
Clinical Topics

A plain man's guide to the management of migraine

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Several family practitioners have told me that their hearts sink when a patient comes into the surgery complaining of migraine. I can sympathise with this feeling if the waiting room is full; it takes several minutes to establish a diagnosis, and a few more to find out the particular patient's triggering factors and then to explain and prescribe the appropriate treatment. The patient may therefore need to come another time when 10-15 minutes can be set aside for the consultation.

I suggest some simple guidelines that may help with managing migraine.

Making the right diagnosis

The foremost objective is to make the correct diagnosis. In migraine an aura is not necessary, occurring in only 15-20% of patients. There are two varieties of migraine, migraine headache and migrainous neuralgia, which must be separated from two other common head pains—tension and muscle contraction headaches. Although tension and muscle contraction headaches are currently considered to be synonymous, I propose a separation as described below.

DEFINITIONS

Migraine—Episodic headache accompanied by visual or gastrointestinal disturbances, or both, attacks lasting hours with total freedom between episodes—The visual symptoms occur as an aura before or photophobia during the headache phase. The

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alimentary symptoms consist of nausea and vomiting. If there are no visual but only gastrointestinal disturbances then vomiting must feature in some attacks.

Migrainous neuralgia—Episodic attacks of pain behind one (the same) eye, which becomes red and waters, the nostril on the ipsilateral side becoming blocked or discharging a watery fluid. Attacks last 20-120 minutes, occur daily for several weeks (hence the term cluster headaches), often wakening the patient in the early morning hours—sometimes so regularly as to merit the term "alarm-clock headaches." During a cluster period alcohol provokes attacks.

Tension headache—A continuous symmetrical headache often described as a pressure, awareness, or discomfort at the vertex, forehead, or occiput, or a coronal distribution or all over the head, not associated with visual or gastrointestinal symptoms. The ache lasts hours, often throughout the waking period, rarely interferes with daily activities, and is unaffected by analgesics.

Muscle contraction headache—A painful tender muscle, or a muscle spasm adjacent to a painful site—for example, cervical spondylosis or a temporomandibular joint. Treating the underlying cause gives partial or complete relief of symptoms.

Multiple diagnoses—Migraine is common (5-7% of the population) and clearly not all migraine sufferers come to the family practitioner. Those patients who seek advice often have complications—for example, migraine and fear of a sinister disease in the head, migraine and tension headache, or migraine and muscle contraction headache. Hence we may need to analyse two varieties of headache, which is not possible in the middle of a busy surgery (but neither is that the time to undertake an antenatal or insurance examination).

What to do if you cannot make a diagnosis

No doctor in general or hospital practice can make a diagnosis every time. With any intermittent symptom a record kept by the patient can help. The chart should have columns for the

date, day of week, time of onset, duration of attack, site of pain, severity (mild, moderate, or severe), associated symptoms, what the patient did during and before the attack, and "other observations." If the chart is divided into morning, afternoon, and evening, and the case is reviewed after two to four attacks have been recorded in this way, the picture becomes clear, particularly whether the pain is continuous or intermittent. You can use the interval to assess the response to abortive therapy (see below).

DIFFERENTIAL DIAGNOSIS

I have said nothing about other headaches, having concentrated on headaches that have recurred or lasted over months or years. Recent headaches present a different problem: glaucoma and temporal arteritis are important conditions that must not be missed because they are remediable. The headache of raised intracranial pressure is progressive and the neoplasm, by infiltrating brain tissue, produces focal neurological symptoms and signs and at times epilepsy.

REFERRAL TO A SPECIALIST

Not all patients with migraine need or can be referred to a specialist. But if at this stage the diagnosis is not reasonably clear then referral to a specialist—a neurologist or general physician with an interest in headache—may be indicated. Unfortunately, no antimigraine drug approaches 100% efficacy, so a "therapeutic test" is not possible.

Ferretting out the fear of migraine

The pain of a severe migraine attack makes many sufferers apprehensive of a sinister underlying disease. This fear must be ferretted out but not suggested in case one adds further problems: tumours, growths, cancer, insanity, or some other cerebral vascular catastrophe often feature in the patient's mind and can be elicited only by direct questioning. Here the family practitioner is often at an advantage because he knows that a neighbour, a member of the family with a cerebral tumour, stroke, or other brain disease has aroused fears in the patient's mind.

Mellontaphobia—I have recently encountered several patients whose "life has contracted" because they are frightened of making arrangements, such as going to the theatre or inviting friends to a dinner party. I am indebted to two Greek scholars who independently have suggested "mellontaphobia" (fear of forthcoming occasions), which elegantly and accurately describes this symptom.

Finding the triggers that provoke migraine attacks

Here the patient with guidance can become a detective discovering his or her own triggering mechanism.

Dietary factors—Having insufficient food, delaying meals, or eating cheese, chocolate, alcohol, or citrus fruits (all common culprits).

Sleep—Having too much or too little, or taking sleeping tablets.

Hormonal changes in women—Premenstrual tension or taking the contraceptive pill.

Local pains arising from the eyes, sinuses, teeth, or neck (the neck is a particularly common source in middle age).

