

Identification and treatment of a patient with pneumocystis pneumonia (case report)

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(Ann. Coll. Med. Mosul 2012; 38 (1): 68-71).

Received: 5th Jun. 2011; Accepted: 4th Dec. 2011.

ABSTRACT

A case of pneumocystis pneumonia was diagnosed on clinical and mycological grounds. A 35 - year - old man was presented with severe chest infection. His medical history included lymphoma for 5 years and under treatment with cytotoxic drugs. The patient diagnosed as a case of pneumonia and treated with antibiotics for one week but with no response. Later on, his sputum was sent for mycological examination that revealed the cysts and trophic forms of pneumocystis jirovecii. Good response with complete healing was achieved after 3 weeks of treatment with co-trimoxazole. Pneumocystis pneumonia may be suspected clinically in hospitalized patients, but this is the first case to be confirmed mycologically in Mosul. Good awareness of the full clinical spectrum of the disease aided by mycological study is needed to minimize the misdiagnosis of cases.

Keywords: Pneumocystis jirovecii, pneumocystis pneumonia, pneumocystosis.

الخلاصة

شخصت حالة مريض متوقع الإصابة بذات الرئة بالمتكيسات الرئوية مختبرياً بالفحوصات الفطرية لتأكيد التشخيص السريري. أحيل رجل عمره ٣٥ سنة إلى قسم العناية المركزة التنفسية في مستشفى ابن سينا التعليمي. حيث كان المريض مصاباً بسرطان الغدد اللمفية منذ ٥ سنوات وتحت العلاج الدوري المستمر. يعاني المريض من التهابات رئوية معقدة للمعالجة التقليدية. تم إرسال عينة من البلغم إلى مختبر الأحياء المجهرية في كلية طب الموصل للتحري عن المتكيسات الرئوية الفطرية. تم التشخيص الفطري بظهور الأكياس الفطرية لفطر pneumocystis jirovecii في البلغم. بعدها كانت استجابة المريض لعلاج co-trimoxazole لمدة ثلاثة أسابيع جيدة وتمثل للشفاء. من المتوقع ان مرض ذات الرئة بالمتكيسات الرئوية يصيب كثير من المرضى ضعيفي المناعة والراقدين في المستشفيات. قد تكون المرة الأولى التي تم التشخيص المختبري في الموصل لمثل هذه الحالة، لذا انه من المؤكد والمفيد أن تكون الإصابة بذات الرئة بالمتكيسات الرئوية من ضمن التشخيص التفريقي لأي حالة مماثلة مستقبلاً.

Pneumocystis pneumonia (PCP) is caused by a yeast-like fungus, *pneumocystis jirovecii*. This type of pneumonia is a condition that could be successfully treatable if diagnosed early ⁽¹⁾. The causative agent first described as a protozoan and reclassified as a fungus in 1988 ⁽²⁾. Delanes named the organism in honor Dr. Carini after isolating it

from infected rats. Years later, Dr. Otto Jirovec and his group isolated the organism from human, and the organism responsible for PCP was renamed as *pneumocystis jirovecii*, which is human specific ^(3, 4).

The disease is relatively rare in normal people although the fungus present among the general population ⁽⁵⁾, but it is commonly

encountered in immunocompromised patients⁽⁶⁾. The patient who have PCP without AIDS typically present with an abrupt onset of respiratory insufficiency that may correlate with an increased dosage of immunosuppressant medication⁽⁷⁾. Extra- pulmonary involvement is rare and systemic spread to many organs as liver, spleen and lymph nodes can result in severe disease refractory to standard therapeutic regimen⁽⁸⁾.

The causative organism establishes latency in immunocompetent individuals. Immunosuppression especially of the T-cell function leads to reactivation of infection resulting in disease⁽¹⁾. The common symptoms of PCP include shortness of breath, low grade fever, non-productive cough and usually no large amount of sputum unless the patient has an additional bacterial infection⁽²⁾. The approach to the diagnosis of PCP and its treatment remains controversial. Because of the critical condition of the patients, some authors advocate the use of clinical criteria alone in the diagnosis of *P. jirovecii*, however, it has become increasingly clear that such an empiric regimen may be associated with an overall worse outcome for the patient⁽⁹⁾.

The commonly used medication is a combination of trimethoprim and sulfamethoxazole (TMP-SMX), but some patients with known allergies to sulfa cannot tolerate this therapy. Other medications that are used include dapson, trimetrexate and clindamycin. For prophylaxis against *P. jirovecii* in immunocompromised patients, cotrimoxazole, dapson/pyrimethamine, or pentamidine nebulizer can be used⁽²⁾.

