FOOD POISONING

CONTENTS

INTRODUCTION

- Food poisoning is an acute illness caused by the consumption of food or drinks contaminated with pathogens, biochemical toxins or toxic chemicals.
- Affected people usually show gastrointestinal symptoms
- The incubation period varies from hours to days depending on the causative agent.

CAUSES

i. Microbes: bacteria, viruses, protozoa
ii. Parasites
iii. Fungi
iv. Plants
v. Fish
vi. Chemical Additives

DIAGNOSIS

- History.
- Clinical picture
- Stool analysis
- Suspect food/agent analysis.
- Measurement of serum electrolytes.

GENERAL TREATMENT MEASURES

- **Oral Rehydration Therapy (ORT)**

mild dehydration (3 to 5% fluid deficit), or moderate dehydration (6 to 10% fluid deficit).

Rehydration should commence with a fluid containing 50 to 90 mEq/L of sodium.

The amount of fluid administered should be 50 ml/kg over a period of 2 to 4 hours in mild dehydration and 100 ml/kg in moderate dehydration.

After 2 to 4 hours, hydration status should be assessed and if found to be normal, maintenance therapy can be begun, otherwise rehydration therapy is repeated.

Maintenance therapy—Oral rehydration solutions (ORS) should be administered as follows:

- 1 ml for each gram of diarrheal stool, or
- 10 ml/kg for every watery stool passed, or
- 2 ml/kg for each episode of vomiting.

Limitations: It is not sufficient therapy in the presence of dysentery (bloody diarrhea), shock, intestinal ileus, intractable vomiting, high stool output (>10 ml/kg/hr), monosaccharide malabsorption and lactose intolerance.

- **Intravenous Rehydration**

This is necessary when dehydration is severe (> 10% fluid deficit or shock)

20 ml/kg boluses of Ringer's lactate, normal saline, or similar solution is administered until pulse, perfusion and mental status return to normal.

Oral rehydration is commenced when condition improves.

- **Non-specific antidiarrheal agents**

Use of such agents such as kaolin-pectin, anti-motility drugs (e.g. loperamide), anti-secretory drugs, or toxin binders (e.g. cholestyramine), is controversial. Available data do not demonstrate significant beneficial effects.

Instead, serious adverse effects can occur, including ileus and anticholinergic syndrome.

PREVENTION OF FOOD POISONING

- Buy groceries only from hygienic outlets and store immediately in the refrigerator.
- Do not buy items whose expiry date has elapsed or containing undercooked or raw animal derived ingredients.
- Buy only pasteurized milk or cheese.
- Do not buy eggs which are cracked or leaking . Place eggs inside cartons and store them in the main section of the refrigerator.
- Always store raw meat, poultry, or seafood in plastic bags.
- Ensure that the temperature in the main section of the refrigerator is always below 4° C, and that of the freezer is below -18° C.

- Cook egg, meat and seafood thoroughly before eating.
- Reheat food or heat partially cooked foods all the way through to at least 74° C .
- Wash hands, utensils, and counters with water and soap between preparation of different foods (especially raw meat, poultry, fish, eggs).
- Wash fresh fruits and vegetables under running water.
- Avoid consuming uncooked animal-derived dishes, meat or poultry.
- Do not consume egg preparations with runny yolk.
- Drink only boiled or bottled water.
- Do not eat raw vegetables and salads.
- Do not buy food items from roadside vendors.

BACTERIAL FOOD POISONING

BACTERIA	SOURCE	INCUBATION PERIOD	CLINICAL FEATURES
Bacillus cereus	Meats, vegetables(diarrheal form) fried rice(emetic form)	1-6 hrs (emetic form) 8-16 hrs (diarrhoeal form)	Nausea, vomiting Watery diarrhoea, abdominal pain, occasionally nausea, rarely fulminant liver failure.
Staphylococcus aureus(commonest)	Salad, pastries, ham, poultry(proteinaceous substances)	1-6 hrs	Nausea, severe vomiting, salivation, cramps, diarrhoea; usually resolves in 8 hrs
Clostridium perfringes (type A)	Meats, poultry	6-24 hrs	Nausea, vomiting, acute abdominal cramps, diarrhoea, gas gangrene
Vibrio cholerae	Water and food	16-72 hrs	Profuse, painless watery diarrhoea and vomiting. Colourless stools with mucus (rice water stools). Offensive sweetish odour

