

Seafood poisoning symptom, treatment and prevention

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Abstract

Food related disease or food poisoning is prevalent worldwide and is associated with high mortality. It can be caused by bacteria, viruses, parasites, enterotoxins, mycotoxins, chemicals, histamine poisoning (scombroid) ciguatera and harmful algal bloom (HAB). Illness can also result by red tide while breathing in the aerosolized brevetoxins (i.e. PbTx or Ptychodiscus toxins). Bacterial toxin food poisoning can affect within 1-6 hours and 8-16 hours, and illness can be with or without bloody diarrhea. The common symptoms of food poisoning include abdominal cramps, vomiting and diarrhea. Diagnosis includes examination of leftover food, food preparation environment, food handlers, feces, vomitus, serum and blood. Treatment involves oral rehydration, antiemetic, and anti-peristaltic drugs. Antimicrobial agents may be needed in the treatment of shigellosis, cholera, lifesaving invasive salmonellosis and typhoid fever. Proper care in handling and cooking is important to prevent any food borne diseases.

Keywords: Seafood poisoning, Harmful algal bloom (HAB), Scombroid, Ciguatera

Introduction

The actual incidence of foodborne disease (infection) is unknown but estimated in 1997 to be approximately 76 million cases, with 325,000 hospitalization and 5,000 deaths each year in the United States (Mead et al., 1999). Analysis of surveillance data from baseline period from 1996 to 1998 through 2007 has shown a decrease in incidence of illness due to several pathogens (Centers for Disease Control and Prevention, 2008). Foodborne illness, also referred to as food poisoning, is the illness resulting from consumption of food spoiled by pathogenic bacteria, viruses, parasite and other contaminants (Centers for Disease Control and Prevention, 2018). Pathogens vary in minimum infectious dose; *Shigella sonnei* has a low minimum dose of <500 cfu, while *Staphylococcus aureus* relatively high and *Salmonella* a high inoculum of 1 million to 1 billion dose (Greig et al., 2010; Owens and Warren, 2017). Harmful algal blooms (HAB) are often associated with mortality of many marine animals, and shellfish poisoning (Center for Disease Control and Prevention, 2009). Marine dinoflagellates that produce red tide are the source of ichthyotoxins (White, 1981). Humans are affected by ingesting harvested shellfish affected by the toxin, breathing in aerosolized brevetoxins (i.e. PbTx or Ptychodiscus toxins) and in some cases skin

contact (Pierce and Henry, 2008). Universiti Malaysia Sabah (UMS) researchers are working on the relationship between environmental factors and HAB species occurrences in the coastal waters of Singapore and Sabah (Yoshida, 2017). The incubation period ranges from hours to days (rarely months in case of listeriosis or bovine spongiform encephalopathy). If symptoms occur within one to six hours after eating food, it suggests that it is caused by a bacterial toxin or chemical rather than live bacteria (Center for Disease and Prevention, 2018). Frequent symptoms of food poisoning include, abdominal cramps, vomiting and diarrhea. Most diarrheal diseases can be managed with oral rehydration. Antiemetic and anti-peristaltic agents also provide symptomatic relief, although later were contraindicated in patients with high fever, bloody diarrhea, or fecal leucocytes indicative of an invasive infection (Guerrant et al., 2001). This paper presents a review of some common cases of seafood-borne illness.

Worldwide prevalence

Asymptomatic subclinical infection may help spread these diseases, particularly *Staphylococcus aureus*, *Salmonella*, *Shigella*, and *Yersinia* (Greig et al., 2010). For example, as of 1984 it was estimated that in the United States, 200,000 people were asymptomatic carriers of *Salmonella* (Greig et

al., 2010). Infants are particularly vulnerable to food related disease. World Health Organization has issued recommendations for preparation, use and storage of prepared formulas. Breastfeeding remains the best preventive measure for protection of food related infections in infants (FAO/WHO, 2006).

