Migrainous infarction

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Introduction

This article includes discussion of migrainous infarction, complicated migraine, migraine-induced stroke, migraine with cerebral infarction, migrainous stroke, and migraine-related stroke. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

In this article, the author updates the topic of migrainous infarction, including the diagnostic criteria proposed by the International Classification of Headache Disorders, 3rd edition (beta version), 2013. A patient with recurrent migrainous infarction was reported—one in the cerebellum, the other in occipital lobe.

Key points

• Migrainous infarction is a rare complication after usual attacks of migraine with aura with a documentation of neuroimaging findings, such as MRI. Cortical laminar necrosis is one of the MRI findings.
• The incidence of migrainous infarction is very rare, estimated as 3.36 per 100,000 person-years according to the strict criteria proposed by the International Headache Society.
• Migrainous infarction mostly occurs in the posterior circulation and in younger women with a history of migraine with aura.
• The majority of patients present with visual prolonged aura, and the stroke severity is mild with a good outcome.
• The pathologic mechanisms responsible for migrainous infarction remain unproven. One case report suggests a continuum between migraine aura and stroke by cortical spreading depolarization.

Historical note and terminology

Migraine attacks are occasionally accompanied by stroke. Permanent neurologic deficits associated with attacks of migraine were reported as early as the 19th century. Charcot first used the term “complicated migraine” (Charcot 1890), and Galezowski reported persistent visual sequelae (Galezowski 1881). Hunt wrote a classic paper concerned with permanent paralysis along with other neurologic complications of migraine (Hunt 1915).

The diagnosis of migrainous infarction is based on the abrupt onset of a neurologic deficit during a migraine attack associated with evidence of cerebral infarction on neuroimaging. Other causes of stroke must be excluded. Strict criteria for the diagnosis of migrainous infarction must be applied because migraine is common and patients with migraine may suffer from other causes of stroke. The diagnosis of migrainous infarction should be made only when a patient with an established history of migraine suffers a cerebral infarction during a typical migraine attack (Rothrock et al 1988).

Welch proposed the following expanded International Headache Society classification to encompass all migraine-related strokes:
(1) Coexisting stroke and migraine
(2) Stroke with clinical features of migraine
   (a) Symptomatic migraine
   (b) Migraine mimic
(3) Migraine-induced stroke
   (a) Without risk factors
   (b) With risk factors
(4) Uncertain (Welch 1994).

Clinical manifestations

Presentation and course

The diagnostic criteria of migrainous infarction were just revised by the International Classification of Headache Disorders, third edition (ICHD-III) (beta version), as follows (coded as 1.4.3) (Headache Classification Subcommittee of the International Headache Society 2013):

(A) A migraine attack fulfilling criteria B and C
(B) Occurring in a patient with 1.2 Migraine with aura and typical of previous attacks except that 1 or more aura symptoms persists for more than 60 min
(C) Neuroimaging demonstrates ischemic infarction in a relevant area
(D) Not better accounted for by another diagnosis

The diagnostic criteria, in fact, do not differ from those proposed by the International Classification of Headache Disorders, second edition, code 1.5.4 (Headache Classification Subcommittee of the International Headache Society 2004), except for some wording.

