



## CASE REPORT

# *Burkholderia pseudomallei* infection of a spinal implant following traumatic spinal injury: A rare case of implant-associated melioidosis

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### ABSTRACT

Melioidosis, caused by *Burkholderia pseudomallei*, is an important endemic infection in Southeast Asia. Pulmonary infection and bacteremia are most common presentations whereas musculoskeletal involvement is less frequent and infection associated with orthopedic implants is rarely reported. We describe a case of a 47-year-old man who was presented with swelling in the back following a complicated recovery from a prior unstable chance fracture of T12 and L1 vertebrae which he acquired during an industrial injury at his workplace. He underwent a Posterior Sacro-iliac Spine Fusion for the vertebral function. He also sustained multiple medical and psychiatric conditions including paraplegia, neurogenic bladder and Major Depressive Disorder (MDD) following the spinal injury. After discharged, he had experienced recurrent episodes of infections related to spinal implant. On this current presentation, his pus culture grew *Burkholderia pseudomallei*, which was a rare pathogen that could be associated with implant infections. Patient had also developed hyperkalemia which was one of the adverse effect from trimethoprim-sulfamethoxazole which was one of the antimicrobial therapy used for *Burkholderia pseudomallei*. This case illustrates the challenges in handling a rare infection associated with spinal implant and in managing adverse effect of hyperkalemia associated with Trimethoprim-sulfamethoxazole.

**Keywords:** *Burkholderia pseudomallei*; spinal injury; implant-associated infection; Trimethoprim-sulfamethoxazole-associated hyperkalemia.

### INTRODUCTION

*Burkholderia pseudomallei* is a Gram-negative bacterium that causes melioidosis. The organism is commonly found in soil and surface water, and infection typically occurs through percutaneous inoculation, inhalation, or ingestion. Risk factors underlying include diabetes mellitus and environmental exposure. The clinical manifestations of melioidosis are highly variable, ranging from localized infection to severe disseminated disease with septicemia. Pneumoniae and visceral abscess are most frequently reported presentation. Implant-associated infections due to *B. pseudomallei* are rare, particularly those involving spinal instrumentation. The presence of foreign material may predispose to persistent infection and poses additional challenges in diagnosis and management. Here, we report a rare case of implant-associated melioidosis involving a spinal implant following traumatic spinal cord injury in a non-diabetic patient. He also developed hyperkalemia due to trimethoprim. This case highlights the diagnostic challenges and therapeutic considerations in managing this uncommon presentation.

### CASE PRESENTATION

A 47-year-old man presented to our hospital in June 2024 with a 2-day history of central back swelling. He reported that the

swelling was similar to his previous episodes of spinal implant infection. Otherwise, he denied fever, pain, shortness of breath or gastrointestinal symptoms. Otherwise he was tolerating orally well and ambulating with a wheelchair. His medical history was significant for an alleged industrial injury sustained in October 2023, when construction material fell on his back, resulting in unstable chance fracture of the T12 and L1 vertebrae. He also sustained multiple ribs fracture and lung contusion. His hospital course was further complicated by pulmonary embolism, *Staphylococcus aureus* bacteremia, *Pseudomonas aeruginosa* urinary tract infection and multiloculated pleural effusion, leading to a prolonged hospitalization for about 2 months. He subsequently underwent a posterior spinal instrumentation and fusion of T10 to L3 in November 2023. Due to the spinal injury, he acquired paraplegia and neurogenic bladder. He subsequently developed Major Depressive Disorder (MDD) and was under Psychiatry follow up.

He had a history of two prior admissions to another hospital for surgical site infection. During the first admission in 30 January 2024 to 6 February 2024, he underwent wound debridement, wound exploration, synovectomy and Collatamp insertion. Tissue culture and sensitivity at that time grew *Acinetobacter* sensitive to ampicillin/sulbactam. During second admission from 12 February 2024 to 14 February 2024, he was admitted for infected wound. A six-week course of ampicillin/sulbactam was administered. Otherwise,

for the social history, he was married and blessed with a daughter. He used to work as a lorry driver for a construction company.

