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Severity and outcomes of *Pneumocystis pneumonia* in patients newly diagnosed with HIV infection: an observational cohort study

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Abstract

It is unclear whether patients who are unaware of their HIV infection have different severity or outcomes of *Pneumocystis pneumonia* (PCP) compared to patients who have been previously diagnosed with HIV. In this retrospective observational cohort study of consecutive HIV-infected patients with microscopically diagnosed PCP at San Francisco General Hospital between 1997 and 2006, 121 of 522 patients (23%) were unaware of their HIV infection prior to their diagnosis of PCP. The proportion of patients with concurrently diagnosed HIV and PCP each year remained unchanged during the study period. Patients with newly diagnosed HIV had a significantly higher alveolar-arterial oxygen gradient at presentation (median 51 versus 45 mm Hg, $p=0.03$), but there were no differences in mortality, frequency of mechanical ventilation, or admission to intensive care compared to patients with previously diagnosed HIV infection. In multivariate analysis, patients who reported a sexual risk factor for HIV infection were more likely to be newly diagnosed with HIV than patients who reported injection drug use as their only HIV risk factor (odds ratio = 3.14, 95% confidence interval 1.59–6.18, $p = 0.001$). This study demonstrates a continued need for HIV education and earlier HIV testing, particularly in patients with high-risk sexual behavior.

Introduction

In the combination antiretroviral therapy (CART) era, *Pneumocystis jirovecii pneumonia* (PCP) remains the most common AIDS-defining illness in HIV-infected patients and is a frequent cause of morbidity and mortality [1,2]. Earlier studies of PCP reported that 27–68% of patients were diagnosed concurrently with PCP and HIV [3–8]. A more recent study, however, found that a lower proportion of persons presenting with PCP in the CART era (1996–2006) were diagnosed concurrently with PCP and HIV compared to prior periods, suggesting improved HIV education and earlier testing efforts [8]. Whether the proportion of patients with PCP who are unaware of their HIV infection continues to decline or has remained constant during the CART era is unclear.

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Ethics: This study was approved by the University of California, San Francisco Committee on Human Research and was performed in compliance with the Helsinki Declaration.

Statistical validity: Leah G. Jarlsberg is a co-author on this manuscript and a statistician in the Division of Pulmonary and Critical Care Medicine at San Francisco General Hospital. She is the statistician and co-author for 13 other publications and agrees that the statistical analysis in this manuscript is valid.

The severity and outcomes of HIV-associated PCP may be different in patients who are newly diagnosed with HIV compared to those with known HIV infection. One study from the pre-CART era found that PCP patients with newly diagnosed HIV had more severe PCP manifested by a lower arterial oxygen pressure (PaO₂) and higher risk of assisted ventilation compared to patients with known HIV [9]. Similarly, patients with newly diagnosed HIV who were admitted to an intensive care unit (ICU) were found to have a lower CD4 count and serum albumin and require a longer length of ICU stay compared to patients with previously diagnosed HIV [10]. In contrast, studies of PCP outcomes found either no difference in PCP mortality [3,8], or a trend towards an increase in mortality in patients who were known to be HIV infected prior to presentation with severe PCP (68% versus 43%, $p = 0.069$) [6].

There are several potential reasons why PCP severity and outcomes may be different based on prior knowledge of HIV infection. Patients with known HIV may be taking CART or PCP prophylaxis, the latter of which can cause “breakthrough” PCP to present with atypical clinical or radiographic manifestations [11]. Patients who are taking PCP prophylaxis have a lower CD4 count at presentation with PCP infection compared to those on no prophylaxis [9], and a lower CD4 count is associated with increased risk for opportunistic infections and mortality. Exposure to sulfa prophylaxis has been clearly associated with *Pneumocystis* dihydropteroate synthase gene mutations and possible antibiotic resistance [12–16]. Despite this association, studies evaluating the effect of PCP prophylaxis on mortality from subsequent PCP infection have yielded conflicting results [17,18]. At present, it is unknown whether patients with newly diagnosed HIV have different severity or outcomes of PCP compared to patients with previously diagnosed HIV infection.

Therefore, we performed a retrospective study with case note review of over 500 consecutive HIV-infected patients microscopically diagnosed with PCP at San Francisco General Hospital (SFGH) in the CART era. Our goals were to determine the proportion of patients who were unaware of their HIV infection and assess whether this proportion remained constant or changed during this period; and to determine whether the clinical presentation, severity, and outcomes of PCP were different based on prior knowledge of HIV infection.

