Case Report

Alcalegenes Faecalis-Induced Emphysematous Cystitis, Pyelonephritis, and Bacteraemia in a Diabetic Patient. Is there Anything Else?

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Abstract
Alcalegenes faecalis is a gram-negative bacterium that typically causes opportunistic infections in humans. The growing resistance of A. faecalis to different antibiotics makes it difficult to treat in most cases. We present a case of 71-year-old male patient with type II diabetes mellitus who diagnosed with emphysematous cystitis, pyelonephritis and bacteraemia caused by Alcaligenes faecalis. There were not any optimal antibiotics available to initiate based on sensitivities. Intravenous vancomycin and ceftolozane-tazobactam were two of the antibiotics that were used to treat the patient successfully. Once it is found in cultures, it should be treated as a real pathogen rather than just a contaminant. Alcaligenes faecalis infections have become highly resistant to many regularly used antibiotics. A recommended course of antibiotic treatment for A. faecalis infection has not been sufficiently defined in the literature. It is advised to start empirical antimicrobial treatment for the most prevalent bacteria.

Keywords: Alcaligenes faecalis infection, multi drug-resistant, bacteraemia, emphysematous cystitis, pyelonephritis.

Introduction

Alcaligenes faecalis is an obligatory aerobic, nonfermentive, oxidase-positive, Gram-negative bacterium that is a member of the Alcaligenaceae family (Huang, 2020) (Hasan, 2019). It is primarily found in soil, water, and hospital settings. Specifically, it has been isolated from a range of clinical specimens, including blood, urine, stool, wound discharge, cerebrospinal fluid, and respiratory secretions. (Samia, 2024) Notably, it is present in the human gut microbiota and can spread to the respiratory and circulatory systems, where it can lead to infection (Hasan, 2019). Limited studies indicate that Alcaligenes faecalis is typically spread by droplets through nebulizers and ventilation apparatus. Direct contact is another way that it can spread (Rana Al-Zakhari, 2020). Several illnesses, including bacteraemia, meningitis, endophthalmitis, endocarditis, skin and soft tissue infections, urinary tract infections, peritonitis, otitis media, and pneumonia, have been linked to A. faecalis infection (Zumrut Sahbudak Bal, 2023). Alcalegenes faecalis cases have been noted in both the community and healthcare environments. (Hasan, 2019). While opportunistic infections are the main outcome, it can also lead to life-threatening medical conditions especially in immunocompromised patients (Rana Al-Zakhari, 2020, Aisenberg, 2004). Current

research indicates that this pathogen is becoming resistant to several broad-spectrum antibiotics, such as quinolones, trimethoprim/sulfamethoxazole, penicillin, cephalosporins, nitrofurantoin, carbapenems, and colistin (Samia, 2024). We report a rare case of emphysematous cystitis, pyelonephritis and bacteraemia caused by multidrug resistance Alcalegenes faecalis.

Case Report

A 71-year-old male patient arrived at our emergency room with weakness and a decreased degree of consciousness that began four hours ago. His medical history includes benign prostate hyperplasia, idiopathic tremor of the upper limbs, obesity (body mass index, kg/m2), diabetes mellitus type II since 2019, on oral hypoglycaemic drugs, and bedridden status for the past two months due to referral depression. He never underwent a colonoscopy. There was no reference to previous hospitalisations. The patient was a retired mathematician.

When the patient first arrived at the emergency room, he was hemodynamically stable but febrile (39.5 ºC) and tachycardic (110 beats per minute). Moreover, his SpO2 on room air was 87%. The first blood gas readings were 33 mm Hg for partial pressure of carbon dioxide, 49 mm Hg for partial pressure of oxygen, and 7.41 mm Hg for pH. His oxygenation gradually improved when he was put on an oxygen delivery system (venturi mask, 35%) right away. His systolic blood pressure dropped to 75 mmHg, indicating hemodynamic instability, thus intravenous resuscitation therapy was initiated using crystalloid fluids. Since, hemodynamics remained unchanged, levophed was started to maintain his blood pressure and stabilize his hemodynamics. The patient's condition began to improve with the combination of oxygen therapy and fluid resuscitation. A Foley catheter was inserted in the emergency, and 500 millilitres of cloudy urine were immediately drained. Urosepsis was the initial diagnosis, and 1.5g of ceftolozane-tazobactam was given. The clinical examination revealed reduced level of consciousness and no signs of meningism. The abdominal examination revealed tenderness and pain in the suprapubic region. Of note, a second-degree burn was noted on the patient's genitalia, inner thighs, and left gluteal region, caused by hot water.

The results of the blood tests showed that the white blood cell count was 9,000/µl (4,000–10,000), the C-reactive protein level was 18.10 mg/dl, the platelet count was 230.000x10^3/µL, the procalcitonin level was 30.28 ng/mL, the serum creatinine level was 2.78 mg/dL, the urea level was 96 mg/dL, haemoglobin 12.30 g/dL and the haematocrit level was 38.20% Furthermore, hyponatremia was also found (128mmol/L), whereas the results of the rest of blood tests were unremarkable. The HbA1c levels were 6.2%. A urine analysis was sent, and the results suggested a UTI. A urine test for Legionella antigen came out negative. Unfortunately, a lack of laboratory facilities prevented the testing of the urinary streptococcus pneumoniae antigen.

