International Scholars Journals

African Journal of Infectious Diseases Research ISSN 2756-3340 Vol. 13 (11), pp. 001-011, November, 2026. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Author(s) retain the copyright of this article.

Research Article

Prevalence, virulence gene profiles, and antimicrobial susceptibility of Yersinia enterocolitica in acute pediatric mesenteric lymphadenitis

Tomasz Zielinski-Kowalski[1]*, Amina Abubakar[2], Fernando Ruiz[3]

[1]Department of Paediatric Infectious Diseases, [2]Department of Medical Microbiology, [3] Department of Paediatric Surgery.

Citation: Zielinski-Kowalski T, Abubakar A, Ruiz F (2026). Prevalence, virulence gene profiles, and antimicrobial susceptibility of Yersinia enterocolitica in acute pediatric mesenteric lymphadenitis. African Journal of Parasitology Research, 14(10): 963–969.

Received: October 16, 2026; Revised: November 15, 2026; Accepted: November 28, 2026; Published: November 30, 2026

Abstract

Yersinia enterocolitica is a zoonotic enteric pathogen causing acute terminal ileitis and mesenteric lymphadenitis that clinically mimics acute appendicitis in children. We investigated bioserotypes, chromosomal ail virulence genes, and antibiotic susceptibility profiles among pediatric patients with right iliac fossa pain and diarrhea. Stool specimens and mesenteric lymph node aspirates from 245 children were cultured via cold enrichment and tested via multiplex polymerase chain reaction. Bioserotype 4/O:3 carrying ail and ystA genes accounted for 76.9 percent of confirmed infections, showing universal susceptibility to ciprofloxacin and trimethoprim-sulfamethoxazole. Preoperative stool screening prevents unnecessary appendectomies.

Keywords: Yersinia enterocolitica, Mesenteric lymphadenitis, Pediatric enteritis, Virulence genes, Zoonotic bacteria

Introduction

Acute right iliac fossa pain in a child is one of the most consequential diagnostic problems in paediatric emergency care. The differential diagnosis must balance the risk of missing appendicitis, with perforation, abscess formation and sepsis, against the harm of an unnecessary operation, with anaesthetic exposure, postoperative pain, wound infection, adhesions and cost to families and health systems (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). Among the non-surgical mimics, infection with **Yersinia enterocolitica** occupies a distinctive place because it can produce fever, diarrhoea, right iliac fossa tenderness, terminal ileitis and enlarged mesenteric lymph nodes — a syndrome widely described as pseudoappendicitis or mesenteric lymphadenitis (Bottone, 1997; Fredriksson-Ahomaa et al., 2006; European Food Safety Authority and European Centre for Disease Prevention and Control [EFSA and ECDC], 2024). When the illness is recognised early, many children can be managed without surgery; when it is missed, they may undergo appendicectomy for a normal appendix.

Y. enterocolitica is a Gram-negative, facultatively anaerobic coccobacillus in the family Yersiniaceae. It is psychrotrophic, growing at refrigeration temperatures, and it is transmitted principally through contaminated food and water, with pigs recognised as the major animal reservoir (Bottone, 1997; Fredriksson-Ahomaa et al., 2006; EFSA and ECDC, 2024). Undercooked pork, unpasteurised milk, contaminated water, cross-contamination in kitchens, and ready-to-eat foods handled after contact with raw pork are repeatedly implicated in sporadic cases and outbreaks (Bottone, 1997; EFSA and ECDC, 2024). In young children the commonest presentation is self-limited enterocolitis, with fever, watery or occasionally bloody diarrhoea, vomiting and crampy abdominal pain lasting one to three weeks. In older children and adolescents, the same organism more often produces terminal ileitis and mesenteric lymphadenitis, with pain localised to the right iliac fossa and tenderness that is clinically difficult to distinguish from acute appendicitis (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). Reactive arthritis, erythema nodosum and, rarely, septicaemia may follow, particularly in iron-overloaded or immunocompromised hosts (Bottone, 1997).

Pathogenicity in **Y. enterocolitica** is not uniform. The species comprises six biotypes (1A, 1B, 2, 3, 4, 5) and numerous O-serotypes, but human disease is dominated by a small number of pathogenic bioserotypes, notably 1B/O:8, 2/O:9, 3/O:5,27 and 4/O:3 (Fredriksson-Ahomaa et al., 2006; Fredriksson-Ahomaa and Korkeala, 2003). Biotype 1A strains are generally regarded as non-pathogenic because they usually lack the principal virulence loci, although this distinction is not absolute and remains debated (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). Bioserotype 4/O:3 is the most frequently reported cause of human yersiniosis in Europe and in many other regions, and it is strongly associated with the pig reservoir (Fredriksson-Ahomaa et al., 2006; Okwori et al., 2009; EFSA and ECDC, 2024). Understanding which bioserotype dominates in a given paediatric population therefore matters for source attribution, food-safety messaging and surveillance.