Case report

A 35-year-old man was admitted to the Respiratory Care Unit (RCU) in Ibn-Sina Teaching Hospital referred from the oncology unit with severe chest infection. He had nausea, malaise, fever of 37.6°C, dyspnea,

dry cough to start with, then the patient started to have productive cough with white sputum and then haemoptysis for several days. His medical past history included lymphoma for 5 years and he was under regular treatment with courses of cytotoxic drugs. Radiological examination showed diffuse infiltrates in both lungs and was diagnosed as a case of broncho pneumonia. The blood picture showed: Hb 92 g/L; PCV 33%; total WBC $11.4 \times 10^9/L$ (N 59%, L 38%, M 3%) and ESR 110 mm/hr. The patient first was treated in the oncology unit with ampicillin-cloxacillin and ceftriaxone for nearly one week but with no response and then referred to the RCU.

The diagnosis of the present case as PCP was made on the bases of chest x-ray findings and clinical examination, with the exclusion of other possible causes and was treated accordingly with TMP-SMX. At the same time, the early morning sputum from the patient was sent to the Department of Microbiology, College of Medicine, University of Mosul, for laboratory confirmation.

Laboratory report: The whitish bloody sputum was subjected to direct microscopical examination of stained slides with Giemsa and Toludin blue O stains. The trophic forms and cysts of *pneumocystis jirovecii* were revealed by these stains respectively (Figure I- b & c). In addition, other wet mounted slide with 20% KOH solution and calcofluor stain showed the cysts of the fungus when examined under fluorescent microscope (Figure I- a).

Treatment: The patient was started on TMP-SMX (120 mg/kg p.o) in 4 divided doses. Because the pneumonia was severe, intravenous hydrocortisone 100 mg 4 times daily was added. The treatment was continued for 3 weeks, with full recovery of the patient.

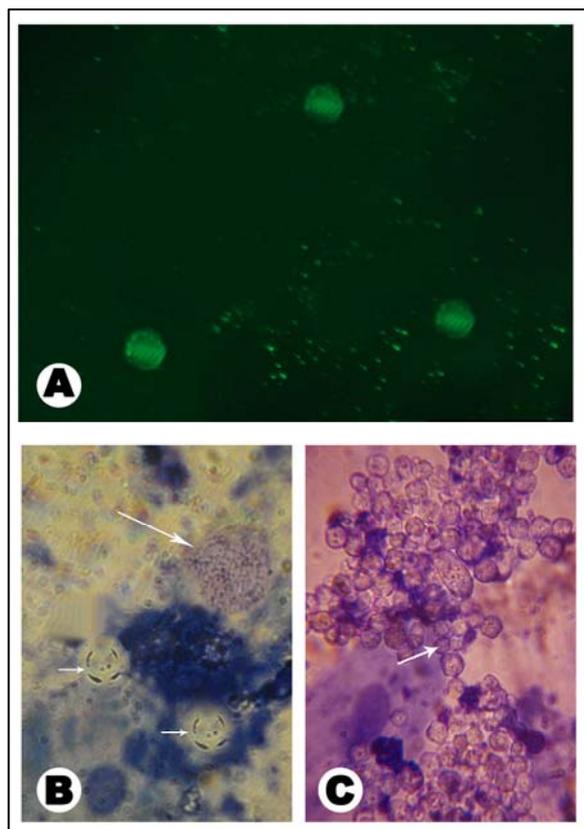


Figure (1): *Pneumocystis jirovecii* seen in sputum:

- A. 20% KOH and calcofluor stain showing the spherical cyst (cyst wall and thickening intensely fluorescent) by fluorescent microscopy (40X).
- B. Giemsa stained smear showing intracystic bodies (short arrowed), 100X, and extracellular, trophozoites (long arrowed), 100X.
- C. Toluidine blue stained smear showing many spherical violet cysts (arrowed), 100X.

Discussion

Pneumocystis pneumonia is usually considered as a secondary infection in immunocompromised patients. It remains the most prevalent opportunistic infection in patients infected with the immunodeficiency virus⁽¹⁰⁾. The number of patients who are receiving chronic immunosuppressive medication or who have an altered immune system and are thus at risk for PCP is rapidly growing⁽²⁾. The studied patient had lymphoma

and under cytotoxic therapy for 5 years presented with severe pneumonia in the RCU. The PCP is an increasing common infection in cancer patients, such as those with lymphoma and leukemia⁽¹¹⁾. The development of worsening pneumonia with respiratory failure in patients with hypoxaemia is the most common reason for admission to an intensive care unit⁽²⁾.

In hospitalized patients, the clinicians depend on the clinical diagnosis of PCP and start treatment empirically. The diagnosis of such an infection requires the identification of the organism from the clinical specimens microscopically⁽¹²⁾, because until now the organism cannot be propagated in culture⁽¹³⁾. This had confirmed the clinical interpretation and treatment were continued. The diagnosis of the reported patient was confirmed as a case of PCP by the detection of the trophic forms and cysts of *P. jirovecii* in his sputum. This is the first patient confirmed to have PCP and to be reported in Mosul, by utilizing the different staining methods. The patient was treated with co-trimoxazole with a good clinical response, complete healing was achieved after 3 weeks of treatment.

In conclusion, *Pneumocystis pneumonia* can be diagnosed by clinical interpretation and confirmed by the laboratory identification of the causative agent.

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