CASE REPORT

Melioidosis-associated rasmussen aneurysm in a patient with progressive CKD: Successful management with bronchial artery embolization and adapted eradication therapy

You Hui Wen, MMed (Internal Medicine)[1], Muhammad Danish Syahmi Saha, MBBS[1], Noorlina Nordin, MMed (Internal Medicine)[2], Salinawati Bakin, MMed (Radiology)[3], Adib Amir (FRCR, UK)[4], You Hui Wei, PhD[5]

[1]Department of Internal Medicine, Hospital Bentong, Pahang, Malaysia, [2]Infectious Disease Unit, Hospital Tengku Ampuan Afzan, Pahang, Malaysia, [3]Interventional Radiology Unit, Hospital Kuala Lumpur, Malaysia, [4]Interventional Radiology Unit, Hospital Sultanah Bahiyah, Kedah, Malaysia, [5]School of Economics and Management, Xiamen University Malaysia

SUMMARY

Melioidosis, caused by Burkholderia pseudomallei, is endemic in Southeast Asia and often complicated by comorbidities such as chronic kidney disease (CKD). Rasmussen aneurysm is a rare but life-threatening vascular complication, typically linked to tuberculosis but occasionally seen in destructive lung infections. A 52-year-old man is reported with poorly controlled diabetes, stage 5 CKD, and prior pulmonary tuberculosis, admitted with fever and productive cough. Blood cultures grew B. pseudomallei, confirming melioidosis. While on renal dose-adjusted ceftazidime, he developed haemoptysis; CT angiography revealed a right upper lobe Rasmussen aneurysm, successfully managed with bronchial artery embolization. Antimicrobial therapy required adaptation due to hyperkalaemia and transaminitis from trimethoprim-sulfamethoxazole (TMP-SMX), prompting temporary switch to amoxicillin-clavulanate. Following dialysis initiation and biochemical stabilisation, TMP-SMX was safely reintroduced and completed for 12 weeks per 2020 Darwin guidelines. The patient remained stable without recurrence at follow-up. This case illustrates the complexity of managing melioidosis in the setting of advanced CKD, the need for tailored antimicrobial strategies, and the lifesaving role of early vascular imaging and intervention in Rasmussen aneurysm. Multidisciplinary coordination across district and tertiary care was important to the favourable outcome.

INTRODUCTION

Melioidosis, caused by *Burkholderia pseudomallei*, is a Gram negative bacillus endemic in Southeast Asia, including Malaysia. It manifests variably, from asymptomatic exposure to fulminant septicemia, and commonly affects individuals with risk factors like diabetes mellitus, chronic kidney disease, and environmental exposure to soil or water.[1] Our patient had multiple such risk factors, including poorly controlled diabetes and CKD.

Pulmonary involvement is frequent, yet haemoptysis is an uncommon presentation, when present, it may signify vascular complications such as Rasmussen aneurysm.[2] Though classically associated with cavitary tuberculosis, similar vascular lesions can occur in destructive lung disease regardless of etiology.

Treatment follows a biphasic approach, with an IV intensive phase followed by oral eradication therapy, typically guided by the Darwin melioidosis protocol. The duration of IV therapy varies based on disease severity and focus, while oral trimethoprim-sulfamethoxazole for at least 12 weeks is recommended during eradication phase.[3] CKD complicates this approach, as it necessitates renal-adjusted dosing, close monitoring for adverse effects (e.g. hyperkalaemia, hepatotoxicity), and occasional substitution of TMP-SMX when intolerance arises.[4]

CASE PRESENTATION

A 52 year old Malay man, current smoker with a 30 pack year history, presented to Hospital Bentong, a district hospital in Pahang with fever and productive greenish cough for two days. He reported previous employment in plantation-based manual labour involving frequent contact with wet soil, a known risk factor for melioidosis exposure. His background includes insulin dependent diabetes mellitus (DM) (poorly controlled), hypertension, dyslipidemia, stage 5 chronic kidney disease (CKD), and a right below knee amputation in 2020 due to diabetic foot ulcer. He completed a 6 month course of anti-tuberculous therapy for pulmonary tuberculosis in 2017.