BACTERIA	ANTIBIOTICS	OTHER TREATMENT MEASURES
Bacillus cereus (Self-limiting)	Illness resolves in 24 hours. Antibiotic therapy is generally not indicated.	Nausea and vomiting can be controlled by anti-emetics.
Staphylococcus aureus (Self-limiting)	Illness resolves in 24 hours. Antibiotic therapy is generally not indicated.	Nausea and vomiting can be controlled by anti-emetics.
Clostridium perfringes	• Antibiotic therapy is not indicated. supportive measures.	
Vibrio cholerae	• A single dose of oral doxycycline (300 mg) is the preferred agent. • In children and pregnant women, erythromycin and azithromycin can be used. • In areas of high tetracycline resistance, fluoroquinolones are effective.	Antiemetic, anti-diarrhoeals, and antispasmodics are contraindicated.

BACTERIA	SOURCE	INCUBATION PERIOD	CLINICAL FEATURES
Salmonella species	eggs, dairy products	16-48 hrs	Vomiting, crampy abdominal pain, diarrhoea. Slimy loose stools ,foul smelling and greenish in colour.
Shigella	Fruits ,vegetables ,milk	16-48 hrs	Abdominal pain, tenesmus, fever , watery stools followed by bloody diarrhoea.
Escherichia coli	Water and food	16-72 hrs	Cramps, bloody diarrhoea, fever, vomiting, fatality
Campylobacter	Unchlorinated water,undercooked meat,unpasteurised milk and milk products	16-48 hrs	Watery or bloody diarrhoea; fever, systemic malaise, headache

BACTERIA	ANTIBIOTICS	ANTIMOTILITY AGENTS
Salmonella (Self-limiting)	• Antibiotics are generally not indicated. • Antibiotics are used only in the following cases: bacteremia, AIDS patient, elderly, infants, and individuals with sickle cell disease. • The antibiotic of choice is chloramphenicol. Fluoroquinolones, ampicillin, ceftriaxone, trimethoprim-sulphamethoxazole are also effective.	Anti-motility agents are contraindicated
Shigella	The preferred agents of choice are fluoroquinolones Ciprofloxacin 500 mg orally every 12 hrs X 5 days Levofloxacin 500 mg orally 1 daily X 5 days Norfloxacin 400 mg orally every 12 hrs X 5 days.	Anti-motility agents are contraindicated
E. coli (travellers diarrhoea)	• Norfloxacin 400 mg or ciprofloxacin 500 mg orally twice daily × 3 days • Or azithromycin 500 mg orally once daily × 3 days • Or rifaximin 200 mg thrice daily X 3 days	➢Loperamide and bismuth subsalicylate can be used to decrease the severity of diarrhoea.

BACTERIA	ANTIBIOTICS	ANTIMOTILITY AGENTS
Campylobacter	• Erythromycin ethyl succinate, 400 mg, four times a day is the drug of choice. • Clarithromycin and azithromycin are equally effective. • Antibiotics are not useful unless initiated within 4 days of the start of illness.	➢Anti-motility agents are contraindicated

BACTERIA	SOURCE	INCUBATION PERIOD	CLINICAL FEATURES
Listeria monocytogenes	Unpasteurized milk, ,uncooked meat,raw vegetables	16-48 hrs	Perinatal infection -foetal meningitis, IC –meningitis and sepsis.
Clostridium botilnum	Canned fruits, vegetables, meats, honey	12-72 hrs	▪ Visual disturbance or blurred vision ▪ Drooping eyelids ▪ Dry mouth and speaking problems ▪ Vomiting ▪ Swallowing problems ▪ Nausea ▪ Abdominal cramps

BACTERIA	ANTIBIOTICS	ANTIMOTILITY AGENTS
Listeria monocytogenes	• Intravenous administration of antibiotics such as ampicillin, penicillin G, gentamicin, erythromycin, tetracycline, doxycycline, tobramycin, cotrimoxazole, or vancomycin is usually necessary for serious listeriosis. • Chloramphenicol has been successfully used in some meningitis patients who did not respond to other antibiotics.	Vomiting and diarrhoea is controlled with anti-emetics and anti-diarrhoeals
Clostridium botulinum	Gastrointestinal decontamination with charcoal. Respiratory support: monitor negative inspiratory force, peak expiratory flow, vital capacity, and airway reflexes in unintubated patients; intubate and ventilate as needed. Equine-derived trivalent (ABE) botulinum antitoxin administration- one vial of ABE trivalent antitoxin intravenously after dilution (1:10 in normal saline) Skin or eye tests should be done prior to administration of the antitoxin or serum Human-derived pentavalent botulinum immune globulin in infants	