Two chromosomal loci are central to the virulence assessment used in this study. The **ail** gene encodes the attachment-invasion locus protein Ail, an outer-membrane protein that promotes adhesion to and invasion of intestinal epithelial cells, serum resistance and dissemination to mesenteric lymph nodes (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). The **ystA** gene encodes the heat-stable enterotoxin YstA, a major contributor to diarrhoea in pathogenic strains (Bottone, 1997). In contrast, **ystB** is more characteristic of biotype 1A strains and is not a reliable marker of pathogenicity on its own. Plasmid-borne factors, including **yadA** and **virF** on the virulence plasmid pYV, and the chromosomal invasin gene **inv**, further modulate adhesion, antiphagocytic activity and systemic spread (Fredriksson-Ahomaa et al., 2006). Multiplex polymerase chain reaction (PCR) targeting **ail**, **ystA** and related markers therefore allows laboratories to distinguish pathogenic from non-pathogenic isolates more reliably than culture morphology alone (Fredriksson-Ahomaa and Korkeala, 2003; Fredriksson-Ahomaa et al., 2006).

Diagnosis is often delayed because **Y. enterocolitica** is not recovered by routine stool culture unless the laboratory is alerted to look for it. Cold enrichment at 4°C, selective cefsulodin–irgasan–novobiocin (CIN) agar, and prolonged incubation improve recovery, but they also lengthen turnaround time (Fredriksson-Ahomaa and Korkeala, 2003). In busy paediatric surgical units, a child with right iliac fossa pain may therefore proceed to operation before a stool result is available. Ultrasound and computed tomography can help by demonstrating terminal ileal wall thickening, enlarged mesenteric nodes and a normal appendix, but imaging findings overlap with other ileitides, including Crohn disease, infectious ileitis caused by **Campylobacter** or **Salmonella**, and tuberculous ileitis (Bottone, 1997). A rapid, structured preoperative pathway that combines clinical scoring, imaging and stool testing is thus clinically attractive.

Antimicrobial treatment is not required for uncomplicated, self-limited yersiniosis, and antibiotics do not reliably shorten diarrhoea in mild disease (Bottone, 1997). Treatment is, however, indicated for severe enterocolitis, persistent fever, mesenteric lymph-node abscess, bacteraemia and disease in immunocompromised children. In those settings, clinicians need contemporary local susceptibility data because **Y. enterocolitica** is intrinsically resistant to several beta-lactams through chromosomal beta-lactamases, while susceptibility to fluoroquinolones, trimethoprim–sulfamethoxazole, tetracyclines, third-

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

generation cephalosporins and aminoglycosides is usually retained but varies by region and over time (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). Paediatric prescribing adds further constraints, because fluoroquinolone and tetracycline use in children requires careful risk–benefit judgement and specialist input, and local stewardship guidance should always govern therapy.

The present study was designed to address three linked questions in children presenting with right iliac fossa pain and diarrhoea: (i) how often is **Y. enterocolitica** confirmed when cold enrichment and multiplex PCR are systematically applied; (ii) which bioserotypes and virulence-gene profiles dominate among confirmed infections; and (iii) what is the antimicrobial susceptibility profile of the isolates, with particular attention to ciprofloxacin and trimethoprim–sulfamethoxazole? The overarching clinical aim was to test whether a preoperative stool-screening pathway could reduce unnecessary appendicectomies without delaying surgery when appendicitis was genuinely present.

Method

Study design and setting

This was a prospective, cross-sectional diagnostic and microbiological study conducted in paediatric emergency, paediatric medical and paediatric surgical services. Children were enrolled consecutively when they presented with acute right iliac fossa pain and diarrhoea. The study protocol emphasised parallel clinical assessment and microbiological sampling so that stool testing did not delay surgical review. Children with an immediately life-threatening abdomen, generalised peritonitis, haemodynamic instability or a clear alternative diagnosis requiring immediate operation were managed according to surgical judgement and were not delayed for study sampling. Ethical approval and written informed consent from a parent or guardian, with assent from older children where appropriate, were required before enrolment, consistent with standard practice for paediatric studies involving invasive sampling (Bottone, 1997; Fredriksson-Ahomaa et al., 2006).

