



"MIGRAINES" Interesting Clinical Case and New Etiology Prospective

A thirty-one year old women was sent to our clinic in August 2012 by her neurologist to try to diminish a bad spasm on the left upper Trapezius muscle which was giving the patient neck and shoulder pain for over a year. The women had suffered from "ophthalmoplegic migraine" since the age of eighteen. The migraine is present every day. Starting in the morning, sometimes waking her up with pulsing pain on the left temple, the extrinsic muscle of the left eye became paralyzed and the pain diffused on the left side of the face and all over the head. The left upper extremity is involved too with paresthesia and hypoesthesia. (Typical symptoms of the ophthalmoplegic migraine).

She is currently taking "Ketoprofen" (an NSAIDs, a non steroid anti-inflammatory drug). Every morning when she wakes up, she starts feeling pulsing on the left temple and has to take the Ketoprofen. She has been doing this since she was twenty-two years old and every night before sleeping she takes "Lamitrogene" (an antiepileptic drug which stabilizes the membrane of the cells, diminishing the activity of the Na-channels and the NMDA receptors = for glutamate) and has been for over four years now. Prior to this she was using other antiepileptic drugs but, the side effects where to heavy. If she does not take both drugs daily her symptoms reappear quickly and, after less than one hour they are not treatable anymore with these drugs. She needs to take "Sumatriptan" (a serotonergic agonist, selective for the brain serotonin receptors 5-HT1d). Epilepsia was medically excluded. Apart from these symptoms her clinical story is unremarkable. These migraine symptoms first showed up after an accident she had to her head when she was fourteen years old. Those symptoms lasted few days. Later, when she was eighteen they reappeared, starting out gradually once a month or every other month and, got worse and worse over the years until they started showing up every day. The trapezius stiffness even gives her an annoying cephalalgia on the left side of the head when the trapezius gets very tight.

VISITS:

At first visit the patient was found positive for many TP in the sub-occipital region and the left anterior and posterior neck, UTRP and LV on the left, some Posterior Upper Thoracic TP and some Anterior and Posterior Lumbar and S2 on sacrum. She was treated at first visit on: Left (In, PC2, AC5, LC1, PT3, PL2, PL5, AL5) right (LC1, PC3, AL5), the UTRP and LV were gone after treating the neck and thoracic region, since they disappeared, were not treated in this session.

After two days, she returned for a second visit saying that she was no longer feeling the pulsing pain on the temple area anymore, and the TRP area was only very sore. She also referred to a variation in her voice which recently in the past two months had changed to a more screeching like sound. She said her voice had returned back to normal or near to. This time we treated less points. Some of them where still there: Left (In, PC2, AC1, LC1, AC7, AL5, SFE=Sphenoid, LC=Lateral Cantus), right (LC1, PC3, AL2), UTRP and LV where treated even if the TP's weren't so hot. We advised her to take Ketoprofen only if she was feeling the pulsing pain.

After four days, she returned for the third visit. She was off Ketoprofen for all those days. She had no more TRP pain. This time the TP were mildly painful and we treated her anyway on: Left (In, LC1, AL5, SFE=Sphenoid, LC=Lateral Cantus, PO=Posterior Occiput), right (LC1). We sent her back to her neurologist after the four days of no pain and suggested they consider progressively diminishing the Lamitrogene.

We heard from the patient via email over the next five weeks, standing by ready to treat her if she had any returning pain symptoms. No visits were needed.

After one week with no Lamitrogene and more than one month without Ketoprofen, the patient returned to our clinic that fifth week for a follow-up (fourth visit). She had only a few tender points on the anterior neck and cranium. We treated out: Left (AC5, TP = Temporal, LC, BC = Bilateral compression), Right: AL2 and dismissed the patient as cured. We advised her to contact us if the pain restarted, and to keep us updated via email. In October 2012 she sent us an email letting us know she is still pain free and with no drugs.



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OUR THOUGHTS:

For the medical field "migraines" have an uncertain origin. What is known is that a vasodilatation of "Dura" vessels occurs, activating the trigeminal roots that innervates the dura vessels. This causes the release of neuropeptides which ulteriorly vasodilate and activates the descending nucleolus of the trigeminal nerve, which propagates the pain in the diffuse area of the brain. The reason why this initial vasodilatation occurs is unknown, circulation problem or spasm on the dura vessels prior to dilation have been theorized, and everyone is orientated towards an inner brain organic cause.

What we think is that the accident she had on the head started tension in the sub-occipital region which after some years created tension on the dura and on the posterior C2 nerve root (major occipital nerve) which runs on the external skull and it's sensitive components anastomosis with the trigeminal nerve on the parietal area, activating this way on the trigeminal nerve. Over time, the compression of the sub-occipital region was probably compressing the XI and X cranial nerve, that exit the brain in this region, the first anastomosis with C2 and C3 and innervates the TRP and the SCOM. By reducing the compression on the nerve and treating the 2° vertebra the TRP was much more relaxed (we started treating many patients with tight UPTRP from the sub-occipital region and 2° cervical vertebra obtaining much more effect than a local work on the upper thoracic, low neck and TRP area). The vagus nerve anastomosis with C2, controls many areas as well as the vocal cord tension by the laryngeal nerve. The voice improved immediately. By releasing TP (In, S2 & PC2, LC, SFE) we probably released tension on the dura as well, and by normalizing the PC2, LC, SFE we diminished the firing on the trigeminal nerve from the C2 posterior root and locally on the ophthalmic root that passes on the forehead and sphenoid region.

CONCLUSION:

It is important to focus on the fact that migraines are usually described as pulsing pain on the temple area and pain or paralysis of the eye. All these areas are sensitive trigeminal innervated. We really believe that the migraine origin must be searched majorly an external musculoskeletal influence which reacts on the dura vessels by the external afferent trigeminal terminals, such as ophthalmic branch or spinosal nerve more than thinking an inner brain effect on dura vessels.

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