

Recurrent Lung Abscesses in Diabetes Mellitus - Most Likely Cause Being Immune Dysfunction

Dr. Jofin George^{1*}, Dr. Anika Parrikar², Dr. Sanjivani Keny³, Dr. Durga Lawande⁴

¹Junior Resident, Pulmonary Medicine, ²Senior Resident, Pulmonary Medicine, ³Associate Professor, Pulmonary Medicine, ⁴Professor and Head, Pulmonary Medicine Goa Medical College, Bambolim, Goa, India

DOI: [10.36347/sjams.2019.v07i12.029](https://doi.org/10.36347/sjams.2019.v07i12.029)

| Received: 09.12.2019 | Accepted: 17.12.2019 | Published: 22.12.2019

*Corresponding author: Dr. Jofin George

Abstract

Case Report

Diabetes Mellitus (DM) incidence is increasing globally at an alarming rate. The risk of infections is high in presence of DM. This is likely due to immune dysfunction which makes them prone to develop infections. Here we present a case of uncontrolled Diabetes mellitus admitted thrice with lung abscess with involvement of different lung lobes every time.

Keywords: Lung abscess, Diabetes Mellitus, Immune dysfunction.

Copyright © 2019: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

INTRODUCTION

It is estimated that by the year 2025, 380 million persons will suffer from Diabetes Mellitus (DM) globally. WHO report suggests that China and India will account for 130 million out of this [1].

It is well established that diabetics are prone to get infections, and risk of infection increases when DM is uncontrolled. This may be due to reduced response of T cells, neutrophil dysfunction, and disorders of humoral immunity among diabetics [2]. Here we present a case of Diabetes mellitus admitted thrice with lung abscess with involvement of different lung lobes every time. Patient responded very well to antibiotics every time as insulin administration brought blood sugar level under control. However patient would not monitor DM after discharge

CASE REPORT

A 41 year old male patient with DM was admitted in pulmonary medicine ward with history of cough with muco-purulent expectoration, low grade fever and loss of appetite and weight for 2 months. Patient was on oral metformin 500mg OD.

There was no history of anti-Tuberculosis (anti-TB) treatment in the past and no contact with TB. He gave past history of being admitted seven years back for left upper lobe lung abscess with uncontrolled DM. (Image no.1) He was started on Intravenous

Cefoperazone-sulbactam and linezolid along with insulin for Diabetic control. Patient showed radiological as well as clinical improvement. Second admission was four years back as a case of left lower lobe lung abscess with uncontrolled DM. (Image no.2) He received a course of cefoperazone-sulbactam and amikacin along with insulin to which he responded well.

On examination he was febrile with pulse rate 100/minute, respiratory rate 24/minute, Blood Pressure of 110/70 mm of Hg in right upper limb. Pallor was present, no clubbing, cyanosis and lymph nodes. On examination of respiratory system no abnormality was found. Other systems were normal.

Blood investigations showed Haemoglobin of 10.5gm%, Total WBC count of 24,200/cmm, with Neutrophils 81%, lymphocytes 8%, monocytes 10% and eosinophils 1%. Fasting blood sugar was 139mg%, post-prandial sugar 231mg% and HbA1c 9%. Sputum smear microscopy for Mycobacterium Tuberculosis bacilli (MTB) and CBNAAT were negative.

His renal and liver function tests were normal.

Chest radiograph at the time of admission (Image no.3) showed a thick walled cavity with fibrotic extensions in right upper and mid zones.

As patient was having uncontrolled diabetes and history was of long duration, with radiological picture of a thick walled cavity, patient was empirically started on anti TB drugs with 4FDC as per weight band.

As blood counts were markedly raised antibiotics ceftriaxone and azithromycin were also given. However patient continued to have temperature spikes. Repeat blood counts after 72hours were further raised. His sputum culture grew *pseudomonas aeruginosa* sensitive to cefoperazone-sulbactam, amikacin and resistant to ceftriaxone. Patient was also subjected to Fiberoptic bronchoscopy which was normal. Bronchial washings were negative for MTB on microscopy as well as CBNAAT. Therefore antibiotics were changed to Cefoperazone-sulbactam, amikacin and anti-TB drugs were discontinued. His blood sugars were monitored and were controlled with short acting insulin.

