PREVALENCE OF CARDIAC SHUNT LESIONS IN PATIENTS WITH MIGRAINE WITH AURA AND WITHOUT AURA

Dissertation Submitted in partial fulfillment of the regulations for the award of the degree of

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DECLARATION

I, **Dr. S. ELANGOVAN,** solemnly declare that dissertation titled, **"PREVALENCE OF CARDIAC SHUNT LESIONS IN PATIENTS WITH MIGRAINE WITH AURA AND WITHOUT AURA "** is the bona fide work done by me at Govt. Stanley Medical College and Hospital during the period January 2006 to March 2008 under the expert guidance and supervision of **Prof. A. MURUGESAN, M.D.D.M.,** Head of the Department, Department of Neurology.

The dissertation is submitted to the **Tamil Nadu Dr. MGR Medical University** towards partial fulfillment of requirement for the award of D.M Degree (Branch I) in Neurology**.**

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CERTIFICATE

This is to certify that this dissertation entitled **"PREVALENCE OF CARDIAC SHUNT LESIONS IN PATIENTS WITH MIGRAINE WITH AURA AND WITHOUT AURA "** is a bona fide original work of **Dr. S. ELANGOVAN** in partial fulfillment of the requirement for **D.M (Branch I) Neurology** examination of the Tamil Nadu Dr.MGR Medical University to be held in August 2008.

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INTRODUCTION

Migraine headache is a common disabling condition which represents a significant healthcare burden. It is characterised by recurrent disabling attacks of headache associated with nausea, vomiting, hypersenstivity to light, sound, and smell and in a third of patients neurological aura symptoms. These aura symptoms are fully reversible visual, sensory or speech disturbances. Visual disturbances are common and last for 5 – 60 mins. The underlying neurological mechanism for aura is cortical spreading depression[32].

Recent evidence found a significant association between patent foramen ovale and transient global amnesia. This finding prompted the suggestion that paradoxical microembolisation in the vertebrobasilar territory may cause transient global amnesia. Like transient global amnesia, migraine with visual aura is a paroxysmal disturbance in which a sudden dysfunction of cortical areas fed by the terminal branches of the basilar artery is believed to trigger the attack. Arteriovenous shunting of vasoactive substances from the venous to the arterial system through a patent foramen ovale in patents with migraine may affect endothelial function (van mokt et al)[45]. In individuals with a right to left shunt, a lower dose of venous trigger substances may be needed to induce migraine because the shunt permits the pulmonary filter to be bypassed.[5,45,49].

Many studies have shown migraineurs who experience an aura are more likely to have a patent foramen ovale (48%) than the general population (20%). Right to left cardiac shunt at rest through a patent foramen ovale (PFO) is more common in migraineurs with aura (15%) than in control patients with patent foramen ovale who do not experience migraine (0%). This suggest that interatrial communication may play a role in the pathogenesis of migraines. Closure of patent foramen ovale had resolved symptoms of migraine with aura in 50% of patients and improved symptoms in other 50% of subjects [5,29,53]. Many studies have demonstrated closure of patent foramen ovale reduce migraine symptoms [3,5,11,14,15,29,37].

This study investigated the prevalence of cardiac shunt lesions in migraine patients with and without aura. The transesophageal contrast echocardiography has been done to detect intracardiac shunts in migraine patients with and without aura.

AIMS AND OBJECTIVES

1. To evaluate the frequency of cardiac shunt lesions in patients with migraine with aura and migraine without aura.

2. To correlate the variables of migraine with variables of shunt.

3. To compare the results of our study with that reported by other studies performed in various parts of the world.

REVIEW OF LITERATURE

Migraine is a common disabling primary headache disorder. Epidemiological studies have documented its high prevalence and high socio economic and personal impacts. It is now ranked by the world health organisation as number 19 among all diseases worldwide causing disability. It is estimated that 2,50,000 patients in the U.S. have at least one migraine per week, with a lifetime prevalence of 18% . Migraine is a risk factor for cryptogenic stroke, particularly in young patients without artherosclerosis. The last 20 years have witnessed a great deal of advances in understanding of the pathophysiology, pharmacology, epidemiology and genetics of migraine. The International headache society (IHS) classified headache into primary headache disorder and secondary headache disorder. Migraine with and without aura, tension type headache and other trigeminal autonomic cephalalgias from the majority of primary headache disorders.Migraine is a primary headache disorder characterised by various combinations of neurological, gastrointestinal and autonomic changes. The word Migraine is derived from the Greek word “hemicrania” (galen, circa 200 AD). The diagnosis is based on the headache’s characteristics and associated symptoms.

Differential diagnosis of primary beadacbes

Clinical Features	**Migraine**	**Tension-type headache**	**Cluster headache**
Men:women	25:75	40:60	90:10
Lateralisation	60% unilateral	Diffuse bilateral	100% unilateral
Location	Frontal, periorbital, Periorbital temporal, hemicranial	Diffuse	
Frequency	1-4 per month	1-30 per month	1-3 per day for 3-12 months
Severity	Moderate/severe	Mild/moderate	Extremely severe
Duration	4-72 hours	Variable	15minutes-3 hours
Pain character	Throbbing, pulsating	Dull	Sharp, boring
Periodicity	±	-	+++
Family history	+++	±	±
Associated symptoms			
Aura	+++	-	-
Autonomic features	±	-	+++
Nausea/vomiting	+++	-	±
Photo/phonophobia	+++	-	±
Exacerbation by movement	+++	-	-

The International headache society diagnostic criteria for headache disorders (1988) [19] have been revised (2004) 2nd edition[20] (cephal 2004) First revision 2nd edition published in may 2005 - ICHD II R1 [43] and describe a total of seven sub types of Migraine .

Epidemiology of Migraine

American Migraine study I conducted in1989 revealed that approximately 18% women and 6% of men in the united states have migraine, resulting in an estimated 23.6 million Americans experiencing migraine. Migraine prevalence varies with the age, the highest being found among persons between 35 and 45 years of age. The prevalence of migraine was found to be higher in the lowest income group and whites than in blacks. The American migraine study II conducted 10 years later had similar prevalence estimates but estimated number of migraineurs had increased to approximately 28 million due to population growth.

Migraine prevalence varies by age, gender, race and income. Before puberty, the migraine prevalence approximately 4%. After puberty the prevalence increases more rapidly in girls than boys. It increases until approximately age 40, then declines. The prevalence is similar in the European countries and in the United states. The prevalence is lowest among Asian Americans, intermediate among African Americans and highest among white persons.

MIGRAINE ASSOCIATED DISABILITY

Migraine substantially affects quality of life. The world health organization ranks Migraine among the world's most disabling medical illnesses[48]. Approximately 28 million Americans have severe disabling migraine headaches. Migraine's annual cost to employers is approximately $13 billion and related annual medical costs exceed $1 billion. 81% of migraineurs reported functional impairment with the headache in the American Migraine study II [24]. 53% reported that severe headache caused extreme impairment in activities and required bed rest.

