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Case report

A rare cause of cavitatory pneumonia

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ABSTRACT

Radiographic findings of thick walled cavities in the lungs are typically seen in mycobacterial infections, malignant lesions, fungal infections, pulmonary vasculitis or other inflammatory lesions of the lungs. Necrotizing infections of the lungs caused by gram negative bacteria (*Klebsiella*, *Psudomonas*, *Legionella*) and *Staphylococcus aureus* may also form cavities of varying thickness, with consolidation. *Escherichia coli* pneumonia causing pulmonary cavities is very rare and the few cases reported are of pneumatocele formation. Here we present an unusual case of *Escherichia coli* infection as a rare cause of bilateral cavitating necrotizing pneumoniae, in a 67 year old male with uncontrolled type 2 diabetes mellitus.

1. Case

A, 67 year old male, with no previously known comorbidities presented to our hospital with fever, cough, generalized weakness and low back ache since 1month. He gave a history of receiving oral antibiotic treatment during the past month, but his symptoms did not resolve. He was febrile, had tachycardia (108 beats/min), blood pressure was normal (120/80 mm of Hg), and maintained oxygen saturation of 96% in room air. A routine random blood sugar (RBS) testing revealed very high blood glucose level (RBS- 704.2 mg/dl).

He was admitted for further investigation and management. His blood sample was sent for analysis and he was started on a broad spectrum antibiotic (intravenous ceftriaxone). His blood glucose levels were controlled with insulin and oral anti-hyperglycemic agents. He was also symptomatically treated with antipyretics, bronchodilators and expectorants.

Upon investigation his chest radiograph showed bilateral multiple thick walled air filled cavities [Image 1]. High resolution computed tomography (HRCT) of his lungs revealed multiple evolving thick walled cavities in both lungs suggestive of Koch's [Images 2, 3]. Based on the nature and duration of symptoms, relentless course, diabetic status and classical radiographic findings a clinical diagnosis of pulmonary tuberculosis was made. His sputum samples were sent for acid fast bacilli (AFB) smear and he was started on standard anti tubercular treatment (ATT) (Rifampicin 600 mg, INH 300 mg, Ethambutol 1 gm, and Pyrazinamide 1.5 gm, along with Pyridoxine). Two consecutive sputum samples (early morning and evening of the same day) did not show any AFB on smear. So the next day a further sample was sent for AFB smear, bacterial culture and sensitivity, and PCR assay (GeneXpert-MTb/Rif) for *Mycobacterium tuberculosis*.

His blood sugars were controlled with insulin, and oral anti-hyperglycemic agents. He was also symptomatically treated with antipyretics, bronchodilators and expectorants. His renal and liver functions were evaluated and found to be normal.

Meanwhile the blood counts showed an elevated total count (12,440) with relative neutrophilia (85.6%). His erythrocyte sedimentation rate (ESR) was 62 mm/hr. The sputum culture grew *Escherichia coli* (E. coli), resistant to most first line antibiotics including ceftriaxone. It was sensitive to gentamicin, amikacin, meropenem and piperacillin/tazobactam. The GeneXpert-Rif report was negative for *Mycobacterium tuberculosis*. His blood culture was sterile and no growth was noticed after 72 hours of incubation. He continued to be febrile with fever spikes (between 100 and 102 deg F).

In view of the conflicting evidences the scope of investigations were broadened. A bronchoscopy was performed and bronchoalveolar lavage (BAL) fluid was obtained. The BAL fluid sample was analyzed for AFB smear and culture, gram's stain, bacterial culture & sensitivity, fungal stain and fungal culture along with study for presence of malignant cells. Ceftriaxone was stopped and he was started on piperacillin/tazobactam. The BAL sample studied

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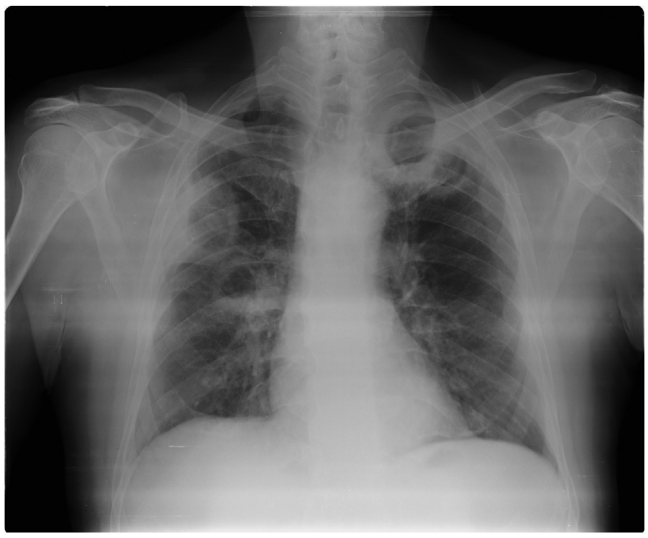

Image 1. Chest X-ray PA view showing bilateral cavities.

