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Editorial

The Mystery around Suboccipital Myofascial Alterations and their Correlated Ailments. Could the Atlasprofilax Method be a Therapeutic Option?

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Abnormalities in the craniocervical junction (CCJ) structures can potentially promote biomechanical and metabolic dysfunction leading to several neurological, myofascial and musculoskeletal pathologies. Because of their subclinical character or because physicians lack particular understanding regarding this issue, CCJ and suboccipital myofascial alterations may frequently go unnoticed and uninvestigated. Some authors have coined the terms Craniocervical Syndrome (CCS) and Craniocervical Junction Syndrome [1]. Some pathological manifestations and CCS-associated symptoms may arise as a consequence of abnormal mechanical stress within the atlanto-occipital hinge. Suboccipital muscles alterations such as trigger points [2], modified cross-sectional area [3,4], or fatty infiltration [5] could cause a probable excess of tension coming from the myodural bridge (MDB) pulling the dura mater, which may end up with inflammation, soreness, and dysfunction in this region. Fibroblasts are at the basis of the fascial system playing an important role in the muscle tension transmission and are involved in the interstitial and transcellular fluids organization [6]. Fibroblasts play a big role in skeletal muscle regeneration and are essential for the proper assembly of proteins into functional fibrillar ECM. But fibroblasts' overactivity is involved in muscle disease resulting in debilitating fibrosis [7]. Increased nociceptive and proprioceptive information involving the MDB [8] could be linked to an excessive proliferation of fibroblasts within the myofascia in the CCJ region. This phenomenon has been defined by some authors as "fascial armoring" and results from abnormal amounts and layout of fibroblasts, normally leading to abnormal fascial tensegrity, playing a role in syndromes such as fibromyalgia (FMS) and myofascial pain [9]. Pathophysiological changes in the vertebral arteries angles and CSF flow obstruction may also play a role in some neurological CCS-associated problems that would involve the MBD [10], increasing the pressure within the intracranial and subarachnoid space [11]. Induced fibrous hyperplasia of the MDB in animal models demonstrated compensatory and distorted compliance of anatomic arrangements in the CCJ [12]. This dysfunctional mechanism has been proposed as etiology for Arnold-Chiari type I malformation [13].

Several studies seem to establish a correlation between CCJ abnormalities and multiple musculoskeletal pain syndromes of neck and head, such as headache [3,4,14-20] and migraine [21]. The posterior atlanto-occipital membrane (PAOM) can be compromised through excess of tension coming from the MDB [14,22], which has several connections towards the dura [23-29]. In addition, meningovertebrodural connections and the PAOM may play a big role in the probable etiology of several ailments [29]. PAOM expands from the occiput to C3 forming a membrane-dura complex with the craniocervical dura mater. The MDB has a proprioceptive feed-back mechanism for dural tension monitoring [8] and is linked through vertebrodural ligaments to the PAOM [22,30]. PAOM has been revealed through confocal microscopy to be part of the rectus capitis posterior minor, the fascia, the tendon, and the perivascular cover, vertically bypassing vertebral arteries grooves, venous plexuses, and the suboccipital nerve [30]. PAOM and MDB merging in the AO space has been defined as "posterior atlanto-occipital membrane-spinal dura complex" (PAOM-SDC) providing dura stabilization and is associated with headache and neck pain [23,29]. The PAOM, as per the case of the MDB, has often been neglected and has a very complex anatomy. This fact may difficult the assessment of its relevance and its probable role in several clinical ailments [31].

The question here is to raise awareness of this problem by quantifying the prevalence of probable abnormalities in the CCJ and elucidating pathomechanic or metabolic suboccipital myofascial alterations. Establishing clear clinical criteria not only for better detecting these anomalies but also for validating their clinical or subclinical significance through objectively measurable standards would help in drawing a diagnostic line. This may help in establishing differential diagnoses and probably also in the etiological understanding of some pathologies associated to benign pain. The prevalence of chronic benign pain and multiple related disorders among adults varies from 2% to 40% [32]. Often, its etiology remains unclear and has a difficult management. Chronic myofascial pain of benign origin is commonly related to joint and muscle overload due to asymmetries and deformities in the myofascial chains. Muscular and fascial mechanoreceptors, proprioceptors and nociceptors send various signals, especially pain signals, through the peripheral nervous system for biomechanical body adaptation. It cannot be excluded that central sensitization in chronic benign pain disorders, when probably linked to CCS, may also occur as a result of small fiber neuropathy originating from abnormalities coming from fascial and muscle receptors with dural connections in the suboccipital myofascial and the CCJ complex. It would be interesting to clearly elucidate which cellular mechanisms would modulate such processes in the absence of viral or bacterial infections. In the case of FMS, several studies revealed that central sensitization is linked with longstanding or permanent changes of nociceptors [33,34]. FMS can be described as the far end of a continuum beginning with local musculoskeletal pain and fascia affectation turning into in a widespread chronic disabling condition. An hypothetical correlation between central sensitization, as per in the case of FMS, and an involvement of CCS abnormalities should be explored. Metabolic and/or mechanical affectation of soft tissue in the CCJ region could turn in altered action potentials and nociceptive signals over the time explaining partially the wind-up phenomenon observed in central sensitization syndromes such as FMS [35,36]. Alterations of the suboccipital myofascial involving MDB stress and dura affectation could lead to a kind of mechanical-electrical "short-circuit" coming from suboccipital myofascial structures via some pain pathways, ending up in a hypersensitization of higher pain centers of the brain. Central sensitization seems to play a role in temporomandibular joint disorders (TMJD) as well [37].

In previous studies, the Atlasprofilax method has already shown benefits in TMJD [38], in FMS [39] and in misalignments of the C0-C1-C2 segment [40,41]. Those pathologies where the Atlasprofilax intervention showed therapeutic gains have a correlation with postural control deficits and imbalance [42-44]. The Atlasprofilax method is a therapeutic intervention focused on the improvement of altered biomechanical and metabolic patterns in the complex structures of the CCJ. This method uses device-mediated non-invasive vibropercussive mechanotransduction in the suboccipital myofascial for about 10 minutes. The aim is to induce a long-term positive metabolic effect by mechanotransduction to free various structures of the suboccipital myofascial from subclinical alterations such as excess fibroblasts, the presence of trigger points, compression of Arnold's nerve, improving fascial tensegrity in this important complex CCJ-region as well as in the deep cervical fascia. The concept of mechanotransduction is closely linked to the fascia and may reveal many still unknown mechanisms related to benign chronic pain. A multi-molecular bridge formed by integrins shapes the cytoskeleton; some actin and myosin filaments are involved in producing mechanical tension within the cell as well as to the extracellular matrix through integrins. This phenomenon also extends to epithelial cells, nerve cells, immune cells, bone cells, and fibroblasts [45]. Thus, primary or secondary distortions of metabolic or biomechanical origin in the CCJ could probably underlie several undetected painful pathologies.

In conclusion, there is still much to learn about the mystery of suboccipital myofascial alterations and their role in various chronic benign painful pathologies. This mystery should be explored deeply. The use of device-mediated external mechanotransduction used by the Atlasprofilax method on the suboccipital myofascial seems to offer interesting potential in this field. This could help elucidate some unexplored characteristics that link fascia and mechanotransduction in pathological conditions associated with chronic benign pain.

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