He had previously been admitted to our centre for evaluation of renal anaemia and acute on chronic kidney disease. He presented with lethargy and haemoglobin of 6.6 g/dL (baseline ≈8.7 g/dL). He denied bleeding, supplement use, constitutional symptoms, chronic cough, or TB contact. At admission, creatinine was 746 µmol/L (eGFR 6.7 mL/min/1.73 m²). He received one unit of packed red cells. Iron studies showed iron and folic acid deficiency. Kidney function improved to creatinine 457 µmol/L (eGFR 12 mL/min/1.73 m²) by discharge.

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Corresponding Author: You Hui Wen
Email: hwyou0707@gmail.com

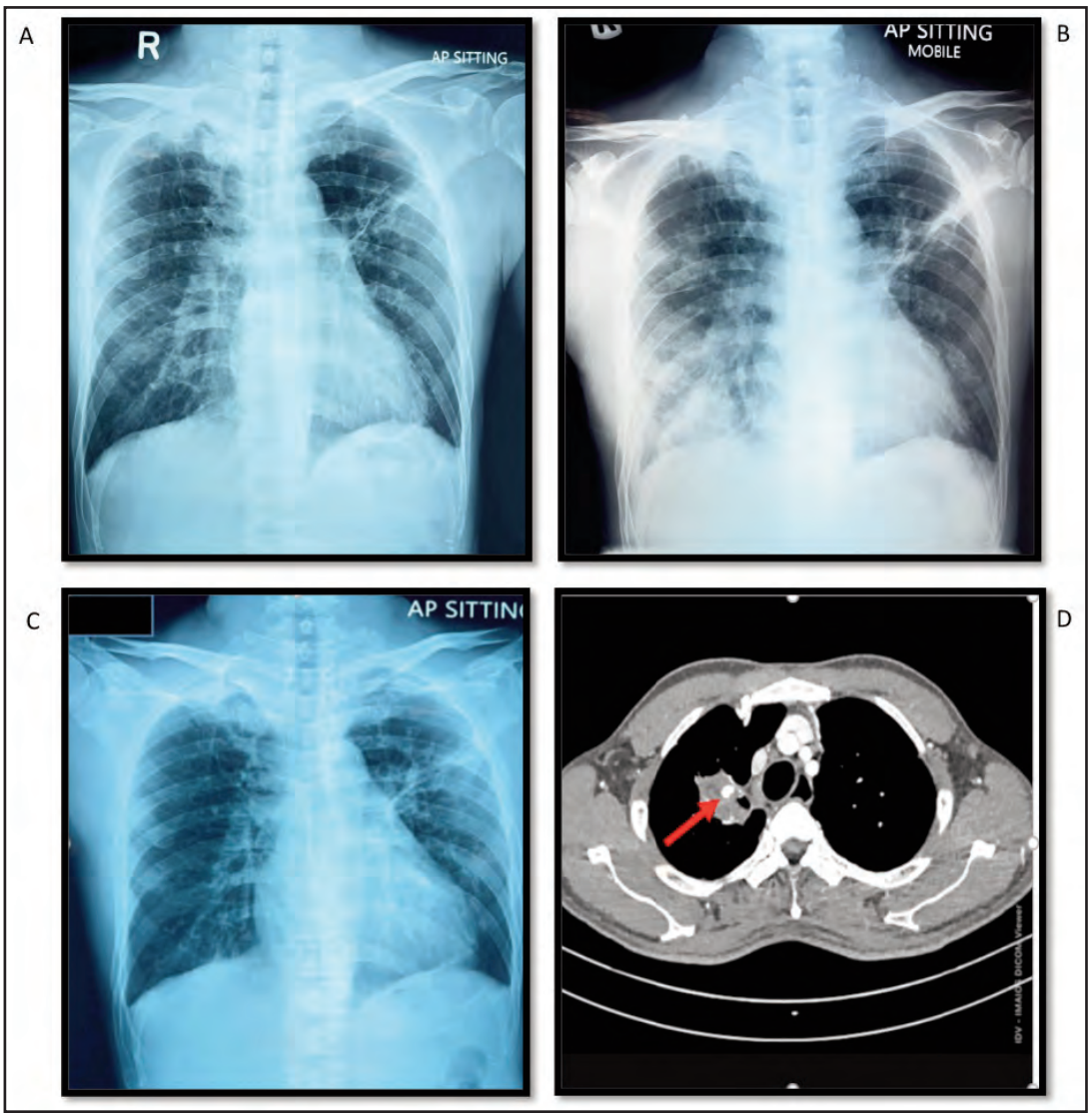

Fig. 1: Serial imaging of the chest demonstrating disease progression and response to treatment.
(A) Initial chest radiograph (CXR) at first presentation shows right upper zone haziness.
(B) CXR during current admission reveals persistent right upper zone haziness with new right lower zone consolidation.
(C) CXR on Day 5 of admission demonstrates radiological improvement following intravenous Ceftazidime therapy.
(D) Axial CT thorax angiography identifies an arterial enhancing lesion (0.6 × 0.9 × 0.6 cm, AP × W × CC), consistent with an aneurysm arising from a segmental branch of the right lobar pulmonary artery.

