

Submitted: 15/06/2025

Revised: 25/08/2025

Accepted: 09/09/2025

Published: 31/10/2025

## Nonsurgical resolution of renal cortical abscesses in a dog with multidrug-resistant *Klebsiella*-induced urosepsis

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### ABSTRACT

**Background:** Urinary tract infections caused by *Klebsiella* can significantly affect the renal parenchyma, with urosepsis being a potential outcome. Renal abscesses may also occur, and surgical interventions may often be required. This article reports a case of urosepsis and renal abscess caused by multidrug-resistant *Klebsiella*, describing a successful outcome of renal abscess resolution using antimicrobial therapy alone.

**Case Description:** A 3-year-old female Shih-Tzu was referred to the intensive care unit of a teaching hospital in Brazil. The patient exhibited anorexia, vomiting, pale mucous membranes, jaundice, halitosis, hyperthermia, hypoglycemia, and hypotension. Stabilization included the administration of fluid resuscitation, vasopressor, antipyretic, and glucose. Laboratory findings included anemia, neutrophilic leukocytosis, thrombocytopenia, hyperfibrinogenemia, and elevated serum concentrations of creatinine, urea, phosphorus, alanine aminotransferase, alkaline phosphatase, and total and fractionated bilirubin. Abdominal ultrasonography revealed images compatible with chronic kidney disease (CKD) and renal abscesses. Empirical antibiotic therapy was initiated with enrofloxacin (5 mg/kg IV q 24 hours) and ceftriaxone (30 mg/kg IV q 12 hours), followed by a switch to meropenem (8.5 mg/kg SC q 12 hours for 10 days) based on positive urine culture results for *Klebsiella* spp. and susceptibility testing indicating sensitivity exclusively to carbapenems. The patient underwent peritoneal dialysis as renal replacement therapy. The treatment successfully resolved the uremic crisis, induced renal abscess regression, and reversed urosepsis.

**Conclusion:** Conservative clinical management with antibiotic therapy proved effective in resolving urosepsis and abscesses located in the renal cortex, measuring approximately 4–5 cm, without the need for percutaneous drainage or surgical intervention. This finding highlights the need for further research into the efficacy of different therapeutic strategies based on the anatomical location of renal abscesses.

**Keywords:** Meropenem, multidrug-resistant bacteria, renal abscess, sepsis, urinary tract infection.

### Introduction

Multidrug-resistant bacterial infections present a significant challenge to both animal and human health as potential disruptors of the renal parenchyma (Li *et al.*, 2022; Smoglica *et al.*, 2022). *Enterobacteriaceae* is of particular concern globally because of its capacity to harbor plasmid-encoded enzymes that inactivate a broad range of antibiotics (Donati *et al.*, 2014; Pomba *et*

*al.*, 2017). In healthcare settings, reduced antimicrobial efficacy contributes to increased healthcare costs, prolonged hospitalization, and increased morbidity and mortality rates (Simmering *et al.*, 2017; Kennedy *et al.*, 2019).

*Klebsiella* is a genus of gram-negative bacteria commonly found as commensals in the gastrointestinal tract of humans and animals. However, the innate

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ability of these microorganisms to acquire resistance genes, coupled with the inappropriate use of antibiotics, facilitates the spread of resistant strains (Araújo *et al.*, 2023), a concerning factor given the significant role of this genus in nosocomial infections (Tandogdu *et al.*, 2013).

In this context, urinary tract infections (UTIs) caused by *Klebsiella* are particularly notable (Yu *et al.*, 2020; Darwich *et al.*, 2021). Although UTIs are typically resolved with outpatient antibiotic therapy, the increase in AMR has transformed these infections into potential precursors of urosepsis, an emerging cause of septicemia (Flaherty *et al.*, 2014; Motzkus and Luckmann, 2017). Both bacterial colonization of the urinary tract and septic conditions can trigger acute kidney injury or exacerbate preexisting CKD (Prabhu *et al.*, 2021), negatively impacting prognoses.

In this context, renal abscesses are a possible outcome. Management generally involves the use of antimicrobials in combination with surgical interventions, such as unilateral nephrectomy, partial nephrectomy, or surgical and percutaneous drainage (Hess and Ilan, 2003; Kitshoff *et al.*, 2011; Hwang *et al.*, 2020; Petchell *et al.*, 2021; Johnson *et al.*, 2024). However, these methods do not preserve renal integrity and necessitate anesthetic procedures (Johnson *et al.*, 2024). Notably, Dalla Palma *et al.* (1999) and Lee *et al.* (2010) reported the resolution of renal and perirenal abscesses up to 5 cm in diameter using antimicrobial therapy alone in humans. Reports of successful outcomes with this conservative medical approach are scarce in veterinary medicine.

