale varies for the expansion chemical action strains are between 4.6 and 4.8. ; The limit for non-proteolytic strains is pH scale 5. However, some dietary proteins, like soy and beef, could have a protecting impact on these bacteria at pH scale 4.6 or less. Additionally, bound food preparations could contain “low acid” baggage wherever the pH scale could also be high spare to resist the assembly of poisons. The low activity of water (aw) prevents the progress of this pathogen. Atleast of ~ 0.94 is required to maintenance the expansion and creation of poisons. The water activity are often restricted by dehydration, however is typically organized by the addition of NaCl. Atleast aw of 0.94 matches to associate in nursing approximate resolution of 10 percent NaCl. The high oxidation-reduction potential (Eh) is typically owing to the existence of O₂. The most effective EH for the evolution of this pathogen is low (~350 mV) however a poison production at EH of +250 mV was found. Owing to this varying expansion of this pathogen and poison production may additionally occur in merchandise thought-about to be O₂ wealthy. Additionally, vacuum packaging, that is employed to cut back food preservation, will increase anaerobic conditions and may thus support the assembly of poisons. Numerous food additives (nitrites, carboxylic acid, parabens, synthetic resin antioxidants, polyphosphates and ascorbates) stop the expansion of this pathogen plus restrict the assembly of poisons. Lactic acid bacterium, as an example: *Pediococcus* and *Lactococcus*, are revealed to provide acid and thereby inhibit the growth of this pathogen7,11.
Background information on botulism.

Neuro paralytic food poisoning may be a unwellness ensuing from the accomplishment of a forceful poison made by the organism, in food and animal feed. E. van Ermengem initially reported of this germ in 1897 once a study of a food-borne happening in Ellezelles, Belgium. Food poisoning is occasional, however it will kill quickly and contaminated product will expose many of us. Food poisoning is thus a medical and community health emergency that promotes speedy and effective communication between physicians and health officers. "Botulism" arises from the Latin "botulus", denotation sausage. Initially recognized food poisoning was in Europe and plenty of cases were owing to home-cured sausages. This distraction, although traditionally vital, has lost abundant of its importance as a result of plants square measure additional common vehicles than animal product. Sausage is never the reason for food poisoning within the North American country (12,13). Foodborne botulism is a serious disease. It is a poisoning caused by the consumption of BoNT in food. At least 30ng of neurotoxin is enough to cause disease and even death. The consumption of at least 0.1ng of food in which botulinum is grown can lead to botulism. Foodborne botulism is mainly associated with two physiologically and heritably different clostridia, namely \textit{C. botulinum} (proteolytic) and \textit{C. botulinum} (not proteolytic). The proteolytic one is a mesophyll with a growth temperature of at least 10°C to 12°C, while the non-proteolytic is a growing psychophore and forms a toxin at 3.0°C (14).

In the last decade, sales of modified atmosphere vacuum and refrigerated foods have increased significantly. These foods meet consumer demand as they are of high quality and require little preparation time. The microbiological care of these foods normally depends on one or more factors including heat, cold, pH control, water activity control and limited shelf life. The food botulism presented by non-proteolytic \textit{C. botulinum} (14), represents the main risk to the microbiological care of these foods.

Given the severity of botulism, regulators and industry are working hard to reduce the risk of occurrence. This led to the expansion of several guidelines, recommendations and procedural rules for the control of \textit{C. botulinum} in food, including refrigerated VP and MAP foods. Sales of refrigerated VP / MAP foods have increased significantly over the past two eras (14).

Risk assessment is a commonly used term that can have many definitions. The formal risk assessments for Codex are clearly defined, require extensive data and are formally recorded to allow transparent assessment of the risks connected with the assembly of certain products (14). Depending on the character of the poisonous substance uptake, four totally different styles of gastrointestinal disorder are often produced:

i. Gastrointestinal disorder within the kind of food outcomes from the assimilation of foods containing a preformed poisonous substance.

ii. Injury kind of a unwellness is caused by organisms that replicate and turn out a poison in an exceedingly contaminated wound.
iii. Child kind of gastrointestinal disorder is owing to the endogenous production of the poisonous substance by germination of bacterial spores within the bowel of the child.

iv. Gastrointestinal disorder in kids or adults through establishment is symbolized by the circumstances beneath that no food vehicle are often identified; there's no proof of gastrointestinal disorder owing to injury and risk of colon establishment in an exceedingly person over one year.$^{11}$

