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Review

Semiology of food poisoning

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Abstract. Food poisoning is a frequently underdiagnosed public health problem, which should be differentiated from foodborne infections or intoxications. Clinical aspects suggesting food poisoning are insufficiently known by the general population and even by the medical world. Food poisoning generates a wide range of diseases with a great variety of symptoms, in the absence of a syndrome characteristic of all these pathological conditions. The symptoms dominating the clinical picture are frequently non-specific, and doctors can misinterpret some clinical signs, particularly in the case of incomplete anamnesis. This review aims to synthesize the clinical elements of food poisoning, without developing paraclinical diagnostic, treatment or prevention elements.

Key Words: semiology, food poisoning, clinical picture

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Introduction

Food poisoning is a pathological condition due to ingestion of food contaminated with toxins (produced by bacteria, fungi, plants) or chemical substances (Morris et al 2013; Davis et al 2008). The essential element in the clinical evaluation of food poisoning is differential diagnosis with foodborne infection or intoxication, in which management is different (Morris et al 2013). Foodborne infection is caused by ingestion of food in which pathogenic organisms have multiplied. Foodborne intoxication is due to pathogens that multiply and produce toxins inside the human body. There are common clinical elements to these pathological conditions, but there are also particular signs and symptoms that help in differentiating them (Roberts 2007). Food poisoning (caused by toxins or chemicals) is induced by microorganisms (bacteria), is the result of chemical contamination, or is due to plants, fungi or fish and seafood. There are four main bacterial strains that cause poisoning: *Clostridium botulinum, Clostridium perfringens, Staphylococcus aureus* and *Bacillus cereus*. Fungal poisoning is due to mycotoxins, of which the best known are aflatoxins from Aspergillus species. Plant toxins are termed phytotoxins. The best known phytotoxins are alkaloids. The most frequent poisoning induced by fish consumption is ciguatera, caused by ciguatoxin (produced by marine algae). Chemicals proven to cause food poisoning include some metals (copper, lead, cadmium, etc.), insecticides/pesticides, and cleaning products (Morris et al 2013; Davis et al 2008; Roberts 2007).

The clinical picture of these various types of poisoning is polymorphic and comprises elements that help in making the certain diagnosis.

Bacterial toxin poisoning

Clostridium botulinum

Human disease due to the *Clostridium botulinum* bacterium is called botulism and is classified as poisoning because it is due to botulinum neurotoxins produced outside the human body by this anaerobic bacterium. Contaminated food is mainly of animal origin, but it can also be of plant origin (processed meat, ham, cheese, canned meat, fish, canned beans etc.) (Popoff 2014).

At 12-36 hours after the consumption of contaminated food, the clinical picture of botulism develops (Glass et al 2013). Its central element is symmetrical flaccid paresis or paralysis with acute onset. Cranial nerves are mainly affected (diplopia, palpebral ptosis and facial paresis), with descending symmetrical progression towards the neck, trunk and limb muscles. Mild forms with muscle weakness alone are also possible (Glass et al 2013; CDC 2016; Lawrence et al 2007). Patients are afebrile and exhibit dysphagia, xerostomia, dysarthria and dysphonia. In severe forms, diaphragmatic paralysis with consecutive respiratory failure develops (McLauchlin et al 2007). Differential diagnosis is made with infant botulism (a foodborne intoxication), wound botulism (particularly in drug addicts), myasthenia gravis, Guillain-Barré syndrome, encephalitis, poliomyelitis, cerebrovascular accidents and chemical poisoning (methyl alcohol, organophosphorus compounds) (McLauchlin et al 2007; Fox et al 2005; Passaro et al 1998).

Clostridium perfringens

Clostridium perfringens is a ubiquitous bacterium, which has 5 subtypes (A-E) capable of producing enterotoxins responsible for animal and human disease (McLauchlin et al 2007). The bacterium needs an anaerobic environment and amino acids for growth, and the foods favoring bacterial multiplication are those