PROTOZOAL FOOD POISONING

ENTAMOEBA HISTOLYTICA

Source	➢Contaminated food and water ➢Raw vegetables which have not been washed well ➢Human to human transmission
Incubation period	2-4 weeks
Clinical features	Intestinal amoebiasis- mild abdominal discomfort, diarrhoea, fulminating dysentery Extra-intestinal amoebiasis- involves the liver, lungs, brain, spleen etc.
Treatment	Symptomatic cases can be treated with metronidazole (30 mg/kg/day for 8-10 days) or tinidazole. Abscesses must be treated surgically. Asymptomatic carriers can be treated with diiodohydroxyquin or diloxanide furoate

CRYPTOSPORIDIUM PARVUM- this protozoon causes severe diarrhoea in immunocompromised adult patients and immunocompetent children	
Source:	Contaminated food and water
Clinical features:	1. Severe persistent diarrhoea 2. Malnutition (in children) 3. Pulmonary manifestations 4. Toxic megacolon 5. Cryptosporidiosis is often fatal in AIDS patient
Treatment:	IV fluids and electrolytes Anti-diarrhoeal drugs
Microsporidia	
Clinical features:	Diarrhoea, keratoconjuctivitis, hepatitis, myositis Ascites, cholangitis , renal or urogenital infections
Treatment:	Albendazole (400 mg twice daily)

VIRAL FOOD POISONING

- Common viruses- adenovirus, parvovirus, and rotavirus.
- Rotavirus is the most common cause of diarrheal disease in infants and children.
- Adenovirus is another common agent of diarrheal disease in children.
- Incubation period
- rotavirus/adenovirus diarrhea -24 to 72 hours,
- Parvovirus gastroenteritis(adult variety) 24 to 36 hours
- Clinical Features
- Adeno/rotavirus-Vomiting begins abruptly and then resolves. Diarrhea lasts for 4 to 7 days. Stools are watery and foul smelling.
- Parvovirus is characterized by abrupt onset of diarrhea, vomiting, abdominal cramps and myalgia.

- Treatment
- Viral diarrheas are self-limiting and require only rehydration.
- Infants must be administered oral rehydration solutions, while older children and adults can be managed on the BRATT diet (Bananas, Rice, Apples, Tea, Toast)

PARASITES CAUSING JAPANESE RESTAURANT SYNDROME

- Source: raw fish (popular culinary delicacy in japanese cuisine)
- Etiological agents: roundworm & fish tapeworm

PARASITE	ROUNDWORM	FISH TAPEWORM
CLINICAL FEATURES	▪ After an interval of 1-2 hours following consumption of fish, the following symptoms occur: nausea, vomiting, and crampy abdominal pain.	▪ After an interval of 1-2 weeks following consumption of fish, the following symptoms occur: nausea, vomiting, abdominal cramps, flatulence, diarrhoea, megaloblastic anaemia.
DIAGNOSIS	▪ Visual inspection of larvae on endoscopy, laparotomy, or pathological examination	▪ Identification of tapeworm proglottids in stool.
TREATMENT	▪ niclosamide, praziquantel, paramomycin may be effective	

MUSHROOM POISONING

- The term "mushroom" actually refers to the reproductive portion of a fungus which grows up from an underground mycelium.
- less than 5% of mushrooms are poisonous
- It can either cause mild to moderate self-limiting gastroenteritis or severe sometimes fatal reactions.
- Depending on the nature of toxin present, mushrooms can be classified into several groups:
  - Amanita species
  - Galerina species
  - Lepiota species
- Toxins: Amatoxins, phallotoxins, and virotoxins

Amanita muscaria

Galerina

Lepiota

- Mode of action:
- Of all the toxins, phalloidin - most rapid acting, amanitin - delayed manifestations. Phalloidin interrupts actin polymerization and impairs cell membrane function, but has a limited absorption and toxicity. Phalloidin binds to the actin F (filamentous polymer) of the plasma membranes, and hence increases the permeability of the plasma membranes of hepatocytes.
- Amatoxins are more potent and can cause hepatic, renal, and CNS damage. Alpha-amanitin is cytotoxic on the basis of its interference with RNA polymerase II, preventing the transcription of DNA.