Methods

Patients

Patients were consecutive HIV-infected adults over 21 years of age diagnosed with PCP at SFGH between January 1, 1997 and December 31, 2006. PCP was diagnosed by identification of *P. jirovecii* cystic or trophic forms on microscopic examination of Giemsa-stained (Diff-Quik stain, Dade Behring, Newark, Delaware) induced sputum or bronchoalveolar lavage specimens. Eligible patients were identified by reviewing a registry of all diagnostic tests performed for PCP in the Division of Pulmonary and Critical Care Medicine. Because the Pulmonary Division performs all diagnostic procedures (sputum induction and bronchoscopy) for PCP and the standard of care at SFGH is to confirm the diagnosis of PCP, it is unlikely that any cases were treated empirically without further evaluation or diagnosed without our involvement or knowledge [19]. Patients who had PCP diagnosed by other means (e.g. *Pneumocystis* in pleural fluid) were excluded from this study. The University of California, San Francisco Committee on Human Research approved the study protocol.

Data Collection

Data were collected by chart review using standardized forms and predetermined definitions. Demographic, clinical, laboratory, hospital course, and outcomes data were recorded. We noted dates of PCP diagnosis and initiation of PCP antimicrobial therapy. Mortality was defined as death in the hospital or after transfer to hospice. Patients who had more than one identified

HIV risk factor (e.g., history of injection drug use [IDU] and sex with other men) were categorized as having multiple HIV risk factors.

Statistical Analysis

Statistical analysis was performed using Stata version 9.2 (STATA Corporation, College Station, Texas, USA). The number of patients with concurrently diagnosed PCP and HIV as well as the total number of patients with PCP were measured by year. A test for trend in the percentage of total PCP cases with newly diagnosed HIV and the total number of PCP cases annually was performed using Poisson regression with a variable for calendar year, setting 1997 equal to 0 and increasing the value by 1 for each subsequent year [20]. Univariate analysis was performed using chi-squared or Fisher's exact test for dichotomous variables and Mann-Whitney rank-sum or Student's *t*-test for continuous variables. Unadjusted and adjusted odds ratios with 95% confidence intervals were calculated using logistic regression. All statistical tests were two-sided, and a *p*-value of < 0.05 was considered statistically significant.

Results

From January 1, 1997 to December 31, 2006, 525 cases of PCP were diagnosed at San Francisco General Hospital. We excluded one patient who was diagnosed with PCP from a sample of pleural fluid and two cases where we were unable to determine whether HIV infection was known or unknown at time of admission, leaving a total of 522 patients in our analyses. Because of a high prevalence of homelessness, injection drug use, and other risk factors for loss to follow-up in our patient population, the majority of cases (96%) were admitted for inpatient diagnosis and initiation of antimicrobial therapy.

Patients newly diagnosed with HIV

Overall, 121 of the 522 patients (23%) were newly diagnosed with HIV. The number of PCP cases diagnosed at SFGH each year as a proportion of the total population of the city of San Francisco decreased during the study period ($p = 0.0001$). Reflecting the decreasing incidence of PCP, the number of PCP patients per year with newly diagnosed HIV decreased from a high of 19 patients in 1997 to a low of six patients in 2005. However, there was no significant change in the proportion of PCP cases with newly diagnosed HIV by study year, which ranged from 15% to 33% ($p = 0.23$, Figure 1).

In the group of patients previously diagnosed with HIV infection, 64% had a history of opportunistic infection and 34% had a history of PCP. A minority of patients, 14% and 19% respectively, were taking CART or PCP prophylaxis. The median CD4 count was similar in patients who were taking CART compared to patients who were not taking CART (36 versus 28 cells/ μ L, $p = 0.89$).

Demographics

There were no significant differences in age, race/ethnicity, or housing status between patients who were newly diagnosed and patients previously diagnosed with HIV infection (Table 1). However, gender and HIV risk factor were significantly associated with having a new versus previous diagnosis of HIV infection in both bivariate and multivariate analysis (Table 2). In multivariate analysis, patients who reported a sexual risk factor for HIV infection were more likely to be newly diagnosed with HIV than patients who reported IDU as their only HIV risk factor (odds ratio = 3.14, 95% confidence interval 1.59–6.18, $p = 0.001$).

