



FOOD POISONING management

BY

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General Medicine (PG)

FOOD POISONING

- Food poisoning is an acute gastroenteritis caused by the ingestion of the food or drink contaminated with either living bacteria or their toxins or inorganic chemical substances and poison delivered from plant and animals

DIAGNOSING FOOD POISONING

- History of outbreak
- History of exposure to contaminated food
- Detailed clinical history
- Rectal examination
- Stool examination
- Blood examination

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- Isolation rate of pathogen from stool 3%
 - Stool leukocytes is inexpensive test to differentiate infectious vs non- infectious types
 - Sigmoidoscopy indicated for Proctitis, C diff.

Character of diarrhea also helps guide evaluation

- **Non-infectious**

- Watery diarrhea, no blood or mucus or pus in stool, no fever or systemic signs
- Secretory or osmotic mechanism
- Dehydration may occur
- Generally self-limited and more benign
- Therapy generally supportive

- **Infectious**

- Frequent lower volume stool, mucoid, bloody, or purulent. Often with fever or systemic signs, tenesmus, urgency
- Exudative mechanism
- Dehydration rare
- Less benign
- Specific therapy may be indicated

Aims/Goals of management


- Prevent, identify and treat dehydration
- Eradicate causative pathogens
 - By Antibiotics
- Prevent spread by early recognition and institution of infection-control measures
 - immunization, chemoprophylaxis, good hygiene, improve sanitation

Treatment of Dehydration

- **Oral rehydration therapy**
 - Oral Rehydration Salt –standard or reduced osmolarity
 - Home solutions
- **Intravenous therapy**
 - Ringer's Lactate solution (Hartmann's soln)
 - Normal saline/ Half normal saline with 5-10% glucose
 - Half strength Darrow's solution

Assessment of severity of dehydration

Degree of dehydration	Plan A: No dehydration	Plan B: Some dehydration	Plan C: Severe dehydration
General condition	Calm, alert	Restless irritable	Lethargic, unconscious
Eye manifestation	Normal	Sunken	Sunken
Ability to drink	Normal	Thirsty, eager to drink	Poor
Skin pinch	Goes back quickly	Slowly	Very slowly
Loss of fluid approximately	<2 litres	2-3 litres	>3 litres

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- Mild dehydration can be treated in a primary care, by giving ORS.
 - 2 litres of ORT in the first 24 hours,
 - unrestricted normal fluids with 200 ml of ORT for every loose stool or vomit
 - Moderate / severe dehydration is an indication for admission.

Oral Rehydration Therapy

- Oral Rehydration Therapy today is at core of management of diarrhoea for prevention of dehydration and treatment of mild dehydration
- Term ORT includes
 1. Oral rehydration salt solution recommended by WHO
 2. Solutions made from sugar(40g) and salt(4g)
 3. Food based solutions(50g)
 4. Home fluids without insisting on both glucose and salt
 - Plain water, lemon water, coconut water, soups

Comparison between low osmolarity ORS and WHO-ORS

Ingredients of ORS solutions	Low osmolarity 245 mmol/L	WHO-ORS (old) 311mol/L
sodium	75	90
potassium	20	20
chloride	65	80
citrate	10	10
glucose	75	111

severe dehydration treatment

- RL OR NS - 10-20 ml/kg or 1 to 2 liter IV given in the first hour
- May repeat bolus until circulation stable
- Maintenance given as
 - 50-100 ml
 - + urine output per hour
 - + ongoing losses per hour

or

Calculate 24 hour maintenance requirements

- Formula:
 - First 10 kg: (100 cc/kg/24 hours)
 - Second 10 kg: (50 cc/kg/24 hours)
 - Remainder: (20 cc/kg/24 hours)

Example: 35 Kilogram

- Daily: 1000 cc + 500 cc + 300 cc = 1800 cc/day



ROLE OF ANTIBIOTICS:

- Indicated in bacterial diarrheas

INDICATIONS:

- Presence of crampy abdominal pain with fever
- Presence of dysentery
- Persistence of diarrhea after 48hrs
- Early onset severe diarrhea
- Rice water stools

Why not treat everyone with antibiotics?

