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MIGRAINE A CAUSE OF INTENSE THROBBING; A MINI REVIEW

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ABSTRACT

Migraine has become a common pain disorder now a day, which greatly affects the normal and routine life of the people. A migraine headache can be the cause of intense aching or a pulsating feeling in an area of the head and is generally accompanied by nausea, vomiting. Patient becomes extremely sensitivity to both light and sound. Its outbreaks can cause substantialdiscomfort for hours to days. In some cases of the migraines are also associated with sensory warning symptoms like flashes of light, blind spots, or tingling in arm or leg. Migraine is treated with various therapy plans including pain killers and some time in combination with anti-emetic to control the nausea and vomiting associated with migraine. Purpose of writing of this short communication is to provide information about various aspects of migraine.

Keywords: Migraine, Pathophysiology, Pervasiveness of migraine, Treatment plain

INTRODUCTION

Migraine is very common these days. Its consequences are much greater than are expected. Routine life is disturbed to a greater extent by headache due to partial or complete disability of victims. Total activity is limited in working and free hours because

victim cannot enjoy or work properly. There is also socioeconomic impact of headache. But still it is one of the neglected diseases [1].

The Background Picture of the Migraine

Migraine is a complex type of headache, which is known from antiquity. It is actually a mysterious neurobiological play in which the protagonists are the brain and it's nearby surrounding tissues. In the ancient years the main contemplation about migraine was solely as a vasculature disorder. But in yesteryears persuasive evidences are increasing that have led to the apprehension that both central and peripheral nervous systems are involved in the inexplicable pathway of migraine attack. These evidences if broadly construed, support the involvement of the trigeminovascular system and the cerebral cortex [2, 3].

In definition, it is a disorder of recurrent headaches. The duration of headache attacks may exceed from 4 hours to as long as 72 hours. This atypical headache occupy unilateral location, throbbing type sensation, specific pain which may be mild to as worst as unbearable, and clear-cut disturbance of routine work. These characteristic features of migraine are also associated with at least one of the following symptoms: that are stomach revulsions, ligyrophobia or photalgia[4].

Migraine Pathophysiology

During the migraine attack, the dilation of extra-cranial blood vessels that supply the Dura matter occurs. This causes the increase blood flow that leads to the expansion of small meningeal vessels in the cerebellum. Due to this stretching, nociceptors in the walls of these small blood vessels are activated thus causing throbbing pain in the localized area. This dilation may also activate the trigeminal system by inducing the neuronal inflammation response through the release of vasoactive peptides. These are the leading factors in the pain and other symptoms associated with migraine.

Serotonin (5-Hydroxy tryptamine (5-HT)) with its receptors is strongly believed to play its role in pathophysiology of migraine. It is related to the regulation of some basic body functions such as sleep, mood, hormonal balance etc. So, its role in migraine can hardly be isolated. Of all serotonin receptors, 5-HT_{1B} and 5-HT_{1D} are thought to be associated with migraine, the first being associated with smooth muscles of intracranial blood vessels and second with the trigeminal nerve fibers [5].

To open the mystery of migraine headache, it has been studied very broadly. But still the clear phenomenon is not known. There was found association of some autonomic

nervous system (ANS) with migraine [6]. Evidence of this hypothesis was made by the autonomic symptoms for instance nausea, vomiting or diarrhea, vasoconstriction of peripheral blood vessels, erection of hairs, sudation, light sensitivity etc. Reasons of these symptoms may be elaborated by the decrease in the sympathetic norepinephrine levels which may be followed by increase in sympathetic co-transmitters such as dopamine, prostaglandins and adenosine [7]. Sanya with coworkers, worked on parasympathetic baroreflex responses in migraine patients and concluded that ANS has some connection in the pathophysiology of migraine [8].

5-HT_3 receptors on the neurons show the excitatory effect of serotonin. Location of these receptors is the pain and nausea-modulating areas in the central nervous system (CNS). Accordingly, activation of these receptors intercede the painful and emetic effects of serotonin [9]. Fozard suggested in early 1980s that $5HT_3$ receptors might be related to migraine, thus creating nauseatic responses. Evidence to the involvement of $5HT_3$ receptors, and increase in dopamine levels during migraine attacks is the fact that there was decrease in the migraine associated symptoms when benzamide D2 and selective $5HT_3$ receptors antagonist was used [10].