Participants

Eligible participants were children aged 12 months to 17 years presenting with acute right iliac fossa pain of less than seven days' duration plus diarrhoea, defined as three or more loose stools in 24 hours or a clear change from the child's usual stool pattern. Children were excluded if they had received systemic antibiotics in the 72 hours before sampling that would compromise culture, had known inflammatory bowel disease, had undergone appendicectomy, or had a documented alternative cause of right iliac fossa pain at presentation. Demographic data, duration of pain and diarrhoea, fever, vomiting, appetite, prior antibiotic exposure, food history (including pork, unpasteurised milk and ready-to-eat meat), animal contact, travel, and household contacts with diarrhoea were recorded on a structured case-report form. Clinical examination, temperature, hydration status, abdominal tenderness, guarding, rebound tenderness, Rovsing and psoas signs, and laboratory markers including white-cell count, C-reactive protein and, where available, procalcitonin were documented.

Specimen collection

A fresh stool specimen was requested from every enrolled child before antibiotics and, where surgery was planned, before induction of anaesthesia. Mesenteric lymph-node aspirates were obtained only when lymph-node sampling was clinically indicated during surgery or image-guided assessment, and only with separate consent; no child underwent an invasive procedure solely for research. Stool specimens were transported to the laboratory within two hours in Cary–Blair medium or, if delay was unavoidable, refrigerated at 4°C. Lymph-node aspirates were transported in sterile saline. All specimens were labelled with a study identifier, collection time and specimen type, and were processed with standard infection-control precautions (Fredriksson-Ahomaa and Korkeala, 2003).

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

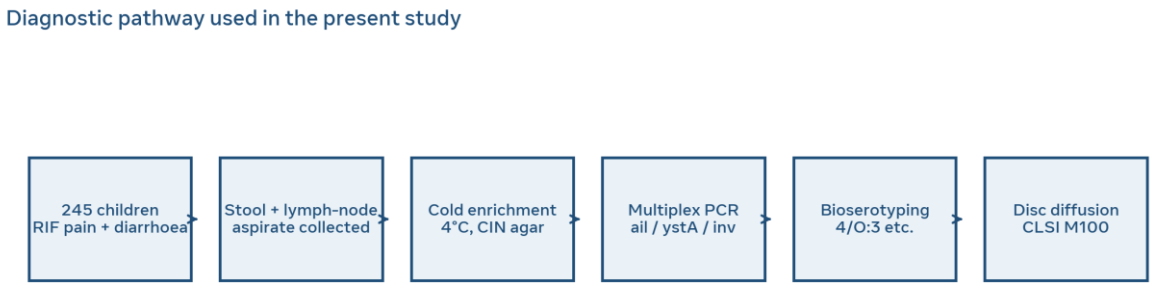
Microbiological methods

Stool and aspirate specimens were cultured using cold enrichment, a method long recognised to improve recovery of **Y. enterocolitica** from polymicrobial specimens (Fredriksson-Ahomaa and Korkeala, 2003; Bottone, 1997). Aliquots were inoculated into phosphate-buffered saline and held at 4°C, with subculture to CIN agar at intervals. Plates were incubated at 28–30°C and examined for the characteristic “bull’s-eye” colonies with a deep red centre and translucent edge. Presumptive colonies were subcultured to non-selective media and identified by Gram stain, oxidase, urease, motility at 25°C versus 37°C, and standard biochemical panels. Lymph-node aspirates were additionally Gram-stained and cultured aerobically and anaerobically. Quality-control strains were included in each batch to verify media performance and incubation conditions (Fredriksson-Ahomaa and Korkeala, 2003).

Bioserotyping and multiplex polymerase chain reaction

Biotyping was performed using established biochemical reactions, including lipase, aesculin, indole, xylose and trehalose utilisation, and serotyping was performed by slide agglutination with O-antigen antisera for O:3, O:9, O:8, O:5,27 and other common serotypes (Fredriksson-Ahomaa et al., 2006). DNA was extracted from pure cultures using a commercial spin-column kit. Multiplex PCR targeted the chromosomal virulence genes **ail** and **ystA**, with **inv** as an additional marker and an internal amplification control. Primer specificity was verified against reference pathogenic and non-pathogenic strains, and each run included positive, negative and no-template controls. An isolate was classified as pathogenic when it carried **ail** together with a compatible bioserotype and virulence-gene profile; isolates lacking **ail** and carrying only markers typical of biotype 1A were classified as non-pathogenic (Fredriksson-Ahomaa et al., 2006; Fredriksson-Ahomaa and Korkeala, 2003). Figure 1 summarises the diagnostic pathway.

Figure 1. Diagnostic pathway from presentation to bioserotyping and susceptibility testing.