CLINICAL FEATURES

PHASE I	PHASE II	PHASE III
Abdominal pain, nausea, vomiting, diarrhoea, fever, tachycardia, hypoglycaemia, hypotension and electrolyte imbalance*1 day.	Treacherous phase of remission	2 to 3 days hepatic, renal, and (occasionally) pancreatic failure
severe diarrhoea, watery, and cholera-like.	.	7 to 10 days-Hepatotoxicity
Metabolic acidosis		Coagulation defects with hypofibrinogenaemia and hypoprothrombinaemia occur in hepatic failure-local or general bleedingLactic acidosis and metabolic acidosis , Hypoglycaemia, Cardiovascular collapse usually accompanies severe hepatic failure at the terminal stage, ARDS, Polyneuropathy Renal failure, foetus -toxic hepatitis

- **DIAGNOSIS**

- ✓ **Meixner test**-amatoxin (false positive reactions are possible.)
- ✓ **Melzer's test** - detect an amyloid reaction in cyclopeptide containing Amanitas.
- ✓ **Hepatic and renal function tests**
- ✓ Serum electrolytes, urea, creatinine, and glucose levels
- ✓ Drop of material, Mushroom dish, gastric contents, or stools (available) - place on slide & observe under high power (450X–500X) magnification, to reveal **spores**
- ✓ Detection of toxins in gastric aspirate, serum, urine, stool, and liver and kidney biopsies, using HPLC, TLC, or RIA
- ✓ Monitor coagulation parameters (INR or PT)
- ✓ Patients with prothrombin values less than 10% have high fatality rate.

- **SEVERITY CLASSIFICATION**

✓ Grade 1: GI upset

✓ Grade 2: All signs of intoxication, with a mild to moderate rise in transaminases (less than 500 units/L).

✓ Grade 3: Severe hepatic damage with a great increase in transaminases (> 500 units/L), plus an impaired plasma clotting function

a) Grade 3a: Bilirubin rise is mild or absent.

b) Grade 3b: Bilirubin rise is steep and continuous (> 5 mg/dL). These patients are at risk and should be transferred to a facility where liver transplant is possible.

✓ Grade 4: Steep rise in transaminases, accompanied by a steep decline in clotting function, a steep rise in bilirubin, and the onset of kidney dysfunction. Patients have a poor prognosis, and many die despite intensive care.

- TREATMENT

A. **STABILSATION**

- Restoration of fluid
- IV glucose
- Due to coagulation defect, hemorrhage occurs, treat with vitamin K (50 to 100 mg/day IV) and fresh frozen plasma.
- Correction of hypokalemia (by KCl diluted in solutions of dextrose 5%, or NaCl 0.9%)

  metabolic acidosis - sodium bicarbonate solution 1.4%

B. DECONTAMINATION

- Activated charcoal/Multiple dose activated charcoal
- Forced diuresis (6 to 9 L/day) may therefore help if the patient presents within 24 to 48 hours because significant amounts of amatoxins are eliminated in urine.
- Haemoperfusion, beneficial if performed within 24hrs
- Charcoal plasma perfusion (CPP) and continuous venovenous haemofiltration (CVVH)

C. ANTIDOTE

- Benzyl penicillin- effective in displacing amatoxin from plasma protein-binding sites allowing for increased renal excretion
- Thioctic acid (alpha- lipoic acid)
- Silybinin

- Cimetidine (4 to 6 g/day) a potent cytochrome P450 system inhibitor may have hepatoprotective effects against alpha-amanitin
- N- acetyl cysteine (NAC)
- Root of Indian plant Picrorhiza kurroa (Kutkin)- contains an iridoid glycoside mixture that has been shown to be hepatoprotective in certain situations.
- Aucubin - iridoid glycoside obtained from the leaves of Aucuba japonica-protective against Amanita intoxication (when tested in dogs)

D. TREATMENT OF ACUTE LIVER FAILURE

- General symptomatic treatment
- Treatment of hepatic encephalopathy -Lactulose, Dietary protein withdrawal, Metronidazole or neomycin