Severity of HIV infection and PCP

At the time of PCP diagnosis, there was no significant difference in severity of HIV infection between patients who were newly diagnosed with HIV infection and those previously diagnosed with HIV infection (Table 3). In contrast, there was a significant difference in oxygenation between these two groups. The median alveolar-arterial oxygen gradient (A-a O₂ gradient) was significantly higher in patients with newly diagnosed HIV (51 versus 45 mm Hg, $p = 0.030$). Other markers of PCP severity and organ dysfunction including admission vital signs, serum lactate dehydrogenase (LDH), sodium, creatinine, bilirubin, and albumin were similar between the two groups.

Hospital course and outcomes

The only significant difference in hospital outcomes was a longer length of stay in patients who were newly diagnosed with HIV (median 9 versus 8 days, $p = 0.035$, Table 3). The overall in-hospital mortality was 10.3%. There were no significant differences in frequency of ICU admission, mechanical ventilation, or in-hospital mortality between the two groups (unadjusted odds ratio for mortality in patients newly diagnosed with HIV, 0.64, 95% confidence interval 0.30 – 1.34). When the analysis was limited to patients presenting with their first episode of PCP, there were no substantive changes to the results. Prior studies have reported increased mortality during repeat episodes of PCP compared to first episodes of PCP, which could potentially inflate the observed mortality in our patients with known HIV [8]. However, mortality in patients with known HIV during their first episode of PCP was 11.7%, which did not differ substantively from the observed mortality of 11.2% in the original, unrestricted analysis.

PCP severity and outcomes based on receipt of PCP prophylaxis

One potential mechanism by which knowledge of underlying HIV infection could produce differences in PCP severity or outcomes is through treatment of HIV (e.g., PCP prophylaxis or CART). Therefore, we examined whether the severity of HIV infection, severity of PCP, or hospital course and outcomes differed in patients with previously diagnosed HIV based on receipt of PCP prophylaxis. Of the 75 patients who reported taking PCP prophylaxis, 28 (37%) were taking trimethoprim-sulfamethoxazole, 35 (47%) were taking dapsone, four (5%) were taking atovaquone, and one (1%) was taking aerosolized pentamidine. Among patients who were previously diagnosed with HIV, there were no differences in CD4 count, HIV viral load, A-a O₂ gradient, or serum LDH based on the use of PCP prophylaxis (Table 4). However, those who were receiving PCP prophylaxis had other laboratory indices which suggested more severe organ dysfunction, including a significantly lower mean sodium and albumin and higher bilirubin. Patients who were receiving PCP prophylaxis had a mortality of 14.7% from PCP infection. In comparison, patients with previously diagnosed HIV who were not taking PCP prophylaxis had a mortality of 10.6%, with the lowest mortality of 7.4% in patients who were newly diagnosed with HIV.

Discussion

Although we are well into the third decade of the HIV epidemic, this study found that 23% of HIV-infected patients diagnosed with PCP at San Francisco General Hospital in the CART era came to medical attention late in the course of their HIV disease and were diagnosed concurrently with HIV and PCP. We found no significant change in the proportion of PCP cases with newly diagnosed HIV by study year. Patients with newly diagnosed HIV had more severely impaired oxygenation manifested by a higher alveolar-arterial oxygen gradient. Nevertheless, there were no differences in clinically important outcomes between the two groups except for an increased length of stay in patients newly diagnosed with HIV. To our knowledge, this is the largest single-center study of HIV-associated PCP in the CART era. No

study since the early AIDS epidemic has specifically evaluated the severity and outcomes of PCP in patients with newly diagnosed HIV [9].

One explanation for the similar outcomes between the two patient groups was that few patients with previously diagnosed HIV infection were taking PCP prophylaxis or CART, and therefore these individuals were no different physiologically or immunologically from those who were newly diagnosed with HIV. The median CD4 count was similar and not significantly increased even in the few patients who were taking CART. Because of the retrospective nature of our study, we were unable to verify whether patients who were classified as taking PCP prophylaxis or CART were adherent to their HIV regimens as prescribed.

