TYPHOID AND ENTERIC FEVER

TYPHOID AND ENTERIC FEVER;OVERVIEW AND GUIDELINE FRAME

Enteric fever is an acute systemic febrile illness caused by Salmonella enterica serovar Typhi and, less commonly, serovars Paratyphi A, B, or C. In Indian pediatric practice, it should be considered in any child with fever for at least 3–5 days without an alternative focus, particularly when gastrointestinal symptoms, hepatosplenomegaly, or a toxic appearance are present. Management should be guideline-led and antimicrobial-resistance aware, using national recommendations (such as those from ##NCDC## and ##ICMR##) and pediatric consensus statements where available. Obtain blood culture before antibiotics whenever feasible, treat promptly when clinical suspicion is high, and reassess response clinically rather than changing antibiotics solely for persistent fever in the first few treatment days. Diagnostic over-reliance on serology (especially the Widal test) drives misdiagnosis and unnecessary antibiotics; a culture-first approach with disciplined reassessment is preferred. Empiric fluoroquinolones are generally avoided in children in India because of high resistance and pediatric safety considerations, with third-generation cephalosporins and azithromycin forming the backbone of therapy. This document integrates triage (outpatient versus inpatient), order-set-ready pediatric dosing, escalation when resistance is suspected, management of key complications, and prevention through typhoid conjugate vaccination and water, sanitation, and hygiene measures.

References:

- National Treatment Guidelines for Antimicrobial Use in Infectious Diseases (India) — Enteric fever section (NCDC) — https://ncdc.mohfw.gov.in/wp-content/uploads/2025/08/NTG-Version-31st-July-final.pdf
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TYPHOID AND ENTERIC FEVER;EPIDEMIOLOGY AND TRANSMISSION IN INDIA

India has a high burden of enteric fever, with transmission sustained by fecal–oral contamination of water and food in settings with inadequate sanitation, crowded housing, and unsafe municipal water storage. Population surveillance from Indian cities has reported very high annual incidence among children (often in the hundreds to over a thousand cases per 100,000 in dense urban settlements), while many rural areas have substantially lower rates, underscoring the role of infrastructure and household water handling. Children and adolescents carry a substantial share of cases; school-age children are commonly affected, but clinically important disease also occurs in preschool children and occasionally in infants. Salmonella Typhi remains the dominant cause, while Salmonella Paratyphi A contributes a meaningful minority of cases in some regions and may cause illness comparable in severity