- Some have no effective specific treatment
- Treatment may not change disease duration or severity
- Treatment may predispose to carrier state
- Treatment may produce complications (HUS, antibiotic resistance, C. difficile, toxic megacolon)

Antibiotics of choice

- Empirical treatment
 - Ciprofloxacin 500mg two times a day for 3-5 days and Metronidazole 400mg two times a day for 7 days for suspected giardiasis
- Invasive bacterial Enteritis- esp. Shigellae
 - Quinolone orally twice daily for 3 days
 - Pevmecillinam 100mg 4 times/day PO and Azithromycin are second line drugs
- Traveller's diarrhea
 - Prophylactic- not recommended
 - A single dose of oral Quinolone at onset

Antibiotics of choice

- Clostridium difficile
 - Metronidazole 500mg tid for 10-14 days PO
 - Vancomycin 125 mg qid for 10-14 days PO
- E. coli
 - Quinolone
- Vibrio cholera
 - A single dose of Doxycycline 300mg PO
 - Tetracycline 12.5mg/kg four times a day for 3days
 - Pregnant women and children are treated with erythromycin or azithromycin

Antibiotics of choice

- Salmonella
 - Ciprofloxacin 500mg bid PO for 5-7 days or TMP-SMX (160mg bid PO for three days)
- Campylobacter
 - Erythromycin 250 mg 4 times a day PO for 5-7 days or Ciprofloxacin 500mg bid PO for 5-7 days
- Yersinia
 - tetracycline, TMP-SMX,.

VIRAL FOOD POISONING:

- No role of antibiotics / antiviral drugs
- Supportive management

PARASITIC FOOD POISONING:

- Giardiasis , cryptosporidium – Metronidazole
- Toxoplasma – clotrimaxazole

FISH POISONING:

- Supportive management




Antimotility agents

- Should be avoided
- Concern for promoting bacterial invasion or prolonging the infection

Clostridium Botulinum

- **Signs and Symptoms**
- The distinctive clinical syndrome of botulism consists of symmetric cranial nerve palsies followed by symmetric descending flaccid paralysis that may progress to respiratory arrest and death.
- In *food-borne botulism*, the incubation period from ingestion of food containing botulinum toxin to onset of symptoms is usually 18–36 h but, depending on the toxin dose, can range from a few hours to several days.
- The extent of paralysis (from a few cranial nerves only to quadriplegia) also depends on the toxin dose.
- The illness ranges from a mild condition for which no medical advice is sought to severe disease that can result in death within 24hrs

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- Cranial nerve involvement, which almost always marks the onset of symptoms of botulism, usually produces diplopia, dysarthria, dysphonia, and/or dysphagia.
 - In food-borne botulism, nausea, vomiting, and abdominal pain may precede or follow the onset of paralysis. Constipation due to paralytic ileus and urinary retention is also common.
 - Extraocular muscle paralysis manifests as blurred vision or diplopia and an inability to accommodate near vision. Ptosis and facial paralysis are frequent; the pupillary reflexes may be depressed, and fixed or dilated pupils are noted in half of patients


Clostridium Botulinum

- With disease progression
 - descending paralysis
 - respiratory weakness
 - respiratory failure
 - oculobulbar symptoms

Botulism:

