

MIGRAINE: DIAGNOSIS AND MANAGEMENT

Giles Elrington

ii10

J Neurol Neurosurg Psychiatry 2002;**72**(Suppl II):ii10-ii15

One in 10 people have migraine. The patient's history is the essential diagnostic tool. Treatment options include acute rescue, lifestyle strategies, alternative remedies, and prophylactic drugs. Most patients are managed in primary care; many never consult a doctor. The triptans have improved acute treatment, and renewed scientific interest in migraine. Overuse of acute rescue medication can lead to chronic daily headache.

PATHOPHYSIOLOGY

Spontaneous overactivity and abnormal amplification in pain and other, predominantly sensory, pathways in the brainstem, leads to migraine. Current opinion favours a primarily neural cause, involving feedback loops through innervation of cranial arteries in the trigeminovascular system.¹ A relative deficiency of 5-hydroxytryptamine (5-HT) may be near the root cause, and is linked to the action of most drug treatments. Ongoing research is studying the relevance of calcium channel abnormalities, and peptides such as calcitonin gene related peptide, which may be closer than 5-HT to the underlying cause, offering hope for improved treatment in the future. Migraine is usually polygenic. Uncommon migraine variants, such as familial hemiplegic migraine and CADASIL, are single gene disorders. These are neurodegenerative, not primarily headache conditions.

DIFFERENTIAL DIAGNOSIS

Migraine is typically manifest by episodic disabling headache, though it is more than just head pain. Differential diagnosis is from tension type headache (TTH), with which migraine is co-morbid (this differential is also discussed from the other perspective elsewhere in this supplement). Migraine lasts hours or days, and is absent more often than it is present; the average attack frequency is once a month. TTH is often chronic and it is present more often than it is absent. Migraine should be distinguished from cluster headache, which is relatively rare and causes recurrent unilateral headache with autonomic dysfunction. The third, common though often challenging differential diagnosis is medication overuse headache (MOH). This typically complicates migraine which is then transformed into a chronic daily headache similar to chronic TTH often with some migrainous features.

DIAGNOSIS

The International Headache Society criteria² are very helpful in the diagnosis of migraine. An abbreviated version is shown in table 1. These criteria can be too restrictive and therefore may be interpreted flexibly by experienced clinicians. There are two main types of migraine: migraine without aura (MO), and migraine with aura (MA). Many people have both; MO is at least three times as common as MA. Note that family history, trigger factors, and treatment response have no additional diagnostic value.

Migraine without aura

Formerly called common migraine, the diagnosis of MO is suggested by a history of episodic disabling headache lasting between a few hours and a few days, accompanied by gastrointestinal symptoms or by heightened special senses. The International Headache Society criteria may be fulfilled when pain is mild or generalised: it does not have to be severe or unilateral. It is unusual to be able to distract oneself from MO with exercise or hard work, in contrast to TTH. The frequency and periodicity of migraine is important: migraine-like headache more than twice every week is unlikely to be MO alone, but it may be MO complicated by MOH and/or TTH. This is common in patients referred with "intractable migraine" or "status migrainosus".

Migraine with aura

In MA, formerly called focal or classical migraine, the aura evolves over time, usually many minutes; one aspect of the aura improves while another is deteriorating. Visual aura usually leads to easy diagnosis. Auras affecting sensation, movement, cognition, vestibular function, or consciousness may be difficult to distinguish from thromboembolism, or from epilepsy (especially occipital seizures). People presenting with recent onset MA often give a longer history of MO, mistakenly

Correspondence to:
Dr Giles Elrington, 119 Harley
St, London W1G 6AU;
elrington@aol.com

Table 1 Abbreviated International Headache Society criteria for the common primary headaches²

