On the segmental and tagmatic phenomena inferred from referred pain and autonomic concomitants

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SUMMARY
Visceral injury induces referred pain, autonomic concomitants, skeletal muscle spasm, and hyperalgesia of segmental pattern on the specific area of somatic tissue. Therefore, since visceral referred pain does not mean only a feeling which is considered as misperception of brain but accompanies accessory phenomena, referred phenomenon can be more reasonable expression. Visceral referred phenomenon has segmental pattern and is shown on the segmental constituents related to viscus and further spreads to multi-segments through central sensitization if visceral injury become severe and chronic. Segmental pattern observed in our body is not a feature developing only in human. Segmentation of animals is a general concept in body organization from Annelida to Vertebrata. The evolutionary advent of segmentation made possible development of much greater complexity in structure and function. Segmentation increased efficiency of movements, and further made fine neural control of movements possible. It is definite that segmentation of body bestows obvious advantages to an animal. If abnormality in a segment develops, to lessen energy expenditure of an organism consisting of multi-segments, the organism sacrifices the abnormal segment for the whole segments via defunctionalization. Defunctionalization of abnormal segment is just segmental phenomenon. Functional unit of grouped segments called as tagma in Arthropoda or its equivalent in Vertebrata could also show this phenomenon if abnormality develops in tagma or its equivalent. Visceral referred pain, referred pain originating from musculoskeletal structure, complex regional pain syndrome I and II are representatives of segmental or tagmatic phenomena.

Referred phenomenon

Referred pain is pain perceived elsewhere than at the site whence it originates [1]. When the afferent visceral pain stimuli are sufficiently intense, discharges at synapses within the spinal cord cause reflex phenomena, usually transmitted by peripheral nerves of the same spinal segments. These include (1) automatic reflexes which cause changes in sweating and skin blood flow along the distribution of the somatic nerve; (2) reflex contraction of skeletal muscles innervated by the same or adjacent neural segments; and (3) increased sensitivity of pain perception received from cutaneous nerve stimulation ("hyperalgesia" – the threshold to recognizing cutaneous stimuli as pain is lowered in the spinal cord or peripheral nerve) [2]. Therefore, referred phenomenon is more reasonable expression because referred pain usually accompanies accessory phenomena described above. If the original problem of viscus is not solved, referred phenomenon could develop on the segmental constituents related to the viscus and spread to up and downward segments of the spinal cord through central sensitization [3]. Hyperalgesia and atrophy of skeletal muscles, and dystrophic skin changes develop in the same segments with causative viscus [2]. These findings are considered as defunctionalization of the segments related to the viscus. Then, does the segmental pattern of referred phenomenon to induce such defunctionalization occur only to human?

Metamerism (segmentation) and segmental phenomenon

Metamerism is a serial repetition of similar body segments along the longitudinal axis of the body [4]. Each segment is called a metamere, or somite. In forms such as earthworms and other annelids, in which metamerism is most clearly represented, the segmental arrangement includes both external and internal structures of several systems. There is repetition of muscles, blood vessels, nerves, and setae of locomotion. Some other organs, such as those of sex, may be repeated in only a few somites. Evolutionary changes have obscured much of the segmentation in many animals, including humans. True metamerism is found in only three phyla: Annelida, Arthropoda, and Chordata, although superficial segmentation of ectoderm and body wall may be found among many diverse groups of animals [5]. Segmentation brings more
varied specialization because segments, especially in arthropods, have become modified for different functions. The evolutionary advent of metamerism was highly significant because it made possible development of much greater complexity in structure and function. Metamerism increased efficiency of movements, and made fine neural control of movements possible. Moreover, repetition of body parts the organisms a built-in redundancy, as in some human-made systems. Redundancy is provided as safety factor: if one segment should fail, other could still function. Thus an injury to one part would not necessarily be fatal [6]. The injured segment undergoes defunctionalization and its energy is redistributed to other segments, or it is got rid of like proglottides of Platyhelminthes. Likewise, defunctionalization could be applicable to the segments in vertebrates including humans. Referred pain and accessory phenomena occurring in the same segments with injured viscerca are segmental phenomena analogous to defunctionalization in Annelida or Platyhelminthes. In humans, segments are mainly discernible in the embryo stage but segmental features can be sought in vertebrae, ribs and deep truncal muscles of adults. If pathologic processes in viscerca occur, the segmental nature of referred phenomenon is evoked in the somites related to viscerca. All the somatic derivatives manifest segmental phenomena which include visceral referred pain, skeletal muscle spasm and autonomic concomitants on skin [2]. At first, these phenomena can protect a pathologic viscus from the external impact by hyperalgesic skin and muscle spasm but in long-lasting visceral problem, permanent atrophic changes in skin and skeletal muscle develop [2]. The meaning of these features is defunctionalization of the entire constituents of the segments related to pathologic viscus.

