



A Case Report: Overview and Therapy of Migraine with Photophobia

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ABSTRACT

Background. Migraine is a recurring headache with attacks lasting 4-72 hours. Characteristic of Migraine are unilateral, throbbing with moderate or severe intensity, worsening with routine physical activity and accompanied by nausea and/or photophobia and phonophobia. This article aimed to present overview and therapy of patient with migraine accompanied by photophobia.

Case Presentation. A 25 year old female patient who came to the Hospital polyclinic was examined, with a diagnosis of migraine with aura and photophobia. Clinical diagnosis was made based on the history and physical examination. The history shows headache, especially on the right side of the head, pain like throbbing and pulling, continuous, lasting approximately thirty minutes, headache occurs and increases when seeing bright lights (photophobia), feeling stress or smelling cigarette smoke. Patient also sometimes saw glare when the migraine came with severe intensity.

Conclusion. Migraine with Aura and Photophobia can be happened if the pain intensity was moderate to high, and difficult to heal completely without avoid the triggered factors of the migraine.

Keywords: Migraine, Aura, Photophobia, Severe, Therapy

BACKGROUND

Cephalgia is pain felt in the head or is an uncomfortable sensation felt in the head area.¹ Headaches are classified according to the International Headache Society into primary and secondary headaches. Primary headaches are Tension Type Headache, migraine, cluster Headache and other primary headaches, for example hemicrania continua. Primary headaches account for almost 90% of all headache complaints. Headaches can also occur secondary, which means they are based on a disease.^{1,2}

Migraine is a complex and recurring disorder that is one of the most common health complaints. In the United States, more than 30 million people suffer from one or more migraines per year. About 75% of all people who experience migraines are women. The term migraine comes from the Greek word hemicrania. Migraine was previously thought to be a vascular phenomenon caused by intracranial vasoconstriction followed by rebound vasodilation. However, currently, neurovascular theory describes migraine primarily as a neurogenic process with secondary changes in cerebral perfusion associated with neurogenic inflammation.^{3,4}

A genetic component to migraines is demonstrated by the fact that approximately 70% of patients have a biological family history of migraines. In addition, various environmental and behavioral factors can trigger migraine attacks in people with predisposing factors. Migraine is characterized most often by unilateral headache that is moderately severe, throbbing, and worsens with activity. Migraines can also be associated with a variety of visual or sensory symptoms, which most often occur before the headache, but can also occur during or after the headache, and are also known as auras. Most commonly, auras consist of visual manifestations, such as scotoma, photophobia, or visual disturbances (e.g., bright zigzag lines).^{5,6}

Headaches can also be associated with weakness. This form of migraine is called hemiplegic migraine. However, in practice, migraine headaches can be unilateral or bilateral and may occur with or without aura. In the International Headache Society's most recent categorization, headaches previously described as classic migraines are now known as migraines with aura, and migraines described as common migraines are now called migraines without aura. Migraines without aura are the most common, accounting for more than 80% of migraines. Migraine diagnosis is clinical, based on criteria established by the International Headache Society. A complete neurological examination should be performed during the first visit, to rule out other disorders; Findings are usually normal in migraine sufferers. Neuroimaging is not necessary in typical cases, but other diagnostic surveys may be used to guide management.¹

In general, migraine was classified into two by the International Headache Society (IHS) in 1988, namely migraine without aura or common migraine and migraine with aura or classic migraine. The most common one is migraine without aura, which is around 80% of all migraine sufferers.^{2,3} The first is migraine with aura or classic migraine which begins with a focal neurological deficit or impaired nerve/aura function. These deficits are in the form of visual and sensory smells such as seeing wavy lines, bright lights, dark spots, followed by unilateral headaches, nausea and sometimes vomiting. These events are generally sequential and the manifestation of pain usually lasts no more than 60 minutes. The second is migraine without aura or common

migraine, namely pain on one side of the head and is pulsatile accompanied by nausea, photophobia and phonophobia, the intensity of the pain is moderate to severe. Pain is worse during activity and lasts for 4 to 72 hours.²

