

2018년 CPX

두통



박광열

중앙대학교 신경과학교실

1 Introduction

- Migraine
- Secondary headache

2 Case

3 Video

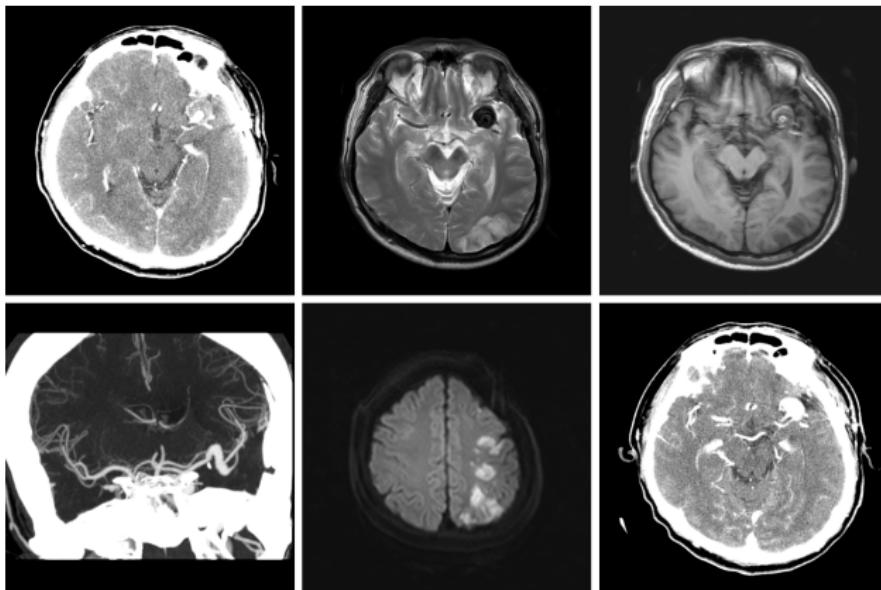
Global burden of Disease in 2010

	Prevalence (both sexes)		Male prevalence		Female prevalence	
	Total (thousands)	Proportion of population (%)	Total (thousands)	Proportion of population (%)	Total (thousands)	Proportion of population (%)
Dental caries of permanent teeth	2 431 636	35.29%	1 194 051	34.37%	1 237 585	36.23%
Tension-type headache	1 431 067	20.77%	655 937	18.88%	775 131	22.69%
Migraine	1 012 944	14.70%	371 072	10.68%	641 873	18.79%
Fungal skin diseases	985 457	14.30%	516 167	14.86%	469 291	13.74%
Other skin and subcutaneous diseases	803 597	11.66%	417 129	12.01%	386 468	11.32%
Chronic periodontitis	743 187	10.79%	378 407	10.89%	364 780	10.68%
Mild hearing loss with perinatal onset due to other hearing loss	724 689	10.52%	386 147	11.11%	338 543	9.91%
Acne vulgaris	646 488	9.38%	311 349	8.96%	335 140	9.81%
Low back pain	632 045	9.17%	334 793	9.64%	297 252	8.70%
Dental caries of baby teeth	621 507	9.02%	352 085	10.13%	269 421	7.89%
Moderate iron-deficiency anaemia	608 915	8.84%	269 596	7.76%	339 319	9.93%
Other musculoskeletal disorders	560 978	8.14%	262 779	7.56%	298 199	8.73%
Near sighted due to other vision loss	459 646	6.67%	235 052	6.77%	224 593	6.58%
Mild iron-deficiency anaemia	375 438	5.45%	152 523	4.39%	222 915	6.53%
Asthma	334 247	4.85%	160 346	4.61%	173 901	5.09%