Duration of Migraine associated activity restrictions was greater among women migraineurs. Approximately 31% of all migraineurs missed more than a day of work or school because of Migraine the 3 months prior to the survey [24]. 51% reported that work or school activity was reduced by more than 50%. Household and family or social activities are more likely than work or school activity to be disrupted by Migraine. 67% reported that household productivity was reduced by more than 50%. Many migraineurs live in fear, knowing an attack will disrupt their ability to work, to take care of their families or to meet social obligations. Health related quality of life measurements have shown that compared with other chronic illnesses like hypertension, diabetes, and coronary artery diseases, migraine has lowest scores in physical functioning, bodily pain and other health aspects. Clinically

validated migraine disability scales are available for clinical use. These include the Migraine disability assessment scale (MIDAS)[26, 41] and Headache impact scale (HIT)[47] . The data from a population based sample of about 2000 migraineurs, 50% of migraineurs account for more than 90% of all work loss due to migraine.These finding imply that health care intervention should be directed to the most disabled segment of the migraine population [39]. Because Migraine remains prevalent, disabling,undiagnosed and untreated in India,public health initiatives to improve diagnosis and treatment are needed.

Diagnosis

Migraine is a syndrome of recurrent headaches with a wide variety of neurological and non neurological manifestations. It is not simply a headache. Diagnosis of Migraine is not obtained solely by exclusion of other disorders. Positive diagnosis is possible. This should be based on information about the attack profile, on identification of probable triggers and on understanding of the clinical spectrum. About 70% of Migraineurs have a positive family history.

IHC – 2 Classification of Migraine

IHC- 2 Code and Syndrome

1. Migraine

    1.1 Migraine without aura

    1.2 Migraine with aura

        1.2.1 Typical aura with Migraine headache

        1.2.2 Typical aura with Non migraine headache

        1.2.3 Typical aura without headache

        1.2.4 Familial hemiplegic Migraine

        1.2.5 Sporadic hemiplegic Migraine

        1.2.6 Basilar type Migraine

    1.3 Childhood periodic syndromes that are commonly precursors of migraine

        1.3.1 Cyclical vomiting

        1.3.2 Abdominal Migraine

        1.3.3 Benign paroxysmal vertigo of childhood

    1.4 Retinal Migraine

    1.5 Complications of migraine

        1.5.1 Chronic Migraine

        1.5.2 Status migrainosus

        1.5.3 Persistent aura without infarction

1.5.4 Migrainous infarction

1.5.5 Migraine triggered seizure

1.6 Probable Migraine

1.6.1 Probable migraine without aura

1.6.2 Probable migraine with aura

1.6.3 Probable Chronic migraine

Migraine can be divided into four major sub types. 1.1 Migraine without aura is a clinical syndrome characterised by headache with specific features and associated symptoms. 1.2 Migraine with aura is primarily characterised by focal neurological symptoms that usually precede or sometimes accompany the headaches. Migraine without aura is the most common subtype of migraine. It has a higher attack frequency and is usually more disabling than migraine with aura. Only less than 30% of attacks are migraine with aura. Migraine without aura is the disorder most prone to become chronic and this chronic is aided by excessive use of immediate relief medications and comorbidities.

Migraine with aura

Definitions

The syndrome of migraine with aura (MA) has been known by a variety of terms over the years, including classic or classical migraine, migraine accompanee, complicated migraine and depending on the nature of the aura symptoms, ophthalmic, hemiparesthetic, hemiplegic, or aphasic migraine.

The current International Headache Classification (IHC-2) defines aura as follows:

A recurrent disorder manifesting in attacks of completely reversible focal neurological symptoms with a mix of positive and negative features, which usually evolve over 5-20 minutes and lasts for less than 60 minutes, which may or may not be followed by headache.

This definition allows that the accompanying headache may be either of typical migraine type or another primary headache form, and also that aura may not necessarily be associated with headache at all [defined as "typical aura without headache" under IHC-2, and sometimes called acephalalgic or (erroneously) acephalgic migraine].

Incidence

The incidence of neurological auras in the community is not known, but a recent study of 952 migraine patients attending a tertiary care center in Atlanta found that just over one-third of migraineurs reported aura with about 20% of all migraine attacks being associated with aura. The average aura duration was about 27 minutes, and the headache phase began about 10 minutes after the onset of aura. More than 90'% of auras were visual.

Migraine with aura may affect only 5 to 10% of the population, but auras without headache are much more common and frequently go unrecognized or undiagnosed.

Knowledge of the characteristics of migraine headache and aura almost certainly influence the observed prevalence. For example, Alvarez recorded 618 instances of migrainous auras preceding headache in migraineurs. Overall, 12% of his male cases reported visual auras occurring in isolation, but among physicians (almost all male), 87% had experienced auras without headache . He concluded that aura without headache might be much more common than "classical" migraine.

Characteristics

Like epileptic auras, migraine auras usually begin with an excitatory phase with positive symptomatology, believed to reflect the advancing excitatory wave of spreading depression. For example, excitation of the visual

cortex results in auras such as "flashing lights," whereas auras arising in the primary sensory cortex cause paresthesia on the opposite side of the body, and so forth. The neuronal inhibition and, possibly the associated oligemia that follows, cause negative symptoms such as scotoma, dysphasia, numbness, and paresis. Such symptoms are not a feature of epileptic auras, although Todd's paresis might be analogous. This neural inhibition phase lasts much longer than the excitatory phase, and indeed can sometimes persist for weeks or months-persistent aura without infarction and migraine coma.

Migraine auras occur in clear consciousness and are perceived by patients as "real," although recognizing that they are not. They are generally considered to be hallucinations. A hallucination is the apparent perception of an external stimulus or object that is not actually present-perceiving things that are not there-as opposed to an illusion, the perception of an external object as a result of a "false belief' or misinterpretation of real events or inputs.Migraine auras can also produce illusions, such as vertigo and metamorphopsias. The distinction is arguably an exercise in arcane semantics, but when interpreting visual symptoms, it is important to distinguish auras from vivid imagery, i.e., images that appear "in the mind's eye." Such perceptions are largely under voluntary Control.

Classification of Auras

"Typical" auras- Visual	Simple Positive, e.g., phosphenes and teichopsia Negative, e.g., scotoma and hemianopia Complex e.g., visual metamorphopsia
Somesthetic	Simple Positive, e.g., tingling Negative, e.g., numbness Complex Somesthetic metamorphopsia
Aphasic	Expressive dysphasia Receptive dysphasia Dyslexia
"Atypical" auras	
Primary sensory	Olfactory Auditory Visceral Kinesthetic Limb pain
Vestibulocochlear	Vertigo Deafness Drop attacks
Motor	Chorea Dystonia Hemiplegia
Higher integrative functions	Memory Mood Perception and planning

"Typical" Auras

Visual Auras

Visual auras originate in the visual cortex and are generated by cortical spreading depression (CSD)[32]. Such auras are typically simple, involving the

functions of the striate and extrastriate cortex. Complex auras, with symptoms suggesting involvement of visual and parietal cortical-association areas, are rare. It is characteristic of all visual auras that the images persist when the eyes are closed. The visual phenomena are often perceived as confined to one eye and described as such by the patient but are actually hemianopic.

