The immediate effect of neuromobilisation in stage 2 idiopathic adhesive capsulitis: a preliminary study

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Abstract
Introduction: The restriction of neural mobility and neuromobilisation on adhesive capsulitis has not been paid attention in previous studies despite joint contracture via an inflammatory process in the natural history of adhesive capsulitis. We hypothesized that neurodynmics could be affected shoulder range of motion in stage 2 adhesive capsulitis due to nerve fibrosis or stiffness of non-neural tissue surrounding a nerve. This study aimed to clarify the neural restriction and the effect of neuromobilisation in patients with idiopathic adhesive capsulitis.
Subject & Method: 16 patients (four males, twelve females, 59.4 ± 11.1 years old, duration period 7.9 ± 4.8 months) were recruited in this study. Their elbow extension range of movement was measured bilaterally at their end range of the Upper Limb Neurodynamic Test1. Subjects were then randomly allocated to a neuromobilisation group, or a control group. The neuromobilisation group received neuromobilisation, whereas the control group received quasi-neuromobilisation after general physiotherapy. The elbow extension range of movement in the Upper Limb Neurodynamic Test 1 and shoulder range of motion were measured before and after general physiotherapy and neuromobilisation interventions.
Results: The elbow extension range of movement was statistically improved from -26.9 ± 29.1° (pre-neuromobilisation) to -15.0 ± 18.7° (post-neuromobilisation) in the symptomatic side in neuromobilisation group but not in control group. Shoulder extension (45 ± 10.7° to 50.6 ± 12.7°), external rotation at arm by side of the body (21.9 ± 18.1° to 27.5 ± 19.6°) and at 90° flexion (65.6 ± 26.9° to 75.6 ± 22.1°), internal rotation at 90° abduction (38.8 ± 13.8° to 45.0 ± 12.0°) were significantly improved after neuromobilisation in neuromobilisation group.
Conclusion: The results of this study might indicate the neural restriction in neurodynamic test, and the improvement of shoulder range of motion with neuralmobilisation for adhesive capsulitis. that neurodynamic assessment and neuromobilisation should be considered in the management of adhesive capsulitis.

Introduction

Adhesive Capsulitis (AC) provokes shoulder pain and restrictions in both active and passive range of shoulder motion. The restricted range of shoulder motion is improved approximately 60 to 80 % of shoulder elevation comparing with asymptomatic shoulder at the final follow-up[1]. Physiotherapy is a primary choice of conservative treatment for AC[2-4]. Joint mobilisation, capsular stretching, and muscle stretching was applied to improve shoulder range of motion (SROM) for AC due to peri- and intraarticular pathology[5-10]. Such physiotherapy intervention was based on pathophysiological evidence in AC, focused on capsuloligamentous inflammation and fibrosis or contracture with associated SROM restrictions[11-15]. An MR imaging studies reported a thickness of coracohumeral ligament in AC[16)17)]. Increment of cytokines and growth factor induced in rotator interval of AC was reported in a histological study[18)].

SROM restriction due to nerve involvement in AC is less knowledge despite joint contracture via an inflammatory process in the natural history of AC[1)]. There was observed abnormal nerve growth factor in the glenohumeral capsule in AC[19)]. Another histological study found fibroblasts in nerve tissue in chronic AC[20)]. This pathophysiological process may provoke a neuro-contracture and mechanosensitivity, related to pain and movement restriction in AC as mechanical elongation and angulation of peripheral nerve during joint motion sensitizes itself after inflammation[21)]. The neural restriction and its improvement by neural mobilisation for carpal tunnel syndrome, and breast cancer were demonstrated[22)23)]. The clinical evidence of neural mobility and neuromobilisation (NM) on AC has not been clarified in previous studies. We hypothesized that neurodynmics could be affected SROM in stage 2 AC due to nerve fibrosis or stiffness of non-neural tissue surrounding a nerve. The purpose of this pilot study was to clarify the restriction of neurodynamics and the effect of a single session of NM in patients with idiopathic AC in stage 2.