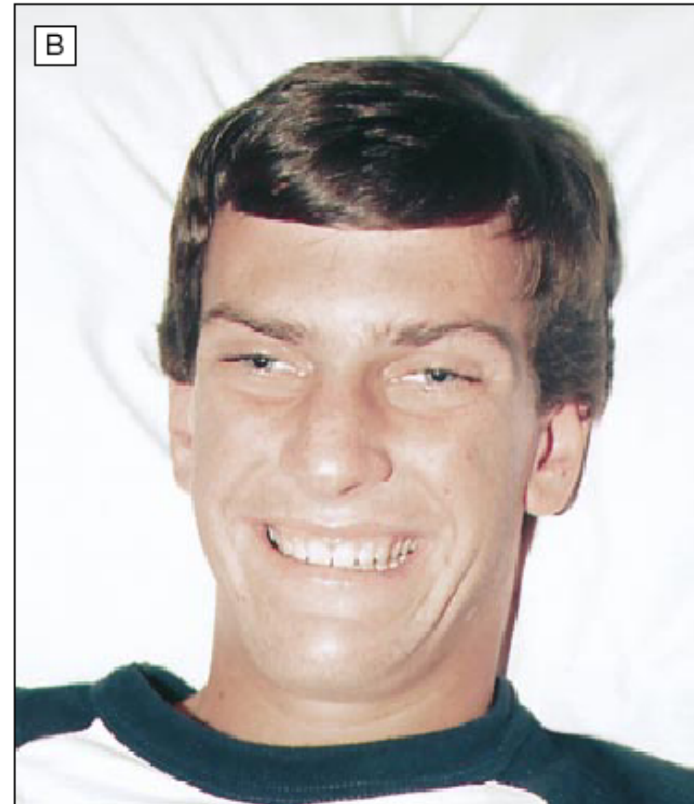
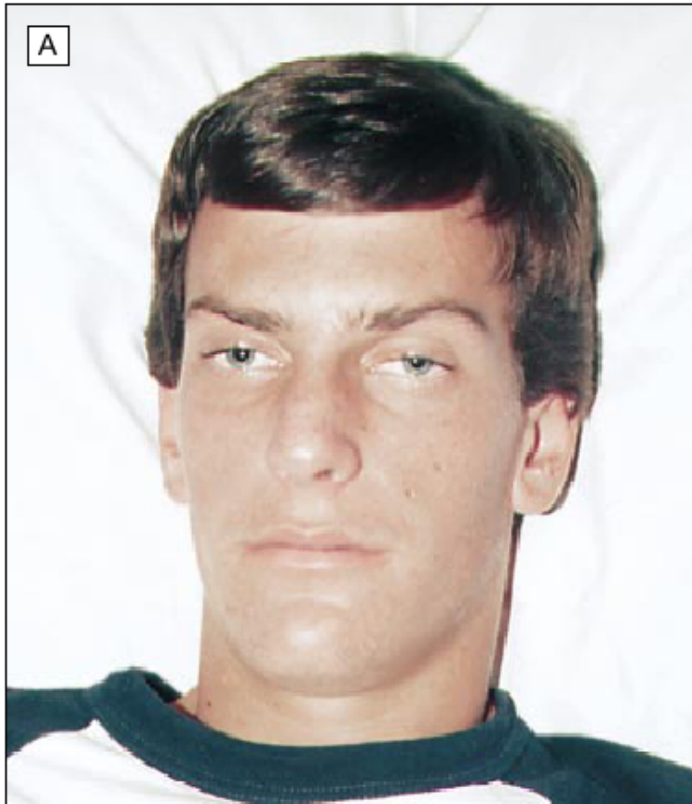
Differential Diagnoses

- Neuromuscular disorders
 - Stroke syndrome
 - Myasthenia gravis
 - Guillain-Barre syndrome (Miller-Fisher variant)
 - Tick paralysis
 - Atropine poisoning
 - Paralytic shellfish/puffer fish poisoning
- Diagnosis based on history, clinical presentation and confirmation by the demonstration of toxin in clinical specimens (serum, stool, sterile water or saline enema, gastric aspirates, wound material) or in samples of ingested foods.