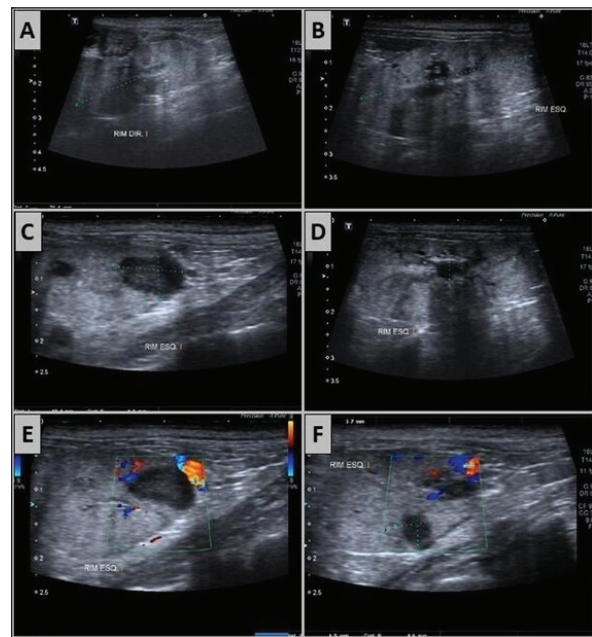
Therefore, given the relevance of *Klebsiella* in urinary tract infections, understanding the clinical manifestations associated with urosepsis caused by this bacterium, as well as the available therapeutic strategies, is essential to guide more accurate diagnostic and treatment decisions, especially in the presence of renal abscesses.

This study aimed to report a clinical case of urosepsis and renal abscesses in a dog infected with multidrug-resistant *Klebsiella*, describing a successful outcome of renal abscess resolution using antimicrobial therapy alone.

### Case Description

A 3-year-old spayed female Shih-Tzu weighing 3.8 kg was presented to the Veterinary Hospital of the UFLA. The owner reported anorexia and vomiting for the past 2 days, along with polyuria and polydipsia. Physical examination revealed pale mucous membranes, jaundice, halitosis, heart rate of 100 beats per minute (bpm), respiratory rate of 55 respirations per minute (rpm), hyperthermia (39.6°C), and hypotension (80 mmHg). SBP was measured using indirect Doppler sphygmomanometry. The SBP value was determined by averaging five consecutive measurements. The Accu-Chek® device was used to detect hypoglycemia (45 mg/

dl). The patient was referred to the intensive care unit, where clinical stabilization was initiated. Biological samples were collected for complete blood count, venous blood gas analysis, and serum biochemistry analyses [alanine aminotransferase (ALT), aspartate aminotransferase, alkaline phosphatase (ALP), gamma-glutamyl transferase, total and fractionated bilirubin, total protein, albumin, globulin, creatinine, urea, phosphorus, chloride, potassium, sodium, and calcium]. Urinalysis, urine culture, blood culture, and antibiotic susceptibility testing were additional diagnostic methods. Urine was obtained via cystocentesis. Abdominal ultrasonography was performed after initial stabilization. Hyperechoic renal cortex was noted, accompanied by a loss of corticomedullary differentiation and definition in both kidneys. Hypoechoic nodular structures lacking vascularization on Doppler mapping were observed in the left kidney, presumed to be renal abscesses (Fig. 1). The complete blood count revealed anemia, neutrophilic leukocytosis, lymphopenia, thrombocytopenia, and hyperfibrinogenemia (Table 1). Blood gas analysis revealed no abnormalities. Serum biochemistry revealed elevated levels of creatinine, urea, phosphorus,



**Fig. 1.** Ultrasound images of the right and left kidneys on the day of the patient's admission to the intensive care unit (ICU). (A) Right kidney showing a hyperechoic cortex with loss of corticomedullary distinction and definition. (B, C, D, E and F) Left kidney also displayed a hyperechoic cortex with loss of corticomedullary distinction and definition. Four hypoechoic, nonvascularized nodular structures, the largest measuring 1.04 cm × 0.65 cm, were identified in the cortical region via Doppler mapping. These findings are consistent with the findings of renal abscesses.

