

A Case of Spreading 'Jumping Stump' Syndrome

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We present a case of myoclonus in the stump of an 81-year-old who had undergone recent through-hip amputation for chronic osteomyelitis. The patient complained of uncontrollable movements of the other limbs in response to the stump twitching, and was observed to have myoclonic-like jerks in the ipsilateral limb. All movements were attenuated with benzodiazepines. The pathogenesis of the "jumping stump", thought to be a form of spinal myoclonus localised to the amputation site, is discussed.

Introduction

Some of the earliest descriptions of the 'jumping stump' syndrome are attributed to the American Neurologist Silas Weir Mitchell (1829-1914) during his observations of soldiers' injuries during the Civil War,¹ although a similar case report by Hancock was published in 1852.² The condition, described both in the immediate post-operative period and with delayed onset,³ has traditionally been thought to represent a form of spinal reflex myoclonus limited to the amputation site. In the present case the patient reported that the sensation of twitching was only in the leg amputation stump, which occurred on several occasions in the immediate post-operative period. On observation it seemed to be 'spreading' to involve the remaining limbs.

Case history

An 81-year-old female patient underwent a left through-hip amputation of the leg. Prior to this she had a three-year history of chronic infection of a left knee prosthesis by Methicillin-resistant *Staphylococcus aureus* (MRSA), requiring three attempted joint revision procedures. Two years prior to this she had undergone left shoulder replacement after a fall, also complicated by infection with MRSA.

There was no significant past medical history and no family history of neurological disease. Her regular medication included furosemide, lansoprazole, aspirin, iron sulphate and lactulose.

In the immediate post-operative period the patient consistently described uncontrollable 'jumping' of all her limbs but this was initially unwitnessed. Long-term intravenous vancomycin and meropenem therapy was commenced for infection of the amputation stump. Four days post-operatively she was noted to have a 'restless leg' in a setting of clear consciousness. Oral diazepam was administered and the symptoms disappeared within two hours. The patient had remained afebrile throughout all this and all subsequent occasions, and no derangements of biochemical blood tests were noted. After this episode gabapentin and amitriptyline therapy were commenced for coincident 'phantom' left foot pain. Approximately three weeks later the patient reported a rapid onset generalised limb shaking, again in a setting of clear consciousness and for which she again received oral diazepam. On this occasion the symptoms lasted approximately twelve hours.

One month after the original surgery, debridement and closure of the amputation skin flap was carried out. Two days post-operatively the limb movements were again reported. At this stage the gabapentin dose was reduced but the symptoms persisted for 24 hours. One week later, after another rapid onset of limb movements, a Neurology ward consultation was sought.

On examination the patient was lucid and in clear consciousness, although highly distressed by the symptoms. She was writhing on the bed, reporting uncontrollable movements of the limbs and jaw and neck which she felt were being triggered by a constant 'jumping' sensation in the stump of her left leg amputation. On inspection of the stump there were widespread and continuous myoclonic jerks visible. The movements of the three intact limbs initially appeared disorganized and asynchronous; however at one point during the observation, the left arm (ipsilateral to the stump) briefly demonstrated myoclonus (see video).

She had received a dose of oral temazepam just after the onset, and the symptoms settled within six hours. The patient was then prescribed oral clonazepam, and when seen five days later remained asymptomatic with no myoclonus visible in the stump.

Discussion

In this case, the 'jumping stump' appeared to trigger activity in other limbs, with the ipsilateral upper limb briefly demonstrating what appeared to be myoclonus similar to the activity seen at the amputation site. Propagation of segmental spinal myoclonus to multiple different myotomes through propriospinal pathways has been reported.^{4,5} We were unable to carry out electromyographic studies on the patient and so our conclusions are cautious.

Steiner and colleagues reported five cases of stump movement disorder and noted common features of pain and infection prior to amputation,⁶ consistent with our own case. However, the authors specifically noted the lack of involvement of the ipsilateral upper limb. There have been other similar case reports. Pain has not always been a consistently associated feature,⁷ though our patient reported marked phantom pain post-operatively. Voluntary movement and cutaneous stimuli have also been implicated as a trigger to episodes.^{3,8}

Steiner and colleagues suggested that the phenomenon represented an 'alternating reflex segmental myoclonus' with afferent pathways reducing inhibitory spinal interneuronal influence over motor neuron activity. Muscle contraction thus results in a cycle of stimulation. In another case, electromyography was performed in an attempt to demonstrate the spinal origin of the myoclonus.⁹ The authors demonstrated that the myoclonic movements observed were limited to the segments involved with the amputated limb. However, they also observed that, by tapping, it was possible to induce spread to the contralateral

limb. Finally, the authors recognised two clinical periods - one immediately post-amputation during which pain and phantom limb sensations were associated with the phenomenon, as in our patient, and a later phase one month later when it occurred in the absence of these.

Devetag and Bernardi noted no association between the stump myoclonic activity and time-locked recordings of cortical potentials.⁹ Alusi however, in a series of three patients with 'jumping stump' concluded that the phenomenon might have a central component and that "psychogenic factors related to the preamputation illness seemed to play a role".⁸

Our initial assessment was that these movements of the intact limbs probably represented a behavioural response to the intense discomfort brought about by the 'driving' amputation site myoclonus. That said, the observation of apparent myoclonus in the ipsilateral upper limb raises the possibility of retrograde propagation of spinal myoclonus from the amputation site. It is interesting that it was only observed in this limb, which had also been affected by prosthesis infection with MRSA but had not required amputation. Steiner and colleagues raised the common theme of infection in their cases, and such chronic infection might conceivably contribute to local afferent hyperexcitability.

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VIDEO LEGEND

The patient is oriented so that the camera is facing the left leg amputation site from below, with the patient's head out of view to the top right-hand corner. There are continuous myoclonic jerks of the stump visible throughout the recording, associated with writhing movements of the remaining three limbs and jaw with intermittent opisthotonus (out of view). As the camera's field of view expands, the jerking movements of the left arm are briefly visible and appear myoclonic.