CASE REPORT

Physiotherapy intervention in two people with HIV or AIDS-related peripheral neuropathy

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INTRODUCTION

As people with HIV or AIDS are living longer, they are beginning to have more chronic health problems. Peripheral neuropathy is one such condition associated with functional limitations. In this case report, the physiotherapy intervention and its outcome for two individuals with HIV-related peripheral neuropathy are described. The patients were two males, ages 37 and 52, with symptoms of HIV-related peripheral neuropathy for three and six years, respectively. Both had decreased functional ambulation as a result of the neuropathy. They received physiotherapy intervention using joint mobilization, soft tissue mobilization, microcurrent, stretching and instruction in self-management. Treatment continued two to four times monthly for 12 and 18 months, respectively. Both patients showed decreased symptoms of neuropathy and increased functional abilities and independence after physiotherapy intervention. One patient returned to community ambulation and the other returned to full-time employment. Physiotherapy intervention may be effective in the management of HIV-related peripheral neuropathy.

As a result of new drug therapies and revised treatment guidelines, HIV-related mortality has decreased (Wood et al., 2000), and AIDS is becoming a chronic rather than a terminal disease. Patients will, therefore, begin to have more chronic health problems (McClure, 1993). One such health problem related to HIV or AIDS is peripheral neuropathy. Here, we describe the physiotherapy intervention and the changes in functional abilities in two individuals with HIV-related peripheral neuropathy.

The Centers for Disease Control and Prevention estimate that by the end of 2001 there were 344 178 people living with AIDS in the USA, an increase of 5% over 2000 (HIV/AIDS Surveillance Report, 2001). Both the incidence of AIDS and the number of deaths from AIDS are decreasing. In 1996, the number of people dying from AIDS decreased for the first time since the epidemic was identified. Since that time, the number of deaths from AIDS has continued to decline (HIV/AIDS Surveillance Report, 2001). Worldwide, however, the incidence of HIV infection and AIDS continues to increase. At the end of 2002 there were 42 million people living with HIV or AIDS (UNAIDS, 2002).

Because people diagnosed with AIDS are living longer, it has become necessary to deal with health problems that are not considered

life-threatening. Peripheral neuropathy is one such condition, affecting quality of life, not survival itself (Klaus, 1996). The most common form of neuropathy in patients with HIV infection is distal symmetric peripheral neuropathy or distal sensory polyneuropathy (DSPN) (Cornblath and McArthur, 1988; Simpson and Olney, 1992; Norton et al., 1996; Markarian et al., 1998; Tagliati et al., 1999). It is estimated that neuropathy occurs to some degree in 30–50% of patients with AIDS (Cornblath and McArthur, 1988; Bouhassira et al., 1999). DSPN occurs most frequently in advanced HIV infection and is inversely correlated with CD4 counts (Norton et al., 1996; Tagliati et al., 1999). CD4 count, the number of CD4 cells per cubic millimetre of blood, reflects how functional the immune system is. In healthy individuals, CD4 count ranges from 500 to 1800 cells/mm^3. In patients with HIV infection, CD4 count can vary from normal to <50 cells/mm^3. When the CD4 count falls below 200, a patient with HIV infection is diagnosed with AIDS (Centers for Disease Control, 1992).

The cause of peripheral neuropathy in people with HIV or AIDS is not known; however, several theories have been suggested. In a study by Norton (1996), it was proposed that neurotoxins produced by infection with *Mycobacterium avium* complex, a disorder that affects people with HIV or AIDS, may cause peripheral neuropathy. Infection with cytomegalovirus (CMV) has also been linked to DSPN (Mastroianni et al., 1994; Said et al., 1997). More recently, it has been found that many of the anti-retroviral medications used to treat HIV are associated with DSPN (Said et al., 1997; Markarian et al., 1998; Wulff et al., 2000). Sixteen to 20% of patients taking ddI (didanosine) reported this side effect (Perry and Balfour, 1996; Project Inform, 1998), whereas 22–35% of those taking ddC (zalcitabine) and 15–21% of those using d4T (stavudine) developed peripheral neuropathy (Project Inform, 1998). Less commonly, it is reported with use of 3TC (lamivudine) (Said et al., 1997; Markarian et al., 1998). There are other factors possibly associated with the disorder, including nutritional deficiencies such as inadequate vitamin B12 or thiamine, alcohol abuse and co-morbidities, such as diabetes mellitus, hypothyroidism and chronic renal failure (Klaus, 1996; Thomas et al., 1997; Tagliati et al., 1999).