There was haziness in the right upper zone on the Chest radiograph (Figure. IA) which raised the concern for TB reactivation. Empirical amoxicillin-clavulanate was initiated. TB work-up (three sputum AFB smears, GeneXpert, culture), Mantoux test, HIV, HBsAg, and HCV was negative. After 5 days, he was discharged stable and observed at CKD clinic.

Three months following his initial presentation, the patient re-presented with fever (39.0 °C), productive cough, and bilateral lower limb oedema. He appeared septic but remained alert. He was tachypneic (respiratory rate 22 breaths per minute) and maintained a SpO_2 of 96% on a 60% Venturi mask. Physical examination revealed bilateral basal crepitations, more prominent on the right, along with minimal pitting pedal oedema. Chest radiography (Figure 1B) showed right lower zone consolidation and right hilar prominence. Differential diagnoses at admission included community-acquired pneumonia, reactivation of pulmonary tuberculosis, and bronchogenic carcinoma.

Empirical treatment was initiated with intravenous amoxicillin-clavulanate, basal-bolus insulin, felodipine, antipyretics, cough suppressants, oral sodium bicarbonate, and pantoprazole. Given the presence of bilateral lower limb oedema and advanced chronic kidney disease, intermittent low-dose intravenous frusemide was administered for volume control with prompt clinical and radiographic response. Blood cultures obtained on day 1 grew *Burkholderia pseudomallei*, sensitive to ceftazidime, confirming a diagnosis of pulmonary melioidosis. Amoxicillin-clavulanate was discontinued, and renal dose-adjusted IV ceftazidime (2 g