In the United States, using FoodNet data from 2000-2007, the CDCP estimated that there were 47.8 million foodborne illnesses per year (16,000 cases per 100,000 inhabitants) (Scallan et al., 2011a), with 9.4 million of these caused by 31 known identified pathogens (Scallan et al., 2011b), leading to hospitalization of 127,839 people, amounting to 43 per 100,000 inhabitants per year (Nixon, 2015) and death of 3,037 people (1.0 per 100,000 inhabitants per year) (Tavernise, 2013). In France, foodborne illness causes 10,200-17,800 hospitalizations per year, with Salmonella being the most frequent cause (5,700-10,200 cases) (Vaillant et al., 2005). A study by the Australian National University published in November 2014, found in 2010 that there were an estimated 4.1 million cases of food borne gastrointestinal ailments acquired in Australia on average each year, along with 5,140 cases of non-gastrointestinal illness (Kirk et al., 2014). The recent study replaces a previous estimate of 5.4 million cases of foodborne illness in Australia every year (The OzFoodNet Working Group, 2004) causing: 180,000 hospitalization, and 120 deaths (0.5 deaths per 100,000 inhabitants). Most foodborne disease outbreaks in Australia have been linked to raw or minimally cooked eggs or poultry (Astridge et al., 2011). The Australian Food Safety Information Council estimates that one third of cases of food poisoning occur in the home (British United Provident Association, 2010).

Agents of food related disease

Food related disease could be caused by a variety of bacteria, viruses, parasites, enterotoxins, mycotoxins, and toxic substances such as poisonous mushrooms or reef fish (Diogène et al., 2017). In the United Kingdom during 2000, bacteria involved in food related illness include, *Campylobacter jejuni* 77.3%, *Salmonella* 20.9%, *Escherichia coli* 0157:H7 1.4% and all others less than 0.56% (Food Standards Agency, 2014). Other common bacterial pathogens are: *Campylobacter jejuni*, which leads to Guillain-Barre syndrome and periodontitis (Humphrey et al., 2007), *Clostridium perferingens*, the “cafeteria germ” (United States Department of Agriculture, 2013), and *Escherichia coli* 0157:H7 enterohemorrhagic (EHEC) (Tribe et al., 2002). Bacterial enterotoxins can produce illness even when the microbes that produce them have been eliminated. The enterotoxins of *Staphylococcus aureus* symptoms appear in two to six hours (Hennekinne et al., 2012).

Viruses involved in the illness include, enterovirus, Hepatitis A, Hepatitis E, Notovirus and Rotavirus. The virus has been found to cause infection due to the consumption of fresh-cut produce, which has fecal contamination (Dubois et al., 2006). Various parasites involved in the illness are *Cyptosporidium*, *Cyclospora*, *Giardia*, and *Trichinella* (Lew et

al., 1990). Chemicals involved in the illness include Ciguatera, histamine fish poisoning (scombroid), heavy metals, Monosodium-L-glutamate, paralytic shellfish poisoning, and neurotoxic shellfish poisoning (Lew et al., 1990).

Seafood poisoning

Ciguatera food poisoning

Ciguatera is a foodborne illness caused by eating certain reef fish whose flesh is contaminated with the toxin produced by dinoflagellates. These dinoflagellates adhere to coral, algae and seaweed, where they are eaten by the prey fish, which in turn are eaten by larger carnivorous fish like barracudas and shark (Diogène et al., 2017). *Gambierdiscus toxicus* is the primary dinoflagellate responsible for the production of a number of similar toxins, including ciguatoxin maitotoxin, gambieric and saritoxin, as well as the long chain alcohol palytoxin (Faust and Gullledge, 2002). Other dinoflagellates that may cause ciguatera include *Prorocentrum* spp., *Ostreopsis* spp., *Coolia monotis*, *Thecadinium* spp. and *Amphidinium cartetarae* (United States National Office for Harmful Algal Blooms, 2016). Predator species near the top of the food chain in tropical and subtropical waters are most likely to cause ciguatera poisoning, although other species may also cause occasional outbreaks of toxicity (FAO, 2004). Ciguatera is odorless, tasteless and cannot be removed by conventional cooking (Swift and Swift, 1993).

