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Urinary Tract Infection Due To *Salmonella Enterica* Subsp. *Enterica*: A Case Report And Literature Review

Zineb Mostafi[1·2], Assia El Rouini[1·2], El Mostafa Benaissa[1·2], Yassine Benlahlou[1·2], Mariama Chadli[1·2]
(Central Bacteriology Laboratory, Mohammed V Military Hospital, Rabat, Morocco)
(Faculty Of Medicine And Pharmacy, Rabat, Morocco)

Abstract:
Background*: Urinary tract infections caused by typhoidal and non-typhoidal Salmonella are rare and typically occur in patients with underlying conditions that should be systematically investigated, such as immunosuppression or congenital and acquired urological abnormalities.*
Materials and Methods*: We report the case of a 67-year-old Moroccan woman with type 2 diabetes complicated by end-stage chronic renal failure. She was admitted to the nephrology department with febrile lower back pain associated with pyuria. A uro- CT scan was performed. Urine, blood, and stool cultures were collected. Bacterial identification was carried out using mass spectrometry, and an antibiogram was performed using the agar disk diffusion method (Kirby-Bauer).*
Results*: The patient was diagnosed with acute pyelonephritis on lithiasic kidneys. Urine cultures revealed the presence of Salmonella enterica subsp. enterica in association with Enterobacter cloacae. 3 sets of blood cultures performed at admission were sterile. A stool culture, carried out as part of the etiological workup on the 5th day of antibiotic therapy, was also negative. The antibiogram guided therapeutic management, and the patient received appropriate treatment with piperacillin-tazobactam (Tazocillin®) 4 g/500 mg.*
Key Word: *Urinary tract infection; Acute pyelonephritis;* Salmonella enterica *subsp.* enterica*; Renal calculi; Diabetes mellitus; Chronic kidney disease*

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I. Introduction

Salmonella are Gram-negative bacilli belonging to the family Enterobacteriaceae and the genus Salmonella. Non typhoidal salmonellosis (NTS) accounts for approximately 90 million infections worldwide each year, with gastroenteritis being the most common clinical presentation. However, it may lead to invasive forms such as bacteremia, meningitis, and other infections of normally sterile sites in at-risk individuals [1]. In 2017, it was estimated that there were 535 thousand cases of invasive NTS infections, with 76 thousand associated deaths worldwide [2]. Typhoidal and paratyphoidal salmonellosis (TS) are responsible for typhoid fever, with an estimated 9.2 million cases and 110 thousand associated deaths annually worldwide, according to WHO [3]. In addition to general symptoms and digestive complications (peritonitis, cholecystitis...) frequently caused by typhoid fever [4], the pathogenesis of TS shares several features and predisposing factors with NTS infections. According to several authors, urinary tract infections (UTIs) due to Salmonella are rare and usually associated with immunosuppression, underlying urinary tract abnormalities, or co-infections with parasites [1]. This work discusses, through our case report and a literature review, the epidemiological, clinical, and therapeutic aspects of this condition.

II. Case Report

Ms A.F., a 67-year-old woman, had a medical history of type 2 diabetes complicated by diabetic nephropathy and end-stage chronic kidney disease. She was admitted to the nephrology department with fever, severe lumbar-abdominal pain, dysuria, pyuria, and vomiting. On physical examination, she appeared in poor general condition: dehydrated, anxious, tachycardic, hypotensive (BP 100/70 mmHg), and febrile (39.7°C). A uro-CT scan revealed nephrolithiasis and chronic nephropathy with bilateral pyelocaliceal dilatation suggestive of acute bilateral pyelonephritis. Laboratory investigations showed pancytopenia with hemoglobin 6.2 g/dl, neutrophils 2400/mm³, and platelets 124,000/mm³. Biochemical tests revealed hyponatremia (126 mEq/l), elevated serum creatinine (72 mg/l), and urea (1.59 g/l). Urine cytobacteriological examination (UCBE) revealed cloudy urine. Microscopy showed significant leukocyturia (10,560 ×10³ elements/ml), hematuria (20 ×10³ elements/ml), and low epithelial cell count (2 ×10³ elements/ml), with no crystals or urinary casts observed.

