

Point of view

Avoiding nightmares in migraine management

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Journal of the Ceylon College of Physicians, 1994, 27, 54-55

In a recent survey on migraine among medical students and doctors I found some misapplications in management which in some instances could make migraine worse.

My letter will only attempt to highlight some management points that may be helpful in relieving the migraine sufferer, but are often missed.

Prophylaxis

The mind of many physicians is focussed on the impressive array of drugs available for prophylaxis which includes low dose aspirin, beta blockers, serotonin antagonists, antidepressants, calcium antagonists (flunarizine), and even feverfew (*Tenacetum parthenium*). In most double blind trials clonidine has not proved to be of value¹. However the most important aspect of migraine prevention should be the identification of trigger factors and their elimination. The frequency of attacks can be reduced by up to 50% by identifying and avoiding trigger factors². Well documented trigger factors are: foods containing amines (eg. cheese, sausages, yeast extracts, meat extracts), chocolate, citrus fruit, red wine, other alcoholic beverages, peanuts, cadjunuts, food flavoured with monosodium glutamate; consumption of large quantities of tea, colas and coffee; odours emanating from cigars or cigarette smoke, paint, diesel or petrol fumes, tar, perfumes; bright light (TV watching); changes in barometric pressure; excessive or inadequate amounts of sleep and psychological stress; mental and physical fatigue; poor posture (neck position) during office work or study should be looked for and corrected. The menstrual period is a well known trigger factor in women, as are oral contraceptives and oestrogen replacement therapy after menopause. Often, migraine appears during rest that follows intense activity. Fasting must be avoided, regular meal times are mandatory.

The clinician must take time to discuss the problem with the patient and not limit himself to prescribing a new drug every time a migraine attack comes on. The patient must be helped to identify the trigger factors in his lifestyle or food habits that aggravate migraine. It is important to

teach the patient how to reduce workload and to have leisure times. A detailed history should be taken to identify trigger factors and time should be taken to advise the patient. Clearly the family physician is more adept at this than the busy consultant. It is unfortunate that these patients (even doctors and medical students) go through so many drugs and numerous physicians without attention to preventable factors.

Secondly a drug tried for prophylaxis is terminated too soon, and a new one commenced, when the former ought to have been prescribed at least for 4-6 weeks. Longer the list of drugs available for treatment, greater the risk of mismanagement ought to be a pharmacological axiom. It must be noted that beta blockers with intrinsic sympathomimetic activity (eg. pindolol, oxprenolol, acebutalol) should not be prescribed, though this is not uncommonly done. What could be prescribed are propranolol, metoprolol, atenolol, nadolol and timolol³. A person who may not respond to one beta-blocker may respond to another. The dose of propranolol that may produce the desired effect may vary from 20 mg to 240 mg/day orally.

Thirdly, low dose aspirin therapy (300 mg/d) is useful in prophylaxis⁴. In this dose aspirin blocks the synthesis of thromboxane (agregatory factor) in platelets. Higher doses of aspirin will block synthesis of prostacyclin (platelet antiaggregatory factor) in arterial walls and may make migraine worse. Hence it is unwise to prescribe repeated doses of aspirin or other NSAIDS even for an acute attack.

The other drugs available for prophylaxis and their schedules are well documented¹. Aspirin 300 mg/D, naproxen 500 mg twice a day, fenoprofen 200 mg thrice a day have proved effective in prophylaxis. Drugs are recommended for prophylaxis when migraine headaches occur more than two a month or when a single attack causes significant loss of work days. Prophylaxis is usually tailed off after six months¹.

Treatment of the acute attack

The patient must be taught to recognise the prodromal symptoms — dull heaviness of a side of the head, food-craving, yawning and sleepiness. The activity that brought this on must be stopped. (eg. heavy office schedule, studying for an exam). A migraine attack is better

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prevented at this stage rather than allow it to fully blow up. The patient should be advised to take an effective dose of a drug early. Taking aspirin 600 mg or paracetamol 500 mg with food and sleeping may be all that is necessary. Topical counter — irritants viz. oil of wintergreen, hot and icy cold fermentation, massage of the scalp greatly relieve the patient and promotes sleep. An attempt must be made to abort the attack completely. The patient (often a busy professional or a student) must be made to understand that resting with the first prodromata can save many hours of unnecessary work loss. In my analysis the major cause of rebounding migraine headaches was inadequate leisure time given to recover from the first attack.

Besides aspirin ibuprofen (1.2 - 1.8 g in divided doses) and naproxen (750 mg at on set; 250 - 500 mg every 30 to 60 minutes; upto 1250 mg/D) are extremely potent in aborting acute attacks. Inducing vomiting before swallowing the analgesic brings significant relief. The patient discovers this by himself. The food taken with the analgesic promotes absorption and reduces the nausea. Lack of response could be due to vomiting; often metoclopramide 10 mg orally or by intramuscular injection promotes gastric emptying and reduces nausea. In children and young women domperidone is preferable because metoclopramide is associated with dyskinetic reactions. Cyclizine as an antiemetic is a cheaper alternative.

If vomiting is a problem indomethacin 50-100 mg or diclofenac 50-100 mg as suppositories will be useful. Many physicians do not adequately make use of the rectal route before switching to other drugs. I make the point that with whatever NSAIDS used an attempt must be made to abort the attack with the first or second dose. Continuing drug dosing is the chief cause that converts an episodic migraine headache into a dull continuous one¹. With the wide variety of NSAIDS available for this purpose opioid analgesics have no place, considering the increased nausea and their addictive qualities. Though the migraineur becomes familiar with many drugs, he

must be strongly discouraged from self-medication with many combined preparations available over the counter. If the first dose of simple analgesic fails to abort the headache, it is best that he consults his family physician.

I would discourage the use of many ergotamine tartrate combined preparations available over the counter. Ergotamine preparations should not be tried till the above NSAIDS have been tried with proper attention to food, sleep and other preventable factors. I have encountered patients ingesting a tablet of an ergotamine combine preparation twice a day for a few days; the physician had failed to warn the patient that the maximum for a week should be 10 tablets (i.e. 10 mg) and that there should be a four day hiatus after a day's therapy with ergotamine before anymore is administered. A patient must not use ergotamine on the second day of a migraine attack. The caffeine in some ergotamine combines can prevent sleep and make migraine worse¹.

Sedation promotes sleep and relieves anxiety. A short — acting benzodiazepine (eg. lorazepam 1-2 mg) for a short period is useful.

Sumatriptan (5 Hydroxy tryptamine-1 agonist) is not available in this country for use. It is effective orally (100 mg) and by subcutaneous injection (6 mg). Transmural myocardial infarction has been reported with its use in a woman who had no previous history of ischaemic heart disease or Prinzmetal's angina⁵.

References

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