Migraine: a headache specialist’s perspective

When I tell colleagues that I run a specialist headache clinic I am often met with a wry smile. I have always being perplexed by this curious reaction, as headache is one of the most common neurological complaints encountered in daily practice. Getting the management right can make a big difference in quality of life to many patients. This is because headache disorders represent a huge burden in terms of disability and lost economic productivity. Migraine may affect up to 1 in 6 of the population at some stage while potentially 4% of the population suffer from chronic daily headache. Analgesia overuse is a major problem and many people who would benefit from headache preventive treatments are not receiving them. In this article I am going to briefly cover some of the issues I encounter in managing migraine, one of the leading cause of neurology referrals.

Genetic and Environmental Influences

Patients often ask me why they have migraine. For the majority it is something they were born with. If you dig deeply enough there is often a history of a first-degree relative who also suffered from migraine and that can help with the diagnosis. There may also be a history of motion sickness or unexplained abdominal pain in childhood. Migraine has a genetic basis and it probably represents the end product of a complex interaction between a number of genes and the environment. Individual genes have being identified as causing hemiplegic migraine but this represents only a small proportion of the total number of people with migraine. The role that the environment plays in triggering migraine is poorly understood. As a general rule people who have migraine prefer routine. Change seems to be a trigger for attacks. A regular sleep pattern is important – too much sleep can be just as much of a trigger as too little. Hunger rather than exclusion of specific food groups in the diet per se seems to be more of a provocation. Hormonal fluctuations as part of the menstrual cycle or menopause may also be a significant determinant in females. Common sense behavior modification may therefore help with preventing some headaches. If a reliable trigger can be identified then it is best to avoid it if possible, but generally speaking I find that dramatic life-style changes are neither warranted nor particularly beneficial.

Migraine Aura

Migraine aura can often be a source of diagnostic confusion. Aura is experienced by a minority of migraineurs, perhaps only 15% of patients. It is important because though relatively uncommon, it may trigger unnecessary investigations for stroke. The history is crucial to avoid misdiagnosis. There is good evidence to suggest that aura corresponds to the experimental phenomenon of cortical spreading depression. Most commonly it manifests as a visual disturbance such as fortification spectra or a shimmering scotoma. It can also cause motor, sensory or speech symptoms depending on the area of cortex involved. Aura typically precedes the onset of the headache but this is not always the case. Isolated aura is well recognized and is perhaps more common in older males. Aura symptoms evolve slowly over minutes unlike the sudden instantaneous onset experienced during an ischaemic event. They may last for some time, but often no more than an hour, before they resolve. Once again this is often a gradual process. Patients often feel tired and “washed-out” following an isolated aura and this can be an additional clue in the history.

Treatment

The treatment of migraine can be divided into two arms: a) acute treatment to alleviate the immediate pain, b) prophylactic to reduce the severity and number of attacks.

Acute Treatment

Acute treatment may involve a combination of medications, depending on the severity and constellation of symptoms. The important thing is to treat the pain aggressively from its onset. Leaving treatment until the pain becomes intolerable is not a good strategy. It is my experience that this approach only allows the pain to become more entrenched and difficult to treat. Simple analgesics such as paracetamol, NSAIDs or codeine can all be used and may be adequate for many patients. Patients should also be offered an anti-emetic as gastric emptying is delayed during a migraine attack.

Triptans can be an invaluable acute treatment option for some, though not all, patients. Seven triptans are available. Each has its own particular characteristics in terms of speed of action, efficacy, side effect profile and risk of headache recurrence. They also come in a variety of formulations e.g. an intranasal sprays can be helpful if a rapid speed of onset is required or early onset of vomiting prevents enteric absorption. A given triptan should be used to treat at least three attacks before deciding if it is effective. Failure to respond to a triptan does not mean that the entire class will be ineffective. It is always worthwhile trying an alternative to see if it is better tolerated or more efficacious. The triptans can have unpleasant side effects including chest/neck tightness or excessive sedation. They are contraindicated if there is a history of coronary artery disease, coronary vasospasm or uncontrolled hypertension. Later generation triptans such as almotriptan may be better tolerated but they tend to be more expensive than sumatriptan. It is worth remembering that a triptan can be taken in combination with analgesics, especially NSAIDs. The combination of the two drugs can be synergistic. Some patients may be able to sense at the onset of a migraine whether or not it is going to be a severe attack. If so, I would suggest they take a triptan and a NSAID such as naproxen (provided it is not contra-indicated) together in an effort to terminate the attack.

