Section: Miscellaneous

Case Series

UNRAVELLING PATTERNS AND UNUSUAL PRESENTATIONS OF BURKHOLDERIA-AN INSIGHT

Sandhya Varshini.S[1], R.M. Nivetha[1], Umapathy. P[2], Kennedy Kumar[1]

[1]Department of Microbiology, Sri Ramachandra Institute of Higher Education and Research, Chennai, India.
[2]Department of Pediatrics, Sri Ramachandra Institute of Higher Education and Research, Chennai, India.

Received : 05/06/2025
Received in revised form : 20/07/2025
Accepted : 08/08/2025

Keywords:
Erector Spinae Plane Block, PECS Block, Mastectomy, Postoperative Pain, Regional Anaesthesia, Analgesia.

Corresponding Author:
Dr. Sandhya Varshini. S,
Email: sandhyavarshini.98@gmail.com

DOI: 10.47009/jamp.2025.7.4.176

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (4); 933-939

ABSTRACT

We present you two cases of Burkholderia sepsis with different species having clinical manifestations being two ends of a spectrum. Both these are pediatric cases, similar age group. Usually, Burkholderia sepsis is known to occur in cases of Cystic fibrosis more frequently, but in this case series both the cases are from non-cystic fibrosis patients. The reports of Burkholderia infection in non-cystic fibrosis individuals is very few and this can be an addition to the sporadic cases. Burkholderia species are known opportunistic pathogens and are present in moist soil. Around 17 species of Burkholderia have been discovered and the most commonly encountered ones are B.pseudomallei and B.cepacia complex which comprises of around 17 sub-species. In this case series the species encountered are Burkholderia cenocepacia in a child with suspected Sero-negative Autoimmune Encepahalitis with DRESS syndrome and Burkholderia gladioli in a child with Acute exacerbation of Bronchial Asthma. B.cepacia and B. gladioli are species that are known to infect and colonise immunocompromised individuals.[1] In a study conducted in the UK for comparing the prevalence of Burkholderia species in cystic fibrosis(CF) vs non-cystic fibrosis cases, in the non-CF category, the highest number was B. cepacia followed by B. cenocepacia1. From this we can say that the rising number of cases of B.cenocepacia amongst non-CF cases is very alarming and appropriate management has to be carried on.

B.gladioli infections that have been mentioned in the literature say that the infection with this organism often occurs in newborns and individuals who are immunocompromised, and often bring in high mortality.[2]

INTRODUCTION

CASE 1

CASE PRESENTATION

The patient is a developmentally normal school going child, admitted initially in another institution for 2 days fever. He also had 5 episodes of vomiting and chills 10 days back, following which the parents noticed painful swelling over the neck. The child was treated with IV medications locally in a hospital and the symptoms seemed to have resolved. During the hospital course, the child had 3 episodes of vacant stare followed by tonic-clonic movements of the limbs, associated with post- ictal drowsiness lasting for half an hour, also the mother noticed the child being disoriented. Hence, he was taken to another tertiary care center for further management. The child was admitted to PICU where his Glasgow coma scale (GCS) was noted to be 12/15 and was treated with Inj. Levetiracetam. Imaging, basic blood investigations were ordered and were reported normal. In the PICU, the child continued to have altered behavior with inappropriate screaming and violent behavior, suspected to have encephalopathy, for which he was treated with Intravenous antibiotics and antiviral medications. On the same day child had another episode of seizure, which was managed with IV Phenytoin and Sodium Valproate. Subsequently, MRI (Magnetic resonance imaging) Brain, Cerebrospinal fluid(CSF) analysis were done and reported normal. CSF multiplex Polymerase chain reaction (PCR), CSF culture and sensitivity were also done and reported negative for organisms; hence, antibiotics and antivirals were stopped. Electroencephalogram (EEG) done, had Bilateral Diffuse Cerebral Dysfunction. A provisional diagnosis of Autoimmune encephalitis was made, after sending Acute Disseminated Encephalomyelitis (ADEM) workup. Child was started on Pulse Methylprednisolone (4 doses given) and Intravenous Immunoglobulin.

