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CASE REPORT

The First Case of Antimicrobial-Resistant *Salmonella* Stanley ST29 Diagnosed Secondary to Acute Cholecystitis

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Objective: To analyze the phenotypic and genomic characteristics of a non-typhoidal *Salmonella* bile isolate from a patient with chronic diarrhoea secondary to acute cholecystitis.

Methods: The patient presented with chronic diarrhoea lasting for two weeks, which was secondary to acute cholecystitis. The non-typhoidal *Salmonella* bile isolate was identified using biochemical tests, mass spectrometry, serum agglutination tests, and antimicrobial susceptibility testing. Subsequently, whole-genome sequencing (WGS) was performed to predict and annotate the serotype, multilocus sequence typing (MLST), antimicrobial resistance genes (AMR), mobile genetic elements (MGEs), and virulence genes of the isolate.

Results: The isolate was identified as *Salmonella* spp. by biochemical tests and mass spectrometry. The serotype of the isolate identified by serum agglutination tests was consistent with WGS prediction and was identified as *Salmonella* Stanley (1,4,5,12: d:1,2, ST29). It was resistant to six antimicrobial agents, including ampicillin, ciprofloxacin, cefotaxime, azithromycin, trimethoprim/sulfamethoxazole, and chloramphenicol. Five major classes of antimicrobial agents, comprising a total of 14 resistance genes were screened, including β-lactam resistance genes: $bla_{\text{TEM-1B}}$, $bla_{\text{OXA-1}}$, and $bla_{\text{DHA-1}}$; quinolone resistance gene: *qnrB4* and *aac(6')-Ib-cr*; macrolide resistance genes: *mph(A), mph(E)*, and *msr(E)*, and folate pathway inhibitor resistance genes: *sul1, sul2*, and *dfrA14*; chloramphenicol resistance genes: *floR, catA2* and *catB3*. T57S mutation in the *parC* gene was detected in the quinolone resistance-determining region (QRDR). Three plasmid replicons (*IncHI2/IncHI2A, IncR, IncN*) and three insertion MGEs were predicted. Forty-two virulence genes were predicted, of which 25 were secretion and transporter genes, and ten were fimbrial adherence genes. Only one invasive protein-regulated gene(*inv*) was found in *Salmonella* Pathogenicity Islands 1(SPI-1), and no Typhoid toxin genes were predicted.

Conclusion: The case of *Salmonella* Stanley ST29 isolated from a patient with diarrhoea lasting 55 days secondary to cholecystitis aligns with the characteristics of high drug resistance and relatively low virulence. This highlights the need for increased vigilance in clinical practice regarding invasive cases and the significant disease burden associated with the intestinal migration of antimicrobial-resistant *Salmonella* isolates.

Keywords: *Salmonella* Stanley, acute cholecystitis, antimicrobial-resistant, whole-genome sequencing, invasive

Introduction

Salmonella is a significant foodborne pathogen that is widely distributed worldwide. According to the White-Kauffmann-Le Minor scheme, over 2,600 serovars of *Salmonella* have been identified.[1] Based on their ability to cause specific diseases in humans, *Salmonella* strains are categorized into two groups: Typhoidal *Salmonella* and Non-typhoidal *Salmonella* (NTS).[2] Over the past few decades, NTS infections have emerged as a growing public health concern, with a notable increase in

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incidence rates.[3] While NTS typically caused diarrhea in humans, it also led to invasive infections such as bacteremia, urinary tract infections, and meningitis, particularly in vulnerable populations like the elderly and infants.[4–6]

Acute cholecystitis is an acute inflammatory condition of the gallbladder, characterized by severe abdominal pain, fever, and nausea.[7] It is most commonly caused by gallstone obstruction of the cystic duct, resulting in bile stagnation and subsequent bacterial infection.[8] Gallbladder disease affected approximately 20 million individuals in the United States, with 20% of patients with gallstones developed gallstone-related complications at an annual incidence rate of 1% to 4%.[9] Calculous acute cholecystitis is the first clinical presentation in 10% to 15% of all patients with gallstones.[9]

The pathogenesis of acute cholecystitis involves a complex interplay between bacterial infection, inflammation, and host immune response.[7] The etiology of acute cholecystitis is multifactorial. Previous studies have reported that approximately 20% of acute cholecystitis cases were due to intestinal bacterial infections, with the most common pathogens being *Escherichia coli*, *Klebsiella* spp., and *Enterococcus* spp.[7,10] Nevertheless, several recent studies have underscored the emerging role of NTS as a pathogen in acute cholecystitis, particularly among immunocompromised individuals or those with pre-existing biliary tract diseases.[11–13] For instance, notable cases included emphysematous cholecystitis caused by *Salmonella* Derby in the United States, *Salmonella* Enteritidis cholecystitis in Japan, and Group D NTS cholecystitis in China. *Salmonella*, with its unique virulence factors and ability to invade host tissues, may contribute to the severity and progression of acute cholecystitis. Despite these reports, the clinical impact of NTS-associated acute cholecystitis and characteristics of the isolates remained poorly documented and understood.

