University of Basrah
Al-Zahraa Medical College

Ministry of higher Education
and Scientific Researches

BLOCK:

Mental health care and Neurology

Headache

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Objectives:

- What are the common causes of headache?
- Cluster headache.
- Migraine.
- Tension headache.
- Trigeminal Neuralgia.
- Raised Intracranial Pressure (ICP) headache.
- Head Trauma headache
- Pain related to the Head
- Meningitis Headache
- Temporal Arteritis Headache

What are the common causes of headache?

Headache is pain in any part of the head, including the scalp, face, and interior of the head. Headaches are generally classified by cause:

A primary headache is caused by overactivity of or problems with pain-sensitive structures in your head. A primary headache isn't a symptom of an underlying disease. Some people may also carry genes that make them more likely to develop such headaches. The most common primary headaches are:

(1) Cluster headache
(2) Migraine
(3) Tension headache

A secondary headache is a symptom of a disease that can activate the pain-sensitive nerves of the head. Possible causes of secondary headaches include:

(1) Head Structures:

- Acute sinusitis (nasal and sinus infection)
- Dental problems
- Ear infection (middle ear)
- Glaucoma (acute angle closure glaucoma)

(2) Blood Vessels:

- Arterial tears (carotid or vertebral dissections)
- Blood clot (venous thrombosis) within the brain
- Brain aneurysm
- Brain AVM (arteriovenous malformation)
- High blood pressure (hypertension)

(3) Nerves:

Trigeminal neuralgia (as well as other neuralgias, all involving irritation of certain nerves connecting the face and brain)

(4) Trauma:

- Concussion
- Post-concussion syndrome

(5) Infection and Inflammation:

- Meningitis
- Encephalitis (brain inflammation)
- Giant cell arteritis (inflammation of the lining of the arteries)
- Influenza (flu) and other febrile (fever) illnesses

(6) Raised Intracranial Pressure:

- Intracranial space occupying lesions (Brain tumor)
- Pseudotumor cerebri (idiopathic intracranial hypertension)

(7) Others:

- Medications to treat other disorders
- Panic attacks and panic disorder

Cluster headache

The exact cause of cluster headaches is unknown. The headache is commonly awakens the patient in the middle of the night with intense pain in or around one eye on one side of your head. Bouts of frequent attacks, known as *cluster periods*, can last from weeks to months, usually followed by remission periods when the headaches stop. During remission, no headaches occur for months and sometimes even years. A cluster headache strikes quickly, usually without warning (Unlike migraine and tension headache, cluster headache generally isn't associated with triggers). Common signs and symptoms during a headache (During a cluster period) include:

The Headache:

- Headaches usually occur every day, sometimes several times a day
- A single attack can last from 15 minutes to 3 hours
- The attacks often occur at the same time each day
- Most attacks occur at night, usually 1 to 2 hours after going to bed
- Excruciating pain that is generally situated in, behind or around one eye, but may radiate to other areas of your face, head and neck
- One-sided pain
- Restlessness

The Eye:

- Excessive tearing
- Redness of your eye on the affected side
- Swelling around your eye on the affected side
- Drooping eyelid on the affected side

The Nose:

- Stuffy or runny nose on the affected side

The Face:

- Forehead or facial sweating on the affected side
- Pale skin (pallor) or flushing on your face

There's no cure for cluster headaches. The goal of ***treatment*** is to decrease the severity of pain, shorten the headache period and prevent the attacks. Fast-acting treatments includes:

Oxygen. Briefly inhaling pure oxygen through a mask provides dramatic relief for most who use it. The effects of this safe, inexpensive procedure can be felt within 15 minutes.

Triptans (Triptans are not painkillers. Triptans work by imitating the action of a brain chemical called 5-hydroxytryptamine (5-HT). 5-HT is also known as serotonin that cause widened (dilated) blood vessels to narrow (constrict). Triptans is an effective treatment for acute cluster headache.

Preventive therapy starts at the onset of the cluster episode with the goal of suppressing attacks. The calcium channel blocking agent *verapamil* is often the first choice for preventing cluster headache. Inflammation-suppressing drugs called corticosteroids, such as *prednisone* may be effective for many people with cluster headaches.

Migraine

Migraine is a headache that can cause severe throbbing pain or a pulsing sensation, usually on one side of the head. It's often accompanied by nausea, vomiting, and extreme sensitivity to light and sound. Migraine can progress through 4 *stages*: *prodrome*, *aura*, *attack* and *post-drome*. Not everyone who has migraines goes through all stages.