Child continued to have multiple episodes of vacant stare and focal seizure, and was managed with Locasamide and Midazolam infusion for 24 hours.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Following Midazolam infusion, no seizure was noted, hence, tapered and stopped after 36 hours. On the 8th day of admission, he again developed seizure with respiratory depression and poor GCS, for which he was intubated and was started on Midazolam and Ketamine infusion.

In view of Refractory seizures, Tab. Pyridoxine and Syp. Carnitine were added to the existing medications and the child was referred to our institution for further management.

COURSE OF TREATMENT AND INVESTIGATIONS:

The child was received in PICU, the basic blood workup was done, serial monitoring of renal function and liver function was done in the PICU. Post-admission, he was seizure- free for 24 hours, then he developed another seizure episode and was managed with Phenobarbitone. He was started on T. Tetrabenazine via Nasogastric tube, and in view of involuntary movements T. Pacitane was added. EEG done on 7th day of admission showed "burst suppression pattern". Gradually Ketamine was tapered as child was seizure free for more than 24 hours. Post Ketamine stoppage, 24 hours later the child developed clusters of seizures, for which he was loaded with Topiramate and Ketamine.

He also developed hypotension which was managed by inotropic support; after securing a line for invasive BP monitoring.

He was continued to be on ventilator support which was gradually weaned off, once he was seizure-free was 48 hours after stopping Midazolam infusion.

Child was currently on Topiramate, he developed new onset of fever, hence infectious disease workup, that is culture and sensitivity testing of blood, urine and endotracheal aspiration was done.

Blood cultures were sent from central and peripheral lines showed growth of Pseudomonas aeruginosa which was sensitive to Cefepime, Ceftazidime, Ciprofloxacin, Levofloxacin, Piperacillin-tazobactam, Tobramycin, Amikacin, Cefaperazone-sulbactam; The endotracheal secretion that was sent on the same day for culture showed 1000000 col/unit of Acinetobacter species sensitive to Amikacin, Cefaperazone- sulbactam, Cefepime, Ciprofloxacin, Levofloxacin, Tobramycin, Piperacillin-tazobactam). The child was treated according to the Antibiotic susceptibility testing. Repeat blood cultures were sent from central and peripheral lines, after antibiotic treatment showed No Growth.

He was extubated and was maintained on HFNC, 2 weeks after which he was weaned to nasal prongs; later was maintaining saturation on room air. Repeat Endotracheal secretion was sent for culture, it showed the growth of 100000 col/ unit of Klebsiella pneumoniae that was sensitive only to Polymyxin.

During the course of the PICU stay, the child developed maculopapular rash over the trunk and face; a drug allergic reaction was suspected and was diagnosed as DRESS Syndrome. Phenytoin was changed to Brivaracetam. The child developed fever, increased rashes (Figures1-5 the new onset rashes can be seen on the body and Figures 6-8 show the healed rashes), elevated Liver enzymes and increased AEC pointing to Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) Syndrome, Sodium valproate was stopped and Perampanel was added instead. He was given 5th dose of Pulse Methylprednisolone and a course of IVIG 2mg/kg and was told to continue for 6 weeks. In view of hyperpigmentation due to the rashes, the child was advised to start on Topical agents.

