The increased distensibility of the wall of cerebral arterial network may play a role in the pathogenic mechanism of migraine headache

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Background- It is now well accepted that migraine headache is mediated by the increased sensitivity and ensuing activation of trigemino vascular nociceptive afferents that innervate the dura mater and their related blood vessels. One of the fundamental questions is to determine which processes actually play a role in promoting such condition.

Objective- The aim was to evaluate whether patients with episodic migraine with (MA+) and without aura (MA-), during the interictal period of migraine would have an altered distensibility of the wall of cerebral arterial network and whether it would play a role in migraine headache.

Methods- To evaluate the distensibility of the wall of cerebral arterial network, we measured the time-delay in milliseconds (ms) between the R-wave of an electrocardiogram and the arterial pulse wave (pulsatile arterial blood) of cerebral microcirculation (R-APWCMtd) on the frontal cortex (Fig. 1,2,3) detected by near-infrared spectroscopy (NIRS) in 10 patients with MA+ (age 39.5 ± 12.2 years), in 10 with MA- (age 40.3 ± 10.2 years), according to ICHD-3 criteria 2012, during the interictal period of migraine, and in 15 age-, sex- and height-matched healthy control subjects.

Discussion- In young, healthy adults, the aorta has a high distensibility and first-generation arteries are relatively stiff. This abrupt transition from the aortic distensibility (low impedance) to the stiff (high impedance) branch vessels represents an impedance mismatch that represents a protective mechanism that limits the transmission of excessive flow pulsatility into the microcirculation. The longer R-APWCMtd may lead to the reduction of the impedance mismatch and thereby may facilitate the transmission of excessive pulsatile energy or flow pulsatility into the cerebral microcirculation of the dura (via the middle meningeal artery). The excessive flow pulsatility may more easily sensitize the mechano-sensitive nociceptor TRPV4 of the trigemino vascular system reducing the threshold for migraine headache.

The meningeal arteries lie mainly in the outer, or endotheal layer of the dura, which in humans is a tough membrane that is rigid, resistant to stretching and is tightly attached to the cranial bones. We suppose that this condition may facilitate the mechanical stimulation of this pain-sensing neuron (nociceptor) innervating the dura and their related blood vessels.

Results - The patients with migraine had a significantly longer R-APWCMtd than the control subjects F = 13.4, p<0.001; MA-:+38.3 ms; MA+:+34.7 ms (Fig. 4). In the multiple regression analysis, the R-APWCMtd was significantly associated with migraine (coefficient of determination R² = 0.50, multiple correlation coefficient 0.71, p<0.0001) but not with age, gender, height, migraine attack frequency and disease duration. The two groups were matched (no significant difference) for age, sex and height. There was no significant difference between the right and left R-APWCMtd of frontal cortex in the migraine patients p = 0.77 and controls p = 0.70.

Conclusion- In conclusion, in our migraine patients, a longer R-APWCMtd is independently associated with migraine and indicates an increased distensibility of the wall of cerebral arterial network. The increased distensibility leads to an increased flow pulsatility into intracranial dural meningeal vessels that may lead to a mechanical stimulation of the nociceptors that innervate the dural vasculature. This condition may play a role in promoting the sensitization of trigemino vascular afferents and a sterile inflammation within the dura mater that are fundamental to the pathogenesis of migraine.