rich in proteins, particularly meat products (37% beef meat) (McLauchlin et al 2007; Heikinheimo et al 2006).
The clinical picture appears as diarrheal disease that occurs 8-24 hours after ingestion of food contaminated with toxins. Profuse diarrhea (usually without mucus or blood) and abdominal pain are the only manifestations of the usually self-limited poisoning (lasting up to 24 hours). Early signs of dehydration may occur. In elderly or immunosuppressed patients, the general state rapidly worsens, resulting in death. The most severe form of poisoning is necrotic enteritis and is caused by *Clostridium perfringens* type C. Untreated necrotic enteritis has a mortality rate higher than 85% (McLauchlin et al 2007; Seike et al 2016). Differential diagnosis is made in the first place with diarrheal syndrome, caused by *Clostridium perfringens* infection, which occurs almost exclusively in elderly patients hospitalized and treated with antibiotics and which is characterized by diarrhea with mucus and blood, unlike food poisoning (Borriello 2005). Any other cause of diarrheal syndrome that evolves with abdominal pain should be excluded (foodborne infections or intoxications).

Staphylococcus aureus
Staphylococcal food poisoning is one of the most frequent forms of food poisoning. *Staphylococcus aureus* is the main enterotoxigenic microorganism of the Staphylococcus genus, which produces the toxins responsible for the development of this form of food poisoning (Hennekinne et al 2012). The foods involved in its appearance are those processed inadequately: meat or fish cooked in poor hygiene conditions, spoiled cheese, but other foods can also be implicated. The disease is frequently seasonal, occurring during summer (particularly in July), because of cooling difficulties encountered in warehouses (Gillespie 2007; Johler et al 2015).
The clinical picture of this poisoning consists of nausea followed by vomiting (usually incoercible), cramping abdominal pain and diarrhea with mucus, without blood. Symptoms occur 2-4 hours after ingestion of contaminated food. In severe cases, under marked dehydration conditions, asthenia, headache and muscle cramps occur. Patients have arterial hypotension. Symptoms last between 6 and 24 hours, and complete recovery is obtained in 24 hours. The sensation of weakness and fatigue may persist for more than 24 hours. Death is extremely rare (Hennekinne et al 2012; Gillespie 2007; Johler et al 2015; Argudin et al 2010). Differential diagnosis is made particularly with enterocolitis caused by staphylococcal colonization in patients treated with antibiotics for a long time, where severe diarrhea with mucus and blood is predominant (Okuyama et al 2012).

Bacillus cereus
Bacillus cereus food poisoning induces two types of clinical syndromes: a clinical syndrome dominated by the presence of diarrhea, and a clinical syndrome dominated by the presence of vomiting (emetic syndrome). The clinical syndrome with the predominance of diarrhea is due to toxins formed after colonization of the human digestive tract; consequently, it is more accurately classified as a foodborne intoxication. The clinical syndrome with the predominance of vomiting is due to a preformed bacterial toxin, and is real poisoning from *Bacillus cereus* (Kramer et al 1989; Granum et al 1997). The foods frequently involved in this poisoning are rice, prepared pasta and dairy products (Logan 2011). About 1-5 hours after ingestion of contaminated food, nausea and vomiting accompanied by cramping epigastric pain appear, which are usually self-limited and last from 6 to 24 hours (usually approximately 12 hours). The differential diagnosis of *Bacillus cereus* toxin poisoning is mainly made with the foodborne intoxication due to the same microorganism, which predominantly manifests as a diarrheal syndrome similar to *Clostridium perfringens* toxin poisoning (Granum et al 1997; Logan 2011; El-Arabi 2013).

Mycotoxin poisoning

Mycotoxins are metabolites of fungi (molds), which cause both animal and human disease. Mycotoxins are toxic products of fungi, not mushrooms. The best known mycotoxins are aflatoxins, ergot derivatives, ochratoxin A, fumonisin, deoxynivalenol and zearalenone (Pitt 2013).

