of suspicious lesions or adjacent lymph nodes may help in prompt diagnosis. BTB usually masquerades as cholangiocarcinoma and a prompt diagnosis helps to avoid major surgery and its ensuing morbidity. When in doubt a diagnostic laparoscopy with biopsy of suspicious lesions can be attempted. Frozen section biopsy prior to resorting to major surgical resections is a useful armamentarium when BTB is suspected. All surgically removed gall bladders must be carefully inspected followed by histopathological examination of suspicious foci. Post operative persistence of symptoms must not be branded as Post Cholecystectomy Syndrome and BTB must be considered in the appropriate setting. Treatment of BTB is medical therapy along with interventional endoscopic procedures to relieve biliary obstruction. Patients treated for TBS should be followed up long term for timely detection and prompt treatment of late strictures prior to the development of secondary biliary cirrhosis.

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References

1. Xu X-F, Yu R-S, Qiu L-L,et al. Gallbladder Tuberculosis: CT Findings with Histopathologic Correlation. *Korean Journal of Radiology*. 2011;12(2):196-202.
2. Padhiari RK, Ramesh M., G.P P, Ahmed N. Tuberculosis Biliary Stricture Simulating as Cholangiocarcinoma. Journal of Clinical and Diagnostic Research : *JCDR*. 2015;9(3):PL01-PL02.
3. Epstein D, Mistry K, Whitelaw A, Watermeyer G, Pettengell KE.The effect of physiological concentrations of bile acids on in vitro growth of Mycobacterium tuberculosis. *SAfr MJ*. 2012;102(6):522-4.
4. S Cheddie, T Bisetty, B Singh. Isolated Tuberculosis of the Gallbladder. *Internet J Surgery*. 2012 Volume 28 Number 2.
5. Sharma K, Sinha SK, Sharma A, et al. Multiplex PCR for Rapid Diagnosis of Gastrointestinal Tuberculosis. *Journal of Global Infectious Diseases*. 2013;5(2):49-53. doi:10.4103/0974-777X.112272.

A case of fatal septicaemic melioidosis from Odisha

Melioidosis, caused by Gram negative environmental saprophyte *Burkhloderia pseudomallei*, a disease of public health importance in Southeast Asia and Northern Australia, of late has shown an increasing trend in India, particularly South India.[1] Intravenous ceftazidime remains the drug of choice in the Intensive phase.[2] We report a fatal case of persistently septicaemic melioidosis that failed to respond to intravenous ceftazidime despite in vitro susceptibility.

Case Report

A 42 years old man, hailing from Puri district of Odisha was referred to the emergency department of our hospital with high grade fever and worsening abdominal pain in the left hypochondrium for last one month. The patient was a known alcoholic and his past medical history was insignificant except a recently diagnosed Type II Diabetes mellitus. On admission, physical examination revealed a temperature of 104.5°F, BP of 102/60 mm of Hg, a respiratory rate of 130/min, and oxygen saturation was 95% in room air. He was pale, icteric and dehydrated. Examination of the abdomen revealed hepatosplenomegaly. Crepitation was present on left infrascapular region. Laboratory evaluation showed anaemia, Hb 8gm/dL, random plasma glucose of 253 mg/dl

and raised inflammatory indexes as follows: white blood cells at 14100 cells per µL, 83% neutrophils,15% lymphocytes, erythrocyte sedimentation rate 125 mm/ first hr in association with deranged liver function tests (Serum bilirubin total 1.6 mg/dl, (direct) 1.20 mg/dl, (Indirect) 0.40 mg/dl, SGOT=124 U/L, SGPT=65 U/L, Alkaline phosphatase 284 U/L. Renal function tests were within normal limits [Urea=30 mg/dl, Creatinine 1mg/dl]. Malaria, enteric fever, viral hepatitis were ruled out. Peripheral smear revealed normocytic normochromic anaemia with presence of target cells, tear drop cells, moderate anisocytosis. USG of the abdomen and pelvis revealed splenomegaly with multiseptate lesion measuring 10x12 cm, and hepatomegaly with decreased echogenicity of liver. Chest X-ray revealed alveolar opacities in the left side of the lungs. A provisional diagnosis of sepsis/hepatitis/anaemia/hypoproteinaemia was made and patient was started on I.V Ceftazidime 1 gm BD after sending blood, sputum, and urine cultures. CECT of the abdomen revealed extensive involvement of entire parenchyma of moderately enlarged spleen with innumerable abscess cavities. Few of the abscesses were giving way near greater curvature of stomach and pancreatic tail likely due to thin wall. (**Figure 1**) Bilateral lung parenchyma showed multiple well defined nodules likely representing infecting seedling with a larger pleural based deposit in right lung lower lobe posterior basal segment. First blood culture sent on the day of admission was sterile up to 72 hours of incubation and was positive after 5 days of incubation. Gram negative coccobacilli exhibiting bipolar staining [Safety pin appearance] (**Figure 2**) were seen in the smear made from positive blood culture bottle. On subculture, small, smooth circular non-hemolytic colonies were obtained on sheep blood agar on 24 hours of incubation, which later changed to large, silver white, dry, wrinkled with central umbonation. (**Figure 3**) Pale pink colonies on Mac Conkey agar was obtained overnight which turned dry and wrinkled on day 4. (**Figure 3**) The isolate was motile, oxidase positive, had a neutral- alkaline reaction on triple sugar iron agar, grew at 42°C, Arginine dihydrolase positive and was resistant to colistin and gentamicin. Isolate identification was also confirmed by Vitek 2 system (Bio Merieux, France). The isolate was sensitive to Ceftazidime, Imipenem,

