MICROBIOLOGY

بسم الله الرحمن الرحيم

MID | Lecture 4

Enteric G-Rods (Pt.2)

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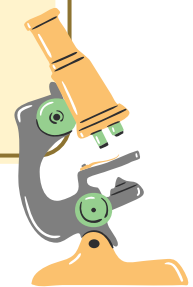
Written by: Laith Alhuniti

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Shigellosis (Bacillary dysentery)

Dysentery means WBCs and RBCs with mucus.

- The natural habitat of shigellae is limited to the intestinal tracts of humans and other primates, where they produce bacillary dysentery.

- Shigellae are slender gram-negative rods; coccobacillary forms occur in young cultures. Shigellae are facultative anaerobes but grow best aerobically. Convex, circular, transparent colonies with intact edges reach a diameter of about 2 mm in 24 hours.

- All shigellae ferment glucose. With the exception of Shigella sonnei, they do not ferment lactose. The inability to ferment lactose distinguishes shigellae on differential media

Shigella sonnei is the only lactose fermenter among Shigella species; however, it is a slow fermenter.

- Non-motile,non-lactose fermenters,do not produse H2S, and produce a colorless colonies in EMB.

The most common causes of bacterial gastroenteritis are Shigella, Salmonella, and Campylobacter, regardless of ethnicity or age.

- Shigella infection is primarily a pediatric disease, with 60–70% of cases occurring in patients younger than 10 years. Children under the age of 5 years are particularly more susceptible to infection due to their immature immune systems and increased exposure risks. However, Shigella can also occur in adults, particularly in specific groups. These include men who have sex with men (MSM), especially during diarrheal episodes; residents or staff of mental health institutions; and adult household contacts of infected children. In these situations, prophylactic antibiotic treatment is recommended for close contacts to prevent further transmission.
- Adults living in endemic areas may carry the infection asymptomatically and shed the bacteria in their feces, thereby continuing the transmission cycle of Shigellosis.
- Shigellosis and typhoid (caused by Salmonella typhi) are **exclusively human diseases.**

Epidemiology

- Man and certain primates are the only host.
- Age: any age but commonly under 5 y/o.
- It occurs in warm months, temperate climates and rainy seasons in tropical countries.
- Asymptomatic infection in endemic areas.

Shigella sonnei is the most commonly isolated species in developed countries, whereas in our country (developing) , Shigella flexneri is more frequently isolated.

- In industrialized countries , S.sonnei is most common with S.flex second.
- Transmission: feco-oral route, person to person, toilet seat, door handles, contaminated food and water supply and a vector causing outbreaks: flies maybe . (highly communicable)

Etiology

Shigella species possess an O antigen as part of their outer membrane. Based on serotyping of the O antigen, they are classified into four species: Shigella dysenteriae, Shigella flexneri, Shigella boydii, and Shigella sonnei.

- The genus shigella is subdivided into 4 species (A,B,C and D) according to their biochemical reaction and antigenic composition . Low number are required to cause disease : 10-1000.

Very successful pathogen.

- Group A Shigella Dysenteriae 12 Serotypes, most imp. type 1 shiga, most severe disease.
- It is the most potent producer of shigella toxin.
- Group B Shigella flexneri 8 serotypes mild disease.
- Group C Shigella boydii 18 serotypes. (From mild to severe shigellosis)
- Group D Shigella sonnei single , intermediately sever disease .

- Experimental evidence shows that mutant strains of Shigella lacking the Shiga toxin (Stx) gene **can** still cause shigellosis, indicating that toxin production is not essential for disease development. In contrast, mutant strains that are defective in their invasion machinery, even if they still produce Shiga toxin, are **unable** to cause shigellosis. Therefore, Shigella is considered a prototype of invasive gastrointestinal pathogens, where the ability to invade intestinal epithelial cells is critical for pathogenesis. The Shiga toxin, while not essential for initiating infection, has a synergistic effect that exacerbates disease severity and invasive process.

Pathogenesis

- Shigella infections are almost always limited to the gastrointestinal tract; bloodstream invasion is quite rare. Shigellae are highly communicable; the infective dose is on the order of less than 10^3 organisms (it usually is 10^5–10^8 for salmonellae and vibrios).

- The essential pathologic process is invasion of the mucosal epithelial cells (eg, M cells) by induced phagocytosis, escape from the phagocytic vacuole, multiplication and spread within the epithelial cell cytoplasm, and passage to adjacent cells.