Originally, a brain CT scan revealed no abnormalities. Pneumonic infiltration was not visible on the chest X-ray, and the costodiaphragmatic angles were hazy. The abdominal X-ray showed nothing unusual. However, upon further examination, a chest CT scan was conducted, which revealed bilateral pleural effusions and consolidation in the lower lobes of both lungs. (Figure 1) Additionally, mediastinal lymphadenopathy was observed. The cardiology team was consulted, and a cardiac ultrasound indicated dilatation of the left atrium and a left ventricular ejection fraction (LVEF) of 35%. On a US of the kidneys, there was no sign of hydronephrosis, nephrolithiasis, or pyelonephritis; instead, there were bilateral cystic renal lesions. There was evidence of fatty liver on the upper abdominal US.

On day one, broad spectrum intravenous antibiotics, specifically vancomycin and ceftolozane-tazobactam, were started as empirical therapy. To prevent thrombosis, anticoagulants were also started. It was proposed that the burn wound in the genital area could be the cause of urosepsis and bacteraemia. Thus, a swab culture of the burned wound was obtained to identify any agent that might be causing the infection. The wound culture was positive for Klebsiella pneumoniae, Candida albicans, and Acinetobacter baumannii complex. Within 24 hours of his hospital admission, blood

cultures were obtained, and they revealed the presence of multidrug-resistant Alcaligenes faecalis and Staph lugdunensis bacteraemia. The culture of urine revealed the presence of Alcaligenes faecalis. The antibiotic sensitivity tests revealed multidrug resistance. Based on the antibiogram of blood cultures, antimicrobial therapy was modified, with vancomycin (IV) being continued and ceftolozane-tazobactam stopped. The patient's clinical response was closely monitored to ensure the effectiveness of the new antibiotic regimen. Levophed was withdrawn 3 days later. A CT scan of the abdomen was performed to rule out any known complications of an A. faecalis infection as well as other possible causes of sepsis. The results revealed findings of emphysematous cystitis (Figure 2) and pyelonephritis (Figure 3) accompanied by perirenal fat inflammation.

Regular cultures were also obtained to track any changes in susceptibility patterns. On day 19 of hospital stay, blood cultures showed complete eradication of Alcaligenes faecalis, Staphylococcus lugdunensis, and Staphylococcus aureus, as well as no other bacterial growth. The patient's clinical symptoms improved significantly following the successful treatment of the identified bacterial infections. His laboratory results demonstrated a consistent improvement in renal function, inflammatory markers, and leucocytosis. Continued monitoring for any signs of a recurrent infection was done.

He also experienced an episode of acute abdominal pain, primarily in the hypogastrium followed by multiple episodes of watery diarrhoea. There were no detectable signs of peritonitism. Upon suspicion of CDI, a stool sample was tested. Both the stool culture and the parasitic testing were negative. Conversely, testing for toxin A and B for the detection of CDI came back positive for toxin A. The administration of oral vancomycin at a dose of 125 mg every 6 hours was initiated. Following a 10-day course of vancomycin treatment, the patient's symptoms subsided, and his stools returned to normal.

Septic shock episodes were observed to be recurring in this patient despite antimicrobial therapy. Leucocytosis, thrombopenia, and progressive renal failure were discovered through repeated blood testing. On days 19 and 26 of hospitalisation, Acinetobacter baumannii was cultured in blood. Antimicrobial susceptibility testing was performed on it and found to be exclusively polymyxin E-susceptible. Meropenem and polymyxin E were started in place of intravenous vancomycin. A repeated abdominal CT scan showed improvement of the findings of emphysematous cystitis. (Figure 4)

On day 32, he rapidly deteriorated, and incubated due to reduced level of consciousness and respiratory failure type II. Four days after his incubation, the patient was discharged from the intensive care unit (ICU) back to the ward in a stable condition.

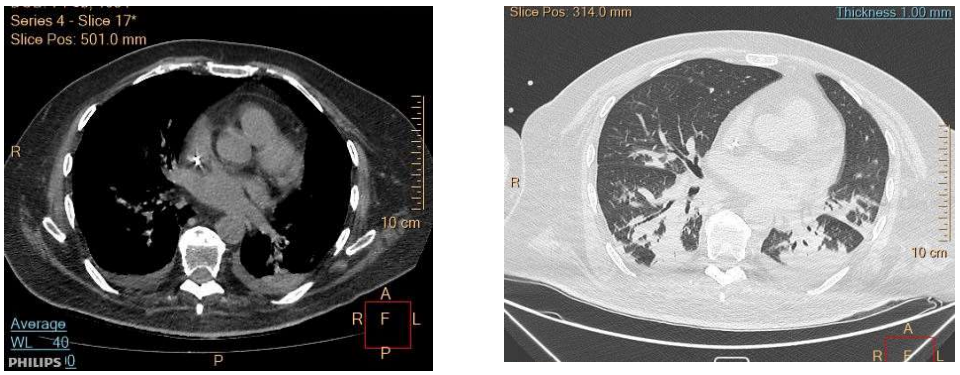

Figure 1. CT scan, axial view of mediastinal and lung parenchyma window demonstrating pleura effusions and consolidations in both lower lobes.