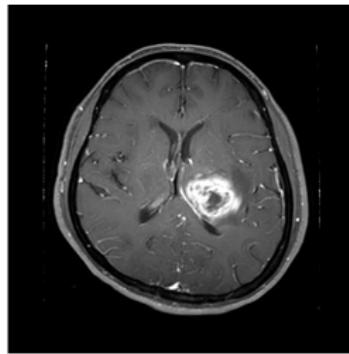
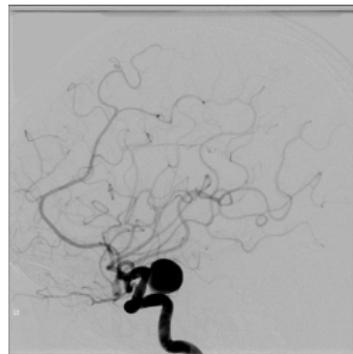
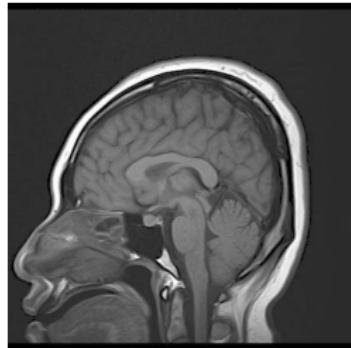
Vos T, et al. Lancet. 2012

- Headache is a symptom.
- Primary headache: no other causative disorder
- Secondary headache: new headache occurring in close temporal relation to another disorder that is a known cause of headache

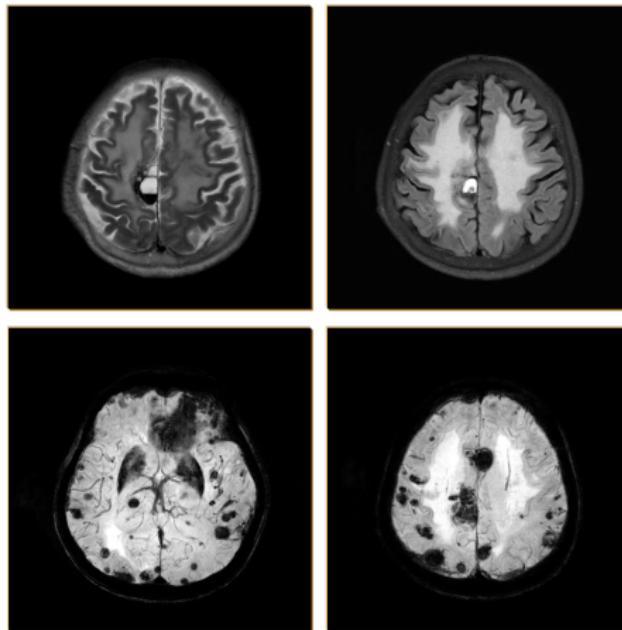
Secondary headache



Secondary headache



Secondary headache



The International Classification of Headache Disorders, 3rd edition (beta version)

Part one: the primary headaches

1. Migraine
2. Tension-type headache
3. Trigeminal autonomic cephalgias
4. Other primary headache disorders

Part two: the secondary headaches

Introduction

5. Headache attributed to trauma or injury to the head and/or neck
6. Headache attributed to cranial or cervical vascular disorder
7. Headache attributed to non-vascular intracranial disorder
8. Headache attributed to a substance or its withdrawal
9. Headache attributed to infection
10. Headache attributed to disorder of homeostasis
11. Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cervical structure
12. Headache attributed to psychiatric disorder