Aflatoxins
Aflatoxins are produced by fungi of the Aspergillus genus (mainly *A. flavus* and *parasiticus*). There are 5 types of aflatoxins. Aflatoxin B1 is the most potent hepatic carcinogen known, particularly in patients with chronic viral hepatitis B. Fungi affect plant cultures in tropical and warm regions (especially peanuts and corn), and aflatoxins are produced in dangerous amounts under inadequate drying or storage conditions (Pitt 2013; Bennett et al 2007).
The clinical picture comprises the symptoms and signs of aflatoxicosis. This occurs as a severe, rare, acute poisoning that leads to death in 25% of the cases, or as a paucisymptomatic chronic poisoning (Williams et al 2004; Peraica et al 1999). Acute poisoning manifests by hemorrhagic necrotic hepatic impairment, the patient presenting an altered general state with asthenia, lethargy, fever, vomiting. Objective examination evidences jaundice of the skin and sclera, coluric urine, lower limb edema and rapidly increasing ascites. Chronic aflatoxicosis manifests particularly in alcohol drinkers (synergistic effect) or in children with viral hepatic disease and malnutrition. Symptoms are non-specific and are due to recurrent infections caused by toxin-induced immunosuppression. Also, chronically exposed persons are susceptible to develop liver carcinoma, presenting with abdominal pain and hepatomegaly. Regarding the differential diagnosis, Reye syndrome (clinical context suggestive of aspirin use, post-viral infections, with the predominance of manifestations due to encephalopathy), and other liver diseases of various causes (autoimmune, viral, alcoholic) should be excluded (Pitt 2013; Williams et al 2004; Peraica et al 1999; Dhanasekaran et al 2011).

Other mycotoxins
Ergot alkaloid poisoning has been known since the Middle Ages as ”Saint Anthony’s fire”. Ergot infecting cereal crops, particularly oats and rye, leads to animal and human poisoning. The symptoms of ergotoxicosis include seizures, paresthesia, diarrhea, headaches, psychosis, nausea and vomiting. During evolution, the effects of peripheral vasoconstriction, with distal trophic disorders, diminution of pulses, edema and gangrene of fingers and toes occur (Peraica et al 1999; Schardl 2015).

Ochratoxin A is a nephrotoxin produced by fungi of Aspergillus and Penicillium species. Cereals and grapes are the most common vectors of transmission to humans. Renal impairment symptoms vary depending on the degree of renal impairment from a mild nephritic syndrome to acute or chronic kidney failure requiring hemodialysis. This poisoning is demonstrated to be involved in the etiopathogenesis of Balkan endemic nephropathy (Pitt 2013; Castegnaro et al 2006).

Fumonisins are toxins produced by Fusarium species, and chronic poisoning has been associated with an increased risk of esophageal neoplasm (Pitt 2013; Zain 2011).

Zearalenone and deoxynivalenol food poisoning is due to high contamination of cereals with toxins produced by fungi of the Fusarium genus. Zearalenone is a mycotoxin that affects humans following the consumption of meat from pigs fed with contaminated cereals. Deoxynivalenol (DON, also known as vomitoxin) is a toxin found in cereal-derived foods. These two toxins are known for their synergistic effect; their coexistence potentiates their harmful effect several times. Zearalenone induces symptoms similar to hyperestrogenism (being a toxin with a structure similar to that of estradiol), and DON causes a syndrome dominated by nausea and vomiting, with associated digestive hemorrhage only in severe forms (Pitt 2013; Peraica et al 1999; Zain 2011). Differential diagnosis between various mycotoxicoses is difficult because of non-specific or subclinical manifestations in mild chronic poisoning and because they are frequently intricate, the synergistic effect potentiating the aggressiveness of toxins (Pitt 2013; Peraica et al 1999; Zain 2011; Pitt 2000).

Phytotoxin poisoning

Phytotoxins are toxins produced by plants. Phytotoxin poisoning occurs due to the ingestion of non-edible plants, to the consumption of insufficiently prepared plants or to plant toxins resistant to the food preparation process. There are 4 main classes of phytotoxins: alkaloids, terpenes, glycosides and phenols (Galal Osman et al 2013; Wittstock et al 2012).

Alkaloids from plants are amino acid derivatives used since ancient times as poisons, narcotics, stimulants or medicines. The best known alkaloids are nicotine, caffeine, morphine, cocaine and atropine. Pyrrolizidine alkaloids are the main plant metabolites that cause poisoning in humans (Galal Osman et al 2013; Stegelmeier et al 1999). The plants producing these metabolites belong to the Crotalaria, Symphytum, Heliotropium and Senecio genera. These alkaloids induce hepatic veno-occlusive disease, with the following signs and symptoms: asthenia, pain in the right hypochondrium, jaundice, edema, ascites and oliguria (Huxtable 2000). Other possible impairments caused by alkaloid poisoning are: pulmonary arterial hypertension and cor pulmonale, with clinical elements characteristic of right cardiac failure; teratogenic and carcinogenic effects (Galal Osman et al 2013).