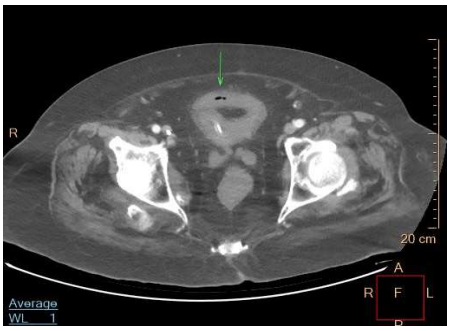

Figure 2. Small air densities are seen in the urinary bladder wall inferring with emphysematous cystitis. There is also pericystic fat strading.

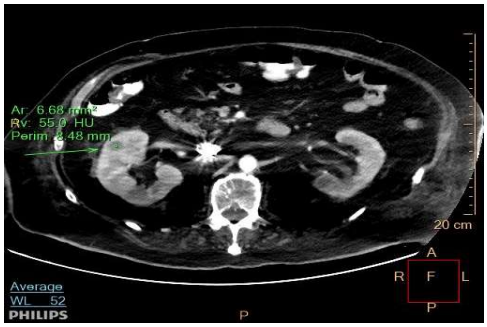

Figure 3. The abdominal CECT scan revealed several hypodense focal lesions in both kidneys. These findings are consistent with small abscesses – pyelonephritis. There are also hazy areas in the perinephric space consistent with perirenal fat inflammation.

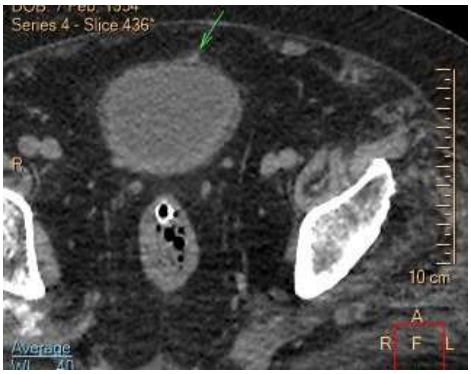

Figure 4. CT scan in axial view showing a decrease of the air densities in the urinary bladder wall and pericystic fat stranding.

Discussion

Alcaligenes faecalis is a gram negative, oxidase positive rod bacterium with peritrichous flagella. It is found in soil, water, and the human digestive system as a harmless saprophyte. Approximately, 5-19% of the human population is colonised by this bacterium. It is quite rare for this bacterium to cause systemic illness (Kahveci, 2011). The literature states that there have been 130 isolated reports of A. faecalis infections, with most prevalent A. faecalis infection sites being the bloodstream, urinary tract, skin, and soft tissue (of which 56% are diabetic foot

ulcers), and middle ear, in order of frequency (Huang, 2020). According to studies, the most common risk factors for A. faecalis-caused cystitis were diabetes mellitus, hypertension, dementia, cerebrovascular accidents, chronic renal disease, catheter-associated infections, and intravenous antibiotic exposure during the previous 90 days. Most patients were males, with a mean age of 76.9 years (Huang, 2020). Furthermore, obstructive uropathy and antibiotic use within the previous 90 days were shown to be prevalent risk factors in cases of A. faecalis-induced acute pyelonephritis (Huang, 2020). In our case, these two risk factors were excluded (Huang, 2020). Bloodstream infections attributed to A. faecalis were initially documented in immunocompromised cancer patients, date back to 2004. Resistance to aminoglycosides, levofloxacin, ciprofloxacin, and monobactam had been identified by antibiotic susceptibility testing. (Hasan, 2019) Evidence of coinfections with other bacteria has been referred in literature, with Enterococcus species, Proteus vulgaris, and Pseudomonas aeruginosa being the most prevalent pathogens. Bloodstream coinfections with Enterococcus faecalis and Morganella morganii have been observed in patients diagnosed with A. faecalis bacteraemia (Huang, 2020). Interestingly, coinfection with distinct Staphylococcus species was present in our patient, specifically Staphylococcus aureus and Staphylococcus lugdunensis. Remarkably, in our case, the multidrug-resistant A. faecalis was completely eradicated from the bloodstream and urine of the patient following intravenous antibiotic treatment with vancomycin and ceftolozane-tazobactam.

Conclusion: Although A. faecalis can rarely lead to fatal infections, it should always be considered a potential pathogen, especially in patients with compromised immune systems. Alcaligenes faecalis's susceptibility to frequently used antibiotics is declining, and data indicate growing resistance to multiple antibiotic drugs since 2018. Therefore, A. faecalis should be regarded as a pathogen rather than a contaminant. This is explained by the emerging and potentially fatal infections caused by multidrug resistant strains of this microbe. It is important for healthcare providers to be vigilant and proactive in treating infections caused by this bacterium.

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