Headache, Migraine

Alan David Kaye | Diana Mekler | Adam Shomstein

Risk

- Incidence in USA: >28 million; maximum prevalence is in 25–55 y of age.
- Can start as early as 1 y of age; 10–20% of instances occur in children by 20 y of age, with males and females being equally affected.
- More frequent in women after age 11 y; ratio is approx 3:1 for female: male; prevalence declines after age 40 y.
- Familial aggregation: CACNA1A (P/Q voltage-gated calcium channel), ATP1A2 (Na^+-K^+ ATPase), and SCN1A (Na_v 1.1 voltage-gated sodium channel) genes are implicated in genetic predisposition for variations of familial hemiplegic migraine.
- Can be associated with sinusitis; AVM; stroke; patent foramen ovale; epilepsy; ischemic myocardial infarction; depression; anxiety disorder; sensitivity to foods rich in tyramine, phenylethylamine, or octopamine (chocolate, wine, dairy products); and electroencephalographic abnormality.
- Socioeconomic status: Migraines are inversely related to household income and education.

Perioperative Risks

- Increased incidence of Htn, stroke, CAD
- Gastric stasis
- Drug toxicity and side effects

Worry About

- Toxic and side effects of antimigrainous preparations, adverse interaction with anesthetic drugs
- Associated intracranial disorders
- Increased aggregation of platelets with increased risk of stroke and CAD

Overview

- Recurrent, frequently unilateral, throbbing head pain with strong family Hx
- Often associated with increased sensitivity to touch, N/V, phonophobia, and/or photophobia.
- May be preceded by a visual, sensory, or motor aura; headache and aura may present independently.
- Dx is Hx dependent in the absence of secondary causes.
- Migrainous infarction with permanent neurologic damage is rare.

Etiology

- Central or peripheral mechanisms can be incited by internal or external stimuli.
- Lowering Mg^{2+} levels increase the affinity and release of serotonin at cerebrovascular and neuronal sites as well as NO production and activation of NMDA receptors.
- Can be precipitated by trigger factors.
- Cerebral and extracerebral arteries are the most likely sources of pain.
- Pain results from exaggerated pulsations in association with trigeminal release of sP, CGRP, and VIP and sensitization of nociceptors around blood vessels.

Usual Treatment

- There is no permanent cure for migraines.
- Elimination of trigger factors when possible can reduce incidence, including regular sleep, meals, and hydration, along with decrease in stress.
- Abortive therapy includes NSAIDs, barbiturates, ergotamines, triptans, phenothiazines, dihydroergotamine, sphenopalatine ganglion block, nonopioid and opioid analgesics, single-pulse transcranial magnetic stimulation, and vagal nerve stimulation.
- Prophylactic therapy:
  - Effective: β-blocking agents (metoprolol, propranolol, timolol), TCA (amitriptyline), antiepileptic drugs (AED; topiramate, divalproex sodium), and serotonin agonists.
  - Ineffective: ACE inhibitors (lisinopril, candesartan), AED (gabapentin), β-blocking agents (atenolol, nadolol), antidepressants (fluoxetine, venlafaxine), serotonin agonists (naratriptan, zolmitriptan; short-term prevention in menstrual migraine), histamine, cyproheptadine, MIG-99 (feverfew), and vitamins (riboflavin, CO-Q10, Mg^{2+}).
  - Possibly effective: α-agonists (clonidine, guanfacine), Ca^{2+}-channel blockers (verapamil, nicardipine, nifedipine, nimodipine), AED (carbamazepine). Conflicting evidence exists for the efficacy of MAOIs. Botulinum toxin is probably not effective. CGRP antagonist and antibodies are in the experimental stages.
- Behavioral treatment involves biofeedback, self-hypnosis, relief by dark surroundings, and sleep.

Assessment Points (Mainly Side Effects and Toxicity of Antimigrainous Therapy)

System	Effect	Assessment by Hx	PE	Test
CV	Ergotamine, sumatriptan: Worsening of Htn, ischemic heart disease, PVD, serotonin syndrome	Symptoms of angina and peripheral vascular insufficiency		ECG Stress ECG
	β-adrenergic receptor blocking agents and Ca^{2+}-channel blockers: Excessive depression of myocardial function	Symptoms of CHF	S_3 Rales Decreased heart sounds	CXR CXR, ECHO
	Methysergide (no longer available): Pericardial fibrosis, cardiac valvular fibrosis	Syncope		ECG
	TCAs and Ca^{2+}-channel blockers: Cardiac conduction abnormalities		Q-T prolongation	
RESP	β-blockers: Worsening of COPD	Dyspnea	Expiratory wheezing	CXR ABGs
	Methysergide (no longer available): Pleuropulmonary fibrosis	Dyspnea	Rapid shallow breathing	PFTs
GI	Gastroparesis	Early satiety		
CNS	Intracranial disorders TCAs Anticonvulsants MAOIs: Anticholinergic and CNS stimulation	Tachycardia, dry mouth Blurred vision, urinary Somnolence, diplopia, ataxia, cognitive impairment Retention, delayed gastric emptying	Focal deficit	NeuroimagingECG

Key References: Weatherall MW: The diagnosis and treatment of chronic migraine, *Ther Adv Chronic Dis* 6(3):115–123, 2015; Chatterjee S, Rudra A, Sengupta S: Current concepts in the management of postoperative nausea and vomiting, *Anesthesiol Res Pract* 2011:748031, 2011.

Perioperative Implications

Preoperative Preparation

- Detailed pharmacotherapy Hx
- D/C MAOIs 14–21 d in advance if possible (see Monoamine Oxidase Inhibitors)
- Gastroparesis: Metoclopramide (10 mg/70 kg pt)

Monitoring

- Routine, unless signs of ischemic heart disease

Airway

- None

Preinduction/Induction

- Pts receiving β-blockers and Ca^{2+}-channel blockers may develop reduced CO and hypotension.

Maintenance

- Exaggerated response to indirect-acting vasopressors may occur with pts on ergotamine, sumatriptan, TCAs, and MAOIs.
- Hyperpyrexic coma reported after administration of narcotic to pts receiving MAOIs.

Extubation

- Increased risk of CNS stimulation with sumatriptan, ergotamine, TCAs, and MAOIs

Postoperative Period

- Pain management may be critical.
- Avoid withdrawal syndromes.
- Increased risk of postop N/V.

Anticipated Problems/Concerns

- Possible adverse interactions of anesthetic drugs and antimigrainous preparations
- No unique hazards of anesthesia administered to pts with migraines