Migraine without aura	
▶	Headache lasting 4 hours to 3 days
▶	Nausea/vomiting and/or light and noise sensitivity
▶	Two of the following:
	Unilateral pain
	Moderate or severe intensity pain
	Aggravation by simple physical activity
	Pulsating pain
Migraine with aura	
▶	At least 3 of the following:
	Reversible focal brainstem or cortical dysfunction
	Aura develops over >4 minutes, or 2 auras in succession
	Each aura <60 minutes
	Headache <60 minutes following aura
Episodic tension-type headache	
▶	Duration 30 minutes to 7 days
▶	At least 2 of the following:
	Mild or moderate intensity pain
	Bilateral pain
	No aggravation by simple physical activity
	Pressing or tight (non-pulsating) pain
▶	No nausea/vomiting; may have light or noise sensitivity (not both)
Chronic tension-type headache	
▶	>15 days pain per month, for >6 months
▶	At least 2 of the following:
	Mild or moderate intensity pain
	Bilateral pain
	No aggravation by simple physical activity
	Pressing or tight (non-pulsating) pain
▶	No vomiting; one only of nausea, light sensitivity, noise sensitivity

All of the above diagnostic criteria require the exclusion of secondary (that is, pathologically verifiable) causes for pain. This is normally achieved on clinical grounds.

diagnosed as “bilious attacks”, “sinusitis”, or “normal headaches”. Visual symptoms are positive (seeing things which are not there), homonymous and binocular, though some patients insist that visual aura is monocular, raising the possibility of retinal origin. Aura typically precedes migraine headache, though can occur at any time in relation to pain. Aura is not always contralateral to pain. Migraine aura without headache is common, especially in middle age: a flurry of MA episodes without headache often triggers referral fearing transient ischaemic attacks (TIA). Thromboembolism may be accompanied by headache (especially with vertebral or carotid dissection, in which pain usually precedes impairment), but is distinguished from MA by abrupt, non-evolving associated impairment, confined to a single vascular territory. MA is much more common than TIA at all ages: around 40 years of age, MA is about 2500 times more prevalent than TIA, and is still about 15 times more common at 70.

HISTORY

The patient’s spontaneous account is the most important part of the history. This rarely takes more than a couple of minutes. Establish the duration, frequency and periods of freedom from pain: a diary can be very helpful. Ask carefully about other symptoms: many who deny nausea will readily acknowledge queasiness. Light and noise sensitivity may not be described by a patient who retreats to the dark and quiet. Enquire what the patient does during an attack. If they can carry on as normal then migraine is unlikely. Can they identify more than one type of attack? People with migraine and TTH will usually volunteer

Table 2 Suggested brief central nervous system examination for routine, outpatient headache practice

▶	Romberg’s sign
▶	Tandem gait
▶	Drift of outstretched hands
▶	Finger–nose test
▶	Finger dexterity
▶	Binocular visual fields, to confrontation
▶	Eye movements
▶	Facial weakness
▶	Pupillary responses and Horner’s syndrome
▶	Tendon reflexes and plantar responses
▶	Funduscopy

the migraine history, but may say that their daily muzzy head is the more debilitating symptom.

Document previous and current treatment strategies: do not be stumped by “I’ve tried everything and nothing works”. Note drugs, doses, and duration: many take ineffective or weak agents, at insufficient dose, at inappropriate times. Overuse of acute medication is often the reason why prophylaxis is ineffective. This is revealed only by cunning enquiry. Those on repeat daily doses of acute rescue drugs often dissemble: “I only use it when the pain is bad” or “I keep it to a minimum”. Efficacy is characteristically reported as “it takes the edge off it”. Establish frequency and quantity of prescription or purchase (for example, “200 Co-proxamol a month”; “12 extra-strong painkillers twice a week”). Stash lists may be revealing: those who are biochemically or emotionally dependent store analgesics in their handbag or briefcase, the bedroom, bathroom, kitchen, office or car.

EXAMINATION

The main goal of examination is to consider structural brain disease. It also provides an opportunity to screen for co-morbid disease such as hypertension and depression (neither of which commonly cause headaches), and to reassure the patient, their family, and the referring doctor.