On examination of the current admission, the patient was alert, afebrile, and stable, with a blood pressure of 117/78 mmHg, pulse rate of 98 beats per minute, and respiratory rate of 18 breaths per minute. His fingertip glucose level was normal at 6.1 mmol/L and SPO<sub>2</sub> 98% under room air. His lungs were clear and per abdomen was soft without any guarding or rigidity. Local examination of the back showed a healed central wound scar. There was swelling over upper back measuring 4x2cm, mobile, fluctuant and no discharge. The overlying skin was warm to touch but not erythematous. On musculoskeletal examination, the power of both limbs was found to be 0/5 from L1 to S1, with decreased sensation from L1 to L2 and absent from L3 to S5 dermatomes. The deep anal pressure and voluntary anal contraction was absent.

Initial laboratory investigations indicated anemia (Hb 9.00 g/dL), thrombocytosis (Platelet  $777 \times 10^3/\mu\text{L}$ ), mild leukocytosis ( $12.6 \times 10^3/\mu\text{L}$ ) with predominance of neutrophils (78.50%). He had elevated inflammatory markers with CRP 82.00 mg/L (0.00-5.00mg/L) and ESR 110 mm/hr (0-22mm/hr)). His renal profile showed mild hyponatremia with sodium 129 mmol/L while his potassium was normal at 4.5mmol/L. His X-ray of thoracolumbar/sacral showed implant was in situ and no loosening.

A provisional diagnosis of infected implant of spine was made based on the history and physical examination. He was admitted to ward and started on IV ampicillin/sulbactam 3g three times per day based on his previous culture results that grew *Acinetobacter*. He was also seen by Rehab Team for his physiotherapy and Psychiatry Team for to monitor his MDD symptoms. Along the course of his admission, he was also given intravenous drip and renal function monitoring for his hyponatremia.

His Computed Tomography (CT) Thoracolumbar showed that that there was no evidence of loosening implant and there was T12 and L1 chance vertebral fracture which was similar to previous CT done before. However there was a multiloculated rim enhancing intramuscular collections involving bilateral erector spinae muscles from T8/T9 until L4/L5 levels. There was also minimal pelvic free fluid. Magnetic Resonance Imaging (MRI) Thoracolumbar done confirmed the presence of multiloculated bilateral erector spinae intramuscular collections with surrounding inflammatory changes, and subcutaneous collection extension. The collection also surrounding the spinal implant, raising suspicion of peri-implant extension (Figure 1). Although there was evidence of a possible infection, the surgeons were unable to remove the implant as the spine fracture hadn't reached adequate fusion.

Subsequently on day two of admission, a bedside aspiration was done and noted thick pus discharge about 10cc. Pus was sent for Mycobacterium tuberculosis culture and sensitivity, Mycobacterium tuberculosis PCR, Acid Fast Bacilli (AFB) smear, bacterial and fungal culture and sensitivity. His bacterial culture demonstrated pure growth of a whitish non hemolytic mucoid colony on blood agar and scanty pinkish colony on MacConkey agar (Figure 2). Direct smear of the colony showed Gram negative rods with safety pin appearance (Figure 3). Colony identification was performed using MALDI-TOF MS (Bruker Biotyper) which identified the organism as *Burkholderia pseudomallei* with a confidence score of 2.29. Antimicrobial susceptibility testing done indicated that the organism was sensitive to ceftazidime, amoxicillin/clavulanate, trimethoprim-sulfamethoxazole, gentamicin, and meropenem. The antimicrobial regimen was switched to IV ceftazidime 2 grams 6 hourly and oral trimethoprim-sulfamethoxazole 4 tabs twice per day as per intensive phase regimen for *Burkholderia pseudomallei*. He was planned to continue the regimen for 6 to 8 weeks, and to be followed by eradication phase regimen with oral trimethoprim-sulfamethoxazole

for another 6 months. Otherwise, his Mycobacterium tuberculosis PCR showed no MTB detected, AFB smear was also negative of acid fast bacilli. Fungal culture yielded no growth after fourteen days of incubation. His Mycobacterium tuberculosis culture and sensitivity however came back as contaminated.