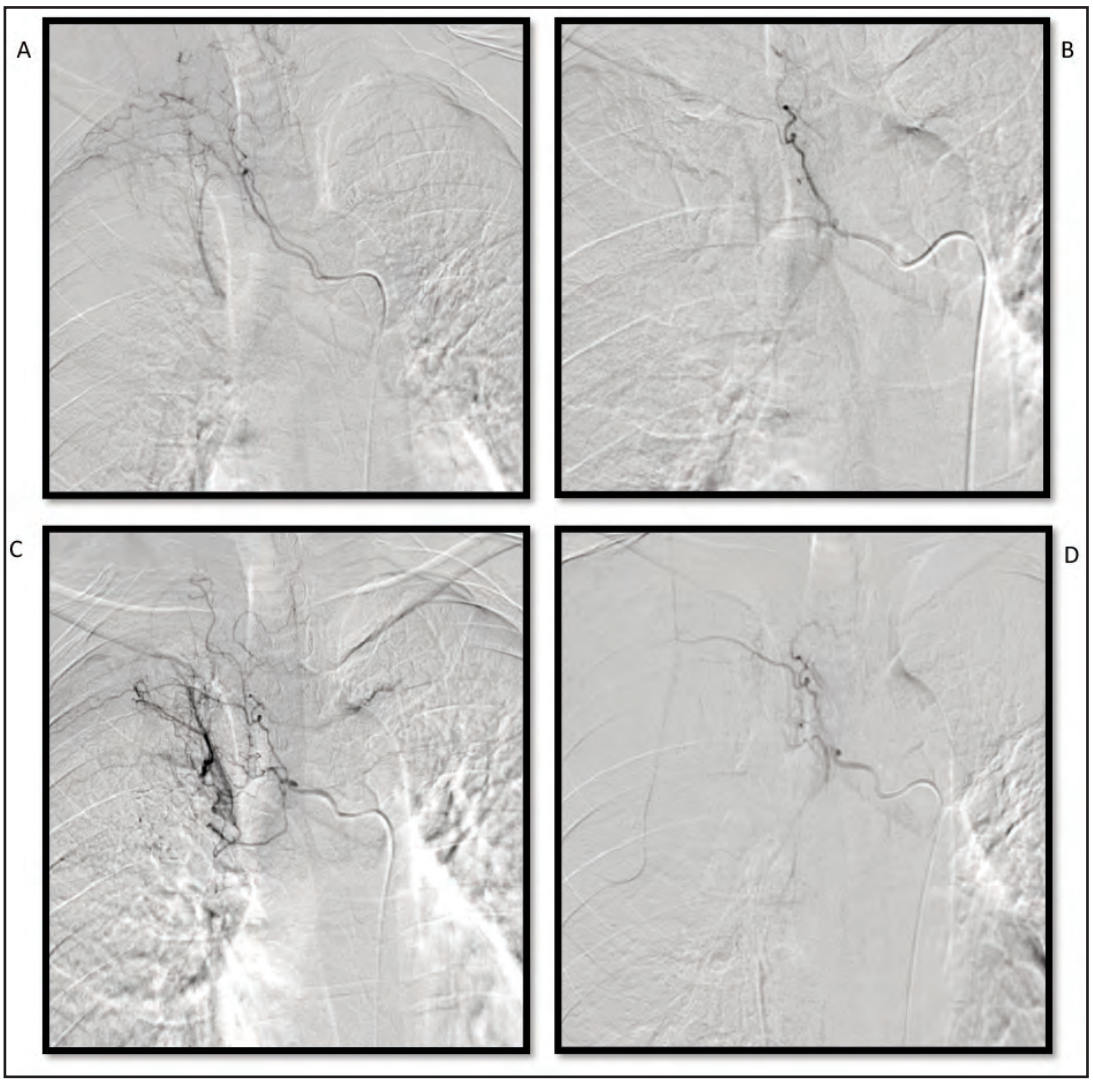

Fig. 2: Angiographic images demonstrating pre- and post-embolization findings of the right superior intercostal and right intercostobronchial arteries.
(A) Right superior intercostal artery, pre-embolization – abnormal vascularity with evidence of shunting.
(B) Right superior intercostal artery, post-embolization – complete occlusion of the abnormal vascularity and shunting.
(C) Right intercostobronchial artery, pre-embolization – abnormal hypervascularity with shunting.
(D) Right intercostobronchial artery, post-embolization – complete occlusion of the abnormal hypervascularity and shunting.

once daily) was initiated on day 3 in accordance with the Darwin guidelines for melioidosis management.

By day 5, his respiratory status improved, allowing oxygen to be discontinued. A follow-up chest radiograph (Figure 1C) demonstrated significant radiological improvement. Abdominal ultrasound on day 6 showed no evidence of intra-abdominal abscesses. Formal transthoracic echocardiography was not performed, as there were no clinical features suggestive of overt cardiac failure, and the patient demonstrated rapid clinical and radiological improvement following antimicrobial therapy and volume optimisation.

On day 12, eradication therapy with oral trimethoprim-sulfamethoxazole (TMP-SMX) was commenced while intravenous ceftazidime was continued. TMP-SMX was discontinued within 24 hours due to hepatotoxicity and hyperkalaemia with metabolic acidosis. Supportive management with oral calcium polystyrene sulfonate and sodium bicarbonate was provided, and eradication therapy was deferred until clinical parameters had stabilised.