The “full neurological examination” is not possible in the time available in primary or routine secondary care, nor is it often necessary. Table 2 lists the author’s personal screen, which has been criticised both for its brevity and its length. Papilloedema and ataxia are probably the two most important physical signs.

INVESTIGATION

Almost everyone with migraine needs no investigation. The goal of investigating is to exclude other causes of migraine-like symptoms, not to confirm migraine—which tests can never do.

Abbreviations

5-HT:	5-hydroxytryptamine
MA:	migraine with aura
MO:	migraine without aura
MOH:	medication overuse headache
NNT:	number needed to treat
NSAIDs:	non-steroidal anti-inflammatory drugs
OCP:	oestrogen-containing contraceptive pill
TIA:	transient ischaemic attack
TTH:	tension type headache

Table 3 Brain imaging strategies for headache patients

First, worst, thunderclap headache	Mandatory imaging: CT (emergency)
Exertional, cough or valsalva headache	MRI
Headache + signs suggesting brain lesion	MRI or CT
Headache + known malignancy	MRI or CT with contrast
	Optional imaging:
New headache in older person	MRI or CT
Headache, not MO, MA, TTH, MOH or cluster	MRI or CT

CT, computed tomography; MA/MO, migraine with/without aura; MOH, medication overuse headache; MRI, magnetic resonance imaging; TTH, tension type headache.

Table 4 Triptan costs^{6,7}

		Cost per tablet	NNT*	Cost per pain-free patient
Sumatriptan	50 mg	£4.70	4.0	£15.92
	100 mg	£8.00	4.3	£34.07
Naratriptan	2.5 mg	£4.00	9.2	£36.75
Zolmitriptan	2.5 mg	£4.00	4.9	£19.49
	5 mg	£8.00	3.3	£26.25
Rizatriptan	10 mg	£4.46	3.1	£13.85
Almotriptan	12.5 mg	£3.25	4.6	£15.00

*Number needed to treat to achieve one pain-free patient 2 hours post-dose.

Imaging

A "brain scan" is often requested by the patient or the non-specialist. Imaging may reassure and, conversely, may generate concern. Arguably, every painful head should be imaged. This would result in an impossible workload: 18% of women and 6% of men have migraine; 3% have chronic daily headache. If imaged once with normal results, when should imaging be repeated? The author's experience is that about one third of UK headache patients in secondary or tertiary care are imaged, two thirds of which occurs before neurological referral. In practice it can be difficult and unhelpful to resist demand for imaging in a patient centred health care system. Magnetic resonance imaging is always preferable to computed tomography because of better resolution and lack of exposure to ionising radiation, except for emergency presentation with possible cerebral haemorrhage. Table 3 provides some suggested imaging strategies.

Other tests

Older people with new headache should have their erythrocyte sedimentation rate assessed to address the possibility of giant cell (temporal) arteritis, which is uncommon in headache practice. A chest x ray should be considered in smokers, or in those who may have metastatic cancer.

TREATMENT

Rest is important for almost all migraine attacks; sleep, if possible, can abort migraine. Some people with infrequent or relatively mild attacks may prefer simply to rest while the migraine attack settles naturally, rather than take medication.

It is important to differentiate between acute rescue, and prevention using lifestyle or drugs. Ask your patient which of these they would like to discuss first. Guidelines have been published in the UK³ and the USA.⁴

Acute rescue

Analgesics and antiemetics

Analgesics and antiemetics are effective for many migraines. Impaired absorption from gastric stasis during migraine is helped by the addition of a prokinetic antiemetic (even if nausea or vomiting are not prominent), and by the use of a large dose of aspirin, 900–1200 mg, dissolved before ingestion, and taken as soon as a migraine appears to commence. Some patients prefer a non-steroidal anti-inflammatory drug (NSAID), or paracetamol 1000 mg. Fixed drug combinations of aspirin or paracetamol, with an antiemetic, are expensive. Domperidone has fewer side effects than other antiemetics, and is available over the counter. Rectal antiemetics or NSAIDs are worth considering.