Terpenes are formed by lipids synthesized from glycolysis products and comprise the majority of essential plant oils. Borneol, menthol, camphor, turpentine, vitamin A and taxanes (used as cytostatic drugs – paclitaxel) are the best known representatives of this class. At the onset of poisoning with these compounds, patients complain of nausea and vomiting. In prolonged and severe poisoning, patients experience seizures (usually single, self-limited episodes), respiratory symptoms due to bronchial aspiration, or cardiac arrhythmias (Poppenga 2010; Kashani et al 2015).

Cyanogenic glycosides induce severe poisoning potentially resulting in death (Galal Osman et al 2013; Yu 2015). The roots of cassava (a plant from South America, Africa) produce the toxins linamarin and lotaustralin. These are the best known cyanides and are responsible for poisoning directly related to the ingested amount. This poisoning causes hepatic impairment (cellular edema, vacuolization) and renal impairment (proteinuria), the plant also containing an agent with goitrogenic potential. Acute toxicity manifests as headache, dizziness, confusional syndrome or mental clouding, seizures, tachypnea, tachycardia, hypotension, cyanosis, and finally, coma and death. Patients have peripheral neuropathy with ataxia or gastrointestinal symptoms (nausea, vomiting). Cyanides are detoxified in the human body in case of mild poisoning and converted to thiocyanate, a compound similar to the iodine molecule. Patients with goiter or cretinism caused by iodine deficiency experience an aggravation of their disease in the context of cyanogenic glycoside poisoning (Vetter 2000; NZFSA 2007). A particular disorder also induced by cassava consumption is konzo, a paralytic disease occurring in foci, in poor rural areas of Africa. Typically, symmetric spastic tetraparesis suddenly develops in the lower limbs, patients walk on tiptoes and exhibit ankle clonus (Tylleskar et al 1992).

Phenol compounds produced by plants are extremely varied and have the property of autoxidation in time. Furanocoumarins, lignins and tannins are representatives of this class. Furanocoumarins are common components of citrus fruits, carrots and some spices. The most important representatives of the class are psoralen, isopsoralen and xanthotoxin. These substances associated with UV radiation are phototoxic, inducing skin lesions (non-painful white spots that will change their color to red, then red-brown) or even skin tumors (Galal Osman et al 2013).

Fish and seafood poisoning

Substances produced by marine organisms may cause poisoning, which induces persistent symptoms and chronic sequelae in about 3% of the cases (Sobel et al 2005). The responsible toxins are produced by algae or bacteria and accumulate in fish, molluscs and more rarely, crustaceans. Toxins do not alter food quality and are frequently resistant to the preparation process. The most important forms of food poisoning caused by the consumption of fish or shellfish are: ciguatera, paralytic shellfish poisoning, diarrhetic shellfish poisoning, neurotoxic shellfish poisoning, tetrodotoxin poisoning, and scombroid poisoning (Sobel et al 2005; Grattan 2013).

Ciguatera is food poisoning due to ciguatoxin produced by algae and transmitted by coral reef fish (e.g., barracuda). Ciguatoxin is a neurotoxin that leads to the inhibition of synaptic transmission, causing neurological symptoms: facial, perioral paresthesia, pain in the limb extremities, headache, reversal of temperature sensation, and myalgia. Patients are cyanotic and complain of insomnia. These symptoms start between 6 hours and 30 hours after consumption of contaminated fish. In half of the patients, the clinical picture starts with gastrointestinal manifestations (abdominal cramps, nausea, vomiting), which precede neurological ones. Patients are hypotensive and bradycardic. Reversal of temperature sensation is characteristic; patients describe cold objects as being warm. Mortality is less than 1%, but in many

patients some symptoms may persist after healing (Sobel et al 2005; Grattan 2013; Dickey et al 2009; Friedman et al 2008; Chan 2014).

Paralytic shellfish poisoning is caused by saxitoxin, a toxin produced by algae and transmitted to men by edible molluscs and mussels captured in cold seas. Saxitoxin blocks voltage-dependent sodium channels in nerve and muscle cell membranes. Symptoms start 30 minutes after ingestion of contaminated food, with a sensation of numbness in the perioral area, spreading to the face and neck. Subsequently, headache, nausea, vomiting and possibly mild diarrhea occur. In case of severe poisoning, diaphragmatic paralysis develops, resulting in death within 2 hours of onset (Sobel et al 2005; Grattan 2013; Hurley et al 2014).