- Micro abscesses in the wall of the large intestine and terminal ileum lead to necrosis of the mucous membrane, superficial ulceration, bleeding, and formation of “pseudomembrane” on the ulcerated area. This consists of fibrin, leukocytes, cell debris, a necrotic mucous membrane, and bacteria. As the process subsides, granulation tissue fills the ulcers, and scar tissue forms.

- Upon reaching the intestinal mucosa, Shigella invades the epithelial lining by first crossing the mucus layer and then penetrating microfold (M) cells within Peyer's patches. Inside these cells, Shigella triggers apoptosis, facilitating its spread to adjacent epithelial cells. The bacteria then disseminate laterally by hijacking the host's cytoskeletal system, primarily through actin polymerization. Clinically, Shigellosis is characterized by the formation of superficial ulcers that are limited to the mucosal layer of the intestine.
- Blood cultures in patients with Shigellosis are typically negative, as Shigella rarely enters the bloodstream. In contrast, infections caused by Salmonella species, particularly Salmonella Typhi and Salmonella Paratyphi, frequently result in positive blood cultures.
- Unlike Shigella, Salmonella invades more deeply, creating profound mucosal ulcers, penetrating the intestinal wall, and accessing the circulatory system. This invasive process can lead to severe complications such as bacteremia and intestinal perforation.

Toxins

- A. Endotoxin
- Upon autolysis, all shigellae release their toxic lipopolysaccharide. This endotoxin
  probably contributes to the irritation of the bowel wall.

Irritation of the bowel wall can occur in either the small intestine or the large intestine, depending on the underlying cause.

- B. Shigella Dysenteriae Exotoxin
- S dysenteriae type 1 (Shiga bacillus) produces a heat-labile exotoxin that is neurotoxic, cytotoxic and enterotoxic .
- Acting as an enterotoxin, it produces diarrhea as does the E coli Shiga-like toxin, perhaps by the same mechanism.

- In humans, Acting as a “neurotoxin,” this material may contribute to the extreme severity and fatal nature of S dysenteriae infections and to the central nervous system reactions observed in them (ie, meningismus, coma).
- The toxic activity is distinct from the invasive property of shigellae in dysentery. The two may act in sequence, the toxin producing an early nonbloody, voluminous diarrhea and the invasion of the large intestine, resulting in later dysentery with blood and pus in stools.

The Shigella toxin can be considered a neurotoxin because patients may present with neurological manifestations. For example, children might not come to you with diarrhea, but instead with seizures, meningismus, or coma.

Clinical Findings

- After a short incubation period (1–2 days), there is a sudden onset of abdominal pain, fever, and watery diarrhea. The diarrhea has been attributed to an exotoxin acting in the small intestine. A day or so later, as the infection involves the ileum and colon, the number of stools increases; they are less liquid but often contain mucus and blood.

•Each bowel movement is accompanied by straining and tenesmus (rectal spasms), with resulting lower abdominal pain.

Tenesmus is the feeling that you need to pass stools, even though your bowels are already empty.

- In more than half of adult cases, fever and diarrhea subside spontaneously in 2–5 days.

However, during their recovery, they may continue to shed the bacteria in their feces, even if they feel well or have received appropriate treatment.

- However, in children and elderly adults, loss of water and electrolytes may lead to dehydration, acidosis, and even death. The illness caused by S dysenteriae may be particularly severe.

- On recovery, most persons shed dysentery bacilli for only a short period, but a few remain chronic intestinal carriers and may have recurrent bouts of the disease. Upon recovery from the infection, most persons develop circulating antibodies to shigellae, but these do not protect against reinfection.

- In addition to hemolytic uremic syndrome (HUS), invasive Shigella infections can lead to another complication: reactive arthritis. This condition, also known as post-infectious reactive arthritis or Reiter's syndrome, is an autoimmune phenomenon triggered by infection. Reactive arthritis typically manifests as a combination of arthritis (often monoarthritis or oligoarthritis), urethritis, and uveitis.

- Reiter's syndrome, is typically preceded by either a urogenital infection caused by Chlamydia trachomatis or by an enteric infection with bacteria such as Shigella, Salmonella, Yersinia, or Campylobacter.

Diagnostic Laboratory Tests

- A. Specimens
- Specimens include fresh stool, mucus flecks, and rectal swabs for culture. Large numbers of fecal leukocytes and some red blood cells often are seen microscopically.