Opiates should not be used because they worsen gastrointestinal symptoms, and have abuse potential especially in the genesis of MOH.

Triptans

Five triptans are currently licensed* in the UK. They are expensive (table 4) and are only slightly more effective than simple analgesia with an antiemetic,⁵ on the number needed to treat (NNT) basis. These data conceal substantial inter- and intra-patient variation. Some people respond very well to triptans, and poorly to other agents; response varies from attack to attack. Triptans should be used only to treat migraine pain of at least moderate severity; they should not be used during prodrome, or during aura in anticipation of headache.

Three meta-analyses of triptans allow comparison of efficacy⁶⁻⁹ and tolerability.⁹ Rizatriptan is consistently the most effective, though almotriptan has a good balance between tolerability and efficacy. Sumatriptan (Imigran) was the first to market in the UK and is familiar to most doctors. Sumatriptan is the only injectable triptan, offering the most rapid, effective, and expensive treatment; the nasal spray is relatively rarely used. Naratriptan (Naramig) is the least potent, but is well tolerated. Zolmitriptan (Zomig) has potentially the lowest NNT (at the extreme of the 95% confidence interval), using its top dose of 5 mg.⁵ Rizatriptan (Maxalt) has the lowest mean NNT, and the lowest cost per effectively treated migraine.^{6,7} Almotriptan (Almogran) costs less than the other triptans, with good sustained freedom from pain, and tolerability equivalent to naratriptan.⁹

Mouth dispersible preparations (Maxalt melt and Zomig rapimelt) have no proven advantage over tablets; they are not absorbed through the buccal mucosa. This formulation may be intuitively patient friendly, without cost disadvantage.

The potential cardiac toxicity of triptans has caused concern, which has rarely been realised in clinical practice. Triptans, however, should not be used in those with, or at risk of, cardiac ischaemia. The problems with triptans are:

- ▶ cost
- ▶ incomplete delayed benefit
- ▶ recurrence of migraine
- ▶ tendency for a minority, perhaps as many as 10%, to overuse triptans.

There is certainly no place for near daily triptan use, though such users are not uncommon in secondary and tertiary

*A sixth triptan, eletriptan, has recently been licensed. Comparative data are available,^{7,9} and do not cast doubt upon the recommendations for earlier triptans given this article.

Table 5 Prophylactic drug treatments for migraine

	Start dose (mg)	Maximum dose (mg)	Side effects, comments
Amitriptyline	5–10	200	Sedation and dry mouth Consider other tricyclics; SSRIs
Propranolol	20	320	Cold limbs, nightmares Atenolol may be better tolerated
Pizotifen	0.5	3	Weight gain, sedation, low efficacy
Valproate	500	1500	Weight gain, teratogenicity Consider gabapentin, topiramate
Methysergide	1	8	Limb pain, visceral fibrosis 6 months treatment, 1 month break

Many of these drugs are unlicensed in this indication.
SSRIs, selective serotonin reuptake inhibitors.

headache practice. Such patients often have an established history of analgesic or ergot overuse, and triptans should be used with caution in such cases. Management is the same as for any other MOH: cure follows drug withdrawal, and is not facilitated by further prescriptions.

Ergots

Ergot alkaloids may still have an occasional place in the acute management of migraine. In headache clinics, ergot users usually have ergot dependency—yet another type of MOH.

Other acute rescue treatments

Unlicensed options for refractory cases include high dose oxygen (100% if possible), parenteral steroids (for example, dexamethasone 4 mg), and parenteral phenothiazines (for example, chlorpromazine 5–50 mg).

Recurrence

Recurrence after initial improvement affects a third of migraineurs. This is a complex and poorly understood phenomenon: at the simplest level, an ineffective treatment has zero recurrence. Recurrence risk with different triptans does not simply reflect drug half life. It is arguable that triptans access their site of action only when there is migraine pain—so they terminate their own action. Some authorities try to reduce recurrence with the combination of an NSAID with triptan treatment, though this is not evidence based.