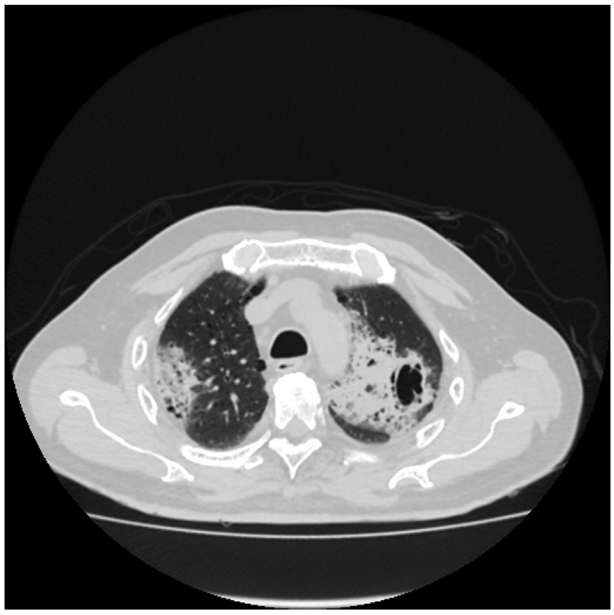

Image 2. HRCT-Thorax showing bilateral consolidation with a cavity in left upper lobe.

did not show the presence of any AFB, fungal elements or malignant cells on smear. On bacterial culture the BAL sample grew E. coli with a colony count of 10^4 cfu/ml, resistant to most first line antibiotics including ceftriaxone, cefuroxime and cefixime, with in-vitro sensitivity to levofloxacin, gentamycin, amikacin, meropenem and piperacillin/tazobactam. Over the next 1 week the patient improved clinically. His cough decreased and he became afebrile. A diagnosis of necrotizing pneumonia due to E. coli was made and ATT was stopped. His blood sugars were well controlled and dose of his oral anti-hyperglycemic drugs titrated and he was discharged from the hospital.

He continued to receive parenteral piperacillin/tazobactam on an out-patient basis for 10 days and he was followed up regularly. His cough steadily decreased and serial chest radiographs on follow up, over the next 4 months showed gradually resolving cavities. During follow up, the BAL culture reports were received and they

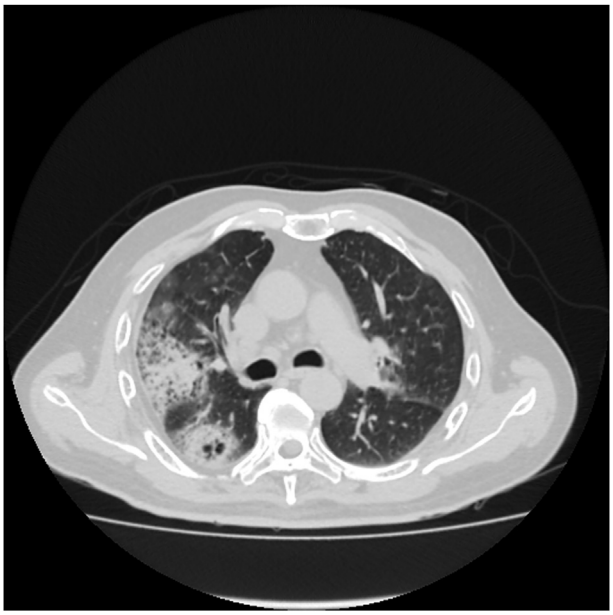

Image 3. HRCT-Thorax showing consolidation in right upper lobe and a cavity in the apical segment right lower lobe.

did not grow any fungus and the culture was negative for AFB. The cavities completely resolved at 4 months [Image 4].

2. Discussion

Cavitatory lesions in radiographs of the lungs can be seen in infections, inflammation or malignancy of the lungs. However certain conditions are more frequently associated with cavities than others. Infection with *Mycobacterium tuberculosis* and fungi tend to be chronic and are common causes of cavity formation in the lungs [1]. *Mycobacterim tuberculosis* infection is classically known to be associated with findings of cavities, both thick (wall size >4 mm) and thin walled (wall size <4 mm) in chest radiograph, more frequently in patients with diabetes as a co-morbidity. Finding of pulmonary cavitation, along with clinical history