Positive symptoms: Simple auras usually include "positive" elemental phenomena, such as dazzling stars, sparks, or flashes, which are usually white but sometimes spectral, occurring singly or in multiple showers. Patients usually describe them as "flashing lights." These primitive hallucinations are generally referred to as scintillations or phosphenes. Other descriptions of positive visual auras given by patients include "electric light bulb clement" and "like a wriggling snake." As noted above, the more formed auras sometimes have colored edges. Less commonly, they may become progressively more pronounced with kaleidoscopic effects. A common experience is of "shattering" of images into an irregular crystalline mosaic.

Visual Auras	
Simple	Elemental (scintillations, phosphenes, kaleidoscopic)
Positive	Blurring, heat haze, "water on glass" Teichopsia (scintillating scotoma and fortification spectra)
Negative	Hemianopia/ Tunnel vision Blindness
Complex	Visual metamorphopsia Agnosia Distortion of motion and space

Negative symptoms: Negative visual aura symptoms are actually

more common than positive symptoms and often occur in isolation. Typical impressions include "blurring" of vision, a rippling effect in the central fields like water running down glass, a "corona" around the image, or a heat haze. More dramatic negative symptoms include the classical expanding translucent scintillating scotoma. This may have a jagged edge, in which case the image is referred to as teichopsia or fortification spectrum, as first clearly illustrated by Hubert Airy.The fortification spectrum, so called by Dr. John Fothergill (1712-1780) because of its similarity to the castellations of a battlement, such as surrounds the French town of Lille or the Dutch town of Naarden, starts as a zigzag figure near the fixation point and gradually spreads right or left (depending on which visual cortex is generating the image), assuming a laterally convex shape with an angulated scintillating edge (the expanding angular spectrum of Gowers), leaving variable degrees of absolute or relative scotoma in its wake. The zigzag edge, the positive part of the aura, may be colored.

Migraine specks, geometrical forms and shimmering in the visual field are Scotoma without positive phenomenon and is usually enlarges gradually. More complicated visual auras include teichopsia, or fortification spectra (the characteristic aura of migraine) metamorphopsia, micropsia, macropsia, zoom vision and mosaic vision. The fortification spectrum that is zig zag figure near the point of fixation that may gradually spread right or left and assume laterally convex shape with an angulated scintillating edge leaving variable

degree absolute or relative scotoma in its wake.

Next in frequency are sensory distribuances in the form of pins and needles moving slowly from the point of origin and affecting a greater or smaller part of the side of body and face. Parathesias are often cheiro aural. Numbness starts in hand, migrates up the arm and jumps to involve the face, lips and tongue. Weakness is rare, occurs in association with sensory symptoms. Weakness is unilateral and it may be classified as either familial hemiplegic migraine (1.2.4) or sporadic hemiplegic migraine (1.2.5) .Less frequent are speech disturbances, usually dysphasic but often hard to characterise. Apraxia, aphasia, agnosia,states of altered consciousness associated with deja vu or jamais vu and elaborate dreamy, nightmarish, trancelike or delirious states can occur.

The Migraine aura was believed to be caused by cerebral vasoconstriction and the headache by reactive vasodilatations. This explained the throbbing quality and its relief by ergots. It is now believed that aura is caused by neuronal dysfunction. Headache often begins while cortical blood flow is reduced [32]. This headache is not caused by simple reflex vasodilatation.

The migrainous fortification spectrum corresponds to an event moving across the cortex at 2 - 3 mm / min. Noxious stimulation of the rodent cerebral cortex produced a spreading decrease in electrical activity that moved at 2 – 3

mm / min. Cortical spreading depression (CSD) is characterised by shifts in cortical steady state potential; transient increases in potassium, nitric oxide and glutamate levels and transient increases in cerebral blood flow followed by sustained decrease in cerebral blood flow (CBF). Olesen and colleagues [32] found 17% - 35% reductions in posterior cerebral blood flow which spread anteriorly at 2 – 3 mm / min. Reduced cerebral blood flow persisted from 30minutes to 6 hrs and then cerebral blood flow slowly return to baseline or even increased. The rates of progression of spreading oligemia are similar to those of migrainous scotoma and cortical spreading depression which suggests that they are related[32]. Additional studies support the hypothesis that cortical spreading depression produces the aura. During visual auras, cerebral blood flow decreased 15 % - 53 %, cerebral blood volume decreased 6% - 33% and mean transit time increased 10% - 54% in the occipital cortex contralateral to where the aura was experienced. The perfusion defect moved anteriorly. There would appear to be preferential pathways for the propagation of CSD, because certain auras, defined under IHC-2 as “typical” (visual hallucinations, somasthetic and dysphasic symptoms) are more common than other forms of aura. Motor auras, for example, are distinctly uncommon. It seems likely that CSD is normally triggered by a critical increase in interstitial K^+ concentration. The control of interstitial K^+ concentration depends on glia. Neurons are a source of K^+, while glia represent a K^+ “sink”, so it would therefore be expected that CSD would

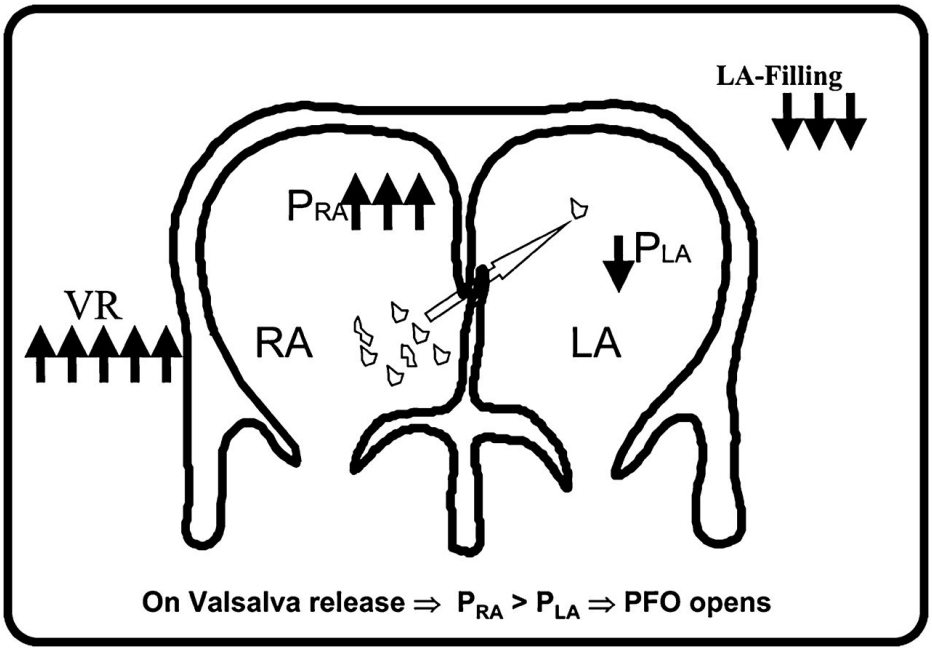
occur more readily in cortical sites with a low glia-to-neuron ratio. The susceptibility of brain areas to CSD probably depends on local glia-dependent K^+ homeostasis, and that the occipital cortex might be a "hot spot" for CSD because of its relatively low gila-to-neuron ratio compared, for example, to the frontal cortex . It is also clear that CSD is more readily observed in lissencephalic than gyrencephalic animals such as primates, which have larger cortical glia-to-neuron ratios; and it cannot be elicited at all in the spinal cord, which has comparatively few nerve cell bodies . Visual stimulation was used to trigger the headache in migraineurs. A wave of increased BOLD (Blood oxygen level dependent) functional magnetic resonance imaging signal and then decreased BOLD signal propagated into contiguous occipital cortex at 3 – 6 mm / min.