The most common clinical scenario is of a patient who has had frequent attacks of migraine headache preceded by temporary hemianopia. Following a particularly severe attack, the deficit persists and the patient is left with a permanent loss of vision in 1 visual field. This visual field deficit may regress gradually over the following months, ultimately leaving a clearly defined area of permanent visual field loss. The next most common signs of migrainous infarction are hemiparesis, monoparesis, and hemisensory symptoms, characteristically having the cheiro-oral distribution (ie, involving the digits and the side of the mouth). Less frequently, neurologic deficits include ataxia or persistent dysphasia. Although persistent neurologic symptoms associated with migraine are usually visual and localized to the region of the cerebral cortex supplied by the posterior cerebral arteries, ischemic strokes attributable to migraine also commonly occur within the territory of the middle cerebral artery. A rare case with right-side occipital infarction presented as persistent visual aura for 1 week without visual defect (Thissen and Koehler 2014). Lee and colleagues reported rare auditory symptoms as initial symptoms in 2 patients with migrainous infarctions (Lee et al 2003). Brain MRI showed infarction over the upper pons and cerebellum in 1 patient and negative findings in the other. It is of note that the negative neuroimaging finding in the latter case did not fulfill the criteria of migrainous infarction based on the ICHD-II (Headache Classification Subcommittee of the International Headache Society 2004). Hoekstra-van Dalen reported that migrainous infarction is a stroke entity that causes mostly infarcts in the occipital lobe (Hoekstra-van Dalen et al 1996). Migrainous infarction involving the bilateral anterior cerebral artery territory has been reported (Demirkaya et al 1999). Tang and colleagues reported 2 patients with migrainous infarctions that involved 2 different cerebral arteries (Tang et al 2004). A 29-year-old woman developed infarctions over the right posterior cerebral artery and right anterior choroidal artery simultaneously, and a 47-year-old man developed 2 episodes of migrainous infarction within 4 years involving the territories of the middle and posterior cerebral arteries sequentially. Even though migrainous infarction is considered more common in those younger than 45 years old, a 93-year-old woman was reported to suffer from migrainous infarction (Tzoulis et al 2006). In 1 unusual case, a 47-year-old man with visual aura was reported, who developed early recurrence of migrainous infarction—one in the cerebellum and the other in the occipital lobe (Renard et al 2015).

Arboix and colleagues reported 9 consecutive patients with migrainous infarction diagnosed according to the strict criteria of the International Headache Society (Headache Classification Committee of the International Headache Society 1988; Arboix et al 2003). Their mean age was 35.7±12 years. Six patients (67%) were women. All suffered from headache at the onset of neurologic deficit. The stroke manifested as limb weakness in 5 patients, sensory
symptoms in 5 patients, hemianopia in 4 patients, nausea and vomiting in 4 patients, and aphasia in 1 patient. Six patients had a cerebral infarct visible on neuroimaging studies (in the territory of the middle cerebral artery in 3 patients, posterior cerebral artery in 2 patients, and superior cerebellar artery in 1 patient). The neuroimaging studies of the remaining 3 patients showed negative findings even though the patients' neurologic deficit lasted at least 7 days.

Two large series, one from Germany (n=17) (Wolf et al 2011) and the other from Finland (n=33) (Laurell et al 2011), showed that migrainous infarction most commonly occurs in younger women with a history of migraine with aura. Most patients presented with prolonged visual aura (82%). The severity of the stroke was mild, with an NIH Stroke Scale score of 2. A total of 70.6% to 82% of patients had acute ischemic lesions in the posterior circulation. The prognosis was usually good. The differentiation between prolonged aura and migrainous infarction was difficult and was associated with delayed admission of patients to the hospital.

Prognosis and complications

In general, the long-term prognosis for patients with migrainous infarction is good. Milhaud and colleagues, in a prospective stroke registry, found that the outcome at 1 month was favorable in more than 70% of migraineurs with ischemic stroke (Milhaud et al 2001). Arboix and colleagues reported that the mean length of hospital stay of 9 consecutive patients with migrainous infarction was 9.75±6.2 days. No patients died during hospital stay, and 67% were symptom-free at discharge (Arboix et al 2003). Rothrock and colleagues followed 28 patients with migrainous stroke for a mean of 25.3 months. There were 6 recurrent strokes. The researchers reported that migrainous stroke itself showed comparatively poor prognosis for risk of recurrent stroke, and stroke recurrence rates of previous reports have been lower because the patient populations involved were either smaller than theirs or were followed for shorter intervals (Rothrock et al 1993). It is of interest that Linetsky and colleagues reported 6 patients with migrainous infarction whose headache frequency and severity decreased after ischemic stroke (Linetsky et al 2001). They hypothesized that the improvement in migraine may be due to reduced nociceptive transmission as the result of loss in vasoreactivity of the affected cerebral blood vessels. Two large series both showed a very favorable prognosis in patients with migrainous infarction (Laurell et al 2011; Wolf et al 2011).

