Pneumocystis Jiroveci Pneumonia in an HIV Infected Patient

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Abstract

A 46 year old male patient presented with dry cough, fever, increasing breathlessness since two weeks. He was seropositive for HIV 1 antibodies. Induced sputum showed crescent shaped clusters of cysts in Gomori's Methenamine Silver stain and diagnosed as a case of *Pneumocystis jiroveci* pneumonia.

Introduction

Pneumocystis jiroveci pneumonia is the commonest pulmonary infection in patients infected with HIV infection in western countries.[1] However tuberculosis is the commonest pulmonary infection in AIDS patients in India, *Pneumocystis jiroveci* pneumonia is less frequently reported.[2-4] The present case report describes a case of *Pneumocystis jiroveci* in an AIDS patient with normal radiological presentation.

Case Report

A 46 year old male, truck driver by occupation presented with a history of dry cough, increasing breathlessness since two weeks. He was tested for HIV antibodies and was found to be seroreactive for HIV 1 infection. His sputum samples were tested for presence of acid fast bacilli (AFB), but smears did not reveal AFB.

On examination he was febrile with a pulse rate of 90/min, respiratory rate of 34/min, pallor was present. Oral candidiasis was observed. No icterus, cyanosis or palpable lymphnodes noted. Other systemic examination revealed no obvious abnormality.

Investigations revealed Hb 10 gm%, TLC 4000/ mm^3, with 70% polymorphs, 28% lymphocytes and 2% eosinophils. X-ray chest PA view was within normal limits. The CD_4 counts were 200/ml. Sputum was sent for culture and routine microscopy. No growth of pathogenic organisms was reported. He was started on broad spectrum antibiotics for one week but did not improve. Induced sputum was collected by administering hypertonic saline and Gomori's Methenamine Silver staining was done which revealed clusters of brownish black cysts, cup, crescent and banana shaped navicular bodies against a greenish background (Fig. 1). The patient was switched over to oral cotrimoxazole therapy to which the patient responded with a marked improvement after 48 hours of start of therapy. The treatment was continued for three weeks.

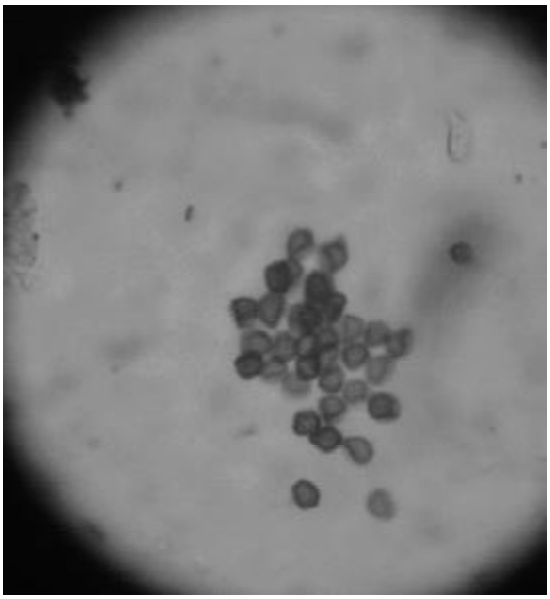

Fig. 1 : GMS staining showing cysts of Pneumocystis jiroveci.

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Discussion

Prior to 1980's, *Pneumocystis jiroveci* pneumonia was a sporadic cause of pneumonia occurring mainly in few immunocompromised patients. In 1981 the outbreak of PCP among homosexuals in Los Angeles (USA) led to the recognition of AIDS.[5] In the west, 75% of individuals infected with HIV developed *Pneumocystis jiroveci* pneumonia, sooner or later. The wide spread use of primary or secondary prophylaxis led to a decline in *Pneumocystis jiroveci* pneumonia after 1988. In India, although the incidence of HIV infection is rapidly increasing yet case reports are scarce in literature.[4,6,7] This may be due to the lack of diagnostic facilities or the Indian patients already suffering and dying due to pulmonary tuberculosis. Rani *et al*[4] showed that out of 23 clinically suspected cases, diagnosis of *Pneumocystis jiroveci* pneumonia was confirmed in 6 cases (26%). A reent study from Maharashtra by Mishra *et al*[7] showed a rise in detection of *Pneumocystis jiroveci* pneumonia from June 1999 to May 2005. The study showed that GMS staining was positive in 100% cases, toluidine blue in 98.2% cases while Giemsa in 91.77% cases. GMS stains the merozoites as well as the cyst walls and detection is earlier but it is cumbersome and expensive to perform. Our patient's sample was tested with GMS and was positive for *Pneumocystis jiroveci* pneumonia.