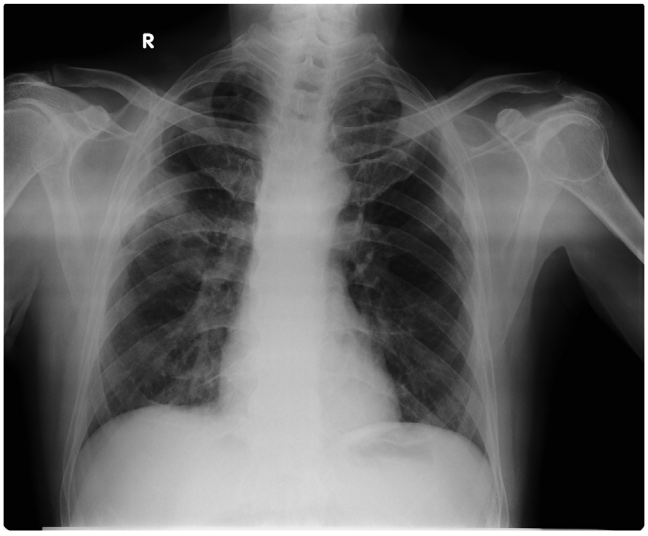

Image 4. Repeat chest X-ray during follow up shows resolution of cavities.

suggestive of pulmonary Koch's has significant diagnostic value in public health setting and prognostic value for the patient, especially in endemic areas [1]. Indian guidelines for management of tuberculosis recommends that in individuals with clinical symptoms suggestive of tuberculosis (cough for more than two weeks not responding to antibiotic therapy) and showing radiographic findings of pulmonary tuberculosis, if the sputum is negative for AFB should be classified as 'sputum negative pulmonary tuberculosis' and started on ATT [2].

Our patient came with clinical history of fever and cough for a month inspite of receiving treatment. He had radiographic finding of multiple thick walled cavities along with uncontrolled diabetes status. His condition was strongly suggestive of pulmonary tuberculosis. Also a high resolution computerized tomography of his lungs showed multiple evolving thick walled cavities suggestive of pulmonary Koch's. He was started on ATT as per Indian Guidelines. However two consecutive sputum smears were negative for AFB which was very unlikely in cavitating tuberculosis.

So his sputum sample was sent for the gene-eXpert RIF test, which again did not detect any *Mycobacterium tuberculosis*. Therefore we looked at the possibility of other common causes for thick walled cavities namely fungal infection and malignancy. Both aspergilloma of the lung and primary malignancy of the lung are also frequently known to present with cavitation in the lungs with thickening of the cavity walls [1]. The sputum sample was sent for fungal stains and it was found to be negative for fungal elements.

Meanwhile the sputum sample grew E. coli (moderate growth). His blood counts also showed high total count with relative neutrophilia, indicating a possible bacterial infection rather than a mycobacterial infection.

A bronchoalveolar lavage (BAL) was performed and the BAL fluid analyzed for presence of AFB, fungal elements, malignant cells and bacterial culture. The BAL fluid was also negative for AFB, fungal elements and malignant cells. Again the bacterial culture of the BAL fluid showed significant growth of E. coli (10^4 cfu/ml), with a very similar sensitivity pattern to the sputum culture, strongly suggestive of E. coli being the causative organism. We also sent a sample of the BAL fluid for fungal culture and AFB culture in our attempts to arrive at a definitive microbiological diagnosis.

In light of the overwhelming negative evidence against *Mycobacterium tuberculosis* as the possible infective agent, and the positive evidence suggestive of *Escherichia coli* infection of the lung, a diagnosis of necrotizing E. coli pneumonia was made and his ATT was discontinued. It is not uncommon for acute bacterial infections of the lung to cause necrotizing pneumoniae with cavitation. The organisms generally implicated are *Staphylococcus aureus* and few gram negative bacteria (*Klebsiella*, *Psudomonas*, *Legionella)* [1,3]. E. coli infections of the lungs are relatively rare [4], but there is a reported rise in incidence of E. coli pneumonia in recent years, both in hospital and community settings [5]. Our patient did not have E. coli bacteremia and his blood culture was sterile. However it is documented that E. coli pneumonia may occur due to 'micro-aspiration of upper airway secretions' due to previous airway colonisation by E. coli, especially in patients with underlying conditions like diabetes mellitus [6]. Thick walled cavities in E. coli pneumonia seem to be a rarity, although we came across few reported cases of pneumatocele (thin walled air filled cavity) formation due to E. coli infection [6]. A review of cavitary lesions of the lung published by the American Society for microbiology does not list E. coli as a causative organism [1].

In this background we wish to report this case of E. coli infection as a rare cause of cavitating pneumonia in an individual with uncontrolled diabetes mellitus. As E. coli infections become common in community settings it becomes important to consider this organism as a potential cause for necrotizing pneumonia with cavity formation, albeit rare.

In practice we frequently encounter cavitating lesions of the lung with a clinical picture suggestive of mycobacterial etiology. Although pulmonary Koch's would be the first in the list of differential diagnosis, especially in endemic areas, in sputum negative cases an attempt should be made to aggressively look for a possible microbiological cause of the infection, other than Koch's.

Through this case we wish to emphasize the role of advanced investigation techniques like flexible bronchoscopy in arriving at a definitive etiological diagnosis. By using all the diagnostic methods available at our disposal it may be possible to pin the diagnosis in many cases for effective treatment and to avoid over treatment. It will also help us in identifying atypical presentations and add to the evidence base.

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