**Gastrointestinal disorder of food origin**

**Impact**

For surveillance purposes, we have defined an outbreak transmitted by food as one or more suitcases of botulism in which a source of tainted food was involved.$^{15}$ Between 1899 and 1949, there have been 477 gastrointestinal disorder of epidemics of food broadcast within the us among 1950 to 1996, extra 444 were informed to Center for Disease Control and Prevention for a complete of 921. The common range of households year has modified very slight, 9.7 each year for the previous amount and 9.4 each year since 1950. For the era from 1899 to 1949, 1281 cases of gastrointestinal disorder are conveyed and between 1950 and 1996 conveyed 1087 circumstance a complete of 2368 suitcases$^{15}$. The common range of cases per epidemic remained constant: a 2.6 suitcases / epidemics within the primary a part of the century and a 2.5 suitcases / epidemics to this point within the succeeding half. Of the 444 epidemics of gastrointestinal disorder foodborne since 1950, 37.6% were owing to neurolysin blood group, type B 13.7%, 15.1% for sort E, type F 0.7% or 32.9% of unidentified suitcases, form of poisonous substance. The proportion of epidemics that the kind of poisonous substance wasn't determined decreases. In 76.9% of households within the amount 1950 to 1959, the poisonous substance sort wasn't resolute, against fifty nine of the outbreaks of the amount 1960-1969, 12.5% in 1970-1979 and a 2.5% in 1980-1989 and 3.6% for 1990-1996. From 1990 to 1996, epidemics were a lot of usually triggered by blood group (44.6%), the subsequent sort (35.7%) and sort B (12.5%)$^{15}$.

**Mortality**

During the era 1899-1949, the proportion of deaths was high at around 60%, however around 1950 mortality step by step cut. For the era 1950-1996 the mortality was 15.5%. This reduction in mortality is principally owing to improved metabolic process care and intensive support, and probably speedy administration of antibody. The general death rate for every kind of poisons has diminished over the years$^{15}$.

**Terrestrial dissemination**

Between 1899 and 1996, epidemics of gastrointestinal disorder within the kind of food were described in forty six states, Puerto RICO and Washington DC. Exclusively four states have constantly described this illness: Delaware, New Hampshire, South geographic area and American state. Five Western states (California, Washington, Colorado, Oregon and Alaska) accounted for quite 0.5 (53.8%) of households described food transfers since 1950. Uniquely Last Frontier has 16.2% of households across the nation. This has been owing to the actual public pathological state of the autochthonic population of Last Frontier, wherever all cases of gastrointestinal disorder are connected with inadequate preparation and storage of ancient foods from Last
Frontier (16). Within the USA, there's a marked geographical distribution of the categories of neurolysin. From 1950 to 1996, 167 epidemics were registered and one hundred forty four (86.2%) occurred within the states west of Mississippi. California, Washington, Oregon and Colorado accounted for fifty eight (7%) of all described blood group epidemics. Twenty states, most notably within the east of the country, didn't report the type-A epidemic, group B gastrointestinal disorder outbreaks were described by twenty six states. Of the sixty one group B outbreaks according from 1950 to 1996, 37 (60.7%) were described by the eastern states; New York and Kentucky described thirteen epidemics of group B. Epidemics of sort E were described in ten states. The geographical distribution shows a preference for Last Frontier, with fifty six (83.6%) described epidemics from 1950 to 1996. The regional distribution of the poisonous substance sort with epidemics results from a study of soil samples that systematically evidenced a prevalence of blood group spores in Last Frontier Western soil samples and B preponderantly spores enter the soil of the northeastern and intermediate states (17). E-spores were found in marine life and sediments within the geographical region and countless lake (18, 19).

