Letter to the Editor

Non-lateralizing brain PET changes in migraine: phenomenology versus pharmacology?

Vinod Kumar Gupta

Dubai Police Medical Services, Dubai, United Arab Emirates

Correspondence to: Dr Vinod Kumar Gupta, MD Physician, Dubai Police Medical Services, PO Box 12005, Dubai, United Arab Emirates
E-mail: docgupta@emirates.net.ae
DOI: 10.1093/brain/awh182

Matharu et al. (2004) believe that the dorsal rostral pons is probably involved in the pathophysiology of chronic migraine. The clinical distinction from episodic migraine is rather semantic and one that is based simplistically on the frequency of headache attacks rather than any fundamental variation of phenomenology. To maintain perspective, it is important to underscore that the International Headache Society classification as well its revisions (Silberstein et al., 1996) are based purely on phenomenology. Similar PET findings in chronic migraine and episodic migraine are, therefore, expected and not surprising.

A series of assumptions sustained by neglect of key pharmacological absolutes underlie current understanding of the pathophysiology of migraine. It cannot be argued that migraine prophylactic agents affect primary pathogenetic processes and, therefore, such drugs must cross the blood–brain barrier (BBB) freely in order to influence significantly any brain neuronal function.

Well controlled studies (Stensrud and Sjaastad, 1980; Forssman et al., 1983; Johannsson et al., 1987) and case reports (Kowacs and Werneck, 1996) have demonstrated the unexpected but consistent efficacy of atenolol in preventing migraine with or without aura, and the issue has been critically reviewed (Andersson and Vinge, 1990; Emilian and Maloteaux, 1998). Atenolol is included in the list of drugs of first choice for migraine prophylaxis (Massiou and Bousser, 1992; Beubler, 1994; Emilian and Maloteaux, 1998). Again, in combination with the monoamine oxidase inhibitor, phentolamine, atenolol remits migraine headache (Merikanganas and Merikigans, 1995). Atenolol, nevertheless, does not freely cross the BBB (Cruickshank, 1980) and, therefore, cannot critically influence brain neuronal function(s). Similarly, verapamil, a widely used agent with limited evidence of benefit (Goadsbey et al., 2002), does not cross the BBB (Agnoli, 1988). Some therapists prefer to use verapamil prophylactically in patients with migraine aura, with or without headache, particularly when auras are frequent or associated with hemiparesis (Silberstein and Young, 1995). Verapamil, nevertheless, does not alter brain neuronal function in any clinical circumstance. Nadolol is regarded as one of the drugs of first choice for migraine prophylaxis (Emilian and Maloteaux, 1998), but a Medline review does not indicate that it crosses the BBB. Finally, there is much interest in prophylaxis of migraine headache (Wang et al., 2003) and treatment of migraine aura (Rozen, 2003) with magnesium supplementation, but magnesium does not cross the BBB (Ko et al., 2001).

A critical mass of available pharmacological evidences strongly suggests that PET recording of brain neuronal function is part of the vast phenomenology of migraine and does not indicate pathophysiological involvement of the relevant brain areas. Importantly, the absence of lateralization of PET changes in this study (Matharu et al., 2004) largely attenuates clinically such putative pathogenetic implications (Gupta, 2003a,b). Remarkably, while the experiment of Matharu et al. (2004) is a modulation of terminal events of the migrainous process, these authors theoretically extrapolate their findings to early primary events of the pathogenetic process. For any meaningful advance in migraine pathophysiology, the apparent disconnection between phenomenology on the one hand and pharmacology as well as clinical neuroscience fundamentals on the other hand has to be addressed.

References


Letters to the Editor


Ko SH, Lim HR, Kim DC, Han YJ, Choe H, Song HS. Magnesium sulfate does not reduce postoperative analgesic requirements. Anesthesiology 2001; 95: 640–6.