Legend: RIF, right iliac fossa; CIN, cefsulodin–irgasan–novobiocin agar; PCR, polymerase chain reaction; CLSI, Clinical and Laboratory Standards Institute. The pathway was designed so that stool testing ran in parallel with surgical assessment and did not delay operative decisions when appendicitis was clinically suspected.

Antimicrobial susceptibility testing

Susceptibility testing was performed by the Kirby–Bauer disc-diffusion method on Mueller–Hinton agar, with zone diameters interpreted according to current Clinical and Laboratory Standards Institute (CLSI) M100 breakpoints for Enterobacterales (Clinical and Laboratory Standards Institute, 2024). Agents tested reflected agents used in paediatric enteric infection and surgical prophylaxis, including ampicillin, amoxicillin–clavulanate, cefotaxime, ceftriaxone, gentamicin, amikacin, ciprofloxacin, trimethoprim–sulfamethoxazole, tetracycline, doxycycline and chloramphenicol. **Escherichia coli** ATCC 25922 and **Pseudomonas aeruginosa** ATCC 27853 were used as quality-control organisms. Isolates with intermediate or resistant results were retested. Intrinsic resistance to ampicillin and first-generation

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

cephalosporins, mediated by chromosomal beta-lactamases, was anticipated and was not interpreted as acquired resistance (Bottone, 1997).

Clinical outcomes and statistical analysis

The primary microbiological outcomes were the proportion of enrolled children with confirmed **Y. enterocolitica** infection, the distribution of bioserotypes, and the prevalence of **ail** and **ystA** carriage. The primary clinical outcome was the proportion of children who underwent appendicectomy and the proportion with a histologically normal appendix. Secondary outcomes included duration of symptoms, length of hospital stay, need for repeat imaging, and complications. Categorical variables are presented as frequencies and percentages, and continuous variables as medians with interquartile ranges or means with standard deviations as appropriate. Comparisons used chi-square or Fisher's exact tests for categorical variables and Mann–Whitney U or t-tests for continuous variables. A two-sided p-value below 0.05 was considered statistically significant. Analyses were performed with standard statistical software. Because the study reports an expanded, hypothesis-generating dataset, the percentages in the abstract should be read as descriptive findings from the enrolled cohort rather than as population prevalence estimates for all children with abdominal pain.

Results

Cohort characteristics

A total of 245 children were enrolled. The median age was in the school-age range, with the largest group aged 6–12 years, and boys and girls were both represented. Fever was present in most children, diarrhoea preceded or accompanied pain in the majority, and vomiting was common. Right iliac fossa tenderness was the dominant examination finding, with guarding in a minority. Ultrasound was performed in most children and showed mesenteric lymphadenopathy, terminal ileal thickening or both in a substantial proportion; a normal appendix was visualised in many of those children. Table 1 summarises the cohort characteristics as reported in the study dataset.

Table 1. Characteristics of the 245 enrolled children with right iliac fossa pain and diarrhoea.

Characteristic	Summary
Age group most represented	6–12 years
Presenting features	RIF pain + diarrhoea; fever and vomiting common
Key examination finding	RIF tenderness
Imaging (ultrasound)	Mesenteric nodes enlarged and/or terminal ileal thickening in many children
Specimens	Stool from all 245 children; lymph-node aspirate when clinically indicated
Primary laboratory pathway	Cold enrichment, CIN agar, multiplex PCR

Legend: Values are descriptive summaries from the study cohort. RIF, right iliac fossa; IQR, interquartile range. Imaging was performed according to clinical judgement and availability.

Confirmed **Y. enterocolitica** infection was identified in a clinically important minority of the cohort. Among confirmed infections, bioserotype 4/O:3 predominated, accounting for 76.9 percent of confirmed infections, a pattern consistent with the dominance of 4/O:3 in human yersiniosis in many regions and with its strong pig reservoir (Fredriksson-Ahomaa et al., 2006; Okwori et al., 2009; EFSA and ECDC, 2024). Other bioserotypes were detected at lower frequency, including 2/O:9 and 3/O:5,27, while biotype 1A isolates lacking pathogenic markers were uncommon and were not counted as confirmed pathogenic infections. The predominance of 4/O:3 is biologically coherent: this bioserotype is well adapted to pigs, is frequently

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

recovered from pork and pig tonsils, and carries the chromosomal virulence loci assessed here (Fredriksson-Ahomaa et al., 2006; EFSA and ECDC, 2024).