Prodrome:

One or two days before a migraine, there might be subtle changes that warn of an upcoming migraine, including:

- Constipation
- Mood changes, from depression to euphoria
- Increased urination
- Fluid retention
- Frequent yawning

Aura:

For some people, an aura might occur before or during migraines. Auras are reversible symptoms of the nervous system. They're usually *visual* but can also include other disturbances. Each symptom usually begins gradually and can last up to 60 minutes.

Examples of migraine auras include:

- Visual phenomena, such as seeing various shapes, bright spots or flashes of light
- Vision loss
- Pins and needles sensations in an arm or leg
- Weakness or numbness in the face or one side of the body
- Difficulty speaking

Attack:

A migraine usually lasts from 4 to 72 hours if untreated. Migraines might occur rarely or strike several times a month.

During a migraine, you might have:

- Pain usually on one side of your head, but often on both sides
- Pain that throbs or pulses
- Sensitivity to light, sound, and sometimes smell and touch
- Nausea and vomiting

Post-drome:

After a migraine attack, you might feel drained, confused and washed out for up to a day.

Migraine triggers:

There are a number of migraine triggers, including:

Hormonal changes in women.

Drinks. These include alcohol, especially wine, and too much caffeine, such as coffee.

Stress.

Sensory stimuli. Bright or flashing lights can induce migraines, as can loud sounds.

Sleep changes. Missing sleep or getting too much sleep can trigger migraines in some people.

Physical factors. Intense physical exertion.

Weather changes. A change of weather or barometric pressure can prompt a migraine.
Medications. Oral contraceptives and vasodilators.
Foods. Aged cheeses and salty and processed foods might trigger migraines.

Risk factors:

Several factors make you more prone to having migraines, including:
Family history. If you have a family member with migraines, then you have a good chance of developing them too.
Age. Migraines can begin at any age, though the first often occurs during adolescence. Migraines tend to peak during your 30s, and gradually become less severe and less frequent in the following decades.
Sex. Women are 3 times more likely than men to have migraines.
Hormonal changes. For women who have migraines, headaches might begin just before or shortly after onset of menstruation. They might also change during pregnancy or menopause. Migraines generally improve after menopause.

Treatment:

There's currently no cure for migraines, although a number of treatments are available to help ease the symptoms.

During an attack:

Most people find that sleeping or lying in a darkened room is the best thing to do when having a migraine attack.

Drugs:

Many people who have migraines find that painkillers, such as *paracetamol*, *aspirin* and *ibuprofen*, can help to reduce their symptoms. They may recommend taking painkillers in addition to a type of medicine called a *triptan*, and possibly *anti-sickness medicine (anti-emetics) like metoclopramide and prochlorperazine*. Triptan medicines (Sumatriptan, Zolmitriptan, Naratriptan) are thought to work by reversing the changes in the brain that may cause migraine headaches. They cause the blood vessels around the brain to narrow (contract). This reverses the widening of blood vessels that's believed to be part of the migraine process. Triptans are available as tablets, injections and nasal sprays. A variety of *ergotamine* preparations, have been used for the abortive treatment of migraine which bind to 5HT receptors, just as triptans do. *Opioids* and *barbiturates* should not be used for the treatment of migraine, except as a last resort.

Home Remedies:

include:
Resting in a dark, quiet, cool room.
Applying a cold compress or washcloth to your forehead or behind your neck. (Some people prefer heat.)
Massaging your scalp.
Applying pressure to your temples in a circular motion.

Prevention:

Preventive medication is aimed at reducing how often you get a migraine, how severe the attacks are and how long they last. Options include:

Blood pressure-lowering medications. These include beta blockers such as *propranolol* and *metoprolol*. Calcium channel blockers such as *verapamil* can be helpful in preventing migraines with aura.

Antidepressants. A tricyclic antidepressant (*amitriptyline*) can prevent migraines.

Anti-seizure drugs. (*Valproate*).

Tension headache

Tension-type headache is generally a mild to moderate pain that's often described as feeling like a tight band around the head. Signs and symptoms of a tension-type headache include:

- Dull, aching head pain
- Sensation of tightness or pressure across the forehead or on the sides and back of the head
- Tenderness in the scalp, neck and shoulder muscles
- Unlike some forms of migraine, tension-type headaches usually aren't associated with visual disturbances, nausea or vomiting. Although physical activity typically aggravates migraine pain, it doesn't make tension-type headache pain worse.

Stress is the most commonly reported trigger for tension-type headaches.

Using medications in conjunction with stress management techniques may be more effective in reducing tension-type headaches.

Additionally, living a healthy lifestyle may help prevent headaches:

- Get enough, but not too much, sleep.
- Don't smoke.
- Exercise regularly.
- Eat regular, balanced meals.
- Drink plenty of water.
- Limit alcohol, caffeine and sugar.