Here, we report the first case of antimicrobial-resistant *Salmonella* Stanley ST29 diagnosed in a patient with acute cholecystitis in Shanghai, China. We also provide detailed phenotypic and genomic analyses of the isolate, offering valuable scientific evidence for the prevention and control of NTS-related acute cholecystitis infections and for assessing the associated disease burden.

Materials and Methods

Case Presentation

On October 12, 2023, a 59-year-old male patient was admitted to Zhoupu Hospital, presenting with a 15-day history of diarrhea (occurring three times per day), loose stools, and abdominal distension. However, he did not experience vomiting or fever. The patient had a medical history of diabetes. Laboratory tests revealed elevated white blood cell (WBC) count at 18.08×10^9/L, with neutrophils comprising 87.40% of the total. Additionally, C-reactive protein (CRP) levels were at 103 mg/L, and serum amyloid A (SAA) was above the measurable limit at >320 mg/L. Despite the clinical indications, the patient refused to undergo pathogen isolation testing. As a result, empirical treatment with cefazoxime sodium (administered at 2.25 g per dose, twice daily) was initiated as a symptomatic intervention.

On November 21, the patient was admitted to the emergency room with unprovoked right upper abdominal pain, accompanied by radiating pain to the right shoulder and chest tightness. Upon admission, the patient's temperature was 36.8°C, and the heart rate was 75 beats per minute. Laboratory tests revealed WBC: 11.90×10^9/L, with neutrophils comprising 75.00% of the total. CRP and urinary amylase levels were within normal limits. Imaging studies, including ultrasound, X-ray computed tomography (CT), and magnetic resonance imaging (MRI), diagnosed acute cholecystitis and the presence of stones in the neck of the gallbladder (Figure 1A). The following day, the patient underwent laparoscopic cholecystectomy, during which bile was collected for pathogen detection. On November 24, the laboratory reported the isolation of *Salmonella* spp. from the bile sample, which was subsequently sent to the public health laboratory for further identification (Figure 1B).

Confirmation and Phenotypic Identification

The *Salmonella* isolate (GenBank ID: SAMN42186027) was confirmed by both VITEK 2 Compact (BioMérieux, France) and MALDI-TOF-MS (Bruker, Germany) in accordance with the protocols outlined in the Standard for Infectious Diarrhea (WS271-2007), Appendix B.1. Serum agglutination tests were conducted using *Salmonella* diagnostic serum and H antigen-inducing medium (SSI, Denmark) and were determined by referring to the White-Kauffmann -Le Minor scheme (9th edition). The antimicrobial susceptibility testing of the isolate was conducted using the minimum inhibitory concentrations (MICs) according to the guidelines of the Clinical and Laboratory Standards Institute (CLSI)

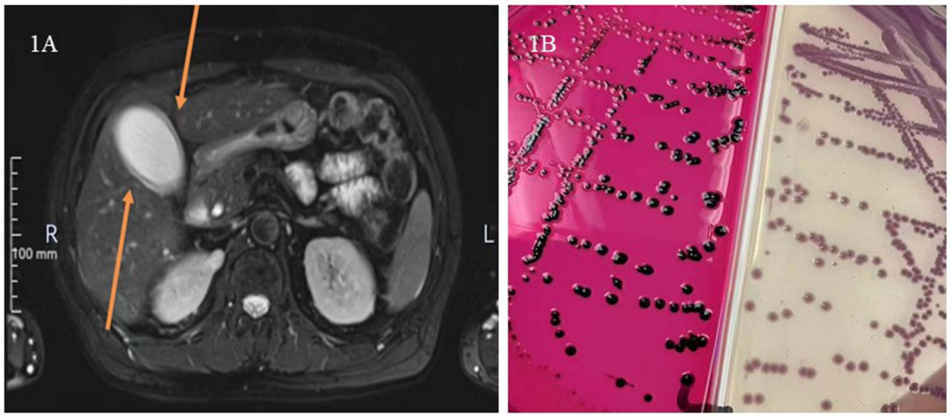

Figure 1 Imaging of the patient with the arrow pointing to the location of the gallbladder (**A**), and the morphology of *Salmonella* after incubation at 37°C for 24 hours (**B**).