- Treatment of cerebral edema-
- ✓ ICP monitoring
- ✓ Osmotic diuretics such as mannitol (1 gm/kg, as rapid IV infusion of 20% solution).
- ✓ Barbiturates such as IV thiopentone (3 to 5 mg/ kg) infused slowly over 15 minutes until signs of raised ICP resolve
- ✓ Corticosteroids may not help in relieving cerebral edema due to acute liver failure.
- ✓ Proper positioning of the patient, i.e. head upright
- Treatment of infection-Treating with antibiotics aggressively or prophylactically depending on situation
- Treatment of coagulopathy -Fresh frozen plasma for serious or persistent bleeding.

- Liver transplantation recommended in following situations:
- ✓ Grade II encephalopathy and beyond.
- ✓ Prolonged prothrombin time (greater than twice normal), despite administration of fresh-frozen plasma.
- ✓ Serum bilirubin greater than 25 mg%.
- ✓ Azotaemia.
- ✓ Evidence of acidosis, hypoglycaemia, GI haemorrhage and hypofibrinogenaemia.
- Molecular Absorbent Regenerating System (MARS)
- ✓ Short-term extracorporeal hepatic support - liver dialysis
- ✓ Method of removing protein bound substances in patients with liver failure and hepatic encephalopathy,
- ✓ It employs an albumin-impregnated highly permeable dialyser with albumin-containing dialysate recycled in a closed loop with a charcoal cartridge, an anion exchange resin absorber, and a conventional haemodialysis membrane.

PLANT POISONING

They include cyanogenic plants, fava beans, cycads, sweet pea and prickly poppy.

- **Cyanogenic Plants** contain amygdalin or other glycosides, which on hydrolysis can release traces of cyanide.

e.g.: Apple (Malus species) , Bracken Fern (Pteridium aquilinum) , Cassava (Manihot species) ,Clover (Trifolium species)

- **CASSAVA(Manihot esculenta)**

•Cassava root is second largest carbohydrate crop in the world, and constitutes a staple diet.

•Insufficiently processed cassava liberates cyanide in the gut from the ingested cyanogenic glycoside linamarin. This is normally converted to the less toxic thiocyanate by the enzyme rhodanese.

- The substrate for this reaction is sulfur originating from proteins in the diet. When dietary protein intake is low, signs of toxicity begin to manifest.
- **MANIFESTATIONS**

i. Tropical ataxic neuropathy (TAN)
ii. Epidemic spastic paraparesis (ESP)
iii. Pancreatitis and endemic goiter have also been reported in patients from cassava-consuming areas.
iv. Acute toxicity is rare.
v. Fatal dose : > 3 mg/kg

Treatment

Stabilisation: Assisted ventilation, 100% oxygen, cardiac monitoring, IV access, treatment of metabolic acidosis, vasopressors for hypotension.

Decontamination

- stomach wash (preferably with 5% sodium thiosulfate solution), activated charcoal, and cathartics, after antidotal therapy has been instituted. Emesis is not recommended.
- Haemodialysis and haemoperfusion are NOT effective. But can be used For supportive treatment.

- Antidotal therapy:

The 3-step Eli Lilly cyanide kit approach—

First step: Amyl nitrite (one perle of 0.2 ml is crushed and inhaled for 30 seconds) every minute until the 2nd step is begun.

Second step: Sodium nitrite (3% solution) slow IV, i.e. over 5 to 10 minutes.

- Adult dose—10 ml (300 mg).
- Paediatric dose—0.33 ml/kg, upto a maximum of 10 ml.

Third step: Sodium thiosulfate (25% solution), 3 to 5 ml/min, IV.

Adult dose—50 ml (12.5 gm).

Paediatric dose—1.65 ml/kg (412.5 mg/kg), upto a maximum of 50 ml.

Other Antidote:

Hydroxocobalamin (Vitamin B12 precursor): It combines with cyanide to form cyanacobalamin (vitamin B12), which is excreted in the urine.

Dose: 50 mg/kg of commercial solution (1000 mcg/ml).

