# Case Report

# *Pneumocystis Jiroveci* Pneumonia and H1N1 Co-infection in an Immunocompetent Patient

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## Abstract

*Pneumocystis jiroveci* pneumonia is frequently diagnosed in immunocompromised patients but it has rarely been reported in immunocompetent individuals without any predisposing factors. Immunocompetent individuals who are exhibited steroids or other immune-suppressive drugs, may become susceptible and may develop clinical *Pneumocystis carnini* pneumonia (PCP) infection. Cases of co-infection of *P. jiroveci* and H1N1 influenza in patients with human immunodeficiency virus (HIV) infection and organ transplant recepients have been reported. However, case reports of *P. jiroveci* and H1N1 influenza co-infection are rare in immunocompetent individuals. We report a case of a young immune-competent man diagnosed with H1N1 influenza and *P. jiroveci* pneumonia which posed significant diagnostic and management dilemma. **[Indian J Chest Dis Allied Sci 2019;61:219-220]** 

Key words: P. jiroveci pneumonia, Immunocompetent, PCP-H1N1 co-infection.

# Introduction

*Pneumocystis jiroveci* is being diagnosed increasingly among non-human immunodeficiency virus (HIV)-infected immunocompromised patients, however its diagnosis in immunocompetent individuals is rarely reported. The H1N1 influenza and P. jiroveci co-infection is one such entity where a significant overlap of symptoms exists and can be difficult for the clinician to distinguish the pathogen. We report a case of a young immune-competent male diagnosed with H1N1 influenza and *P. jiroveci* pneumonia (PCP).

#### **Case Report**

A 25-year-old male was admitted to our hospital with complaints of fever, sore throat, cough with streaky haemoptysis, fatigue and progressively worsening breathlessness of three days duration. He was a resident of Delhi, which was on high alert following the H1N1 influenza pandemic. He denied any history of contact with a known/ suspect case of influenza or high-risk behaviour. Clinically the patient was breathless at rest, febrile, normotensive with a heart rate of 110/min. He had tachypnoea and oxygen saturation on room air was 80%, augmented to 90% with oxygen at FiO<sub>2</sub> 50%. Respiratory system examination revealed fine crackles in the bilateral infra-scapular regions. Haematological parameters revealed bi-cytopaenia with total leucocyte count of 980/µL, absolute lymphocyte count 392/µL and absolute neutrophil count of 558/µL. Platelet count was 83000/mm<sup>3</sup>. Arterial blood gas analysis showed type 1 respiratory failure. Serum lactate dehydrogenase levels were also raised (696 Units/L). Chest radiograph

(postero-anterio view) showed bilateral middle and lower zone acinar opacities (Figure 1). Throat swab for H1N1 was positive on polymearase chain reaction.



Figure 1. Chest radiograph (postero-anterior view) showing bilateral mid and lower zone acinar opacities with perihilar predominance.

At the same time, sputum microscopy on Grocotts stain revealed cysts of *P. jiroveci* (Figure 2). The CD 4 count was  $161/\mu$ L and CD4:CD8 ratio was 1.76. Patient was HIV seronegative on enzyme linked immunosorbent assay.

Patient was treated with tab Oseltamivir 150 mg/day for five days and tab Trimethoprim (TMP)/Sulfamethoxazole (TMP 1400mg/d) for two weeks. He was given high flow

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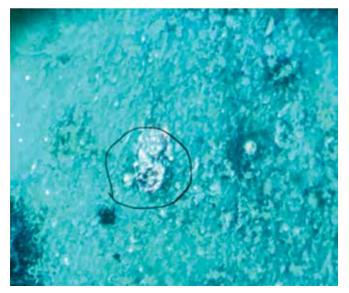


Figure 2. Cyst of *Pneumocystis jiroveci* stained with Grocott's methenamine silver stain (X100).

oxygen and barrier-nursing care. He showed considerable improvement over the next 10 days and was gradually weaned off. The haematological parameters also normalised and the patient had an uneventful recovery. Repeat CD4 counts also normalised to  $710/\mu$ L after two months.

#### Discussion

Pneumocystis jiroveci pneumonia has been rarely reported in immunocompetent individuals without any predisposing factor. Various studies have revealed that Pneumocystis organisms can replicate in the lungs of immunocompetent hosts, thus, humans are reservoirs for P. jiroveci and person-to-person aerosol transmission has also been reported.1 When such immunocompetent individuals are immunosuppressed with glucocorticoid use or other cell-mediated immunity defects, they may become susceptible and may develop clinical PCP infection, thereby predisposing them to *P. jiroveci pneumonia*.<sup>2</sup> There have been reported cases of co-infection of P. jiroveci and H1N1 influenza with HIV infection and organ transplant.3,4 However, case reports of P. jiroveci and H1N1 influenza coinfection are rare in immunocompetent individuals. The clinical manifestations of H1N1 virus infection range from self-limited, uncomplicated, sub-febrile respiratory illness to severe and even fatal respiratory disease and extrapulmonary disease.<sup>5</sup> Approximately 50% of individuals with H1N1 influenza infections may develop lymphopaenia and a decrease in ratio of CD4 to CD8, usually during the early course of the illness. This state of immunosuppression might be responsible for this co-infection with P. jiroveci.6 Our patient also showed lymphopaenia, low CD4 count with a decreased CD4:CD8 ratio. Lewis et al7 found that the

lymphopaenia which is typical of an influenza infection was shown to be secondary to reduction in both T and B cells; however, without alteration in the CD4:CD8 ratio. Depletion of lymphocytes is observed with the onset of illness in acute phase, and recovery is observed soon after the fever subsides.<sup>7</sup> Cao et al<sup>8</sup> who noted a decreased CD4:CD8 ratio in half of those who were tested positive for A/H1N1, similar findings were observed in our case.8 Common clinical presentation of P. jiroveci pneumonia includes onset of subtle dyspnoea which may be progressive, non-productive cough and fever. However, at times, patients may have acute dyspnoea, hypoxaemia and respiratory failure which require intensive care admission.9 Our case presented with similar symptoms of cough, sore throat, breathlessness and was found to be in respiratory failure. Diagnosis for H1N1 was made by a positive PCR on throat swab while P. jiroveci cysts were demonstrated on sputum microscopy. He was managed with both treatment for H1N1 influenza as well as *P. jiroveci* with a favourable outcome. It remains difficult to distinguish a true P. jiroveci infection from colonisation without a tissue biopsy, the treatment for *P. jiroveci* may aid to clinical improvement in such co-infection cases, as was observed in our case also.<sup>6</sup> The present case was unusual as the patient was neither a HIV infected nor he was on immunosuppressive therapy or long-term glucocorticoid treatment.

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