

4th International Conference on Healthcare and Health Management

March 08, 2022 | Webinar

Clonazepam as an alternative drug for the treatment of Phantom Limb Pain

Stefano Brunelli

Fondazione Santa Lucia Scientific Institute for Research, Italy

The phantom limb pain syndrome (PLP) is a common complication in amputees. PLP incidence varies from 4% to 83% and about 14% of amputees referred PLP-related disability. The management of PLP often involves a combination of medications, such as NSAIDs, opioids, anticonvulsants, antidepressants, anesthetics, and NMDA receptor antagonists that target multiple mechanisms. Currently there are no standard guidelines in the pharmacologic management of PLP as reported by a Cochrane review of 2011. Other reviews reported a limited evidence for opioids and high dosage of Gabapentin. A recent retrospective study from our group has showed the efficacy of clonazepam for PLP management. The motivation to carry out this study arises from the experience of 25 years of use of this drug with clinical evidence of efficacy and few side effects. The study analyzed retrospectively the clinical charts of all patients admitted after lower limb amputation for inpatient rehabilitation for 3 years. Twenty-three of 82

amputees suffered for PLP and were treated exclusively with clonazepam. After about 1 month of treatment the PLP significantly decreased. The PLP was measured with the Numeric Rating Scale (NRS). The median NRS before the treatment was 7(2), the median NRS after 31±5 days of treatment was 3(3.5). Limited side effects were observed but not so intense to reduce the dosage of clonazepam. Those few incidences of side effects might be due to the low dosage required for PLP reduction. The average dosage of clonazepam used was 1.5±1 mg per day. The highest dosage administered was 3.5 mg per day. Only 4 patients were “not responders” to clonazepam and needed to combine other drugs. The effect of clonazepam may be explained through its agonistic action at the inhibitory GABA-A receptor, decreasing cortical inhibition mediated through excitatory callosal neurons, which act on local GABAergic neurons.

e: s.brunelli@hsantalucia.it