(M100-Ed34, 2024). *Escherichia coli* (ATCC25922) and *Klebsiella pneumoniae* (ATCC700603) were used as reference strains for quality control. Ten antimicrobial agents used in our study were as follows: ampicillin; ciprofloxacin; cefotaxime; azithromycin; imipenem; meropenem; ertapenem; trimethoprim/sulfamethoxazole; chloramphenicol and tetracycline.

Whole-Genome Sequencing (WGS) and Genomic Analysis

DNA libraries were constructed using KAPA SYBR® FAST qPCR kits (Kapa Biosystems, USA) and sequenced using the Illumina HiSeq PE150 sequencing platform (Illumina, USA). Paired-end reads (150 bp) were generated, and the sequencing depth was ≥100x. The raw data were assembled and evaluated using CLC Genomics Workbench 24.0.1 software (Qiagen, Germany). *Salmonella* serotype prediction was performed using SISTR and SeqSero2. Multilocus sequence typing (MLST) was confirmed using PubMLST database (https://pubmlst.org/databases/). Antimicrobial resistance (AMR) genes, mobile genetic elements (MGEs) and virulence genes were screened using ResFinder 4.3.3 (http://genomicepidemiology.org/services/) and VFDB database (http://www.mgc.ac.cn/VFs/), respectively.

Results

Serotyping and Antimicrobial Susceptibility Testing

The serotype of the isolate was identified as *Salmonella* Stanley (1,4,5,12: d:1,2), which was consistent with the serotype predictions by SISTR and SeqSero2 based on WGS. The isolate exhibited resistance to six antimicrobial agents, including ampicillin, ciprofloxacin, cefotaxime, azithromycin, trimethoprim/sulfamethoxazole, and chloramphenicol. However, it remained susceptible to imipenem, meropenem, ertapenem, and tetracycline (Table 1).

MLST, AMR Genes, MGEs and Virulence Genes

The MLST of the isolate was identified as ST29. Five major classes of antimicrobial agents, comprising a total of 14 resistance genes were screened, including β-lactam resistance genes: $bla_{\text{TEM-1B}}$, $bla_{\text{OXA-1}}$, and $bla_{\text{DHA-1}}$; quinolone resistance gene: *qnrB4* and *aac(6')-Ib-cr*; macrolide resistance genes: *mph(A), mph(E)*, and *msr(E)*, and folate pathway inhibitor resistance genes: *sul1, sul2*, and *dfrA14*; chloramphenicol resistance genes: *floR, catA2* and *catB3*. Additionally, T57S mutation in the *parC* gene was detected in the quinolone resistance-determining region (QRDR). The isolate carried three plasmid replicons: *IncHI2/IncHI2A, IncR*, and *IncN*, as well as three insertional MGEs: *ISSpu10, IS6100*, and *ISVsa3*. The AMR genes *mph(E)* and *msr(E)* were located in *ISSpu10, mph(A)* in *IS6100*, and *floR* in *ISVsa3*. A total of 42 virulence genes were predicted, including 25 secretion and transporter genes, ten fimbrial adherence determinants, four nonfimbrial adherence determinants, three other virulence genes, as well. The *Salmonella* Pathogenicity Islands 1 (SPI-1) only contained *inv* gene, no highly pathogenic enterotoxin gene, such as Typhoid toxin (*cdt* or *plt*) gene, was found (Table 2).

Table 1 Antimicrobial Susceptibility of *Salmonella* Stanley ST29 (μg/mL)

Antimicrobial Category	Antimicrobial Agent	MIC Breakpoints	MIC
Penicillins	Ampicillin	≥32	>64
Quinolones	Ciprofloxacin	≥1	4
Cephalosporins	Cefotaxime	≥4	16
Macrolides	Azithromycin[a]	≥32	>64
Carbapenems	Imipenem	≥4	0.5
	Meropenem	≥4	≤0.125
	Ertapenem	≥2	≤0.25
Folate pathway inhibitors	Trimethoprim/sulfamethoxazole	≥4/76	>8/152
Chloramphenicol	Chloramphenicol	≥32	>64
Tetracyclines	Tetracycline	≥16	4

Note: [a]Refer to determination criteria for *Salmonella* Typhi.