Culture on CLED (Cystine Lactose Electrolyte Deficient) medium yielded bimorphic growth: transparent colonies (10^5 CFU/ml) and opaque colonies (10^4 CFU/ml) (Figure no 1). The transparent colonies were identified by MALDI-TOF mass spectrometry (VITEK MS Prime) as Salmonella enterica subsp. enterica (SEE), while the opaque colonies were identified as Enterobacter cloacae (ENCL) using the same method. Antibiotic susceptibility testing was performed using the disk diffusion method on Mueller-Hinton agar and interpreted according to the guidelines of the French Society for Microbiology (CASFM) [5]. The antibiotic susceptibility profiles of both strains are summarized in Table 1. The SEE strain was susceptible to beta-lactams but resistant to ciprofloxacin. The ENCL strain was an extended-spectrum beta-lactamase (ESBL) producer, resistant to third-generation cephalosporins and quinolones. 2 additional control UCBE samples were performed, yielding identical results. 3 sets of blood cultures collected at admission remained sterile. A stool culture, performed on day 5 of treatment with piperacillin-tazobactam 36 µg (TAZOCILLINE 4/0.5 g), was negative for Salmonella. The patient died a few days after admission during a dialysis session. Although death was not directly related to the infection, her subsequent clinical course remains unknown.

Figure no 1: Bimorphic growth of SEE and ENCL on CLED medium.

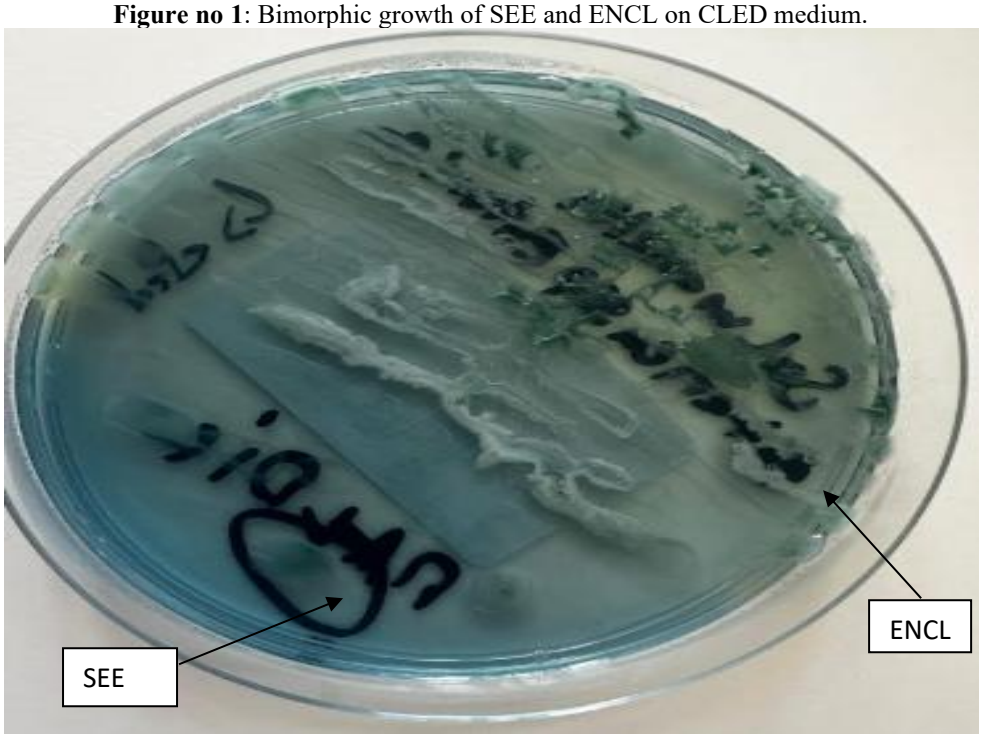

Table no 1: Presents the antibiotic susceptibility profiles of the two bacterial isolates, SEE and ENCL. According to CASFM (2024) guidelines, S indicates susceptible, R indicates resistant, R¹ corresponds to known intrinsic resistance despite apparent in vitro susceptibility, and R² indicates low-level resistance to ciprofloxacin identified by pefloxacin disk testing (inhibition zone diameter < 24 mm) with a ciprofloxacin MIC > 0.6 mg/L.