Association of interatrial shunts and migraine headaches

The foramen ovale is composed of the septum primum (left atrial side, fenestrated cranially) and the septum secundum (right atrial side, fenestrated caudally) joined in parallel forming a slit-like valve that shunts oxygenated blood to the systemic circulation during fetal development. A patent foramen ovale (PFO) results from lack of normal closure postnatally. Atrial septal defects (ASD) and patent foramen ovale (PFO) are present in approximately 25% of patients at autopsy[17] and 45% of patients with cryptogenic stroke [2,23,42]. An underlying mechanism of stroke in such patients

includes transient right-to-left shunt during the release phase of Valsalva with passage of venous micro emboli systemically (i.e., paradoxic embolism). It is estimated that 70,000 strokes are associated with patent foramen ovale annually in the U.S.

Neurological decompression illness is more frequent in divers with right to left shunt than those without.This right to left shunt allows bubbles to bypass the filter and bubbles may then embolise neurological tissues [51,53].



Diagramatic representation of opening of PFO during valsalva release

Del sette and colleagues showed that right to left cardiac shunt where present in 41% patients with migraine with aura compared with 16 % of controls[9]. Anzola of and colleagues obtained similar results [20,16] which showed

that right to left shunts were present in 48% of individuals with migraine with aura compared with 20% in controls. Dalla Volta and colleagues showed that patent foramen ovale was present in 61.9% of patients with migraine with aura and 16.2% of patients with migraine without aura[7]. These observations suggest that an atrial shunt merely fascilitates migraine with aura. It may cause aura by micro embolisation or vasoactive chemicals which bypassed the lungs.

Peter Wilmshurst and Simon Nightingale postulated a venous agent possibly 5 Hydroxy tryptamine bypass the lung filter through persistent foramen ovale and that generates attacks migraine[52]. Peter Wilmshurst and Simon Nightingale concluded that individuals with a history of migraine with aura should be screened for the presence of right to left shunt before they dive or be exposed to subatmospheric decompression[51].

Azarbal et al. retrospectively evaluated changes in migraine patterns in 89 patients with cryptogenic stroke following ASD/PFO closure and showed a 42% migraine prevalence, with 62% having migraine with aura[5]. At one-year follow-up, there was complete resolution of migraine, starting immediately postprocedure, in 60% of patients (75% in those with aura), and improvement in the remaining 40%. Reisman et al. in a study of similar methodology, treated 162 patients with presumed paradoxic embolism and showed a 35% prevalence of migraine, with 68% having migraine with aura[29].

Complete resolution of migraine was noted in 56% and significant improvement in 14%. These two studies corroborate several recent studies showing similar results, with enhanced improvement in patients with migraine with aura [15,16,22,30,37,55].

Effect of PFO Closure Devices on Migraine Headache

Authors (Ref.)	# of Subjects	# (%) With Migraine	Mean Follow-Up	% Improved	Resolution %
Wilmshurst et aI. (53)	37	21 (57)	17 months	48	38
Morandi et al. (11)	17	17 (100)	12 months	29	59
Post et aI. (30)	66	26 (39)	6 months	84	N/A
Schwerzmann et aI. (37)	215	47 (22)	24 months	N/A	83
Azarbal et al. (1)	89	37 (42)	6 months	60	16
Reisman et al. (29)	162	57 (35)	12 months	56	14
Total	**586**	**205 (35)**		**55**	**42**

N/ A = not applicable; PFO = patent foramen ovale.

Phases of migraine

Four different phases of migraine recognised are prodrome, aura, headache(during which associated symptoms are experienced) and recovery or resolution phase.

1. **Prodrome**

The prodromal symptoms may be present in some patients during some attacks but it is not universally present. It may be difficult to identify but with patients are specifically asked about the premonitory symptoms in the 24 hr period before the headache, they often describe symptoms such as irritability, excitability, hyperactive or depression and helpful in diagnosis. Premonitory symptoms may also include hypoactivity, craving for certain particular foods, repetitive yawning, neck tightness and so on.

2. **Aura**

The diagnosis is usually evident by careful history alone, although there are secondary mimics including carotid dissection, seizure and arteriovenous malformations.

Visual aura is the most common type presenting as fortification spectrum or scotoma. Next in frequency are sensory disturbances in the form of pins and needles(positive features) and / or numbness (negative features). Less frequent are speech disturbances – dysphasia.

Symptoms usually follow one another in succession begining with visual than sensory symptoms and dysphasia but reverses and other orders have been noted. After an initial consultation, use of an aura diary may help in

diagnosis.

If the aura begins after the age of 40, when negative features are predominant (hemianopia) or when aura is prolonged or very short,other causes should be ruled out espescially transient ischemic attacks.

3. **Headache phase**

About 60% of headaches in migraines are predominantly unilateral. It is important to note that headache occur alternating side. It can start on one side and go to other side during the same attack or it may be different sides in different attacks. Approximately 40 – 45 % of patients may have bilateral headaches. A pulsating headache is very characteristic of migraine but not always diagnostic.

Migraine headache is of moderate to severe intensity unlike tension type headache which is usually mild. Migraine headaches interfere with patients ability to function whereas the majority of tension type headaches do not.

Migraine headache is aggravated by any activity or posture that increases intracranial pressure such as coughing, sneezing, bending down or climbing stairs or physical exercises. Therefore typically patients with

migraine generally do not like to move and prefer to lie down without much movement of the head or body. The location of the headache does not help in the diagnosis. Headache even though typically in the temples, could be frontal, occipital or suboccipital. It may be unilateral or bilateral. Associated symptoms are also important. Anorexia, nausea and / or vomiting often accompany migraine headache and are very helpful diagnostic features. Any condition with increased intracranial pressure or meningeal irritation cause nausea and vomiting but major difference between such disorders and migraine is that migraine is recurrent.

