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Title	A case of long thoracic nerve palsy
Type	Article
URL	https://clock.uclan.ac.uk/id/eprint/20413/
DOI	
Date	2010
Citation	Chance-Larsen, Kenneth and Littlewood, Chris (2010) A case of long thoracic nerve palsy. International Journal of Physiotherapy and Rehabilitation, 1 (1). pp. 41-43. ISSN 2349-5987
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CASE REPORT

A case of long thoracic nerve palsy

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Abstract

A 31 year old male developed unilateral neck pain associated with increased kyphotic posture 24 hours after having general anaesthesia for ankle surgery. The pain and postural adaptation resolved, but a painless shoulder dysfunction developed. Following assessment a provisional diagnosis of long thoracic nerve palsy was made, and a 'wait and see' approach to management was taken. The dysfunction gradually resolved over a 12 month period without further investigation or intervention. The case study is discussed in light of possible neurophysiological mechanisms involved and of published literature on the management of long thoracic nerve palsy.

Keywords: long thoracic nerve palsy; post-operative; case study.

Case History

A 31 year old male lecturer presented with a sudden onset of right sided neck pain and kyphotic deformity, 24 hours after a lateral ligament repair of the ankle (Modified Broström repair). The surgical procedure was carried out under general anaesthesia and required non-weight bearing mobilisation with elbow-crutches (EC's) in the early post-operative period. The pain and deformity relating to the neck resolved rapidly over a 24 hour period but a residual painless shoulder dysfunction persisted.

Immediately following resolution of the pain, the individual was unable to elevate his arm greater than 90 degrees due to weakness. These movements were associated with excessive lateral translation and winging of the medial border of the scapula. At this time it was evident that the right serratus anterior (SA) muscle was flaccid. A full and painless passive range of movement (ROM) was maintained. Resisted testing of abduction, flexion and lateral rotation in the neutral glenohumeral joint position did not provoke pain or identify weakness but was associated with winging of the scapula. Neurological testing was negative.

The individual was medically well and had an unremarkable past medical history. Analgesics (paracetamol) were discontinued through choice 48 hours post-surgery. At this stage the case was limited functionally when elevating the arm, e.g. dressing, or reaching forwards and was uncomfortable when leaning against hard surfaces due to the scapular protrusion.

Based upon this, a provisional diagnosis of long thoracic nerve (LTN) palsy was made. A definite cause of the palsy could not be identified. A decision was made to 'wait and see' based upon the acute nature of the presentation (only a few days since onset), recovery to date (reduced pain and slight increase in active ROM) and suggestions from relevant literature (Elders et al 2001).

Despite no further intervention or investigation, the dysfunction gradually resolved over a 12 month period. Within the limits of the pathology, the subject functioned as normally as possible during this time and began to notice minor improvements in active ROM and reduced discomfort when resting on hard surfaces. Over a number of months, a full active ROM was gradually regained. Initially this gain in active ROM was associated with persistent scapula winging and lateral translation. By 12 months post-onset the individual was fully functional with no evidence of significant winging of the scapula with movement.

Discussion

The LTN functions as a motor nerve innervating the SA (Marin 1998). The SA, in conjunction with trapezius, serves to rotate the scapula upwardly during upper limb elevation whilst stabilising the medial and inferior border of the scapula (Hamada et al 2008). In the absence of an effectively functioning SA upper limb function is markedly restricted (Hamada et al 2008).

Shoulder complaints are common (Luime et al 2004), but dysfunction due to LTN palsy is rare (Chaney et al 2000) and may be frequently missed (Belville and Seupaul 2005). Prognosis is variable and may not be favourable in the absence of signs of early recovery (Kauppila and Vastamäki 1996, White and Witten 1993). Current estimations for functional recovery range from 6 months to 2 years with some patients requiring reconstructive surgery (Wang et al 2008).

Reports of SA weakness due to LTN palsy have been presented detailing both traumatic and non traumatic onsets (Marin 1998, White and Witten 1993), although the causative factor is often elusive (Chaney et al 2000). Direct trauma, heavy lifting or traction injury (Elders et al 2001), certain positions, e.g. prolonged supine, or just moving a patient from the operating table (Chaney et al 2000, White & Witten 1993) have been reported as direct causes. Reports of insidious onsets include exposure to cold, child birth or post anaesthesia (Belville and Seupaul 2005, Marin 1998) but the mechanisms involved are not understood. It is impossible to identify the causative factor in this case but it is possible that heavy lifting/ brachial plexus traction through use of EC's was a factor. Also, onset as a result of post anaesthetic complications cannot be ruled out, neither can a prolonged position during surgery or the transfer from the operating table.

Due to the limited numbers of these clinical presentations it is difficult to move towards management consensus or evidence based guidelines. Various suggestions have been put forth without adequate justification. Some advocate the use of immobilisation early in the process to facilitate recovery (White and Witten 1993) whilst others advocate early mobilisation, including physical therapy (Belville and Seupaul 2005). The use of functional braces to prevent the scapula winging whilst enabling functional activity has been investigated and recommended (Marin 1998). However, it should be highlighted that the majority of these ideas are anecdotal or stem from case series designs, which, alone, cannot provide sufficient evidence of efficacy. It does seem sensible to suggest that a programme of exercise designed to maintain strength and ROM

in the early phases before gradually increasing the functional demand upon the shoulder complex would be safe and appropriate.

Implications

This case report has detailed the presentation, diagnosis and protracted recovery of a person presenting with SA weakness secondary to LTN palsy. In this case, after showing early signs of recovery, the shoulder dysfunction gradually resolved over a period of 12 months. This report is a reminder of this presentation and offers perceptions into diagnosis, prognosis and possible approaches to conservative treatment.

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