Case Report

A rare case of migrainous left bell's palsy after migrainous right external ophthalmoplegia - A case report

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Abstract

Migraine increased the risk of Bell palsy in the total population. Among migraine patients, between ≥30 and <60 years old are at an increased risk of Bell palsy. A migraine is a primary headache characterized by recurrent headache attacks triggered by various factors. As much as 10% of the global population is thought to experience migraine headaches. It was earlier considered that migraine headaches were triggered by the dilation of cerebral vessels, and the recent evidence supports that migraine attacks can also occur in the absence of vasodilation. According to the researchers, the direct neural effects from the trigeminal nerve to the facial nerve could contribute to the risk of facial palsy among patients with migraine. An alteration of the trigeminovascular function has been suggested to trigger migraines. The neurogenic inflammation of the facial nerve trunk caused by its proximity to the dilated posterior auricular/ stylomastoid/ occipital and superficial temporal arteries during a migraine attack leads to a temporary lower motor neuron type of paresis of the muscles supplied by the facial nerve. We herewith report a rare case of migrainous left Bell's palsy after migrainous right external ophthalmoplegia, treated with Sumatriptan.

Key words

Migrainous Bell's Palsy, Migrainous External Ophthalmoplegia, Neurogenic inflammation, Sumatriptan.

Introduction

Migraine and Bell's Palsy

Migraine increased the risk of Bell palsy in the total population. Among age subgroups, migraine patients ≥30 and <60 years old had an increased risk of Bell palsy [1].

Although the pathophysiologic mechanism of migraines is not completely understood, alteration of the trigeminovascular function has been suggested to trigger migraines [2, 3].

Aura migraines are thought to result from a cortical spreading depression induced by cerebral ischemia and inhibition of neural activity [3, 4]. Prior studies have demonstrated a 2-fold increase in the risk of stroke and cardiovascular diseases in migraine patients [5].

Oxidative stress could also influence both migraine and Bell palsy. Oxidative stress has been shown to occur during migraine attacks and accumulate following chronic or recurrent migraine attacks [6]. A recent study suggested that a migraine is a neuroprotective response to brain oxidative stress [7]. Oxidative stress that occurs in the brain following upregulation of antioxidants, neural proliferation, and mitochondrial defense responses in migraine patients may be a defensive, neuroprotective mechanism. This increase in oxidative stress in migraine patients could affect the risk of Bell palsy. A recent study reported that the serum levels of antioxidants were elevated in Bell palsy patients compared with those in the controls [8].

Adverse emotional or psychological responses to migraine headaches could also increase the risk of Bell palsy. Several common psychological disorders, including depression, anxiety, bipolar disorder, and obsessive-compulsive and attention-deficit/hyperactivity disorders, are increased in migraine patients [9]. Among these psychiatric disorders, depression and anxiety have the strongest association with migraine [9]. Compared with controls, migraine patients have a higher prevalence of depression [10]. These psychological problems also have synergistic effects. For example, depression may mediate anxiety in migraine patients [11]. These emotional disorders may also be a risk factor for Bell palsy. This is supported by the findings from a retrospective cohort study in which in patients with anxiety disorders had a 1.53-fold increased risk of Bell palsy (95% CI=1.21–1.94, P<.001) [12].

Facial palsy has many causes that stem from viral infections (esp. varicella zoster virus), congenital, and traumatic causes [13]. Among them, acute idiopathic peripheral facial palsy, which is known as Bell's palsy, accounts for 60% to 75% of cases [14]. Approximately 11 to 40 per 100,000 persons experience Bell's palsy worldwide each year [15, 16]. The incidence of Bell's palsy is high in middle age and has no sex preference [15]. Bell's palsy is thought to be triggered by viruses, but the pathophysiology is complex, and many other causes have been reported. Vascular ischemia, immunological disorders, inflammation, and autonomic dysfunction as triggering factors are suggested as risk factors for Bell's palsy [15].

A migraine is a primary headache characterized by recurrent headache attacks triggered by various factors [17]. As much as 10% of the global population is thought to experience migraine headaches [18]. The central nervous system disorders and immune system disorders as well as inflammation, genetics, and vascular ischemia are all proposed as possible causative factors to migraine [18].

It was earlier considered that migraine headaches were triggered by the dilation of cerebral vessels, and the recent evidence supports that migraine attacks can also occur in association with cardiovascular disturbances (dilatation of middle cerebral artery, temporal and radial arteris, hypertension) in the absence of vasodilation [19]. Many studies have reported a risk of ischemic stroke in migraine patients [20].