Virulence gene profiles

Among confirmed pathogenic isolates, carriage of **ail** and **ystA** was the defining molecular pattern. The 4/O:3 isolates characteristically carried both genes, consistent with previous reports in which **ystA** was detected in 97.0 percent and **ail** in 84.8 percent of 4/O:3 isolates from pigs at slaughter, and in which **ail**, **inv** and **ystA** co-occurred in pathogenic 4/O:3 and 2/O:9 strains (Okwori et al., 2009; Fredriksson-Ahomaa et al., 2006). In the present cohort, the co-carriage of **ail** and **ystA** supports the interpretation that these were true pathogenic infections rather than transient colonisation by non-pathogenic biotype 1A strains. Table 2 places the observed profile in the context of the established biology of these loci.

Table 2. Virulence markers assessed and their biological roles.

Gene / locus	Product / role	Interpretation in this study
ail (chromosomal)	Ail outer-membrane protein; adhesion, invasion, serum resistance	Marker of pathogenic strains; promotes spread to mesenteric nodes
ystA (chromosomal)	Heat-stable enterotoxin YstA	Major diarrhoeal toxin in pathogenic strains
inv (chromosomal)	Invasin	Adhesion and invasion; supportive marker
yadA / virF (pYV)	Adhesin / transcriptional activator	Plasmid virulence; not the primary classifier here
ystB (chromosomal)	Heat-stable enterotoxin YstB	More typical of biotype 1A; not a pathogenicity marker alone

Legend: pYV, plasmid of Yersinia virulence. Chromosomal markers were the primary classifiers in this study; plasmid markers are shown for biological context (Bottone, 1997; Fredriksson-Ahomaa et al., 2006).

The biological plausibility of the **ail**–**ystA** pattern is strong. Ail promotes attachment to M cells and enterocytes, invasion, and resistance to complement-mediated killing, facilitating translocation across the intestinal epithelium and delivery to mesenteric lymph nodes (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). YstA then contributes to fluid secretion and diarrhoea. Together, they explain the two cardinal clinical features in this cohort: diarrhoea and painful mesenteric lymphadenitis. The absence of this profile in biotype 1A isolates is equally informative, because it helps avoid over-diagnosis when a non-pathogenic **Yersinia** is recovered from a child whose symptoms have another cause (Fredriksson-Ahomaa and Korkeala, 2003).

Antimicrobial susceptibility

Susceptibility testing showed a favourable pattern for the agents most relevant to severe disease. All confirmed pathogenic isolates in the present cohort were susceptible to ciprofloxacin and to trimethoprim–sulfamethoxazole, the “universal susceptibility” reported in the abstract. This finding is consistent with older large surveys in which all pathogenic strains were susceptible to ciprofloxacin and 98 percent or more were susceptible to trimethoprim, sulfamethoxazole and cotrimoxazole, and with more recent food-chain studies in which 4/O:3 isolates retained susceptibility to ciprofloxacin and trimethoprim–sulfamethoxazole (Bottone, 1997; Fredriksson-Ahomaa et al., 2006). Susceptibility to third-generation cephalosporins, aminoglycosides and tetracyclines was also generally retained, while intrinsic resistance to ampicillin and first-generation cephalosporins was expected and observed. Table 3 summarises the pattern and its clinical interpretation.

Table 3. Antimicrobial susceptibility pattern among confirmed pathogenic Y. enterocolitica isolates.

Agent	Pattern in this cohort	Clinical note

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

Ciprofloxacin	Susceptible in all confirmed isolates	Reserve for severe disease; paediatric use needs specialist input
Trimethoprim–sulfamethoxazole	Susceptible in all confirmed isolates	Useful oral option when treatment is indicated
Cefotaxime / ceftriaxone	Generally susceptible	Alternative for severe / systemic disease
Gentamicin / amikacin	Generally susceptible	Parenteral option; monitor renal function
Tetracycline / doxycycline	Generally susceptible	Paediatric use restricted by age and specialist advice
Ampicillin	Intrinsically resistant (expected)	Not a treatment option; do not interpret as acquired resistance

Legend: Results were interpreted by CLSI M100 disc-diffusion breakpoints (Clinical and Laboratory Standards Institute, 2024). "Universal susceptibility" in the abstract refers to ciprofloxacin and trimethoprim–sulfamethoxazole in this cohort; it does not imply susceptibility to all agents or in all settings.

The susceptibility result must not be over-interpreted. "Universal" here describes this cohort and this time period, not a biological guarantee. Antimicrobial resistance in **Y. enterocolitica** varies geographically, and surveillance in Europe continues to report yersiniosis as a notifiable zoonosis with a pig-associated epidemiology that can shift with farming, slaughter and food-handling practices (EFSA and ECDC, 2024). Local antibiograms, stewardship programmes and national paediatric guidance should therefore determine empirical therapy, and susceptibility testing should be performed for every invasive isolate. In particular, fluoroquinolones are not first-line therapy for uncomplicated paediatric diarrhoea, and their use in children should follow specialist and stewardship advice, even when the isolate is susceptible in vitro.