Overall, the clinical manifestations of migraine sufferers vary between individuals. There are four general phases that occur in migraine sufferers. But not everything has to be experienced by each individual.⁶ First is the Prodromal Phase. This phase is experienced by around 40-60% of migraine sufferers. Symptoms include changes in mood, irritability, depression or euphoria, feeling weak, sleeping excessively and wanting certain types of food. These symptoms appear several hours or days before the headache. Second is the Aura Phase. Aura of complex focal neurological symptoms that precede or accompany a migraine attack. This phase appears gradually over 5-20 minutes. Aura can be a motoric, sensory, visual sensation or a combination of these. Visual aura occurs in 64% of patients and is the most common neurological symptom. The aura in migraines usually disappears for a few minutes and then headaches appear. Third is the Headache Phase. Migraine headaches are usually throbbing, on one side or unilateral and often start in the frontotemporal and ocular areas. After 1-2 hours the headache spreads diffusely posteriorly. Attacks last 4-72 hours in adults and in children usually 1-48 hours. The intensity of the pain is moderate to severe and interferes with daily activities. The last is the Postdromal or recovery Phase where the patient feels tired, irritable, concentration decreases and mood changes occur. Patients can fall asleep for long periods of time.^{7,8,9}

CASE PRESENTATION

A 25 year old female patient went to the hospital polyclinic with the main complaint of headache since three days ago, pain especially on the right side of the head. The pain felt like throbbing and pulling, continuously, the pain lasted for approximately 30 minutes, headaches arose and increased when thinking about something too hard. Sometimes when a headache comes, glare could also be seen. Pain was reduced by resting or sleeping. The patient had more than 4 headaches in 72 hours. The patient was nauseous and vomited once, the vomiting did not spray, it contained what she had eaten. The patient's appetite has decreased since being sick. The patient has had frequent headaches since she was a teenager. From the time she was a teenager, the patient often suffered from headaches that came and went, and felt increasingly frequent and more severe if the patient was stressed and had a lot on his mind. Every time she has a headache, the patient went to sleep and after waking up the headache disappeared. There was no history of hypertension in the patient. The patient's younger brother complained of the same headache as the patient. The patient was a new employee at a private company in the journalism sector so she was often pressed for deadlines, with light to medium daily activities. When patient was asked between a score of 1 to 10, where 1 is no pain at all and 10 is the most pain, what is the perceived score? The patient answered 8 to 9.

From the physical examination, it was found that the patient was conscious, there were no signs of meningeal stimulation, there were no signs of increased intracranial pressure, and other neurological examinations were within normal limits. The patient did not get laboratory tests. Based on a complete history and physical examination, the patient was diagnosed as migraine with aura and photophobia et causa idiopathic. The patient was given treatment in the form of pharmacological treatment, Ibuprofen 3 x 400 mg (orally) and Cafergot 3x1 tab (orally). Next, the patient was asked to rest and avoid trigger factors such as excess activities, stress and especially seeing excessive bright light, for example from a laptop or room lights that are too bright.

DISCUSSION

A 25 year old female patient who came to the Hospital polyclinic was examined, with a diagnosis of migraine with aura and photophobia. Clinical diagnosis was made based on the history and physical examination. The history showed headache since three days ago, pain especially was felt on the right side of the head, pain was like throbbing and pulling, continuous and pain lasting approximately thirty minutes. Headache occurred and increased when seeing bright lights, smelling cigarette smoke and with increased physical activities. The pain decreased with rest or sleep. The patient had more than 5 headaches in 72 hours. The patient was nauseous and vomited once, the vomiting did not spray, it contained what she had eaten. The patient had suffered from headaches since she was a teenager. Since then, the patient had frequently suffered from headaches, coming and going, becoming more frequent and more severe when the patient was stressed. Every time she was sick, the patient slept and the patient's headache would disappear.

The patient's sign and symptoms meet the Diagnostic Criteria for Headache based on PERDOSSI 2011 adapted from the IHS (International Headache Society), namely headaches that occur more than 5 times in 4-72 hours. Headache meets 2 of the following characteristics: unilateral, throbbing, moderate or severe intensity, worsens with physical activity.¹⁰ In this patient, unilateral headache was found, with moderate to severe intensity, the headache increased with stress and physical activity. During headaches accompanied by one of the following: nausea and/or vomiting, photophobia and phonophobia. These symptoms arose because there is hyperactivity in the brain's electrical impulses which can increase blood flow in the brain, resulting in widening of the brain's blood vessels and an inflammatory process occurs. This widening and inflammation causes pain and other symptoms, such as nausea and vomiting. The more severe the inflammatory process is, the more severe the headache or migraine will be. In this patient, nausea, vomiting and photophobia were found. From a pain intensity score of 1 to 10, the patient felt pain of 8 to 9 with a moderate-severe interpretation. Migraine is often accompanied by photophobia if the intensity of the pain felt is moderate to severe.¹¹