Food causes and merchandises that cause epidemics

By the first sixties, the majority gastrointestinal disorder epidemics within which sorts of poisonous substances were determined for toxin sorts A or B and were sometimes canned with the activity of vegetables, fruits, and meat product. Sort E gastrointestinal disorder wasn't documented as a heavy troublesome within the USA till 1963 once twenty two cases were described (20, 21). Sixty one of the sixty seven sort E gastrointestinal disorder described in 1950-1996 epidemics was discovered in marine product (fish or marine mammals); the beaver has been linked many suitcases. Remainder comes from an indefinite supply. Exclusively 3 epidemics of sort F gastrointestinal disorder were described during this country, one amongst that was determined reception games (22). Between 1950 and 1996, 289 (65.1%) epidemics of gastrointestinal disorder are found in processed foods reception and thirty one (7%) in commercially manipulated foods, together with food served within the edifice. The kind of food handling that accounts to 124 (27.9%) epidemics is mysterious. The vegetables were vulnerable to neurolysin within the US from 1950 to 1996. The fish and marine mammals have additionally been the supply of the many gastrointestinal disorder epidemics throughout that point. Of the eighty seven epidemicstriggered by food (fish or marine mammals) were sixty one by E sort, 15 types A, group B eight and three were unknown. Beef, dairy, pork, poultry and alternative vehicles have triggeredto fewer epidemics (23).

Management interventions

In the USA, food gastrointestinal disorder owing to marketable foods has been mostly precise by safe canning and food production approaches. Industrial food products are heated to a temperature decent to get rid of spores. Untreated industrial foods in jars or cans are often secured by action or alternative manipulations that inhibit the expansion of the body (for example, the addition of H₃PO₄ acid with garlic in oil). Generally industrial foods still cause gastrointestinal disorder after they are able to permit the assembly of poisons. Within the US, several gastrointestinal disorder epidemics are linked to the activity of poorly preserved canned foods (3). People that work with food product and alternative foods ought to be alert of the time, pressure, and temperature needed to kill spores, the necessity to properly cool unprocessed foods, and
therefore the effectiveness of preparation. Boiling water; the addition of canned vegetables destroys toxins yielded by this pathogen (20). Autoclave can safely store vegetables reception as they reach temperatures higher than boiling (> 212°F [>100°C]), necessary to kill the spores of gastrointestinal disorder (24). Although botulism spores are thermally stable, botulinum toxin is heat labile. Botulinum toxin can be inactivated by heating to 80°C (176°F). Heating canned foods before ingestion may therefore decrease the hazard of botulism intoxication. *C. botulinum* may swell the caps of the containers and the contents must have a “bad smell”. Commercially available cans or jars with a protruding lid must not be opened, and foods that do not smell should not be eaten or “tested” (24). Additional data on home safe canning practices connect with the native district extension officer or the North American country Department of Agriculture's educational activity website is very important (25).

**Child gastrointestinal disorder**

Since 1980, child gastrointestinal disorder has been the foremost common variety of gastrointestinal disorder within the US. It’s epidemiologically totally different from foodborne gastrointestinal disorder, that doesn’t create the uptake of the poison in contaminated food; however establishment (contagion) of the gut with spores of this pathogen, with ulterior formation of poisons in vivo (26). Though child variety of gastrointestinal disorder has been delineated for the primary time in 1976 (27,28), previous cases in recent years have solely been discovered on reflection and known advances in diagnostic talents instead of the start of the coated illness. The new illness well-tried syndrome is delineated by the looks of constipation followed by a neuromuscular paralysis that begin with the cerebral nerves and accomplish peripheral and metabolic process muscles. The severity spectrum ranged from delicate lethargy and slow food intake to severe cardiovascular disease and metabolic process failure (29,30).

**Medical specialty and birthplaces of *C. botulinum***

Since the popularity of child variety of gastrointestinal disorder in 1976, additional and extrasuitcases are detected. Within the US, 1,442 suitcases were sent to CDCs in forty six states between 1976 and 1996. Group A accounted for 46.5% of those circumstances and sort B 51.9%. Since the reports arose toward steady in 1980, the regular twelve-monthly incidence of gastrointestinal disorder in infants within the North American country was concerning ~2 / 100,000 live births. Since 1976, 47.2% of all suitcases of gastrointestinal disorder in infants are supposed in CA. Between 1976 and 1994, the incidence of child gastrointestinal disorder was highest in Delaware, Hawaii, Utah, and CA (9.0, 8.8, 6.3, and 5.7 per 100,000 live births), in respectively. The explanations for the plain geographical variations area unit unknown. The characteristics of gastrointestinal disorder suitcases in infants are processed in recent years. Hospital infants typically have a better birth weight than alternative kids, and their mothers’ area unit typically white, older and higher educated than mothers within the overall residents. Affected kids also are additional typically breastfed and breastfeeding is related to early-onset B-type un wellness (29, 30). Generally, affected babies area unit the merchandise of traditional gestation and childbearing. Inherent anomalies weren't related to gastrointestinal disorder in infants and kids were typically in healthiness till gastrointestinal disorder occurred. Regarding constant variety of men World Health Organization were tormented by ladies. The mean age of onset was thirteen weeks and ranged from one to sixty three weeks. In some jap suburbs of the u. s. plus in certain little
cities and rural areas within the west, an accumulation of suitcases of child gastrointestinal disorder has been discovered\(^{(31)}\).