Along the course of antimicrobial therapy, patient developed hyperkalemia after day eight of trimethoprim/sulfamethoxazole where his potassium level increased to 5.0mmol/L. His potassium normalized to 4.9mmol/L after two times of lytic cocktail. However the potassium level rose up to 5.7mmol/L on subsequent blood taking. He was suspected to develop an episode of persistent hyperkalemia which was a known adverse reaction to trimethoprim/sulfamethoxazole. Thus, the regimen was changed to graded dosing (1/2 tab twice per day for 2 days, then 1 tab twice per day for 2 days, then 2 tab twice per day for 2 days and 3 tab twice per day for 2

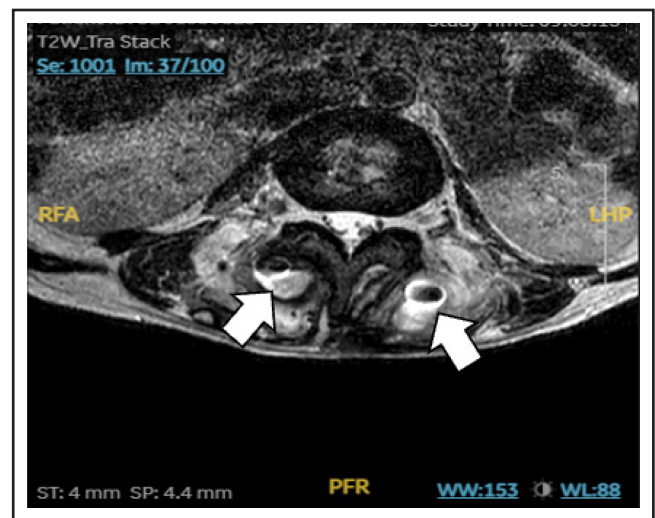


**Figure 1.** Axial T2-weighted MRI of thoracolumbar spine demonstrating multiloculated rim enhancing intramuscular collections involving bilateral erector spinae muscles (white arrow).