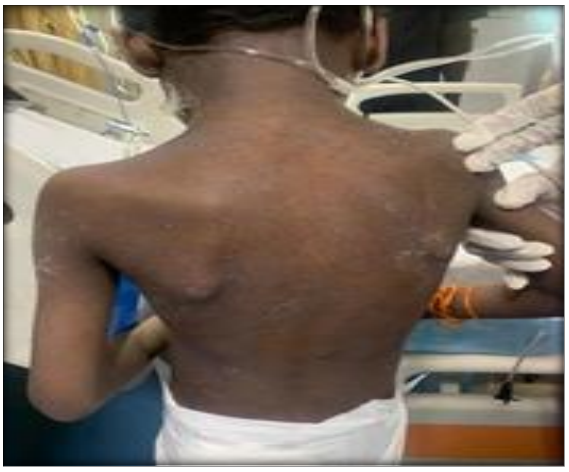

Figure 1

Figure 2

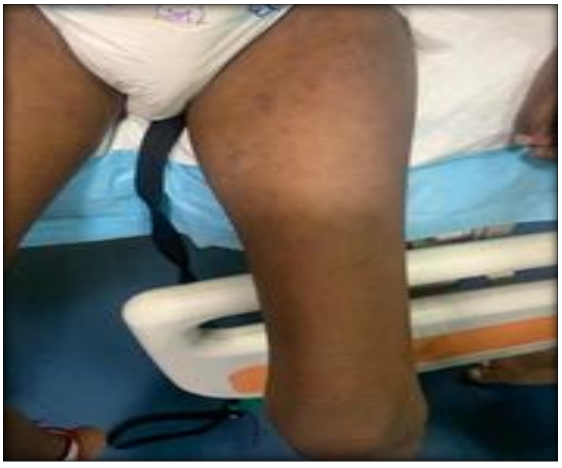

Figure 3

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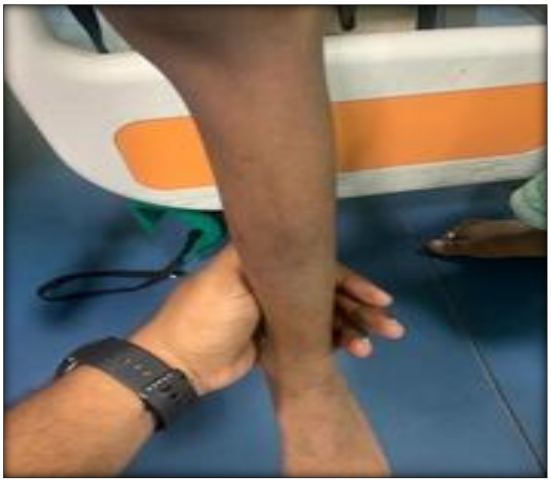

Figure 4

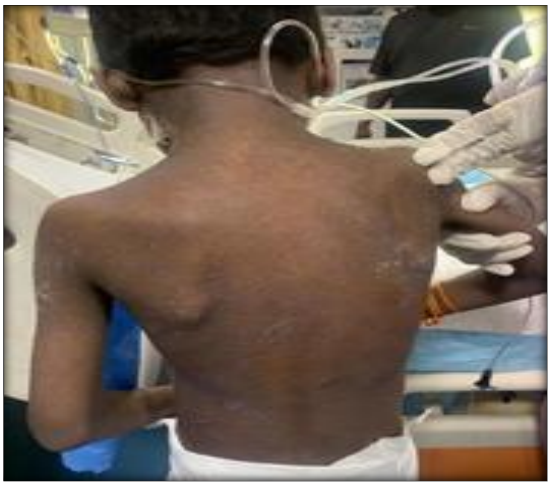

Figure 5

Figures 1 to 5 show the rashes during their onset.

In view of fever spikes again, blood cultures were sent from central and peripheral line which showed the growth of Burkholderia cenocepacia **Sensitive to Ceftazidime, Cotrimoxazole, Levofloxacin, Piperacillin- tazobactam, Cefaperazone-sulbactam**, following which the antibiotics were changed to Cefaperazone- Sulbactam.

Since there were no signs of sepsis, the child was shifted to the ward and was continued on IV antibiotics, Nasogastric feeds and anti- epileptics. The child was on keto- diet and urine ketone levels were monitored. The anti-epileptics and muscle relaxants were tapered and the child was started on chest and physiotherapy.

Repeat Chest Xray done in view of cough was normal, steroids were stopped and the child was discharged with anti-epileptic drugs and multivitamin supplements.

Figure 6

Figure 7

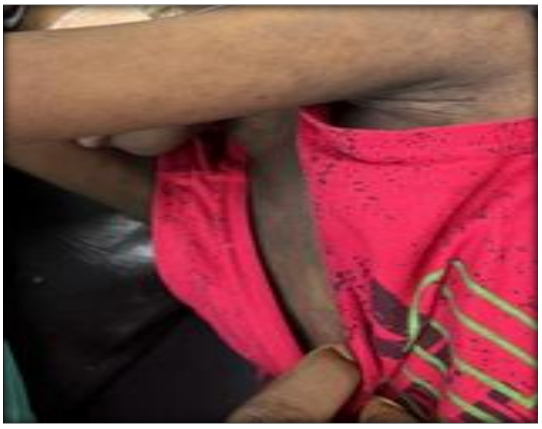

Figure 8

The above images show the healed rashes

Investigations

Routine blood tests, MRI brain, and cerebrospinal fluid (CSF) studies were all included in the workup during admission, and all were normal. Infectious pathogens were not identified by CSF multiplex PCR, culture, or sensitivity. Initially, the electroencephalogram (EEG) showed diffuse cerebral dysfunction bilaterally. Renal and liver function tests were serially checked after the patient was referred to a referral center. On the seventh day of admission, a follow-up EEG showed a burst

