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Central and Peripheral Sensitization in Patients with Chronic Shoulder Pain

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Abstract: ***Chronic shoulder pain present complex pathomechanical situations, frequent difficulties in identification of the pain causing factors leading to lack of effectiveness in treatment. Thus the purpose of this study is to identify the role of neurophysiological changes altering pain sensitivity leading to hyperalgesia and allodynia in such patients. Pressure pain threshold values of the affected side of patients were compared to the unaffected side and to controls and were analysed statistically, significant difference in the readings were noticed pointing towards central nervous system playing a role and leading to central and peripheral sensitization.***

Keywords: Shoulder pain, central sensitization, peripheral sensitization

1. Introduction

Shoulder pain is a common musculoskeletal condition affecting around 15-30% of adults at anytime.[1] Chronic shoulder pain is a pain that lingers for more than 3 months continuously or intermittently associated with restricted range of movement. Chronic pain can range from mild, to severe, to disabling and can last from a few weeks to few months to many years.[2] There is now evidence that alterations in the central and peripheral nervous systems may play a role in chronic pain, [3, 4, 5] and may explain why some patients fail to improve in spite of treatment and lack of evidence for persistent pathology.

Central hypersensitivity is an augmentation of the nociceptive pathways of the central nervous system that is characterized by local and generalized lowered pain thresholds and an exaggerated pain response to painful and non-painful stimulation. Central hypersensitivity is a normal response of the central nervous system to injury that encourages protection of injured tissue to allow healing.[6] After the injured tissue has healed, the hypersensitivity to pain typically resolves; however, the central hypersensitivity may persist in some individuals, resulting in a chronic pain syndrome.

There is evidence of secondary hyperalgesia in those who experience chronic shoulder pain, providing indirect evidence of central hypersensitivity.[7] Peripheral and central abnormalities of nociception have been described in musculoskeletal pain patients.[8,9,10,11] Important nociceptive systems in the skin and deep tissues of these patients seem to undergo profound changes, resulting in sensitization of the hyperpolarization-activated cyclic nucleotide-gated ion channels,[12] transient receptor potential channels, acid-sensing ion channel receptors, and purinoreceptors.[13] Tissue mediators of inflammation and nerve growth factors can excite these receptors and cause extensive changes in pain sensitivity.[14]

The most distressing feature of these pathological processes is that they persist long after healing of the damaged peripheral tissue. Peripheral neural mechanisms, such as nociceptor sensitization and neurogenic responses are likely to contribute to pathological pain at early stages following injury when tissue damage and inflammation are prevalent. However, the persistence of pathological pain after the healing of damaged tissue suggests that changes in CNS function may also play a significant role.

Terrence J. Coderre et al in his study on contribution of central neuroplasticity to pathological pain concluded that clinical and experimental evidence suggests that noxious stimuli may sensitize central neural structures involved in pain perception. An increased understanding of the central changes induced by peripheral injury or noxious stimulation should lead to new and improved clinical treatment for the relief and prevention of pathological pain.

Tracy Maria Paul et al in her study on Central Hypersensitivity in Patients with Subacromial Impingement Syndrome that subjects with SIS may be experiencing central hypersensitivity. There is still a paucity of research in Indian population on central and peripheral sensitization in chronic shoulder pain patients. Thus with this understanding our research investigates the presence of central and peripheral sensitization in chronic shoulder pain.

2. Material and Methodology

Material: Pressure Algometer (Wagner) Proforma

Methodology:
Research Design: Cross sectional study
Sample size: 80 (25 with Adhesive capsulitis, 25 with Impingement syndrome and 30 controls)
Duration of study: 4 months
Inclusion Criteria: Adhesive capsulitis and Subacromial Impingement Syndrome patients with pain history of more than 3 months.
Exclusion Criteria: Any other neuromuscular problem in shoulder

Institutional ethics committee approval was taken. Subjects consent was taken prior to the study. Subjects were evaluated for myofascial trigger points in various muscles like Upper trapezius, Middle trapezius, Lower trapezius, levator scapulae, Subscapularis, Supraspinatus, Serratus anterior, Pectoralis Minor, Infraspinatus, Deltoid and Teres minor, Tibialis anterior. Pressure pain thresholds (PPT) of

above muscles were taken on the affected and unaffected side and we used tibialis anterior as the distal site of which PPT was taken on the ipsilateral side of pain.The PPT values of proximal muscles on the affected side and unaffected side were then compared to each other and the values of unaffected side were compared with controls. For tibialis anterior the values on the ipsilateral side were compared to controls.The data thus collected was statistically analysed for the level of significance.

Table 1

	MEAN	SD(±)
AGE	43.1	14.12
BMI	24.22	2.44
NRS(REST)	2.7	1.20
NRS(ACTIVITY)	7.18	1
DURATION	5.5	1.82

Table 2

Muscle	*Mean*	*SD(±)*	*Lower 95% CI*	*Upper 95% CI*	*P value*
Tibialis Anterior					
Ipsilateral	5.13	0.10	4.86	5.40	0.0385 S
Controls	5.62	0.74	5.34	5.90	