Clinical vignette

A 32-year-old woman who worked as a laborer suffered from severe migraine for 1 week. This was followed by weakness in her limbs on the left side. She denied hypertension or any other cardiovascular risk factors and was not on oral contraceptives. Her usual pattern was 1 or 2 attacks of migraine without aura per month and, rarely, migraine with visual aura. During the week she had severe headaches and noted a visual aura (bright light) over her left visual field that lasted for 30 minutes. This lasted longer than her previous auras, and 2 hours later she noted transient left limb weakness that lasted for approximately 30 minutes. When she awoke the next day, her headache was less severe, but her left limbs were paralyzed. Neurologic examination showed left hemianopia and left hemiparesis (muscle power grade 1 to grade 2) with hyperreflexia and Babinski sign. MRI of the brain showed acute cerebral infarction over the right frontotemporal region compatible with MCA occlusion. MRA showed occlusion of the M1 segment of the right middle cerebral artery but no vasospasm. The patient had a normal electrocardiogram, cardiac echogram, carotid artery duplex sonography, ANA, prothrombin time, APTT, protein S, protein C, and anticardiolipin antibody. She was discharged on verapamil 240 mg once per day and aspirin 100 mg per day. In 6 months, she had recovered enough to walk without aid. At the 4-year follow-up, she still had occasional migraine without aura and infrequent migraine with aura. No recurrence of stroke was noted.

Biological basis

Etiology and pathogenesis

The exact cause of migrainous infarction is still not certain. Based on studies using cerebral angiography during attacks, the most important underlying mechanism for the stroke is believed to be carotid or vertebral arterial spasm resulting in a critical degree of cerebral hypoperfusion (Featherstone 1986; Rothrock et al 1988; Sanin and Mathew 1993). However, this may not always be true, so other factors should be considered.

Occasionally, drug therapy is considered to be a contributing or precipitating factor. For many years, ergotamine overdoses have been thought to cause constriction of the cerebral vessels. Propranolol has also been reported to be
related to permanent neurologic defects in some patients, particularly if the drugs induced postural hypotension among elderly patients (Prendes 1980). This relationship, however, is not well established. Mendizabal and colleagues reported a case of migrainous stroke in which treatment with propranolol was associated with stroke onset and may have played a causative role (Mendizabal et al 1997). Oral contraceptives, particularly in high doses, have been found to greatly increase the incidence of migrainous cerebral infarction, especially in young women (Chang et al 1999; Milhaud et al 2001). Serotonergic medications were perhaps responsible for ischemia and the subsequent stroke (Molaie 1997; Singhal et al 2002).

It is possible that some migraineurs’ strokes are a result of microemboli associated with these disorders (Nozari et al 2010). Patent foramen ovale, a potential source of paradoxical cerebral embolism, was found to be more frequent in young patients with migraine with aura (Anzola et al 1999). Milhaud and colleagues found that patent foramen ovale is an independent risk factor for ischemic stroke in young migraineurs (Milhaud et al 2001). Wolf and colleagues found a high frequency in a German series (64.7%) (Wolf et al 2011). However, Laurell and colleagues did not show an increased frequency (40%) (Laurell et al 2011). Carerj and colleagues reported a higher prevalence of atrial septal aneurysm in patients with migraine with aura than in patients with migraine without aura and controls, suggesting that atrial septal aneurysm may play a part in cryptogenic stroke in migraineurs (Carerj et al 2003). Other investigators have related carotid or vertebral artery dissection to cerebral infarction in patients with migraine.