ISSN (O): 2687-5365; ISSN (P): 2753-6556

suppression pattern. Endotracheal cultures grew Acinetobacter species, and central and peripheral line blood cultures had growth of Pseudomonas aeruginosa sensitive to several antibiotics because of repeated fever spikes. Repeat blood cultures after initiation of antibiotics had no growth. A follow-up endotracheal secretion culture, however, had Klebsiella pneumoniae that was only sensitive to polymyxin. A second collection of blood cultures from a subsequent episode of fever yielded Burkholderia cenocepacia, which was treated appropriately with cefaperazone-sulbactam. A chest X-ray obtained in response to a cough was normal. Maculopapular rash, fever, abnormal liver enzymes, and abnormal absolute eosinophil count were utilized to raise the suspicion of DRESS syndrome. Upon admission, an ADEM workup was sent out, but it was negative.

Treatment

The refractory seizures of the child and other systemic issues required a complex and dynamic treatment regimen. Levetiracetam, Phenytoin, Sodium Valproate, Lacosamide, Midazolam, Ketamine, Phenobarbitone, Topiramate, Tetrabenazine, Pacitane, Brivaracetam, and Perampanel were some of the antiepileptic medications employed in the course. He was also given multivitamin supplements and supportive drugs such as carnitine and pyridoxine. He began a ketogenic diet with his urine ketones being monitored. Ketamine and midazolam were infused continuously as part of sedation to manage seizures.

Five pulses of intravenous methylprednisolone and a number of IVIG sessions were utilized as immunotherapy, specifically for DRESS syndrome and possible autoimmune encephalitis. Multiple antibiotics, including Cefepime, Piperacillin-tazobactam, Cefoperazone-sulbactam, Amikacin, Ciprofloxacin, Levofloxacin, Polymyxin, and Ceftazidime, were altered according to culture sensitivity results. He required inotropic support and mechanical ventilation during periods of importance and was weaned gradually. Other management of symptoms involves chest physical therapy to ensure pulmonary function and topical therapy for hyperpigmentation with the rash. The child was provided with dietary supplements and oral anti-epileptic medication upon stabilization.

OUTCOME AND FOLLOWUP

Following a prolonged PICU course, sensorium and seizure control of the child improved, and they were discharged in a clinically stable condition. He was seizure-free and maintaining oxygen saturation on room air at the time of discharge. Topiramate, Brivaracetam, Perampanel, and supportive multivitamin supplements were some of the anti-epileptic medications continued. Steroids were advised for six weeks followed by tapering. For residual hyperpigmentation from DRESS syndrome, topical therapy was advised. The family was educated about adherence to drugs and prevention of seizures.

Follow-up included routine monitoring of liver function tests, complete blood counts, and therapeutic drug levels in an attempt to monitor the success of therapy as well as any side effects caused by drugs.

CASE 2

CASE PRESENTATION

An early childhood age, developmentally normal boy, who is a known case of Multi-triggered wheeze, came with complaints of cough and increased work of breathing associated with noisy breathing with low-grade fever and rhinorrhoea for the past 2 days. There was decreased food intake and activity for one day. The child was seen in the Emergency Room, and admitted to the PICU in view of respiratory distress and hypoxia (SpO_2 <90%) and was provisionally diagnosed as Acute Severe Asthma.

In the PICU, he was managed on HFNC, Chest X-Ray showed bilateral perihilar infiltrates with hyperinflation, appropriate management for Acute severe asthma/ status asthmaticus was initiated. On day 4 of admission the child was started on Oseltamivir.

During the serial monitoring of vitals and ABG, the child was found to have hypokalemia, hence was treated for the same. During the PICU stay, the patient continued to have fever spikes for which blood cultures were sent and was prophylactically treated with Ceftriaxone. The blood culture showed the growth of Burkholderia gladioli that was sensitive to Cefaperazone- sulbactam, Ceftazidime, Levofloxacin and Meropenem. Following the blood culture report the child was treated with Cefaperazone- sulbactam for 4 days. The repeat blood cultures showed no growth.