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- The cornerstones of treatment for botulism are meticulous intensive care and immediate administration of botulinum antitoxin
 - The decision to administer botulinum antitoxin—the only specific treatment—must be based on a clinical diagnosis and cannot be postponed while laboratory confirmation is awaited.
 - stabilization of airway
 - upper and lower GI decontamination
 - trivalent antitoxin
 - call for CDC (centre for disease control)

Botulism: Treatment/Prophylaxis

- **Ventilatory assistance and supportive care**
- **Botulinum antitoxin**
 - **Trivalent equine product against types A,B, and E available from CDC**
 - **Most effective if given early**
- **Antibiotics for wound botulism**
 - **Penicillin**
- **Recovery may be prolonged with supportive care necessary**

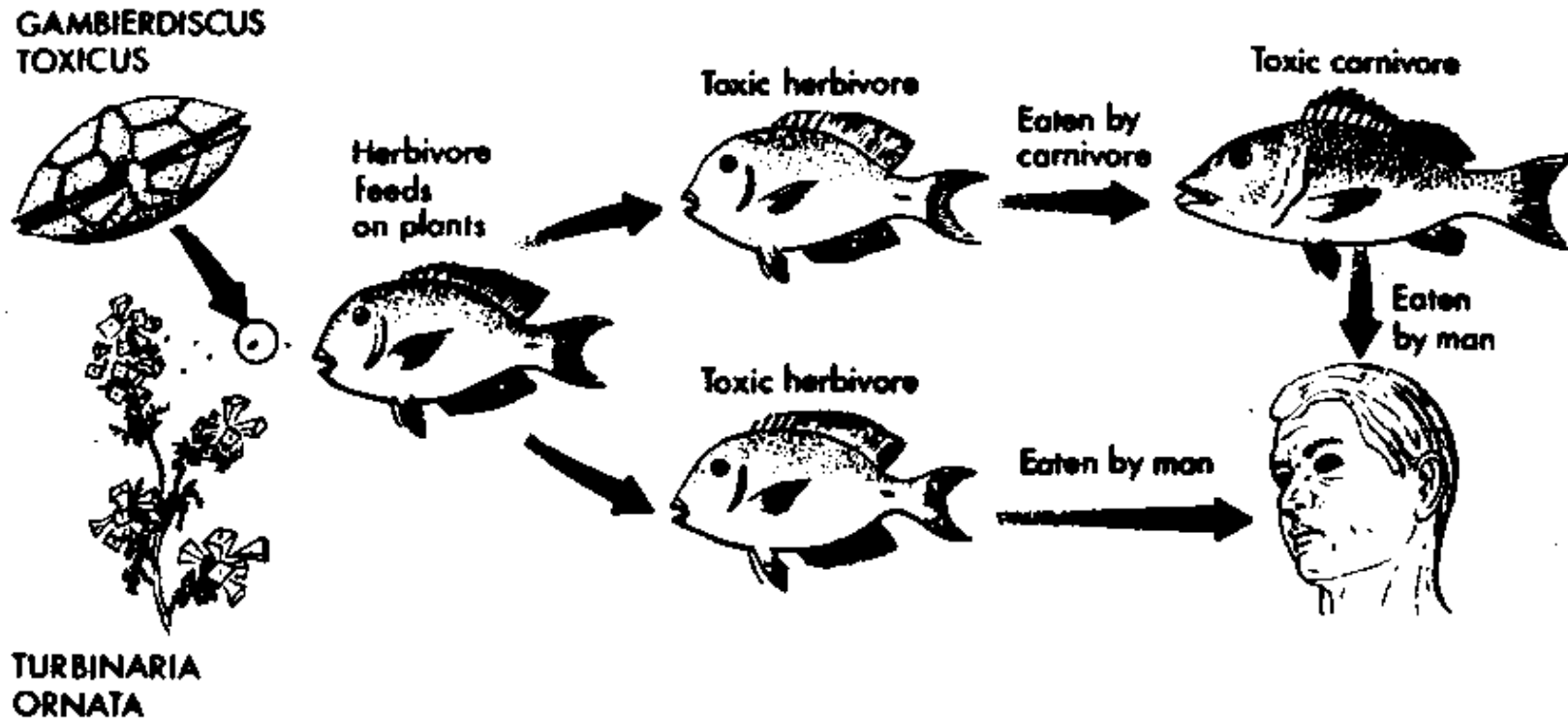


A, Patient at rest. Note bilateral mild ptosis, dilated pupils, disconjugate gaze, and symmetric facial muscles. B, Patient was requested to perform his maximum smile. Note absent periorbital smile creases, ptosis, disconjugate gaze, dilated pupils, and minimally asymmetric smile. As an indication of the extreme potency of botulinum toxin, the patient had 40×10^{-12} g/mL of type A botulinum toxin in his serum (ie, 1.25 mouse units/mL) when these photographs were taken.