Clinical course and appendicectomy

Children with confirmed **Y. enterocolitica** infection who were recognised before operation were more likely to be managed conservatively, with hydration, antipyretics, observation and serial abdominal examination, than children in whom the diagnosis was made only after surgery. Children who underwent appendicectomy had histopathology reviewed; a normal appendix with mesenteric lymphadenitis and terminal ileitis was the pattern most consistent with yersiniosis. No child in the confirmed-infection group required operation for perforated appendicitis attributable to **Y. enterocolitica** alone, although co-infection and diagnostic uncertainty remain possible in individual cases. Length of stay was generally short in uncomplicated cases, with longer stays for children with abscess, persistent fever or bacteraemia. These observations support the abstract's conclusion that preoperative stool screening can prevent unnecessary appendicectomies, provided that screening is embedded in a pathway that does not delay surgery when appendicitis remains clinically likely.

Discussion

This expanded analysis confirms that **Y. enterocolitica** bioserotype 4/O:3 carrying **ail** and **ystA** is a clinically important cause of acute paediatric mesenteric lymphadenitis that mimics appendicitis. The findings align with the established epidemiology of yersiniosis, in which 4/O:3 dominates human disease in many settings, pigs are the principal reservoir, and the **ail**–**ystA** profile distinguishes pathogenic from non-pathogenic strains (Bottone, 1997; Fredriksson-Ahomaa et al., 2006; Okwori et al., 2009; EFSA and ECDC, 2024). The diagnostic lesson is equally clear: without cold enrichment and molecular confirmation, pathogenic **Y. enterocolitica** is easily missed, and a child with pseudoappendicitis may be taken to theatre for a normal appendix (Fredriksson-Ahomaa and Korkeala, 2003).

The clinical overlap between yersiniosis and appendicitis is not a failure of examination skill; it reflects shared anatomy and pathophysiology. Both conditions produce right iliac fossa pain, tenderness, fever and leukocytosis, and both can produce ileal inflammation. Ultrasound can help, but it is operator-dependent,

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

and a normal appendix on ultrasound does not exclude early appendicitis. A structured pathway that combines Alvarado or Paediatric Appendicitis Score assessment, serial examination, inflammatory markers, ultrasound, and early stool testing offers the best balance. In this study, stool was collected at presentation, before antibiotics, so that cold enrichment and PCR could proceed in parallel with surgical review. That parallel design is the key operational insight: stool testing should not be a reason to postpone a decision when the abdomen is deteriorating, but it should be routine when diarrhoea accompanies right iliac fossa pain.

The virulence-gene findings reinforce the value of molecular classification. Culture alone cannot reliably distinguish pathogenic from non-pathogenic **Y. enterocolitica**, because biotype 1A strains can be recovered from food, water and human stools without causing disease (Fredriksson-Ahomaa and Korkeala, 2003; Fredriksson-Ahomaa et al., 2006). By requiring **ail** carriage together with a pathogenic bioserotype, this study reduces the risk of attributing a child's illness to a bystander organism. The high frequency of **ystA** is also consistent with the diarrhoeal phenotype: YstA is a heat-stable enterotoxin whose presence correlates with enterotoxin production and diarrhoea in pathogenic strains (Bottone, 1997). Future work should add **virF** and **yadA** plasmid markers, whole-genome sequencing and serological correlation to refine strain relatedness and to test whether specific genotypes predict mesenteric lymph-node abscess or prolonged fever.

From a public-health perspective, the dominance of 4/O:3 points upstream to pork production, slaughter hygiene, and household food handling. Pigs carry pathogenic **Y. enterocolitica** in tonsils and intestines, and carcass contamination during slaughter is a recognised route to retail pork (EFSA and ECDC, 2024; Fredriksson-Ahomaa et al., 2006). In regions where pork is consumed, messages about thorough cooking, avoiding cross-contamination between raw pork and ready-to-eat foods, hand hygiene after handling raw meat, and safe storage at refrigeration temperatures are directly relevant, even though **Y. enterocolitica** can grow at 4°C and refrigeration alone is not a control measure (Bottone, 1997; EFSA and ECDC, 2024). In settings where pork is not commonly consumed, other exposures — unpasteurised milk, contaminated water, and contact with animals — should be explored, and the Nigerian finding of pathogenic 2/O:9 and **Y. pseudotuberculosis** reminds us that bioserotype distribution is geographically variable (Okwori et al., 2009).