Step care versus stratified care

Step care means starting with simple analgesia, usually with an antiemetic, escalating in one or more steps toward triptans. With stratified care, low impact migraine is treated with simple analgesia and an anti-emetic, but high impact migraine is treated first with a triptan. Stratified care results in significantly better clinical outcomes than step care,¹⁰ but at a higher price. This comparison assumes that the two strategies are equivalent. It is arguable that analgesia/antiemetic is appropriate to use very early in a migraine attack, perhaps before pain is significant—for example, during aura or prodrome, a time when triptans are known to be ineffective.

PREVENTION OF MIGRAINE

Lifestyle management

Management of lifestyle can appear to be very helpful, though evidence is largely anecdotal. Regularity of biorhythms is the key. The avoidance of relative hypoglycaemia, with a regular,



fibre containing diet is probably the most helpful strategy. Dietary exclusion is rarely helpful, and should be abandoned if ineffective. Change in sleeping times at weekends and irregular shift work may usefully be avoided, as is the abrupt let-down from stress. Trigger factors often summate—for example, at times of international travel.

Alternative therapies

These are often acceptable to patients though are rarely evidence based. Large doses of vitamin B2, and magnesium, may be effective with several months' latency to benefit. Manipulative treatments seem to be helpful for soft tissue pain or tenderness. Homeopathy is ineffective.¹¹ Acupuncture probably has only an acute analgesic effect.

Prescribed drugs

Daily drug treatment to prevent migraine should be considered after acute treatment has been optimised, medication overuse abolished, lifestyle modification tried, and a migraine diary recorded for a month or three. Most agents offer partial benefit only, which may take 1–5 months to achieve; those most commonly used are listed in table 5. Patients must be carefully advised that the occurrence of migraine after starting prophylaxis does not mean treatment failure. Co-morbid disease may suggest initial drug choice. It is unusual to offer prophylaxis for less than two attacks a month. Treatment should be titrated first for tolerability, then for efficacy. Cycle through single drug regimens to find the best agent, with ongoing diary monitoring; occasionally, combine partially successful drugs. After six months of effective treatment, phased withdrawal should be considered.

Hormones and migraine

Migraine is more common in women, the sex difference beginning at puberty. Menstruation is a migraine trigger in 10% of women with migraine. This is often overestimated by the patient: true menstrual migraine can be diagnosed only after examining a few months of the headache and menstrual diary. The oestrogen-containing contraceptive pill (OCP) may lead to an improvement, but this ameliorating effect is then lost during the pill-free week. Tricycling the contraceptive pill, and using transdermal oestrogen, can be helpful.

Migraine, especially MA, is possibly an independent risk factor for stroke, though confidence intervals of case-control studies are wide, making risk assessment difficult. For example, one study quotes an odds ratio of 34.4 for stroke among migraineurs who smoke and use the OCP, but the 95% confidence interval is 3.3 to 361! This leads many authorities to

question the wisdom of OCPs for women with migraine. The risk is probably very low in MO. MA is a relative contraindication to the OCP. Intrauterine devices and progesterone only contraceptives affect neither migraine nor stroke risk, so are preferable to OCPs for women with any form of migraine.

Migraine typically improves during the second and third trimesters of pregnancy, though can be troublesome in the puerperium.

The climate and menopause are associated with worsening of migraine, as often as with improvement. Transdermal, not oral, hormone replacement therapy is often helpful, though high doses may trigger MA.

MIGRAINE IN CHILDREN

The prevalence of migraine in childhood rises with age; under 12 years, it is more common in boys. Compared with adult migraine, attacks are shorter (as short as two hours), pain is seldom unilateral, and aura is less common. Children do not usually describe light or noise sensitivity though this is sometimes inferred from their behaviour; they can rarely describe the pain as throbbing or pulsating. Gastrointestinal symptoms are more prominent than in adults. Head pain is absent in the migraine variants—cyclical vomiting and abdominal migraine in which the attack is identical to MO except that pain is in the abdomen, not the head.