- B. Culture (Definitive diagnosis)

Eosin methylene blue

- The materials are streaked on differential media (eg, MacConkey or EMB agar) and on selective media (Hektoen enteric agar or Salmonella –Shigella agar), which suppress other Enterobacteriaceae and gram-positive organisms.

  On MacConkey agar and EMB (Eosin Methylene Blue) agar, Shigella produces colorless colonies because it does not ferment lactose.

- C. Serology
- Normal persons often have agglutinins against several Shigella species. However, serial determinations of antibody titers may show a rise in specific antibody. Serology is **not used** to diagnose Shigella infections .

Treatment

- Ciprofloxacin, ampicillin, doxycycline, and trimethoprim–sulfamethoxazole are most commonly inhibitory for Shigella isolates and can suppress acute clinical attacks of dysentery and shorten the duration of symptoms.

- Antibiotic treatment for shigellosis is generally reserved for individuals at higher risk of developing complications, such as the elderly, very young children, and immunocompromised patients. Treatment is also given to reduce transmission, particularly to adult household contacts. Antibiotic therapy shortens the duration of symptoms and decreases the fecal shedding of Shigella in affected children. However, antimicrobial susceptibility testing should always be performed before or soon after starting treatment, due to the increasing rates of antibiotic resistance.

- They may fail to eradicate the organisms from the intestinal tract.
- Multiple drug resistance can be transmitted by plasmids, and resistant infections are widespread. Many cases are self-limited.
- Opioids should be avoided in Shigella dysentery.
- Opioids are not recommended because opioids like morphine might mask the real problem when a patient has bloody diarrhea. Bloody diarrhea has a very serious differential diagnosis, so the clinician must be aware of it.

Prevention, and Control

- IgA antibodies in the gut may be important in limiting reinfection

- Serum antibodies to somatic Shigella antigens are IgM.

- Antibodies against the somatic O antigen are generated, but they are not protective against triggering infections. Therefore, control focuses on preventing fecal-oral transmission, mainly through contamination of food, water, fingers, and flies.

- Shigellae are transmitted by “food, fingers, feces, and flies” from person to person. Because humans are the main recognized host of pathogenic shigellae, control efforts must be directed at eliminating the organisms from this reservoir by (1) sanitary control of water, food, and milk; sewage disposal and fly control; (2) isolation of patients and disinfection of excreta; (3) detection of subclinical cases and carriers, particularly food handlers; and (4) antibiotic treatment of infected individuals.

The Salmonella-group

- Salmonellae are often pathogenic for humans or animals when acquired by the oral route.
- They are transmitted from animals and animal products to humans, where they cause enteric fever ,gastro- enteritis and systemic infection.
- The disease caused by Salmonella is known as salmonellosis. Salmonellosis has four clinical types. The first is known as enteric fever or typhoid fever. The second, and the most common form, is Salmonella gastroenteritis, which often presents with bloody diarrhea and usually resolves spontaneously. The third type is bacteremia with focal lesions. The fourth type is the carrier state, where patients continue to harbor Salmonella in their biliary or intestinal tracts, or in the kidneys. If the gallbladder contains stones, the patient may remain a chronic carrier even after antibiotic treatment.

- Most isolates are motile with peritrichous flagella. They almost never ferment lactose or sucrose. They form acid and sometimes gas from glucose and mannose. They usually produce H2S.
- They survive freezing in water for long periods. Salmonellae are resistant to certain chemicals (eg, brilliant green, sodium tetrathionate, sodium deoxycholate) that inhibit other enteric bacteria; such compounds are therefore useful for inclusion in media to isolate salmonellae from feces.
- Sodium tetrathionate can be used in tetrathionate broth or in media such as Selenite F broth to create an enrichment culture for *Salmonella*. This medium allows the preservation and selective multiplication of *Salmonella* while inhibiting the growth of gram-positive bacteria and other gram-negative organisms.

Go to the next slide to see enterica subspecies.

- Salmonellae are named by genus (Salmonella), species (enterica), and subspecies (e.g., typhi or enteritidis) .

The nomenclature of Salmonella is complex, but at our level, we are required to understand two main categories: typhoidal Salmonella and non-typhoidal Salmonella.

Typhoidal Salmonella includes Salmonella Typhi and Salmonella Paratyphi (groups A, B, and C), which cause typhoid fever and paratyphoid fever. Paratyphoid fever is considered a milder form of typhoid fever.