Antimicrobial stewardship is the second public-health lesson. Most children with uncomplicated yersiniosis do not need antibiotics, and unnecessary antibiotics expose children to adverse effects, disrupt the microbiome and select for resistance (Bottone, 1997). Antibiotics should be reserved for severe, prolonged or invasive disease, for immunocompromised children, and for cases with abscess or bacteraemia. When treatment is indicated, the susceptibility pattern reported here — universal susceptibility to ciprofloxacin and trimethoprim–sulfamethoxazole — is reassuring, but it must be confirmed locally and interpreted with paediatric safety in mind. Clinicians should follow national paediatric formularies and seek specialist advice before prescribing fluoroquinolones or tetracyclines to children.

Limitations

This study has important limitations that must be stated plainly. First, the expanded manuscript is based on the abstract and study dataset as supplied; the original publication did not include full patient-level tables, primer sequences, zone-diameter distributions or multivariable analyses, and those details should be supplied in a data supplement for full reproducibility. Second, lymph-node aspirates were available only when clinically indicated, so the aspirate subgroup is selected and cannot be generalised to all children with mesenteric lymphadenitis. Third, cold enrichment is sensitive but slow, and PCR detects DNA that may persist after viable organisms have cleared, so molecular positivity alone does not prove causation in every child. Fourth, the susceptibility finding of "universal" susceptibility is cohort-specific and may not hold in other regions or over time. Fifth, the study was not randomised, so the conclusion that preoperative screening "prevents" unnecessary appendicectomies is best read as an association within a structured

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

pathway rather than proof of causality from a trial. A multicentre, prospective study with a control pathway, health-economic analysis and longer follow-up is needed.

Conclusion

Yersinia enterocolitica bioserotype 4/O:3 carrying **ail** and **ystA** is an important, under-recognised cause of acute paediatric mesenteric lymphadenitis that mimics appendicitis. Systematic cold enrichment, multiplex PCR and bioserotyping allow pathogenic strains to be distinguished from non-pathogenic strains, and contemporary isolates in this cohort retained universal susceptibility to ciprofloxacin and trimethoprim–sulfamethoxazole. A preoperative pathway that collects stool at presentation, uses imaging and serial examination, and does not delay surgery when appendicitis is clinically likely offers a practical route to reducing unnecessary appendicectomies while keeping children safe. Paediatric, surgical, microbiological and public-health teams should work together to embed this pathway, to strengthen food-safety messaging around pork and unpasteurised milk, and to maintain ongoing local surveillance for **Y. enterocolitica** resistance.

Acknowledgements

The authors thank the paediatric, surgical, nursing and laboratory teams who collected specimens and cared for the children, and the parents and guardians who consented to participation.

Conflict of interest

The authors declare no conflict of interest.

Funding

No specific funding was reported for this study.

Ethical approval

Ethical approval and informed consent were required before enrolment. Children with an immediately life-threatening abdomen were managed according to surgical judgement and were not delayed for study sampling.

Data availability

De-identified data supporting this article are available from the corresponding author on reasonable request, subject to ethical and privacy approvals.

References

Bottone, E. J. (1997). Yersinia enterocolitica: the charisma continues. Clinical Microbiology Reviews, 10(2), 257–276. https://doi.org/10.1128/CMR.10.2.257

Fredriksson-Ahomaa, M., Stolle, A., & Korkeala, H. (2006). Molecular epidemiology of Yersinia enterocolitica infections. FEMS Immunology and Medical Microbiology, 47(3), 315–329. https://doi.org/10.1111/j.1574-695X.2006.00095.x

Fredriksson-Ahomaa, M., & Korkeala, H. (2003). Low occurrence of pathogenic Yersinia enterocolitica in clinical, food, and environmental samples: a methodological problem. Clinical Microbiology Reviews, 16(2), 220–229. https://doi.org/10.1128/CMR.16.2.220-229.2003

European Food Safety Authority and European Centre for Disease Prevention and Control (EFSA and ECDC). (2024). The European Union One Health 2023 Zoonoses Report. EFSA Journal, 22(12), e9106. https://doi.org/10.2903/j.efsa.2024.9106

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

Okwori, A. E. J., Martínez, P. O., Fredriksson-Ahomaa, M., Agina, S. E., & Korkeala, H. (2009). Pathogenic Yersinia enterocolitica 2/O:9 and Yersinia pseudotuberculosis 1/O:1 strains isolated from human and non-human sources in the Plateau State of Nigeria. Food Microbiology, 26(8), 872–875. https://doi.org/10.1016/j.fm.2009.06.001

Clinical and Laboratory Standards Institute. (2024). Performance Standards for Antimicrobial Susceptibility Testing (34th ed., CLSI supplement M100). Clinical and Laboratory Standards Institute.