There is visually heightened sensory perception manifested by photophobia, phonophobia and dislikes for smells. Patients seek a dark quiet room. Behaviour of the patient during attacks may be altered. They may be irritable and their memory & concentration may be impaired. When behavioural symptoms occur they may be misdiagnosed as a psychological condition. Depression, fatigue, anxiety, nervousness, irritability and impairment of concentration are common.

Recent research has clearly indicated that significant percentage of patients with migraine would develop cutaneous allodynia of the scalp or extracephalic areas of the body such as limbs. Non painful stimuli becomes uncomfortable or painful. It is the result of central sensitisation of the trigeminal pathways. The allodynic brushing / coombing the hair, wearing

ponytails, and rubber bands, and difficulty lying on the side of headache beacuse of soreness and tenderness of the scalp. The allodynic symptoms in the extracephalic areas include tingling and increased sensitivity as manifested by difficulty wearing neck chains and discomfort on having the blanket touch the body.

The practical importance of recognizing allodynia has recently been pointed out by Berstein et al. . Central sensitization and resultant allodynia begins its process in the first hour of migraine attack and becomes established by 4 hours.The best chance of making a patient pain free with triptan is giving the drug before the allodynic process becomes established . Therefore, early treatment with triptan before allodynia develops is extremely important.

Identification of provocational trigger for a headache is evidence in support of the classification of headache as benign. It is unusual for an organic disorder to show sensitivity to triggers.

Common provocational triggers for migraine

Hormonal triggers	Menstruation, ovulation, oral contraceptive, Hormonal replacement
Dietary triggers	Alcohol, nitrite-laden meat, monosodium glutamate, aspartame, chocolate, aged cheese, missing a meal.
Psychological triggers	Stress, period after stress (weekend or vacation),

	anxiety, worry, depression
physical / environmental	Glare,flashing lights, visual stimulation, fluorescent lighting, odors, weather changes, high altitude
sleep-related triggers	Lack of sleep, excessive sleep
Miscellaneous triggers	Head trauma, physical exertion, fatigue,
Drugs	Nitroglycerin, histamine, reserpine, hydralazine,ranitidine, Estrogen.

Resolution phase

After the headache, the patient often feels tired, washed out, irritable, or restless and can have impaired concentration, scalp tenderness, or mood changes. Some feel unusually refreshed or euphoric after an attack; others experience depression and malaise.

Migraine Variants

Various migraine variants are recognized and classified in the IHS classification.

Familial Hemiplegic Migraine (FHM)

FHM characterized by familial occurrence of hemiparesis associated with migraine like headaches. The aura consists of fully reversible motor weakness and at least one of the following :

(a) fully reversible visual symptoms (including flickering lights, spots, or lines) or negative features (i.e., loss of vision);

(b) fully reversible sensory symptoms including positive symptoms (i.e., pins and needles) or negative features (i.e., numbness); and

(c) fully reversible dysphasic speech disturbances. Usually the auras develop gradually over 5 minutes and different auras occur in succession over a period of 5 minutes or more each aura symptom usually lasts >5 minutes, but <24 hours. The most important diagnostic feature is that at least one first or second-degree relative has had attacks of similar kind. So the familial occurrence of such attacks with hemiparesis is diagnostic. It may be difficult to distinguish weakness from sensory loss.

Now genetic data have allowed a more precise definition of FHM than previously. Specific genetic subtypes of familial hemiplegic migraine have been identified.

In FHM 1, there are mutations in CACNA1A gene on chromosome 19 and in FHM2, mutations occur in ATP1A2 gene on chromosome 1q21-23 .

It has been shown that FHM 1 very often has basilar type symptoms in addition to the typical aura symptoms and that headache is virtually always present. During FHM1 attacks disturbances of consciousness (sometimes including coma, fever, Cerebrospinal fluid pleocytosis, and confusion) can occur. FHM1 attacks can be triggered by (mild) head trauma. In approximately 50% of FHM1 families, chronic progressive cerebellar ataxia occurs independently of the migraine attacks. FHM is very often mistaken for epilepsy and unsuccessfully treated as such.

Sporadic Hemiplegic Migraine

In sporadic hemiplegic migraine, attacks occur with a migraine aura including motor weakness usually on one side without a family history of similar illness in first - or second degree relatives. The main manifestation is fully reversible motor weakness, sometimes associated with visual or sensory or dysphasic aura. Studies have shown that sporadic cases occur with approximately the same prevalence as familial cases. Attacks have the same clinical characteristics as those of familial hemiplegic migraine.

Sporadic cases always require neuroimaging and other tests to rule out other causes. Lumbar puncture is also necessary to rule out pseudomigraine with temporary neurological symptoms and lymphocytic neutrocytosis in the

Cerebrospinal fluid.This condition is more prevalent in males and often associated with transient hemiparesis and aphasia.

Basilar- Type Migraine

In basilar-type migraine, the migraine aura symptoms clearly originate from the brain stem and/or from both hemispheres simultaneously, but there is no motor weakness. The aura in basilar - type migraine consists of at least two of the following fully reversible symptoms, but no motor weakness a) dysarthria, (b) vertigo, (c) tinnitus, (d) hyperacusia, (e) diplopia, (f) visual symptoms simultaneously in both temporal and nasal fields of both eyes, (g) ataxia, (h) decreased level of consciousness, and (i) simultaneously bilateral paresthesias. All the auras developed gradually over 5 minutes or more, and different aura symptoms may occur in succession over more than 5 minutes. Each aura symptom lasts usually 5 minutes or more and usually less than 60 minutes. The headaches following these are similar to those seen in patients with migraine without aura. In all these types of migraine, any other cause that could produce similar symptoms have to be ruled out. Basilar-type attacks are mostly seen in young adults. Many patients who have basilar type attacks also report attacks with typical aura. Differential diagnosis of basilar-type of migraine is anxiety and panic attacks, which may cause some of the symptoms just described. The associated severe headache and the development pattern of the aura symptoms differentiates basilar-type attacks

from a panic attack causing similar symptoms.

Childhood Periodic Syndromes: Migraine Precursors

Cyclical Vomiting

Cyclical vomiting occurs in recurrent episodes, usually stereo typed in individual patients, of vomiting and intense nausea. The attacks are associated with pallor and lethargy. There is complete resolution of symptoms between attacks. The clinical features of this syndrome resemble those found in association with migraine headaches.

Multiple threads of research over the last years have suggested that cyclical vomiting is a condition related to migraine and may run as a precursor of migraine in children.

Abdominal Migraine

This is an idiopathic recurrent disorder seen mainly in childern and characterized by episodic midline abdominal pain manifesting in attacks lasting 1 to 72 hours with normality between episodes. Pain is moderate to severe in intensity and associated with vasomotor symptoms of nausea and vomiting. The abdominal pain has the following characteristics: (a) mid-line location, periumbilical or poorly localized, (b) dull or just sore quality, and (c) moderate or severe intensity. During the abdominal pain, at least two of the

following symptoms may be present: (a) anorexia, (b) nausea, (c) vomiting, and (d) pallor. All other organic conditions have to be ruled out in order to make a positive diagnosis. Pain may be severe enough to interfere with normal daily activities. Children may find it difficult to distinguish anorexia from nausea. The pallor is often accompanied by dark shadows under the eye. In a few patients, flushing is a predominant vasomotor phenomena. Most children with abdominal migraine develop migraine headache later in life.

