

Mycobacterium abscessus causing native valve endocarditis due to peripherally inserted central catheter line

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ABSTRACT

Infections due to rapidly-growing mycobacteria (RGM) are increasing worldwide, especially in immunocompromised hosts. However, data on the clinical features of patients with RGM bacteremia are limited [1]. Data on the incidence of clinically significant non-tuberculous mycobacteria (NTM) infections from India are scarce as these are frequently under-diagnosed due to either under recognition by clinicians because of the nonspecific nature of their clinical manifestations, and/or the inadequacy of laboratory services [2]. We present a case of *Mycobacterium abscessus* native tricuspid valve endocarditis in a patient who had a peripherally inserted central catheter line (PICC).

Clinicians need to be aware of RGM as a cause of prolonged fever in patients who have chronic indwelling intravenous catheters [3].

Case report

A 61-year-old female, diagnosed with non-Hodgkins lymphoma (NHL) in June 2011, for which she had received 6 chemotherapy cycles (Adriamycin, Bleomycin, Vincristine, Doxorubicin + Rituximab) from July 2011 to January 2012, started having low-grade fever from February 2012 which persisted for 6 months. She had a peripherally inserted central catheter line of 6 months duration which was removed at this stage. Blood cultures at this time were negative. Low grade fever persisted. A positron emission tomography–computed tomography (PET/CT) scan showed a new small splenic lesion. She received 14 courses of irradiation to the spleen on a suspicion of relapse of NHL. Seven months later, in July 2012, a repeat PET/CT scan still showed increased splenic uptake; the patient underwent splenectomy in September 2012. Histopathology of the spleen was normal. Fever persisted post-splenectomy. On reevaluation, her absolute neutrophil count was $700/\text{mm}^3$, blood cultures were repeatedly negative. Two-Dimensional echocardiography was done twice, and was normal. Elevated serum galactomannan was detected at this time (1.1, normal < 0.5); she received empirical voriconazole based on this. The patient continued to deteriorate and was admitted to Lilavati Hospital in January 2013. On admission, she had tachycardia (pulse—110 beats/min), hypotension, (blood pressure 90/70 mm/Hg), was slightly drowsy, and had bilateral edema of feet.

Laboratory findings included: hemoglobin 7.5 g/dL, white blood cell count $14.6 \times 10^9/\text{l}$, platelet count $47,000/\mu\text{L}$, international normalized ratio (INR) 2.0, partial thromboplastin time (PTT) 27.6 s, total

bilirubin 0.96 mg/dL, serum glutamic oxaloacetic transaminase (SGOT) 37 U/L; serum glutamic pyruvic transaminase (SGPT) 16 U/L, alkaline phosphatase 513 U/L, total protein 3.9 g/L, albumin 1.9 g/L, serum ferritin 1120 $\mu\text{g/L}$, serum triglycerides 182 mmol/L, lactate dehydrogenase (LDH) 239 U/L, C-reactive protein (CRP) 146 (normal < 5). The differential diagnosis at this time included hemophagocytic lymphohistiocytosis (HLH), granulomatous hepatitis, reactivation of lymphoma, and sepsis.

Blood cultures were repeated and patient was initiated on meropenem on a suspicion of sepsis. Two-dimensional echocardiography showed a large 2 cm vegetation of on the anterior tricuspid leaflet. The patient was started on liposomal amphotericin B in view of suspicion of fungal endocarditis. Blood cultures eventually turned positive for non-tuberculous mycobacteria, further identified as *Mycobacterium abscessus*. Patient subsequently underwent tricuspid valve replacement. (Figs. 1 and 2 show the surgically removed tricuspid valve with vegetations). Histology of the native tricuspid valve showed “Acute pyogenic inflammation with associated mycobacterial infection with 4+ Acid fast bacilli seen” (Figs. 3 and 4). Cultures further confirmed *Mycobacterium abscessus*. Polymerase chain reaction assay (PCR) of the tricuspid valve for *Mycobacterium tuberculosis* complex was negative. Post-operatively, patient improved clinically transiently, but had recurrence of fever and became drowsy. Cerebrospinal fluid (CSF) examination showed $450/\mu\text{L}$ total nucleated cells (80% neutrophils, 20% lymphocytes), glucose 31 mg/dL, proteins 228.3 mg/dL, LDH 34 mg/dL. CSF cultures were negative. Patient was initiated on intravenous amikacin, linezolid, moxifloxacin and clarithromycin. Intrathecal

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Fig. 1. Surgically removed tricuspid valve showing vegetations.