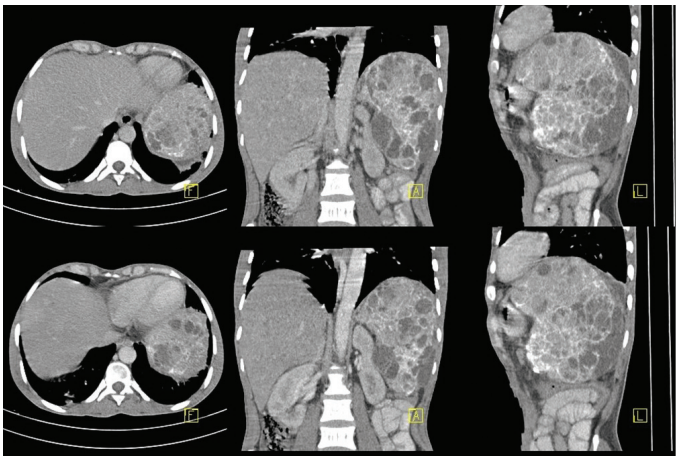

Figure 1: Extensive involvement of entire splenic parenchyma of moderately enlarged spleen with innumerable abscess cavities. Few of the abscesses are giving way near greater curvature of stomach and pancreatic tail, likely due to thin wall.

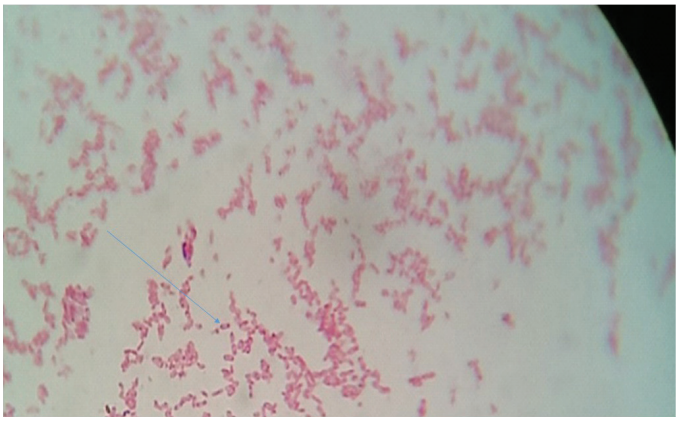

Figure 2: Gram negative coccobacilli exhibiting bipolar staining, safety pin appearance.

Figure 3: Large, flat , dry , wrinkled, colonies on sheep blood agar (left) with central umbonation at 96 hours of incubation. Pale pink colonies on MacConkey agar (right).

Meropenem, Co-trimoxazole and Amoxicillin-clavulanic acid by disc diffusion technique. Ceftazidime MIC [by E test, (Bio Merieux, France] of the 1st and 2nd blood isolates were 3µg and 8 µg respectively [S≤8, I=16, R ≥32 µg/ml]. (**Figure 4**)

Despite treatment with I.V ceftazidime and other supportive therapy, patient continued to remain in a state of sepsis [with TLC 29.35x10^3/µl on 7th day of I.V antibiotics] and developed rupture of abscesses to peritoneal cavity. I.V meropenem was added on 5th day and patient had to be taken for emergency splenectomy after obtaining high risk surgery consent. About 200 ml of frank pus was present in the perisplenic area. It was aspirated and sent for culture. (**Figure 5**) A second blood culture sent on day 5 of IV Ceftazidime as well as intra-operative pus yielded pure growth of *B. pseudomallei*. The patient expired on the 2nd post-operative day due to sepsis and cardiac arrest.

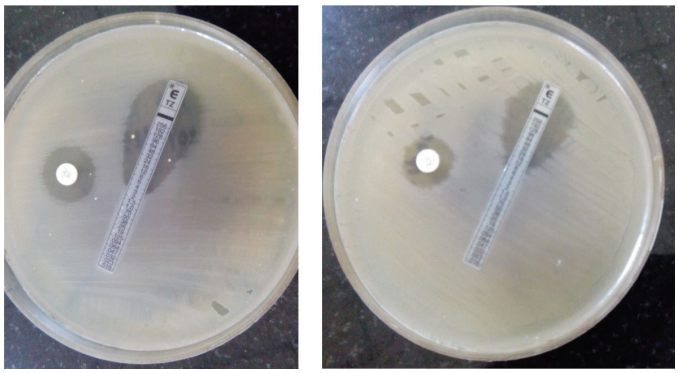

Figure 4: **Ceftazidime MIC 3µg/ml, 1st blood Isolate (left) Ceftazidime MIC 8µg/ml, 2nd blood isolate (right).**