Peripheral neuropathy is diagnosed by history and physical examination. Symptoms of DSPN are usually symmetrical in the feet but may be more pronounced on one side and can occur in the hands as well. Symptoms generally progress from distal to proximal. Signs and symptoms include burning, paresthesia, allodynia or dysaesthesia, hyperaesthesia, painful numbness, decreased temperature and vibratory sensation in a stocking or glove distribution, areflexia at the ankles, trophic changes (hair loss, oedema, thinning of the skin), mild weakness of the intrinsic musculature of the feet and gait abnormalities associated with all of the above (Parry, 1988; Simpson and Olney, 1992; Simpson and Tagliati, 1994; Klaus, 1996; Markarian et al., 1998; Tagliati et al., 1999). Nerve conduction studies can be used to diagnose DSPN. Decreased amplitude of nerve action potentials can be seen in patients with DSPN; however, nerve conduction velocities may be normal or only slightly decreased (Simpson and Olney, 1992; Bouhassira et al., 1999).

The medical management of DSPN is limited. Tricyclic antidepressants, such as amitriptyline, have been used with some success. Neurontin (gabapentin), an anticonvulsant medication, provides some decrease in symptoms, but its effects appear to decrease over time. Narcotic analgesics, non-steroidal anti-inflammatory agents and

topical capsaicin provide incomplete pain relief (Simpson and Olney, 1992; Klaus, 1996; Markarian et al., 1998; Wulff et al., 2000). Acupuncture has been used with varying results. A large study by Shlay (1998) found that neither acupuncture nor amitriptyline was more effective than a placebo in decreasing the symptoms of peripheral neuropathy. Shlay (1998) concluded that 'additional clinical trials are needed because there are no effective treatments for this chronic debilitating condition'. Said (1997) advocates pharmacological intervention in combination with 'psychological support and physiotherapy'. Here, we described the physiotherapy intervention and document functional changes in two individuals with HIV-related peripheral neuropathy. The names used in the case descriptions are not the patients' actual names.

CASE DESCRIPTIONS

Subject 1

S was 37 years of age when he first presented to our clinic for treatment. He had been diagnosed with AIDS for three years, and was known to have been HIV-seropositive for one year beforehand. He had been disabled for the past year because of frequent illnesses, pain in his feet and failing eyesight secondary to CMV retinitis. His viral load was 7700 copies/ml and his CD4 count was 107 cells/mm^3, reflecting fairly well-controlled HIV replication but a compromised immune system. To control the HIV infection and the opportunistic infections as well as the side effects of the medications, S had been prescribed 13 different drugs. These medications, their uses and possible side effects are summarized in Table 1. S was

TABLE 1: Medications, Subject 1

Medication	*Use**	*Side effects**
Invarise	HIV infection	Nausea, diarrhoea, abdominal pain
Novir	HIV infection	Oral tingling, diarrhoea, nausea, vomiting, fatigue, flushing
d4T	HIV infection	Peripheral neuropathy, neutropenia
Viramune	HIV infection	Rash, fever, nausea, headache, increased liver enzymes
Sulfadiazine	Urinary tract infection toxoplasmosis	Anaemia, decreased white cell count, decreased platelets, headache, peripheral neuropathy, depression
Daraprim	Toxoplasmosis	Anorexia, vomiting, anaemia, decreased white cell count
Leucovorin	Anaemia, antibiotic toxicity	Allergy to drug
Ganciclovir	Cytomegalovirus retinitis	Decreased white cell count, decreased platelets
Fluconzale	Crytoccal meningitis, candidiasis, histoplasmosis	Nausea, headache, rash, vomiting, abdominal pain, diarrhoea
Pred Forte	Ocular diseases	Blurred vision
Darvocet	Pain	Light-headedness, drowsiness, nausea, vomiting
Tylox	Pain	Light-headedness, drowsiness, nausea, vomiting
Amitriptyline	Depression, insomnia, pain	Dizziness, dry mouth, drowsiness, headache

**Project Inform (1998)*

not prescribed any new medications during the course of his physiotherapy care, and did not discontinue any except the narcotic analgesics. He did, however, have vitracert, a slow-release gancyclovir pellet, inserted into each eye on two occasions, in an attempt to control CMV retinitis.