Fig. 2. Surgically removed tricuspid valve showing vegetations.

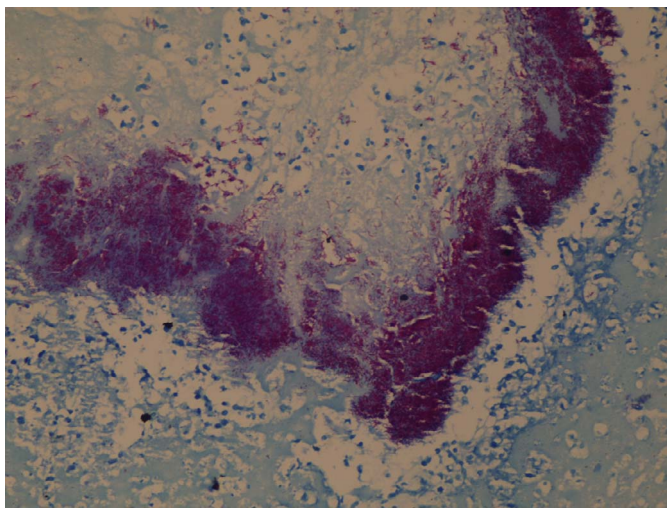


Fig. 3. Ziehl-Neelsen staining showing clumps of AFB.

amikacin was also given. Patient finally deteriorated and subsequently succumbed to her illness.

Discussion

Rapidly growing mycobacteria (RGM) have emerged as important pathogens in catheter-related infections, especially in immunocompromised hosts with malignancies and long-term indwelling

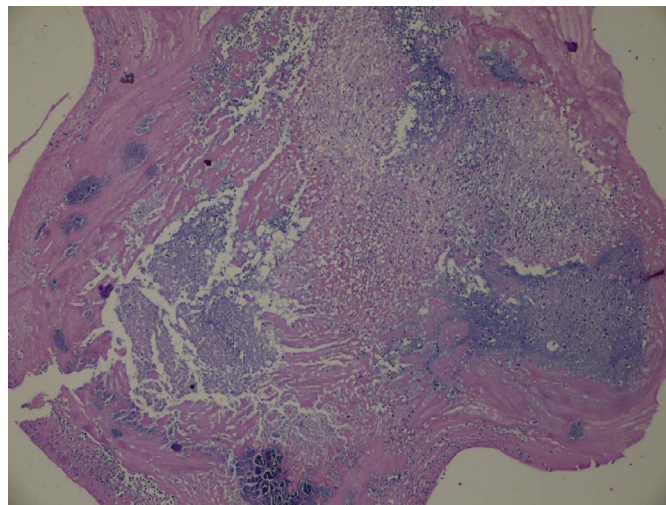


Fig. 4. H and E stain showing caseous necrotic material and neutrophils.

catheters [4]. The incidence of infections with NTM has been increasing due to the growing population of patients with therapeutic interventions and induced immunocompromised status. In 2010, Redelman-Sidi and Sepkowitz reviewed 36 studies reporting on 151 cancer patients with RGM bacteremia, wherein central venous catheter (CVC) was present in 97% patients; *M. mucogenicum* was the predominant species [5].

NTM infections are also underreported in the laboratory since many times gram positive bacilli may be dismissed as diphtheroids and not processed further for identification [3]. The microbiological similarities of rapidly growing mycobacteria and corynebacteria impose potential for misidentification in the laboratory [6]. When NTMs are suspected, it is very important to alert the laboratory so that appropriate procedures are applied. Appropriate treatment prompted by antibiotic susceptibility test results and catheter removal (if present) helps to successfully eradicate infection.

The combination of an underlying immunocompromised state and the presence of a long-term central venous catheter are predisposing risk factors for RGM septicemia, as was seen in our patient. In such patients, who often have fever with no localizing signs, the clinician must have a high suspicion for RGM.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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