The cerebral venous system in migraine: the not so innocent bystander?

The role of the venous circulation of the brain has long been underestimated in clinical practice as well as in research into neurological diseases. Indeed, for many years the cerebral venous system was regarded simply as a series of vessels that collect venous blood and channel it back to the heart. In recent years, abnormalities of the venous system have been implicated in a variety of neurological disorders, including multiple sclerosis, leukoaraiosis, vascular dementia, and normal-pressure hydrocephalus (Beggs, 2013).

In this volume of Functional Neurology, Petolicchio et al. (2016) present intriguing data on the higher occurrence of cerebral venous hemodynamic abnormalities in migraineurs than in controls. Furthermore, the presence of chronic cerebrospinal venous insufficiency emerges as a potential factor for migraine chronification. Chronic cerebrospinal venous insufficiency is characterized by multiple intraluminal stenotic malformations of the principal venous drainage pathways, particularly in the internal jugular veins (Beggs et al., 2014).

The physiological mechanisms associated with cerebral venous outflow are poorly understood, but it is generally recognized that cerebral venous outflow plays an important role in intracranial hemodynamics, which in turn appears to influence the perfusion of the brain parenchyma and the dynamics of the cerebrospinal fluid system. There is evidence that occlusion of the venous drainage pathways causes blood to accumulate within the cranium: compression of the internal jugular veins results in an up to 20% increase in intracranial blood volume together with an increase in the pulsatility of the pial arteries (Frydrychowski et al., 2012; Kitano et al., 1964).

Considering these observations from the perspective of migraine pathophysiology, it becomes apparent that alterations in cerebral venous hemodynamics may play a role in the mechanisms involved in migraine attacks at several levels: increase in intracranial pressure, facilitation of inflammatory processes, and activation of the trigeminovascular system via the distension of blood vessels or of dural sheaths (Burgos-Vega et al., 2015).

Not explored by Petolicchio et al. but definitely worth future consideration is the effect of sex hormones (and the expression of their receptors in cerebral veins), considering that cerebral venous thrombosis is more common in women and associated with hormonal factors, and that migraine is a typical disease of the female gender closely connected with hormonal fluctuations (Serra et al., 2016; Koopman et al., 2009; Borsook et al., 2014).

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References