Table 2 Genomic Analysis of *Salmonella* Stanley ST29

Characteristics	*Salmonella* Stanley ST29
Serotypes prediction	
SISTR	*Salmonella* Stanley
SeqSero2	*Salmonella* Stanley
MLST	ST29
Resistance genes	
β-lactams	bla_{TEM-1B}, bla_{OXA-1}, bla_{DHA-1}
Quinolones	*qnrB4, aac(6')-Ib-cr*
Macrolides	*mph(A), mph(E), msr(E)*
Folate pathway inhibitors	*sul1, sul2, dfrA14*
Chloramphenicol	*floR, catA2, catB3*
Mutation	*parC*(T57S)
Plasmid Replicons	*IncHI2/IncHI2A, IncR, IncN*
MGEs	*ISSpu10, IS6100, ISVsa3*
Virulence gene	
Secretion and transportation genes	*Pho, HilA, Iac, Iag, Inv, Org, Prg, Sic, Sip, Spa, Spr, Ssa, Ssc, Sse, Ssr, Slr, Avr, Sip, SopA, SopB/SigD, Sop, Spt, Pip, Sif, Sse*
Fimbrial adherence determinants	*Agf/Csg, Fim, Lpf, Stb, Stc, Std, Ste, Stf, Sth, Sti*
Nonfimbrial adherence determinants	*MisL, RatB, ShdA, SinH*
Other virulence genes	*EhaB, Mig, Mgt*

Discussion

The World Health Organization (WHO) has estimated that the annual global incidence of *Salmonella* infections was approximately 550 million cases.[14] While the number of cases and deaths caused by typhoid and paratyphoid (14.3 million) exceeded that of NTS (53.5 thousand),[15] the overall disease burden of the latter remained higher due to its significant contribution to infectious diarrhea. Acute cholecystitis is a relatively common condition with a high disease burden, particularly among middle-aged and elderly populations worldwide. It was reported that more than 200,000 people in the United States were diagnosed with acute cholecystitis each year, with direct costs exceeding $1.6 billion annually.[7] Accurate diagnosis of the etiology of acute cholecystitis and understanding the evolutionary characteristics of its pathogenic microorganisms are of great significance for the development of disease intervention policies.

The patient in this case had a history of diabetes and had been suffering from diarrhea for two weeks before the initial consultation. It was speculated that the patient had been infected *Salmonella* Stanley via fecal-oral route, which led to the diarrhea. Due to the lack of further epidemiological investigation, the source of *Salmonella* infection in the patient had not been determined. However, pathogen isolation testing was not performed simultaneously when the patient sought medical attention. The empirical treatment for three weeks failed to eliminate the antimicrobial-resistant pathogen, which persisted in the gut. A similar case was reported in Guangxi province, China.[16] Subsequently, the pathogen migrated retrogradely from the gut, triggering an episode of acute cholecystitis. As one of the etiological factors for acute cholecystitis and other extra-intestinal infections, the persistence of antimicrobial-resistant *Salmonella* in the gut of high-risk populations should be a new focus in clinical practice.[17] Accurate diagnosis of the etiology of diarrhea is an effective measure to prevent potential secondary cholecystitis. Strengthening the pathogen isolation and diagnostic capabilities of clinical laboratories is crucial for identifying and interrupting the translocation of antimicrobial-resistant, persistent pathogens within the gut that may lead to invasive diseases.

The commonly used antimicrobial agents for NTS are ciprofloxacin and third-generation cephalosporins. In recent years, multidrug-resistant NTS has been increasingly reported in clinical, environmental, and food samples.[18–20] The resistance rates to ampicillin, tetracycline, and sulfamethoxazole exceed 40%, while the resistance rates to ciprofloxacin, cefotaxime, and azithromycin were below 10%.[21] The recent research indicated that *Salmonella* Stanley ST29 isolated from pig farms harbored the super-resistant gene $bla_{\text{NDM-5}}$ via the *IncHI2/ST3* plasmid in Guangdong Province, China.[22] In 2024, the WHO updated the Bacterial Priority Pathogens List, categorizing fluoroquinolone-resistant NTS as a high-priority pathogen.[23] This classification reflected the increasing global concern regarding antimicrobial resistance. In this context, the antimicrobial-resistant *Salmonella* Stanley, implicated in secondary invasive cholecystitis cases, represented a convergence of both a pandemic priority pathogen and a high-priority pathogen for resistance control. This dual status significantly increased the complexity of clinical treatment and also imposed a greater disease burden on patients. Despite multidrug-resistant *Salmonella* being susceptible to carbapenems,[24] physicians rarely used these antibiotics for treating diarrheal *Salmonella* infections. Therefore, culture-based antimicrobial strategies are essential to reduce empiric antibiotic use and spread of these pathogens in human populations.[25]