Despite clinical improvement of respiratory symptoms, and after TMP-SMX had already been discontinued, on day 14 of ongoing IV ceftazidime therapy (completing a 14-day intensive phase), the patient developed new-onset haemoptysis with a drop in haemoglobin from 8.3 to 7.6 g/dL, requiring transfusion of one unit of packed red blood cells. A CT thorax angiogram (Figure 1D) performed in week 4 revealed multiple cavitating lesions with surrounding consolidation in the apical segment of the right upper lobe. Notably, a 0.6 × 0.9 × 0.6 cm arterially enhancing lesion consistent with a Rasmussen aneurysm was seen, arising

from a branch of the right upper lobe pulmonary artery. There was no radiological evidence of tuberculosis reactivation, and chronic changes were attributed to prior TB infection.

Due to persistent haemoptysis and concern for aneurysmal rupture, the patient underwent successful bronchial artery embolization in week 5, performed by the Interventional Radiology team at Hospital Kuala Lumpur (Figure 2A–IID). Selective catheterization of the abnormal right intercostobronchial artery and right superior intercostal artery was performed, and the feeding vessel to the aneurysm was occluded with polyvinyl alcohol particles (350–500 µm), achieving complete stasis. Haemoptysis resolved immediately, and no post-procedural complications occurred.

The patient completed a total of 28 days of intravenous ceftazidime treatment in the ward. The prolonged intensive phase was chosen due to bacteraemia, cavitating pulmonary disease, and the development of a vascular complication. Following biochemical stabilisation and after discussion with the infectious disease team at HTAA Kuantan via remote consultation, eradication therapy was reinitiated on day 28 using oral amoxicillin-clavulanate (1875 mg once daily) as a temporary bridging alternative pending biochemical stabilisation.

One week later, the patient experienced deterioration in renal function and hemodialysis was initiated via a right internal jugular catheter. This intervention resulted in normalisation of serum potassium and correction of metabolic acidosis, which allowed the safe reintroduction of TMP-SMX as eradication therapy (Table. I). The patient subsequently completed a full 3-month course of eradication therapy with TMP-SMX without further complications. At follow-up, he remained clinically stable and asymptomatic, with no signs of disease recurrence.

DISCUSSION

Melioidosis, caused by Burkholderia pseudomallei, is endemic to Southeast Asia and northern Australia. Pneumonia and bacteraemia are among its most frequent clinical manifestations.[5] In Malaysia, the disease disproportionately affects individuals with underlying comorbidities especially diabetes mellitus, present in over half of all cases, and chronic kidney disease (CKD), which is associated with a threefold increase in infection risk.[6]

Our patient carried several high-risk features, including poorly controlled diabetes, advanced CKD, and a past history of pulmonary tuberculosis. The combination of pneumonia and bacteraemia placed him at particularly high risk for complications. Local and regional studies have shown that bacteraemic melioidosis can carry mortality rates up to 65%, particularly when complicated by acute kidney injury.6 Both CKD and bacteraemia are known to independently increase the risk of AKI, which is strongly associated with ICU admission and higher mortality.[7]

Diagnosing pulmonary melioidosis can be challenging given its nonspecific symptoms and overlap with TB or malignancy. In our patient, early blood cultures were key to confirming the diagnosis and starting the right treatment, highlighting the importance of prompt cultures and clinical suspicion in endemic areas.

Although in melioidosis pneumonia and bacteraemia without a focus, adding trimethoprim-sulfamethoxazole has not been part of the initial intensive therapy, however, commencement during the intensive phase was necessitated in this case to enable monitoring of adverse events. Trimethoprim-sulfamethoxazole (TMP-SMX) remains the first-line agent for melioidosis eradication; however, its use in advanced CKD is complicated by risks of hyperkalaemia and hepatotoxicity, both of which our patient experienced early in the course.[3] This necessitated a temporary switch to amoxicillin-clavulanate as an alternative agent, albeit one associated with a higher relapse rate. Dosing, however, presents a clinical dilemma in patients with renal impairment.

