Disseminated Melioidosis with Spinal Intraosseous Abscess

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SUMMARY

Melioidosis is endemic in the State of Sabah, Malaysia. We report a case of a 34-year-old man with one-week history of fever and cough, three days history of diarrhoea and vomiting, which was associated with a loss of appetite and loss of weight for one-month. Clinically, he had hepatosplenomegaly and crepitation over his right lower zone of lung. Chest radiograph showed right lower lobe consolidation. Ultrasound abdomen showed liver and splenic abscesses. Ultrasound guided drainage of splenic abscess yielded Burkholderia pseudomallei. Magnetic resonance imaging (MRI) lumbosacral confirmed right sacral intraosseous abscess after he developed back pain a week later. He received 6 weeks of intravenous antibiotics and oral co-trimoxazole, followed by 6 months oral co-trimoxazole and had full recovery.

INTRODUCTION

Melioidosis is an infection caused by the gram negative bacterium *Burkholderia pseudomallei*. It is endemic in Sabah, Malaysia. The important risk factors associated with melioidosis are diabetes mellitus (DE), chronic kidney disease, chronic lung disease, underlying immunosuppression, thalassaemia, and underlying malignancy. Melioidosis is usually acquired via percutaneous inoculation with contaminated soil or water, inhalation, aspiration, and ingestion. Here, we report a case of a male with underlying diabetes who had disseminated melioidosis involving the spine, lungs, multiple liver abscesses and splenic abscesses.

CASE REPORT

A 34-year-old man with underlying diabetes mellitus, presented at the Queen Elizabeth Hospital, Kota Kinabalu, Sabah, Malaysia with one-week history of fever and productive cough, three days history of diarrhoea and vomiting, and one-month history of loss of appetite and loss of weight. He worked as a security guard and did not engage in recreational activity involving soil or contaminated water. On examination, he was febrile with temperature 38.7 ^{0}C and was haemodynamically stable. He had hepatosplenomegaly and crepitations over his right lower zone of lung. Other clinical examinations were unremarkable. Results of relevant blood investigations are shown in Table I. His blood results showed hypochromic microcytic anaemia along with leucocytosis, hyponatremia and raised C-reactive protein. His random blood sugar was 16.7 mmol/L. Chest X-ray showed right lower lobe consolidation. Ultrasound abdomen showed a 10 cm x 9 cm heterogenous collection over the posterior pole of spleen and a heterogenous collection seen within segment III, VII, and VIII of the liver, with a largest 2.8 cm x 3.8 cm abscess seen at segment III of the liver.

He was treated empirically with intravenous Ceftazidime 2g every 8-hourly for melioidosis in view of underlying DE and because melioidosis is endemic in Sabah. His first blood culture was sterile. Ultrasound guided drainage of splenic abscess was done, and 30 ml of blood-stained pus was aspirated. Pus culture from splenic abscess grew *B.pseudomallei*, which was sensitive to Ceftazidime and Meropenem. His antibiotic was escalated to intravenous Meropenem 1g every 8-hourly in view of persistent fever and bacteraemia despite abscess drainage from the spleen. Contrasted CT thorax and abdomen showed multiple hypodense liver lesions of varying sizes (largest at segment IVa measuring 3.2 cm x 2.8 cm x 2.4 cm), multiple scattered heterogeneously enhancing hypodense splenic lesions (largest measuring 4.6 cm x 4.6 cm x 4.6 cm) and multiple lung nodules at apical segment of right upper lobe.

During the second week of admission, he developed lower back pain which was aggravated by walking and was tender on palpation. There was no neurological deficit. MRI lumbosacral showed right sacral intraosseous abscess (2.3 cm x 1.4 cm x 2.2 cm) with inflammation of the adjacent right sacral nerve root. A smaller intraosseous abscess was also seen in the superior endplate of S1 sacral body measuring 1.5 cm x 2.2 cm x 0.8 cm (Figure 1). Patient completed 6 weeks of intravenous Meropenem which was later de-escalated to intravenous Ceftazidime together with oral co-trimoxazole, followed by another 6 months of eradication phase with oral co-trimoxazole. Reassessment scan with MRI lumbosacral after intensive phase treatment showed resolving right sacral intraosseous abscess (1.4 cm x 0.6 cm x 0.8 cm) and sacral body of S1 abscess (1.3 cm x 1.0 cm x 0.5 cm)(Figure 2). He made full recovery and was well in 2020, two years after discharge.

DISCUSSION

Melioidosis is endemic in Southeast Asia and northern Australia.[1] In Sabah, the incidence rate of melioidosis is 2.7

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Table I: Blood Investigations

Investigation	Day 1 of admission	After complete treatment	Normal range
Haemoglobin (g/dl)	11.4	16.8	13 -17
White cell count (10 ^3/uL)	11.9	6.9	4 -10
Neutrophil (10^3/uL)	10.6	4.3	2 -7
Platelet (10^3/uL)	276	185	150 – 410
CRP (mg/L)	184.4	1.1	< 5
Na (mmol/L)	112	134	136 -145
K (mmol/L)	3.6	4.2	3.5 – 5.1
Cl (mmol/L)	74	101	98 – 107
Urea (mmol/L)	2.6	5.4	3.2 – 7.4
Creatinine (umol/L)	73.4	109.7	63.6 – 110.5
Total protein (g/L)	68	85	
Total bilirubin (umol/L)	39.6	9.4	3.4 – 20.5
Albumin (g/L)	25	46	35 – 50
Globulin (g/L)	43	39	
ALP (U/L)	1085	108	40 – 150
ALT (U/L)	82	60	0 -55
AST (U/L)	154	45	5 – 34