**Table 1.** Hematological values of a canine patient during the *Klebsiella* infection period.

| Parameter                                     | Reference value* | Blood Transfusion ☼ |         |         | Peritoneal Dialysis ☼ |         | Meropenem ✎ |        |         |
|-----------------------------------------------|------------------|---------------------|---------|---------|-----------------------|---------|-------------|--------|---------|
|                                               |                  | D1                  | D2☼     | D3☼     | D4☼✎                  | D5☼☼✎   | D6✎         | D10✎   | D14     |
| Red blood cells ( $\times 10^6/\text{mm}^3$ ) | 5.5–8.5          | 3.79                | 2.15    | 4.22    | 2.6                   | 1.70    | 4.46        | 4.78   | 2.59    |
| Hemoglobin (g/dl)                             | 12–18            | 8.67                | 4.63    | 9.6     | 5.90                  | 3.70    | 9.33        | 10.06  | 5.33    |
| Hematocrit (%)                                | 37–55            | 26                  | 16      | 29      | 19                    | 11      | 28          | 30     | 17      |
| M.C. V                                        | 60–77            | 68.60               | 65.12   | 68.72   | 73.08                 | 64.71   | 62.78       | 62.78  | 65.67   |
| M.C.H                                         | 19–24            | 10.6                | 11.4    | 11.7    | 12.40                 | 11.60   | 11          | 11.90  | 13.80   |
| M.C.H.C                                       | 30–36            | 33.33               | 33.08   | 33.10   | 31.05                 | 33.64   | 33.33       | 33.53  | 31.37   |
| Leukocytes (/mm <sup>3</sup> )                | 6,000–17,000     | 28,700              | 43,600  | 33,800  | 50,800                | 49,800  | 49,300      | 36,600 | 16,625  |
| Segmented neutrophils (/mm <sup>3</sup> )     | 3,000–11,500     | 21,238              | 33,572  | 29,406  | 45,212                | 45,816  | 43,877      | 33,672 | 14,170  |
| Band neutrophils (/mm <sup>3</sup> )          | 0–300            | 0                   | 0       | 676     | 1016                  | 0       | 0           | 0      | 0       |
| Total lymphocytes (/mm <sup>3</sup> )         | 1,000–4,800      | 3,157               | 8,224   | 1,352   | 2,032                 | 820     | 1,479       | 732    | 1,200   |
| Eosinophils (/mm <sup>3</sup> )               | 100–1,250        | 0                   | 0       | 0       | 0                     | 205     | 0           | 732    | 205     |
| Basophils (/mm <sup>3</sup> )                 | 0–100            | 0                   | 0       | 0       | 0                     | 0       | 0           | 0      | 0       |
| Monocytes (/mm <sup>3</sup> )                 | 150–1,350        | 4,305               | 1,744   | 2,366   | 2,540                 | 3,984   | 3,944       | 1,464  | 1050    |
| Platelets (/mm <sup>3</sup> )                 | 160–430          | 320,400             | 280,800 | 205,200 | 142,200               | 316,800 | 198,000     | 72,000 | 252,000 |
| Fibrinogen (mg/dL)                            | <300             | 400                 | 500     | 500     | 500                   | 100     | 600         | 400    | 500     |

The table presents the fluctuations in hematological parameters according to the days of hospitalization (D) and the types of interventions performed (antibiotic therapy, blood transfusion, and peritoneal dialysis) over the 14-day hospitalization period (D1–D14). ☼ Indicates a day with a blood transfusion. ☼ Indicates the day that peritoneal dialysis was conducted. ✎ Indicates the administration of meropenem.

\*Kaneko, Harvey, and Bruss (1997); Meyer and Harvey (2004); Stockham and Scott (2008); Weiss and Wardrop (2010).

ALT, ALP, total bilirubin, direct bilirubin, and indirect bilirubin (Table 2). Urinalysis revealed proteinuria, a specific gravity of 1.012, and leukocyte and epithelial casts. For chemical parameters, urine urinalysis was performed using a test strip, while urine-specific gravity was measured by spectrophotometry.