S was diagnosed with peripheral neuropathy one month before beginning physiotherapy. He described constant painful numbness over both the plantar and dorsal surfaces of the feet to the ankles. He had intermittent shooting, electrical pain in the toes. He woke at night with allodynia or dysesthetic pain in both feet from contact with the sheets. These symptoms had been gradually increasing over the past three years, with marked increase over the three to four months before initiating physiotherapy treatment. The symptoms were aggravated by walking even short distances, such as crossing the room. Because of the limitations in his ability to ambulate, S required assistance with some of his activities of daily living. He reported that warm water and rubbing the feet eased his pain.

Observational gait analysis revealed short stride length bilaterally. His gait pattern was flat-footed with almost no heel strike or push-off noted. On physical examination, S had allodynia with light touch on both the plantar and dorsal surfaces of the feet, right greater than left. Manual muscle testing revealed strength of 4/5 throughout the lower extremities, with the exception of plantar flexion, which was 3/5 bilaterally. Upper extremity strength was 5/5. The toe extensor musculature was contracted, drawing the toes into hyperextension at the metatarsal–phalangeal joints. Testing of the passive accessory movements revealed hypomobility in the intertarsal joints. Temperature and colour of the feet were normal. Ankle jerk was absent bilaterally.

Subject 2

At the time of initial contact, R was 52 years of age. He had had known HIV disease for a total of 12 years, and had been diagnosed with AIDS for the past eight years. He had been disabled for two years because of frequent illness and pain in his feet with weightbearing. His viral load was undetectable, indicating that the virus was well controlled, and his CD4 count was 250 cells/mm^3. Table 2 summarizes his medications. He was prescribed no new medications during this physiotherapy episode of care; amitriptyline was the only medication discontinued.

R had been diagnosed with peripheral neuropathy for three years when he began physiotherapy treatment, although his symptoms had been present for six to seven years. At the time of his initial contact R described constant numbness and tingling on the plantar surfaces of the feet, stiffness in the feet and toes and intermittent sharp, shooting pains through the distal feet and toes. He reported that his symptoms were aggravated by walking short distances and eased slightly by amitriptyline.

R exhibited little push-off during observational gait analysis. Physical examination revealed decreased sensation to light touch on the plantar surfaces of the feet and reports of increased tingling with palpation. Manual muscle testing of the lower extremities revealed strength was 4/5 to 4+/5. Upper extremity strength was 5/5. Passive accessory movements were hypomobile in the intertarsal, tarsal–metatarsal, intermetatarsal, metatarsal–phalangeal and inter-

TABLE 2: Medications, Subject 2

Medication	*Use**	*Side effects**
Crixivan	HIV infection	Nausea, elevated bilirubin, abdominal pain, headache, diarrhoea, vomiting, kidney stones, fatigue, insomnia
d4T	HIV infection	Peripheral neuropathy, neutropenia
3TC	HIV infection	Nausea, vomiting, headache, fatigue, diarrhoea, peripheral neuropathy, decreased white cell count, anaemia
Dapsone	*Pneumocystis carini*, pneumonia, mycobacterial infection, protozoal infection	Anaemia, peripheral neuropathy, weakness nausea, vomiting, abdominal pain, pancreatitis, vertigo, blurred vision
Amitriptline	Depression, insomnia, pain	Dizziness, dry moth, drowsiness, headache
Erythropoietin	Anaemia	Fever, fatigue, headache, cough, diarrhoea, rash, congestion, nausea, shortness of breath, muscle weakness, dizziness

* Project Inform (1998)

phalangeal joints. Passive great toe extension was to neutral only in both feet. The feet were cool to the touch and normal in colour. Ankle jerk was absent bilaterally.