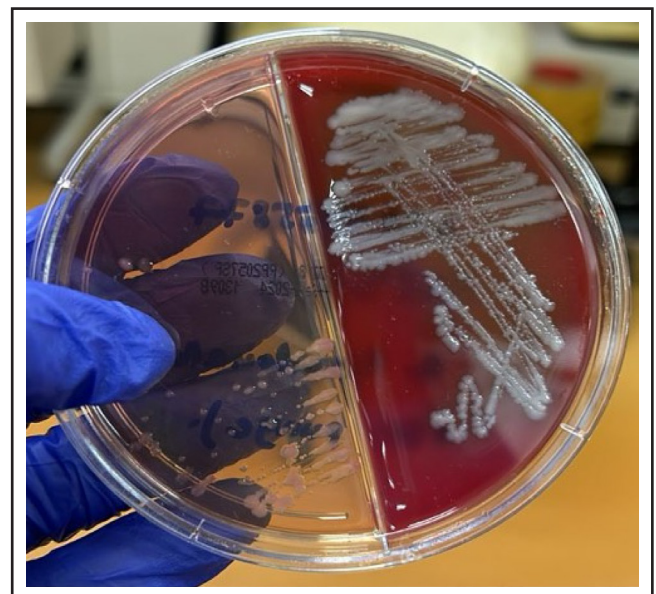


**Figure 2.** Creamy whitish colony on blood agar and scanty pinkish colony on MacConkey agar.



**Figure 3.** Gram stain of colony shows gram negative rod with safety appearance.

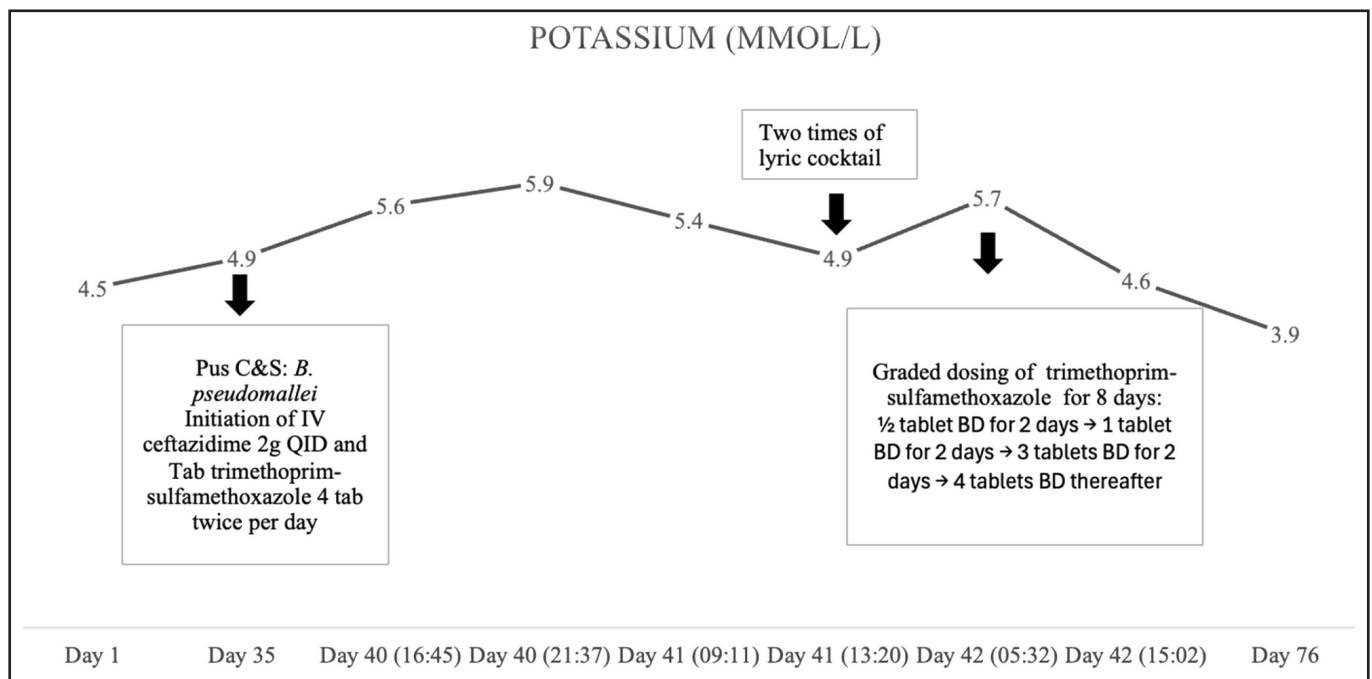


**Figure 4.** Trend for potassium.

days and subsequently 4 tabs twice per day afterward). The graded dosing for trimethoprim/sulfamethoxazole was given over a course of 8 days. His potassium level subsequently normalized afterwards, ranging from 3.9mmol/L to 4.5mmol/L (Figure 4).

On day thirteen of admission, his wound showed punctum with pus discharge. He undergone an emergency operation for wound debridement and Collatamp insertion. Intraoperative finding showed there was abundant fibrous tissue over previous incision site with slough and biofilm over implant. There was approximately 20cc of pus from deep tissue. Tissue and pus cultures sent intraoperatively however yielded no growth. His tissue for histopathology

examination revealed granulomatous reaction, with presence of many multinucleated giant cells of foreign body type.

He completed effective intensive phase (combination IV ceftazidime and trimethoprim/sulfamethoxazole) for a total of 44 days (approximately 6 weeks). He was discharged with monotherapy of trimethoprim/sulfamethoxazole to be completed for 6 months. A follow up visit to the clinic revealed that he had no new complains, was adherent to treatment and his spinal wound healed. His full blood count, renal function test and liver function test were also within normal parameters. Figure 5 illustrates the chronology of the patient's hospital course.