For initial stabilization, the patient was managed hemodynamically with two fluid challenges of 10 ml/kg lactated Ringer's solution infused over 10 minutes each. These boluses fail to achieve an adequate pressor response. Subsequently, norepinephrine infusion (0.3 mcg/kg/minute) was initiated, which successfully normalized the systolic arterial pressure (92 mmHg). After 2 hours, the vasopressor infusion was gradually tapered and discontinued. Hypoglycemia (45 mg/dl) was corrected with a bolus of 1 ml/kg of 50% dextrose, followed by continuous infusion of 5% glucose solution.

The patient was diagnosed with urosepsis based on clinical and laboratory findings. Empirical antibiotic

therapy included enrofloxacin (5 mg/kg IV q 24 hours) and ceftriaxone (30 mg/kg IV q 12 hours). Supportive treatments for hepatopathy include S-adenosylmethionine (20 mg/kg PO q 24 hours on an empty stomach) and silymarin (8 mg/kg PO q 8 hours). Antimicrobial de-escalation was performed following the urine culture results, which identified *Klebsiella* spp. as the causative agent. Susceptibility testing revealed a multidrug resistance profile, with sensitivity to only imipenem or meropenem (Table 3). Consequently, meropenem was administered at 8.5 mg/kg SC q 12 hours for 10 days. Antibiotic therapy was conducted in accordance with the guidelines of the International Society for Companion Animal Infectious Diseases (Weese *et al.*, 2019). No bacterial growth was observed in the blood cultures.

For the initial management of the uremic condition, a conservative clinical approach was adopted, which included aluminum hydroxide (30 mg/kg PO q 8 hours post-meal), cyproheptadine (Apevitin® 0.2 mg/kg PO

Table 2. Serum biochemistry values of a canine patient during the *Klebsiella* infection period.

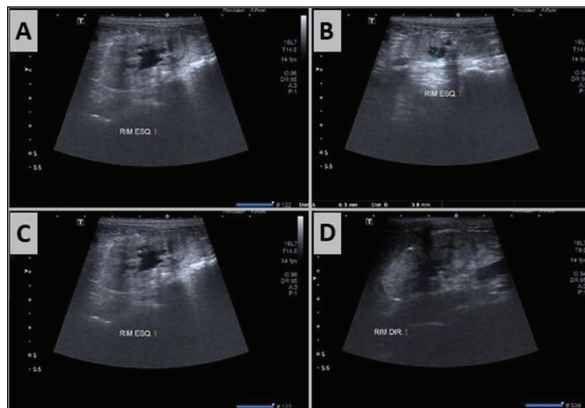
| Parameter                        | Reference value* | Blood transfusion ⊕ Peritoneal dialysis 🗑 Meropenem 🚑 |        |       |        |       |        |       |        |       |        |       |        |  |  |
|----------------------------------|------------------|-------------------------------------------------------|--------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|--|--|
|                                  |                  | D1                                                    | D2⊕    | D3    |        | D4🗑   |        | D5🗑   |        | D6🗑   |        | D10🗑  | D14    |  |  |
|                                  |                  | Pre 🗑                                                 | Post 🗑 | Pre 🗑 | Post 🗑 | Pre 🗑 | Post 🗑 | Pre 🗑 | Post 🗑 | Pre 🗑 | Post 🗑 | Pre 🗑 | Post 🗑 |  |  |
| Creatinine (mg/dl)               | 0.5–1.5          | 8.5                                                   | 5      | 5.5   | 4.07   | 3.6   | 2.7    | 2.3   | 2      | 3.1   | 1.9    | 2.3   |        |  |  |
| Urea (mg/dl)                     | 21.4–59.92       | 390                                                   | 386    | 460   | 368    | 387   | 273    | 299   | 243    | 198   | 160    | 233   |        |  |  |
| Phosphorus (mg/dl)               | 2.2–5.5          | 14                                                    | 10     | 11.7  | 8.9    | 9     | 6.9    | 7     | 6.1    | 7.4   | 7.0    | 7.4   |        |  |  |
| Potassium (mEq/l)                | 4.37–5.65        | 5.3                                                   | 5      | 3.8   | 3.9    | 4.4   | 3.4    | 3.9   | 3.5    | 3.7   | -      | -     |        |  |  |
| Total calcium level (mg/dl)      | 8.6–11.2         | 10.2                                                  | 10     | 9.3   | 9.2    | 8.6   | 7.9    | 8.9   | 9.4    | 8.1   | -      | -     |        |  |  |
| Ionized calcium (mg/dl) level    | 5.2–6            | 7.1                                                   | 6.9    | 5.61  | 5.61   | 5.47  | 5.32   | 5.47  | 5.37   | 5.39  | -      | -     |        |  |  |
| Sodium (mEq/l)                   | 141.1–152.3      | 150                                                   | 148    | 144   | 142    | 142   | 143    | 143   | 147    | 146   | -      | -     |        |  |  |
| Chloride (mmol/l)                | 109–122          | 118                                                   | 117    | 109   | 108    | 109   | 107    | 109   | 103    | 104   | -      | -     |        |  |  |
| Albumin (g/dl)                   | 2.6–3.3          | 2.6                                                   | 2.6    | 2.7   | 2.6    | 2.4   | 2.5    | 2.6   | 3.1    | 2.8   | -      | -     |        |  |  |
| Globulin (g/dl)                  | 2.7–4.4          | 3.9                                                   | 3.5    | 2.7   | 2.8    | 2     | 3.8    | 2.4   | 2.9    | 2.9   | -      | -     |        |  |  |
| Total protein (g/dl)             | 5.4–7.1          | 6.5                                                   | 6.1    | 5.4   | 5.4    | 4.4   | 6.3    | 5     | 6      | 6.7   | -      | -     |        |  |  |
| ALT (U/l)                        | 21–73            | 264                                                   | -      | 260   | 283    | 344   | 344    | 344   | 344    | -     | 269    | 1,100 |        |  |  |
| ALP (U/l)                        | 20–156           | 1,007                                                 | -      | 4,221 | 4,207  | 3,700 | 3,700  | 3,700 | 3,700  | -     | 1,116  | 7,435 |        |  |  |
| Direct bilirubin (mg/dl)         | 0.06–0.12        | 0.81                                                  | -      | 0.81  | 0.74   | 0.75  | 0.75   | 0.75  | 0.75   | -     | 0.08   | 0.39  |        |  |  |
| Indirect bilirubin level (mg/dl) | 0.01–0.49        | 1.22                                                  | -      | 1.22  | 1.37   | 1.37  | 1.37   | 1.37  | 1.37   | -     | 0.56   | 0.51  |        |  |  |
| Total bilirubin level (mg/dl)    | 0.1–0.3          | 2.03                                                  | -      | 2.03  | 2.11   | 2.12  | 2.12   | 2.12  | 2.12   | -     | 0.64   | 0.9   |        |  |  |