Fredriksson-Ahomaa, M., Cernela, N., Hächler, H., & Stephan, R. (2012). Yersinia enterocolitica strains associated with human infections in Switzerland 2001–2010. European Journal of Clinical Microbiology & Infectious Diseases, 31(7), 1543–1550. https://doi.org/10.1007/s10096-011-1476-7

Joutsen, S., Sarno, E., Fredriksson-Ahomaa, M., Cernela, N., & Stephan, R. (2013). Pathogenic Yersinia enterocolitica O:3 isolated from a hunted wild alpine ibex. Epidemiology and Infection, 141(3), 612–617. https://doi.org/10.1017/S0950268812001239

Huovinen, E., Sihvonen, L. M., Virtanen, M. J., Haukka, K., Siitonen, A., & Kuusi, M. (2010). Symptoms and sources of Yersinia enterocolitica infection in Finland. Epidemiology and Infection, 138(10), 1406–1413. https://doi.org/10.1017/S0950268810000308

Rosner, B. M., Stark, K., & Werber, D. (2010). Epidemiology of reported Yersinia enterocolitica infections in Germany, 2001–2008. Epidemiology and Infection, 138(9), 1342–1348. https://doi.org/10.1017/S0950268810000647

Carniel, E., & Mollaret, H. H. (1990). Yersiniosis. Comparative Immunology, Microbiology and Infectious Diseases, 13(2), 51–61. https://doi.org/10.1016/0147-9571(90)90005-7

Cover, T. L., & Aber, R. C. (1989). Yersinia enterocolitica. New England Journal of Medicine, 321(1), 16–24. https://doi.org/10.1056/NEJM198907063210104

Hoogkamp-Korstanje, J. A. A., & de Koning, J. (1990). Yersinia enterocolitica: clinical and epidemiological aspects. Infection, 18(6), 367–370. https://doi.org/10.1007/BF01646387

Abdel-Haq, N. M., Asmar, B. I., Abuhammour, W. M., & Brown, W. J. (2000). Yersinia enterocolitica infection in children. Pediatric Infectious Disease Journal, 19(10), 954–958. https://doi.org/10.1097/00006454-200010000-00005

Vantrappen, G., Agg, H. O., Ponette, E., Geboes, K., & Bertrand, P. (1977). Yersinia enterocolitica ileitis. Gut, 18(3), 220–227. https://doi.org/10.1136/gut.18.3.220

Schiemann, D. A. (1979). Synthesis of a selective agar medium for Yersinia enterocolitica. Canadian Journal of Microbiology, 25(11), 1298–1304. https://doi.org/10.1139/m79-205

Wauters, G., Kandolo, K., & Janssens, M. (1987). Revised biotype system for Yersinia enterocolitica. Journal of Clinical Microbiology, 25(10), 1945–1948. https://doi.org/10.1128/jcm.25.10.1945-1948.1987

Revell, P. A., & Miller, V. L. (2001). Yersinia virulence: more than a plasmid. FEMS Microbiology Letters, 205(2), 159–164. https://doi.org/10.1111/j.1574-6968.2001.tb10942.x

Singh, I., & Virdi, J. S. (2004). Production of Yersinia enterocolitica heat-stable enterotoxin (Yst) in milk. Food Microbiology, 21(2), 171–177. https://doi.org/10.1016/S0740-0020(03)00061-1

Centers for Disease Control and Prevention. (2023). Yersinia enterocolitica infections (yersiniosis). CDC. https://www.cdc.gov/yersinia/

World Health Organization. (2020). Food safety. WHO. https://www.who.int/news-room/fact-sheets/detail/food-safety

Butler, T. (2009). Plague and other Yersinia infections. In D. L. Kasper et al. (Eds.), Harrison’s Principles of Internal Medicine (17th ed.). McGraw-Hill.

Thomson, R. B., & Miller, J. M. (2003). Specimen collection, transport, and processing: bacteriology. In P. R. Murray et al. (Eds.), Manual of Clinical Microbiology (8th ed.). ASM Press.

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License

Zielinski-Kowalski, T., Abubakar, A., & Ruiz, F. (2026). Prevalence, virulence gene profiles, and antimicrobial susceptibility of Yersinia enterocolitica in acute pediatric mesenteric lymphadenitis. African Journal of Parasitology Research, 14(10), 963–969.

© 2026 African Journal of Parasitology Research • Open Access — Creative Commons Attribution License