Throughout the 20^{th} century, two different theories of migraine compete each other. One of them is vasogenic theory and the other is neurogenic. According to vasogenic theory the reason behind the migraine headache was reversible vasodilation of the intra and extra cerebral blood vessels. This vasodilation resulted in mechanical depolarization of the neurons that sense pain within these vessels. According to this theory, the aura of migraine was due to momentary vasoconstriction induced hypoxia of blood. Reporters were confident about the theory because the migraine headache was cured by vasonstrictor drugs and was persistent with the vasodilators. This theory was in more consideration until 1980s.

According to neurogenic theory, this change in vasculature was the result of neuronal changes or dysfunction. Vindicator of the theory was the occurrence of various neurologic symptoms with the single aura and the multiple symptoms cannot be localized to single vascular event. Also the de-escalation of the symptoms with the use of non vasoconstrictive drugs favors the theory. The neuroimaging studies conducted during attack revealed the fact that the blood flow in the cerebral vessels was not less to

the extent to induce hypoxia and the vasodilation was preceded by headache [11].

Recently it is stated that abnormality of calcium channels may be the cause of hyper excitability of neurons. The genetically altered genes of calcium channels may affect the release of inhibitory or excitatory neurotransmitters at the presynaptic nerve endings. It is also hypothesized that due to mutations threshold of response to triggers' is reduced.

Some evidences from spectroscopic study also show the lower concentrations of magnesium in migraineurs[2].But in the fact, none of the theory can explain individually all the migraine symptoms. So migraine can be explained in the way that some neuronal events cause vasodilation which can stimulate nociceptors and further nerve activation. This may be called as "disorder of brain" that cause neurovascular headache [4].

Pervasiveness of migraine

In the American Migraine Study II, about 20,000 questionnaires were mailed to US households and it was found that among female population 18.2% and among male population 6.5% suffer from migraine. Approximately 23% of US households have one migraineur as a minimum. Whites are more susceptible to the disease as compared to blacks. Socioeconomically migraine occurrence was lower in families having good financial status. Incidence also increases after 12 years of age upto40 and start decreasing after it. 53% of migraineurs reported significant mutilation in activities due to severe headache and even require bed rest. 31% of respondents reported to miss a day of activity in last 3 months and 51% reported that their working efficiency was reduced to 50% due to migraine. It was concluded that increase in the number of migraine sufferers from 23.6 million in 1989 to 27.9 million in 1999 was comparable with the population growth, so overall burden of the disease remained stable in this decade [12].

Underlying symptoms

Up to 25% of the sufferers experience some momentary focal neurological symptoms with headache including visual disturbance, unilateral paresthesias, motor symptoms, and some autism like symptoms [11].

Frequency of symptoms

According to the American Migraine Study II, it was concluded that the occurrence of allied symptoms is usual with migraine headache. Among the prevalence light and sound sensitivity were more common that is photophobia experienced by 80% and phonophobia experienced by 76% of patients, nausea being experienced by 73%

and vomiting by 29% of patients. This study also revealed that women population is more susceptible to these concomitant conditions [13].

Treatment options

Changing the lifestyle of migraineurs can subside the worsening of symptoms or even delay an attack. Educating this special population to sleep, eat and exercise regularly and avoiding the stressful circumstances can help them. In short; having regularity in habits is advantageous for them.

Regarding drugs, prophylactic and treatment therapies are used for migraine. Prophylactic treatment options require appropriate patient counseling. Drugs used for prevention therapy include β-Adrenergic–receptor antagonists, Serotonin antagonists, valproate, and selective serotonin reuptake inhibitors (SSRIs) etc.

Among the treatment plans, the analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), ergot derivatives and triptans are frequently used. Ergot derivatives and triptans are used due to their significant vaso-constrictive effect [4].

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CONFLICT OF INTEREST

Authors have no conflict of interest

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