

VASCULAR: COMPLEX VASCULAR AND NEURONAL ASPECT OF MIGRAINE

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Migraine headaches have a complex pathophysiology; both vascular and neuronal mechanisms have been proposed. Cortical spreading depression (CSD) can cause both migraine aura and trigeminal activation, which in turn, promotes neuropeptide release and triggers peripheral and central mechanisms that results in headache. The characteristic unilateral pulsating headache is caused by a neurogenic inflammation in the meninges. Dilatation of cerebral and dural arteries causes a throbbing, migraine-like pain. Both calcitonin gene-related peptide (CGRP) and nitrogen oxide (NO) are potent vasodilators that can induce migraine. However, vasoactive intestinal peptide (VIP) mediates a marked dilation of cranial arteries, but does not trigger migraine attacks in migraineurs. These data provide further evidence against a purely vascular origin of migraine. Furthermore, CGRP does not cause the familial hemiplegic migraine (FHM) phenotype in FHM patients. The nitroglycerin (NTG) model produces substantially different vascular effects than those seen with spontaneous migraine headache. Current specific drugs used in the acute treatment of migraine interact with vascular receptors. The first oral neuropeptide CGRP antagonist, MK-0974 has recently been shown to be highly effective in the treatment of migraine attacks. Furthermore, a recent study revealed that pretreatment of experimental animals with L-kynurenine (a tryptophan metabolite) plus pobenecid significantly mitigates the NTG-induced increase in the number of c-fos-immunoreactive nerve cells in the caudal trigeminal nucleus. Some evidence suggests that kynurenine system influences cerebral blood flow (CBF) under both normal and pathological conditions. Therefore, the alterations of the kynurenine system may be involved in the pathogenesis in migraine. "