Patient gradually improved clinically. Repeat chest radiograph after 14 days of antibiotic course showed marked decrease in size of cavity as well as surrounding consolidation. (Image no.4) His repeat blood counts after 14 days were: Total counts 14100/cmm with neutrophils 82%, lymphocytes 12% monocytes 6%.He was continued on same antibiotics for total of 21 days and then discharged.

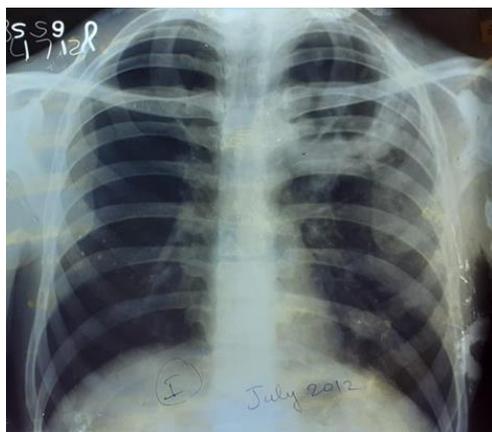


Image-1: Chest x-ray shows left upper lobe lung abscess

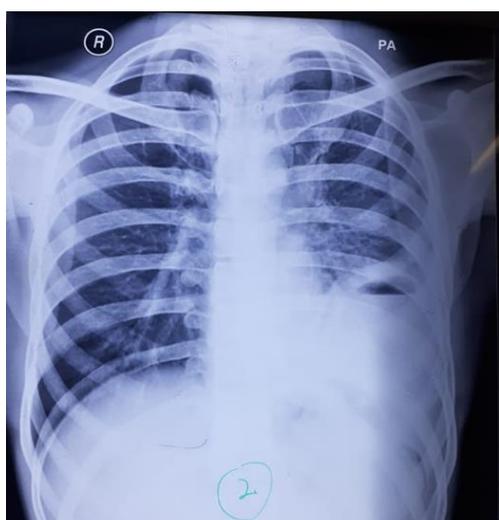


Image -2: Chest x-ray shows left lower lobe lung abscess

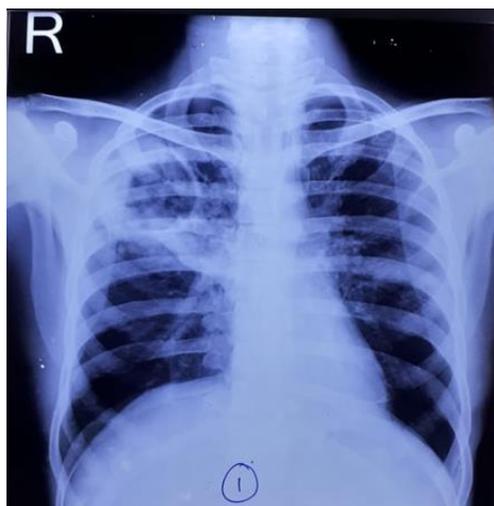


Image-3: Chest xray shows right upper lobe lung abscess

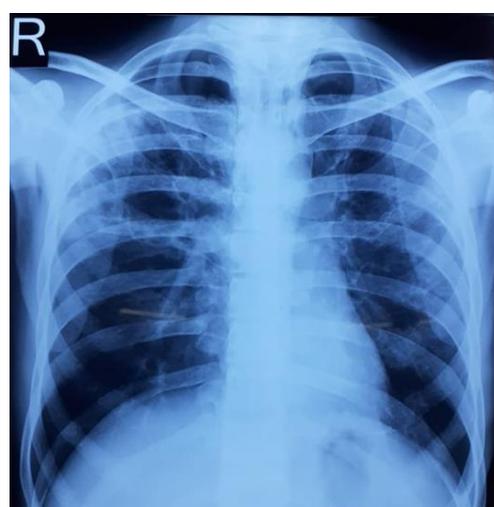


Image-4: Repeat chest x-ray after antibiotics shows improvement

DISCUSSION

DM incidence is increasing globally at an alarming rate. The risk of infections is high in presence of DM. This is mainly due to disordered humoral immunity, dysfunction of neutrophils, lower response by T lymphocytes and less secretion of inflammatory cytokines in presence of DM [2]. Due to intrinsic defect in the cells of individuals with DM there is less secretion of Interleukin 1 (IL-1) and IL-6 by mononuclear cells and monocytes. Also there is decreased production of IL-10 by myeloid cells and IFN-gamma and TNF-alpha due to increased glycation [2]. There is impairment of chemotaxis, adherence, phagocytosis and the phagocytic killing due to hyperglycemia impairing functioning of neutrophils and macrophages. NADPH which is essential for the production of free radicals is diverted to the polyol pathway to metabolise the excess glucose which enters the cells. This leads to impairment of the “respiratory burst” with decreased production of free radicals [3]. There is also an inhibitory effect on G6PD leading to increased apoptosis of neutrophils and reduced transmigration across the endothelium [4]. Long

standing DM also gives rise to microangiopathy in pulmonary vasculature leading to impaired oxygen dissociation and tissue hypoxia which in turn predisposes to anaerobic infections [3].