A novel recent development in the acute management of migraine is the use of neurostimulator devices. Transcranial magnetic (TMS) and vagal nerve stimulation (VNS) are two methods of neuromodulation that are non-invasive and generally well tolerated. Their precise mechanism of action is not clear. TMS delivers magnetic pulses to the occipital cortex using a hand–held device while VNS delivers electrical pulses to the vagal nerve in the neck.

Analgesic and Triptan Overuse Headache

Overuse of acute treatments is a serious and common problem. Any analgesic – even paracetamol – will make migraine worse if used too frequently. Triptans are even more prone to causing over-use headache if taken excessively. Patients may suffer a “double hit” as overuse of acute treatments will not only make migraines worse, but may also block the action of prophylactic treatments. What however constitutes overuse? **I consider consumption of painkillers on more that 2 days out of each week, or triptans on more that 6 days per month is too much.** Patients will have to stop their acute treatments for at least 4-6 weeks for the effects of overuse to wear off. They may experience a rebound worsening of their headache in that time but there are ways of dealing with this. It is very important to ask specifically about this issue and sometimes a bit of probing is required. Patients may be reluctant to accept this and it often needs to be addressed gently but firmly. It must be remembered that some people have come to rely on their acute treatments. Being told to stop them – even temporarily – because they are making their headaches worse is anathema to them. It is important to stress that they will not improve until the overuse stops. It may help to point out that the very act of acute treatment withdrawal on its own may lead to a 50% reduction in headache days in 50% of patients.

Prophylactic treatments

I generally offer a prophylactic treatment to patients when they suffer four or more debilitating headache days per month. There are numerous treatment options but there are some general principals that must be observed. Firstly prophylactic treatments may not work quickly. A patient may need to be on an effective dose of a prophylactic agent for up to four months before deciding it is working or not. There are therefore no quick fixes. They are usually started at a low dose but then it must be increased until either:

1. the maximum dose is reached
2. side effects prevent further dose increases
3. the headaches improve

Dose increases should be made every two to four weeks depending on tolerability, but one has to be flexible. Some patients may need to go more slowly but there is little point in maintaining a patient on a low dose if their headaches are not improving. If they get to an effective dose and are maintained on this for 3-4 months without improvement (and they are not overusing acute treatments) then they should be moved on to another treatment. With the exception of the CGRP-antagonists currently in development, no migraine prophylactic treatment was specifically designed for this purpose. Serendipity has played a part and this sometimes needs to be explained to patients. Some may be concerned to learn that they are receiving an anti-depressant or anti-epileptic medication and explaining this early on avoids confusion. Treatments range from Vitamin B2 to botulinum toxin but there is no “wonder-drug”. It may entail a process of trial and error before a preventive is found for an individual that is both tolerable and effective.

Conclusion

Migraine is a very common, debilitating but eminently treatable condition. Acute treatment often depends on using a combination of drugs to bring the pain and other symptoms under control as quickly as possible. Acute treatments may be a doubled edged sword however, and if used too frequently will ultimately lead to a worsening of headache. In such cases prophylactic treatments should be prescribed. Remember that there are a wide variety of prophylactic options but they may not work quickly. Perseverance is required and the dose should be increased if there is no response. It may involve a process of trial and error to find a drug that works and is tolerable. If it becomes apparent that a drug is ineffective, move on to another. It is worth the effort as getting it right can make a big difference to patient’s quality of life.

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