The pathologic mechanisms responsible for cerebral infarction remain unproven; arterial vasospasm (with cerebral edema), arterial wall dissection, and increased platelet aggregation are the most likely candidates. Other factors may contribute to migrainous infarction, but extracranial or intracranial vasospasm is believed to play the major role.

Fifty years ago, Wolff and his coworkers suggested that arterial vasospasm was the primary cause of the aura preceding the migraine attack (Wolff et al 1953). This vascular theory of migraine has been supported by studies of cases of “complicated migraine” and migrainous infarction where prodromes have persisted and cerebral infarction has been documented. All investigators agree that attacks are accompanied by reduced cerebral blood flow during the aura. Meyer and colleagues reported significant reductions in regional cerebral blood flow during the prodromal interval with 133Xe inhalation (Meyer et al 1991). The evidence that vasospasm is the primary cause of the symptoms of migrainous aura is supported by regional cerebral blood flow measurements, arteriographic data, and limited autopsy studies demonstrating the absence of intrinsic vascular or cardiac disease despite extensive brain infarction.

It is possible that patients with migrainous infarction have rheological abnormalities predisposing to platelet aggregation, coagulation disorders, and intravascular thrombosis. Welch contends that “spreading oligemia” can lead to platelet accumulation in hypoperfused cerebral areas (Welch 1987). Vasoactive substances released by platelets could further reduce cerebral blood flow to ischemic levels. The proportion of platelet microaggregates in the circulation has been shown to be greater than in control individuals during the prodromal period of an attack. Augmentation of this intrinsic platelet abnormality by extrinsic factors (eg, oral contraceptives, alcohol abuse, cigarette smoking, and diabetes mellitus) combined with intrinsic conditions (eg, mitral valve prolapse or migrainous vasospasm) are probable factors in the genesis of migrainous strokes. The frequent presence of antiphospholipid antibodies and lupus anticoagulant in young patients with stroke has suggested that these factors play an important part in migrainous stroke (Silvestrini et al 1994). However, this finding remains controversial (Tietjen et al 1998). Santos and colleagues reported a 43-year-old woman who developed migraine aura-like symptoms before developing right-side, middle cerebral artery ischemic infarction (Santos et al 2012). During operation, several episodes of cortical spreading depression were recorded with increasing levels of glutamate and lactate/pyruvate ratio. This case provided a link between migraine aura and stroke by cortical spreading depolarization.

The von Willebrand factor, a large, multimeric glycoprotein acting as a procoagulant via stimulation of platelet adherence, activation, and aggregation, has been purported to be an independent risk factor for ischemic stroke. The role of von Willebrand factor in migrainous infarction was explained by Tietjen and colleagues, who found that migraineurs with or without prior stroke had significantly higher von Willebrand factor antigen and activity than controls (Tietjen et al 2001). Proinflammatory platelet adhesion to leukocytes occurring during the headache-free interval in patients with migraine is similar to that seen in those with acute coronary or cerebrovascular syndromes (Zeller et al 2005). This may support the hypothesis that migraine attacks predispose to stroke by inducing platelet-related hypercoagulability.

According to the ICHD-2, migrainous infarction has to occur in patients with migraine with aura. It is possible that the mechanisms underlying migrainous infarction and migraine with prolonged aura might be the same. However, a study
adopting 31P-MR spectroscopy found the cortical energy reserve in patients with migrainous infarction was similar to that in controls but higher than in patients with migraine with prolonged aura (Schulz et al 2009).

Wolf and colleagues found that acute ischemic lesions in patients with migrainous infarction were often multiple and located in distinct arterial territories and that there were no overlapping ischemic lesions; therefore, they suggest that hemodynamic compromise during the development of migraine is unlikely to be the cause of infarction (Wolf et al 2011).

