Catheter associated mycobacteremia: Opening new fronts in infection control

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**Abstract**

*Mycobacterium fortuitum* is a rapidly growing *Mycobacterium* ubiquitous in nature, known to form biofilms. This property increases its propensity to colonize the *in situ* central line and makes it a prospective threat for nosocomial infection. We report a case of 48-year-old female with carcinoma cecum who reported to us with clinical illness and neutropenia while on chemotherapy via totally implanted central venous device, postlaparoscopic-assisted right hemicolectomy.

**Keywords:** Biofilm, chemo port, mycobacteremia, *Mycobacterium fortuitum*, totally implanted central venous device

**Introduction**

Central venous catheter (CVC)-related bloodstream infection is associated with significant mortality and morbidity in critically ill patients, much of the focus, however, has remained on the bacterial pathogens. Catheter-associated mycobacteremia is a rare entity, even in oncology patients undergoing chemotherapy via a totally implanted central venous device (TID), its diagnosis starts with disbelief and is followed up with enormous diagnostic and clinical challenge. It stares the clinician with the myriad treatment options antimicrobial regimen, duration of therapy, and CVC management, remains unclear. Catheter-associated mycobacteremia is usually caused by rapidly growing Mycobacteria. They are ubiquitous in nature and can be found in soil, bioaerosols; they are known to form biofilms in lungs, a property conducive to colonizing central venous device (TID) and subsequently causing infections.

**Case Report**

A 48-year-old diabetic female was admitted to our hospital with a history of fever with chills for past 10 days. A known case of carcinoma caecum, she underwent a laparoscopic-assisted right hemicolectomy 6 months back. Thereafter she was put on chemotherapy regimen FOLFOX (Folinic acid), fluorouracil (5-FU) and oxaliplatin of which she has already received three cycles.

Her total leukocyte count (TLC) was $2.5 \times 10^9/L$, differential leukocytes count-N45, L50, and M5. A provisional diagnosis of febrile neutropenia was made, and growth factor was given following which TLC increased, but the fever still persisted.

X-ray chest revealed chemo port tip in the right atrium. An echocardiography was confirmatory. To rule out fever due to embolization prothrombin time and international normalized ratio were measured, and were found to be normal. So, a full-fledged fever work up was done, in view of the immunocompromised state of the patient. All the tests, urine R/E, urine culture, malarial antigen, Dengue NS1 and IgM, Widal, Anti HAV IgM, Anti HEV IgM, cytomegalovirus IgM, anti Epstein–Barr virus (viral capsid antigen) IgM, Anti-herpes simplex virus (1 and 2) IgM; were negative.

Blood cultures were also sent, on 4th day the blood sample which was taken from the chemo port flashed positive in BacTAlert, a Gram-stain revealed irregularly staining Gram-positive bacilli [Figure 1]. An Erlich-Ziehl-Neelsen
stain was done, and acid-fast bacilli were seen [Figure 2]. Subcultures were done on blood agar, MacConkey agar without crystal violet, LJ media, Middlebrook 7H9 broth in mycobacteria growth indicator tube.

Three days later pale and opaque colonies grew on blood agar, which acquired yellowish-pink tinge on further incubation [Figure 3]. Acid-fast bacilli were seen on Erlich-Ziehl-Neelsen staining. A MPT64 antigen assay was negative.

Upon the suspicion of a rapidly growing nontuberculous Mycobacterium (RGM) spp. the isolate was sent to a reference NABL accredited private lab for further speciation, where it was identified as M. fortuitum.

The patient had a past history of tuberculosis for which she had completed treatment (25 years back). However, the current illness seemed unrelated to it. To search for an active focus of tuberculosis, further tests were done. However, chest high-resolution computed tomography did not show active lesion or cavity.

Erlich-Ziehl-Neelsen stained sputum and urine were negative for acid fast bacilli. There was no evidence of tuberculosis in abdominal ultrasonography either.

Therefore, Pulmonologist’s consultation was taken, and anti-tuberculous therapy (ATT) was started. As the patient was discharged and advised to be followed up in out-patient department with a baseline liver function test to monitor ATT related toxicity.

15 days later the patient was readmitted for the next cycle of chemotherapy. This time chemotherapy was given via a peripheral line, as a matter of caution to prevent the possible impending bacteremia. Repeat blood cultures from the chemo port and peripheral line were collected. On the 4th day the chemo port cultures were positive again for acid fast bacilli. The diagnosis of chemo port colonization was made and removal of chemo port was planned.

Discussion

Although RGM are not highly virulent or life threatening, they have a high predisposition to create biofilms and thus to colonize and infect intravascular catheters. Catheter-related RGM infection nearly always, occur in patients who have a CVC in situ, in a 10 years review of oncology patients, Gil et al. reported that CVC was present at diagnosis in 136 (97%) of 140 patients, the underlying malignancies were commonly hematologic. M. mucogenicum was the predominant species, perhaps due to its association with outbreaks. Pediatric and male patients were more predisposed; chemotherapy, corticosteroids, and lymphopenia were other important risk factors.[1,2]

Fever with or without chills and rigors in a patient who has received chemotherapy via a TID/CVC in the past 3 months is a subtle indicator of the underlying infection. Paired blood cultures should be drawn
from the catheter site as well as periphery and send in BACTEC or BacTAlert bottles for continuous monitoring of blood cultures. Although insertion site is usually normal, redness or oozing at the catheter exit site is a proclamation of an infection underneath. Therefore in such cases a tip off should be sent to the microbiologist to look out for non-tuberculous acid-fast bacilli in the background of patient’s history and a negative Gram-stain. Cutaneous abscess and catheter hubs should also be cultured for mycobacteria. Auramine-rhodamine staining and nucleic acid assays have higher sensitivity and specificity and can hasten the diagnosis as well. Disseminated mycobacteremia can occur if not timely detected and treated.[1,3]

Prognosis is excellent when removal of catheter is done in addition to administration of systemic antibiotics. Most commonly used antibiotics are amikacin, clarithromycin, linezolid, cotrimoxazole, and Ciprofloxacin.[4] Species identification should always be attempted as response to amikacin, clarithromycin, linezolid, cotrimoxazole and ciprofloxacin may vary with species. In 96% of the patients the infection is resolved after appropriate treatment.[4] The optimum duration of treatment is 4 weeks, longer treatment offers no additional benefit however, treatment has to be individualized as no guidelines are currently available.[4]

Rapidly growing mycobacteria have been frequently isolated from tap water. Outbreaks of bacteremia from oncology units and subsequent isolation from the tap and municipal water have been reported. Therefore, patients should be advised to cover the catheter sites while taking a bath. In hospitals catheter care is usually limited to heparin flush, before the administration of chemotherapy. Routine antimicrobial locks in the absence of clinical infection are not recommended.[5]

**Conclusion**

Clinicians need to be aware of the significance of nontuberculous *Mycobacteria* (NTM) infections in immunocompromised patients with TIDS which are usually present for long times and in the event of fever, blood cultures can enhance detection of such infections. NTM infections are also underreported in the laboratory as aerobic blood cultures are usually sent and many times Gram-positive bacilli may be misclassified as diphtheroids and not processed further for identification.

**References**


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