INTERVENTION TECHNIQUES

There has been little research on physiotherapy intervention in people with peripheral neuropathy. McReynolds (1995) reported improved gait and decreased pain perception in individuals with HIV-related peripheral neuropathy after treatment with microcurrent. McReynolds (1995) cites clinical experience as the basis for combining microcurrent with joint mobilization and massage in decreasing pain and increasing function. In a population of patients with anti-retroviral drug-induced neuropathy, Galantino et al. (1999) documented improvement in functional activities and decreased muscle response latency after treatment with electro-acupuncture.

The patients in the cases presented here were treated using a combination of joint mobilization, soft tissue mobilization, microcurrent, stretching and instruction in a home programme.

Joint mobilization

Both S and R presented with decreased mobility in the joints of the feet. To promote more normal gait and decrease pain related to stiffness, joint mobilization or passive accessory movement, as defined by Maitland (1993), was used. Specific techniques were grade III and grade IV postero-anterior and antero-posterior movements of tarsal on tarsal, tarsal on metatarsal, metatarsal on metatarsal, metatarsal on proximal phalanx and phalanx on phalanx. Longitudinal distraction of the proximal phalanx on metatarsal was also used.

Soft tissue mobilization

Massage has long been associated with mechanical and physiological changes, including increased venous flow, increased flow of nutrients, removal of waste products and metabolites, pain relief and increased

extensibility of connective tissue (Tappan, 1988; DeDomenico and Wood, 1997). Soft tissue mobilization was used in these cases to decrease sensitivity, improve soft tissue pliability and promote circulation. Both petrissage and effleurage were applied to the limit of patient tolerance, working from distal to proximal. Firm touch was best tolerated.

Stretching

In the first subject, S, the toe extensor musculature was tight and he lacked heel strike during ambulation. Because of these findings, two muscle groups, the toe extensors and the triceps surae, were passively stretched. The toe extensor musculature was stretched beginning with the ankle in neutral and progressing to plantar flexion. The triceps surae musculature was stretched with the knee in extension and the ankle in maximum dorsiflexion. The elongated positions were held for 30 s. Following a brief rest, the stretch was repeated. Passive range of motion with overpressure was used to regain mobility in the joints of the feet.

Microcurrent

Although the efficacy of microcurrent in pain management is poorly documented, it has been shown to decrease pain in individuals with degenerative joint disease of the temporomandibular joint (Bertolucci and Grey, 1995). In the cases presented here, microcurrent was applied using the Ultima Xs Microamperage unit (American Imex, Irvine, CA). Electrodes were placed over the acupuncture points for chronic pain in the lower extremity described by Wu and Carroll (1982). These points are: gall bladder 34 (GB34) anterior and inferior to the fibular head; bladder 40 (B40) at the centre of the popliteal crease; spleen 5 (Sp5) anterior and inferior to the medial malleolus; and bladder 60 (B60) posterior to the lateral malleolus. The microcurrent unit was set at a frequency of 0.5 Hz and intensity was sub-threshold for sensation. At each treatment session, microcurrent was applied for 15 min to each lower extremity concurrent with the joint and soft tissue mobilization. In both cases, however, the patients initially presented with allodynia or increased tingling with touch and were unable to tolerate these manual techniques. Therefore, microcurrent was applied for five to ten minutes before beginning joint and soft tissue mobilization. This pre-treatment with microcurrent was only necessary for the first two sessions.

Home programme

Both subjects were instructed in a home programme to be performed once daily. This included stretching of the toe extensor and triceps surae musculature for two bouts of 30 s for each muscle group. The toe extensors were stretched with the foot in plantar flexion, moving the toes into flexion. The triceps surae musculature was stretched in standing, legs astride, with the knee of the posterior leg extended; the patient leaned forward, keeping the heel on the floor. Desensitization techniques for the feet using a terry towel after a warm shower or bath were also to be performed daily.

OUTCOMES

Subject 1

S was treated once every other week as he had to travel for one and a half hours each way to attend the physiotherapy clinic. Because of his poor eyesight, he relied on his parents to drive him to his appointments. He reported decreased pain and numbness

following the first treatment session. After two months (five treatment sessions) he exhibited inconsistent heel strike and push-off during gait and reported decreased pain. At six and at 12 months after initiation of physiotherapy, S had clearly apparent changes in functional abilities. He exhibited normal stride length, was able to ambulate within the community without pain and was no longer taking any narcotic analgesics. Results of treatment are summarized in Table 3. S was lost to follow-up after one year of treatment.