Fluctuations in serum biochemistry parameters and hemogram during hospitalization were observed. Changes in biochemical parameters and hematological profiles across days of hospitalization (D1 to D14) are presented in this table. ⊕ Indicates a day with a blood transfusion. 🗑 Indicates a day with peritoneal dialysis. 🚑 Indicates a day of meropenem administration. \*Kaneko, Harvey, and Bruss (1997); Meyer and Harvey (2004); Stockham and Scott (2008).



**Table 3.** Antibigram results of a canine patient with *Klebsiella* infection.

| Susceptible | Resistant                           |
|-------------|-------------------------------------|
| Meropenem   | Amikacin                            |
|             | Ampicillin                          |
|             | Amoxicillin + potassium clavulanate |
| Imipenem    | Sodium Ceftriaxone                  |
|             | Cefazolin                           |
|             | Enrofloxacin                        |
|             | Marbofloxacin                       |
|             | Gentamicin                          |
|             | Clindamycin                         |
|             | Ciprofloxacin                       |
|             | Aztreonam                           |
|             | Metronidazole                       |
|             | Sulfamethoxazole/trimethoprim       |



**Fig. 2.** Ultrasound images of the right and left kidneys following treatment with meropenem (8.5 mg/kg SC twice daily for 10 days) in a canine patient with *Klebsiella* infection. (A–D) Both kidneys show a loss of corticomedullary distinction and definition. (A–C) In the left kidney, there was noticeable remission of the hypoechoic nodular structures, which were previously identified as consistent with renal abscesses.

q 12 hours), sucralfate (Sucrafilm® 5 ml PO q 8 hours), maropitant citrate (Cerenia® 1 mg/kg SC q 24 hours), ondansetron (Emedron® 1 mg/kg IV q 8 hours), B vitamins (Hyplex® 1 ml IV q 12 hours), telmisartan (Semintra® 1 mg/kg PO q 24 hours), recombinant human erythropoietin (Hemax Eritron® 100 IU/kg SC three times weekly), iron (Ferropurum® 100 mg IM every 21 days), and dexamethasone (Dexaflan®