Although standard renal dosing guidelines typically call for reducing amoxicillin-clavulanate in patients with CKD to minimise toxicity, the consensus statements by Cheng et al. and Chierakul et al. highlight a different priority in melioidosis ensuring adequate drug levels to effectively eradicate the infection, even if higher than standard doses are required.[8] A practical approach was adopted in this patient by administering 1875 mg once daily before dialysis to strike a balance between therapeutic efficacy and safety, particularly given the limitations of a district hospital setting. A particularly rare but life-threatening complication in this case was the development of a Rasmussen aneurysm, a pseudoaneurysm of a pulmonary artery branch adjacent to cavitary lung lesions. Though classically associated with tuberculosis, similar vascular lesions may occur in necrotizing pneumonias, including melioidosis. When ruptured, it can cause massive haemoptysis and carry a mortality rate exceeding 50%.[9] In this case, early detection via CT angiography and prompt bronchial artery embolization were lifesaving.

In Malaysia, melioidosis case fatality rates range from 33% to 54%, with delays in diagnosis and treatment often contributing to recurrence or death.6 Several poor prognostic indicators were identified in this patient, including bacteraemia, leukocytosis, chronic kidney disease, and metabolic acidosis, all of which have been associated with worse outcomes.[10] Despite this, early diagnosis, tailored antimicrobial therapy, and a flexible treatment plan helped avoid adverse outcomes.

This case highlights the complexity of managing melioidosis in patients with severe CKD, especially in resource-limited or district hospital settings. Antimicrobial adjustments, managing renal-related complications, and coordinating referrals to tertiary centres required close collaboration between internal medicine, infectious disease, nephrology, and interventional radiology teams. Remote consultation proved invaluable in decision-making when subspecialty support was not readily available on site. It highlights that

with careful monitoring and interdisciplinary coordination, even high-risk patients with multiple comorbidities can achieve favourable outcomes, even outside of tertiary academic centres.

This case highlights the multidimensional challenges of managing melioidosis in a patient with advanced chronic kidney disease and complex comorbidities in a district hospital setting. From diagnostic uncertainty to antimicrobial toxicity, and the rare complication of a Rasmussen aneurysm, this case demonstrates the importance of early microbiological confirmation, tailored therapy, and timely escalation of care. Despite resource limitations, coordinated collabouration across district and tertiary hospitals, including remote infectious disease consultation and interventional radiology, enabled a favourable outcome. In melioidosis-endemic regions, this case reinforces how timely clinical recognition, personalised antibiotic choices, and adaptable care approaches can make a critical difference for vulnerable patients with underlying CKD. Ultimately, the success of this case reflects not only medical management, but also the strength of interdisciplinary teamwork and continuity of care across levels of the healthcare system.

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Appendix

Table I: Selected Laboratory Parameters during Hospitalization

Biochemical and Hematological Parameters	Admission (Day 1)	TMP-SMX initiation (Day 12)	Post TMP-SMX toxicity	CTA thorax (Week 4)	Post BAE (Week 6)	Post Dialysis (Week 7)
Hemoglobin (g/dL)	8.3	8.0	7.6 ↓ after haemoptysis	8.3	8.4	8.9
White Blood Cell ×10³/µL	14.8	6.9	7.0	6.9	12.7	8.9
Platelets×10³/µL	320	241	236	193	130	183
Erythrocyte sendimentation rate (mm/HR)	>140	-				-
Serum urea (mmol/L)	19.8	30.3	31.0	33.4	39.2↑	12.0 ↓
Serum creatinine (µmol/L)	417 (eGFR ~14 mL/ min/1.730	445 (eGFR ~14 mL/ min/1.73)	443 (eGFR ~14 mL/ min/1.73)	499 (eGFR ~12 mL/ min/1.73)	869 ↑ (eGFR ~5.6 mL/ min/1.73)	450 ↓
Serum Potassium (mmol/L)	3.6	5.3	5.7 ↑	3.6	4.6	3.5
AST (U/L)	32	56	166 ↑	23		23
ALT (U/L)	16	53	120 ↑	19		29
CRP (mg/L)	117.3	51.65		5.0		
Microbiological Culture						
Blood culture	Positive for B. pseudomallei	Repeat cultures negative	-	-	-	-
Sputum AFB/ GeneXpert/ Culture	Negative	-	-	-	-	-
Other test: HIV, HBsAg, HCV	Negative	-	-	-	-	-
PH	7.369	-	7.325	7.25	7.10 ↓	7.30
Bicarbonate	19	-	15	14.3	8.3 ↓	18