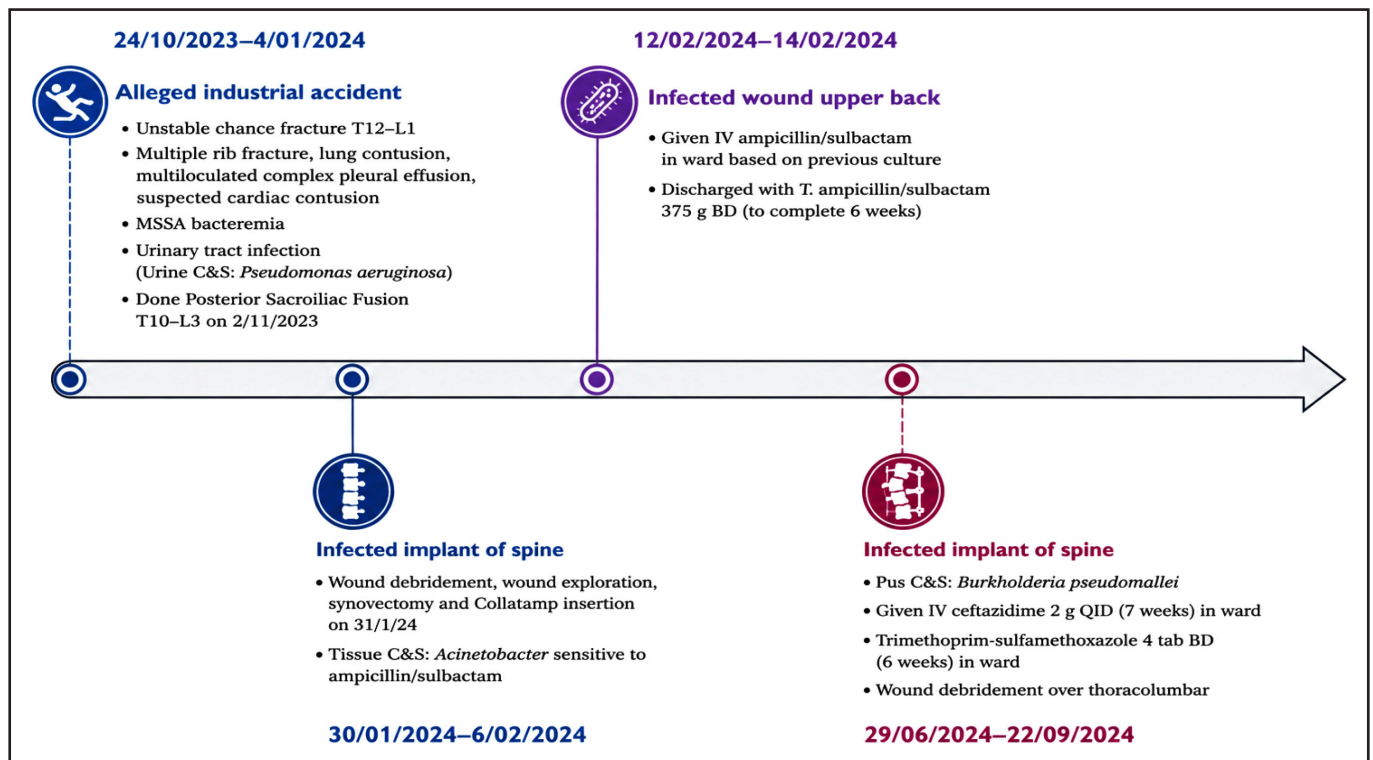


**Figure 5.** Chronology of clinical events, microbiological findings and antimicrobial management.

Abbreviations: C&S, culture and sensitivity; MSSA, methicillin-susceptible *Staphylococcus aureus*; IV, intravenous; T., tablet; BD, twice daily; QID, four times daily.

## DISCUSSION

Melioidosis is endemic in many tropical countries including Southeast Asia and Australia. The infection is caused by gram-negative bacterium *Burkholderia pseudomallei*. Soil is natural reservoir for the organism. The bacterium enters the body via inhalation, inoculation through skin breaches or ingestion (Cheng & Currie, 2005). Consequently, when host immunity is impaired, opportunistic infection or reactivation of latent bacteria may ensue. Clinical manifestations is variable and can range from localized soft tissue infection to severe septicemia. In Malaysia, more than 90% of cases were acute onset, presenting as acute respiratory infection or acute bacteremia whereas musculoskeletal involvement was infrequent (Nathan et al., 2018). Implant associated infection had been rarely documented. Clinically, it can present as localized pain over implant site, swelling, erythema or warmth around surgical area, or chronic wound with sinus discharge (Arockiaraj et al., 2016). The presentation is usually chronic in nature and overall mortality is low (Raja & Scarsbrook, 2016). Hematogenous spread following percutaneous inoculation was postulated as the major mode of transmission. Bacterium may also reach by direct spread from adjacent organs and soft tissue (Raja & Scarsbrook, 2016). In our patient, the most likely mode of transmission was percutaneous inoculation following environmental exposure to soil, particularly given his occupational history as a lorry driver at a construction site. Presence of implant and biofilm may have supported the growth of this intracellular pathogen (Gassiep et al., 2020).