Management must address the concerns of the child, parents, and sometimes teachers as well—which may mean three different agendas. The child's estimate of attack frequency may be different from the parent's, and neither may tally with frequency of days off school. A diary is invaluable in such cases.

Acute rescue medication may be unhelpful for short attacks; otherwise, ibuprofen is preferable to paracetamol (aspirin is contraindicated in children under 12 years). The child should be allowed to rest in a quiet place, and to resume normal activities as soon as this is comfortable. It is rarely necessary for the child to be sent home from school, though teachers may disagree. Zolmitriptan may be used from the age of 12 years. Sumatriptan nasal spray is safe and effective, though unlicensed in children.

Preventative strategies should focus on lifestyle and diet, especially ensuring regular adequate sleep, and three meals containing fibre a day (constipation may cause headache in children). Dietary exclusion is less disappointing in children than adults, though this must be carefully negotiated and foods reintroduced if migraine is unchanged.

Drug prophylaxis is occasionally necessary. Pizotifen seems to be better tolerated and more effective in children than in adults. β Blockers are typically the drugs of second choice. In refractory cases, the same drugs that are used in adults may be considered.

POST-TRAUMATIC MIGRAINE

All forms of primary headache, especially migraine, may appear or become worse after head injury, or after more controlled forms of trauma, such as neurosurgery. MOH is common in these patients, who are typically offered combination analgesics as inpatients, which are continued on discharge. Management is identical to that for patients in whom there is no history of trauma.

In medicolegal practice, claimants state that migraine has been triggered by minor head injury and deny any migraine before injury. This history must be corroborated by review of primary care notes, and of employment records as time may be

missed from work because of migraine that does not present to the general practitioner. Where the records cast no doubt upon the patient's history, post hoc propter hoc is an argument whose weakness increases with time. It seems reasonable to attribute up to a year of excess migraine to mild head injury; beyond that time, a causal link is less likely.

INTRACTABLE MIGRAINE

Some patients keep coming back to clinic saying that nothing has changed, and that their migraine is just as bad as ever. Retake the headache history; compare current frequency and severity with that recorded in the notes. Sometimes, the patient has forgotten just how bad things were, and has mistaken incomplete cure for unchanged migraine. An ongoing diary can be very helpful in assessing change.

The most common error is failure to diagnose MOH: probe carefully for this history, as no amount of new prescriptions will help these challenging patients. Has prophylaxis been taken at maximum tolerable doses for a good few months, in the absence of MOH? Is there co-morbid depression requiring specific management independent of migraine? Is the headache diagnosis correct? It is possible to mistake for migraine the trigeminal autonomic cephalgias, such as cluster headache or chronic paroxysmal hemicrania.

Review the fine detail of acute rescue strategies:

- ▶ Are triptans being taken incorrectly during aura or prodrome, or shortly before vomiting?
- ▶ Are simple analgesics such as aspirin being used too late, without an antiemetic, or as whole tablets rather than being dissolved first? Some authorities recommend dissolving aspirin in a sweet fizzy drink.
- ▶ Have at least three attacks been treated with each of the triptans, one by one, using the full range of doses and formulations?
- ▶ Have a range of NSAID tablets suppositories and injections been tried? An ergot preparation may be worth trying.
- ▶ Is the patient resting while waiting for acute treatment to work, or expecting benefit from a miracle treatment while they try to carry on as normal?
- ▶ A combination of aspirin or NSAID, antiemetic, triptan, and hypnotic can be tried, though triptans should not be combined with each other.

A second opinion can be very helpful, if only to reassure the patient that nothing important has been overlooked.

Patients who claim dissatisfaction with treatment may change their mind when offered medication whose threat is perceived as greater than that of migraine: "These tablets (methysergide) can work well, but may be taken for six months only, because they occasionally cause scarring around the vital organs"; "Thank you doctor, I'll stay as I am, but can we think again at my next appointment?" This helps the patient feel in control of treatment options, and therefore of their migraine. The move from an external to an internal locus of control may be critical to quality of life, even if experience of pain is unchanged.