Typhoid fever, also known as enteric fever, is a multisystemic disease. All other Salmonella serotypes – over 1,600 in number – are classified as non-typhoidal Salmonella. These typically cause gastroenteritis, but some can also cause bacteremia with focal lesions.

Subspecies of Medical Importance

- S. enterica subsp. Typhi.
- S. enterica subsp. Enteritidis
- S. enterica subsp. Typhimurium
- S. enterica subsp. Choleraesuis
- S. enterica subsp. Paratyphi
- S. enterica subsp. Dublin

The CDC classifies Salmonella into the genus Salmonella, which contains two species: Salmonella enterica and Salmonella bongori. Salmonella enterica affects humans as well as other warm-blooded animals and accounts for about 99% of human infections. Salmonella bongori is less commonly associated with human disease.

Salmonella enterica is further divided into six subspecies. Among its serotypes, Salmonella Enteritidis (subspecies enterica, serotype Enteritidis) is the most common cause of Salmonella gastroenteritis, which is the most frequent clinical form of salmonellosis.

Salmonella Typhi and Salmonella Paratyphi cause enteric fever (typhoid and paratyphoid fever). Salmonella Typhimurium is the second most common cause of gastroenteritis after Enteritidis.

Salmonella Choleraesuis is a serotype more frequently associated with bacteremia with focal lesions, often without gastrointestinal symptoms, and can spread to sites like the lungs, meninges, and bones.

The “Enteric Fevers” (Typhoid Fever)

- Four serotypes of salmonellae that cause enteric fever can be identified in the clinical laboratory by biochemical and serologic tests. These serotypes should be routinely identified because of their clinical significance.

- Salmonella Paratyphi A (serogroup A), Salmonella Paratyphi B (serogroup B), Salmonella Choleraesuis (serogroup C1), and S Typhi (serogroup D).

- Salmonella serotypes Enteritidis and Typhimurium are the two most common serotypes reported in developed world.

- Patients infected with Salmonella Typhi or Paratyphi may not exhibit gastrointestinal symptoms during the first two weeks of infection. Afterward, gastrointestinal symptoms may appear.
- About 5% of individuals who recover from typhoid or paratyphoid fever become convalescent carriers, continuing to shed the bacteria after clinical recovery.

Epidemiology

- Typhoid fever is sever systemic disease.

- Incidence differ significantly developing vs developed counties 0.2-4 cases to up to 500 /10^5 population.

- Humans are the natural reservoir. The feces of persons who have unsuspected subclinical disease or are carriers are a more important source of contamination than frank clinical cases that are promptly isolated, such as when carriers working as food handlers are “shedding” organisms.
  Vertical transmission from an infected mother to a susceptible newborn can also occur, leading to transmission of the disease.

- Many animals, including cattle, rodents, and fowl, are naturally infected with a variety of salmonellae and have the bacteria in their tissues (meat), excreta, or eggs.

- Food, water contaminated with human faeces, vertical transmission (trans- placental).

- Salmonella is commonly found in the intestinal tracts of a wide variety of animals, including poultry, domesticated animals, and reptiles. These animals can serve as reservoirs for Salmonella species. However, Salmonella Typhi and Salmonella Paratyphi are exceptions, as they are **strictly human pathogens**.

Pathogenesis

- The vast majority of salmonellae, however, are chiefly pathogenic in animals that constitute the reservoir for human infection; these include poultry, pigs, rodents, cattle, pets (from turtles to parrots), and many others
- Stomach acidity and normal intestinal microbiota are important determinants of susceptibility.
- The salmonella invades peyer paches and transported to other intestinal L.N. where they multiply in Mononuclear cells to mesenteric L.N. to blood through thoracic duct (transient bacteraemia).
- Circulating organism reach reticule-endothelial cells in liver ,spleen and bone marrow and circulating endo -toxin cause prolonged fever.
- Inflame mucosa and lymphatics .Necrosis and sloughing of overlaying epithelium producing ulcer that may bleed. Ulcers heal without scarring.
- Cell mediated immunity is important

- The main targets of **Shigella** are the epithelial cells of the gastrointestinal tract. **Salmonella** invades the epithelial cells, induces apoptosis, and is then taken up by resident macrophages. Inside the macrophages, the bacteria can survive and multiply. They are subsequently disseminated by macrophages and circulating monocytes to the reticuloendothelial system. This process leads to transient bacteremia and systemic manifestations characteristic of typhoid fever.