Table no 1: Antibiotic susceptibility profiles of both isolates

Antibiotics	Disc content (µg)	SEE	ENCL
Ampicillin	10 µg	S	R
Amoxicillin-clavulanic acid	30 µg	S	R
Piperacillin-tazobactam	36 µg	S	S
Mecillinam	10µg	S	S
Ertapenem	10 µg	S	S
Cefoxitin	30 µg	S	R
Cefadroxil	30 µg	S	R
Cefixime	5 µg	S	R
Gentamicin	10 µg	R¹	S
Amikacin	30 µg	R¹	S
Trimethoprim-sulfamethoxazole	25 µg	R	R
Nalidixic acid	30 µg	R	R
Ciprofloxacin¹	5 µg	R²	R
Nitrofurantoin	100 µg	S	S
Fosfomycin	200 µg	S	S

III. Discussion

Salmonella enterica (SE) is one of the two species of the *Salmonella* genus (*enterica* and *bongori*). SE is subdivided into six subspecies. The subspecies *enterica* (SEE) includes 2610 different serotypes [5], of which the most commonly isolated in urine worldwide are Typhimurium, Heidelberg, Enteritidis, Infantis, Newport, and Typhi [7][8]. Infection with SE can cause gastroenteritis, which is the most frequent clinical manifestation, typhoid fever (mainly caused by ST and Paratyphi), septicemic forms, localized infections, and chronic carriage, which raises both epidemiological and therapeutic challenges [9]. UTIs caused by NTS are rare. NTS was first reported as a causative agent of UTI in 1946 by Seligman et al. [10]. A retrospective study conducted in a microbiology laboratory reported that UTIs due to NTS accounted for only 0.07% (19 cases) of all UTIs diagnosed over a 15-year period [11]. Similarly, in another French study conducted over 18 years, 20 urine cultures tested positive for NTS [12]. ST may be isolated from urine during chronic carriage [13], or as a complication of typhoid fever, estimated to occur in about 23% of cases [14], and more rarely following localized UTIs. Sally Weiding and Gordon Scott reported a rare case of localized UTI caused by SEE, sexually acquired in an HIV-positive patient [15]. UTIs caused by SE occur more frequently in elderly individuals over 60 years old and in children under 3 years old [12]. In our case, the UTI was associated with type 2 diabetes, end-stage chronic renal failure, and bilateral renal calculi. Several studies have demonstrated a strong association between these comorbidities and UTIs caused by SEE. UTIs caused by *Salmonella* often reflect underlying functional or structural abnormalities of the urinary tract (renal failure, vesicoureteral reflux, prostatic hypertrophy, urinary stones) [3], [12] and/or conditions compromising cellular immunity (diabetes, HIV infection, malignancies, immunosuppressive therapy) [17,18], which should be systematically investigated. In a Tunisian study, 9 cases of NTS-related UTIs were identified with various predisposing factors, sometimes combined: diabetes (4 cases), corticosteroid and immunosuppressive therapy (3 cases), chronic renal failure (3 cases), vesicoureteral reflux (1 case), malignancy (4 cases), systemic lupus erythematosus (1 case), and prostatic hypertrophy (1 case) [19]. Diabetes constitutes a major risk factor for *Salmonella* UTI due to reduced cytokine secretion, impaired neutrophil function, defective phagocytosis, glycosuria, and local vascular damage leading to weakened immune defenses [20]. Immunocompromised patients also carry a higher risk of systemic dissemination of *Salmonella* with secondary foci. A case of UTI due to ST complicated by pyelonephritis, septicemia, and spondylodiscitis has been reported, where diabetic peripheral neuropathy was identified as a contributing factor [17]. Renal failure may act both as a risk factor and a complication of SE UTI. Dehydration, rhabdomyolysis, or renal abscess may trigger functional renal failure secondary to *Salmonella* UTI [21][22]. Renal stones promote chronic carriage of *Salmonella*, favoring bacterial persistence around and within urinary calculi due to inflammatory lesions [23]. While other bacteria are known to form biofilms on kidney stones, it remains unclear whether *Salmonella* can do so as well. Bacteriuria due to SE cannot be definitively eradicated by antibiotics alone if urinary calculi are not surgically removed [8]. Chronic