It is reasonable to allow patients to choose alternative or complementary treatments even if there is no evidence to support their choice; and, if these appear helpful, the neurologist should gracefully acknowledge this.

A tiny minority of migraineurs are truly resistant to treatment. The same consultation attitudes useful for other incurable diseases are indicated. Listen to and record symptoms, and accept how hard they are for the patient to bear. It is amazing how grateful people can be for the simple act of interested compassion without cure.

ACKNOWLEDGEMENTS

I am grateful to Dr David Symon for his helpful advice on migraine in children, and to Dr Anne MacGregor for permission to reproduce her cartoon.

Possible conflict of interest: The author has accepted hospitality from, and acted on advisory boards for, all the triptan manufacturers. He is treasurer of the British Association for the Study of Headache and a member of the International Headache Society.

REFERENCES

- 1 **Goadsby PJ.** Migraine, aura, and cortical spreading depression: why are we still talking about it? *Ann Neurol* 2001;**49**:4–6.
- 2 **International Headache Society.** Headache classification committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. *Cephalalgia* 1988;**8**(suppl 7).

- 3 **Steiner TJ, MacGregor EA, Davies PTG.** Guidelines for all doctors in the management of migraine and tension-type headache. Available at: <http://www.bash.org.uk>. Accessed 8 September 2001.
- 4 **Silberstein SD.** Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review). *Neurology* 2000;**55**:754–62.
- 5 **Bandolier 59.** Making sense of migraine. 1999;**6**:1–4. URL:www.jr2.ox.ac.uk/bandolier/band59/659-2.html.
- 6 **Belsey J.** The clinical and financial impact of oral triptans in the management of migraine in the UK: a systematic review. *J Med Econ* 2000;**3**:35–47.
- 7 **Belsey J.** The clinical and financial impact of oral triptans—an updated meta-analysis. *J Med Econ* (in press).
- 8 **Adelman JJ, Lipton RB, Ferrari MD, et al.** Comparison of rizatriptan and other triptans on stringent measures of efficacy. *Neurology* 2001;**57**:1377–83.
- 9 **Ferrari MD, Roon KI, Lipton RB, et al.** Oral triptans (serotonin 5-HT_{1B/1D} agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet* 2001;**358**:1668–75.
- 10 **Lipton RB, Stewart WF, Stone AM, et al.** Stratified care vs step care strategies for migraine. *JAMA* 2000;**284**:2599–605.
- 11 **Whitmarsh TE, Coleston-Shields DM, Steiner TJ.** Double-blind randomized placebo-controlled study of homoeopathic prophylaxis of migraine. *Cephalalgia* 1997;**17**:600–4.

IMAGES IN NEUROLOGY

Colloid cyst of the IIIrd ventricle

Colloid cysts are relatively unusual (1.5% neurosurgically treated tumours) benign intracranial masses located in the rostral aspect of the IIIrd ventricle. As they grow they obstruct the foramen of Munro producing hydrocephalus of the lateral ventricles.¹ This can be acute and catastrophic because of the pedunculated nature of the tumour resulting in brain herniation and death.² Both computed tomography (CT) and magnetic resonance imaging (MRI) will usually show the lesion and its effects on the ventricular system, but the multiplanar capability and higher contrast resolution of MRI will usually define its relations better.

Unless the tumour is an incidental finding³ patients usually present with intermittent headache and less commonly with nausea, short term memory loss, gait disturbance, blurred vision or coma. Non-specific neurological symptoms and signs are common but may be suggestive of raised intracranial pressure. The cause and origin of the cysts are still debated. The cyst size ranges from 2–50 mm and they contain colloidal material including cholesterol fats that are largely responsible for their appearances on MRI. Thin (≤ 5 mm) axial sections on MRI or CT will show the lesion hanging from the roof of the IIIrd ventricle, while sagittal and/or coronal sections will show its relation with the foramen of Munro⁴ (fig 1). On CT the mass is hyper- or isodense with grey matter while on MR T1 weighted imaging it is usually hyperintense, reflecting the colloidal matrix and fatty content. T2 weighted sections show it as hyperintense or isointense with grey matter while FLAIR (fluid attenuated inversion recovery) sequences will show it as hyperintense against a background of hypointense cerebrospinal fluid. Surgical treatment used to be carried out via a transcalsal approach in an attempt to avoid damage to the fornix and resultant memory loss, but more recently stereoscopic and endoscopic approaches to the IIIrd ventricle have proved superior in many cases^{4,5} with less neurological and neuropsychological sequelae.

