Clinical Neurology and Neurosurgery 172 (2018) 124–129

Contents lists available at ScienceDirect

Clinical Neurology and Neurosurgery

journal homepage: www.elsevier.com/locate/clineuro

Peripheral facial paralysis associated with HIV infection: A case series and literature review

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ARTICLE INFO

Keywords:
AIDS
HIV
Facial paralysis
Bell's palsy

ABSTRACT

Objective: The association between peripheral facial paralysis (PFP) and HIV infection has been scarcely explained. The authors aimed to describe the association between PFP and HIV infection status, along with the related co-morbidities and the outcomes of PFP, as well as the literature review on this topic.

Patients and Methods: All HIV-infected patients who experienced PFP, both before and after a positive HIV serology test, between January 2002 and June 2015 were retrospectively reviewed. The patients' demographic data, clinical characteristics, HIV co-morbidities and outcomes of PFP were summarized. A literature review of PFP in HIV infection was also performed. Descriptive statistics were used in the data analysis. The Mann-Whitney U test was performed to compare the parameters between the current case series and cases from literature review to determine statistical significant differences ($p < 0.05$).

Results: Sixteen patients (6 males and 10 females) were enrolled. Their median age was significantly higher than that of the cases in the literature review [46 (38, 49.75) *vs.* 33 (26, 41) years ($p = 0.004$)]. Nonetheless, a non-significant lower median CD4 count was observed [274 (134.5, 425.5) *vs.* 373 (265, 718) cells/μL ($p = 0.058$)]. In our series, unilateral PFP (UFP) was the most frequent, and it typically occurred long after a positive HIV serology test. However, bilateral PFP (BFP) was commonly found in the literature, and a simultaneous positive HIV serology test was reported in almost all cases. Consequently, most of our cases, except for those with HIV-related complications or co-morbidities, experienced a satisfactory recovery from PFP regardless of treatments received.

Conclusions: Most of the cases in our series were UPF with a higher median age and a lower median CD4 count. Moreover, facial paralysis presented later in our series than in the previously reported cases in the literature. Most of our cases experienced satisfactory recovery of facial weakness.

1. Introduction

A variety of neurological disorders are associated with HIV infection. These include encephalopathy, aseptic meningitis, myelitis, brachial plexitis, cranial neuritis, mononeuritis, polyradiculitis and polyneuritis [1,2]. However, the definite pathogenesis of these disorders remains unclear. Because the direct invasion of nervous system by HIV was proposed only in a single study [3], the host's immunological response during seroconversion period of HIV infection or a neurological complication during advanced HIV infection leading to peripheral facial paralysis (PFP) has been extensively investigated [1,4–6].

Facial nerve disorder is the most common cranial neuropathy associated with HIV infection. The association of PFP with HIV infection was first described by Snider et al. in 1983 [7]. Both unilateral PFP (UFP) and bilateral PFP (BFP) can develop at any time throughout the course of HIV infection *via* different pathogenic mechanisms depending on the concurrent competence of the host's immunity. It has been found that PFP presents as early as the seroconversion stage, as an immunologically-induced cranial neuropathy when the patient's immunity is still competent in the asymptomatic stage of HIV infection, or in the advanced stage, as a neurological complication when the host's immunity is markedly compromised by the HIV infection [4,8–10]. To date, only a single study has suggested that the highly neurotropic HIV strains could contribute to an earlier neurological presentation than do the immune-suppression HIV strains [11]. Hence, acute PFP resembling Bell's palsy (an idiopathic facial neuropathy) can particularly occur as a

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https://doi.org/10.1016/j.clineuro.2018.06.033
Received 10 January 2017; Received in revised form 23 June 2018; Accepted 30 June 2018
Available online 02 July 2018
0303-8467/

Table 1
Summary of current case series of facial palsy associated with HIV infection.

No.	Age/sex	Side	Onset related to HIV Dx (months)[x]	CD4 at onset (cells/uL)	Viral load (copies/ml)	Previous ARVs treatment	Treatments for facial paralysis	Outcome	Associated conditions
1	29 F	Right	+ 36	463	NA	No	Prednisolone	Full recovery	No
2	34 F	Right	+ 12	136	< 400	TDF, 3TC, EFV	Prednisolone	Full recovery	No
3	36 M	Right	+ 48	343	< 40	AZT, 3TC, NVP	Prednisolone	Full recovery	No
4	38 F	Left	0	161	NA	No	Prednisolone	Partial recovery	No
5	38 M	Right	+ 13	300	NA	TDF, 3TC, NVP	Prednisolone	Full recovery	No
6	39 F	Right	+ 2	185	NA	No	Acyclovir	NA	No
7	42 F	Left	−10	NA	NA	No	Prednisolone + Acyclovir	Partial recovery	No
8	44 F	Left	+ 240	18	199,000	TDF, 3TC, LPV/RTV	None	No recovery	Yes[a]
9	48 M	Left	+ 96	413	46,800	No	Acyclovir	No recovery	Yes[b]
10	49 F	Left	+ 120	1511	< 40	ddI, EFV, 3TC	NA	Full recovery	No
11	49 F	Right	−8	NA	NA	No	Prednisolone	Partial recovery	No
12	49 M	Right	+ 24	130	NA	TDF, 3TC, NVP	None	No recovery	Yes[c]
13	50 M	Left	+ 120	600	NA	Yes NA	Prednisolone	Full recovery	No
14	52 F	Bilateral	0	412	NA	No	Prednisolone	Full recovery	Yes[d]
15	54 F	Right	+ 11	248	470,338	No	Prednisolone	Full recovery	No
16	55 M	Bilateral	−48	64	225	No	B1 6 12	Partial recovery	No