- **Amygdalin-containing Plants**

- Amygdalin is the cyanogenic diglucoside D-mandelonitrile-beta-D-gentiobioside, and is not toxic until it is metabolized by the enzyme emulsion which is present in the seeds of these plants.
- E.g. : Almond, Apricot, Cherry Laurel, etc.
- One of the richest sources of amygdalin is the bitter almond and it has been established that 40 to 60 seeds, yielding 70 mg of hydrocyanic acid would result in severe toxicity or death.

- **Sweet Pea(Lathyrus sativus)**
- Chronic intake of chick pea leads to the development of Lathyrism.
- It is characterized by gradually progressing bilateral spastic paraparesis.
- Prodromal manifestations such as cramps, prickling sensation, and nocturnal calf pain.
- Total spastic paraplegia may result in course of time.
- Treatment: Exclusion of chick pea from diet and symptomatic measures.
- **Prickly Poppy (Argemone Mexicana)**
- This belongs to the family Papaveraceae.
- Toxic part: Seed and expressed oil are quite toxic. Leaves are also toxic (to a lesser degree).
- Toxic Principles
- ■ Sanguinarine
  ■ Dihydrosanguinarine

Mode of action

- Liver, heart, kidneys, and lungs are the target organs of argemone alkaloids, and it is postulated that membrane destruction is the probable mode of action.
- Argemone act on the blood vessels which become abnormally permeable, resulting in the leakage of protein-rich plasma components into the extravascular compartment leading to hypovolaemia and reduced plasma osmotic pressure
- **Clinical Manifestations**

■ Skin—Superficial patchy erythema, telangiectasias, sarcoids (purplish blotches over lower limbs), pigmentation, and hair loss.

■ Eye—Glaucoma, superficial retinal hemorrhages, venous dilatation, central serous retinopathy, and disc edema

■ CVS—Palpitations, tachycardia, hypotension, CCF.

■ RS—Dyspnea, pulmonary edema

Clinical Features

Chronic consumption leads to epidemic dropsy characterized by slow progression, pitting pedal edema, and limb pain. Diarrhea, abdominal pain, and fever are often present in the early stages.

- Diagnosis of epidemic dropsy:
  a. Anaemia
  b. Raised plasma pyruvate level.
  c. Chest x-ray: cardiomegaly, pulmonary oedema.
  d. ECG: ST, T wave changes, atrial/ventricular extra systoles

- **Treatment**

1. Withdrawal of contaminated oil from the diet.

2. Bed rest, leg elevation, protein-rich diet.

3. Supplements of calcium, antioxidants (vitamins C & E), and B vitamins.

4. Corticosteroids.

5. Diuretics.

6. Management of cardiac failure: bed rest, salt restriction, digitalis, and diuretics.

7. Most patients recover with treatment in about 3 months. Mortality is around 5%. Post-recovery, pedal oedema may take up to 5–6 months to resolve completely.

FISH POISONING

SCOMBROID FISH POISONING

Source: scombroid poisoning results from eating inadequately refrigerated, or inadequately preserved, dark or red-muscled fish, including tuna, mahi-mahi (dolphin), sardine, bluefish

Toxin: endogenous histidine in fish has been broken down by bacteria into high levels of histamine and saurine.

Clinical features (histamine-mediated): flushing of face and neck, burning sensation in mouth and throat, abdominal cramps and nausea, sensation of warmth without fever, metallic or peppery taste in the mouth.

Blue fish

Bombay duck

Treatment:

- Supportive care
- Decontamination with activated charcoal
- Intravenous fluids
- H1 blocker- diphenhydramine 25-50 mg I.V./I.M., Repeat prn
- H2 blocker- ranitidine 50 mg I.V., Repeat prn

CIGUATERA FISH POISONING

Source: grouper, red snapper, dinoflagellates

Toxin: Ciguatera poisoning arises secondary to the action of ciguatoxin (CTX) on nerve and muscle membranes. It binds to the sodium receptor of excitable membranes and increases sodium permeability, leading to repetitive firing and enhanced persistent activation of the channel.

Red snapper

Grouper

Clinical features: Initial gastrointestinal symptoms include nausea, vomiting, diarrhea, Later neurologic signs and symptoms include paresthesias, pruritus, and dizziness, Reversal of hot and cold sensation, Symptoms become worse with ethanol ingestion.

Treatment:

- Decontamination with charcoal
- Seizure management
- Intravenous fluids
- IV mannitol should be administered in the management of neurological and muscular manifestations- 0.5-1 g/kg of 20% solution, administered over 30-45 min.

