

AN UNUSUAL PRESENTATION OF POLYSEROSITIS

Dr. Harini G. L.* and Dr. Nagaraj B. S.

India.

*Corresponding Author: Dr. Harini G. L.

India.

Article Received on 13/10/2016

Article Revised on 03/11/2016

Article Accepted on 23/11/2016

INTRODUCTION

Melioidosis or Whitmore's disease is an infectious saprophytic disease caused by *Burkholderia Pseudomallei*. The disease is most prevalent in South East Asia and Northern Australia. Thailand has the maximum reported cases.^[1] In India, it is more prevalent in the South Western coast; however, there are no data on its prevalence and most of the cases go unrecognized.^[2] The disease is transmitted via exposure of broken skin to contaminated soil, particularly wet paddy fields. Other mode of transmission is via aerosols.

Important risk factors for the disease are occupational exposure, i.e., agriculturist, type 2 diabetes mellitus, immune deficiency state and renal disorder.

Clinical features of melioidosis are highly variable. They range from asymptomatic disease, localized skin ulcers or abscesses and acute fulminant septicaemia to chronic infection. Predisposing factors for melioidosis include chronic diseases such as diabetes, chronic renal failure, chronic lung disease and alcoholism.^[1-3] Timely diagnosis and prompt institution of correct antibiotics will prevent high morbidity and mortality.

CASE REPORT

We present a case of a 52 year old female, from Assam who was a known diabetic from past 15 years on treatment with oral hypoglycemics and no other co morbidities. She presented to us with history of fever from 10days, which was high grade intermittent and associated with breathlessness and distention of abdomen, but there was no history of cough with expectoration or chest pain. No H/o decreased urinary output or No H/o loss of weight.

On examination, she was febrile with temperature of 100.2f, tachypnoeic with respiratory rate of 32/min and tachycardia was present with heart rate of 108/min. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy or pedal edema.

Respiratory system examination was significant in the form of absent breath sounds in bilateral infra axillary and infra scapular areas but no added sounds and per abdomen showed mild hepatosplenomegaly and evidence of free fluid. Other system examination was normal.

Investigations showed increased total count of 13,600cells/cumm with Neutrophilic predominance of 80% and elevated ESR of 92. Her RBS was 206 with HbA1C of 7.6. Her renal and hepatic functions were normal.

Her chest x ray was done which showed bilateral moderate pleural effusion.



USG abdomen showed Hepatosplenomegaly with moderate ascites.

Ascitic fluid analysis was done which was exudative with Neutrophilic predominance. Protein: 4.8g/dl, Glucose: 108mg/dl, Cells: 316, Morphology: 70% neutrophils, ADA: 4.8

Pleural fluid analysis was also done which was also exudative with Neutrophilic predominance. Cells: 1100, predominant neutrophils, Protein: 4.6g/dl, Glucose: 92mg/dl, ADA: 9, Pleural fluid gram stain showed gram

negative bacilli and Culture after 48hrs of incubation grew *B.pseudomallei*.

She was started on intravenous ceftazidime 2g 8th hourly. she became symptomatically better and was afebrile by 10th day of admission. She was discharged after 2 weeks of antibiotics. Repeat USG after 2 weeks showed minimal ascites with mild splenomegaly.

Repeat chest x ray



DISCUSSION

Melioidosis is endemic in Southeast Asia and northern Australia. In parts of northeastern Thailand, it is the most common cause of severe community-acquired sepsis.¹ The first reported case in the northern territory of Australia occurred in 1960 and melioidosis is now recognized as the most common cause of fatal community-acquired bacteremic pneumonia.^[1] In several published series, pneumonia is the most frequent presentation of melioidosis and is involved in approximately half of all cases.^[1-3]

In our report, co-morbidity, and age were most relevant. *B. pseudomallei* is visualized as a slender Gram-negative bacillus with rounded ends and bipolar staining, and are vacuolated; it is often described as having a safety-pin appearance. It is oxidase positive, and on culture, the organisms demonstrate different colony morphology.¹

In this case, on Gram stain the pathogen was observed as Gram-negative bacilli. Culture in blood agar yielded a small, dry, earth-smelling colony.

Isolation of *B. pseudomallei* from the body fluids of patients remains the gold standard in the diagnosis and requires the use of selective media for non-sterile specimens. Gram stain and other histopathological stains are not specific for the organism.

Skin and soft tissue infection is a common manifestation of Melioidosis, and may result from hematogenous spread and can be the source of systemic infection. Presentation may be of rapid progression, similar to necrotizing fasciitis. Bone and joint infections are uncommon (4–12%) and may be difficult to diagnose, and systemic manifestations are prominent. Surgical drainage and long courses of antibiotics are required.¹⁻⁵

In our case, the patient developed polyserositis without involvement of lung parenchyma which is rare in reported cases of Melioidosis Currie et al. reported seven cases of septic shock with high mortality (100%).^[2] Regarding other non-bacteremic forms; they reported five cases of osteomyelitis and septic arthritis with no mortality (0%). In published series from Thailand and Australia, other frequent presentations have been suppurative parotitis, prostatic abscesses, urinary infection and encephalomyelitis syndrome.^[3,5]

Several randomized, controlled trials have examined intensive phase interventions in severe Melioidosis and are the basis of ceftazidime-based regimens.^[1,5] Ceftazidime has been shown to be of benefit in the treatment of this disease in a sequential open-label randomized trial of ceftazidime against chloramphenicol–doxycycline–trimethoprim–sulfamethoxazole (known as conventional therapy) in severe disease, at least for 6 months.¹ Other trials have used amoxicillin–clavulanate and imipenem, granulocyte colony stimulating factor (G-CSF) and activated protein C in severe cases of sepsis.¹ For maintenance therapy, amoxicillin–clavulanate vs. chloramphenicol, doxycycline, trimethoprim–sulphamethoxazole and ciprofloxacin have been recommended, with a duration of 12–20 weeks.^[1]

CONCLUSIONS

Melioidosis is a common disease but rarely suspected and detected. Especially in tropical countries like India. In our case the associated co morbidity diabetes was one of the predisposing factors. It s rare presentation in the form of high fever and polyserositis without parenchymal pulmonary involvement is one of the interesting things to be noted in our case.