Benign Paroxysmal Vertigo of Childhood

The probably heterogeneous disorder of benign paroxysmal vertigo of childhood is characterized by recurrent brief episodic attacks of vertigo occurring without warning and resolving spontaneously in otherwise healthy children. Neurological examination and audiometric and vestibular function tests are normal. The attacks are usually short lived. It may be associated with nystagmus or vomiting. Unilateral throbbing headache may occur in some patients.

Retinal Migraine

Retinal migraine manifests as repeated attacks of monocular visual

disturbances including scintillations, scotoma, or blindness, associated with migraine headache. These are fully reversible episodes and always confined to the monocular visual field. Headache would meet the criteria for migraine without aura. The ophthalmological examination is usually normal between attacks. Other causes of transient monocular blindness have to be ruled out. One of the confusing issues is that some of the patients who complain of monocular visual disturbances may infact have hemianopia. Some cases without headache have been reported, but in these cases, their migrainous nature cannot be ascertained. Other causes of transient monocular blindness (amaurosis fugax), such as optic neuropathy or carotid dissection, must be excluded.

COMPLICATIONS OF MIGRAINE

Chronic Migraine

Chronic migraine is defined as migraine occurring for 15 or more days per month for more than 3 months in the absence of medication overuse

Status migrainosus

Status migrainosus are debilitating migraine attacks lasting more than 72 hours. Usually these occur in patients with migraine without aura who may have unremitting headache for more than 72 hours. They are of severe

intensity. These are the type of patients who end up in the emergency room for treatment. Note that status migrainosus can be caused by medication overuse and that factor has to be evaluated. The patients who are in status migrainosus usually are very sick with extreme nausea and vomiting and often dehydrated. Rehydration is an essential part of the treatment.

Persistent Migraine Aura without Infarction

These are patients with aura symptoms persisting for more than 1 week without any radiographical evidence of cerebral infarction. This aura could be visual, sensory, or motor. The visual aura is often bilateral and may last for months or years. It is important to exclude organic conditions like posterior leukoencephalopathy in persistent cases.

Migrainous Infarction

An occasional patient with migraine with aura may develop cerebral infarction with some persistent neurological symptoms. Their attacks are typical of previous attacks of migraine with aura, but the difference is that the aura symptoms persist for more than 60 minutes and neuroimaging would demonstrate an ischemic infarction in the relevant areas. Ischemic stroke in migraineurs can only be made if the infarction occurs during a typical attack of migraine with aura. Increased risk for stroke in migraine patients has been demonstrated in women under the age 45 in several studies. Evidence for

association between migraine and stroke in older women and in men is inconsistent. Younger women with migraine with aura, who are heavy smokers and who are on birth control pills, may increase their risk for cerebral infarction. The usual area of cerebral infarction is the posterior occipital lobe and is typically a wedge-shaped infarction. In patients with migrainous infarction, other predisposing causes for cerebral infarctions, such as cardiac abnormalities, anticardiolipin antibody syndrome, and collagen disease, have to be ruled out.

Migraine- Triggered Seizure

A seizure triggered by a migraine aura can occasionally occur. Migraine and epilepsy are paroxysmal brain disorders. Migraine like headaches are quite frequently seen in the postictal period of an epileptic attack, and sometimes a seizure occurs during or following a migraine attack. This phenomenon, some times referred to as migralepsy, has been described in patients with migraine with aura. This is a rare condition.

Probable Migraine

A previously used term was migrainous disorder . These are attacks missing one of the features needed to fulfill all the criteria for a disorder described in the criteria for migraine with aura and without aura. The prevalence of probable migraine is quite high in the clinical population. They

sould be considered as migraine for all practical purposes and treated as such.

Menstrual Migraine

The International Headache Society recognizes two forms of menstrual migraine (1) pure menstrual migraine without aura and (2)menstrually related migraine without aura.

Pure Menstrual Migraine without Aura

These are migraine attacks in a menstruating woman, fulfilling the criteria for migraine without aura. The attacks should occur exclusively on day 1, plus or minus two, that is, days -2 to +3 of menstruation in at least two out of three menstrual cycles and no other times of the cycle. The first day of menstruation is counted and the preceding day as -1. There is no day zero. For the purposes of classification, menstruation is considered endometrial bleeding resulting from either normal menstrual cycle or from withdrawal of exogenous progesterones as in the case of combined oral contraceptives and cyclical hormone replacement therapy.

Menstrually Related Migraine without Aura

In this entity, attacks in a menstruating woman should occur on day 1 plus or minus two, that is, days minus to plus three of the menstruation in at least two out of three menstrual cycles and additionally at other times of the

cycle. In other words, these patients have migraines around the menstrual time plus other migraines at other times of the month. The importance of distinguishing between pure menstrual migraine and menstrually related migraine without aura is that the hormone prophylaxis is more likely to be effective for pure menstrual migraine. In order to make a certain diagnosis, a respectively documented evidence by a proper diary is necessary.

Menstrual migraines are mostly migraine without aura in a woman who has migraine both with aura and without aura Migraine aura does not appear to be associated with menstruation. Menstrual migraines are usually prolonged, can occur for 4 or 5 days, as long as the menstruation lasts. It is more difficult to treat. Recurrence of headache even with triptans can occur.

Transesophageal contrast echocardiography:

Transesophageal contrast echocardiography is the most sensitive diagnostic test available for detecting a persistent foramen ovale followed by transcranial doppler and transthoracic cardiography(33,9,18).Intravenous saline contrast was given during transesophageal contrast echocardiography. Persistent foramen ovale is judged to be present if any microbubbles seen in left side chambers within 3 cardiac cycles from the maximal right atrial opacification after injection of agitated saline. Agitated saline is prepared by

mixing 0.5 -1 ml of air with 10ml of normal saline through the 2 syringes in the peripheral intravenous line.Injection is performed with and without valsalva maneuver.Cough during injection increase the sensitivity of detecting persistent foramen ovale over that achieved by valsalva maneuver

MATERIALS AND METHODS

In a cross sectional case control study, transesophageal contrast echocardiography was done in 42 migraine patients. Among them 21 patients were migraine with aura and 21 patients were migraine without aura. We perform transesophageal contrast echocardiography and assessed anatomy and size of the shunt lesions. Cardiologist was blinded for migraine history. All the patients were the out patients of the Neurology department.