**Gastrointestinal disorder because of injury**

Injury gastrointestinal disorder could be a rare unwellness that results from the expansion of spores for this pathogen during a wound that's contaminated with the assembly of inactive toxins\(^{(40)}\). Neurologic findings area unit indistinguishable from those discovered in food gastrointestinal disorder. However, duct symptoms don't occur. The injuries might not be obvious or severely infected. Between 1943, the year during which the syndrome initially became best-known, and in 1985, thirty three cases of gastrointestinal disorder were supposed within the US. Of these, twenty five were confirmed within the laboratory; seventeen suitcases A, seven suitcases B and one were classified as a mix of group A and sort B organisms\(^{(41)}\). The mean age of the patients was twenty one years (range half dozen to forty four years); eighty one were men. The injuries were typically deep and enclosed vascular zones; several ill people had composite fractures and four patients had been severely eviscerated by hand. The common period for trauma was seven days (range: 4-21 days)\(^{(42)}\). Since 1980, suitcases of gastrointestinal disorder are wounded in folks that have used illicit medicine. They need been related to sites of needle perforation or nasal or sinus lesions because of chronic inhalation of cocaine\(^{(43)}\). From 1986 to 1996, there have been seventy eight supposedsuitcases of gastrointestinal disorder per injury within the USA. Most involved injecting drug use, particularly the alleged "black tar heroin". Sixty-sixA cases were typed , nine were group B and therefore the residualthree were mysterious poisonous substance varieties. The mean age of the ill people was thirty eight years (range 5-65 years); 60% were men\(^{(43)}\).

**Gastrointestinal disorder in youngsters or adults once enteric employment:**

Isolated gastrointestinal disorder circumstances underneath that associate in-depth investigation doesn't mean that some foods because the reason for the sickness are recorded by the office since 1978 as a case of "unknown origin" rather than food-borne botulism. By 1996, all of those suitcases were in adults, whereas some speculation has taken place since 1920, an intensive investigation has shown that a number of these cases were owing to constitution of the alimentary canal of *C. eubacterium* or *C. baratii* with poisonous substance production in vivo, just like the pathologic process of kid gastrointestinal disorder\(^{(44, 45)}\). The designation of *eubacterium* by constitution of the viscus is expedited by proof of prolonged degradation of neurolysin for this pathogen and faecal matter and by detection of spores of *C. botulinum*, however not preformed toxins in suspect foods. In some suitcases of gastrointestinal disorder, it's been a history of gastrointestinal surgery or diseases, like inflammatory viscos sickness, that dispose them to enteric-coated constitution. No different specific risk factors were known\(^{(46)}\).

**Botox**

The pathogen *C. botulinum* is the same bacterium that is used to yield Botox, a medicinal produce principally injected for clinical and cosmetic usage. Botox treatments custom botulinum neurotoxin type A purified and
highly diluted. The treatment is overseen in a medical setting, adapted to the requirements of the patient and is mostly well tolerated even if occasional side effects are perceived (47).