**Epidemiology**

The incidence of migrainous infarction is rare, according to the strict criteria proposed by the International Headache Society. Henrich and colleagues reported that the incidence rate of first migrainous infarction was 3.36 per 100,000 person-years (Henrich et al 1986). However, in the absence of other stroke risk factors, this estimate was reduced to 1.44 per 100,000 person-years. In a study of the Barcelona Stroke Registry, Arboix and colleagues reported that the group of patients with migrainous infarction accounted for only 0.6% of all first-ever acute strokes, 0.8% of ischemic strokes, 12.8% of ischemic strokes of unusual etiology, and 13.7% of ischemic strokes in young adults 45 years of age or younger (Arboix et al 2003). Therefore, stroke can occur during migraine attacks, but the association between migraine and stroke is infrequent (Bousser 1999; Tzourio et al 2000). It was also reported that ischemia-induced migraine attacks may be more frequent than migraine-induced ischemic insults (Olesen et al 1993). A large series in Germany found 17 patients among 8137 stroke patients (0.2%) over an 11-year period (Wolf et al 2011).

Unlike the rare epidemiological survey of migrainous infarction, the association between migraine and stroke has been well documented (Buring et al 1995; Merikangas et al 1997; Wang et al 2010). A case-control study in young women also revealed that oral contraceptives, high blood pressure, and smoking were high risk factors for infarction in migraineurs (Chang et al 1999). Schwaag and colleagues repeated this association that migraine was a significant risk factor for juvenile stroke in Germany (Schwaag et al 2003). The risk was even higher in people younger than 35 years of age and in females. In a cross-sectional study, migraineurs, particularly those who had migraine with aura, had a higher cerebrovascular risk than individuals without a history of migraine (Scher et al 2005). By following a cohort of the Women's Health Study for a mean of 9.9 years, Kurth and colleagues demonstrated that migraine with aura but not without aura was associated with about a 2-fold increased risk of ischemic stroke as well as myocardial infarction and ischemic cardiovascular death and a 1.7-fold increased risk of coronary revascularization (Kurth et al 2006).

Compared with women without migraine, Kurth and colleagues observed a J-shaped relationship between migraine frequency and cardiovascular events, with a higher risk for very infrequent (< monthly) and frequent migraine attacks (> or = weekly) and a lower risk for monthly migraine (Kurth et al 2009). The risk was only increased for women with migraine with aura. Stroke was associated with migraine with aura and a migraine frequency of at least 1 per week. The prospective data from the Physicians' Health Study also indicate an increased risk of stroke in men with migraine (RR: 1.84; 95% CI: 1.10–3.08) who were aged 40 to 45 years at study entry. This association was not found in older age groups (Kurth et al 2007). The same group further demonstrated that the association between migraine with aura and cardiovascular disease varies by vascular risk status. The association was strongest in the lowest cardiovascular risk score group stratified by the Framingham risk score (Kurth et al 2008). A meta-analysis confirmed the association between migraine aura and stroke but not myocardial infarction (Schurks et al 2009).

**MRI** has been successfully used to evaluate clinical and subclinical lesions in the brain. Subclinical cerebral lesions, especially in the posterior circulation or white matter, were reported to be more frequent in patients with migraine (especially migraine with aura) in a case control MRI study, ie, CAMERA (Cerebral Abnormalities in Migraine, an Epidemiological Risk Analysis) study (Kruit et al 2004). The same group, in another study, demonstrated that most (88%) infratentorial infarct-like lesions had a vascular border zone location in the cerebellum and, further, that a combination of (possibly migraine attack-related) hypoperfusion and embolism is the likeliest mechanism for posterior circulation infarction in migraine (Kruit et al 2005). Scher and colleagues studied the association between migraine in middle age and late-life infarct-like lesions in a prospective, large, population-based cohort, ie, Age Gene/Environment Susceptibility (AGES)-Reykjavik Study from Iceland (Scher et al 2009). After on average 25 years, subjects with migraine with aura had increased odds of late-life infarct-like lesions on MRI (OR=1.4), a result entirely driven by an association of migraine with aura and cerebellar lesions among women (OR=1.9). There was no association between migraine without aura or nonmigraine headache with the infarct-like brain lesions.
**Prevention**

Featherstone reported that patients with migrainous stroke were usually young adults with a past history of migraine with aura whose headache attacks had worsened in severity and duration just before the stroke (Featherstone 1986). Therefore, it is wise to initiate prophylactic measures for stroke during these prodromal intervals. Comorbid cardiovascular risk factors (including angina pectoris, diabetes mellitus, hyperlipidemia, hypertension, collagen disease, blood dyscrasias, and cigarette smoking) should also be controlled.