Table 3

Muscle	*Mean*	*SD(±)*	*Lower 95% CI*	*Upper 95% CI*	*P value*
Upper Trapezius					
Affected	1.97	0.47	1.84	2.10	<0.0001 ES
Unaffected	2.91	0.52	2.76	3.06	
Middle Trapezius					
Affected	2.84	0.60	2.67	3.01	<0.0001 ES
Unaffected	2.23	0.51	3.10	3.38	
Lower Trapezius					
Affected	3.24	0.52	3.09	3.39	0.0003 ES
Unaffected	2.40	0.56	3.24	3.56	
Levator scapulae					
Affected	2.22	0.58	2.06	2.39	<0.0001 ES
Unaffected	3.10	0.54	2.94	3.25	
Subscapularis					
Affected	2.85	0.64	2.67	3.30	<0.0001 ES
Unaffected	3.16	0.49	3.02	3.30	
Supraspinatus					
Affected	2.03	0.52	1.90	2.17	<0.0001 ES
Unaffected	3.10	0.45	2.97	2.23	
Serratus anterior					
Affected	2.76	0.55	2.60	2.92	<0.0001 ES
Unaffected	2.99	0.43	2.87	3.11	
Pectoralis minor					
Affected	2.26	0.55	2.10	2.41	<0.0001 ES
Unaffected	3.02	0.55	2.87	3.18	
Infraspinatus					
Affected	2.60	0.56	2.45	2.77	<0.0001 ES
Unaffected	3.22	0.52	3.07	3.71	
Deltoid					
Affected	2.58	0.58	2.41	2.71	<0.0001 ES
Unaffected	3.22	0.57	3.06	3.38	
Teres Minor					
Affected	2.69	0.63	2.51	2.87	<0.0001 ES
Unaffected	3.13	0.45	3	3.26	

Table 4:

Upper Trapezius					
Unaffected	1.97	3.19	1.83	2.09	<0.0001 ES
Controls	3.18	0.45	3.01	3.35	
Middle Trapezius					
Unaffected	2.84	0.59	2.67	3.01	<0.0001 ES
Controls	3.54	0.29	3.43	3.64	
Lower Trapezius					
Unaffected	3.23	0.52	3.08	3.38	0.0008 ES
Controls	3.71	0.50	3.53	3.90	
Levator scapulae					
Unaffected	3.09	0.53	2.94	2.25	<0.0001 ES
Controls	3.34	0.47	3.16	3.51	
Subscapularis					
Unaffected	3.15	0.49	3.02	3.30	0.0061 VS
Controls	3.46	0.40	3.31	3.61	
Supraspinatus					
Unaffected	2.97	0.57	2.80	3.13	0.0098 VS
Controls	3.27	0.37	3.13	3.42	
Serratus anterior					
Unaffected	2.30	0.43	2.87	3.11	<0.0001 ES
Controls	3.46	0.45	3.29	3.62	
Pectoralis minor					
Unaffected	3.02	0.55	2.87	3.18	0.0075 VS
Controls	3.33	0.33	3.20	3.45	
Infraspinatus					
Unaffected	3.22	0.52	3.07	3.37	0.0230 S
Controls	3.56	0.44	3.39	3.72	
Deltoid					
Unaffected	2.58	0.57	2.42	2.74	<0.0001 ES
Controls	3.39	0.36	3.26	3.56	
Teres Minor					
Unaffected	3.13	0.45	3	3.26	<0.0001 ES
Controls	3.55	0.38	3.41	3.70	

3. Discussion

There is currently a large number of studies showing that there are changes, especially in intramuscular microcirculation and in muscle energy metabolism, that could be the excitatory drive for the changes found in nociceptive system in the CNS and for the multifocal pain in the muscles.[15, 16] In, RA, osteoarthritis and chronic idiopathic back pain, decreased PPT has been found in areas outside the area of pain.[17, 18, 19]

The present data demonstrates a significant change in the Pressure Pain Threshold (PPT) values on the affected side than the unaffected side of the patients when found out on trigger points of a number of muscles around the shoulder viz. upper trapezius, middle trapezius, lower trapezius, levator scapulae, subscapularis, supraspinatus, serratus anterior, pectoralis minor, infraspinatus, deltoid and teres minor.(Table 3)

The plausible mechanism of the PPT values being lower on the affected side than the unaffected side after >3 months of initiation of pain explains changes in the neurophysiological mechanisms affecting pain sensitivity at and around the site of pain which is called peripheral sensitization.

One of the main characteristics of central sensitization in patients with musculoskeletal pain is a generalized rather than a localized decrease in their pressure pain threshold. Here, 'generalized' implies more than a segmental spreading of the symptom area, in that it means that the increased sensitivity is localized at sites segmentally unrelated to the primary source of nociception.[20] Hence, PPT values of the unaffected side of the patients shoulder were compared to

controls and to include a distal site PPT value of Tibialis anterior muscle of patients was also compared to controls and significant changes were observed. (Table 2, 4)

Lower pressure pain thresholds at symptomatic areas most often represent primary hyperalgesia due to sensitized polymodal nociceptors within injured musculoskeletal structures. By measuring pressure pain thresholds outside the area of primary nociception, widespread hyperalgesia or secondary hyperalgesia can be detected. Findings of numerous areas of hyperalgesia at sites outside and remote from the symptomatic site, together with a non-segmental general decrease in pressure pain threshold, may imply a generalized hyperexcitability of central nociceptive pathways .[20] Based on this reasoning, research has shown evidence in support of generalized hypersensitivity to mechanical pressure in patients with chronic whiplash associated disorders,[20,21] as well as in a subgroup of the chronic low back pain population[22] and similar findings have been found in chronic shoulder pain patients in our study.

This study emphasizes the presence of peripheral and central sensitization in patients with chronic shoulder pain. It is a need to identify and assess these patients so appropriate therapeutic strategy can be developed for the same.

4. Conclusion

There was presence of central and peripheral sensitization in patients with chronic shoulder pain and further investigations should be conducted in order to develop appropriate treatment strategies.

5. Future Scope

An effective evaluative and treatment strategy needs to be considered in patients with chronic shoulder pain having central and peripheral sensitization. Further studies of the relationship of PPTs and chronic pain syndromes should be conducted including longitudinal studies of central hypersensitivity in subjects with chronic shoulder pain undergoing treatment which would improve our understanding of this association.

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