LETTER TO THE EDITOR

A systematic review of the therapeutic interventions for heterotopic ossification after spinal cord injury

Spinal Cord (2011) 49, 482; doi:10.1038/sc.2010.133;
published online 12 October 2010

Teasell *et al.*¹ assert that various factors including pressure sores and trauma have been associated with this condition but that a causative relationship between them has yet to be determined.

I wish to adduce the evidence that trauma in the form of passive movement is implicated in the development of heterotopic ossification (HO).

In 1969, I postulated in a study of 124 traumatic spinal injury admissions at the Liverpool Regional Paraplegic Centre between 1963 and 1968 that there was a striking relationship of heterotopic bone formation to passive movement and trauma and I wrote:

This series seems to offer some support for the relationship of heterotopic ossification to trauma, either by passive movement or by the use of Stryker frames and turning beds since all three patients who had unilateral movements in the non-swollen leg developed ossification whereas the swollen leg which was not manipulated remained free of ossification.²

When I re-examined the problem in 1993 in relating it directly to the passive movements in the lower limbs, I found in a study of 91 consecutive patients at the National Spinal Injuries Centre (NSIC) between 1983 and 1989 that of the 35 patients who started passive movements in the first 6 days following spinal cord injury, two had radiological evidence of HO but this was not associated with clinical signs or symptoms. Of the 56 patients who started passive movement 7 or more days after injury to the spinal cord, 10 (18%) showed clinical evidence and 12 (21%) showed radiological evidence of HO.³

The missing link in the argument as to how passive movement causes heterotopic bone to occur has been provided by Rossier *et al.*⁴ in a magisterial article on paraosteoaarthopathy (POA) with a combined pathological and biochemical study where they showed, by biopsy, the presence of occasional microfractures that may modify secondarily the morphological aspect of the POA.

Findings by Rossier *et al.* have been substantiated at the NSIC by biopsies showing microtrauma, and in Belgium by sonography, also showing traumatic tears to the muscles in

such cases leading to HO. The cause of these microfractures has been substantiated by subsequent investigators in Spain and at Oswestry.

These microfractures may be related to the patients' spasticity, or perhaps, to passive mobilization.

These reports of trauma in the form of passive movement being associated with pathological evidence of damage to the muscle and precipitating heterotopic bone formation arise from different countries, Switzerland, Belgium, Spain and the UK, and at different times and by different investigators cannot be dismissed. These results are reproducible and consistent and based on careful clinical observation of a series of cases correlated with X-ray and pathological substantiation of the findings.

Prevention is better than cure. If patients are admitted forthwith to a spinal unit, commence anti-coagulants and careful gentle passive movements, they are unlikely to develop ossification. If they are admitted late, when the joints have already developed contractures, passive movement can cause trauma to the tissues, haematoma formation and calcification.

Conflict of interest

The author declares no conflict of interest.

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