Frequent clinical symptoms include gastrointestinal, cardiovascular, and neurological effects (Isbister and Kiernen, 2005), followed by neurological problems such as headache, muscle aches, paresthesia, numbness of extremities, mouth and lips reversal of hot and cold sensation, ataxia, vertigo, and hallucination (Clark et al., 1999; Isbister and Kiernen, 2005). Dyspareunia and other ciguatera symptoms have developed in otherwise healthy males and females following sexual intercourse with partners suffering from ciguatera poisoning, signifying that the toxin may be sexually transmitted (Lange et al., 1989). Death from the condition may occur, but is extremely rare (Telegraph Reporter, 2016). The symptoms may last from weeks to years, and in extreme cases as long as 20 years, often leading to long-term disability. Most people do recover over time (Gillespie et al., 1986; Pearn, 2001).

Scombroid or decayed fish poisoning

This illness results from eating spoiled (decayed) fish (Clark et al., 1999). In 2015, seven people at a café in Sydney became ill after eating John Bull Tuna Chunky Style in safflower oil on tuna salads (International Society for Infectious Disease, 2015). The canned tuna had come from Thailand. Several people became ill after eating tuna sandwiches at a café in Edinburgh in Scotland in 2013. The tuna had come from Ghana (International Society for Infectious Disease, 2013). In 2011, 20 reports of food poisoning at a Stockholm restaurant were thought to be histamine poisoning in tuna from Senegal. In 2012, the UK environmental health authorities in North East Lincolnshire

intercepted and destroyed a shipment of tuna from Vietnam after four crew members were reported to have developed symptoms of histamine poisoning (International Society for Infectious Disease, 2011). Commercially canned tuna was determined to be the cause of the poisoning of 232 persons in the north central United States in 1973 (Merson et al., 1974).

Along with ciguatera, it is listed as a common type of seafood poisoning (Merson et al., 1974; Heller and Zieve, 2017). The toxin believed to be responsible is histamine, formed as the flesh of the fish that begins to decay. The histamine is also the natural agent in allergic reactions, hence scombroid food poisoning often gets misidentified as a food allergy. The syndrome is named after the Scombridae family of fish, which includes mackerels, tunas and bonitos, because early descriptions of the illness noted an association with those species, although the Center for Disease Control and Prevention (CDC) identified that the non-scombroid syndrome can result from inappropriate handling of fish during storage or processing. Cooking does not prevent illness, because histamine is not destroyed at normal cooking temperature (Merson et al., 1974; Heller and Zieve, 2017).

Unlike many types of food poisoning, scombroid-related health problem is not caused by ingestion of a pathogen (Heller and Zieve, 2017). Histidine is an amino acid that exists naturally in many types of food (including fish) and at temperatures above 16°C (60°F). It is converted to the biogenic amine histamine via enzyme histidine decarboxylase produced by symbiotic bacteria such as *Morganella morganii*. This is one reason why fish should be stored in the freezer. Histamine is not destroyed by normal cooking temperature, so the consumption of even properly cooked fish can still result in poisoning (Brenner, 2012). Histamine is the main natural chemical responsible for the allergic reaction, so the symptoms produced are almost identical to food allergy (Otwell, 1989). In rare cases, the poisoning may result in death. Sudden deaths of some tourists in Bali in January 2014 were attributed to scombroid food poisoning (Queensland Courts, 2015).

Red tide and harmful algal bloom

The term red tide is most often used in the United States of America to refer to *Karenia brevis* blooms in the eastern Gulf of Mexico, also called as Florida red tide. In the past decade, it has been elucidated that *K. brevis* is only one of the many different species of the genus *Karenia* found in the world's oceans (Fleming et al., 2011). Red tide is a common name for a worldwide phenomenon known as algal bloom (large concentrations of aquatic microorganisms- protozoans or unicellular algae) when it is caused by species of dinoflagellates and other organisms. Certain species of phytoplankton found in red tide contain photosynthetic pigments that vary in color from brown to red. When algae are present in high concentrations, the water appears to be discolored or murky, varying in color from a rust to pink to blood red. Specifically, red tide species can be found in

oceans, bays and areas where fresh water meets salt water but they cannot thrive in freshwater environments. The growth of the algal bloom depends on wind; temperature, nutrients and salinity (Alcock, 2007). Some red tide algal blooms are associated with fish kills. The production of natural toxins such as ichthyotoxins is harmful to marine life. Generally, red tides are described as harmful algal blooms or HABs. Their conspicuous effects are associated with wildlife mortalities as well as harmful human exposure (Alcock, 2007).