Subject

33 patients with AC (10 men, 23 women, mean age 59.4 ± 11.1 years old, duration of symptoms 7.9 ± 4.8 months) were participated. The inclusion criteria of this study were, (i) no traumatic history of bony lesion, nor AC, (ii) no unstable neurological signs or disease, (iii) unilateral AC, (iv) more than fifteen weeks of duration of symptoms, i.e. likely to be beyond the inflammatory phase[16)17)], and (v) no history of treatment. The exclusion criteria were, (i) more than 50 % loss or less than 30° external rotation at arm by side of the body (0abd)[24)], (ii) more than 30° SROM restriction compare to asymptomatic side in less than one direction[16)17)], or (iii) restricted passive range of elbow extension in physical examination. This study was approved by the Human ethics research committee of the Nobuhara hospital (Approval no.2401).

Method

All subjects' elbow extension range of movement (EEROM) was measured on their

symptomatic and asymptomatic sides at their end range of the Upper Limb Neurodynamic Test 1 (ULNT1) (i.e. structural end range of elbow extension in ULNT1). The ULNT1 maneuver was shown in Figure 2 in appendix. Structural differentiation was undergone with cervical lateral flexion at the end of ULNT1 position by asking the change of participants' symptom. Subjects were then randomly allocated to a NM group, or a control group. The NM group received NM in ULNT1 position. They were randomly undergone 30 repetitions of through-range elbow extension into resistance (i.e. tensioner technique), or 30 repetitions of elbow extension with cervical ipsilateral flexion and wrist dorsal flexion, and elbow flexion with cervical contralateral flexion and wrist volar flexion (i.e. slider technique) after conventional physiotherapy (hot pack, soft tissue mobilisation, and passive end range glenohumeral accessory mobilisation). The control group was undergone quasi-NM (passive range of motion exercises for each component of ULNT1, 30 repetitions of through-range passive range of motion into resistance) after conventional physiotherapy. The passive range of shoulder motion exercises was undergone while elbow, forearm, wrist and fingers were held at start position of ULNT1 so as to minimize the effect of neural components during mobilizing glenohumeral joint. EEROM in ULNT1 and SROM was measured before and after conventional physiotherapy, and NM/quasi-NM respectively. SROM was measured according to the method of Macedo & Magee (2009)[25]. The profile of patients, the effects of the interventions (conventional physiotherapy versus NM) in each group and the effects of interventions between both groups were compared using Wilcoxon signed rank test.

Results

16 patients with AC recruited in this study (Table 1). Six patients excluded due to short duration period and eleven patients were excluded according to SROM criteria in this study. There was no marked difference of subjects' profile, SROM in symptomatic side, and SROM in asymptomatic side between NM and control groups ($p > 0.05$, table 1). A significant restricted SROM in symptomatic side was confirmed in both groups ($p < 0.05$).

A significant difference of EEROM between symptomatic and asymptomatic sides in both

Table 1. The profile of patients. Age and duration periods: the mean ± standard deviation.

	NM group (n = 8)			Control group
	Tensioner	Slider	All	(n = 8)
Age (yo)	56.3 ± 9.6	62.8 ± 9.5	59.5 ± 9.5	56.1 ± 9.7
Male (n)	2	1	3	1
Female (n)	2	3	5	7
Duration periods (mo)	10.0 ± 4.0	9.3 ± 9.8	9.6 ± 7.0	8.9 ± 3.4
Right-handed (n)	4	4	8	8