urinary tract carriage of *Salmonella* may also be favored by co-infection with schistosomes. Several observations have reported this association, which could be explained by the ability of *Salmonella* to colonize the intestinal tract of adult schistosomes and to adhere to the parasite's tegumental surface. Schistosomal co-infection should be suspected in cases of persistent or recurrent *Salmonella* infection despite appropriate antibiotic therapy [24]. According to several authors, no specific clinical features distinguish UTIs caused by *Salmonella enterica* (SE) from those caused by other Enterobacteriaceae. Clinical presentations include cystitis, prostatitis, pyelonephritis (as in our case), asymptomatic carriage, and rare cases of renal abscess [11],[21]. The diagnosis of mixed UTI due to SEE and ENCL was based on suggestive clinical signs, significant bacteriuria (10^5 CFU/ml for SEE and 10^4 CFU/ml for ENCL), and significant leukocyturia (>10^4/ml). To differentiate true UTI from fecal contamination, two additional urine cultures were performed, yielding identical results. Although the patient presented with fever and leukopenia, blood cultures remained sterile, excluding bacteremia-associated UTI. No history of preceding gastroenteritis was reported before the onset of UTI in our case; however, chronic digestive carriage of *Salmonella* cannot be completely ruled out due to inconclusive stool cultures. In contrast to our susceptibility results, a TEM-3 ESBL-producing SEE strain has been previously isolated in Morocco [25]. Another study analyzing multiple SE serotypes showed high resistance rates: 37% to penicillins without beta-lactamase inhibitors (amoxicillin, ticarcillin) and nitrofurans, and 12.5% to quinolones, while resistance to third-generation cephalosporins, aminoglycosides, and sulfamethoxazole-trimethoprim remained low [26]. The resistance profile of Moroccan SE strains appears similar to that observed in Mediterranean countries [27]. The emergence of strains resistant to fluoroquinolones and third-generation cephalosporins is concerning, given that these drugs—due to their excellent tissue penetration, potent bactericidal activity, and urinary excretion in active form—remain first-line treatments for this type of infection [12]. Fluoroquinolone resistance in SEE is attributed to chromosomal mutations affecting DNA gyrase genes, while third-generation cephalosporin resistance results from plasmid-encoded ESBL or class C cephalosporinase (Ambler classification). Excessive use of these antibiotics in both human and veterinary medicine has been proposed as a contributing factor to the emergence of resistant strains [28], [29]. Based on the susceptibility profiles of both isolated strains, antibiotic therapy with piperacillin-tazobactam (Tazocilline® 4/0.5 g) was

initiated. No consensus exists regarding optimal treatment duration. However, several authors recommend treatment lasting 21 to 42 days in pyelonephritis cases, depending on present risk factors [30]. Although antibiotic therapy can sterilize urine, it is often insufficient to prevent recurrence. Surgical intervention was therefore necessary, involving ureterorenoscopy and retrograde intrarenal surgery to manage large and small renal stones [31]. UTIs caused by SE have a high mortality rate, estimated at 22%, likely due to the prevalence of underlying comorbidities in affected patients [12]. In our case, the patient passed away a few days after admission during dialysis. Although death was not directly attributable to the infection, her subsequent clinical course remains unknown.

IV. Conclusion

UTIs caused by *Salmonella* remain rare, despite the high global incidence of salmonellosis. However, they pose a dual challenge: on the one hand, therapeutically, due to increasing antibiotic resistance and the risk of recurrence; and on the other hand, because of the severity of complications and the high mortality they can cause in individuals with predisposing factors.

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