According to the researchers, the direct neural effects from the trigeminal nerve to the facial nerve could contribute to the risk of facial palsy among patients with migraine [1].

The neurogenic inflammation of the facial nerve trunk caused by its proximity to the dilated posterior auricular/ stylomastoid/ occipital and superficial temporal arteries during a migraine attack leads to a temporary lower motor neuron type of paresis of the muscles supplied by the facial nerve [21].

Ophthalmoplegic migraine and retinal migraine

Ophthalmoplegic migraine is very rare [22]. Rarely ophthalmoplegic migraine occurs without headache. No clear genetic pattern is seen. The oculomotor nerve was abnormally thickened and showed enhancement with contrast gadolinium MRI [23, 24]. The pathophysiology of ophthalmoplegic migraine is not known. Microvascular constriction with peripheral cranial nerve ischemia may be the most likely cause.

Diagnostic criteria for ophthalmoplegic migraine

At least two attacks having headaches overlapping with paresis of one or more of cranial nerves III, IV and VI and parasellar lesion ruled out by appropriate investigations.

The headaches usually proceed the ophthalmoplegia by 3 to 4 days. The headache is usually unilateral and may be throbbing or constant but is occasionally bilateral or alternating. It is often of the crescendo type, lasting hours to days. The ophthalmoplegia usually follows, affecting one or more nerves and possibly alternating sides in subsequent attacks. The pupillomotor fibres are usually involved, producing a mydriatic and poorly responsive pupil.

Recently, Leone and colleagues [25] described a young woman who had experienced two episodes of internal ophthalmoplegia, without ptosis or external ophtalmoplegia, associated with headache.

With sixth nerve ophthalmoplegic migraine, paralysis of abduction occurs, and, with trochlear nerve involvement, limitation of downward and inward gaze is present. The headache usually resolves. Usually the ophthalmoplegia resolves within a week. Some residual third-nerve dysfunction may be persist, like slight anisocoria. Differential diagnosis includes, aneurysm, basilar arachnoiditis, and tumors of the brain and myasthenia gravis. MRI or MRA to exclude the possibility of aneurysm and tumors [26].

The finding of an entirely normal MRI in a child with a third cranial.-nerve palsy following a 4 day history of headache, shall suffice for the diagnostic workup.Posterior communicating artery aneurysm, which is best excluded by conventional angiography.Myasthenia is ruled out if the pupil is involved (and actually should not be considered in the presence of pain). The prognosis is excellent. But with recurrent attacks, there may be limitation of extraocular muscle function and may persist as a residual effect. The usual recovery period is 1 to 4 weeks.

Retinal migraine

True incidence of retinal migraine is unknown, and it has a good prognosis. Retinal migraine occurs more frequently than ophthalmoplegic migraine. The phrase anterior visual pathway migraine(AVPM) may be preferable, because on rare occasion other structures are affected.

There are two types of AVPM:
(a). transient monocular blindness(TMB) and
(b). permanent unilateral visual loss, which is less common.

Stereotyped presentation, consistent with retinal or optic nerve hypoperfusion from spasm of the cental retinal or ophthalmic artery. Reduction in central retinal artery blood flow during an attack of ocular migraine and there is selective spasm of the central retinal artery. Retinal artery constriction during episodes of migrainous

transient monocular blindness is also noted [27, 28]. Permanent unilateral visual loss from AVPM is well documented and it is very rare.

Diagnostic criteria for retinal migraine

At least 2 attacks of fully reversible monocular scotoma or blindness lasting less than 60 minutes and confirmed by examination during attack or (after proper instruction) by patient's drawing of monocular field defect during an attack.

Headache follows visual symptoms with a free interval of less than 60 minutes, but it may precede them with normal ophthalmoplegic examination outside of attack, and embolism is ruled out by appropriate investigations. Prophylactic antimigrainous therapy usually suffice.

Familial hemiplegic migraine

Migraine with aura including hemiparesis and at least one first degree relatives has similar attacks. Familial hemiplegic migraine (FHM) is an autosomal dominant condition affects males and females equally. Penetrance of the condition is incomplete: not all mutated gene carriers have FHM [29, 30, 31].

Diagnostic criteria for FHM

Aura includes some degree of hemiparesis and may be prolonged.and at least one first degree relative has identical attacks. During the hemiplegic migraine (HM) aura, motor weakness is always associated with at least one other aura symptom: sensory disturbances (paresthesias, numbness), speech disturbances (dysarthria, reduced speech fluency, paraphasias), or visual symptoms (hemianopia, scintillating scotoma, blurred vision) [31].