The most prevalent predisposing factor for melioidosis is type 2 diabetes mellitus and it is the most common co-morbid condition associated with melioidosis in Malaysia (Arushothy et al., 2024; Wiersinga et al., 2018). In addition, environmental exposure through soil-related activities and high risk occupations has been identified as important risk factor for infection (Arushothy et al., 2024). Although our patient was non-diabetic, his spinal cord injury may have contributed to the functional immunosuppression. Spinal cord

injury has been associated with neurogenic immune suppression, termed spinal cord injury-induced immune deficiency syndrome (SCI-IDS). Recent evidence from the SCIntinel study demonstrated that patients with spinal cord injury may develop impaired immune responses and are at risk of infections due to dysregulation of autonomic-immune pathways (Kopp et al., 2023). The study found that patients with spinal cord injury developed reduced mHLA-DR expression, impaired monocyte function, reduce immunoglobulin levels; which can impairment of both cellular and humoral immunity. This mechanism may have contributed to the susceptibility in our patient to *Burkholderia pseudomallei* infection despite the absence of traditional risk factors such as diabetes mellitus. Nevertheless, in this patient, formal immunological investigations were not performed in our setting, and therefore underlying immune impairment could not be definitely established.

Laboratory investigations in melioidosis commonly demonstrate elevated inflammatory markers, including leukocytosis with neutrophilia, increase CRP and ESR levels all of which were observed in this patient (Arockiaraj et al., 2016). He also demonstrated thrombocytosis, likely representing a reactive response to the underlying infection. These parameters subsequently normalized following appropriate antimicrobial therapy and adequate source control, reflecting favorable treatment response.

Histopathological findings in melioidosis are heterogeneous and may pose significant diagnostic challenges. Reported features include abscess formation, necrotizing and/or granulomatous inflammation, as well as multinucleated giant cells (Arockiaraj et al., 2016). In our patient, histopathological examination was likewise non-specific, demonstrating granulomatous reaction with numerous foreign body-type multinucleated giant cells. Foreign body-type giant cells may reflect implant-associated chronic inflammation. Presence of multinucleated giant cells may be associated with tuberculosis or fungal infection (Savelkoel et al., 2023). In our center, pathogen-specific immunohistochemistry was not done. However, tissue sent for tuberculosis and fungal culture showed no growth, making the

infection by these organisms were unlikely. Nevertheless, culture sensitivity may be limited, with reported rates as low as 60% (Wiersinga *et al.*, 2018).

Several challenges were encountered in the isolation of *Burkholderia pseudomallei* in this patient. Due to the presence of biofilm, localized nature and low bacterial load, the organism may be difficult to isolate. The organism was not isolated during the initial course of infection. This can be related its ability to remain latent before reactivating in the setting of immunosuppression (Gassiep *et al.*, 2020). A comparable case of melioidosis-associated peri-prosthetic infection occurring 10 years after medial malleolar internal fixation has also been described (Jayarajah *et al.*, 2020). Other plausible contributing factors include low organism burden and possible inadequate sampling during the initial evaluation. Secondly, growth of the organism was obtained only from the pus aspirate, while blood cultures and intraoperative cultures remained negative. This reflected the localized nature of the infection, with a higher bacterial concentration confined to the peri-implant collection, thereby increasing the diagnostic yield of the aspirated pus specimen. This observation highlights the importance of obtaining multiple specimen types, including tissue and pus aspirates, when implant associated *Burkholderia pseudomallei* infection is suspected, as this may improve diagnostic yield.

Identification by MALDI-TOF MS (Bruker Biotyper) yielded a score of 2.29, consistent with reliable species-level identification. Although additional confirmatory methods such as molecular testing or biochemical identification systems were not performed as they were not available in our center, the diagnosis was supported by concordant microbiological findings including characteristic colony morphology, bipolar "safety-pin" appearance on Gram stain, and reliable species-level score by MALDI-TOF MS (Bruker Biotyper). Furthermore, taking into account the endemicity, the clinical presentation and epidemiological exposure were compatible with melioidosis. Nevertheless, the absence of molecular confirmation remains a limitation of this report.

Definitive source control was challenging because implant removal was not feasible due to incomplete spinal fusion. In cases where implant retention is unavoidable, early surgical debridement and drainage with appropriate administration of intravenous antibiotics is the cornerstone of management (Gouse *et al.*, 2017). *Burkholderia pseudomallei* is intrinsically resistant to many antimicrobial agents including penicillin, ampicillin, first and second generation cephalosporin and aminoglycosides. For non-neurological presentation, IV ceftazidime 2g given 6 hourly and additional trimethoprim 80mg/Sulfamethoxazole 400mg every 12hours are given in the initial intensive phase. The duration for deep seated infection is 4 to 8 weeks. For the eradication therapy, preferred antibiotic is trimethoprim 80mg/sulfamethoxazole 400mg given for 12 to 20 weeks (Gassiep *et al.*, 2020; Wiersinga *et al.*, 2018).