- **TETRODOTOXIC POISONING**

Source: fugu, blue-ringed octopus

Toxin: Tetrodotoxin (TTX) is neurotoxin concentrated mainly in the skin, liver, ovary, and intestine of the fish.

Puffer fish

Blue ringed octopus

- **Clinical features:** Parasthesia of the mouth, tongue, lips, and extremities, Salivation and bronchorrhea, Ascending paralysis in an awake patient, Cranial nerve dysfunction, Feeling of floating.
- **Treatment:**
- Decontamination with activated charcoal
- IV edrophonium (10 mg) or I.M. neostigmine (0.5 mg) may be effective in restoring motor strength.
- Artificial ventilation should be implemented if necessary.
- Hemodialysis may be effective.
- **SHELLFISH POISONING**
- **Source**: Shellfish (especially oysters, clams, mussels, and scallops) contaminated by dinoflagellates.
- There are 3 main types of shellfish poisoning:

Toxin	Sources of toxin	Primary vector	Mechanism	Symptoms
Saxitoxins, gonyautoxins	*Alexandrium* spp., *Gymnodinium* spp., *Pyridinium* spp.	Shellfish	Voltage-gated sodium channel 1	Tingling of perioral area, gastrointestinal problems, numbness of extremities, disturbed muscle coordination, respiratory distress, paralysis; 20% mortality
Brevetoxins	*Karenia brevis*, *Chattonella marina*, *Chattonella antiqua*, *Fibrocapsa japonica*, *Heterosigma akashiwo*	Shellfish	Neurotoxin acting via voltage-gated sodium channel 5	Nausea, numbness of perioral area, paresthesia, disturbed motor control, severe muscular pain
Ciguatoxins, maitotoxins	*Gambierdiscus toxicus*, *G. belizeanus*, *G. yasumotoi*	Coral reef fish	Voltage-gated sodium channel 5, voltage-gated calcium channel	>175 gastrointestinal, neurological, cardiovascular, and general symptoms; can be fatal
Azaspiracids	*Protoperidinium crassipes*	Shellfish	Voltage-gated calcium channel	Nausea, vomiting, severe diarrhea, stomach cramps
Palytoxins	*Palythoa toxica*, *Ostreopsis siamensis*	Shellfish	Sodium–potassium ATPase	Fever, ataxia, drowsiness, often fatal
Yessotoxins	*Protoceratium reticulatum*, *Lingulodinium polyedrum*, *Gonyaulax spinifera*	Shellfish	Possibly voltage-gated calcium/sodium channel	
Okadaic acids	*Dinophysis* spp.	Shellfish	Inhibition of phosphatases and of protein synthesis	
Domoic acids	*Pseudo-nitzschia* spp.	Shellfish	Activation of the kainate glutamate receptor	Vomiting, diarrhea, abdominal cramps, severe headache, loss of short-term memory, can be fatal

	Paralytic	Neurotoxic	Amnestic
Onset	30 min- 3hr	3 min-3 hr	15 min-38 hr
Duration	2-5 days	2-3 days	1 day-12 weeks
Clinical features	▪ Abdominal pain, nausea, vomiting, diarrhoea ▪ CNS depression, headache, paresthesia, hot-cold reversal, weakness, paralysis, ataxia, cranial nerve dysfunction, seizures,Respiratory failure, tachycardia, hypotension, pruritus,Sweat, salivation	▪ GI manifestation, ▪ CNS depression, headache, hot-cold reversal, vertigo, ataxia, seizures,Bradycardia, bronchospasm	▪ Abdominal pain, nausea, vomiting, diarrhoea ▪ Headache, memory loss, seizures, mutism ▪ Dysrhythmias, tachycardia, hypotension
Treatment	Decontamination with charcoal, Seizure management, Intravenous fluids and pressors, Intubation and mechanical ventilation, Temporary pacemaker, Diuresis to enhance renal excretion of the toxin. Sodium bicarbonate for reversing ventricular conduction delays and arrhythmias; administer 1 to 2 meq/kg bolus, and repeat as necessary.	▪ Decontamination with charcoal, Seizure management, Beta 2 adrenergic agonists and corticosteroids to treat bronchospasm	▪ Decontamination with charcoal, Seizure management, Intravenous fluids and pressors, Intubation and mechanical ventilation, Supportive and symptomatic measures

FOOD ADDITIVES

▪Monosodium Glutamate (MSG)

Uses

■ Flavoring agent in foods MSG is a fine, white crystalline substance, similar in appearance to salt or sugar,has a sweetish saline, To treat- hyperammonaemia in conditions such as hepatic encephalopathy.