Environmental factors—Heat, light, noise, shopping, or a combination of these.

Psychological factors—Anxiety, depression, frustration, too much work, and conflicts at work or in the family.

Allergy—Recent radioisotope immunoassays indicate that some patients may be especially sensitive to wheat and wheat products, milk, eggs, fish, or tea.¹

Exercise.

Travel.

These triggers must be worked out with the patient, whose attention needs to be focused by direct questions about the various factors. For example, "Have you ever found missing or delaying meals can provoke an attack? Or eating cheese, chocolate, or alcohol?" Attacks do not invariably follow exposure to the stimulus and I explain, "It does not always rain when there are clouds in the sky, yet we believe that clouds cause rain."

This then is the approach for preventing attacks. Migraines cannot be avoided altogether, however; so we need a method of effectively aborting or quickly resolving attacks.

Early effective treatment to abort attacks

A major discovery at the City Migraine Clinic has been that metoclopramide (10 mg) followed 10 minutes later by three analgesic tablets (aspirin or paracetamol) provides effective treatment in most cases.² This has now been confirmed in industry: 35 out of 36 attacks of migraine were aborted in this manner in a food factory in the home counties, where workers returned to the bench within one hour.³ Further proof of the efficacy of this combination is that drug companies are profitably selling this antiemetic and analgesic mixture (Paramax and Migravess). Another combination of an analgesic and antihistamine (the latter to counteract nausea), called Migraleve, is also claimed by some patients to be effective.

Ergot like other drugs can be addictive and Wilkinson² has made an important contribution in describing ergotamine overdose headache. In my opinion, however, the danger of ergot poisoning has been overemphasised. Many patients find relief from ergot taken by one route or another—mouth, suppository, or occasionally injection. This group of patients claims that ergot is the only effective measure to stop attacks. I see no objection to otherwise healthy people taking 2-4 mg or even more of ergot a week if that is what they find necessary to stop their attacks.

RESOLUTION OF MIGRAINE ATTACKS

I have recently studied the way attacks end.⁴ Out of 50 subjects, 34 finished their attacks during a night's sleep, some having gone to bed early. Fourteen of these 34 shortened and ended attacks by going to sleep during the day for an average of two and a half hours, aided by lying in a darkened room with a hot water bottle or central heating to warm themselves up. Others needed a variety of antimigraine preparations. The efficacy of sleep in resolving the attacks is not a new idea but it raises again the possibility that migraine is a primary neurological disturbance with secondary vascular manifestations rather than a primary vascular disorder.

Interval treatment: last and least

Only a few patients need interval treatment, taking tablets two or three times a day to reduce the frequency and severity of headaches. The drugs used range from mild sedatives, such as diazepam and stemetil, to antimigraine agents, beta-blockers such as propranolol, or anti-serotonin agents such as pizotifen or methysergide. Folk medicine also has its place, and some patients claim great benefit from taking a few leaves of feverfew. Other patients need antidepressants for depression as a complication of a migraine. Acupuncture, biofeedback, and exclusion diets also claim their successes.

Discussion

"Do something about that migraine" is a fine phrase, coined by Dr K M Hay, a Midland general practitioner with an interest in migraine for many years. The important message to the patient is that something can be done to reduce the severity and frequency of migraine, although at present we cannot cure the condition. Nevertheless, the patient must learn to cope and not expect a tablet to prevent all attacks or reduce worries at home or at work. Sometimes advising a patient to attend a relaxation class can reduce anxiety and tension.

I have not discussed the management of tension headache—a different and more difficult subject. Usually the underlying disturbance is anxiety, depression, or agitated depression that needs to be treated on its own merits along psychological lines. Muscle contraction headache as defined here is helped by treating the underlying cause of pain—for example, arranging physiotherapy to the neck or referring the patient to a temporomandibular joint clinic if appropriate.

The family practitioner seems to me to be the ideal physician to treat patients as a whole and as an individual. We should all avoid becoming "pill pushers," trying another and yet another preparation until the patient says (and they often do), "I have tried every headache tablet in the chemist shop." Undoubtedly many migraine sufferers have only a few attacks, perhaps one or two a year, and treat themselves. Some with more frequent attacks also try to treat themselves or take advice from friends. Of such patients it has been said, "A doctor treating his own illness is dealing with two stupid persons." I advise those patients to let me or someone else take on the responsibility of treatment.

I have left until last the treatment of migrainous neuralgia because this is the easiest of all the four conditions that I have discussed. Prophylactic ergotamine tartrate suppositories, inserted several hours before an attack is due, is effective in

eight out of 10 patients. If the attacks occur at night then the suppository has to be inserted before the patient retires to bed. In diurnal attacks an additional suppository is used in the morning after the bowels have been opened. Migrainous neuralgia is rare, however, and only one family practitioner in four has experienced such a case. Patients may complain of side effects after using a whole suppository and half or two-thirds of a suppository may suffice. Total abstinence from alcohol is recommended during the cluster period.

Conclusion

Once the diagnosis has been clarified, treating migraine and guiding patients to cope with their migraine is rewarding; you can help most patients most of the time. Although a cure is not yet available, active research along many lines is proceeding all over the world—a further comfort to those affected.