**: $p<0.01$

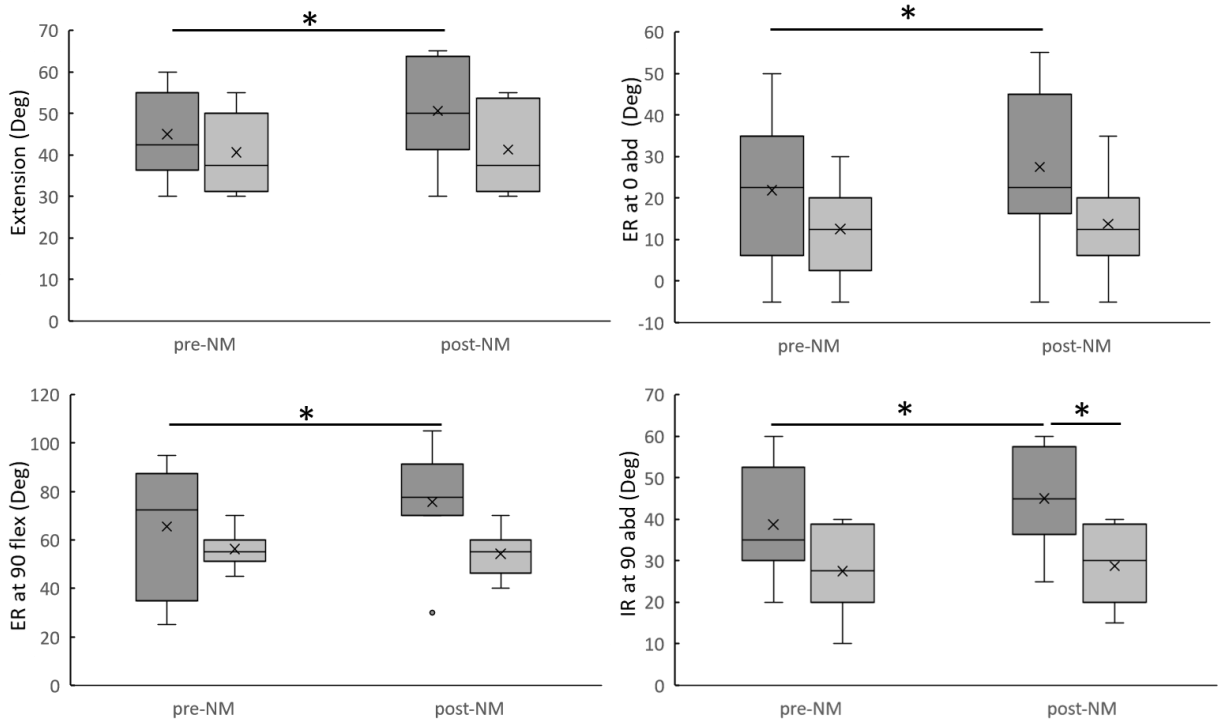

Figure 1. SROM between pre- and post-NM (Deg). ER: external rotation, 0abd: at arm by side of the body, 90 flex: at 90° flexion, 90 abd: at 90° abduction, dark grey boxes: NM group, light dark grey boxes: control group, *: $p < 0.05$. a black dot: an outlier. Cross markers indicate mean value in box-whisker graphs.

groups, with -26.9 ± 29.1° for symptomatic side, -0.6 ± 1.8° for asymptomatic side in NM group, -28.8 ± 29.5 ° for symptomatic side, 0 ± 0.0° for asymptomatic side in control group ($p = 0.042$, 0.043), and no statistical difference of EEROM between both groups was confirmed ($p > 0.05$) in pre-conventional physiotherapy. All participants complained that their pain increased with contralateral flexion and decreased with ipsilateral flexion of cervical spine at end of ULNT1 position. A marked improvement in EEROM was found between pre- and post-NM in the symptomatic side (-26.9 ± 29° to -15.0 ± 18.7°, $p = 0.043$) in NM group but not in control group (-28.8 ± 29.5 ° to -27.5 ± 28.7 °, $p = 0.317$).

Shoulder extension, external rotation at 0abd and at 90° flexion, internal rotation at 90° abduction were significantly improved in post-NM comparing with pre-NM in NM group ($p = 0.024$, 0.034, 0.014, 0.039, Figure 1). A statistical improvement of internal rotation at 90° abduction, and at 90° flexion in NM group were observed comparing with those in control group ($p = 0.029$, 0.017).

Discussion

EEROM in ULNT1 position was restricted more than 10 degrees in patients with AC whilst asymmetrical EEROM of ULNT1 in healthy subjects was within eight degrees[26]. Moreover, the symptom of subjects in ULNT1 was changed with structural differentiation,

implying a positive finding of neurodynamic test[27]. These findings in this study could indicate restricted neural mobility in stage 2 AC.