Specific factors may predispose DM patients to certain types of infections. Patients with diabetes are often nasal carriers of *Staphylococcus aureus* and so at increased risk of pneumonia [3]. Other common respiratory infections in DM are Tuberculosis, H1N1, Streptococcal pneumonia, klebsiella, Psuedomonas, Acinetobacter and Influenza [2, 3]. Also a diminished cough reflex, and disordered sleep patterns [5, 6] oesophageal disorders and altered mental status (hypoglycemic seizures) [3] among Diabetics predispose them to pulmonary infections.

A study published recently showed how DM was risk factor contributing to mortality from Middle East Respiratory Syndrome coronavirus (MERS-CoV). Further research, to understand how DM can increase the severity of respiratory infection which may lead to more fatality, is necessary [7].

CONCLUSION

To conclude immune dysfunction is the most common cause of infections in DM and how to reverse this back to normal may be an important subject for further research.

Also after discharge patient should be followed up for control of DM so that recurrence of such infections can be avoided.

REFERENCES

1. Atkins RC, Zimmet P. Diabetic kidney disease: Act now or pay later. *Kidney International*. 2010; 77(5):375-77.
2. Casqueiro J, Casqueiro J, Alves C. Infections in patients with diabetes mellitus: A review of pathogenesis. *Indian J Endocrinol Metab*. 2012;16 Suppl 1(Suppl1):S27–S36.
3. Ljubic S, Balachandran A, Pavlic-Renar I, Barada A, Metelko Z. Pulmonary Infections in Diabetes Mellitus. *Diabetologia Croatica*. 2004; 33-4:115-24.
4. Peleg AY, Weeraratna T, McCarthy JS, Davis TM. Common infections in diabetes: Pathogenesis, management and relationship to glycaemic control. *Diabetes Metab Res Rev*. 2007; 23:3–13.
5. Antonelli Incalzi R, Fuso L, Giordano A. Neuroadrenergic denervation of the lung in type I diabetes mellitus complicated by autonomic neuropathy. *Chest*. 2002; 121:443–51.
6. Abu-Ashour W, Twells L, Valcour J. The association between diabetes mellitus and incident infections: a systematic review and meta-analysis of observational studies. *BMJ Open Diabetes Res Care*. 2017;5(1):e000336. Published 2017 May 27.
7. Kulcsar KA, Coleman CM, Beck SE, Frieman MB. Comorbid diabetes results in immune dysregulation and enhanced disease severity following MERS-CoV infection. *JCI Insight*. 2019;4(20).