As the child's condition improved the child was shifted to ward and High frequency nasal cannula was tapered to low frequency then to Hudson mask. As the child's condition improved and was maintaining on room air, he was discharged with Levofloxacin as discharge medication.

Investigations

A chest X-ray during the initial workup showed bilateral perihilar infiltrates and hyperinflation that could suggest an asthma attack. After hypokalemia was noted on serial vital sign and arterial blood gas (ABG) monitoring, it was corrected. Blood cultures were sent due to recurring fever spikes. The cultures yielded Burkholderia gladioli, and it is sensitive to levofloxacin, meropenem, cefaperazone-sulbactam, and ceftazidime. The child received four days of cefaperazone-sulbactam treatment in accordance with the sensitivity report. After treatment, follow-up blood cultures revealed no growth, suggesting that the bloodstream infection had been cleared.

Treatment

The child's acute severe asthma was treated in the PICU using supportive care, bronchodilators, and high-flow oxygen therapy. On the fourth day, oseltamivir was started, most likely in response to a suspected or confirmed viral infection. Blood gas

ISSN (O): 2687-5365; ISSN (P): 2753-6556

analysis indicated hypokalemia, which was appropriately managed. The initial empirical antibiotic employed was ceftriaxone; once blood cultures provided the diagnosis of Burkholderia gladioli, Cefaperazone-sulbactam was added to the antibiotics, which were continued for four days. Once sterile repeat blood cultures confirmed the infection had abated and improved the condition of the child, the child was weaned off HFNC and transitioned onto low-flow nasal oxygen prior to being put onto a Hudson mask. He later was able to maintain the oxygenation on room air at adequate levels.

OUTCOME AND FOLLOWUP

After resolving the respiratory distress and weaning the child successfully from oxygen therapy, the child was discharged in stable condition. With proper oral intake and activity levels, he had a normal oxygen saturation on room air. Following culture sensitivity, on discharge, he was prescribed oral Levofloxacin to complete the course of antibiotics. The repeat cultures were sterile, and there were no further episodes of fever.

To assess asthma control and review inhaler technique and avoidance of environmental triggers, a follow-up with the Pediatric Pulmonology Clinic was arranged. Parents were counseled on recognizing respiratory distress early and the importance of taking controller medications as prescribed. Repeat blood tests were advised in the case of a new fever or symptoms.

SOCIO-ECONOMIC STATUS

Both the patients belonged to urban area

SENSITIVITY PATTERNS

Table 1 shows the antibiotic susceptibility pattens of both the species of Burkholderia seen in this case series.

Table 1: Sensitivity patterns of both the Bukholderia species that was reported

ANTIBIOTICS	B. CENOCEPACIA (Case 1)	B. GLADIOLI (Case 2)
Cefaperazone- Sulbactam	Sensitive	Sensitive
Ceftazidime	Sensitive	Sensitive
Chloramphenicol	Resistant	Resistant
Cotrimoxazole	Resistant	Resistant
Levofloxacin	Resistant	Resistant
Meropenem	Sensitive	Sensitive
Cefotaxime	Resistant	Sensitive
Piperacillin- Tazobactam	Sensitive	Sensitive
Amikacin	Resistant	Resistant
Tobramycin	Resistant	Resistant

CULTURE CHARACTERISTICS

Figures 9 and 10 show the growth of Burkholderia cenocepacia and Burkholderia gladioli on MacConkey agar plates.

Figure 9: Burkholderia cenocepacia

Figure 10: Burkholderia gladioli

DISCUSSION

The genus Burkholderia contains over 80 different Gram-negative species including both plant and human pathogens, the latter of which can be classified into one of two groups: the Burkholderia pseudomallei complex (Bpc) and the Burkholderia cepacia complex (Bcc). Burkholderia pseudomallei and Burkholderia mallei are extremely aggressive pathogens, despite not being considered as bioterror agents, the Bcc pathogens Burkholderia cenocepacia, Burkholderia multivorans, and Burkholderia cepacia