The resistance mechanism of the antimicrobial-resistant *Salmonella* Stanley ST29 in this case was related to resistance genes carried by plasmids and MGEs. Specifically, plasmid replicons, such as *IncHI2/IncHI2A, IncR*, and *IncN*, were capable of harboring a diverse array of resistance genes, including bla_{TEM}, $bla_{\text{OXA-1}}$, $bla_{\text{DHA-1}}$, *mph, msr, sul, dfrA14*, and *qnrB4*. These plasmids have been demonstrated to be highly effective in facilitating the horizontal transfer of resistance genes among different serotypes of Enterobacteriaceae and *Salmonella* (such as *S*.Stanley, *S*.London, *S*. Typhimurium, and *S*.Bovismorbificans).[26] For instance, the "superbug" *Klebsiella pneumoniae* K82 was found to possess a conjugative resistance plasmid, pK82-*mcr*-1 (*IncFIA/IncHI2/IncHI2A/IncN/IncR*), which conferred resistance to 12 antibacterial agents, including colistin.[27] The plasmid characteristics and the types of resistance genes carried by this strain were highly similar to those identified in the isolates of this study. This suggests that the antimicrobial-resistant *Salmonella* Stanley ST29 may have acquired its super antimicrobial-resistant phenotype through horizontal gene transfer. Furthermore, studies have shown that the *Inc* series of plasmids carrying multidrug resistance genes, which evolving into stable clonal structures across different species within the Enterobacteriaceae family, including *Klebsiella pneumoniae*,

Escherichia coli, and *Salmonella*.[28] This highlights the significant role of plasmid-mediated horizontal transfer in the dissemination of multidrug resistance among clinically relevant pathogens.

Previous study has confirmed the relationship between gallbladder cancer and *Salmonella* infections.[29] Specifically, it was demonstrated that *Salmonella* Typhi released the virulence factor *Avr* through the Type III Secretion System (TTSS). This process activated the *Wnt-β-catenin* signaling pathway, Janus kinase (JAK), and signal transducer and activator of transcription (STAT) pathway, ultimately leading to genomic instability in gallbladder cells. This study provided direct evidence that *Salmonella* Typhi played a role in the development of gallbladder cancer.[29] In this case, *Salmonella* Stanley ST29 was found to carry the *Avr* virulence gene, indicating its potential carcinogenic risk. Therefore, enhancing the surveillance of *Salmonella* virulence genes associated with carcinogenicity in patients with acute cholecystitis is of great significance for the early prevention of gallbladder cancer. In this case, *Salmonella* Stanley ST29 was found to lack the Typhoid toxin gene. Previous study has shown that *Salmonella* adapted to the host ecological niche and enhanced its invasiveness through genomic degradation, which included gene deletion and pseudogene formation.[30] In this case, it is hypothesized that *Salmonella* Stanley ST29 degraded highly pathogenic genes to adapt to the ecological pressure of the intestinal microbiota and the host immune response. This adaptation allowed the bacteria to persist in the intestinal tract, consistent with the patient's clinical presentation of mild and chronic diarrhoea.

Conclusion

To our best knowledge, this study is the first case of antimicrobial-resistant *Salmonella* Stanley ST29 diagnosed secondary to acute cholecystitis in Shanghai, China, providing prospective insights and biological evidence for disease progression in potentially high-risk populations. It is recommended to conduct pathogen isolation and antimicrobial susceptibility testing to avoid the persistence of pathogens due to ineffective empiric antibiotic use, which may lead to secondary invasive infections. When diagnosing the causes of acute cholecystitis, clinicians should not neglect rare or uncommon etiologies. Additionally, multisource surveillance system of NTS and researches on the characteristics of strains are necessary to alert the spread of antimicrobial-resistant *Salmonella* worldwide. Precision clinical diagnosis and treatment, as well as precision public health measures, can effectively interrupt and warn against the human-to-human transmission of antimicrobial-resistant *Salmonella* cases.[31]

Data Sharing Statement

The data used to support the findings of this study are included within the article.

Ethics Approval and Consent to Participate

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study and the publication of case details were approved by the Ethics Committee of Zhoupu Hospital Affiliated to Shanghai University of Medicine and Health (No.2024-C-154-E01). Written informed consent was obtained from the patient.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically

reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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