a: Diffuse large B-cell non-Hodgkin lymphoma treated with the 4th cycle of CHOP (cyclophosphamide, adriamycin, vincristine and prednisolone).
b: Chronic leptomeningitis combined with unilateral Abducen nerve palsy.
c: Chronic leptomeningitis and ependymitis with bilateral Abducen nerve palsy.
d: Cephalic variant of Guillain Barre syndrome.
x: ± refers to after/before the diagnosis of HIV infection (months), 0: the presentation of facial palsy indicates HIV infection, NA: not available.
Abbreviations: ARVs: antiretroviral agents; TDF: tenofovir; 3TC: lamivudine; EFV: efavirenz; AZT: zidovudine; NVP: nevirapaine; LPV/RTV: lapinavir/ritonavir; ddI: didanosine.

first and sole presenting symptom of asymptomatic HIV infection [8,12,13]. This mode of presentation often results in the physician's under-recognition of the association with HIV infection and, therefore, a missing diagnosis of early HIV infection. Because the acute PFP's association with HIV infection has been rarely mentioned and Bell's palsy is generally more prevalent, the association between them is under-emphasized. Therefore, this case series was aimed to highlight the clinical characteristics of PFP in relation to the course of HIV infection as well as the associated co-morbidities, and the recovery of HIV-associated PFP. The English literature concerning HIV-associated PFP was reviewed and discussed as well.

2. Materials and methods

2.1. Study population

The medical records of the patients who presented with PFP associated with HIV infection (both before and after a positive HIV serology test) to our center, an 800-bed medical teaching hospital in southern Thailand, from January 2002 to June 2015 were reviewed. The authors enrolled cases from the hospital's computerized database system using ICD-10 codes as follow: B20-B24: HIV disease, G51: facial nerve disorders, G51.0: Bell's palsy, G51.8: other disorders of facial nerve, G51.9: disorders of facial nerve, unspecified, G 53: cranial nerve disorders, and G 59: mononeuropathy in diseases classified elsewhere. The patients' demographic data, clinical characteristics of PFP, time between the onset of PFP and the serological diagnosis of HIV infection, the most-recent CD4 count (within three months of the onset of facial paralysis), viral load, associated systemic or neurological co-morbidities, antiretroviral treatments, and treatments of PFP and outcomes were collected and summarized.

A literature review comprising all single-case reports and case series from 1987 to 2015 found in the PubMed.gov using the following keys words: facial paralysis, Bell's palsy, peripheral facial paralysis, HIV infections, human immunodeficiency virus infection, AIDS, and acquired immunodeficiency syndrome was conducted. Only the reports containing the complete clinical profiles required were chosen for comparison with our cases series.

2.2. Statistical analysis

The results were demonstrated using descriptive statistics like number, percentage and median with interquartile range (Q1, Q3). The Mann-Whitney U test was used to determine the significant difference of the clinical parameters between the current and the literature review cases ($p < 0.05$).

2.3. Ethics consideration

The protocol of this study was approved by the Ethics Committee of the Faculty of Medicine, Prince of Songkla University. The authors performed this study in accordance with the standards of the 1964 Declaration of Helsinki and its later amendments.

3. Results

A total of 1736 cases of PFP were diagnosed in our institution during the enrollment period. Sixteen (6 males and 10 females) of them were associated with HIV infection (0.92%). Meanwhile, fourteen of these cases had UFP (Right: Left = 8:6), and two cases had BFP. Their median age (Q1, Q3) was 46 (38, 49.7) years. Three cases (2 UFP and 1 BFP) developed facial paralysis before the diagnosis of the HIV infection over a period of time ranging from 8 to 48 months (case 7, 11, 16). Only 2 patients (1 UFP: case 4, and 1 BFP: case 14) were diagnosed HIV infection at the presentation of PFP, whereas the remaining cases acquired PFP within varying time intervals after the diagnosis of HIV infection. Overall, the median time (Q1, Q3) of the onset of PFP was 12.5 (0, 84) months after the positive serology test. A short course of oral prednisolone was the mainstay treatment in our cases. Acyclovir, either alone or combined with prednisolone, was an alternative. Most of