Dry cough, dyspnoea (grade I or II) low grade fever were the manifestations in our study which was observed in other studies also.[5-7] The CDC criteria allows[8] presumptive diagnosis of *Pneumocystis jiroveci* pneumonia being made in HIV infected person with

1) Dyspnoea on exertion/non productive cough of recent origin.
2) Chest X-ray showing diffuse bilateral interstitial infiltrates.
3) Arterial hypoxaemia.
4) No evidence of bacterial pneumonia.

These criteria may help in the diagnosis of *Pneumocystis jiroveci* pneumonia in HIV infected patient, but less commonly on atypical radiographic presentation may be found in *Pneumocystis jiroveci* pneumonia.[4] In early stage of *Pneumocystis jiroveci* pneumonia, the chest radiograph shows fine bilateral perihilar diffuse infiltrates which progress to interstitial alveolar butterfly pattern. From hilar region the infiltrates spread to apices or bases. However, normal X-ray may be seen in 2-34% of the cases.[4] In the present case X-ray chest was normal but breathlessness, cough and fever was present.

Clinical response to 3 weeks treatment cotrimoxazole (100/75 mg/kg) is good. Cotrimoxazole is well tolerated by patients.[7] Our patient also responded very well to treatment without relapse.

Though presumptive diagnosis of *Pneumocystis jiroveci* pneumonia can be made by CDC guidelines, currently the diagnosis depends upon microscopic visualization of *Pneumocystis jiroveci* cysts/ trophozoites in respiratory secretions or biopsy specimens using GMS, Giemsa or toluidine blue. Though GMS is most sensitive to detect *Pneumocystis jiroveci* pneumonia it is cumbersome.

Non invasive methods as in induced sputum and/or bronchoalveolar lavage analysis are safer and sensitive also.

Induced sputum used in the present case is easier to obtain than other specimens. It is more suitable for moderately equipped setups where special facilities may not be available or affordable by the patient. Many authors have found induced sputum to be useful for *Pneumocystis jiroveci* pneumonia detection.[7,8,9]

We conclude with a remark that clinicians

must be aware of various radiological findings as well as abnormal findings. High index of suspicion is required when an HIV seropositive patient presents with fever, breathlessness and sputum is negative for AFB and sputum culture shows no growth of pathogenic bacteria.

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ENDOSCOPIC TREATMENTS FOR GASTRO-OESOPHAGEAL REFLUX DISEASE

Klaus Monkemuller and colleagues recently systematically reviewed clinical trials from the past three decades on the use of endoluminal therapies to support the use of endoluminal therapies for GORD [gastro-oesophageal reflux disease] in routine clinical practice.

An example of this is the use of the radiofrequency energy technique since 2000 in several thousand patients, without any long-term data to support claims that it “thickens” and “tightens” musculature of the lower oesophageal sphincter (LOS), reduces LOS relaxation, provides a long-term improvement in symptom control, or reduces use of proton-pump inhibitors.

In many studies, therapeutic success with the endoluminal therapies is defined if there is a reduction in use of proton-pump inhibitors of 50% or more. The outcome of this lowering of the threshold for success has been an ***unjustified*** rush to offer such approaches to patients seeking a long-term solution to their problem in the hope of eluding a lifetime of pill taking. As a result, many patients have undergone procedures that have since been abandoned and proven ineffective, and an unfortunate minority has had substantial side-effects or even died.

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