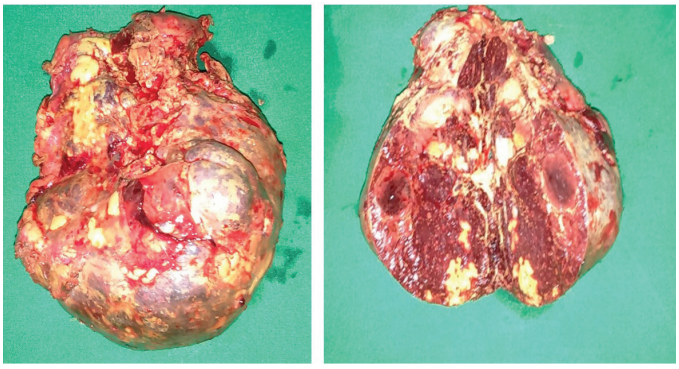

Figure 5: **Multiple abscesses in the spleen.**

Discussion

We want to emphasize on three important aspects, early clinical suspicion, prompt laboratory diagnosis and adequate intensive phase therapy. Our patient was diabetic and was alcoholic, which are two important risk factors for melioidosis.[2] Firstly, as majority of rural population in Odisha work in rice paddy fields, and the incidence of diabetes is also increasing, the possibility of melioidosis should be kept in mind in diabetic patients presenting with community acquired sepsis, pneumonia, soft tissue and internal organ abscesses.

Secondly, as the culture of *B. pseudomallei* from any clinical sample is the sine qua non for the diagnosis of melioidosis,[2] use of continuously monitoring blood culture systems can expedite the recovery from blood cultures. For samples other than blood, prolonged incubation of culture plates at 35°-37°C for up to 5-7 days is required. A delay in the identification of *B. pseudomallei* or a misidentification as another species is not uncommon in laboratories.[3] Therefore, it is strongly recommended that any non-*Pseudomonas aeruginosa*, oxidase positive, gram-negative bacillus isolated from any clinical specimen should be suspected to be *B. pseudomallei*.[3] *B. pseudomallei* is typically resistant to aminoglycosides (e.g., gentamicin), colistin, and polymyxin and this helps in identification.[3]

Thirdly, failure to respond to ceftazidime in our case despite in vitro susceptibility is a cause of huge concern. Rising ceftazidime MIC from 3µg to 8 µg [between the 1st and 2nd blood isolates], though still in the sensitive range, within one week of therapy is also noteworthy. Similar finding of emergence of ceftazidime resistance during therapy and variations in ceftazidime susceptibilities was also reported earlier by Chia Te Kung et al.[4] and Sam et al[5] Reports of high mortality (35%) of melioidosis patients treated with ceftazidime to which the isolates were susceptible, highlights a role of Carbapenems in the intensive phase.[6] There are recent reports of ceftazidime resistance in Indian *B. pseudomallei* also and requires active monitoring.[7,8]

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References

1. Gopalakrishnan R, Sureshkumar D, Thirunarayan MA, Ramasubramanian V. Melioidosis: an emerging infection in India. *J Assoc Physicians India*. 2013;61:612-4.
2. Wiersinga WJ, Currie BJ, Peacock SJ.Melioidosis. *N Engl J Med*. 2012;367:1035-44.
3. Hoffmaster AR, AuCoin D, Baccam P, Baggett HC, Baird R, Bhengsri S et al. Melioidosis diagnostic workshop, 2013. *Emerg Infect Dis*. 2015;21(2).
4. Kung CT, Lee CH, Li CJ, Lu HI, Ko SF, Liu JW. Development of ceftazidime resistance in Burkhoderiapseudomallei in a patient experiencing melioidosis with mediastinal lymphadenitis. *Ann Acad Med Singapore*. 2010;39:945-3.
5. Sam IC, See KH, Puthucheary SD. Variations in ceftazidime and amoxicillin-clavulanate susceptibilities within a clonal infection of Burkholderiapseudomallei. *J Clin Microbiol*. 2009;47:1556-8.
6. Simpson AJ, Suputtamongkol Y, Smith MD, Angus BJ, Rajanuwong A, Wuthiekanun V et al. Comparison of imipenem and ceftazidime as therapy for severe melioidosis. *Clin Infect Dis*. 1999;29:381-7.
7. Behera B, Prasad Babu TL, Kamalesh A, Reddy G. Ceftazidime resistance in Burkholderiapseudomallei: first report from India. *Asian Pac J Trop Med*. 2012:5;329-30.
8. Karuna T, Khadanga S, Dugar D, Sau B, BhoiP.Melioidosis as a cause of acute abdomen in immuno-competent male from eastern India. *J Lab Physicians*. 2015 7:58-60.