Clinical manifestations

- Incubation 7-14 days. Onset is insidious.
- **1st week**
- Fever malaise ,anorexia myalgia headache, abdominal pain ,diarrhoea early and later constipation.
- Temp. increase in a stepwise fashion become unremitting and high (a high platuea).

One of the characteristic features of fever caused by Salmonella (especially in typhoid fever) is the stepwise pattern of temperature increase. In the morning, the patient's temperature is elevated, and by the night, it becomes even higher. On the following day, the morning temperature is higher than the previous night's reading. This gradual, progressive rise in fever is known as the **step-ladder pattern**.

Another clinical sign is known as the Faget sign, which refers to relative bradycardia – a slower heart rate than expected for the degree of fever.

- **2nd week**
- High fever, fatigue, cough ,epistaxis. abdominal symptoms more sever, rose spots and rash.
- Patients may develop a cough and epistaxis (bleeding from the nose) due to involvement of the liver, spleen, and bone marrow. Complications such as disseminated intravascular coagulation (DIC), thrombocytopenia, and anemia can occur. These conditions contribute to the bleeding manifestations, including nasal bleeding.
- Another characteristic finding is the appearance of rose spots, which are small areas of bleeding under the skin. These typically appear as rosy-colored maculopapular lesions on the chest and abdomen.

- **3-4 weeks**
- If no complications, symptoms & signs gradually resolve.
- In the pre-antibiotic era, the chief complications of enteric fever were intestinal hemorrhage and perforation, and the mortality rate was 10–15%. But now it is lower than 5% of cases.

- It is important to remember that some patients will develop a carrier state, typically localized in the biliary tree, particularly in the gallbladder, especially if gallstones are present. These carriers are usually asymptomatic. If they work as food handlers, they can shed Salmonella in their feces and remain contagious, thereby perpetuating the transmission cycle.

Enterocolitis (Salmonella gastroeneritis)

- This is the most common manifestation of salmonella infection.
- In the United States, S Typhimurium and Salmonella Enteritidis are prominent, but enterocolitis can be caused by any of the more than 1400 group I serotypes of salmonellae.
- Eight to 48 hours after ingestion of salmonellae, there is nausea, headache, vomiting, and profuse diarrhea, with few leukocytes in the stools. Low-grade fever is common, but the episode usually resolves in 2–3 days. Inflammatory lesions of the small and large intestine are present.
- Bacteremia is rare (2–4%) except in immunodeficient persons.
- Blood culture results are usually negative, but stool culture results are positive for salmonellae and may remain positive for several weeks after clinical recovery.

- In Salmonella enterocolitis, transmission often occurs through contact with animals, unlike Salmonella Typhi and Paratyphi, which are exclusively human pathogens.

- In typhoid fever, blood cultures are typically positive during the first week of infection, while stool cultures become positive in the second or third week. In contrast, in Salmonella enterocolitis, blood cultures are usually negative, but stool cultures are positive.

Bacteremia with Focal Lesions

- This is associated commonly with S choleraesuis but may be caused by any salmonella serotype. After oral infection, there is early invasion of the bloodstream (with possible focal lesions in lungs, bones, meninges, and so on), but intestinal manifestations are often absent.

• We must be particularly cautious with patients who have comorbidities, such as cancer or sickle cell anemia (trait or disease), as they are more susceptible to Salmonella-induced bacteremia and focal lesions. In patients with sickle cell disease, occluded capillaries cause ischemia and necrosis of bone tissue, increasing permeability and allowing Salmonella to invade through the mucosa and submucosa and reach the circulation. This explains why Salmonella is a common cause of osteomyelitis in these patients.

- Blood culture results are positive.

Diagnostic Laboratory Tests

- A. Specimens

- culture : positive in Blood, Bone marrow, Stool & Urine culture results may be positive after the second week.

- In enteric fevers, the stools yield positive results from the second or third week on; in enterocolitis, the stools yield positive results during the first week. A positive culture of duodenal drainage establishes the presence of salmonellae in the biliary tract in carriers.

B. Bacteriologic culturing for Isolation of Salmonellae

1. Enrichment cultures— The specimen (usually stool) also is put into selenite F or tetrathionate broth, both of which inhibit replication of normal intestinal bacteria and permit multiplication of salmonellae.

2. Differential and Selective medium cultures—EMB, MacConkey, or deoxycholate medium. salmonella-shigella (SS) agar, Hektoen enteric agar and xylose-lysine decarboxylase (XLD) agar.

3. Final identification— Suspect colonies from solid media are identified by biochemical reaction patterns and slide agglutination tests with specific sera.