SITE OF STUDY

Dept of Neurology, Govt. Stanley Medical College & Dept. of Cardiology, Govt. Stanley Medical College

PERIOD OF STUDY : JANUARY 2006 –MARCH 2008

INCLUSION CRITERIA

1. Age > 12 Years
2. Patients who satisfied the criteria of the International Headache Society for Migraine.
3. Patients who are willing to participate in the study & given the written consent.

EXCLUSION CRITERIA

l. Age <12 Years

2. Patients who not satisfied the criteria of the International Headache Society for Migraine.

3. Patients who are not willing to participate in the study & not given the written consent.

TOOLS USED:

1. THE CRITERIA OF THE INTERNATIONAL HEADACHE SOCIETY FOR MIGRAINE.

1.1 ***Migraine without aura***

Diagnostic criteria:

A . At least 5 attacks fulfilling B - D. Migraine days Frequency < 15 days/ month

B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated).

C. Headache has at least two of the following characteristics:

1. Unilateral location
2. Pulsating quality
3. Moderate or severe pain intensity
4. Aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)

D. During headache at least one of the following:

1. Nausea and/or vomiting
2. Photophobia and phonophobias.

E. Not attributed to another disorder

1. 3.1 ***Typical aura with migraine headache***

Description: Typical aura consisting of visual and/or sensory and/or speech symptoms. Gradual development, duration no longer than one hour and complete reversibility characterize the aura which is associated with a headache fulfilling criteria for migraine without aura (1.1)

Diagnostic criteria:

A. At least two attacks fulfilling criteria B . E

B. Fully reversible visual and/or sensory and/or speech symptoms but no motor weakness

C. At least two of the following:

1. Binocular unilateral Homonymous visual symptoms and/or unilateral sensory symptoms including excitation positive features (flickering light, pins and needles) and/or depression negative features (blindness,scotoma or numbness)

2. At least one symptom develops gradually over 5 minutes and/or different symptoms occur in succession.

3. Each symptom lasts >5 minutes and <60 minutes

D. Headache that meets criteria B -D for migraine without aura (1.1) begins during the aura or follows aura within 60 minutes

D. Not attributed to another disorder.

2. MIDAS SCORE

l. How many days in past 3 months -missed work or school because of your headache?

2. How many days in past 3 months-productivity at work or school reduced by half or more because of your headache?

3. How many days in past 3 months-not do household work because of your headache?

4. How many days in past 3 months-productivity at household work reduced by half or more because of your headache?

5. How many days in past 3 months-did you miss family ,social or non work activities because of your headache?

A. On how many days in the past 3 months did you have a headache?

B. On a scale of 0-10, how painful were these headache on average?

Total MIDAS Score: MIDAS Grade- (Grade1minimal -score 0-5,mild II-6-10, moderate III-11-20, severe IV-21+- Sum of lost days due to headache for questions 1-5)

3. TRANSESOPHAGEAL CONTRAST ECHOCARDIOGRAPHY

Transesophageal contrast echocardiography was performed after obtaining informed consent by using multiplane transesophageal echocardiography 5MHZ transducer interfaced with Aloka4000 SSD. Transesophageal contrast echocardiography was performed blind to migraine history.Bubble contrast was produced by pushing about 10 ml Normal saline ,0.5 ml venous blood and 0.5 ml air back and forth between two syringes connected by a three way tap until there were no visible bubbles.This mixture was injected through a 21 gauge butterfly needle into a left anticubital vein.With valsalva manouever transesophageal contrast echocardiography was performed.Shunt is graded according to the number of bubbles.

Small shunts-fewer than 6 bubbles

Medium shunts-6-20 bubbles

Large shunts->20 bubbles

OBSERVATION AND RESULTS

Total no of patients	:	42
Male	:	11
Female	:	31
Female to Male ratio	:	2.8:1
No of Migraine patients with aura	:	21
Male	:	6
Female	:	15
Female to Male ratio	:	2.5:1
No of Migraine patients without aura	:	21
Male	:	5
Female	:	16
Female to Male ratio	:	3.2:1

Age

Range	:	16-55 (years)
Mean ±SD	:	27.35±9.88 (years)
Male	:	29.90±12.10(years)
Female	:	25.5±9.10(years)

t=0.94 p=0.35 NS

Duration of Headache

Range	:	2-10 (years)
Median	:	3(years)
No of Patients with family history	:	10(25%)
Migraine with aura	:	6(29%)
Migraine without aura	:	4(19%)
No of patients with unilateral headache	:	25(60%)
Migraine patients with aura	:	12(57%)
Migraine patients without aura	:	13(62%)
No of patients with autonomic signs	:	40 (100%)
No of patients with severe MIDAS SCORE:		18(43%)
Migraine patients with aura	:	16(76%)
Migraine patients without aura	:	2(10%)
Average MIDAS Score		
Migraine patients with aura	:	22.55±4.44
Migraine patients without aura	:	14.95±4.89
P value=0.001 significant		
Average MIDAS Score	:	18.75±6.01
No of patients with CT Scan abnormalities:		Nil
No of patients with ANA,CRP,RA Factor positivity:		Nil

AGE &SEX DISTRIBUTION

Age group(years)	Migraine with aura			Migraine without aura			Total No .of patients
	Male	Female	Total	Male	Female	Total	
15-24	2	8	10	2	6	8	18
25-34	2	5	7	1	9	10	17
35-44	1	-	1	2	1	3	4
45-54	1	1	2	-	-	-	2
>55	-	1	1	-	-	-	1

MIGRAINE TYPES

Type	Male	Female	Total
Migraine with aura	6	15	21
Migraine without aura	5	16	21

FAMILY HISTORY

Family history	Migraine with aura			Migraine without aura			Total No .of patients
	Male	Female	Total	Male	Female	Total	
Positive	1	5	6	-	4	4	10
Negative	5	10	15	5	12	17	32

MIDAS SCORE SEVERITY

MIDAS Score	Migraine with aura			Migraine without aura			Total No .of patients
	Male	Female	Total	Male	Female	Total	
Minimal	-	-	-	-	-	-	-
Mild	-	-	-	-	5	5	5
Moderate	-	5	5	9	5	14	19
Severe	6	10	16	1	1	2	18

TEE FINDINGS IN MIGRAINE PATIENTS

Sl.No	TEE Findings	Migraine with aura			Migraine without aura		
		Male	Female	Total	Male	Female	Total
1.	Normal	3	11	14	5	15	20
2.	PFO	1	1	2	-	-	-
3.	ASD(0S)	2	3	5	-	-	-
4.	ASD(SVC)	-	-	-	-	1	1
	Total	**6**	**15**	**21**	**5**	**16**	**21**

X2=7.78 p=0.05 SIGNIFICANT

SHUNT LESIONS IN MIGRAINE PATIENTS

Migraine type	PFO	ASD(OS)	ASD(SVC)	Total
Migraine with aura	2 (10%)	5(24%)	-	6(34%)
Migraine without aura	-	-	1(5%)	1(5%)

Correlation of shunt and migraine severity

Migraine Group	Midas Score Severity		
	Mild	**Moderate**	**Severe**
Migraine with shunt	5	18	11
Migraine without Shunt	-	1	7

Correlation of shunt lesions with severity

TEE Finding	MIDAS SCORE GRADING		
	Mild	Moderate	Severe
Normal	5	18	11
ASD(OS)	-	-	5
PFO	-	-	2
ASD(SVC)	-	1	-

DISCUSSION

Out of 42 patients ,21 patients met the diagnostic criteria for migraine with aura and 21 patients met the the diagnostic criteria for migraine without aura. The female to male ratio is 2.8:1.