D M Hadley

gota27@udcf.gla.ac.uk

References

- 1 **Jeffrey RL, Besser M.** Colloid cyst of the third ventricle: a clinical review of 39 cases. *J Clin Neurosci* 2001;**8**:328–31.
- 2 **Stoodley MA, Nguyen TP, Robbins P.** Familial fatal and near-fatal third ventricle colloid cysts. *Aust NZ J Surg* 1999;**10**:733–6.
- 3 **Pollock BE, Huston J 3rd.** Natural history of asymptomatic colloid cysts of the third ventricle. *J Neurosurg* 1999;**91**:364–9.
- 4 **El Khoury C, Brugieres P, Decq P, et al.** Colloid cysts of the third ventricle: are MR imaging patterns predictive of difficulty with percutaneous treatment? *Am J Neuroradiol* 2000;**21**:489–92.
- 5 **Decq P, Le Guerinel C, Brugieres P, et al.** Endoscopic management of colloid cysts. *Neurosurgery* 1998;**42**:1288–94.

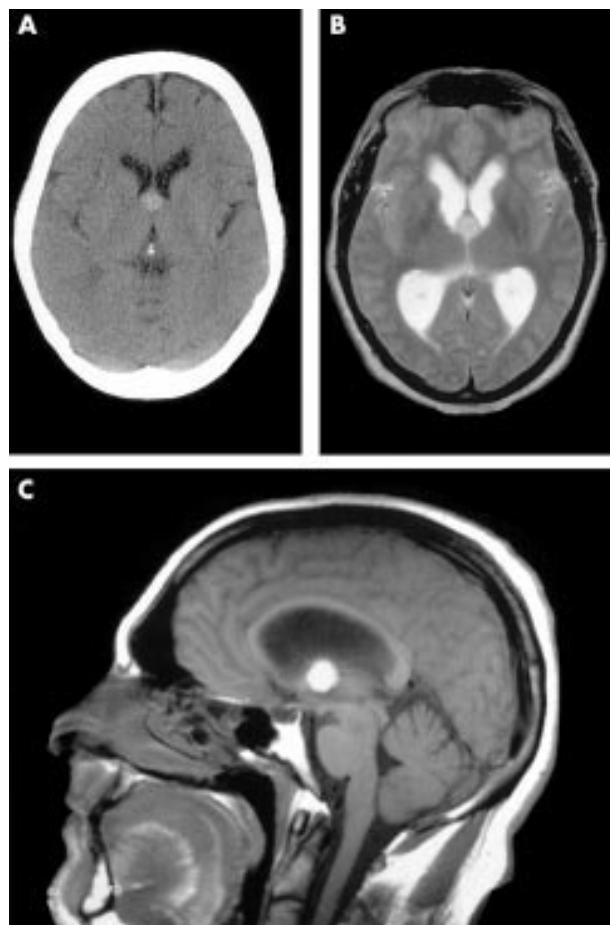


Figure 1 (A) Axial non-contrast CT showing a 8 mm diameter round high attenuation tumour, a colloid cyst in the IIIrd ventricle at the level of the foramen of Munro—as yet no hydrocephalus. (B) T2 weighted axial and (C) T1 weighted sagittal sections showing a 10 mm diameter tumour hanging from the roof of the IIIrd ventricle obstructing the foramen of Munro, causing hydrocephalus of the lateral ventricles with periventricular oedema.