Concept of Virrudhahar - A Lifestyle Factor for Microbial Food Poisoning

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Abstract:
A drastic ation and In India peoples are apply modern culture and their diet also. The improper dietary intake can cause food poisoning. According to Ayurveda if a person taking appropriate diet which he needs then and then only person can live a Healthy life. In Charka samhita there is mentioned jatharagnibala and aaharmatra. If a person taking more quantity of food excepting of his poor Jatharagnibala then person should survive some aaharjanyavikara. There are eighteen types of virrudhaaahar. Deshavirrudha, Kala virrudha, Agni virrudha, Matravirrudha, Satmyavirrudha, Doshavirrudha, Sanskarvirrudha, Viryavirrudha, Avasthavirrudha, Kramavirrudha, Pariharvirrudha, Upacharvirrudha, Pakavirrudha, Sanyogvirudhha, Hridayaviruddha, Sampadviruddha, vidhiviruddha. In today's fast life lots of changes in dietary schedules. In India peoples are apply modern culture and their diet also. The improper dietary intake can cause food poisoning.

Introduction:
According to Ayurveda if a person taking appropriate diet which he needs then and then only person can live a Healthy life. In Charka samhita there is mentioned jatharagnibala and aaharmatra. If a person taking more quantity of food excepting of his poor Jatharagnibala then person should survive some aaharjanyavikara. In Aatreybhadrakapeyaadhaya asthodasha viruddha ahar mentioned If a person can seven virrudhaaaharjanyavikaras can be form. There are eighteen types of virrudhaaahar, Deshaviruddha, Kala viruddha, Agni viruddha, Matraviruddha, Satmyaviruddha, Doshaviruddha, Sanskarviruddha, Viryaviruddha, Avasthaviruddha, Kramaviruddha, Pariharviruddha, Upacharviruddha, Pakaviruddha, Sanyogviruddha, Hridayaviruddha, Sampadviruddha, vidhiviruddha. These types of virrudhaaahar can causes vividha Aaharjanyasamanyarogas like Aamvisha (Visuchika, Alasak, Vilambika), Chardi, Visarpa, Pandu, Grahaniroga, Aamlpitta, Jwara, Pinas, Kushtha, murcha etc.

As compared to today's dietary habit to virrudha ahar. There is lots of similarities are found. Because in today's fast life lots of dietary schedules. In India peoples are apply modern culture and their diet also. The improper dietary intake can cause food poisoning.

Food Poisoning:
Food poisoning can occur in many ways, and may be isolated instances, or may constitute an epidemic. Food born illness are among the commonest health problems encountered world-wide and are particularly rampant in third world countries such as India mainly due to a relative lack of sanitation and public hygiene. Food poisoning syndrome results from ingestion of water and wide variety of food contaminated with pathogenic organisms (bacteria, viruses, parasites, and fungi), their toxin and chemicals. Food poisoning must be suspected when an acute illness with gastrointestinal or neurological manifestation affect two or more persons or animals who have shared a meal during the previous 72 hours. The term generally used to encourage both food related infection and food related intoxication. In microbial food poisoning, the microbes multiply readily in the food prior to consumption, whereas in food born infection, food is merely the vector for microbes that do not grow on their transient substrate. Other considers food poisoning heen and teen types virrudha Sanskar as intoxication of food by chemicals or toxins from bacteria or fungi. Food borne illness commonly called food poisoning, it's caused by pathogens or certain chemicals present in ingested food bacteria, viruses, worms and protozoa which diseases are all pathogens, although there are also harmless and beneficial bacteria that are used to make yogurt and cheese. Some chemicals that causes food borne illness are natural components of food, while other may be accidentally added during production and processing either through carelessness or pollution. The main causes of food borne illness are bacteria (66%), chemicals (26 %), virus (4%) and parasites (4%).

Salmonella
Salmonella species are motile gram-negative rods, and grow both aerobically at anoptimum temperature of 37 degree e, and pH between 4 and 8. Commonest species involved include Heidelberg s.agona, s.arizonae, s.hadr, s.enertidis, sjava, s.muenchen, s.paratyphi B, s.virchow, s. Indiana, s. Anatum.
Pathogenesis
Salmonella often enter the host by ingestion, and even with several system to mediate acid resistance, few survive the stomach and move into the small intestine. Normal flora protects against colonization of administration of oral antibiotics facilitates establishment of infection. Entry of salmonella usually occurs without mucosal damage in systemic infections, but enteric infection is characterized by local damage without septicemia-salmonella infection with M cells in peyer’s patches is facilitated by fimbrial adhesions.