We found that male gender and acquisition of HIV through high-risk sexual behavior were independently associated with newly diagnosed HIV. Prior studies of late HIV diagnosis, defined as diagnosis concurrent with an AIDS-defining condition or with a CD4 cell count less than 200 cells/ μ L, found that late diagnosis was less common in men who have sex with men compared to patients with other HIV risk factors (e.g. heterosexuals and IDU) [21–23]. However, the majority of these studies were performed in European cohorts where the prevalence of injection drug use was low [21–23]. One explanation for our findings may be that, compared to patients with high-risk sexual behavior, injection drug users in San Francisco have more interactions with the medical system before an overt AIDS-defining illness. Injection drug users have medical co-morbidities (e.g., skin and soft tissue infections, drug overdose or withdrawal) which require frequent contact with medical providers [24], resulting in repeated opportunities for HIV testing. Another possibility, described in a study of homosexual men in San Francisco, is that patients with high-risk sexual behavior in San Francisco are more reluctant to undergo HIV testing [25]. This may partly explain why male gender was associated with a new diagnosis of HIV infection, but there are clearly other confounding factors that we were unable to measure or account for. This study demonstrates a continued need for HIV education and earlier testing in all patients, but suggests that focusing on populations with high-risk sexual behavior may be particularly necessary and effective.

Although mortality was no different, physicians took longer to initiate PCP antimicrobial therapy in patients who were unaware of their HIV infection. Knowledge of underlying HIV infection clearly changes the physician's pre-test suspicion of different diseases. At SFGH, initiating PCP therapy is usually done empirically while awaiting a definitive microscopic diagnosis. Physicians likely initiated antimicrobial treatment for other types of pneumonia (e.g., bacterial pneumonia) but delayed PCP treatment in patients who were not known to be HIV infected. In addition, oxygenation was more severely impaired in patients with a new HIV diagnosis. Patients who are unaware of their HIV infection may take longer to present for medical care, either from failure to recognize the severity of their illness or because they lack familiarity with the medical system. A study from the pre-CART era described similar findings, with PCP patients newly diagnosed with HIV having lower PaO₂ compared to patients with known HIV [9]. We did not find any other differences in admission vital signs or laboratory indices of organ dysfunction between the two groups. However, we did not collect data on duration or severity of symptoms or results of chest radiography, and are therefore limited in our ability to conclude that there were no other clinically significant differences in PCP presentation or severity between patients with newly and previously diagnosed HIV.

This study has several other limitations. The study was retrospective and limited to patients with a confirmed diagnosis of PCP at a university-affiliated public hospital. Patients at other institutions who are presumptively diagnosed with PCP may have different clinical manifestations or severity of disease compared to patients in our cohort. Furthermore, there was the potential to have misclassified patients into incorrect categories of newly versus previously diagnosed HIV. While this is a valid concern, we believe that both systematic and

random miscategorization were unlikely. Our criteria for “newly” versus “previously” diagnosed HIV infection were stringent. Classification of HIV status was based on information from detailed histories. Patients classified as “newly” diagnosed with HIV infection had no past medical history related to underlying HIV infection and had no mention of definitive HIV infection in any admission note in the medical record. Subsequent progress notes documented a confirmatory HIV test after PCP diagnosis. For persons classified as “previously” diagnosed HIV infection, admission notes had to clearly document an earlier confirmatory HIV test either by patient self-report or by available medical records, typically with additional supportive information including CD4 count, history of opportunistic infections, and adherence to HIV treatment. Nevertheless, it is possible that a patient new to our medical system could have withheld information regarding an earlier diagnosis of HIV made at a different institution. This would bias our results toward the null hypothesis, but would not explain differences in oxygenation or HIV risk factors that we discovered between the two groups.

In conclusion, a significant number of patients at San Francisco General Hospital continue to be diagnosed with HIV only after presenting with PCP. The proportion of patients with concurrently diagnosed HIV and PCP each year remained unchanged during the study period. While oxygenation was more severely impaired in those with newly diagnosed HIV, there were no significant differences in mortality, ICU admission, or mechanical ventilation. Patients who acquired HIV via high-risk sexual activity were more likely to be newly diagnosed with HIV/AIDS than persons who acquired HIV via injection drug use. This study demonstrates a continued need for HIV education and earlier HIV testing, particularly in patients with high-risk sexual behavior.

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Figure 1.
Cases of HIV-associated Pneumocystis pneumonia at San Francisco General Hospital (n = 522), 1997–2006.