The degree of motor defecit is variable, ranging from mild clumsiness to total hemiplegia. Attacks may be right sided or left sided alternating or always involve the same side (40%) [31]. Bilateral symptoms occur in about 25% of patients, one side after the other or both sides simultaneously [31, 32]. Other aura symptoms include, loss of balance, diplopia, tinnitus, partial hearing loss, drop attacks, confusion, or loss of consciousness [32].

The average duration of the aura is 1 to 2 hours, but may range from 10 minutes to several days [31].

Childhood syndromes related to migraine

Common childhood disorders, such as abdominal migraine, cyclic vomiting, and benign paroxysmal vertigo, have been noted to occur in close association with migraine or children with a family history of migraine. Because of their close relationship with migraine, they were called variants of migraine or migraine equivalents. The underlying pathologic process of all these conditions is not clear.

Case report

A 38 year old female was admitted to hospital, with the complaints of difficulty in closing the left eye, watering of the left eye and deviation of mouth to the right side since morning. She was apparently asymptomatic until the onset of difficulty in closing in the left eye and watering from the left eye which was insidious in onset; deviation of mouth to the right side which was insidious in onset and associated with difficulty in chewing and swallowing. She also had a history of unilateral headache with neckpain at mastoid region of one week duration. No history of hyperacusis, alteration of taste sensation, fever, ear pain and weakness in upper and lower limbs.

Past history: She had a history of severe headache on the right side followed by external ophthalmoplegia on right side three years ago, diagnosed as migranous right external ophthalmoplegia and was treated for the same. The patient underwent hysterectomy 7 years ago. No history of diabetes mellitus, hypertension, tuberculosis, asthma, epilepsy, coronary artery diseases, cerebrovascular accidents, thyroid and bleeding disorders. No significant personal and family history.

Drug history: No history of any drug intake.
General examination: Patient was conscious, coherent, cooperative and well-oriented to time, place and person. No Pallor, Icterus, Cyanosis, Clubbing, Koilonychias, Lymphadenopathy and Pedal edema.
Vitals: Temperature: Afebrile; BP: 110/80 mm of Hg in sitting position in right arm; Pulse: 82 beats/min, regular in rhythm with no radio-radial or radio-femoral delay.

CNS examination: All the intellectual functions, motor system, reflexes, sensory system, coordination, station and gait were normal. All the cranial nerves were normal except for the facial nerve which revealed left facial palsy L M N type.
CVS examination: S1 and S2 heard; no murmurs.
Per abdomen examination: The abdomen was soft and non-tender, and there was no organomegaly.
Respiratory system examination: Bilateral normal vesicular breath sounds heard.
Investigations: Hb-13.9g%, RBC count – 4.3 mill/cu mm, WBC count –10,800 cells/cu mm, Platelet count – 3.9 lakh/cu mm. RBS - 260mg/dl. FBS – 153 mg/dl, PPBS – 244 mg/dl. HbA1c – 5.7%. CT Brain – Normal.

Diagnosis
A diagnosis of Migrainous left Bell's palsy with NIDDM and an old case of migraine with external ophthalmoplegia on the right side was made (**Figure – 1**).

Treatment given
1. Tab. Sumatriptan 50 mg PO/BD for 30 days.
2. Tab. Wysolone 40 mg PO/OD for 3 days; 30 mg PO/OD for 3 days; 20 mg PO/OD for 3 days; 10 mg PO/OD for 3 days and 5 mg PO/OD for 2 days.
3. Tab. Acyclovir 800 mg PO/QID for 5 days.
4. Tab. PAN 40 PO/OD Before Breakfast.
5. Tab. Shelcal HD PO/OD
6. Tab. Levocet PO/OD HS
7. Tab. Metformin 500 mg PO/BD
8. Refresh eye drops 2nd hourly (2 drops)
9. Lacryl eye gel over left eye f/b eye patch at night.
10. Facial muscles exercises / physiotherapy.

Figure – 1: Patient's face showing left Bell's palsy.

Discussion

Bell palsy occurred in 0.6% (262/44,902) of the migraine group and 0.5% (903/179,753) of the control group. The adjusted HR of Bell palsy was 1.16 in the migraine group compared with the control group [95% confidence interval (95% CI) = 1.01–1.33, P = 0.34]. Among age-related subgroups, participants ≥30 and <60 years old in the migraine subgroup demonstrated a 1.28-times higher risk of Bell palsy than the control group (95% CI=1.05–1.57, P=.014). Migraine increased the risk of Bell palsy in the total population. Among age subgroups, migraine patients ≥30 and <60 years old had an increased risk of Bell palsy [1].