Also, since the relapse rate and mortality is high with this disease, there is a need for early diagnosis and treatment particularly in India where it is under diagnosed.

REFERENCES

1. Chaowagul W, Suputtamongkol Y, Dance DA, Rajchanuvong A, Pattara-arechachai J, White NJ. Relapse in melioidosis:incidence and risk factors. *J Infect Dis.*, 1993; 168: 1181-5.
2. Sanford JP. *Pseudomonas* species (including melioidosis & glanders). In: Mandell GL, Bennett JE, Dolin R, editors. *Principles and Practice of Infectious Diseases*. 4th ed. New York: Churchill Livingstone, 1995; 2003-9.
3. Suputtamongkol Y, Hall AJ, Dance DA, et al. The epidemiology of melioidosis in Ubon Ratchatani, northeast Thailand. *Int J Epidemiol*, 1994; 23: 1082-90.
4. Bharadwaj R, Kagal A, Deshpandey SK, et al. Outbreak of plague-like illness caused by *Pseudomonas pseudomallei* in Maharashtra, India. *Lancet*, 1994; 344: 1574.

5. So SY, Chau PY, Leung YK, Lam WK, Yu DY. Successful treatment of melioidosis caused by a multiresistant strain in an immunocompromised host with third generation cephalosporins. *Am Rev Respir Dis.*, 1983; 127: 650-4.
6. So SY, Chau PY, Aquinas M, Gabriel M, Lam WK. Melioidosis: a serological survey in a tuberculosis sanatorium in Hong Kong. *Trans R Soc Trop Med Hyg.*, 1987; 81: 1017-9.
7. National Committee for Clinical Laboratory Standards (NCCLS). Performance standards for antimicrobial disk susceptibility tests. NCCLS document M18-S9. Pennsylvania: National Committee for Clinical Laboratory Standards, 1999.
8. Ip M, Osterberg LG, Chau PY, Raffin TA. Pulmonary melioidosis. *Chest*, 1995; 108: 1420-4.
9. Suputtamongkol Y, Chaowagul W, Chetchotisakd P, et al. Risk factors for melioidosis and bacteremic melioidosis. *Clin Infect Dis.*, 1999; 29: 408-13.
10. Fierer J. *Pseudomonas* and *flavobacterium*. In: Braude AI, Davis CE, Fierer J, editors. *Infectious diseases and medical microbiology*. 2nd ed. Philadelphia: W.B. Saunders Company; 1986; 314-9.
11. Chaowagul W. Melioidosis; a treatment challenge. *Scand J Infect Dis Suppl*, 1996; 101: 14-6.
12. Suputtamongkol Y, Rajchanuwong A, Chaowagul W, et al. Ceftazidime vs. amoxicillin/clavulanate in the treatment of severe melioidosis. *Clin Infect Dis*, 1994; 19: 846-53.
13. Rajchanuvong A, Chaowagul W, Suputtamongkol Y, Smith MD, Dance DA, White NJ. A prospective comparison of co-amoxiclav and the combination of chloramphenicol, doxycycline and cotrimoxazole for the oral maintenance treatment of melioidosis. *Trans R Soc Trop Med Hyg*, 1995; 89: 546-9.
14. Chaowagul W, Suputtamongkol Y, Smith MD, White NJ. Oral fluoroquinolones for maintenance treatment of melioidosis. *Trans R Soc Trop Med Hyg*, 1997; 91: 599-601.
15. Walsh AL, Smith MD, Wuthiekanun V, White NJ. Postantibiotic effects and *Burkholderia* (*Pseudomonas*) *pseudomallei*: evaluation of current treatment. *Antimicrob Agents Chemother*, 1995; 39: 2356-8.
16. Limmathurotsakul D, Wongratanacheewin S, Teerawattanasook N, Wongsuvan G, Chaisuksant S, Chetchotisakd P, et al. Increasing incidence of human melioidosis in Northeast Thailand. *Am J Trop Med Hyg*, 2010 Jun; 82(6): 1113-1117.
17. Vidyalakshmi K, Shrikala B, Bharathi B, Suchitra U. Melioidosis: an underdiagnosed entity in western coastal India: a clinico-microbiological analysis. *Indian J Med Microbiol*, 2007 Jul; 25(3): 245-248.
18. Raja NS, Ahmed MZ, Singh NN. Melioidosis: an emerging infectious disease. *J Postgrad Med*, 2005 Apr-Jun; 51(2): 140-145.
19. White NJ. Melioidosis. *Lancet*, 2003 May; 361(9370): 1715-1722.
20. Marin M, Marty F, Cortesia M, Herrera L, Vainrub B. Melioidosis pulmonary adquirida en Venezuela. Poster presentation at the IX Jornadas Nacionales de Infectología "Dr. Juan Guido Tata' Cumana", San Cristóbal, Venezuela, November 1995; 22-24.
21. Douglas MW, Lum G, Roy J, Fisher DA, Anstey NM, Currie BJ. Epidemiology of community-acquired and nosocomial bloodstream infections in tropical Australia: a 12-month prospective study. *Trop Med Int Health*, 2004; 9: 795-804.