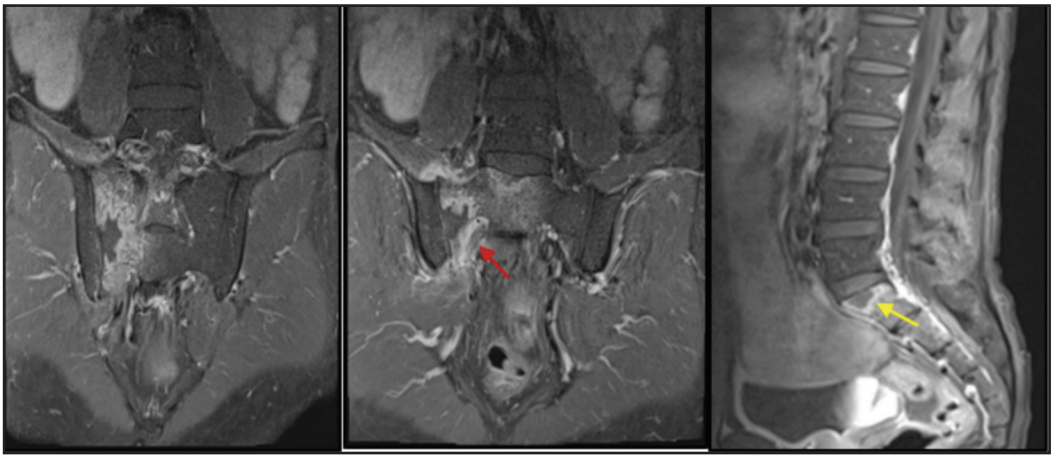

Fig. 1: Coronal T1WFS post contrast image shows a thick-walled abscess in the right sacral ala with minimal enhancement of the adjacent right sacral nerve (red arrow). Another smaller intraosseous collection noted in the superior end plate of S1 sacral body (yellow arrow).

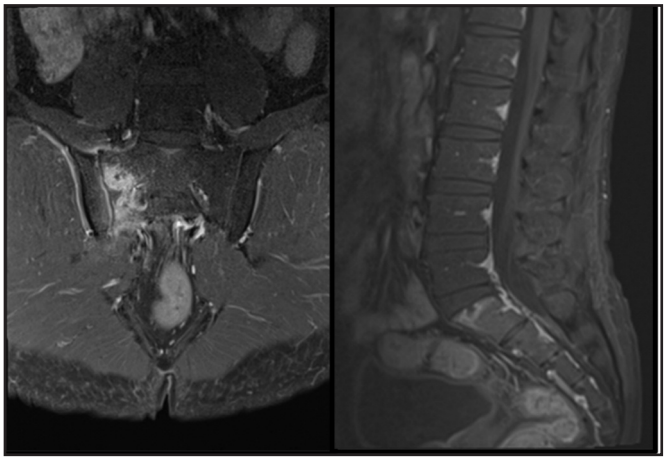

Fig. 2: Coronal T1WFS post contrast image at 1 month follow up shows marked reduction in size of the abscess in both right sacral ala and superior end plate of S1 sacral body.

per 100,000 population.[2] Melioidosis can be acquired via percutaneous inoculation with contaminated soil or water, inhalation, aspiration, and ingestion.[3] Patients with risk factors such as DE, chronic kidney disease, chronic lung disease, thalassemia, malignancy, immunosuppression, hazardous alcohol intake and advanced age are at risk of melioidosis infection. Occupational or recreational exposure involving contaminated soil and water are commonly associated as the infecting event.

Melioidosis can present with various clinical manifestations involving any sites, hence making this disease a great mimicker. Pneumonia is the most common clinical presentation.[4] Musculoskeletal involvement is a well-recognised manifestation of melioidosis. However, B.pseudomallei is a relatively rare causative organism in musculoskeletal infections.[5] Our patient had multiple organs involvement including liver, spleen, lungs, and sacral intraosseous abscesses. He had risk factor of DE, but no infecting event was identified. Development of spinal abscess could be secondary to haematogenous spread from another region, but sacral region involvement is rare.

Detailed history taking and risk factors assessment are important to make timely provisional diagnosis and to start empirical treatment. This is because few intravenous

antibiotics are effective to treat melioidosis during the intensive phase. These include intravenous Ceftazidime, Meropenem or Imipenem which are not usually used as first line antibiotics in community acquired pneumonia or sepsis. Culture isolation from patient bodily fluids remains the gold standard for diagnosis and is important to guide antimicrobial de-escalation therapy later. Delayed treatment of spinal abscess can lead to a permanent neurological deficit and even fatality. Thus, detailed clinical assessment, followed by prompt utilisation of MRI scan is necessary to avoid diagnostic delay in patient who presents with musculoskeletal symptoms and signs. Large deep-seated spinal abscess needs to be drained for good source control.

Antibiotics treatment of melioidosis consists of two phases of therapy, which are the induction phase and the eradication phase. During the initial intensive phase, either intravenous Ceftazidime or Meropenem or Imipenem is used. Oral co-trimoxazole is combined early with intravenous antibiotics in skin and soft tissue infection, septic arthritis, osteomyelitis, prostatic infection, and central nervous system infection in view of good tissue penetration. After successful intensive phase treatment, oral co-trimoxazole is continued as the eradication phase therapy. The duration of both the intensive phase and eradication phase therapy depends on the anatomical organ involvement of the disease and the severity of infections.

CONCLUSION

Melioidosis is a serious neglected tropical disease and is endemic in Sabah, Malaysia. Early recognition and source identification are particularly important for successful treatment of patient. Surgical intervention is often useful in large deep-seated abscess to achieve good source control. Good source control together with appropriate antibiotics and adequate duration of antibiotics are required to achieve successful patient outcome.

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