Regular monitoring of renal function, liver function, serum electrolytes, and full blood count is essential during prolonged trimethoprim-sulfamethoxazole therapy for melioidosis. In our patient, one of the recognized adverse effects of trimethoprim-sulfamethoxazole, namely hyperkalaemia, was observed. Hyperkalaemia is generally defined as a serum or plasma potassium level exceeding the upper limit of normal, typically greater than 5.0–5.5 mEq/L (Nickels *et al.*, 2012).

Importantly, trimethoprim is primarily responsible for the potassium elevation (Perazella, 2000). Trimethoprim exerts an amiloride-like effect by competitively inhibiting epithelial sodium channels (ENaC) located on the luminal membrane of the distal nephron. Under normal physiological conditions, ENaC facilitates passive sodium reabsorption into tubular cells, generating an electrical gradient that promotes potassium and hydrogen ion secretion into the tubular lumen. Blockade of ENaC by trimethoprim reduces sodium entry into tubular cells, resulting in a less negatively

charged tubular lumen and subsequent reduction in urinary potassium excretion, ultimately leading to hyperkalemia.

There was no definite threshold dose of trimethoprim that can cause hyperkalemia. Nevertheless studies had demonstrated that trimethoprim-induced hyperkalemia may occur with both standard and high-dose therapy, although the risk is greater with high-dose regimens commonly employed in melioidosis treatment, typically around 20 mg/kg/day of trimethoprim (Perazella, 2000). Serum potassium elevation generally follows a dose-dependent pattern and commonly develops after 4 to 5 days of therapy (Perazella, 2000). Studies have demonstrated that trimethoprim-sulfamethoxazole exhibits concentration-dependent killing activity against *Burkholderia pseudomallei*, and weight-based regimens are predicted to achieve adequate therapeutic concentrations of both trimethoprim and sulfamethoxazole (Cheng *et al.*, 2009).

In our patient, a graded dose escalation strategy was employed to minimize treatment-related toxicity while maintaining effective eradication therapy. The dose was gradually increased over 8 days from ½ tablet twice daily to the target therapeutic dose of 4 tablets twice daily, with close monitoring of serum potassium and renal function. This approach is supported by Northern Territory melioidosis guidelines, which recommend graded introduction of trimethoprim-sulfamethoxazole in selected patients receiving prolonged high-dose therapy (Mason, n.d.). Previous studies have also demonstrated reduced toxicity with gradual initiation of trimethoprim-sulfamethoxazole therapy as prophylaxis for *Pneumocystis carinii* pneumoniae in AIDS patients (Para *et al.*, 2000).

## CONCLUSION

This case report highlights the complexity of managing rare infections in the context of spinal implants, particularly when caused by *Burkholderia pseudomallei*, a pathogen more commonly associated with endemic regions and soil exposure. This case reinforces the importance of environmental exposure history in diagnosing atypical infections. Additionally, the development of hyperkalemia from trimethoprim-sulfamethoxazole therapy emphasizes the need for vigilant monitoring during the prolonged antimicrobial treatment. The successful resolution of this case demonstrates the efficacy of targeted antimicrobial therapy, combined with surgical source control and individualized patient management.

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## Declaration of Competing Interest

The authors have no conflicts of interest to declare

## REFERENCES

- Arockiaraj, J., Karthik, R., Jeyaraj, V., Amritanand, R., Krishnan, V., David, K.S. & Sundararaj, G.D. (2016). Non-caseating granulomatous infective spondylitis: Melioidotic spondylitis. *Asian Spine Journal* **10**: 1065-1070. <https://doi.org/10.4184/asj.2016.10.6.1065>
- Arushothy, R., Mohd Ali, M.R., Zambri, H.F., Muthu, V., Hashim, R., Chieng, S. & Nathan, S. (2024). Assessing the national antibiotic surveillance data to identify burden for melioidosis in Malaysia. *IJID Regions* **10**: 94-99. <https://doi.org/10.1016/j.ijregi.2023.11.014>

