SHORT REPORT

Spasms of amputation stumps

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Abstract

Two patients are presented with muscle spasms in an amputation stump. Neither patient experienced neuropathic pain nor phantom sensations, though phantom sensory phenomena, severe pain, and lack of response to treatment is characteristic of reported cases. One patient, a 75 year old man, has had myoclonic activity of the stump for more than two years, and the other, a 79 year old woman, recovered spontaneously after three months and is symptom free after a one year follow up. We emphasise the lack of association with pain and the need to consider spontaneous improvement when therapy is evaluated.

After amputation of a limb the remaining stump occasionally presents choreiform or myoclonic involuntary movements, a syndrome known as "convulsive movements of the stump",¹ "trépidation du moignon"² or "painful jumping amputation stumps".³ Phantom sensory phenomena, severe neuropathic pain, persistence of the involuntary movements after long periods of time, and unsatisfactory response to treatment are typical.¹⁻⁵ The pathophysiology is unknown. We describe two patients who developed spasms of amputation stumps in the absence of neuropathic pain or phantom limb sensations; one of them had an unexpected spontaneous recovery.

Case 1

A 79 year old diabetic and hypertensive woman was admitted in January 1990 for an above the knee amputation of the left leg because of vascular insufficiency. Three days after amputation she experienced involuntary movements of the stump. Initially they were intermittent and precipitated by voluntary activity of the remaining limb muscles, but over the following two days they became continuous and disappeared only during sleep. She denied pain or phantom sensations. She seemed to have no control over the stump, which moved in all directions. She was receiving no medication other than subcutaneous insulin. When first seen, a month after amputation, examination showed constant, clonic and choreatic movements of the stump that increased in severity each time she attempted voluntary movements. The only other neurological abnormalities were an absent right ankle reflex and mild

diminution of vibration sense of right hallux. Routine biochemical and haematological studies were normal, as was a CT head scan. Electroneurographic findings were consistent with a sensory-motor axonal neuropathy. Electromyography showed spontaneous arrhythmic discharges of several motor unit potentials in the left iliopsoas, rectus femoris, biceps femoris and adductor muscles.

No pharmacological treatment was prescribed. During the following two weeks the movements varied throughout the day but clearly diminished in amplitude and duration. Choreiform movements disappeared during the second month, but frequent bursts of jerking movements of the stump occurred mainly related to action. Myoclonic activity completely disappeared before the end of the third month. After a one year follow up she has experienced no further episodes of involuntary movements.

Case 2

This 75 year old hypertensive man had an above the knee amputation of his left leg in October 1988 three weeks after an unsuccessful lumbar sympathectomy. Two months after amputation he complained of involuntary jerks of the stump. He denied pain or phantom sensations. The jerks presented in bursts lasting from minutes to hours. They were worse at night, causing insomnia, and at daytime were aggravated by action. Symptoms persisted without remission. Treatment with diazepam, carbamazepine, fluoxetine, and clonazepam, all failed to alleviate the movements.

When first seen by us two years later, neurological examination showed no abnormalities other than episodic, middle amplitude, rhythmic myoclonic jerks of the stump. They could not be elicited by a variety of tactile and action stimuli but presented randomly during rest or sustained activity of the stump. In both situations voluntary movements of the stump could variably abbreviate the duration of the burst.

Discussion

The timing and clinical characteristics of these two patients most resemble the syndrome of involuntary movements of the stump that rarely occurs after amputation of a limb. The pathophysiology of this condition is unknown, although it may represent a variant within the

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spectrum of movement disorders associated with peripheral nerve injury.⁵⁻¹³ It is suggested that many features of this condition are similar to certain aspects of causalgia and related sympathetic algodystrophies.^{6,7,14} Far from producing deafferentation, damaged nerves can amplify and distort naturally generated signals presenting an exaggerated afferent input to the CNS.¹⁵ Neuropathic pain and phantom sensations are common in reported cases.^{1-5,8,16}

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.³⁻⁹ Our cases argue against this hypothesis, as neither patients experienced neuropathic pain or phantom sensation. An alternative explanation is provided by Schwartzman and Kerrigan.¹⁷ Although they had no patients with spasms after amputation, they have noted that the motor manifestations of reflex sympathetic dystrophy may precede pain or occasionally occur in the absence of pain. They propose that a neuroanatomical network linking the somatic and sympathetic nervous system with participation of substance P directly initiates the abnormal depolarisation of anterior horn cells.¹⁷ This does not explain why involuntary movements occurred in our second patient in spite of previous complete lumbar sympathectomy. At present the pathogenesis of the motor abnormalities and the reason for the variable interval between amputation and onset of involuntary movements that may range from two days⁴ to few months^{1,3} is unknown.

We are not aware of any other reports of spontaneous recovery of spasms of amputation stumps, which should be considered when therapy is evaluated in these patients.

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