It has been reported that a history of migraine is a risk factor for cerebral ischemia in patients younger than 45 years of age (Collaborative Group for the Study of Stroke in Young Women 1975; Buring et al 1995; Schwaag et al 2003). Migraine-related stroke is more common in individuals with aura than those without aura, but it does occur in migraine patients without aura (Leira 1997). Patients with a history of migraine-associated stroke are at significantly increased risk for recurrent strokes (Rothrock et al 1993). Avoiding migraine drugs with marked vasoconstrictive action (ergotamine or possibly sumatriptan) and using them in accordance with labeling and removing other vascular risk factors (smoking and oral contraceptives) are additional measures for the prevention of migraine-related stroke, especially in patients with migraine with aura (Leira 1997; Bousser 1999; O'Quinn et al 1999). On the subject of oral contraceptives and hormone replacement therapy in women with migraine, the International Headache Society Task Force recommended that combined oral contraceptives should be stopped when the following conditions exist: (1) new persisting headache, (2) new onset of migraine aura, (3) increased headache frequency or intensity, and (4) development of unusual aura symptoms, particularly prolonged aura (Bousser et al 2000).

**Differential diagnosis**

Because migraine is so common in the general population, it is not unusual to observe patients with cerebral infarction who also have a history of migraine. The fact that a cerebral infarction develops around the time of a migraine headache does not necessarily mean that they are related.

Headache may occur acutely with any stroke; approximately 30% of stroke patients have headaches (Arboix et al 1994). At times, pulsatile headaches follow both atherothrombotic and embolic cerebral infarction. Thus, in a migraineur, headaches at the onset of a cerebral infarction may not necessarily be of the migraine type. Some conditions—in particular, arterial dissection, arteriovenous malformation, cardioembolic stroke, and intracranial neoplasm—can mimic migrainous infarction.

Idiopathic thunderclap headache with vasospasm presenting as severe intractable headache may be followed by a delayed stroke. It mimics migrainous infarction; however, the onset of headache is much more abrupt (within a split second) in patients with thunderclap headache (Dodick 2002).

Shuaib described 5 patients with migraine headache who developed cerebral infarctions (Shuaib 1991). The initial diagnosis in all cases was migrainous infarction, but the diagnosis was later revised. Three patients had arterial dissection (1 proven at autopsy), 1 had marantic endocarditis (proven at autopsy), and 1 had generalized atherosclerosis associated with diabetes.

**Diagnostic workup**

Because distinct diagnostic criteria are lacking, migrainous infarction should be considered a diagnosis of exclusion. The diagnosis demands a well-established history of migraine and exclusion of other conditions that cause stroke. Computed tomography, MRI (including diffusion-weighted image, perfusion-weighted image, MRS, and MRA), cerebral arteriography, transcranial Doppler evaluations, and transesophageal echocardiography evaluations should be performed when possible. Lumbar puncture and blood studies to help exclude vasculitis should also be carried out.