Ciguatera poisoning

- Ciguatera syndrome is due to sodium channel activator toxins that originate in photosynthetic dinoflagellates (*Gambierdiscus toxicus*) and accumulate in the food chain
- Three major toxins are found in the flesh and viscera of ciguateric fishes CTX-1,2,3
- TRPV1 a non selective cation channel expressed in nociceptive neurons may play a role in the unique neurologic disturbances in ciguatera poisoning
- Most common reported fish poisoning
- Hawaii and Florida report 90% of all cases

Ciguatera poisoning



Ciguatera poison


- Ciguatoxins are unaffected by freeze-drying, heat, cold and gastric acid
- Heat stable
- Odorless
- Acid stable
- Tasteless
- Pathognomonic symptom is reversal of hot and cold tactile perception which develops in some persons after 3-5 days and may last for months

Ciguatera poisoning

- Diagnosis
 - Mostly made on clinical grounds
 - ELISA or Radioimmuno assay for Ciguatoxin
 - High-performance liquid chromatography (HPLC)

TREATMENT

- Therapy is supportive and is based on symptoms
- Nausea and vomiting controlled with antiemetic such as
 - Inj Ondansetron 4-8 mg IV
- Hypotension may require the administration of
 - IV crystalloid and pressor drug(Dobutamine, Nor adrenaline, dopamine)
- Cool showers or Hydroxyzine 25 mg po every 6-8 hrs for pruritis
- Amitriptyline(25 mg po twice a day) for dysesthesias
 - Mannitol 1g/kg IV over 45 minutes for the relief of distressing neurologic symptoms.
 - If symptoms improve second dose given with in 3-4 hrs
 - Mannitol reverses ciguatoxin induced schwann cell edema

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- During recovery from the poisoning victim should exclude following from the diet
 - Fish and Fish sauces
 - Alcoholic beverages
 - Nuts and nut oils

SCROMBOID

“Mahimahi Flush”

- This poisoning is entirely preventable with adequate refrigeration soon after it is caught.
- Inadequate preservation or refrigeration, the musculature of these fishes undergoes bacterial decomposition, which includes decarboxylation of amino acid l-histidine to histamine, histamine phosphate, histamine chloride
- The bacteria responsible for decomposition
 - Morganella morganli*
 - Escherichia Coli*
 - Kiebsiella pneumonia*
- Cooking the fish will not destroy the histamine

SCROMBOID POISONING

Symptoms

- The onset of symptoms is usually within minutes of the ingestion
- Headache
- Flush
- Diarrhea
- Diaphoresis
- Abdominal Cramps
- Pruritis & Urticaria

SCROMBOID POISONING

Laboratory & Treatment

- Histamine levels in the fish correlate with toxicity
 - 0.1 mg/100mg = Non-toxic
 - 1.0 mg/100 mg = Toxic
- Intravenous Diphenhydramine
- Intravenous Cimetidine
- Rehydration
- Once treated, symptoms do not reappear

SHELLFISH POISONING

- Paralytic shellfish poisoning is induced by ingestion of aquacultured filter-feeding organisms, including clams, oysters, scallops, mussels, chitons, limpets, starfish, and sand crabs.
- Toxin originate in photosynthetic dinoflagellates(e.g., *Protogonyaulax*, *Ptychodiscus*, and *Gymnodinium*) and protozoan organisms and accumulate in the food chain
- The paralytic shellfish toxins are water-soluble as well as heat- and acid-stable; they cannot be destroyed by ordinary cooking.
- The best-characterized and most frequently identified paralytic shellfish toxin is saxitoxin,