to typhoid. Current typhoid vaccines target Typhi and do not protect against Paratyphi A, so ##WASH## remains critical even when ##TCV## coverage improves. Humans are the only reservoir; transmission is driven by acutely infected individuals and, less commonly in pediatrics, chronic carriers with prolonged fecal shedding. Risk is higher for household contacts of a case, children consuming untreated water or street food, and communities with sewage–water interfaces. Because antimicrobial resistance is an increasing constraint, prevention is central to reducing morbidity, household economic impact, and antibiotic pressure.
References:
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- Geographic pattern of typhoid fever in India: a model-based analysis (Journal of Infectious Diseases) — https://academic.oup.com/jid/article/224/Supplement_5/S475/6433809
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TYPHOID AND ENTERIC FEVER;ETIOLOGY AND PATHOGENESIS
After ingestion of contaminated food or water, Salmonella organisms survive gastric acidity, invade the intestinal mucosa (often via Peyer’s patches in the terminal ileum), and are taken up by macrophages. Intracellular survival allows dissemination through lymphatics into the bloodstream (primary bacteremia) and seeding of the reticuloendothelial system, especially liver, spleen, and bone marrow. The incubation period is usually about one to two weeks but can be shorter or longer depending on inoculum and host factors. With ongoing intracellular replication, a sustained secondary bacteremia develops, driving prolonged fever and systemic symptoms. Salmonella Typhi expresses a Vi capsular antigen that reduces opsonization and dampens early intestinal inflammation, contributing to the insidious onset. Biliary excretion leads to re-entry of organisms into the gut, perpetuating intestinal injury and producing characteristic ulceration of Peyer’s patches. In untreated or late-treated disease, these ulcers may bleed or perforate, typically in the second to third week. Rose spots, when present, reflect skin microembolic seeding, but they are transient and often absent in children. Relative bradycardia can occur but is not reliable in pediatrics. Early effective antibiotics truncate this natural history, reducing complications and mortality; prolonged fecal shedding may still occur during convalescence, while true chronic carriage is rare in children but important for outbreak propagation.
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TYPHOID AND ENTERIC FEVER;CLINICAL PRESENTATION AND EXAMINATION
Fever is the cardinal feature of enteric fever and is usually persistent for days to weeks without treatment. A step-ladder pattern is classically described, but many children present with continuous or fluctuating high fever without a distinct pattern. Early symptoms include malaise, headache, anorexia, myalgias, and sometimes a dry cough. Gastrointestinal manifestations typically emerge or intensify during the first week: abdominal pain, nausea, vomiting, diarrhea or constipation, and abdominal distension. Hepatomegaly and splenomegaly are common on examination, along with a coated tongue and relative dehydration. Relative bradycardia (pulse–temperature dissociation) can be a clue in older children but is inconsistent in pediatrics. Rose spots on the trunk may occur but are uncommon, transient, and easily missed on pigmented skin. Neuropsychiatric involvement ranges from apathy and confusion to frank encephalopathy in severe illness, usually later in the course or with shock. Complications such as gastrointestinal bleeding or intestinal perforation typically occur after more than a week of untreated or partially treated illness and should be anticipated with repeated abdominal examinations. In younger children, the presentation may resemble gastroenteritis or sepsis with fewer “classic” signs, whereas older children and adolescents more often report prominent headache and localized abdominal pain. Because co-infections are possible, clinical assessment should remain broad even when enteric fever seems likely.
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TYPHOID AND ENTERIC FEVER;CLINICAL WORKFLOW, CASE DEFINITION, AND TRIAGE
A practical workflow begins with early recognition, severity stratification, and placement of care. A suspected case is a child in a typhoid-endemic setting with fever for at least 3–5 days without another clear focus, especially with gastrointestinal symptoms,

hepatosplenomegaly, or a typical “typhoidal” appearance. A confirmed case requires laboratory isolation of Salmonella (most often from blood culture). At presentation, stratify severity. Mild illness is characterized by stable vital signs, good perfusion, preserved sensorium, and ability to maintain oral intake without significant dehydration. Severe illness is suggested by toxemia, persistent vomiting, moderate to severe dehydration, altered mental status, bleeding manifestations, marked abdominal distension, or concern for ileus, peritonitis, or shock. Outpatient management is appropriate only when the child can drink, tolerate oral antibiotics, and has no danger signs, with reliable follow-up in 24–48 hours and explicit return precautions for worsening vomiting, abdominal pain, bleeding, or lethargy. Hospital admission is advised for infants, children unable to maintain oral intake, or any child with toxic appearance, neurological features, gastrointestinal bleeding, suspected perforation, or poor access to urgent care. In admitted patients, initiate supportive care immediately (fluids, antipyretics, nutrition, and close abdominal and neurologic monitoring) alongside empiric antibiotics after cultures are drawn.
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TYPHOID AND ENTERIC FEVER;DIFFERENTIAL DIAGNOSIS AND CO-INFECTIONS
In India, enteric fever is a frequent cause of prolonged pediatric fever, but it is also a common label applied to other illnesses, particularly when serology is overused. Differential diagnosis should be driven by tempo of illness, epidemiologic exposure, and targeted testing, and clinicians should avoid diagnostic anchoring on “typhoid” in a child whose course is atypical. Malaria and dengue are high-priority exclusions in many regions; both can present with fever, headache, and hepatosplenomegaly, and both may coexist with enteric fever. Scrub typhus and other rickettsioses can mimic typhoid with fever and gastrointestinal symptoms; look carefully for an eschar and consider region-appropriate tests when cultures are negative or response is atypical. Leptospirosis is suggested by conjunctival suffusion, myalgias, jaundice, or flood-water exposure. Viral hepatitis (A or E) can present with fever early but typically has jaundice and more marked transaminase elevation when fever is waning. Persistent fever beyond two weeks with weight loss, lymphadenopathy, or chronic symptoms should trigger evaluation for tuberculosis, occult abscess, endocarditis, malignancy, or inflammatory conditions such as systemic juvenile idiopathic arthritis or Kawasaki disease. Failure to improve as expected on appropriate anti-typhoidal therapy