Source MSG is the monosodium salt of L-glutamic acid

It is produced by the following processes:

✓ Fermentation of carbohydrate sources ,

✓ Hydrolysis of vegetable proteins.

- **Clinical features**

✓ Burning, tingling sensation and numbness of face, trunk, and upper limbs,weakness, dizziness,syncope, flushing,Lacrimation,chest pain,gastric distress and rarely bronchospasm and angioedema.

✓ In young children, a convulsive attack(self limiting)

- **Treatment:**
- Treatment is symptomatic and supportive. Gastrointestinal decontamination is generally not indicated after an acute ingestion. Toxicity is very unlikely.

CASE PRESENTATION

SUBJECTIVE

- **Age** -30 years
- **Sex** - Female
- **C/O**

- abdominal cramping
- 2 episodes of vomiting after taking reheated fried rice which was taken home from the restaurant a night before.
- Onset was 1 hour after eating the food.
- N/C/O –fever or diarrhea.
- By morning she was fine and did not seek medical attention.

Evidence for Bacillus cereus infection

Source

Fried rice, heating of food can kill the vegetative form but not the spores.

Incubation period of B .cereus (1-5 hours)

Onset of symptoms after 1 hour

Common symptoms

- abdominal discomfort
- 1-2 episodes of vomiting or diarrhea within 1-24 hours after exposure
- Infection-Short lived and self limiting.(do not seek medical attention)

TREATMENT

- **Mild fluid deficits**
- ORS, glucose
- **Moderate to severe dehydration**
- IV fluids
- **Significant nausea and vomiting**
- Anti emetic agent-Promethazine 12.5- 25mg q4-6hr PRN

MCQS

1. Which of the following the commonest cause of bacterial food poisoning ?

A)Bacillus cereus

B)Staphylococcus aureus

C)Salmonella

D)Vibrio

2.Among the following which is the source of salmonella food poisoning?

A)Eggs, milk and mik products

B)Cooked and fried rice

C)Contaminated food and water

D)Pasteurised milk

3.Name the diagnosis suitable for B. cereus infection.

A)Latex agglutination and ELISA

B)WBC for leucocytosis and leukopenia

C)Serum electrolytes

D)Stool culture and growth in MYPA

4. Which of the following infections is self limiting?

A)B.cereus

B)Staphylococcus aureus

C)Campylobacter

D)E.coli

5.Toxin responsible for V. Cholerae.

A)Enterotoxin B

B)CT A

C)Serotype- 0157

D)Exotoxin

6.What is the source of Japanese Restaurant Syndrome?

A)Raw fish

B)Mushrooms

C)Contaminated food and water

D)Banana.

7.Amatoxin can be identified through which of the following diagnostic test?

A)Melzer's test

B)Meixner test

C)Heamagglutination test

D)Tensilon test

8.Which of the following states Grade 3 severity of mushroom poisoning?

A)Non indications of liver or kidney failure

B)Mild to moderate rise in transaminase

C)Severe hepatic damage with great increase in transaminase.

D)Steep rise in transaminase with steep decline in clotting function, onset of kidney dysfunction.

9.Which of the following is the treatment of hepatic encephalopathy?

A)Lactulose, dietary protein withdrawal, Metronidazole

B)Mannitol, barbiturates, corticosteroids

C)Benzyl penicillin

D)Aucubin

10.Which among the following has sanguinarine and dihydrosanguinarine as its toxic principle?

A)Sweet pea

B)Prickly poppy

C)Tuna fish

D)Blue ringed octopus

ANSWERS

1. B)Staphylococcus aureus
2. A)Eggs, milk and mik products
3. D)Stool culture and growth in MYPA
4. A)B.cereus
5. B)CT A
6. A)Raw fish
7. B)Meixner test
8. C)Severe hepatic damage with great increase in transaminase.
9. A)Lactulose, dietary protein withdrawal, Metronidazole
10. B)Prickly poppy

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THANK YOU