SHELLFISH POISONING

- Saxitoxin appears to block sodium conductance, inhibiting neuromuscular transmission at the axonal and muscle membrane levels.
- A toxin concentration of >75 g/100 g of foodstuff is considered hazardous to humans.
- In the 1972 New England "red tide," the concentration of saxitoxin in blue mussels exceeded 9000 g/100 g of foodstuff

Treatment of shell fish poisoning

- Treatment is supportive and is based on symptoms.
- If the victim comes to medical attention within the first few hours after poison ingestion, the stomach should be emptied by gastric lavage and then irrigated with 2 L of 2% sodium bicarbonate
- Activated charcoal (50–100 g) (shellfish toxins are proved to bind well to charcoal)
- The most serious problem is respiratory paralysis. With prompt recognition of ventilatory failure, endotracheal intubation and assisted ventilation prevent anoxic myocardial and brain injury.

Mushroom poisoning

- Mushroom poisoning management and prognosis can be determined by the history and the geographic origin of the mushroom, the initial signs and symptoms, the organ system or systems involved, and coexistent factors or conditions.
- Groups of toxins are identifiable as cyclopeptides, gyromitrin, muscarine, coprine, ibotenic acid and muscimol, psilocybin, general GI irritants, orellinine, allenic norleucine, acromelic acids, and myotoxins.

Amanita mushrooms

- Survival rates in case series of variable numbers of patients poisoned by *A. phalloides* who received any of the following: supportive care, fluid and electrolyte repletion, high-dose penicillin G, dexamethasone, and thioctic acid, are between 70% and 100%.
- Emesis, lavage, and catharsis are not necessary unless the patient is seen within several hours after the ingestion, because the toxin usually induces emesis and catharsis.
- Activated charcoal is safe, logical, and a valuable therapeutic strategy.
- Forced diuresis, hemodialysis, plasmapheresis, hemofiltration, and hemoperfusion may be effective shortly after ingestion

- Fluid and electrolyte repletion and treatment of hepatic compromise are essential.
- Intravenous 0.9% sodium chloride solution and electrolytes usually are necessary because of substantial fluid loss due to vomiting and diarrhea
- *N*-acetylcysteine, benzylpenicillin, cimetidine, thiocetic acid, and silybin used treat hepatic necrosis caused by amatoxins and cyclopeptides

Gyromitra mushrooms

- *Gyromitra* mushrooms contain gyromitrin. which on hydrolysis splits into acetaldehyde and *N*-methyl-*N*-formyl hydrazine.
- The hydrazine moiety reacts with pyridoxine, resulting in inhibition of pyridoxal phosphate-related enzymatic reactions. This interference with pyridoxal phosphate disrupts the function of the inhibitory neurotransmitter γ -aminobutyric acid (GABA).
- The implications of this decrease in GABA, which is thought to contribute to intractable seizures

TREATMENT

- Activated charcoal 1 g/kg body weight should be given.
- Benzodiazepines are appropriate for initial management of seizures.
- Under most circumstances, supportive care is adequate treatment.
- Pyridoxine in doses of 70 mg/kg IV up to 5 g in an adult may be useful in limiting seizures

clitocybe mushrooms

- Clitocybe mushrooms contain muscarine, which is similar to acetylcholine and have comparable clinical effects at the muscarinic receptors.
- Peripheral manifestations typically include bradycardia, miosis, salivation, lacrimation, vomiting, diarrhea, bronchospasm, bronchorrhea, and micturition.
- Central muscarinic manifestations do not occur because muscarine, a quaternary ammonium compound, does not cross the blood–brain barrier.
- No nicotinic manifestations such as diaphoresis or tremor occur.
- The effects of muscarine often last longer than those of acetylcholine. Because muscarine lacks an ester bond, it is not susceptible to acetylcholinesterase hydrolysis

Treatment

- atropine (1–2 mg given IV slowly for adults or 0.02 mg/kg with a minimum of 0.1 mg IV for children) can be titrated and repeated as frequently as indicated to reverse symptomatology



Food

- Do not withhold
 - Withholding food, even for one or two days, greatly exacerbates the malnutrition



THANK U