- Cheng, A.C. & Currie, B.J. (2005). Melioidosis: epidemiology, pathophysiology, and management. *Clinical Microbiology Reviews* **18**: 383-416. <https://doi.org/10.1128/CMR.18.2.383-416.2005>
- Cheng, A.C., McBryde, E.S., Wuthiekanun, V., Chierakul, W., Amornchai, P., Day, N.P.J., White, N.J. & Peacock, S.J. (2009). Dosing regimens of cotrimoxazole (trimethoprim-sulfamethoxazole) for melioidosis. *Antimicrobial Agents and Chemotherapy* **53**: 4193-4199. <https://doi.org/10.1128/AAC.01301-08>
- Gassie, I., Armstrong, M. & Norton, R. (2020). Human melioidosis. *Clinical Microbiology Reviews* **33**: e00006-19. <https://doi.org/10.1128/CMR.00006-19>
- Gouse, M., Jayasankar, V., Patole, S., Veeraraghavan, B. & Nithyananth, M. (2017). Clinical outcomes in musculoskeletal involvement of *Burkholderia pseudomallei* infection. *Clinics in Orthopedic Surgery* **9**: 386-390. <https://doi.org/10.4055/cios.2017.9.3.386>
- Jayarajah, U., Arulanantham, A., Koculen, V., Palkumbura, C., Faleel, A. & Sooriyachchi, R. (2020). *Burkholderia pseudomallei* peri-prosthetic infection following medial malleolar internal fixation: a case report. *BMC Infectious Diseases* **20**: 236. <https://doi.org/10.1186/s12879-020-04967-y>
- Kopp, M.A., Meisel, C., Liebscher, T., Watzlawick, R., Cinelli, P., Schweizerhof, O., Blex, C., Lübster, T., Prilipp, E., Niedeggen, A. et al. (2023). The spinal cord injury-induced immune deficiency syndrome: results of the SCIntinel study. *Brain* **146**: 3500-3512. <https://doi.org/10.1093/brain/awad092>
- Mason, L. (n.d.). Melioidosis NT Health Guideline. NT Health. <https://digitallibrary.health.nt.gov.au/nthealthserver/api/core/bitstreams/78de80ef-afe5-4aea-860a-6ea801354542/content>. Accessed 10/02/2024.
- Nathan, S., Chieng, S., Kingsley, P., Mohan, A., Podin, Y., Ooi, M.-H., Mariappan, V., Vellasamy, K., Vadivelu, J., Daim, S. & How, S.-H. (2018). Melioidosis in Malaysia: incidence, clinical challenges, and advances in understanding pathogenesis. *Tropical Medicine and Infectious Disease* **3**: 25. <https://doi.org/10.3390/tropicalmed3010025>
- Nickels, L.C., Jones, C. & Stead, L.G. (2012). Trimethoprim-sulfamethoxazole-induced hyperkalemia in a patient with normal renal function. *Case Reports in Emergency Medicine* **2012**: 815907. <https://doi.org/10.1155/2012/815907>
- Para, M.F., Finkelstein, D., Becker, S., Dohn, M., Walawander, A. & Black, J.R. (2000). Reduced toxicity with gradual initiation of trimethoprim-sulfamethoxazole as primary prophylaxis for *Pneumocystis carinii* pneumonia: AIDS Clinical Trials Group 268. *JAIDS Journal of Acquired Immune Deficiency Syndromes* **24**: 337-343. <https://doi.org/10.1097/00126334-200008010-00007>
- Perazella, M.A. (2000). Trimethoprim-induced hyperkalemia: clinical data, mechanism, prevention and management. *Drug Safety* **22**: 227-236. <https://doi.org/10.2165/00002018-200022030-00006>
- Raja, N.S. & Scarsbrook, C. (2016). *Burkholderia pseudomallei* causing bone and joint infections: a clinical update. *Infectious Diseases and Therapy* **5**: 17-29. <https://doi.org/10.1007/s40121-015-0098-2>
- Savelkoel, J., Tiemensma, M., Birnie, E., Wiersinga, W.J., Currie, B.J. & Roelofs, J.J.T.H. (2023). A graphical overview of the histopathology of human melioidosis: a case series. *Open Forum Infectious Diseases* **10**: ofad367. <https://doi.org/10.1093/ofid/ofad367>
- Wiersinga, W.J., Virk, H.S., Torres, A.G., Currie, B.J., Peacock, S.J., Dance, D.A.B. & Limmathurotsakul, D. (2018). Melioidosis. *Nature Reviews Disease Primers* **4**: 17107. <https://doi.org/10.1038/nrdp.2017.107>