SROM and EEROM in NM group further improved with NM intervention. The results of this study demonstrated that a single NM treatment can improve neural mobility and SROM in patients with AC. On the other hand, in control group, passive range of motion of ULNT1 components did not improve SROM after conventional physiotherapy although a peripheral nerve is shortened and elongated when a joint mobilized[28]. This result might indicate less effect of passive range of motion exercise for neurodynamics in AC. Our findings could indicate that neurodynamic assessment and NM should be considered in the management of AC.

To our knowledge, this study was the first challenge of NM intervention for AC. The improvement of EEROM in ULNT1 and SROM was observed after NM intervention. Several factors might lead to the improvement of them. Neurodynamic tests could detect and NM could improve surrounding tissue of peripheral nerve or the contracture of peripheral nerve per se in stage 2 AC. Induce of nerve growth factor receptor and nerve fibres were reported in AC capsule[19]. Furthermore, abnormal neovessels were observed in the rotator interval in patients with AC[29]. Nerve ingrowth with neovessels was a cause of pain[30]. NM intervention reorganized nerve collagen fibre and vessels[31]. NM intervention might reduce mechanical irritation to the capsule in AC patients.

The limitation of this study was that there was no biomechanical study to investigate the restricted neurodynamics in AC. Our hypothesis was based on previous pathophysiological studies and clinical experience that some patients with AC complaint pain along peripheral nerve at the end of motion. There might be some mechanisms that SROM improved after NM. Neural mobility could be restricted due to stiffness of neural interface or stiffness of peripheral nerve per se. This study provided the clinical evidence of that neural mobility was restricted and the NM improved ROM in patients with stage 2 AC. Future study is expected to clarify the restricted neural mobility in AC.

Conclusion

A positive finding of ULNT1 was confirmed in the symptomatic side of stage 2 AC. This finding might indicate neural restriction in AC. EEROM in ULNT1 and SROM were significantly improved after NM. The results of this study might indicate that neurodynamic assessment and NM should be considered in the management of AC.

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Appendix

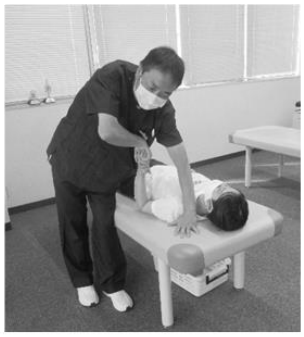

Figure 2-1: Start position

Figure 2-5: Shoulder external rotation

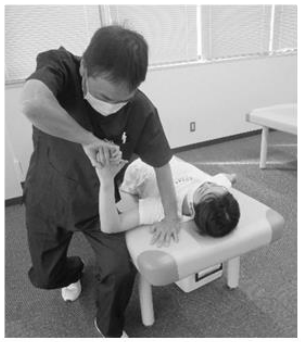

Figure 2-2: Shoulder abduction

Figure 2-6: Elbow extension

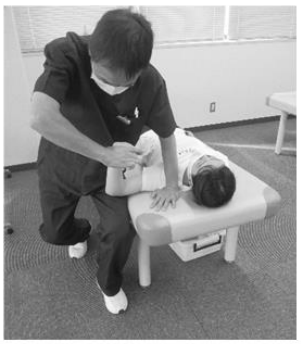

Figure 2-3: Wrist extension

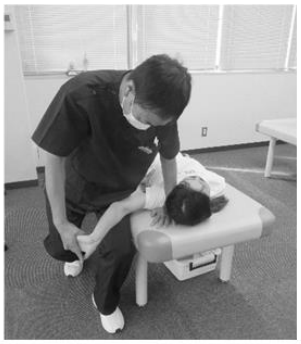

Figure 2-7: Cervvical ipsilateral flexion

Figure 2-4: Forearm spination

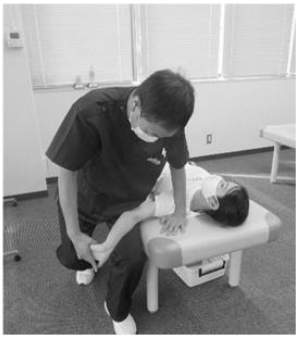

Figure 2-8: Cervvical contralateral flexion

Figure 2: The ULNT1 maneuver