I am grateful to two Greek scholars, Mr John R M Smith of St Paul's School, London, and Mr Stacy Colman, formerly of Shrewsbury School, for their advice about "mellontaphobia," a concept that may be applicable to other disorders.

References

- 1 Monro J, Brostoff J, Carini C, Zilkha K. Food allergy in migraine. *Lancet* 1980;ii:1-4.
- 2 Wilkinson M, Williams K, Leyton M. Observations on the treatment of an acute attack of migraine. *Res Clin Stud Headache* 1978;6:141-6.
- 3 Jones A, Harrop C. Study of migraine and the treatment of acute attacks in industry. *J Int Med Res* 1980;8:321-5.
- 4 Blau JN. Resolution of migraine attacks: sleep and the recovery phase. *J Neurol Neurosurg Psychiatry* (in press).

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For four years a 23-year-old woman has been taking phenytoin 100 mg twice daily for epilepsy. She has also been taking oral contraceptives (a combined preparation containing 50 µg of ethinyloestradiol), but during the past year she has been experiencing intermenstrual bleeding lasting seven days. Her reproductive tract seems normal. I presume that the mechanism of the intermenstrual bleeding is induction of liver enzymes by the phenytoin. How should she be treated?

Breakthrough bleeding among oral contraceptive users taking phenytoin is well recognised,¹ and accidental pregnancies have been reported. Besides phenytoin, phenobarbitone, primidone, and carbamazepine may cause breakthrough bleeding, and the risk is greater with low-dose oral contraceptives. The mechanism is the induction of hepatic microsomal enzymes,² and another factor may be induction of sex hormone binding globulin,³ reducing the proportion of steroid free in the circulation. Again, increased steroid metabolism in the wall of the gut may possibly occur.² There are three possible methods of treatment. One is to change to a non-hormonal method of contraception; this will deal with the breakthrough bleeding, but the failure rates of non-hormonal methods are probably equal to—or higher than—the failure rate of the pill among patients taking phenytoin. The second possibility is to change the antiepileptic medication from phenytoin to sodium valproate, which may be preferable⁴ and which does not seem to affect contraceptive efficacy. Sodium valproate, however, is not suitable for all types of epilepsy. The third possibility is to increase the dosage of the oral contraceptive by prescribing two types of pill simultaneously to bring her total oestrogen dosage up to 80 µg a day.^{2,3} If intermenstrual bleeding still occurs alternative contraception should be recommended. The patient may be worried about the risk of side effects from the high oestrogen dose, and unfortunately she cannot be reassured, since this risk is not yet quantified²; we do not know whether the vascular side effects of the pill are due to the drug itself (in which case she would be safe) or to its metabolites (in which case her risk would be increased).—J O DRIFE, lecturer in obstetrics and gynaecology, Bristol.

¹ Anonymous. Drug interaction with oral contraceptive steroids. *Br Med J* 1980; 281:93-4.

² Back DJ, Breckenridge AM, Crawford FE, MacIver M, Orme MLE, Rowe PH.

Interindividual variation and drug interactions with hormonal steroid contraceptives. *Drugs* 1981;21:46-61.

³ Victor A, Lundberg PO, Johansson EDB. Induction of sex hormone binding globulin by phenytoin. *Br Med J* 1977;iii:934-5.

⁴ Anonymous. Teratogenic risks of antiepileptic drugs. *Br Med J* 1981;283:515-6.

Cerebral oedema is treated with dexamethasone or betamethasone, septic shock with methylprednisolone, acute asthma with hydrocortisone, intradermal injections are usually of triamcinolone, and most of the other steroid-responsive diseases are treated with prednisolone or prednisone. Is there any evidence that any one steroid is better than any other in treating any specific disease? Would it be possible to treat cerebral oedema with prednisolone?

Hydrocortisone possesses both glucocorticoid and mineralocorticoid activity. Anti-inflammatory properties are related to glucocorticoid activity, and modifications of the steroid structure can increase these relative to mineralocorticoid effects. The most potent in this respect are betamethasone and dexamethasone. When normal therapeutic maintenance doses are given for anti-inflammatory purposes, the choice of synthetic corticosteroid is not critical because the disturbance of electrolyte balance that they cause is usually small. Prednisolone is suitable as a standard drug and is cheaper than most newer compounds; prednisone is converted to prednisolone after it is absorbed and is less satisfactory. When high-dose treatment is necessary, however, particularly in patients in whom retention of water would be a disadvantage, betamethasone or dexamethasone should be chosen. For emergency treatment of septic shock or status asthmaticus an injectable preparation should be used; most drugs are available in a suitable form.¹ Cerebral oedema related to an intracranial neoplasm is reduced by corticosteroids leading to an improved neurological state²; betamethasone or dexamethasone is more suitable because of their minimal effect on water balance. The value of corticosteroids in head injury or stroke is doubtful.—ALAN RICHENS, professor of pharmacology, London.

¹ *British National Formulary*, 1981; Number 2:196-201.

² Klawans HL, Weiner WJ. *Textbook of clinical neuropharmacology*. New York: Raven Press, 1981:283-6.