Part three: painful cranial neuropathies, other facial pains and other headaches

13. Painful cranial neuropathies and other facial pains
14. Other headache disorders

Appendix

Definition of terms

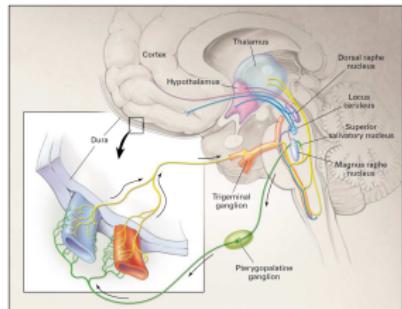
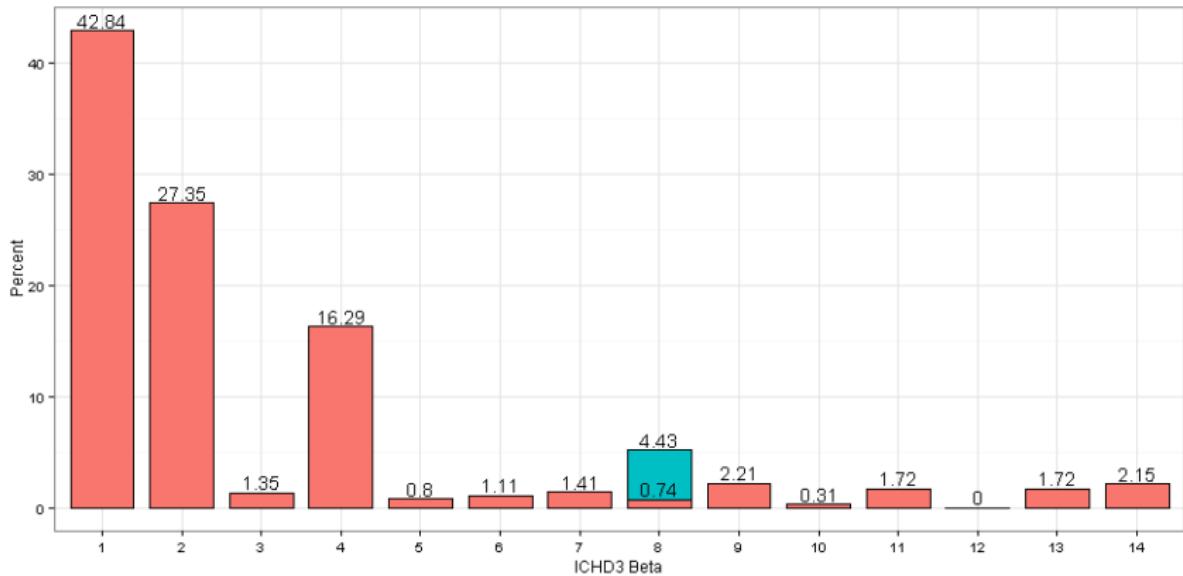


Figure 1. Pathophysiology of Migraine.

Migraine involves dysfunction of descending pathways that normally modulate sensory input. The key pathways for the pain are the trigeminal sensory input from the meningeal vessels, which passes through the trigeminal ganglion and synapses on second-order neurons in the trigeminovascular complex. These axons, in turn, project through the trigeminocervic tract, and after descending in the posterior limb of the internal capsule, synapse in the posterior nucleus of the thalamus. From here, the pain fibers pass in the superior salivatory nucleus, which results in a cranial parasympathetic outflow that is mediated through the pterygopatine, ophthalmic, and maxillary nerves. The pterygopatine nerve also projects to the trigeminal ganglion, where it can modulate pain pathways with trigeminal-autonomic co-opsilches, such as cluster headache and paroxysmal hemicrania. It may be active in response. fMRI imaging studies suggest that increased modulation of the trigeminothalamic receptive input comes from the dorsal raphe nucleus, locus caeruleus, and nucleus raphe magnus.



문화수, 조수진등.

1.1 Migraine without aura

- A At least five attacks fulfilling criteria B - D
- B Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)
- C Headache has at least two of the following four 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 phonophobia
- E Not better accounted for by another ICHD-3 diagnosis

1.2 Migraine with aura

- A At least two attacks fulfilling criteria B - C
- B One or more of the following fully reversible aura symptoms:
 - ① visual
 - ② sensory
 - ③ speech and/or language
 - ④ motor
 - ⑤ brainstem
 - ⑥ retinal
- C At least two of the following four characteristics:
 - ① at least one aura symptom spreads gradually over ≥ 5 minutes, and/or two or more symptoms occur in succession
 - ② each individual aura symptom lasts 5-60 minutes
 - ③ at least one aura symptom is unilateral
 - ④ the aura is accompanied, or followed within 60 minutes, by headache
- D Not better accounted for by another ICHD-3 diagnosis