Defunctionalization of tagma or its equivalent: tagmatic phenomenon

All the referred phenomena do not seem to be expressed segmentally [7]. Referred pain from limb girdle and limb muscles usually has non-segmental nature [8]. Of course, although deep truncal muscles or vertebrae which keep segmental features from embryo to adult can have segmental referred pain, limb girdle and limb muscles lose segmental features according to growing up. These muscles assemble other synergistic muscles to make a functional unit. For example, muscles for retromotion of upper limb are composed of rhomboideus major and minor, trapezius, teres major, deltoid attached to the scapular spine, latissimus dorsi, long head of triceps, lateral and medial head of triceps, anconeus, extensor carpi ulnaris, extensor digitorum, abductor digitii minimi and extensor digiti minimi [9]. Muscular referred pains from teres major, latissimus dorsi or triceps appear on functional unit of synergistic grouped muscles. Muscles which lose their proper segmental features and get together under the same function should also have their pathologic expression, or referred pain, of non-segmental pattern. In Arthropoda, functional unit of grouped segments (tagma) is achieved by means of desegmentation [10]. The evolutionary achievement of tagma made much greater complexity in structure and function develop. For example, insects are divided into three major body sections which are called tagma. These are head, thorax and abdomen which are differentiated more than Annelida. In humans, tagma-equivalents are more complicated. Human has complex functional units composed of head, neck, thorax, abdomen, pelvis and limbs. And further, each limb has six functional grouped muscle units for antemotorion, retromotion, mediomotion, lateromotion, inratorotation and extrarotation [9]. Therefore, abnormality of a part of a functional unit can induce tagmatic phenomenon much like segmental phenomenon. In other words, a muscle belonging to a functional unit or tagma-equivalent has logically referred pain of “functional pattern”, not segmental, because the muscle already lost segmentation.

Clinical considerations of segmental and tagmatic phenomena

Based on the described above, it can be simplified that pathologic processes to start at endoderm (viscus) are transmitted to mesoderm (myotome, osteotome and dermatome) segmentally via referred phenomenon. This pathologic transmission is considered as segmental phenomenon analogically equivalent to the phenomenon of segmentation observed in Annelida or Platyhelminthes. Abnormality of viscus can be easily found out if we are familiar to characteristic patterns of referred pain. Clinical significance of referred pain is that it can lead us to identify injured viscus easily and treat the viscus as possible as early. But it is hard to find out pathogenic muscles to elicit referred pains in limb girdle and limb because they have usually non-segmental pattern [8]. In limb girdle and limb, trigger points and their referred pains develop and spread in a functional unit that consists of synergistic muscles. We commonly observe that associated trigger points, whether secondary or satellite, appear in accordance with functional unit of synergistic muscles [11]. Secondary trigger points are manifested in a synergist of the muscle harboring the key trigger point. And satellite trigger points also develop in the zone of referred pain of the key trigger point, in a synergistic muscle that is substituting for the muscle harboring the key trigger point. From these observations, it could be postulated that there are tagma-equivalents more differentiated in humans, called as functional units of synergistic muscles, analogical to tagmata (head, thorax abdomen) in insects. There are six tagma-equivalents in each limb. That is why myofascial pain and referred pain develop and spread in accordance with functional unit of synergistic muscles [8]. For example, myofascial pain of latissimus dorsi muscle may coexist with lower trapezius, rhomboid, teres major and long head of triceps brachii muscles frequently [11]. These muscles all belong to the functional unit for retromotion of upper limb [9]. Supraspinatus muscle commonly develops myofascial pains together with trapezius and deltoid muscles [11]. These are parts of the functional unit for lateromotion of upper limb [9]. Therefore, the fact that myofascial pain and referred pain are elicited in functional unit means that constituent muscles of tagma-equivalent can be defunctionalized together. This symbiotic dysfunction could be called as “tagmatic phenomenon” similar to segmental phenomenon. Tagmatic phenomenon also has muscular hyperalgesia (myofascial pain) and autonomic concomitants on referred pain area like segmental phenomenon. We can know where pathogenic processes start if we differentiate between segmental and tagmatic phenomena in our body. If an injury is evoked in deep truncual muscles, vertebrae or discs to keep segmental features, pathologic processes develop segmentally in all the mesodermal derivatives of common origin from the trunk to limb. On the contrary, if a problem develops in limb girdle and limb to lose segmentation, tagmatic phenomenon occurs in a functional unit non-segmentally. For example, somatic referred pain originating from the C5 spinal derivatives (the posterior aspect of annulus fibrosus, posterior longitudinal ligament, or anterior aspect of the dural root sleeve) [12] can produce C5 segmental pattern of muscle spasm and pain (rhomboids, supraspinatus, infraspinatus and deltoid muscles), but muscle pain starting at trigger point of supraspinatus muscle spreads non-segmentally to trapezius (accessory nerve) and deltoid (C5-6) muscles that belong to the function unit for lateromotion of upper limb [9]. Referred pain from latissimus dorsi muscle can be mistaken as C7 or C8 somatic referred pain [11]. To differentiate them clinically, it is helpful to find out associated trigger points in lower trapezius, rhomboid (C4-5), teres major (C6-8) and long head of triceps brachii (C6-8) muscles which are all func-
tional constituents for reteromotion of upper limb as well as key trigger points in latissimus dorsi muscle. Thus, it is important to seek associated trigger points arising from constituent muscles in the same functional unit to elucidate the cause of problem.