Age distribution:

The age group ranged from 16 years to 55 years.For the purpose of analysis the study group was divided into five categories.

1.15-24 years 2.25-34 years 3.35-44 years

4.45-54 years 5.>55 years

The maximum distribution of patients were between 15-34 years(33/42).Only three cases belonged to age group above 35 years.The female preponderance more in younger age group 15-34 years(28/34).

Family history

Family history was present in 25% of patients. 29% of migraine with aura patients had family history.But only 20% of migraine without aura patients had family history.

MIDAS Score

MIDAS Score was severe in 76% of migraine with aura patients. But only in 10% of migraine without aura patients,MIDAS Score was severe.

Average MIDAS Score in migraine with aura patients was22.55±4.44. In migraine without aura patients, average MIDAS score was 14.95±4.89. P value was 0.001 which is significant.

In all patients, ANA,CRP and RA Factor were negative and CT scan was normal.

Transesophageal contrast echocardiography findings

Shunt lesion was present in 34% of migraine with aura patients.Osteum secundum type of atrial septal defect was present in 24% of migraine with aura patients.Patent foramen ovale was present in 10% of migraine with aura patients.Only 5% of migraine without aura patients had atrial septal defect of sinus venosus type. P value is 0.05 which is significant.So, transesophageal contrast echocardiography is of immense value in the workup of migraine with aura patients.Since the shunt lesion was present in 34% of patients,closure of the shunt may result in remission of migraine symptoms.

Del sette and colleagues showed that right to left shunts were present

in 41% of migraine with aura patients compared with 16% of controls[9].In our study shunt lesion was found in 34%of migraine with aura patients.The increased prevalence of right to left shunts in migraine with aura patients suggests a causal relationship.

Magalhaes E et al concluded migraine with aura occurred more in atrial septal defect cases and atrial septal defect increase the frequency of migraine crises[2].In our study Osteum secundum type of atrial septal defect was present in 24% of migraine with aura patients.,In individuals with a right to left shunt, a lower dose of venous trigger substances may be needed to induce migraine because the shunt permits the pulmonary filter to be bypassed.[5,45,49].Schwerzmann.M et al found shunt size is larger in migraine with aura patients than control subjects[36].

Anzola and colleagues found the prevalence of patent foramen ovale was 48% in migraine with aura patients,23% of migraine without aura patients and 20% in control subjects[4].Domitrz et al found the prevalence of patent foramen ovale was 53% in migraine with aura patients,25% of migraine without aura patients and 20% in control subjects[10].Dalla volta G et al study showed the patent foramen ovale was present in 61.9% in migraine with aura patients,16.2% of migraine without aura patients and 36.8% in cluster headache patients[7].Schwerzmann.M et al found the patent foramen ovale was present in 47% in migraine with aura patients, and 17% of control

subjects[36].But Patent foramen ovale was present in 10% of migraine with aura patients in our study. Patent foramen ovale was not present in any of migraine without aura patients in our study.

Atrial septal defect of sinus venosus type was observed only in 5% of migraine without aura patients in our study.

We analysed the presence of shunt lesions and severity of MIDAS score. MIDAS score was severe in all migraine with aura patients who had osteum secundum type of atrial septal defect and patent foramen ovale . MIDAS score was moderate in migraine without aura patients who had sinus venosus type of atrial septal defect.

In migraine with aura patients who had shunt lesion ,closure of the shunt may ameliorate migraine symptoms or may improve by reduction in frequency and severity [3,5,11,15,29,37,46].In our study one patient with migraine with aura who had osteum secundum type of atrial septal defect,had undergone closure of atrial septal defect.He was followed up prospectively for one year.He was symptom free after the closure.MIDAS Score before closure was 50 and after closure was 2.So,the closure of shunt has a role in ameliorating symptoms in patients with migraine.However,further larger prospective randomized trials are needed to establish the role of closure of the shunt in managing migraine patients[5,11,37].

CONCLUSION

1. The female to male ratio in our study was found to be 2.8:1.Female predilection was more in younger age group <35 years.
2. Shunt lesion was present in 34% of migraine with aura patients. But only in 5% of migraine without aura patients.
3. Osteum secundum type of atrial septal defect was found to be the most common shunt lesion in our 24% of migraine with aura patients.
4. Patent foramen ovale was found in 10% of migraine with aura patients.
5. Atrial septal defect of svc type was noticed in only 5% of migraine without aura patients.
6. Statistically significant number of migraine with aura patients had shunt lesions.
7. Transesophageal contrast echocardiography is valuable in patients with migraine more so in aura patients to detect shunt lesions.
8. Closure of the shunt in patients with migraine may ameliorate symptoms.

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PROFORMA

Name : Age: Sex:

Address : I.P.No :
Neuro No :

H/O Headache Duration : Frequency :

Periodicity :

Unilateral/Bilateral

Quality of pain : Severity :

Aggravating factors:

Aura: yes/no-
Visual/sensory/Motor/Olfactory/Gustatory

GI Autonomic symptoms:

Nausea :

Vomiting :

Diarhoea :

Abdominal cramps:

Sensory hyperexcitability:

Photophobia:

Phonophobia: Osmophobia:

Unilateral cranial autonomic symptoms :

Lacrimation :

Conjunctival injection

Nasal congestion :

Rhinorhoea :

Systemic:

Faintness :

Fever :

Palpitation :

Anorexia :

Polyuria :

Postural hypotension:

Visual loss :

Memory loss:

Loss of consciousness:

Fits:

Weakness: Sensory loss:

Unsteadiness while walking:

Family history

MIDAS Score:

1. How many days in past 3 months –missed work or school because of your headache?

2. How many days in past 3 months-productivity at work or school reduced by half or more because of your headache?

3. How many days in past 3 months-not do household work because of your headache?

4. How many days in past 3 months-productivity at household work reduced by half or more because of your headache?

5. How many days in past 3 months-did you miss family ,social or non work activities because of your headache?

A. On how many days in the past 3 months did you have a headache?

B. On a scale of 0-10,how painful were these headache on average?

Total MIDAS Score-

MIDAS Grade-

(Grade1minimal/frequent -score 0-5,mild II-6-10,moderate III-11-20,severe IV-21+- Sum of lost days due to headache for questions 1-5)

Any systemic disease:Diabetes mellitus/Hypertension

Medication use

Complete hemogram:

ESR :

ANA :

RA Factor :

CT Scan Brain:

ECG :

Transesophageal contrast echocardiography: