



CASE REPORT

Auricular Therapy for Treating Phantom Limb Pain Accompanied by Jumping Residual Limb: A Short Review and Case Study

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Received: August 5, 2020 / Accepted: January 15, 2021 / Published online: March 4, 2021
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ABSTRACT

Phantom limb pain (PLP) is a common complaint among patients after amputation, while jumping residual limb is a rare post-amputation complication, they rarely happen at the same time and both remain difficult to manage. At present, there is a paucity of literature on this topic, and no treatment has been proven effective for treating both of them. In the present brief report, we described a patient who developed severe PLP accompanied by jumping residual limb after below-the-knee amputation and she was treated by auricular therapy (AT) with satisfactory effect.

Keywords: Auricular therapy (AT); Involuntary movements; Jumping residual limb; Movement disorders; Phantom limb pain (PLP)

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Key Summary Points

Why carry out this study?

Phantom limb pain (PLP) is a common complaint among patients after amputation, while jumping residual limb is a rare post-amputation complication—they rarely happen at the same time and both are difficult to manage.

The aim of this article is to describe a successful auricular therapy (AT) treatment of severe PLP accompanied by jumping residual limb in a 28-year-old female after below-the-knee amputation.

What was learned from the study?

After a 3-week period AT treatment, the intensity of pain, the frequency of jumping stump attacks, and the dosage of analgesic were significantly reduced, meanwhile, the overall condition of the patient was improved.

This case report demonstrates that AT may be an effective alternative therapy for PLP accompanied by jumping residual limb.

We believe that this is the first report of AT for treating PLP accompanied by jumping residual limb.

DIGITAL FEATURES

This article is published with digital features, including a summary slide and video, to facilitate understanding of the article. To view digital features for this article go to <https://doi.org/10.6084/m9.figshare.13574603>.

INTRODUCTION

Phantom limb pain (PLP) is defined as persistent painful sensations perceived in the missing portion of the amputated limb [1]. It is a common complication and the prevalence may be as high as 75–80% in amputees [2, 3]. Most of the amputees suffer from burning, cramping, pinprick, stabbing, itching, or other sensations of pain in the first few weeks following amputation. The onset of pain may be elicited by environmental, emotional, or physical changes. Although the frequency and intensity of pain diminish over time in most PLP patients, severe pain can persist in about 5–10% of amputees [4], affecting individuals' subjective well-being, as well as both physical and mental components of quality of life [5]. Regarding etiology and pathogenesis, PLP is regarded as one kind of neuropathic pain and is caused by a lesion of the somatosensory nervous system. At present, given to a lack of robust evidence to support the efficacy of many treatments, it remains difficult to effectively manage.

On the other hand, movement disorders following amputation is a rare complication and can manifest as the jumping residual limb phenomenon, the nature of which is a form of peripheral myoclonus and is characterized by sudden, brief, and sometimes repetitive muscle contractions, jerking, tremulousness or spasms of the residual limb [6]. Although it is reported that only 1% or less of these post-amputation patients experience spontaneous, involuntary, autonomous movements of the residual limb [7], it is significantly disturbing to patients when it does occur. The “jumping residual limb” syndrome is generally regarded as a form of spinal reflex myoclonus limited to the amputation site. Similarly, the pathophysiology has not been completely elucidated and there

are currently no guidelines or standardized treatment for it.

In clinical settings, PLP and jumping residual limb rarely occur at the same time. Hence, we presented a case of a 28-year-old female suffering from phantom pain accompanied by intermittent myoclonus in her residual limb after amputation. Due to failure to respond to oral medication, we attempted the use of auricular therapy (AT) and achieved satisfactory symptom improvement in this patient.

Pathogenesis of PLP

The underlying mechanisms of PLP have commonly been classified according to the level of the nervous system: peripheral or central. On the one hand, peripheral mechanisms include mechanical stimulation, circulating catecholamines, proinflammatory immune factors, and other sensitive neurochemicals activating the residual limb neuromas [8]. On the other hand, the neural mechanism of PLP is closely related to the brain reorganization after denervation in the sensorimotor system [9]. The sudden absence of afferent nerves following amputation resulted in many changes at the peripheral and spinal levels. It is speculated that the changes of sensory field and cortical reorganization observed after amputation are related to PLP. This may explain why a number of amputees relieve PLP by rubbing their entire limbs, thus stimulating normal afferent inputs at cortical, spinal, and peripheral levels.

As an intractable condition, PLP has been studied by many researchers. As described above, although several hypotheses have been put forward, the accurate mechanisms underlying PLP remain inconclusive. Further in-depth research is warranted.

Treatment of PLP

Currently, PLP remains intractable to be managed. Conventional treatments include pharmacotherapy, surgical intervention, and adjunctive therapy.

Regarding pharmacotherapy, there are a variety of medications to choose from, which

include tricyclic antidepressants, opioids, and nonsteroidal anti-inflammatory drugs (NSAIDs), etc. The most commonly used first-line medication is gabapentin, but a systematic review found that it is only beneficial for short-term analgesic efficacy. Tricyclic antidepressants are also commonly used, however, its role against PLP is not definitive [10] and it has been shown to fail to effectively control the pain [11, 12]. Acetaminophen and NSAIDs are also prevalently used for PLP [13]. Opioids (levorphanol, oxycodone, methadone, and morphine) provide analgesia without causing the loss of proprioception, touch, or consciousness by binding to peripheral and central opioid receptors. Calcitonin can also be used for PLP, but its therapeutic effect is mixed and the mechanism is unclear [14, 15].

Regarding surgical intervention, nerve resection, nerve root cutting, and anterior column cutting are gradually replaced by interventional surgery because of their large trauma and various complications. As a surgical technique, targeted muscle reinnervation (TMR) refers to transferring the residual peripheral nerve to the redundant motor nerve of target muscle, restoring physiological continuity, encouraging organized nerve regeneration, so as to reduce and potentially prevent nerve confusion and directional growth [16] and it presents a surgical solution to this problem as either a primary or secondary intervention [17]. Image-guided percutaneous nerve cryoablation is feasible and safe and may represent a new efficacious therapeutic option for patients with PLP [18]. Brain and spinal nerve stimulation therapy includes invasive intervention of deep brain stimulation (DBS), spinal cord stimulation (SCS), motor cortex stimulation (MCS), and dorsal root ganglion (DRG) stimulation. For those with chronic PLP, transcranial direct current stimulation (tDCS) and noninvasive interventions repetitive transcranial magnetic stimulation (rTMS) are targeted choice. However, current studies on the efficacy and safety of nerve stimulation therapy have not provided reliable results.

Regarding adjunctive therapy, it includes mirror therapy (MT), transcutaneous nerve stimulation, electroconvulsive therapy,

biofeedback, acupuncture, massage, and so on [19]. MT was first reported in 1996, which helped to reduce PLP by eliminating the separation of visual proprioception in the brain [20]. One randomized study [21] also demonstrated its effectiveness and showed that the pain relief associated with MT may be due to the activation of mirror neurons in the hemisphere of the brain contralateral to the amputated limb. Local anesthetics can block peripheral nerves, plexi, dorsal roots, as well as the sympathetic nervous system, however, the effects are usually short-lived. Although longer-lasting blocks may increase the duration of anesthesia, they have the risk of irreversible functional impairment. Long-term epidural administration, such as steroids, midazolam, clonidine and opioids, usually results in serious side effects. Selective nerve root injection of ozone is a safe and effective new approach for the treatment of PLP [22]. Eye movement desensitization and reprocessing (EMDR) therapy is believed to reduce PLP through emotional processing of “painful memories”, which are proposed to maintain PLP [23]. The results show that acupuncture is also beneficial to PLP [24–27]. Acupuncture stimulation in the artificial hand has an enhanced sensory feedback mechanism, which may be a new approach for PLP [28].

Over the past five decades, researchers have explored how PLP might be treated via medication, surgery, and various other approaches [8]. Nevertheless, there is still little high-quality evidence available for PLP treatments and most clinical trials are unconvincing.

Pathogenesis of Jumping Residual Limb

The jerking of the residual limb is generally associated with severe residual limb pain and can occur at variable periods after amputation. Sometimes it occurs in the immediate postoperative period and persists over weeks to months, but they may also develop gradually after a variable latent period and persist indefinitely. Jumping residual limb is usually precipitated by voluntary movement, but can also be triggered by cutaneous stimuli. At present, the pathophysiology of “jumping residual limb” is

still unclear. The latest research has shown that “jumping residual limb” represented a clinical entity in a series of dyskinesia, which was related to peripheral nerve injury or more common peripheral-induced dyskinesia. Peripheral trauma caused by amputation is likely to alter sensory input and induce central cortical and subcortical reorganization, resulting in dyskinesia [29, 30]. During amputation, nerve damage is inevitable. They amplify or distort natural signals and provide exaggerated input to the central nervous system [12]. Some researchers suggest that the neural anatomical network connecting the somatic and sympathetic nervous systems and participating in substance P directly initiates the abnormal depolarization of anterior horn cells. Myoclonus is caused by the transient activation of a muscle, resulting in convulsions in the affected area. This activation may arise from the cortex, subcortical structure, spinal cord, or nerve root and plexus [31–33]. Although some progress has been made in the investigation of its mechanism, jumping residual limb still needs to be further researched.

Treatment of Jumping Residual Limb

The mechanism underlying jumping residual limb is still unclear, nevertheless, several treatment methods have been applied to treat it, such as gabapentin, pramipexole, benzodiazepine, injection of botulinum toxin, and phenol [6, 33–39]. However, current available literature on treating jumping residual limb is all case reports, and there are no strict randomized controlled trials to support their effectiveness. Because of the rarity of jumping residual limb phenomenon and the complexity of its pathogenesis, to date, no consensus-based best practice is recommended.

The Relationship Between PLP and Jumping Residual Limb

Typically, but not in all cases, patients with jumping residual limb experience associated neuropathic pain, and phantom or residual limb pain. According to the literature research, rare patients present jumping residual limb and

PLP simultaneously. A close relationship between pain and the movement disorder could lead to the theory that pain might be a specific trigger for subsequent development and persistence of involuntary movements following peripheral injury [32]. Yet, there is no sufficient evidence to prove that one treatment is effective to treat PLP accompanied by jumping residual limb.

Cases of PLP Accompanied by Jumping Residual Limb from the literature

Jumping residual limb is an uncommon clinical phenomenon, especially in those accompanied by PLP. There is a paucity of published literature about it, only several available case reports on this topic focused on the intractability in treating both of them simultaneously, which are summarized in Table 1.

Compliance with Ethics Guidelines

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. Institutional review board approval is not required for a case report.

CASE PRESENTATION

A 28-year-old female patient was admitted to the Acupuncture Ward of the Third Affiliated Hospital of Zhejiang Chinese Medical University in December, 2019, due to severe PLP accompanied by jumping residual limb. Her medical history was as follows. She had undergone a below-the-knee amputation of the right leg due to a severe carbon fire burn in November 2019. After amputation, she began to suffer from persistent unbearable severe PLP, which attacked as a knife cutting her leg and the most severe pain site was located in her phantom foot, accompanied by intermittent involuntary movements of the residual limb (typical onset of symptoms was displayed in Supplemental Video 1).

Table 1 Case reports on PLP accompanied by jumping residual limb of published literature

Article	Age	Gender	Reason of amputation	Site of amputation	Occurrence time of the jumping residual limb	Manifestations of the jumping residual limb	Treatment history	Effective treatment	Effect
Briand [6]	57	Male	Aneurysm infection after right popliteal artery trauma	Right leg below the knee	1 month after amputation	Repetitive muscle contraction	Carbamazepine, levetiracetam, pregabalin, clonazepam, lidocaine, and dexamethasone injection	Botulinum neurotoxin type A injection	Success
Seidel [33]	68	Male	Arterial hypertension and peripheral arterial occlusive	Left leg in the middle of the thigh	3 years after amputation	Involuntary convulsion	-	Pramipexole	Reduced the involuntary movement, not influenced PLP
Iacono [39]	54	Male	Crush injury	Right leg below the knee	1 month after amputation	Flexion and adduction of the hip joint and flexion of the knee, followed by extension of the hip and knee, and adduction of the hip	Local and regional anesthesia and transcutaneous nerve stimulation, interfascicular coagulation of anterior and posterior tibial nerves, dorsal root entry zone (DREZ) lesions	Baclofen	The involuntary movement stopped
Iacono [39]	45	Male	Electric shock injury	Bilateral upper limb and left upper knee	8 years after amputation	Severe, brisk adduction and flexion of the left shoulder residual limb	diazepam, phenytoin sodium, cyclobenzaprine, meperidine carbamazepine, morphine, methocarbamol, codeine, clonidine, phenothiazines, clorazepate, chlordiazepoxide, ibuprofen and so on	Baclofen	Abolished the involuntary movement, PLP did not change

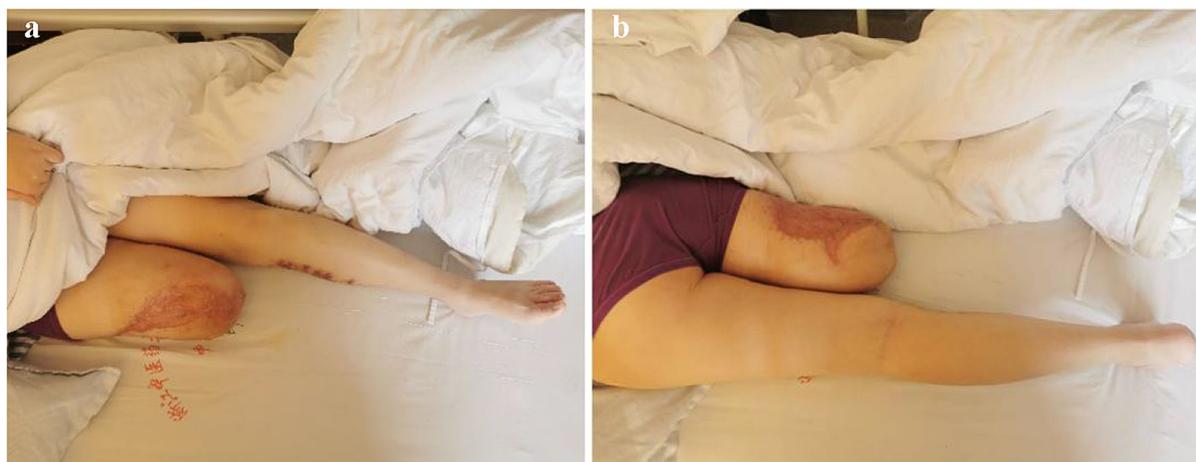


Fig. 1 **a** The patient's condition after amputation (*front view*). **b** The patient's condition after amputation (*back view*)

The pain was usually aggravated at night, without sweat or palpitation. These movements attacked about 13–15 times a day, which lasted from minutes to hours and always worsen the PLP. They were aggravated by action at daytime and usually disappeared during sleep. The pain was so severe that it seriously affected her sleep, the jumping residual limb brought her a lot of inconvenience and discomfort, and psychiatric scale assessment showed very high levels of anxiety and depression (Table 3).

On physical examinations at admission, the scar of the amputation was fully healed and freed from major adhesions (Fig. 1a and b). Hyperpathia was detected in the right leg. Patches of hyperalgesia were measured at the tip of the residual limb. Apart from involuntary, rapid, rhythmic myoclonic jerks of the residual limb, the neurologic examination showed no abnormalities. The results of routine biochemical and hematologic studies were normal. At present, the effect of pharmacological treatment of gabapentin tablets (0.9 g bid plus 0.6 g qn), duloxetine hydrochloride capsules (60 mg bid) was unsuccessful and she had to inject tramadol (100 mg qn) to relieve pain. In addition, she took olanzapine tablets (5 mg qn) for sleep. Given that pharmacological medication could not control her disturbing symptoms, she decided to receive acupuncture as a complementary therapy.

At Treatment Regimen

On the basis of routine oral administration of gabapentin tablets and duloxetine hydrochloride capsules, she was treated with AT using magnetic beads on both ears. The selected points were heel (AH1), toe (AH2), ankle (AH3), knee (AH4), hip (AH5), lumbosacral vertebrae (AH9), Shenmen (TF4) and tender points (the locations of these auricular points are illustrated in Table 2 and Fig. 2a and b. AT was performed by an experienced acupuncturist. Before AT treatment, the acupuncturist disinfected the ears with 75% alcohol cotton ball and pasted magnetic beads on the above auricular points. The acupuncturist pressed against the points with the index finger and the thumb to the extent that the patient could bear the strength and feel distended pain or heat sensation. Each auricular point was pressed constantly for about 30–60 s, and kept pressing for about 5 min. During the retention period, the patient was informed to press these auricular points by herself 5–6 times a day, with 5 min every time, and change the magnetic beads every other day. The frequency of AT treatment was once in 2 days for a total of 3 weeks.

OUTCOME MEASURES

Primary outcomes included the change in dosage of previous medication, change in pain



Fig. 2 **a** The selected points on the right ear. **b** The selected points on the left ear

intensity of PLP assessed by Visual Analogue Scale (VAS), and change in frequency of attacks of jumping residual limb. Secondly outcomes included the level of emotional state measured by Hamilton Anxiety Scale (HAMA) and Hamilton Depression Scale (HAMD) (Table 3).

DISCUSSION

Both PLP and jumping residual limb are difficult to manage, when they occur at the same time and interact with each other—the difficulty is more than doubled. According to comprehensive literature reviews, most of the available treatments in published literature are effective in dealing with either PLP or jumping residual limb, to date, rare treatment modality has been indicated to be effective in treating both of them simultaneously. Fortunately, we used AT to treat them and received satisfactory effect.

As a major part of acupuncture, AT is defined as “a health care modality whereby the external surface of the ear, or auricle, is stimulated to

alleviate pathological conditions in other parts of the body”. AT involves the relationships among the ear, energy lines (i.e., channels and meridians), and muscle regions comprising the whole body, according to the theory of somatic reflexology. This theory regards that when specific symptoms or diseases arise, it is projected onto the ear at a regular and measurable zone [40]. For instance, the auricular acupoints may manifest not only a decrease in auricular cutaneous electrical resistance but also a decrease in pain threshold [41]. Typical types of AT include auricular acupuncture (AA), electroacupuncture stimulation (EAS), and acupressure (AP), etc. AT has been used to treat a variety of disorders, including both psychological and somatic diseases. Because of its safety and effectiveness, as well as negligible side effects, it is receiving increasing acceptance by physicians and patients. Up till now, a great number of modern studies have indicated that AT is an effective therapy in the management of various types of painful conditions [42–49].

Table 2 The location of the auricular points used in the patient

Auricular point	Location
Gen (AH1) heel	In the anteriosuperior part of the superior antihelix crus at area 1 of the antihelix
Zhi (AH2) toe	In the posteriosuperior part of the superior antihelix crus, inferior to the apex, at area 2 of the antihelix
Huai (AH3) ankle	Inferior to AH1 and AH2, at area 3 of the antihelix
Xi (AH4) knee	On the middle 1/3 of the superior antihelix crus, at area 4 of the antihelix
Kuan (AH5) hip	At the lower 1/3 of the superior antihelix crus, at area 5 of the antihelix
Yaodizhui (AH9) lumbosacral vertebrae	Posterior to AH8 at area 9 of the antihelix
Shenmen (TF4)	At the upper part of the posterior 1/3 of the triangular fossa, at area 3 of the triangular fossa

Nevertheless, the exact mechanisms underlying AT for treating pain remain inconclusive. Based on existing literature, AA can restrain the descending pain pathway via activating the descending pain inhibitory pathway along the dorsal side of the spinal cord, so as to exert analgesic effects [50].

Besides, stimulations of auricular acupoints could promote the release of endorphin and enkephalin. Moreover, the neurophysiological connections between auricular acupoints and the human central nervous system (CNS) have been corroborated by functional magnetic resonance imaging (fMRI) [51]. Therefore, the analgesic effect of AT on this patient might be explained by these potential mechanisms, yet more rigorous experiment is required to confirm it.

In our case, although the PLP and jumping residual limb did not completely disappear, the intensity of pain was significantly reduced, the frequency of jumping residual limb attacks was markedly lessened, the dosage of analgesic was significantly reduced and the overall condition of the patient was improved significantly after a 3-week period treatment. After cessation of AT therapy, the symptoms of PLP were not worsened, instead, the oral medicine further reduced and the jumping residual limb rarely re-emerged. This case report demonstrates that AT may be an effective alternative treatment for PLP accompanied by jumping residual limb.

Table 3 Timeline of drug administration, VAS score, frequency of jumping residual limb attacks, HAMA and HAMD scores

Date	Gabapentin (g)	duloxetine hydrochloride (mg)	Tramadol (mg)	VAS score	frequency of attacks (times)	HAMA score	HAMD score
Before treatment	0.9 bid + 0.6 qn	60 bid	100 qn	9	13–15	30	32
1st week	0.6 tid	60 bid	50 qn	8	9–12	23	27
2nd week	0.6 tid	60 qd + 30 qn	50 qon	6	6–7	19	23
3rd week	0.6 tid	30 bid	–	4	3–4	13	16

CONCLUSIONS

In summary, we presented a rare case report of PLP accompanied by jumping residual limb and AT was applied to improve the patient's symptoms significantly. Given that there is a lack of robust evidence to support the efficacy of many conventional treatments, AT may be an alternative therapy for treating PLP accompanied by jumping residual limb. Nevertheless, further studies with large samples are needed to investigate its efficacy and mechanism.

ACKNOWLEDGEMENTS

We thank the patient for participating in the study.

Funding. Sponsorship for this study and Rapid Service Fee were funded by grants from the Special fund for Key talents training of Zhejiang Province (No:[2017]102); Clinical training project of middle-age famous TCM doctor of Zhejiang Province (No:[2017]22); National Natural Science Foundation of China (grant no. 81503645) and Natural Science Foundation of Zhejiang Province (CN) (grant no. Q20H270078).

Authorship. All named authors meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship for this article, take responsibility for the integrity of the work as a whole, and have given their approval for this version to be published.

Authorship Contributions. Hongfang Tian and Hantong Hu contributed equally to this work.

Disclosures. Hongfang Tian, Hantong Hu, Xingling Li, Jing Liu, Qin Guo, Yang Li, and Dexiong Han have nothing to disclose.

Compliance with Ethics Guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. Institutional review

board approval was not required for a case report.

Data Availability. Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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