Complex regional pain syndrome is typically characterized by non-segmental limb pain and disability after a relatively minor injury to a limb but is more severe and lasts much longer than would normally be expected given the injury. The affected area has a tendency to spread outside the territory of the originally injured area non-segmentally even to parts of the body other than the limb or the opposite limb [13]. These areas have associated features similar to referred pain and its accessory phenomena, such as (1) hyperalgesia and allodynia, (2) abnormal vasomotor, sudomotor and pilomotor activities, (3) dystrophic muscles and bones [14]. If analogically extended, complex regional pain syndrome should be viewed as multi-taggmatic phenomenon, exaggerated, because functional disability is combined with autonomic phenomenon.

Pain and autonomic interaction

Pain, in the past, is simply thought as a central activation from convergent somatosensory activity. This activity is conveyed by sensory pathways to neurons located in the thalamus and somatosensory cortex. But in fact, this pain theory cannot explain why neither stimulation nor destruction of the thalamus or somatosensory cortex affects pain. Recent evidence suggests a new view of pain as a homeostatic sensation like temperature, itching, hunger or thirst. Pain is a physical sensation transmitted by homeostatic afferent pathway with direct thalamocortical projections (the anterior cingulated and insular cortices which are each limbic motor and sensory cortices eventually connected to the autonomic nervous system) [15]. Therefore, pain becomes a part of interoception as well as exteroception, and a reason for specific behavior [16]. Since pain have interoceptive function as homeostasis, it is reasonable that pain has intimate connections with the autonomic nervous system. Referred pain also accompanies autonomic phenomena. If it is so, referred phenomena that have pain and its autonomic concomitants simultaneously are thought of as homeostatic processes that are continuously adjusted to pathogenic viscera or musculoskeletal structures. Thus, it seems very probable that these homeostatic processes make pathogenic viscera or musculoskeletal structures protected from the external impact by producing muscle spasm, hyperalgesia, and increased sudomotor and lipomotor activities on referred pain area. But if these processes continue without solving underlying cause, defunctionalization can be manifested as dystrophic changes on the segments or tagmata related to the underlying cause. Although it is certain that defunctionalization stems from the phenomenon of segmentation on evolutionary view, its underlying mechanism is not clear. But evidences of autonomic overactivity on the referred area suggest that autonomic overactivity is related with defunctionalization. Therefore, to functionalize the segments or tagmata related to the cause of problem, we effort to calm down autonomic overactivity as well as to find out and treat the cause of problem.

Conclusion

There is a merit on evolution in that segmentation of animals increases the complex and efficiency of the whole organism and prevents pathologic processes in a segment spread to other segments. But in Vertebrata including human, there can be a demerit in that segmentation makes a segment or tagma having a problem functionless on purpose to use energy effectively for the individual. It seems likely that such defunctionalization of the segment or tagma is mediated by segmental or tagmatic phenomenon. From this, it could be postulated that referred phenomenon showing segmental or tagmatic features originates from the phenomenon of segmentation observed common to Annelida, Arthropoda and Vertebrata. Therefore, since referred phenomenon is thought to develop as a means of defunctionalization, it is important to early detect it and treat its underlying cause before defunctionalization is expressed completely. We must comprehend exactly pathophysiology and patterns of referred pain, whether segmental or tagmatic.

Conflicts of interest statement

None declared.

References