

# Three men, a paint brush and a coronavirus

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**Summary:** Coronaviruses cause respiratory tract infection and a coryzal syndrome. Although described as a cause of gastroenteritis in HIV patients, with the exception of the severe acute respiratory syndrome (SARS), there is little in the literature about respiratory infection in HIV patients. We describe two patients with HIV, exacerbations of chronic obstructive pulmonary disease and proven coronavirus infection. A third patient presented with an upper respiratory tract infection but coronavirus was not isolated. All three men had spent a day decorating the first patient's flat four days prior to presentation. This is the first description of respiratory tract infection with coronavirus in HIV patients. Both patients with coronavirus required prolonged admission to hospital and extensive investigations because they were HIV infected. Coronavirus is often associated with less severe upper respiratory tract infection but can cause more severe disease and should be considered in patients with HIV and respiratory tract infection.

**Keywords:** HIV, coronavirus, chronic obstructive pulmonary disease (COPD)

## INTRODUCTION

Coronaviruses are positive stranded RNA viruses and cause up to 18% of viral upper respiratory tract infections (RTI)<sup>1</sup>. They are associated with exacerbations of airways disease<sup>2</sup> and the severe acute respiratory syndrome (SARS).<sup>3</sup>

Although severe respiratory illness is described in immunocompromised patients<sup>4</sup> and implicated as a cause of diarrhoea in HIV patients,<sup>5</sup> the only description of coronavirus RTI in HIV is in conjunction with SARS.<sup>6</sup> We describe two HIV-positive patients with coronavirus-associated exacerbations of chronic obstructive pulmonary disease (COPD) with close contact prior to presentation. A third HIV-positive man, with close social contact, presented with an acute RTI although coronavirus was not isolated.

## CASE REPORT

Patient 1, a 50 year-old smoker with advanced HIV infection (CD4 count 62/mm<sup>3</sup> (8%)) and COPD was admitted with a three-day history of increasing dyspnoea, cough, purulent sputum and decreased exercise tolerance. Four days earlier, he had been painting his flat with patients 2 and 3. He had previously declined antiretroviral therapy. Chest auscultation revealed widespread wheeze. White cell count and C reactive protein (CRP) were mildly raised and chest radiograph showed emphysematous changes with no focal consolidation. Induced sputum was

negative for *Pneumocystis carinii* pneumonia (PCP) by polymerase chain reaction (PCR). Treatment was empirical with clarithromycin, nebulized bronchodilators and prednisolone. Following weaning from oxygen, he was converted to inhaled bronchodilators and commenced antiretroviral therapy. Throat swab and sputum samples were positive for human coronavirus OC43 by PCR.<sup>7</sup> He was discharged after 13 days.

Patient 2 was admitted 24 h after patient 1. A 58-year-old smoker with HIV (CD4 309 cells/mm<sup>3</sup> (16%), viral load < 50 copies/mL on a atazanavir/ritonavir, didanosine and lamivudine), COPD and bipolar affective disorder, he reported four days of increasing dyspnoea, wheeze and cough with purulent sputum, raised respiratory and heart rate and oxygen saturation of 93% on air. White cell count was normal. CRP and D-dimer were raised. Computed tomography (CT) pulmonary angiogram showed no pulmonary embolus and appearances consistent with COPD. Induced sputum was negative for PCP. He was treated with oral co-amoxiclav, nebulized bronchodilators and a short course of prednisolone. Two sputum samples were positive for coronavirus OC43 by PCR.<sup>6</sup> He was discharged after five days.

Patient 3, a 41-year-old male smoker with HIV (CD4 count 75 cells/mm<sup>3</sup> (10%)) currently off ART, presented with three days of dyspnoea, cough and purulent sputum. Two weeks previously, he had also been decorating patient 1's flat. He was afebrile with audible wheeze and oxygen saturation of 97%. Although a smoker, he had never been diagnosed with COPD but was taking co-trimoxazole for PCP prophylaxis. White cell count and chest X-ray were normal and CRP was minimally raised. He was discharged and PCR for respiratory pathogens was negative.

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## DISCUSSION

Coronaviruses are usually associated with a coryzal syndrome, occasionally in exacerbations of existing cardiopulmonary disease<sup>8</sup> and with SARS. This is the first description of RTI in HIV patients outside SARS. Other respiratory viruses, notably adenovirus, can cause severe disease in HIV.<sup>9</sup> Patients 1 and 2 had severe RTI requiring hospital admission and their HIV status and underlying pulmonary disease may have worsened the severity of disease. The epidemiology of infection is interesting. Both men had been in prolonged close contact before becoming unwell and were infected with the OC43 strain of coronavirus, supporting direct transmission via respiratory droplets or fomites. Although patient 3 did not have coronavirus isolated, it is notable that he presented later than the others and does not have underlying airways disease.

HIV protease inhibitors may inhibit SARS coronavirus replication *in vitro*<sup>10</sup> and small empirical treatment studies suggested a clinical effect from treatment with atazanavir/ritonavir<sup>11</sup>. Interestingly, patient 2 was taking atazanavir/ritonavir at the time of his illness but hospitalization was not prevented.

In summary, coronavirus RTI in patients with HIV may be severe enough to require prolonged hospital admission and extensive investigation due to immunosuppression and the broad differential diagnosis. Lopinavir/ritonavir does not appear to be protective against the OC43 strain.

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(Accepted 22 May 2006)