**Clinical syndrome**

The clinical syndrome gastrointestinal disorder, whether or not for food, suckling, injury or enteric constitution, is conquered by neurologic indicators associated signs derivatives of an anchor-blocking poisonous substance, voluntary motor skills, and involuntary cholinergic junctions, and is fairly paralleled at every reason plus every sort of toxin (48). The time period for gastrointestinal disorder in food is simply half dozen hours or ten days (49). In general, the time between activity of poisons and onset of symptoms is between eighteen and thirty six hours (49, 50). The intake of different bacterium or their toxins in poorly keep foods or enteric motility changes are seemingly to be answerable for abdominal pain, nausea and vomit and diarrhoea, which regularly accompany the neurologic symptoms of gastrointestinal disorder or food preceding them. Dry mouth, incapability to concentrate on a near-point (causing the ill people to criticize of "blurred vision") and visual disorder are typically the primary neurologic disorders. If the sickness is benign, the opposite symptoms might not develop and therefore the initial symptoms bit by bit disappear. The person with gentle gastrointestinal disorder can't attract the doctor's helpfulness. In severe suitcases, however, these preliminary indications could also be followed by speech defect, dysarthria, dysphagia, and weakness of the peripheral muscles. If the ill health is severe, the metabolism muscles are affected, leading to ventilator failure and death if no adjunct treatment is provided. Recovery follows the regeneration of latest fiber bundle compounds. Respiratory support from two to eight weeks is common, though patients would like metabolism support for up to seven months before muscle perform is fixed up (48). Death happens in five to 100% of cases of gastrointestinal disorder caused by food. Premature deaths are seen by the severity of the sickness or general secondary respiratory organ infections, whereas deaths once two weeks are thanks to complications for the management of long-run mechanical ventilation (48). Maybe as a result of babies are unable to complain regarding the first effects of bacterial poisoning, the neurologic dysfunction related to gastrointestinal disorder in infants typically seems to develop suddenly. The most manifestations are poor nutrition, reduced ability to suck and cry cervical and peripheral weakness (infants are typically thought about "soft babies" and ventilator failure (37). Constipation conjointly typically happens in infants with gastrointestinal disorder, and in some cases, many days precede the looks of neurologic abnormalities. The loss of facial expressions, dysfunction of the attention muscles, expanded pupils, and deep sinew reflex depression were additional normally described with B gastrointestinal disorder compared to A gastrointestinal disorder infants (38). Treatment with antimicrobial aminoglycosides could promote fiber bundle weakness in kid gastrointestinal disorder and has been related to enlarged chance of mechanical ventilation. But two of described suitcases of gastrointestinal disorder in infants result in death (51).

**Public health response**

Because botulinum food poisoningis caused by ingesting tainted food that will still be the reason behind the sickness, one botulism circumstance could be a public health emergency that may trigger an epidemic (52).
Therefore, it's crucial for physicians to doubt that botulinum food poisoning is being mentioned directly with epidemiologists and public health communities on the bottom. State public health authorities ought to so now contact the government agency. If marketable diet produce could be a suspected food poisoning vehicle, the Department of Agriculture should conjointly inform the administration of food and medicines. Seeking for a suspected circumstance of food poisoning involves the immediate seek for different probable causes, the documentation of suspected suitcases of food and also the confirmation of the identification. If over one person is affected, a fast and careful epidemiologic investigation is initiated to make sure that the supply is known and controlled. Diagnostic tests for each suitcase and for food ought to achieve as required (47).

CONCLUSION

Occasionally, *C. botulinum* may be present in food and represents a potential risk to public health. *Clostridium botulinum* is responsible for a disease called botulism. Botulism is a lethal disease caused by the ingestion of a powerful neurotoxin formed through the growth of *C. botulinum* bacteria. This neurotoxin is among the peak toxic substances known; Even microscopic amounts can cause illness or death. It can destroy, paralyze or negatively affect nerves or nervous tissue. The prevention of botulism of food can be carried out through good practices in the preparation of food, in particular during heating / sterilization and hygiene. Food botulism can be prevented by inactivating the bacterium and its spores in hot sterilized (for example, replicates) or canned products or by inhibiting bacterial growth and the production of their toxins in other products. Presently, the improvement of sanitation practices at all steps of the food production chain based on the “farm to fork” principle and the reduction of the burden of pathogens in foods of animal origin are highly recommended and seem be the only realistic measures for preventing the spread of foodborne pathogens such as *C. botulinum*.

ETHICAL CLEARANCE

The Research Ethical Committee at scientific research by ethical approval of both environmental and health and higher education and scientific research ministries in Iraq

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

FUNDING: Self-funding

REFERENCES


39. Wells, Carol L., Hiroshi Sugiyama and Sarah E. Bland. "Resistance of mice with intestinal flora limited to enteric colonization by Clostridium botulinum." The Journal of Infectious Diseases, 1982, 146.6: 791-1891; 796th


44. McCroskey, Loretta M. and Charles L. Hatheway. "Laboratory results in four cases of adult botulism indicate colonization of the intestinal tract." Journal of Clinical Microbiology, 1988, 26.5: 1052 - 1054th


49. Louis, Michael E. ST, et al. "Botulism by chopped garlic: Late detection of a major focus." Annals of Internal Medicine, 1988, 108.3: 363-1388; 368th