Symptoms:

The incubation period of the salmonella is 12-36 hours. The clinical sign includes diarrhoea, which may be watery, greenish and foul smelling. This may be preceded by headache and chills Other findings include prostration, muscle weakness and moderate fever. In most cases the symptoms resolve in 2-3 days without any complication. Symptoms are usually gastrointestinal, including nausea, vomiting, abdominal cramps and bloody diarrhea with mucous, headache, fatigue and rose spots are also possible. These symptoms can be severe, especially in young children and elderly Symptoms last generally up to a week, and can appear 12-72 hours after ingesting the bacterium. Reactive arthritis, sickle-cell anaemia and osteomyelitis due to salmonella infection is much more common that in the general population.

Detection of pathogens
Eggs-Even unbroken, unsoiled eggs can be contaminated. Raw eggs, raw eggs-based milk shakes, Caesar salads, homemade ice creams. Milk and milk products.

According to Ayurveda- an correlate with Agni virrudha, sanyogvirrudha, Sanskarvirrudha, Pakavirrudha. Staphylococcus aureus:

Etiology- Staphylococcus aureus is gram positive cocci that occur in singles, short chains, tetrads and irregular grape like cluster Only the strains that produce enterotoxin can cause food poisoning The food handler with an active lesion or carriage later initiates infection.

Pathogenesis Most food poisoning is caused by enterotoxin A followed by type D. These enterotoxins are heat stable, with type B being most heat resistant. Enterotoxin stimulates Central Nervous Systems (CNS) vomiting centre and inhibit water and sodium absorption in the small intestine. Staphylococcal enterotoxins, along with the toxic syndrome toxin and others, are classed as bacterial super antigens relative to in vivo antigen recognition in contrast to conventional antigens.

Symptoms-Food poisoning by S. aureus is characterized by a short incubation period typically 2-4 hours. The onset is sudden and is characterized by vomiting and diarrhoea but no fever. The illness lasts less than 12 hours. In severe cases dehydration, masked pallor and collapse may require treatment (intravenously) infusion. The short incubation periods are the characteristics of intoxication where illness is the results of ingestion of the preformed toxin in the food.

Detection of the organisms - Most food (particularly high in protein) will support staphylococcus aureus. The presence of a large number of staphylococcus growth. Especially custard cream filled in pastries, mayonnaise, dairy products, potato, egg products. Other organism like mushroom, rice, noodles, salad, cooked food keep in room temperature. These organism in a food indicate poor handling or sanitation. The dilution is placed on baird-parker agar or manniot salt agar. The enterotoxin can be detected and identified by gel diffusion.

According to Ayurveda Can be correlate with Deshvирудха, satmyavирудха, Kramvирудха, Avasthavирудха Agni virrudha .

Clostridium botulinum - Etiology-Clostridium botulinum is gram positive anaerobic spore bearing bacilli that is widely distributed in soil, sediments of lakes, ponds and decaying vegetations. Seven different strains of the organisms (A-G) are classified based on serologic specificity and another neurotoxin. Pathogenesis: During their growth C. botulinum strains produce high potent neurotoxin that cause neuroparalytic disease known as botulism in humans and animals without the development of histological lesion Botulism may lead to death due to respiratory muscle paralysis unless treated properly. Botulism toxin is absorbed from the glandular stomach and anterior small intestine or the wound and carried through the blood stream bind to receptor and enters the nerve cell after receptor mediated endocytosis to peripheral cholinergic nerve terminals including neuromuscular junction postganglionic parasympathetic nerve ending and peripheral ganglion.

Symptoms: The incubation of C. botulinum is 12-36 hours. The most common features include vomiting, thirst, dryness of mouth, constipation, ocular paresis (blurred-vision), difficulty in speaking breathing and swallowing. Death occurs due to respiratory paralysis within 7 days). Clinically, botulism recognized as a lower motor neuron disease resulting progressive flaccid paralysis Although deficient’s in somatic neuro-muscular transmission are the most prominent effects, the motor deficient’s in cranial
nerve function, as well as the autonomic nervous system have also been reported.