Table I

Demographics of 522 HIV-infected patients admitted with Pneumocystis pneumonia at San Francisco General Hospital, 1997–2006 ^a

	Newly diagnosed HIV/AIDS (n = 121)	Previously diagnosed HIV/ AIDS (n = 401)	p value
Age, years ^b	41.6 (8.4)	40.4 (7.8)	0.136
Gender			0.043*
Male	112 (93%)	343 (86%)	
Female	9 (7%)	58 (14%)	
Race/ethnicity			
White or Caucasian	51 (42%)	196 (49%)	Referent
Black or African-American	36 (30%)	116 (29%)	0.476
Other	34 (28%)	89 (22%)	0.132
HIV risk factor			
Injection drug use	13 (13%)	98 (27%)	Referent
Men who have sex with men	48 (48%)	140 (39%)	0.004 *
Heterosexual sex	24 (24%)	47 (13%)	0.001*
Multiple	15 (15%)	78 (21%)	0.361
Homeless	14 (12%)	57 (14%)	0.520

^aValues are shown as n (%) unless otherwise stated.

^bMean (standard deviation).

Table II

Multivariate analysis of characteristics associated with newly diagnosed HIV

	Odds ratio (95% CI)	p value
Age, per 5-year increase	1.10 (0.95–1.28)	0.199
Male gender	3.14 (1.25–7.86)	0.015 *
Race/ethnicity		
White or Caucasian	Referent	
Black or African-American	1.11 (0.61–2.01)	0.729
Other	1.05 (0.60–1.86)	0.858
HIV risk factor		
Injection drug use	Referent	
Men who have sex with men	2.42 (1.19–4.89)	0.014 *
Heterosexual sex	5.67 (2.45–13.1)	0.001 *
Multiple	1.42 (0.62–3.24)	0.407
Homeless	0.83 (0.39–1.78)	0.631

Table IIISeverity and outcomes of HIV infection and Pneumocystis pneumonia ^a

	Newly diagnosed HIV/AIDS (n = 121)	Previously diagnosed HIV/ AIDS (n = 401)	p value
Severity of HIV infection at presentation			
CD4+ cell count, cells/ μ L ^b	29 (15–58)	29 (12–64)	0.740
HIV viral load, log copies/ml ^c	5.2 (0.6)	5.1 (0.6)	0.383
Severity of PCP at presentation			
Serum LDH, IU/L ^b	357 (263–509)	358 (270–495)	0.521
A-a O ₂ gradient, mm Hg ^b	51 (40–68)	45 (33–60)	0.030 *
Hospital course and outcomes			
Time to PCP antimicrobial therapy, days ^c	1.5 (3.4)	0.8 (2.1)	0.011 *
Time to PCP diagnosis, days ^c	3.1 (3.7)	2.4 (4.1)	0.086
ICU admission	19 (16%)	62 (15%)	0.866
Mechanical ventilation	13 (11%)	53 (13%)	0.525
Hospital length of stay, days ^b	9 (7–16)	8 (6–15)	0.035 *
Mortality	9 (7.4%)	45 (11.2%)	0.231
Odds ratio, mortality (95% CI)	0.64 (0.30–1.34)	Referent	

^aValues are shown as n (%) unless otherwise stated.^bMedian (inter-quartile range).^cMean (standard deviation).

Table IV

Severity of HIV infection and Pneumocystis pneumonia in patients with previously diagnosed HIV according to PCP prophylaxis ^a

	Receiving PCP prophylaxis (n = 75)	Not receiving PCP prophylaxis (n = 322)	p value
Severity of HIV infection			
CD4+ cell count, cells/ μ L ^b	26 (12–62)	29 (12–64)	0.591
HIV viral load, log copies/ml ^c	5.0 (0.7)	5.1 (0.6)	0.246
Severity of PCP			
A-a O ₂ gradient, mm Hg ^b	46 (37–61)	45 (33–60)	0.573
Serum LDH, IU/L ^b	336 (263–490)	359 (278–499)	0.530
Laboratory values			
Sodium, mmol/L ^c	135 (4)	136 (4)	0.031 *
Albumin, g/dL ^c	2.9 (0.6)	3.1 (0.6)	0.026 *
Bilirubin, mg/dL ^c	0.8 (0.6)	0.6 (0.4)	0.002 *
Hospital course and outcome			
Time to PCP diagnosis, days ^c	2.9 (4.1)	2.3 (4.0)	0.201
Time to PCP antimicrobial therapy, days ^c	1.3 (3.0)	0.6 (1.8)	0.010 *
Mortality	11 (14.7%)	34 (10.6%)	0.312
Odds ratio, mortality (95% CI)	1.46 (0.70–3.03)	Referent	

^a Four patients for whom we were unable to determine use of PCP prophylaxis were omitted from this analysis.

^b Median (inter-quartile range).

^c Mean (standard deviation).