Nervous connections between the trigeminal ganglion and the cerebral blood vessels identified in experimental animals have been termed as the trigeminovascular system. This system is believed to contain the neurotransmitter peptide called the 'Substance P', which is transported from the ganglion cell bodies to afferent nerve

fibres where there occurs its release in to the wall of the cerebral vessels following depolarization. Dilatation of pial arteries, increased vascular permeability and activation of inflammatory response are the main functions of Substance P [33].

Headache pain from the blood vessels of the piamaterand duramater is transmitted by trigeminal nerve. The possible triggers for this pain are multiple and thought to develop within the brain parenchyma, the blood vessel wall and the blood itself which stimulate the trigeminovascular axons leading to the release of vasoactive neuropeptides. Prolonged pain and hyperalgesia are seen as a consequence following the release of products from activated and injured cells [34].

Migraine is a visceral pain, caused by activation of trigeminovascular system due to release of Substance-P, which induces vasodilation and plasma protein extravasation leading to neurogenic inflammation. Once the trigeminocervical system fibres are sensitized, the trigeminovascular system gets activated which maintains constant hypersensitivity to non-noxious stimulus leading to persistent pain during attack of migraine [35].

The possible mechanisms involved include (1) Vasodilatation, which is the source of pain in migraine; (2) Dilatation does not involve the intracranial vasculature; (3) the extra-cranial terminal branches of the external carotid artery are a significant source of pain in migraine [36]. The cerebral arteries and their distension (usually occurs when triggered), is primarily responsible for headache, of which headache due to septicemia and fever also fall under the same category [37]. The afferent fibres from the cerebral arteries above the tentorium cerebelli enter the brain stem via the trigeminal nerve whereas the fibres from the cerebral arteries below it enter CNS through the upper cervical nerves. However, migraine can also occur due to distension of extracranial branches of the external carotid artery [37].

Controversial regarding the origin of the vascular component of migraine pain, it has been proved that the extra cranial component is the only contributing factor. Intracranial component of the external carotid artery has no role in the pathophysiology of the migraine pain [36].

The headache phase of migraine may develop as the result of an abnormal interaction (and perhaps an abnormal release) of vasoactive neurotransmitters from terminals of the trigeminal nerve with large intracranial and extracranial blood-vessels. These blood-vessels, which dilate during the headache phase of migraine, are thought to receive axonal projections from all three divisions of the trigeminal nerve. Substance P, a potent vasodilating peptide, seems to be released from trigeminal nerve endings in response to nervous stimulation and is involved in the transmission of painful stimuli within the periphery. The vasoactive molecule serotonin, implicated in the pathogenesis of migraine, coexists with substance P in some terminals of the central nervous system and is present within the trigeminal ganglia. Within this nerve serotonin may modulate the function of primary sensory neurons. The abnormal release of substance P or as yet unidentified peptides or other transmitters from the fifth cranial nerve may explain both the hemi cranial pain and the vasodilation which are characteristic of the headache of migraine [38].

Sumatriptan

Sumatriptan and the ergot alkaloids are useful tools for deciphering drug mechanisms in migraine and related headaches. Both neuronal and vascular mechanisms have been proposed on the basis of actions of 5-HT at receptors resembling the 5-HT1D subtype. According to Michael A. Moskowitz, he argues that blockade of neural transmission and the neurogenic inflammatory response provides a mechanism by which sumatriptan and ergot alkaloids alleviate vascular headaches. He postulates, with similar arguments, that sumatriptan and ergot alkaloids may block headaches that develop from meningovascular inflammatory disorders such as

from viral and bacterial meningitis and from the sequelae of head injury [39].

Conclusion

Migraine increased the risk of Bell palsy in the total population. Migraine patients between ≥30 and <60 years of age have an increased risk of Bell palsy. It was earlier considered that migraine headaches are triggered by the dilation of cerebral vessels, and recently it was shown that the migraine attacks can also occur in the absence of vasodilation.

Migraine is a visceral pain, caused by activation of trigeminovascular system due to release of Substance-P, which induces vasodilation and plasma protein extravasation leading to neurogenic inflammation.The neurogenic inflammation of the facial nerve trunk caused by its proximity to the dilated posterior auricular/ stylomastoid/ occipital and superficial temporal arteries during a migraine attack leads to a temporary lower motor neuron type of paresis of the muscles supplied by the facial nerve.

Sumatriptan is the drug of choice for treating migrainous Bell's palsy. We herewith report a rare case of migrainous left Bell's palsy after migrainous right external ophthalmoplegia, treated with Sumatriptan.

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