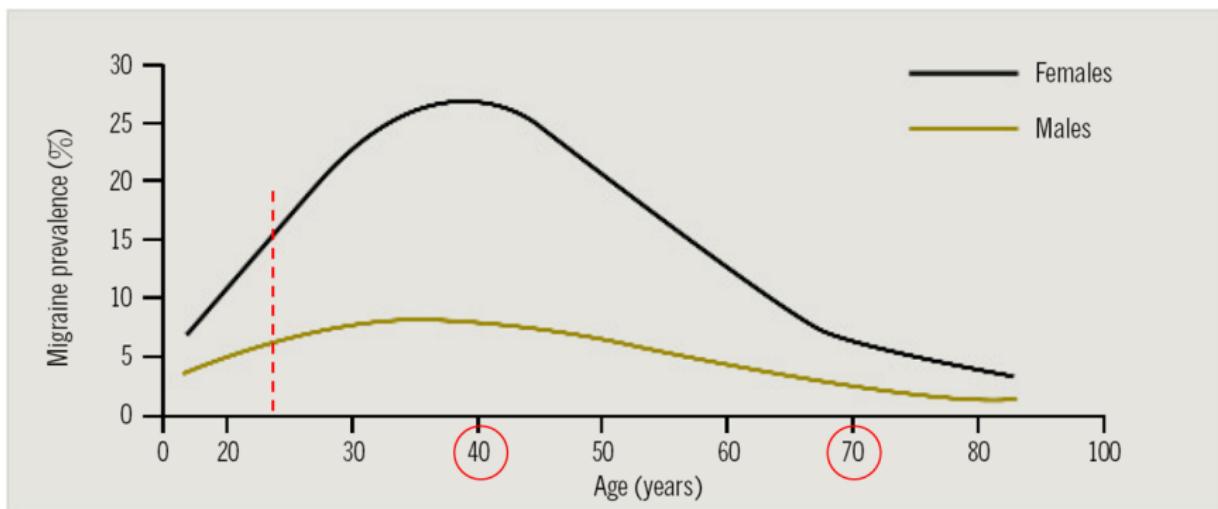
Migraine aura



- 5분에서 60분간 지속
- 전조후 60분이내에 두통이 발생
- 시각증상
- 감각증상
- 언어장애



Migraine Prevalence



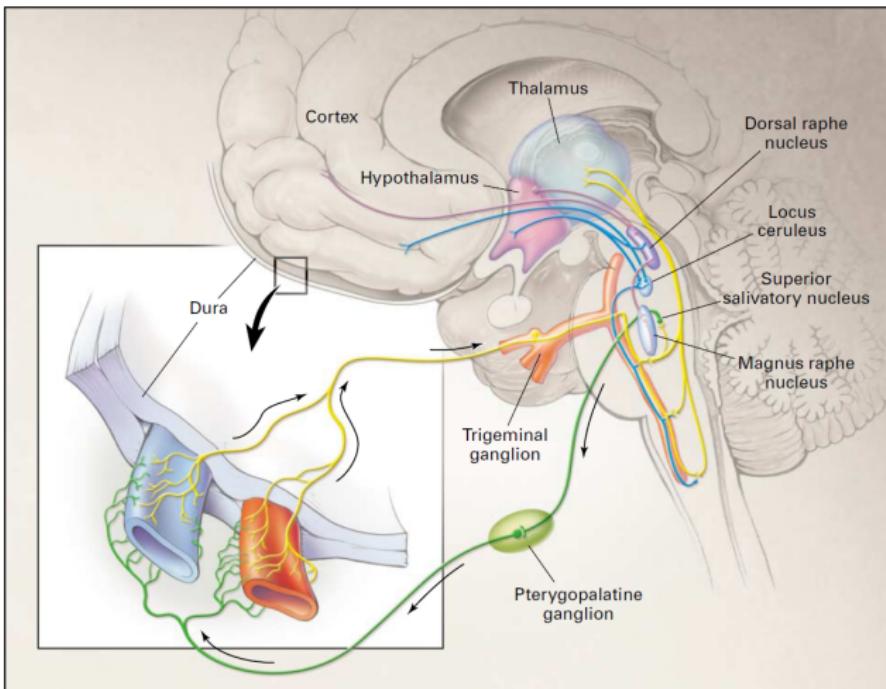
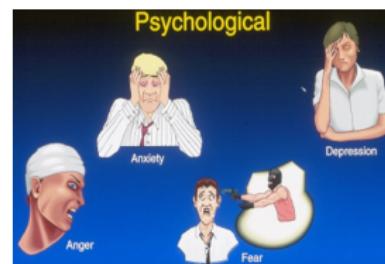
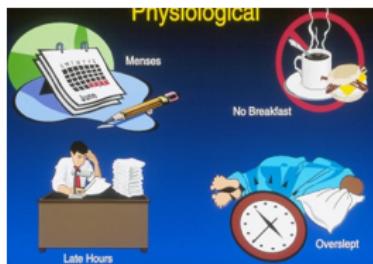


Figure 1. Pathophysiology of Migraine.

Migraine involves dysfunction of brain-stem pathways that normally modulate sensory input. The key pathways for the pain are the trigeminovascular input from the meningeal vessels, which passes through the trigeminal ganglion and synapses on second-order neurons in the trigeminocervical complex. These neurons, in turn, project through the quintothalamic tract, and after decussating in the brain stem, form synapses with neurons in the thalamus. There is a reflex connection between neurons in the pons in the superior salivatory nucleus, which results in a cranial parasympathetic outflow that is mediated through the pterygopalatine, otic, and carotid ganglia. This trigeminal-autonomic reflex is present in normal persons³⁴ and is expressed most strongly