Subject 2

R was treated once a week. After five treatment sessions he reported decreased pain and decreased use of amitriptyline. Six months after initiation of physiotherapy intervention, R was able to return to work part-time. By 12 months and continuing to 18 months, he was working full-time as a carpenter. Approximately 12 months into the intervention, the intrinsic musculature of both feet began to atrophy. This progressed over the next six months despite attempts to

TABLE 3: Outcomes, Subject 1

	One treatment	*Five treatments*	*Six months*	*One year*
Sensation	↓ allodynia in both feet		Resolution of painful numbness	Maintaining positive changes in sensory signs and symptoms
Pain		↓ Frequency electrical pain in toes	Resolution of electrical pain except left great toe	Maintaining positive changes in pain level
Gait		Inconsistent heel strike and push-off	Normal stride length	Heel strike and push-off, normal stride length
Function		Able to walk one block without pain, sleeping through the night	Able to walk several blocks without pain, independent in ADLs	Travelling with difficulty 2^0 vision, maintaining independence
Medication			Taking no narcotic analgesics	Taking no pain medication

strengthen these muscles. Function did not appear to be affected by this occurrence. Outcomes of his treatment are presented in Table 4.

DISCUSSION

Recently, there has been a stigma associated with HIV disease and AIDS. Fear of contracting the disease, perceptions of entitlement to care and attitudes about its mode of transmission (Herek and Capitanio, 1993; Dimick et al., 1996; West et al., 1996) can all be associated with fear of touching or reluctance to touch people with AIDS. A recent study on physiotherapists' use of touch identified six common types, including touch to provide a therapeutic intervention and caring touch. In addition, the authors described many more combinations of touch used by physiotherapists (Roger et al., 2002). Because the physiotherapy intervention described here is focused on touch, its role as an integral part of the treatment should be considered.

This patient population is very knowledgeable about HIV disease and therapeutic innovations for its management. Physiotherapists' knowledge of the disease progression, opportunistic infections and the medications used in treatment of HIV disease, as well as familiarity with the mani-

TABLE 4: Outcomes, Subject 2

	Five treatments	*Six months*	*12 months*	*18 months*
Sensation		↓ sensation to light touch		Maintaining positive changes sensory signs and symptoms
Pain	↓ Frequency of sharp shooting pain	Resolution of electrical sharp shooting pain		Maintaining pain-free status
Gait	Inconsistent push-off during gait			Lacks full push-off during gait
Function		Return to work part-time as a carpenter	Return to work full-time as a carpenter	Maintaining work status
Medication	↓ use of amitriptyline		Occasional amitriptyline	Taking no amitriptyline
Other changes			Atrophy of foot intrinsic muscles	Persistent atrophy of intrinsics

festations of neuropathy itself, is helpful in gaining the trust of the patient with AIDS. The role of collaboration and self-efficacy should also be considered in these cases. The physiotherapist plays a significant part in teaching patients about self-management of their symptoms. This may enhance their self-confidence and further contribute to functional gains (Jensen et al., 2002; Jones et al., 2000).

CONCLUSION

In the cases of these two patients with HIV- or AIDS-related peripheral neuropathy, the combination of joint mobilization, microcurrent, soft tissue mobilization and self-management appears to be an effective intervention when no other methods have provided significant relief. In the first subject, signs and symptoms included pain, allodynia, gait abnormality and contractures. By decreasing these signs and symptoms, the functional limitations — in this case inability to walk one block or sleep through the night — were minimized. As a result, quality of life improved allowing the patient to travel and become more independent. In the second case, numbness, pain, stiffness and gait abnormalities were addressed. Decreasing these signs and symptoms allowed increased functional ambulation. Because of the increased functional abilities, the patient was able to return to work, decreasing disability and promoting independence.

The procedures outlined here were used to decrease signs and symptoms and increase function in two patients with HIV- or AIDS-related peripheral neuropathy. However, further research is needed, using both qualitative and quantitative methods, to demonstrate the long-term effectiveness of physiotherapy management of individuals with peripheral neuropathy related to HIV or AIDS and diabetes mellitus, as well other conditions associated with chronic illnesses.

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