0.5 mg/kg SC q 24 hours for 3 days) to control systemic inflammation indicated by urosepsis and the previously described empirical antimicrobial therapy. Subsequently, peritoneal dialysis was employed as renal replacement therapy based on the criteria set forth by the International Renal Interest Society (Cowgill, 2016). An esophageal feeding tube was also inserted to provide nutritional support (metabolizable energy requirement =  $95-130 \text{ kcal} \times \text{BW}^{0.75}$ ) using Recovery (Royal Canin®). Two blood transfusions were required because of dyspnea associated with low hematocrit, hemoglobin, and red blood cell counts (Table 1). The blood volumes administered on days D2 and D5 were 55.9 and 85.5 ml, respectively, resulting in clinical improvement of dyspnea after the transfusions.

With the administration of meropenem according to the antibiotic susceptibility profile, normalization of the leukogram was observed (Table 1). Renal abscesses, visualized via abdominal ultrasonography (Fig. 2), regressed, leading to urosepsis control. The uremic crisis was resolved. On the 14th day of hospitalization, the patient was discharged for continued treatment at home for the hepatopathy that developed during the infection. The clinical management of CKD was maintained.

## Discussion

This case report describes the development of urosepsis and renal abscesses caused by multidrug-resistant *Klebsiella* infection in a dog and highlights the importance of urine culture and antimicrobial susceptibility testing in guiding the selection of the most appropriate antibiotic for effective infection control. The therapeutic approach used for purulent renal collections demonstrates that antibiotic therapy alone, rather than percutaneous drainage and/or surgical methods, can be effective in resolving renal cortical abscesses measuring approximately 4–5 cm in dogs. This approach avoided the use of invasive therapeutic methods that could contribute to further renal parenchymal injury. Moreover, the regression of the lesions, confirmed by ultrasonographic follow-up, underscores the essential role of imaging in monitoring the response to conservative therapy without percutaneous drainage and in excluding differential diagnoses such as neoplasia.

*Klebsiella* is a ubiquitous pathogen found in the environment, intestinal microbiota, skin, and mouth of both animals and humans. Some of its species, such as *Klebsiella pneumoniae*, are frequently associated with nosocomial infections (Yang et al., 2019). Ribeiro et al. (2022) reported that over 50% of the dogs and cats examined in their study presented with *Klebsiella*-associated cystitis, pyelonephritis, and urethritis, highlighting the high pathogenic potential of this bacterium in the urinary tract. Our study reinforces this evidence, demonstrating the potential for renal abscess formation in dogs infected with *Klebsiella*.

Similarly, Chang *et al.* (2000) described renal or perirenal abscesses, bacteremia, and metastatic septic infections caused by *K. pneumoniae* in humans in Asia, and presented a clinical diagnosis and progression similar to the urosepsis and renal abscesses observed in animals in this report. This highlights the concern of emerging infections caused by resistant uropathogens in the One Health context (Araújo *et al.*, 2023).

The antibiogram identified the antibiotic to which the bacterium colonizing the patient's urinary tract was susceptible. *Klebsiella* showed no susceptibility to beta-lactams, aminoglycosides, or fluoroquinolones. The resistance pattern observed is similar to that described in *Klebsiella* isolates from Thailand (Amphaiphan *et al.*, 2021), Spain (Darwich *et al.*, 2021), the United States (Yudhanto *et al.*, 2022), and Italy (Smoglica *et al.*, 2022). The similarity of antimicrobial resistance patterns of *Klebsiella* in different countries underscores the importance of documenting complications, such as urosepsis and renal abscesses, as well as the therapeutic strategy used in this case report. The resistance pattern identified in different studies suggested that the multiresistance of this bacterium is not limited to this case, which may contribute to the recurrence of clinical scenarios similar to the one reported.

The hypoechoic structures identified in the left kidney did not require percutaneous drainage, suggesting the possibility of renal abscess remission with pharmacological intervention alone (Dalla Palma *et al.*, 1999; Lee *et al.*, 2010). Previous reports failed to successfully treat dogs using antibiotic therapy alone (Cola *et al.*, 2020; Rohini *et al.*, 2021; Véran *et al.*, 2021). The decision to opt for pharmacological treatment over percutaneous drainage or surgical methods was based on previous studies by Dalla Palma *et al.* (1999) and Lee *et al.* (2010), which reported involution of renal and perirenal abscesses up to 5 cm in diameter with antimicrobial therapy alone in humans. In addition, the patient already exhibited renal parenchymal function instability, which contributed to the selection of therapeutic alternatives that would not worsen the existing renal injury. The remission of these structures on ultrasonography after antibiotic treatment supports the hypothesis that these purulent collections were present in the renal parenchyma, ruling out neoplasms as a differential diagnosis.