**Detection of toxin:** Foodborne botulism results form- Canned meat and meat products, fruits, vegetables pickles, fish. On opening the can w peculiar testing contents should raise the suspicion of botulism.

**According to Ayurveda:** Can be correlated vidhivirrudha.

**Escherichia coli :**

**Etiology** Escherichia coli (E. coli) are bacteria which belongsto family enterobacteriaeae and are gram negative rod up to 3 um in length, ferment glucose and wide range of sugars. These lactase fermenters produce pink colonies on Mcconkey agar. Haemolytic activity on blood agar is characteristics of certain strain of E. coli.

**Pathogenesis:** Enterohemorrhagic E. coli (EHEC) strain may produce one or more types of cytotoxins which are collectively referred as shiga- like toxins (SLTs) since they are antigenically and functionally similar to shiga toxin produced by shigelladsynteria. SLTS were previously known as verotoxin. In animals, toxin has been shown to cause danders the heralction, pheral localized fluid accumulation and colonic lesion characterized by sloughing of surface and crypt epithelial cell.

**Symptoms:** The incubation period is 72-120 hours. The clinical sign initially may be diarrhoea with abdominal cramps, which may turn into grossly bloody diarrhoea in a few days. There is however, no fever The symptons of E. coli septicemia are mainly referable to bacteremia, end toxemia and the effect of bacteria localization in a variety of tissue spaces throughout the body.

**Detection of toxin-** Contaminated food; Inadequately cooked beef, raw milk, contaminated water. **According to Ayurveda-** Can be correlated with the Agni virrudha, satmyavirrudha.

**Shigelllosis:**

**Aetiology**- Shigella is a species of enteric bacteria that causes disease in humans and other primates (Shigella is gram-negative rods that are non-motile and non-spore forming. The bacteria are primarily a human disease, but has been found in some primates Shigellas are facultative anaerobes, similar to enteric such as E.coli

**Pathogenesis**- Shigella attaches to and penetrate intestinal cell walls of the small intestines by producing toxins that may promote the diarrhoea characteristic of the disease. The Shiga toxin enables the bacteria to penetrate the epithelial lining of the intestines, leading to a breakdown of the lining and haemorrhage. Shigellas also have adhesions that promote binding to epithelial cell surfaces and invasion plasmid antigens that allow the bacteria to enter target cells, thus increasing its virulence

**Symptoms:** Abdominal pain, cramps, diarrhoea, fever, vomiting, blood, pus or mucus in stools and tenesmus are the common symptoms. Mild infections cause low-grade fever (about 100.4 to 102°F [38 to 38.9°C] and watery diarrhoea 1 to 2 days after people ingest the bacteria. Abdominal cramps and a frequent urge to defecate are common with more severe infections. Severe infections may cause low-grade or moderate fever and watery diarrhoea that progress to dysentery.

**Detection of toxin:** Shigella infection is diagnosed through testing of a stool sample Main sources are Fruits, Vegetables, Milk, Vegetables (Potato salads, mashed potato), tossed salads and milk products **According to Ayurveda-** Can becorrelated with matravirrudha, Agni virrudha.

**Conclusion:**

Bacterial food borne illnesses are among the widest spred global public health problems of recent times, and their implication for health and economy is increasingly recognized. However, the true incidence of bacterial food borne illnesses are unknown for number of reasons, including poor responses from victims duringinterviews with health officials, misdiagnosis of the illness inadequate collection of samples for laboratory analysis and improper laboratory examination. The presence of various pathogenic bacteria in different foods poses a health hazard and rise concerns about the safety of these food products. **According to AyurvedaViridiana also causes vividhaaharajrogas.** We can correlate to the viruddhaahar is one of the causes of food poisoning In addition, there is a need to implement strict hygienic measures inthe manufacturing, handling, storage and selling of food in order to guarantee the quality of these foods so as to minimize or eliminate the risk of FBI. To the best of our knowledge, this is the first review on major bacteria that causes FBI.

**References**

3) Center for Disease Control and Prevention (2011) Estimates of Foodborne Illness in the United States
5) https://www.cde.gov/
7) Ananthanarayan and paniker's textbook of pathology, 10 edition Pg e are cause gressmple. otato with no. 279, 305
9) Ananthanarayan and paniker's textbook of pathology, 10 edition Pg no Chapter no27. 10h edition. Pg no 268.