The patient also has a family history of migraines, which was the patient's own sister. A genetic link to migraine has long been known, although no consistent Mendelian inheritance pattern has been found. This shows that there are varied inheritance patterns and the possibility of genes. It is known that genetic factors play a role in the emergence of migraines which interact with environmental factors in a multifactorial pattern. A clear pattern of inheritance is found in familial hemiplegic migraine, namely the subtype of migraine with aura. From the physical examination, it was found that the patient was conscious, there were no signs of meningeal stimulation, there were no signs of increased intracranial pressure and other neurological

examinations were within normal limits. The patient did not get laboratory tests since the diagnosis of Migraine with aura and photophobia could be made by complete history and physical examination.

The specific management given to the patient was administration of Ibuprofen 3 x 400 mg. Special therapy for patients was given cafergot, which is a group of ergotamine combined with caffeine. Ergotamine stimulates or blocks alpha adrenergic and serotonergic receptors. For example, it stimulates 5HT1 receptors, especially 5HT1D and blocks alpha receptors (alpha blockers) with a mild vasodilation effect. This property is controlled by its strong vasoconstrictive power of cerebral and peripheral arteries based on its antiserotonin power (5HT1 blockade).^{11,12}

After that, patients are given education to rest and carry out stress management. Special therapy for patients is given cafergot, which is a group of ergotamine combined with caffeine. Ergotamine stimulates or blocks alpha adrenergic and serotonergic receptors. For example, it stimulates 5HT1 receptors, especially 5HT1D and blocks alpha receptors (alpha blockers) with a mild vasodilation effect. Furthermore, if a migraine comes, you should sleep, and compress the affected side using cold water with the aim of constricting the blood vessels. Patients are also advised to avoid other trigger factors such as inhaling cigarette smoke or eating certain foods that use monosodium glutamate.^{11,12}

In general, three lines of therapy are recommended for migraines. Drug selection remains based on indications, clinician experience, cost effectiveness, side effects, half-life, affordability and drug availability. First-line therapy is to use oral or intravenous antiemetics, paracetamol, acetylsalicylic acid (ASA), NSAIDs (ibuprofen, naproxen, diclofenac), phenothiazines, intranasal or subcutaneous di-hydroergotamine (DHE), naratriptan.¹¹

Second-line therapy uses antiemetics (intravenous), NSAIDs (eg, intramuscular ketorolac), sumatriptan (subcutaneous), ergotamine, haloperidol, intranasal lidocaine, intranasal opiates, corticosteroids, phenothiazines, or opiates. Next, third-line therapy uses sumatriptan (intranasal), intravenous phenothiazines, barbiturates. The three lines of migraine therapy mentioned above can generally be classified into non-specific acute therapy, namely Analgesics and NSAIDs are the first line acute therapy. Drugs in this group include acetylsalicylic acid (500-1000 mg), diclofenac potassium (50-100 mg), paracetamol (1000 mg), piroxicam SL (40 mg), and tolfenamic acid (200-400 mg). Complemented analgesic efficacy with concurrent administration of metoclopramide (5 mg or 10 mg orally) can be given before or simultaneously with oral analgesics. 9,10 Second are antiemetics PO, IM, or IV) and domperidone (20-30 mg PO or PR). 11,12 Next Specific acute therapies such as triptans and ergot derivatives.¹²

The prognosis for migraines is that they do not occur by avoiding trigger factors and taking regular medication, but if there are trigger factors again, migraines can come back again, especially migraines with moderate-severe intensity like the patient above. In addition, based on research in recent years the risk of suffering a stroke in patients with a history of migraine has increased. About 19% of all stroke cases occur in people with a history of migraine.

Conclusion

Migraine with aura and photophobia can be happened if the pain intensity was moderate to severe, and difficult to heal completely without avoid the triggered factors of the migraine and taking regular medication.

Conflict of Interest

No potential conflict of interest relevant to this article was reported

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