should prompt an explicit diagnostic “time-out” to reconsider these alternatives and to search for complications or co-infections.
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TYPHOID AND ENTERIC FEVER;DIAGNOSIS: CULTURE-FIRST AND CORE INVESTIGATIONS
Blood culture is the preferred diagnostic test for enteric fever and should be obtained before antibiotics whenever possible. Culture yield improves with adequate blood volume and prompt processing; a practical target is a cumulative 5–10 mL in older children (smaller volumes in infants), inoculated into appropriate broth, ideally using automated culture systems where available. Yield is highest in the first week of illness, so early sampling matters. If initial cultures are negative and fever persists with ongoing clinical suspicion, repeating a blood culture can be reasonable before declaring a non-typhoidal diagnosis, particularly if prior antibiotics were not given. Any Salmonella isolate should undergo antimicrobial susceptibility testing, and clinicians should request quantitative reporting such as ##MIC## values for key agents (for example, ceftriaxone) to support stewardship and escalation decisions. Bone marrow culture has the highest sensitivity and may remain positive even after antibiotics; it is reserved for diagnostically difficult, culture-negative cases when confirmation will change management. Supportive laboratory tests are not diagnostic but can support clinical suspicion and detect complications: complete blood count may show a normal or low total leukocyte count with eosinopenia; mild transaminase elevation is common. Imaging and focused tests should be problem-driven: obtain abdominal ultrasound or radiography for worsening abdominal pain or distension, and evaluate cerebrospinal fluid only when meningitis or an alternative neurologic process is plausibly present.
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TYPHOID AND ENTERIC FEVER;DIAGNOSIS: WHAT NOT TO USE AND TEST SELECTION BY ILLNESS STAGE

Serologic testing (including the Widal agglutination test) is a major pitfall in enteric fever diagnosis. In endemic settings, background antibody titers and cross-reactivity lead to false positives, while early illness and prior antibiotics lead to false negatives. As a result, national guidance discourages using a single Widal result to confirm or exclude typhoid; if serology is used at all because culture is unavailable, a paired, rising titer is more meaningful than a single sample, but it rarely informs acute decision-making. Many rapid antibody kits have variable performance and should not override clinical assessment or culture results. Molecular tests (##PCR##) and other novel assays exist, but they are not yet standard in routine care because of cost, variable availability, and limited programmatic validation. Test selection should follow illness stage. In the first week, prioritize blood culture and targeted tests for common alternative febrile illnesses (for example malaria or dengue, depending on local epidemiology). Between days 5 and 14, blood culture remains useful; if cultures are repeatedly negative but suspicion remains high, consider bone marrow culture in selected cases. Stool and urine cultures are not first-line for diagnosing acute disease but may become positive later and are more useful for outbreak investigations and carrier detection. In late or complicated illness, testing should pivot toward detecting complications while still attempting culture confirmation, and avoid broad “shotgun” panels that do not change management.

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TYPHOID AND ENTERIC FEVER;TREATMENT: UNCOMPLICATED OUTPATIENT ENTERIC FEVER

Uncomplicated enteric fever can be managed as an outpatient when the child is clinically stable, can drink, and has no danger signs. Antibiotics should be started after blood culture is collected when feasible, but treatment should not be delayed in a strongly suspected case. A practical first-line oral regimen is cefixime 20 mg/kg/day divided twice daily for 10–14 days.

Azithromycin is an effective alternative, particularly when cephalosporins cannot be used or local susceptibility supports it; dose 20 mg/kg once daily (maximum 1 g/day) for 7 days. Where culture demonstrates susceptibility or local data support use, co-trimoxazole is a stewardship-friendly option; dose by trimethoprim component 8 mg/kg/day divided twice daily for 14 days. Empiric fluoroquinolones are generally avoided in children because of resistance and pediatric safety concerns. Families should be counseled that fever may persist for 4–7 days even with effective therapy; assess response by the child's overall condition, oral intake, and trend of fever spikes rather than expecting immediate defervescence. Changing antibiotics in the first few days is usually unnecessary if the child looks better. Reassessment within 48 hours is recommended, with explicit advice to return urgently for worsening vomiting, abdominal pain, bleeding, lethargy, or poor intake. If the child is not improving by day 5–7, admit for evaluation of complications, alternative diagnoses, or resistance. Supportive care includes oral rehydration, paracetamol for comfort, and nutritional support.
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TYPHOID AND ENTERIC FEVER;TREATMENT: SEVERE OR COMPLICATED INPATIENT ENTERIC FEVER
Hospitalized children require parenteral antibiotics plus close monitoring and supportive care. A common first-line regimen for severe disease is ceftriaxone 100 mg/kg/day (maximum 4 g/day) given intravenously once daily or in divided doses for 10–14 days, tailored to clinical response and susceptibility results. Cefotaxime (150–200 mg/kg/day in 3–4 divided doses) is a reasonable alternative when ceftriaxone is undesirable (for example, concern for biliary sludging or marked hepatitis) or when local protocols favor it. In severely ill children, combination therapy with azithromycin (20 mg/kg/day, oral or intravenous where available) may be used to augment intracellular activity and potentially hasten fever clearance, especially when reduced cephalosporin susceptibility is suspected. For children with serious beta-lactam allergy, seek specialist input; azithromycin-based regimens may be used when the child is stable and susceptibility is expected. If intestinal perforation is suspected, antibiotics must be broadened to cover secondary intra-abdominal flora (for example by adding anaerobic coverage) and surgical consultation should be immediate. Adjunct corticosteroid therapy is reserved for the sickest patients with shock or encephalopathy: dexamethasone 3 mg/kg as a loading dose, followed by 1 mg/kg every 6

hours for 48 hours. Supportive care includes fluid resuscitation, glucose monitoring, temperature control, early nutrition, and frequent abdominal and neurologic examinations to detect bleeding, perforation, or evolving organ dysfunction promptly.
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TYPHOID AND ENTERIC FEVER;MONITORING RESPONSE, CULTURE-NEGATIVE MANAGEMENT, AND STEP-DOWN
Response assessment should combine the fever curve with overall clinical trajectory. With effective therapy, many children show improved appetite, activity, and hemodynamics before fever fully resolves; defervescence may take 4–7 days, especially with cephalosporins. Persistent fever alone in the first treatment week is not equivalent to failure if the child is clearly less toxic and is eating and drinking better. A structured reassessment is warranted when there is clinical deterioration at any time, or when by day 5–7 there is minimal improvement in both fever trend and overall condition. Reassessment includes a repeat history and examination, review of antibiotic dosing and adherence, and focused evaluation for complications (for example perforation, bleeding, abscess) or alternative diagnoses and co-infections. Culture-negative illness is common when children present late or after prior antibiotics; if suspicion remains high and the child is improving, complete the planned course and ensure close follow-up rather than chasing low-yield tests. If suspicion is high but response is poor, repeat blood culture and consider bone marrow culture in selected cases to confirm diagnosis and guide escalation. Routine “test of cure” cultures are not needed in uncomplicated recovery. For hospitalized children who improve on parenteral therapy, step down to an appropriate oral agent (guided by susceptibility when available) once the child is stable and tolerating feeds, to complete a total of 10–14 days of therapy and reduce line-related complications and length of stay.
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TYPHOID AND ENTERIC FEVER;ANTIMICROBIAL RESISTANCE AND STEWARDSHIP
Antimicrobial stewardship is central to enteric fever management in India because resistance patterns evolve and directly affect empiric therapy. Resistance should be suspected when a child has received appropriate doses with good adherence yet shows no meaningful clinical improvement by day 5–7, or when culture demonstrates non-susceptibility. Clinicians should actively engage the microbiology laboratory: ensure susceptibility testing is performed for relevant agents and request ##MIC## values when available, since “susceptible” categories may mask reduced susceptibility that correlates with slower response or failure. Fluoroquinolone resistance and reduced susceptibility are common, so empiric fluoroquinolones are generally avoided in children. If ceftriaxone susceptibility is reduced and the child is clinically stable, azithromycin is often the preferred option when the organism is susceptible. If the child is severely ill, deteriorating, or culture confirms ceftriaxone-resistant or extensively drug-resistant typhoid, escalation to a carbapenem such as meropenem (for example, 40 mg/kg every 8 hours) is appropriate, ideally guided by specialist input. Reserve carbapenems for proven or strongly suspected resistance to preserve their utility. Once culture results are available and the child is improving, de-escalate to the narrowest effective oral agent to complete the course. Stewardship also includes avoiding treatment of non-typhoidal fevers based on unreliable serology and ensuring full-course completion to reduce relapse and ongoing transmission.
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TYPHOID AND ENTERIC FEVER;COMPLICATIONS I: GASTROINTESTINAL BLEEDING AND PERFORATION

Gastrointestinal complications reflect ulceration of Peyer's patches and typically occur after more than a week of illness, especially when treatment is delayed or incomplete. Gastrointestinal bleeding should be suspected with melena, hematochezia, pallor, tachycardia out of proportion to fever, or an unexplained fall in hemoglobin. Management priorities are hemodynamic assessment, intravenous access, type and cross-match, and transfusion when clinically indicated, while continuing effective anti-typhoidal antibiotics. Avoid nonsteroidal anti-inflammatory drugs in severe illness because of bleeding and renal risk. Most bleeds are self-limited with supportive care, but recurrent or massive bleeding warrants urgent senior review and surgical or endoscopic consultation depending on local capability. Intestinal perforation is a surgical emergency. Suspect it in a child with enteric fever who develops sudden severe abdominal pain, guarding or rigidity, progressive distension, or shock; fever may paradoxically lessen as peritonitis evolves. Do not delay: make the child nil per os, begin aggressive fluid resuscitation, obtain urgent imaging when feasible, and involve pediatric surgery immediately. Antibiotics must be broadened to cover secondary intra-abdominal contamination (including anaerobes) in addition to Salmonella coverage, and operative repair with peritoneal washout is usually required. Postoperative care often requires intensive monitoring, ongoing antibiotics, and vigilance for abscess and sepsis.
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TYPHOID AND ENTERIC FEVER;COMPLICATIONS II: NEUROLOGIC, HEPATIC, CARDIAC, AND HYPERINFLAMMATION
Typhoid encephalopathy presents with delirium, confusion, obtundation, or coma in a child with systemic toxicity and requires immediate escalation of care. Stabilize airway and circulation, correct hypoglycemia and electrolyte derangements, treat shock, and ensure effective parenteral antibiotics. Adjunct dexamethasone is recommended for severe disease with shock or encephalopathy using a short, high-dose regimen (3 mg/kg loading dose followed by 1 mg/kg every 6 hours for 48 hours). Exclude alternative neurologic diagnoses when clinically indicated rather than reflexively performing lumbar puncture. Hepatic involvement is common and usually manifests as mild transaminase elevation with hepatomegaly; jaundice should prompt evaluation for viral hepatitis or cholangitis. Management is largely supportive with avoidance of hepatotoxic drugs and continuation of appropriate antibiotics. Myocarditis is uncommon but potentially fatal; suspect it with

persistent tachycardia out of proportion to fever, arrhythmia, gallop rhythm, or unexplained shock. Obtain electrocardiography and echocardiography, involve cardiology, and provide hemodynamic support while treating the infection. Secondary ##HLH## is a rare but critical consideration in children with persistent or recrudescent fever despite therapy, cytopenias, organomegaly, hyperferritinemia, or coagulopathy. Initiate urgent evaluation and specialist consultation; treat the infection and begin immunomodulatory therapy (often corticosteroids) when the syndrome is strongly suspected.
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TYPHOID AND ENTERIC FEVER;AGE AND SPECIAL POPULATIONS
Age modifies both presentation and the threshold for admission. Neonatal typhoid is exceedingly rare but may present as nonspecific sepsis with poor feeding, abdominal distension, or shock; manage in hospital with sepsis protocols and a third-generation cephalosporin such as cefotaxime rather than ceftriaxone in early infancy because of bilirubin-related safety concerns. Infants and toddlers may present with diarrhea, vomiting, and dehydration with fewer classic signs; the threshold for admission is lower because deterioration can be rapid and oral therapy may not be reliable. In preschool and school-age children, abdominal pain, hepatosplenomegaly, and a typical “typhoidal” appearance are more often evident, and selected uncomplicated cases can be treated as outpatients when follow-up is assured. Adolescents approach adult patterns of illness and may be at risk for late complications if diagnosis is delayed. Special populations require additional caution. Children with ##SAM##, chronic disease, or immunocompromise (including ##HIV## or chemotherapy) may have blunted signs, higher bacteremia burden, and slower recovery, and may need longer courses and closer monitoring. In such children, consider broader differentials including invasive non-typhoidal Salmonella. Children with hemoglobinopathies can develop focal Salmonella infections such as osteomyelitis; persistent localized pain or swelling warrants targeted imaging and prolonged therapy. Social factors matter at every age: poor ability to return for review or uncertain adherence should lower the threshold for admission or supervised therapy.
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TYPHOID AND ENTERIC FEVER;FOLLOW-UP, RELAPSE, AND CARRIER MANAGEMENT