The authors believe that differences in antibiotic perfusion across distinct renal regions may explain the varying therapeutic outcomes reported in the literature. Cola *et al.* (2020); Rohini *et al.* (2021) and Véran *et al.* (2021) were unsuccessful in treating subcapsular abscesses using antimicrobial therapy alone. In contrast, Johnson *et al.* (2024) reported the resolution of a corticomedullary abscess using enrofloxacin-based antibiotic therapy combined with percutaneous drainage. Contributing to this body of evidence, the present article describes a successful case of cortical abscess resolution achieved solely through antibiotic

therapy, highlighting the relevance of this approach and encouraging further investigation into the efficacy of therapeutic interventions based on the anatomical location of renal abscesses.

Finally, initial hemodynamic management in the intensive care unit was crucial for reversing the shock observed in the animal. When hypotension is unresponsive to fluid therapy, volume replacement followed by vasopressor administration, in accordance with the Surviving Sepsis Campaign Guidelines (Evans *et al.*, 2021), was crucial for stabilizing the patient. This hypotensive state, concomitant with hypoglycemia, hyperthermia, neutrophilic leukocytosis, and the presence of a UTI, confirmed the sepsis presented by the patient (Evans *et al.*, 2021), and the need for a vasopressor established the diagnosis of septic shock (Evans *et al.*, 2021). These hemodynamic alterations are explained by an unregulated inflammatory response to infections, leading to organ dysfunction (Vincent and De Backer, 2013), which was due to a UTI caused by *Klebsiella* in this clinical case.

The multidrug resistance exhibited by *Klebsiella* represents a significant public health concern. The use of carbapenem-class antibiotics should be avoided and reserved for cases in which there is no susceptibility to other antimicrobials. In the presence of MDR bacteria, strict hospital disinfection measures are essential. The molecular identification of the pathogen, along with comparison with isolates from potential environmental or clinical sources, is desirable to interrupt the transmission chain. Methods, such as polymerase chain reaction (Remya *et al.*, 2019) and next-generation sequencing (Wang *et al.*, 2022), can be employed for this purpose. Adopting these measures helps mitigate the spread of bacteria and their resistance genes, prevent nosocomial outbreaks, reduce healthcare costs, and improve patient prognosis (Altayb *et al.*, 2023; Djordjevic *et al.*, 2024). However, despite performing hospital disinfection, financial constraints prevented the application of these molecular techniques in this case report. The limitation of this case report is the absence of molecular techniques to detect the pathogen species involved in this infection.

This case report describes the development of urosepsis and renal abscesses caused by multidrug-resistant *Klebsiella*, emphasizing the importance of urine culture and antimicrobial susceptibility testing in guiding targeted antibiotic selection for effective infection control. Conservative clinical management using antibiotic therapy instead of percutaneous drainage proved effective in resolving abscesses located in the renal cortex measuring approximately 4–5 cm. The regression of the lesions, confirmed by ultrasonographic follow-up, highlights the relevance of imaging in both monitoring therapeutic response and excluding differential diagnoses such as neoplasia. This finding highlights the need for further research into the

efficacy of different therapeutic strategies based on the anatomical location of renal abscesses.

#### Conflict of interest

The authors have no relevant financial or non-financial interests to disclose.

#### Funding

The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

#### Authors' contributions

DR, RSM, and TALN were responsible for case management. LSM, MLMM, and JAMP contributed to laboratory analyses and case management. AEJB performed the imaging examinations and contributed to the case management. JCNCJ, MSOB, and LGDB assisted in the case management in the intensive care unit. The RBN and PTCGO supervised the clinical management. DR wrote the first draft of the manuscript, with contributions and revisions from all authors. DR, MSOB, and LGDB translated the article. All authors have read and approved the final version of the manuscript.

#### Data availability

All data supporting this study's findings are available within the manuscript.

#### Ethical approval

This case report is part of a study that has been approved by the Committee for Ethics in Animal Use (CEUA) of the Federal University of Lavras (UFLA), under protocol number 4667090823 – 2023.

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