Diarrhetic shellfish poisoning is a self-limited disease with rapid onset, with exclusively gastrointestinal symptoms. The disease is more frequent in Japan and is caused by okadaic acid, produced by certain species of flagellated microorganisms. Symptoms start within 30 minutes of shellfish ingestion, with abdominal pain and diarrhea, and disappear in 3 days, usually without requiring hospitalization (Sobel et al 2005; Grattan 2013; Lloyd et al 2013).

Neurotoxic shellfish poisoning is due to brevetoxin, produced by Karenia brevis, particularly in the warm waters of the Gulf of Mexico. Symptoms appear 3-4 hours after consumption of contaminated shellfish. Patients complain of non-specific gastrointestinal symptoms (nausea, vomiting, diarrhea) and neurological symptoms (oral paresthesia, dysarthria, dizziness or ataxia and walking disorders). Patients rarely need hospitalization and supportive treatment, and symptoms typically disappear within 48 hours of onset (Sobel et al 2005; Grattan 2013; Watkins et al 2008).

Tetrodotoxin poisoning is a potentially lethal poisoning, which occurs after consumption of fugu fish meat (especially on the coast of Japan). The toxin is produced by bacteria in the viscera of this fish and is extremely toxic (0.5 mg toxin are lethal to humans). Symptoms develop within half an hour of ingestion of fugu fish (blowfish or puffer fish) and death can occur rapidly, within 6 hours of ingestion. In the first stage of the poisoning, paresthesia and a sensation of perioral and lingual numbness arise, accompanied by dysphagia, nausea and vomiting. Subsequently, the sensation of numbness progresses, patients developing paresis or paralysis of the extremities, followed by generalized muscle paralysis. Severe forms progress towards respiratory failure, marked hypotension and death. This staging represents the typical form of evolution of tetrodotoxin poisoning. However, patients can also have dizziness, generalized asthenia, chest pain or seizures. Differential diagnosis is made with botulism; the consumption of puffer fish and the differences between neurological symptoms clarify the diagnosis. Thus, in botulism, paresis and paralysis progress in a descending pattern and affect all cranial nerves, while tetrodotoxin affects the head in the mouth area, followed by the trunk and limbs (Sobel et al 2005; Grattan 2013; Bane et al 2014; Ahasan et al 2004; Arakawa et al 2010).

Scombroid poisoning occurs after ingestion of improperly stored fish. The fish involved in the transmission of this poisoning are tuna, mackerel and swordfish. Histidine in fish meat is broken down by bacteria (under improper storage conditions) to histamine. Excess histamine is the factor that mediates the onset of symptoms, which occur within 2 hours of ingestion of contaminated fish meat. Patients experience flushing, skin rash, urticaria, sweating and palpitations. In severe cases, gastrointestinal symptoms (abdominal pain, nausea, vomiting), expiratory dyspnea (due to bronchospasm) or signs due to arterial hypotension are associated. Symptoms remit progressively after 3 to 36 hours. Differential diagnosis is made with food allergies to fish; the certain diagnosis is provided by the suggestive context (fish of the Scombroidae genus, several persons who consumed it having the same symptoms) or by histamine measurements (Sobel et al 2005; Grattan 2013; Berghi 2013).

Chemical food poisoning

Chemical food poisoning is due to a pre-contamination of plants or animals consumed by humans. It comprises a wide range of clinical manifestations, depending on the chemical substance involved in poisoning. The substances that are most frequently involved in the etiology of this type of poisoning are some metals (lead, cadmium, mercury), but there are also cases when toxic chemical products are added during the food preparation process (Gaman et al 1981; Tchounwou et al 2012). Because these types of poisoning are usually occupational and are only rarely due to contaminated food, they are beyond the scope of this review.

Conclusions

Food poisoning comprises a wide range of disorders of various causes, with frequently similar clinical aspects, but also with specific clinical particularities that may help provide clinical diagnosis. Correct clinical diagnosis will facilitate the performance of additional targeted investigations and will accelerate the making of the certain diagnosis. Consequently, clinical elements are essential for the knowledge of food poisoning and represent a mainstay for doctors in finalizing their diagnostic approach.

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