Clinical follow-up after treatment is aimed at confirming recovery, detecting relapse, and reinforcing prevention. Most children improve rapidly once effective antibiotics are started, but fatigue and weight loss may persist for weeks; counsel families on nutrition and gradual return to activity. Emphasize full-course completion even after early improvement to reduce relapse risk. Relapse can occur, typically within 1–3 weeks after completing therapy, and usually presents as recurrent fever with a milder clinical picture. Relapse should be managed as a new episode: obtain culture if possible, review prior adherence and antibiotic choice, and treat with a full course guided by susceptibility. If fever recurs with focal symptoms (for example bone pain or localized tenderness), evaluate for a focal Salmonella complication and extend therapy as needed. Persistent shedding of Salmonella in stool can occur transiently during convalescence; true chronic carriage is uncommon in children, so routine post-illness stool cultures are not recommended for otherwise well pediatric patients. Targeted stool culture follow-up is reasonable in outbreaks, recurrent family clusters, or when an older adolescent is involved in food handling. When a carrier state is confirmed, prolonged antibiotic therapy guided by sensitivity and public health coordination may be required. Children can usually return to school once afebrile, clinically well, and able to participate normally; strict hand hygiene and safe water practices reduce household transmission during recovery.

References:

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- The treatment of enteric fever (review, PMC) — https://pmc.ncbi.nlm.nih.gov/articles/PMC1847736/
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TYPHOID AND ENTERIC FEVER;PREVENTION I: IMMUNIZATION
Vaccination is a key preventive strategy in India, complementing improvements in water and sanitation. The preferred product for children is the typhoid conjugate vaccine (##TCV##), which can be given from 6 months of age and produces T-cell–dependent immunity with longer protection than older polysaccharide vaccines. ##IAP## ##ACVIP## guidance supports a single dose in infancy (commonly administered around 9–12 months, and permissible from 6 months), with catch-up vaccination for older unvaccinated children and adolescents. Current practice generally does not require a routine booster in childhood, although policies may evolve as long-term effectiveness data mature. ##TCV## can be coadministered with other routine childhood vaccines at separate sites; vaccination should be deferred only for the usual reasons such as a severe acute illness or a prior anaphylactic reaction to a vaccine component. The older Vi polysaccharide vaccine is less preferred because it is not immunogenic in children under 2 years and requires repeat dosing for sustained protection. The oral live Ty21a vaccine is less commonly used in Indian pediatrics because of age and dosing constraints. Programmatically, introduction into the public schedule has been recommended by national advisory groups (##NTAGI##) with phased implementation; clinicians should follow local public health schedules while ensuring access through private services when needed. After recovery from acute enteric fever, vaccination can be offered at follow-up once the child is clinically well. Available typhoid vaccines do not protect against Salmonella Paratyphi A, so immunization must be paired with ##WASH## measures and outbreak control.
References:
- Typhoid vaccines — IAP ACVIP (guidance page) — https://acvip.org/parents/columns/typhoid.php
- IAP Advisory Committee on Vaccines and Immunization Practices: recommended immunization schedule (Indian Pediatrics PDF) — https://www.indianpediatrics.net/dec2018/1066.pdf
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TYPHOID AND ENTERIC FEVER;PREVENTION II: WASH, OUTBREAK RESPONSE, AND REPORTING
Enteric fever control depends on interrupting fecal–oral transmission through water, sanitation, and hygiene (##WASH##) and on timely public health action when clusters occur. Clinicians should counsel families on safe drinking water (boiling or reliable filtration), handwashing with soap after toileting and before food handling, and avoidance of high-risk uncooked foods and unsafe street beverages for children. When multiple suspected or confirmed cases occur in a locality, school, or hostel, notify local public health authorities and surveillance systems (such as ##IDSP##) to trigger investigation and source control. Outbreak response typically includes confirmation with cultures from early cases, rapid assessment of common exposures, and inspection and testing of water sources and food