C. Serologic Methods

- 1. Agglutination test— In this test, known sera and unknown culture are mixed on a slide. Clumping, when it occurs, can be observed within a few minutes. This test is particularly useful for rapid preliminary identification of cultures. There are commercial kits available to agglutinate and serogroup salmonellae by their O antigens: A, B, C1 , C2 ,D, and E.

- When a culture is suspected to contain Salmonella, a sample can be mixed with specific antibodies against Salmonella antigens. If agglutination occurs, it provides further confirmation of the presence of a Salmonella colony on selective agar. This method is known as slide agglutination.

Serologic Methods

- 2. Tube dilution agglutination test (Widal test)—
- Serum agglutinins rise sharply during the second and third weeks of S Typhi infection. The Widal test to detect these antibodies against the O and H antigens has been in use for decades.
- At least two serum specimens, obtained at intervals of 7–10 days, are needed to prove a rise in antibody titer.
- Serial dilutions of unknown sera are tested against antigens from representative salmonellae. False-positive and false-negative results occur. The interpretive criteria when single serum specimens are tested vary, but a titer against the O antigen of greater than **1:320** and against the H antigen of greater than **1:640** is considered positive.
- High titer of antibody to the Vi antigen occurs in some carriers. Alternatives to the Widal test include rapid colorimetric and EIA methods.
- Results of serologic tests for Salmonella infection cannot be relied upon to establish a definitive diagnosis of typhoid fever and are most often used in resource poor areas of the world where blood cultures are not readily available .

Immunity

- Infections with S Typhi or Salmonella Paratyphi usually confer a certain degree of immunity.
- In contrast, Salmonella gastroenteritis, which is the most common form of salmonellosis, does not provide protective immunity against reinfection. Therefore, if a person consumes contaminated food again, they can develop the disease once more.
- Reinfection may occur but is often milder than the first infection. Circulating antibodies to O and Vi are related to resistance to infection and disease. However, relapses may occur in 2–3 weeks after recovery despite antibodies.
- Secretory IgA antibodies may prevent attachment of salmonellae to intestinal epithelium.
- Persons with S/S hemoglobin (sickle cell disease) are exceedingly susceptible to Salmonella infections, particularly osteomyelitis. Persons with A/S hemoglobin (sickle cell trait) may be more susceptible than normal individuals (those with A/A hemoglobin).

Treatment

- Although enteric fevers and bacteremias with focal lesions require antimicrobial treatment, the vast majority of cases of enterocolitis do not.
- Antimicrobial treatment of Salmonella enteritis in neonates is important. In enterocolitis, clinical symptoms and excretion of the salmonellae may be prolonged by antimicrobial therapy. In severe diarrhea, replacement of fluids and electrolytes is essential.
- Patients with Salmonella gastroenteritis typically do not require antibiotics, as the condition usually resolves spontaneously.
- However, in very young or elderly patients, there is an increased risk of complications such as acute renal failure and dehydration, which may warrant the use of antibiotics.

- Antimicrobial therapy of invasive Salmonella infections is with fluoroquinolones, ampicillin, trimethoprim–sulfamethoxazole, or a third-generation cephalosporin.
- Multiple drug resistance transmitted genetically by plasmids among enteric bacteria is a problem in Salmonella infections.
- Susceptibility testing is an important adjunct to selecting a proper antibiotic. In most carriers, the organisms persist in the gallbladder (particularly if gallstones are present) and in the biliary tract. Some chronic carriers have been cured by ampicillin alone, but in most cases cholecystectomy must be combined with drug treatment.

Prevention and Control

- Three percent of survivors of typhoid become healthy permanent carriers, harboring the organisms in the gallbladder; biliary tract; or, rarely, the intestine or urinary tract.
- Sanitary measures must be taken to prevent contamination of food and water by rodents or other animals that excrete salmonellae.
- Infected poultry, meats, and eggs must be thoroughly cooked.
- Carriers must not be allowed to work as food handlers and should observe strict hygienic precautions.
- Two typhoid vaccines are currently available : an oral live, attenuated vaccine and a Vi capsular polysaccharide vaccine for intramuscular use.
- Vaccination is recommended for travelers to endemic regions, especially if the traveler visits rural areas or small villages where food choices are limited, efficacy of 50–80%.
- The immunity provided by Salmonella vaccines is typically short-lived, lasting no more than one or two years. As a result, these vaccines are not included in national vaccination programs.

The End

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
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