ISSN (O): 2687-5365; ISSN (P): 2753-6556

have a remarkable effect on the cystic fibrosis population.[20] Infections with BCC are commonly seen in immunocompromised patients, and has a tendency to affect patients with cystic fibrosis and Chronic granulomatous disease.[5] Presence of indwelling catheters in-situ, patients on haemodialysis for renal failure, exposure to frequent bronchoscopy techniques, recent abdominal surgery and prolonged Intensive care unit(ICU) stay forms the major contributing risk factors for acquiring BCC infection.[3,4]

Outbreaks of BCC infections have been reported from both Intensive care and oncology units. BCC is capable of thriving in the aqueous hospital niche such as IV fluids and commonly used chemical disinfectants.[6] The main source for transmission of BCC is through various water sources like: wash basins, while performing oral/ tracheostomy care, ventilator- support care, oro-gastric tube feeds, nasal sprays, gels used for Ultrasound procedures, etc.[6]

BCC exhibits intrinsic resistance towards aminoglycosides and polymyxins, because of which treating them remains a difficult task for the clinicians.[7] The recommended treatment is with 3rd and 4th generation Beta-lactam antibiotics.[8]

Sarkar K et al.(2024) from New Delhi has reported refractive seizures in a pediatric patient who was not responding to treatment, whose blood culture grew BCC who responded later with specific treatment against BCC.[9] Similarly, the first case of this case series also had similar presentation of refractory seizures not responding to anti- epileptic measures later responded following treatment for B. cenocepacia which grew in the culture.

There have been studies where latency has been noticed in B. cenocepacia infections, which was attributed to its adaptive shift to fatty acid metabolism, which helps it to survive in oxygen limited settings. Hypermutation, intracellular survival within host cells like epithelial cells and macrophages, biofilm formation was also cited as reasons for its persistence or latency causing chronic burkholderia infections.[10] A BCC bacteremia was encountered in Command Hospital Airport Bangalore, in a renal transplant recipient who was on a cocktail of immunosuppressants,[11] this could have been a flare-up of a latent Burkholderia infection or a new infection following steroid usage, which is similar to the infection seen in the first case, where the infection occurred post the use of Methylprednisolone.

There have been cases (mainly in known cystic fibrosis patients) where patients have presented with cutaneous vasculitis often typical of BCC infections, where the rashes range from erythematous to maculopapular type and hyperpigmentation; biopsy of one of which revealed the presence of hemosiderin.[12] Here, the patient in the first case presented with a purple, maculo-papular rash, with can rise a suspicion of Burkholderia infection (causing cutaneous vasculitis), for which DRESS syndrome can be a differential diagnosis which was initially suspected in this case.

There have been a very few reports on hyperpigmentation due to BCC; in India, a tertiary hospital in Meghalaya has reported a peculiar violet-coloured pigmentation as a result of BCC infection,[19] similar to one of the cases in this case series.

B. gladioli was initially described as a colonizer in patients with CF13, often seen as an opportunistic pathogen with chronic granulomatous disease, Acquired immunodeficiency syndrome(AIDS), diabetes mellitus and organ transplant recipients.[14] Thompson III GR et al. in 2011 reported a case of Burkholderia gladioli infection was reported in a 48-year-old Chronic obstructive pulmonary disorder (COPD) patient who was a known case of hypocomplementemic urticarial vasculitis syndrome15. There are cases of B.gladioli which were reported in patients with viral respiratory infections like COVID-19.[16]

It has been reported that BCC is an important cause in many cases of pneumonia, bacteremia, urinary tract infection, endocarditis, meningitis, brain abscess in children and according to Peng F et al.'s study, they were most commonly associated with respiratory tract infections.[17]

B. cenocepacia is an aggressive pathogen known to cause infections in immunocompromised and is often intrinsically resistant to many antibiotics, hence combination therapy strategies are often employed to enhance treatment efficacy.

B. gladioli on the other hand is till an emerging pathogen and there have been some cases of bacteremia and infections which have been documented, indicating its potential as an opportunistic pathogen.

CONCLUSION

Recently, Burkholderia complex is being reported often due to advancements in microbiological diagnosis like MALDI- TOF, Microscan, etc. In resource limited settings where these automated systems are not available, a diagnosis of Burkholderia infection can be made with the help of conventional antimicrobial susceptibility, i.e., intrinsic resistance to Polymyxin B.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556