MRI techniques provide more information for acute cerebral infarction and may be the diagnostic tool of choice for migrainous infarction (Neumann-Haefelin et al 2000), particularly if the clinical examination demonstrates brainstem or cerebellar signs. It might also be useful in patients with prolonged aura (Smith et al 2002). It has been reported that elevation of cerebral lactate can be detected among patients with migrainous infarction by localized 1H MRS (Watanabe et al 1994). Liang and Scott (Liang and Scott 2007) reported a patient with migrainous infarction whose MRI showed cortical laminar necrosis. The authors hypothesized that sluggish perfusion combined with a variety of metabolic derangements (spreading depression in the case of migraine) probably accounted for this unusual finding.
Two patients with migrainous infarction showed cortical laminar necrosis in their MRI studies (Arboix et al 2013; Parks et al 2014). Arai and associates reported on the chronological changes in neuroimaging findings in a 64-year-old man with migrainous infarction (Arai et al 2008). Of note, the MRI abnormalities suggested the presence of vasogenic edema, prolonged hyperperfusion, and alteration of the blood-brain barrier followed by irreversible brain damage.

When carried out at the time of the acute process, cerebral arteriography often reveals abnormalities during migrainous stroke. Dukes and Vieth first reported angiographic observations in migraine with aura (Dukes and Vieth 1964). Sequential angiography made during the prodromal phase showed progressive decreases in the caliber of the internal carotid arterial system. Lieberman and colleagues reported a case of complicated “diplegic migraine” with angiographic demonstration of vasospasm of both the cervical carotid and intracranial middle cerebral arteries (Lieberman et al 1984). However, cerebral arteriography carries a significant complication rate, and the risk-to-benefit ratio should be carefully weighed in the acute stages of presumed migrainous infarction.

Gomez and colleagues reported an interesting case of a young woman with a longstanding history of migraine with aura who developed acute cerebellar infarction in association with reversible vasospasm of the vertebral arteries (Gomez et al 1991). The vasospasm was demonstrated by transcranial Doppler sonography. However, the differential diagnosis of thunderclap headache with vasospasm should also be considered in this case (Dodick 2002).

Management

Prophylactic treatment of migrainous infarction in patients with prolonged aura includes platelet antiaggregants or calcium channel blockers. Aspirin administration should be considered as prophylactic treatment against migrainous infarction. Patients who have anticardiolipin syndrome may need anticoagulants. Calcium channel blockers are recommended for patients at risk for migrainous infarction. Nimodipine reduces cerebral vasoconstriction, and several studies have demonstrated the potential effectiveness of calcium channel blockers in the prophylactic treatment of migraine headaches (Meyer 1985).

Ergotamine, triptans, and serotonergic medications may initiate or worsen intracranial vasospasm and dysautoregulation in patients with migrainous infarction. These drugs should not be given to patients with prolonged aura. Some experts believe that peripheral beta-blockers such as propranolol should also be withheld because they may worsen intracranial vasoconstriction. For prophylactic purposes, verapamil should be considered for migraine attacks and for migrainous infarction.

Special considerations

Pregnancy

Pregnancy is clearly a risk factor for stroke, but several mechanisms are involved (Wiebers 1985). However, the risk of migrainous infarction was not studied in pregnant women.

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**References especially recommended by the author or editor for general reading.

**Former authors**

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**ICD and OMIM codes**

**ICD codes**

ICD-9:
Variants of migraine: 346.2

ICD-10:
Complicated migraine: G43.3

**Profile**

**Age range of presentation**

06-12 years  
13-18 years  
19-44 years  
45-64 years  
65+ years

**Sex preponderance**

female>male, >1:1  
female>male, >2:1

**Family history**

family history may be obtained

**Heredity**

heredity may be a factor

**Population groups selectively affected**

none selectively affected
Occupation groups selectively affected
none selectively affected

Differential diagnosis list
arterial dissection
arteriovenous malformation
cardiembolic stroke
intracranial neoplasm
idiopathic thunderclap headache
severe intractable headache
marantic endocarditis
generalized atherosclerosis associated with diabetes

Associated disorders
Blood dyscrasia
Cardiac dysrhythmias
Collagen disease
Diabetes mellitus
Hypertension
Mitral valve prolapse
Prinzmetal angina

Other topics to consider
Headache associated with ischemic cerebrovascular disease
Migraine
Oral contraceptives and stroke