Humans are affected by the red tide species by ingesting contaminated harvested shellfish, breathing in aerosolized brevetoxins (i.e. PbTx or Ptychodiscus toxins) and in some case skin contact. The brevetoxins bind to voltage-gated sodium channels, an important structure of cell membranes. Binding results in persistent activation of nerve cells which interferes with neural transmission, leading to health problems. These toxins are created within the unicellular organism, or as a metabolic product (Pierce and Henry, 2008). There are two major types of brevetoxin compounds with similar but distinct backbone structures. PnTx-2 is the primary intracellular brevetoxin produced by *K. brevis* blooms. However, over time PbTx-2 brevetoxin can be converted to PbTx-3 through metabolic changes (Pierce and Henry, 2008). Researchers found that PbTx-2 has been the primary intracellular brevetoxin that converts over time into PbTx-3 (Pierce and Henry, 2008). When cell ruptures, the extracellular brevetoxins are released into the environment. Some of these substances stay in the ocean while other particles get aerosolized. During offshore winds, brevetoxins can become aerosolized by bubble-mediated transport, causing respiratory irritation, bronchoconstriction, coughing and wheezing among other health problems (Backer et al., 2003). On a windy day, it is best to avoid contact with aerosolized toxin. It has been shown that there is a decrease in respiratory function after only 1 hour of exposure to a *K. brevis* red tide and these symptoms may last for days (Fleming et al., 2005). People with severe or persistent respiratory conditions (such as chronic lung disease or asthma) may experience stronger adverse reactions. The National Oceanic and Atmospheric Administration's National Ocean Service provides a public conditions report identifying possible respiratory irritation impacts in areas affected by red tide (NOAA, 2013).

Food poisoning due to bacterial toxin

Symptoms of food related disease within 1 to 6 hours

The major etiological considerations are *Staphylococcus aureus* and *Bacillus cereus*. *Staphylococcus* food poisoning is characterized by vomiting (82% cases), and diarrhea (68%) but fever is relatively uncommon (16%) (Holmberg and Blake, 1984). More than 99% enterogenic staphylococci associated with food poisoning are coagulase positive, when outbreak caused by enterogenic *Staphylococcus epidermis* has been reported (Breckinridge and Bergdoll, 1971). *Bacillus cereus* can cause two types of food poisoning syndromes, one characterized by nausea and vomiting with

an incubation period of 1 to 6 hours (short incubation "emetic" syndrome) and the second manifests primarily by abdominal cramps and diarrhea with an incubation period of 8 to 16 hours (long incubation "diarrhea" syndrome) (Stenfors-Arnesen et al., 2008).

Food related disease within 8-16 hours

The major etiologic considerations for this syndrome, which is also enterotoxin mediated, are *Clostridium perfringens* and *B. cereus*. Staphylococcal food poisoning and short-incubation *B. cereus* disease are caused by preformed enterotoxin, whereas *C. perfringens* and long incubation *B. cereus* are caused by toxins produced in vivo, which is the reason for the longer incubation period (McDonel, 1979).

Food related disease without bloody diarrhea within 24-48 hours

Noroviruses (formerly Norwalk viruses), are common known foodborne pathogens. They were last estimated to cause 9.2 million foodborne illnesses per year (Mead et al., 1999). Vomiting and diarrhea are often the presenting symptoms with onset 1 to 2 days after exposure. The symptoms progresses to include watery, non-blood diarrhea, abdominal pain and nausea. Vomiting is more common among children, whereas diarrhea is more likely to predominate among adults. However, both symptoms occur in most cases regardless of age (Bresee et al., 2002).