handling sites. Immediate control measures may include chlorination of water supplies, cleaning storage tanks, temporary closure of implicated kitchens, and community messaging on boiling water and hand hygiene. Routine antibiotic prophylaxis for contacts is generally discouraged because it promotes resistance; instead, contacts should be educated to seek care promptly for fever. Targeted stool testing and treatment of identified adult carriers, especially food handlers, may be required, with clearance before return to food preparation. Where feasible, reactive ##TCV## campaigns can be considered to reduce transmission during outbreaks, alongside sustained infrastructure improvement.
References:
- Integrated Disease Surveillance Programme: district surveillance manual — outbreak investigation module (IDSP) — https://www.idsp.mohfw.gov.in/WriteReadData/OldSite/2WkDSOSept08/Resources_files/DistrictSurvMan/Module8.pdf
- Typhoid fever: control & challenges in India (review, PMC) — https://pmc.ncbi.nlm.nih.gov/articles/PMC6977362/
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TYPHOID AND ENTERIC FEVER;CONTROVERSIES, PEARLS, AND PITFALLS
A frequent pitfall is equating persistent fever with antibiotic failure. Fever may take up to a week to resolve on effective therapy, especially with cephalosporins; judge response by toxicity, intake, and hemodynamics, and change antibiotics early only when the child is worsening or clearly not improving by day 5–7. Another common pitfall is diagnostic overconfidence based on serology. A “Widal-positive” result does not confirm typhoid in endemic areas and can distract from malaria, dengue, rickettsioses, tuberculosis, malignancy, or inflammatory disease; culture-first diagnosis and scheduled diagnostic time-outs reduce this harm. Classic findings such as rose spots and relative bradycardia are supportive when present but are often absent in children; their absence should not lower suspicion when the overall syndrome fits. Stewardship requires resisting unnecessary broad-spectrum escalation and using susceptibility data to de-escalate; renewed susceptibility to older agents in some regions creates an opportunity to spare azithromycin and third-generation cephalosporins when culture confirms sensitivity. Empiric fluoroquinolones are generally avoided in children because of resistance and safety considerations, despite occasional debate about their role in exceptional circumstances. Corticosteroids have a narrow indication: reserve high-dose dexamethasone for enteric fever with shock or encephalopathy, not for routine cases. Finally, remember that vaccination reduces typhoid but not paratyphoid; do not dismiss enteric fever in vaccinated children, and maintain ##WASH## measures as the durable prevention layer.
References:
- National Treatment Guidelines for Antimicrobial Use in Infectious Diseases (India) — Enteric fever section (NCDC) —

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TYPHOID AND ENTERIC FEVER;CLINICAL VIGNETTES AND DECISION POINTS
Vignette 1: A 7-year-old with 6 days of continuous fever, mild abdominal pain, and splenomegaly is drinking well and has stable vital signs. Blood culture is obtained and oral therapy is started. The key decision is outpatient versus inpatient care; because the child has no danger signs and follow-up is reliable, outpatient treatment with cefixime or azithromycin is reasonable, with review in 48 hours and clear return precautions.

Vignette 2: A 3-year-old presents after 10 days of fever with vomiting, lethargy, delayed capillary refill, and abdominal distension. This is severe disease until proven otherwise. Obtain cultures immediately, begin parenteral ceftriaxone, aggressive fluids, and close monitoring. If shock or encephalopathy is present, add high-dose dexamethasone while evaluating for co-infections and complications. Escalate promptly if peritonitis is suspected.

Vignette 3: An adolescent treated empirically as “typhoid” on the basis of Widal remains febrile on day 6 of oral cefixime with minimal clinical improvement. The decision point is to avoid reflex antibiotic switching without reassessment. Recheck dosing and adherence, repeat a focused examination, obtain blood culture if not already done, and evaluate for rickettsial disease, malaria, dengue, or focal complications. If culture confirms reduced susceptibility or the child worsens, escalate therapy according to susceptibility and local resistance patterns.

Vignette 4: A vaccinated child develops enteric-fever syndrome. Do not assume vaccine failure or exclude the diagnosis; obtain cultures and treat based on severity, recognizing that paratyphoid and other febrile illnesses remain possible despite ##TCV##.
References:
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