in patients with trigeminal-autonomic cephalgias, such as cluster headache and paroxysmal hemicrania; it may be active in migraine. Brain imaging studies suggest that important modulation of the trigeminovascular nociceptive input comes from the dorsal raphe nucleus, locus ceruleus, and nucleus raphe magnus.

유발요인



비교적 확실

- 스트레스
- 생리
- 카페인 중단

가능성 높음

- 금식
- 수면장애
- Nitrate, MSG
- Wine

가능성이 있음

- 흡연
- 냄새
- 초콜릿

흔한 이차두통

- Meningitis, encephalitis
- Tumor
- Drug - medication overuse headache
- Trauma

Case description

남자/ 30세

두통

체온: 37.4도씨

해야 할 일

병력청취

신체진찰

추가 진단과 치료

병력 청취

- 증상이 언제부터 생겼는지?
- 증상이 어떤 상황에서 생겼는지? - 갑자기?, 이전 병력?
- 증상이 어떤 양상인지? - character, location, severity
- 동반증상 - 열이 나는지? 오심/구토?
- 완화요인? 악화요인?
- 약물 복용 여부
- 외상병력
- 가족력

- 3일전부터 두통
- 열감, 두통약 먹어도 심함
- 오심, 구토
- 두통약 복용
- 외상 없음.

흔한 실수

50%이상 틀림

갑자기 생긴 증상인지

Check light reflex

30 - 50% 틀림

구역, 구토 여부

유발 또는 악화요인 외상력

편두통의 전조증상

Check eyeball movement and fundus

신체 검진도중 불편하지 않는지 배려

감별진단

뇌수막염

편두통

뇌농양, 뇌염, 지주막하출혈, 긴장형두통등

진단계획

뇌영상검사 - CT or MRI

뇌척수액검사

Watching video files.