Food related disease with bloody diarrhea within 72-120 hours

The distinctive syndrome of hemorrhagic colitis has been linked to Shiga toxin-producing *E. coli* (STEC), most often serotype O157:H7 (Griffin et al., 1988). The toxin damages the vascular endothelial cells in target organs such as gut and kidney (Lingwood et al., 1988). The illness is characterized by severe abdominal cramping and diarrhea, which is initially watery but may later be grossly bloody (Griffin et al., 1988). The patients with uncomplicated infection remain afebrile. The mean incubation period in outbreaks is 3-8 days. The duration of incubation in uncomplicated cases ranges from 1-12 days. The development of fever and leukocytosis herald hemolytic uremic syndrome (HUS) which is typically diagnosed about 1 week after the beginning of the diarrheal illness, when the diarrhea is resolving. HUS occurs in about 8% of infected individuals and in people of all ages, but its occurrence is highest among children less than 5 years old and the elderly. The fatality rate for HUS in children is 3% to 5%. Mortality rates as high as 16% - 35% have been observed in nursing homes (Brooks et al., 2005).

Diagnosis, treatment, and prevention

Diagnosis

Appropriate specimens for laboratory confirmations vary with the etiologic agents but include feces, vomitus, serum, and blood. In addition, specimens from leftover food, the food preparation environment, and food handlers may be examined. Special laboratory techniques can be used for

identification of *Clostridium perfringens*, *C. vivrios*, *C. jejuni*, *E. coli* O157:H7, *Cylospora*, and *Y. enterocolitica* so that organisms that may appear similar to the normal flora (e.g. other *E. coli*, *B. cereus*) are not overlooked (Lew et al., 1990). To confirm the etiologic agent in outbreaks, specimens should be collected and tested from multiple ill individuals involved in the outbreak. Foods should also be collected and tested to confirm the etiology and source of contamination. Many of the tests used for outbreak identification and confirmation are available only in specialized health or microbiology laboratories (Lew et al., 1990). Although noroviruses are a common cause of foodborne disease; many clinical laboratories are unable to test specimens for these viruses. The current test for noroviruses uses reverse transcription polymerase chain reaction (RT-PCR) (Horwitz et al., 1977). Many public health laboratories can also perform genetic sequencing of PCR products to identify the specific noroviruses strain involved in the outbreak and potentially link multiple cases and environmental sources (Swaminathan et al., 2001).

Treatment

In any diarrheal illness, gastrointestinal fluid losses should be replaced either orally or parenterally. Antimicrobial agents may be used in the treatment of Shigellosis and cholera, and are lifesaving in invasive salmonellosis and typhoid fever, but they should be avoided in uncomplicated gastrointestinal infection caused by non-typhoid salmonellosis (Guerrant et al., 2001). Tetracycline shortens both the duration of clinical cholera and excretion of *Vibrio cholerae* O1. Early treatment of *Campylobacter* infection with fluoroquinolones can shorten the duration of illness (Lew et al., 1990).

Prevention

Prevention of food-related diseases depends on careful handling of raw and finished products all the way from the farm to the table, and on technologies that reduce or eliminate contamination in food (Osterholm and Potter, 1997). A systematic approach to risk reduction called the Hazard Analysis Critical Control Point (HACCP) program originally developed to ensure the safety of foods used in the space program is suitable for determining the safety of seafood. This approach requires a food producer to identify points where the risk of contamination can be controlled, and to use production systems that eliminate the hazards (Tauxe, 2001). Contamination of seafood by *Salmonella*, *Campylobacter*, *C. perfringens*, *Vibriosis*, *Y. enterocolitica*, and other zoonoses is caused by raw food of animal origin, and not infected food handlers. However, poor personal hygiene by food handlers frequently contributes to norovirus, *Staphylococcus*, *Shigella* and hepatitis A outbreaks (Banatvala et al., 1996). Particular care in handling and cooking raw food is important to prevent many foodborne diseases (Banatvala et al., 1996). Since bacterial pathogens grow in food at temperatures ranging from 40°F to 140°F, their growth may be prevented if the food is adequately refrigerated and hot food is held at temperatures higher than 140°F before serving (Banatvala et al., 1996).

Conclusion

Food-related disease is mainly caused by bacterial toxins and histamine fish poisoning (Scombrotoxin), ciguatera, paralytic shellfish poisoning, harmful algal bloom, marine dinoflagellate, and bacterial toxin. Treatment is done with oral hydration and antiemetic and peristaltic drugs. Hazard Analysis Critical Control Point program is effective for ensuring the food safety. Postharvest treatment of seafood deserves special attention from a human health perspective.

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