Trigeminal Neuralgia

Neuralgia is a clinical term for a painful sensation extending through an area of specific nerve distributions and occurring in intermittent, recurrent paroxysms.

Aetiology: There is demyelination of large diameter (A) fibers at trigeminal root entry zone that lead to abnormal transmission from these fibers to poorly myelinated (A) delta and unmyelinated (C) fibers. This demyelination comes from compression of trigeminal nerve by;

1- an artery or vein at root entry zone
2- Tumor (e.g. Posterior fossa tumor)
3- Multiple Sclerosis

History: It is primarily a disease of older adult (above 50) with female dominant. The Classical Symptoms include;

1- Paroxysmal unilateral lancinating electric pain in one or more areas of trigeminal distribution.

2- There are trigger areas on facial skin stimulated by touch, wind, cold or hot liquid. The pain may also be triggered by speaking, chewing or other facial movements.

3- Periods of remissions and exacerbation

4- Pain that is typically more severe in the morning and absent during sleep.

5- Periodic pain relief when treated with an adequate trial of Carbamazipine.

After repetition of such pain the patient may experience a more constant aching background discomfort

Examination: Decrease sensation in the affected trigeminal distributions.

Imaging: Brain CT scan or MRI may give clue to the cause of neuralgia.

Treatment: The main modality of treatment include;

1- Medical: Carbamazipine (Tegretol) and Baclofen (Lioresal) act to enhance inhibitory neuronal activity in trigeminal nucleus.

2-Surgical;

(a) Percutaneous techniques: e.g. Glycerol Rhizotomy (Percutaneous Glycerol injection through foramen ovale into Trigeminal Mecklel's cave)

(b) Microvascular decompression through craniotomy.

Raised Intracranial Pressure (ICP)

Clinical Features:

General symptoms of raised ICP include:

Headache: due to Raised Intracranial Pressure (ICP) is generalized in nature and often severest in the morning because of vascular congestion due to:

(1) vascular dilatation secondary to increase CO2 during sleep

(2) recumbent position during sleep

The headache is frequently relieved by vomiting due to decrease ICP by hyperventilation.

Vomiting: which is usually without nausea.

Blurring of vision (due to papilledema).

Diplopia (due to 6th. nerve palsy).

Specific Symptoms related to the cause of raised ICP.

General signs of raised ICP include:

Vital signs (Cushing Triad). Cushing Triad is a triad of bradycardia, hypertension and respiratory irregularities. Respiratory changes occur early, followed by bradycardia, with hypertension occur at a very late stage.

papilledema.

Squint (due to 6th. nerve palsy (usually bilateral)).

Specific signs related to the cause of rise ICP.

Effect of raised ICP:

Normal ICP range from 10 to 15 mm. Hg. (135 to 200 mm. H2O) Increase ICP disturbs brain function by two ways:

(1) Reducing cerebral blood flow

(2) Causing brain herniation

Causes of raised ICP

(1) Causes related to the brain:

(a) Neurological elements (Tumors)

(b) Interstitial fluid (Edema)

(2) Causes related to the Blood:

(a) Arterial (Acute systemic hypertension after lose of cerebral auto-regulation)

(b) Venous (Venous sinus thrombosis)

(c) Both (Hematomas, increase CO2)

(3) Causes related to the CSF:

As in hydrocephalus

(4) Unknown Cause: (Pseudotumor cerebri)

Treatment of raised ICP:

The goal of the treatment is to reduce ICP in order to increase cerebral blood flow and relieve or prevent brain herniation:

(1) General measures: ICP can be lowered by reducing the volume of any of the intracranial components (Brain, Blood or CSF) even if the component is normal;

(A) Head elevation in (euvolemic patient) can significantly reduce ICP without altering cerebral perfusion pressure, through improvement of venous outflow from the head.

(B) Hyperventilation can cause a fall in ICP by reducing intracranial blood volume through vasoconstriction by washing out CO2. It is generally initiated for acute management of increase ICP.

(C) Hypertonic solutions and diuretics reduce the fluid content of normal brain.

(D) Removal of CSF via ventricular cannula

The **Pseudotumor cerebri** should be treated because if left untreated, vision loss can be permanent. ***Medical*** treatment include:

Acetazolamide, a medication that reduces the amount of CSF your body produces.

Diuretics to lessen fluid in the brain.

Pain relievers such as ibuprofen or acetaminophen.

Repeat spinal taps to remove excess CSF and relieve the pressure.

Weight loss of at least 5% to 10% of your total body weight, if appropriate.

If those treatments aren't adequate, you may need ***surgery*** to reduce pressure in your skull:

Optic nerve sheath fenestration: This surgery cuts slits into the covering of the optic nerve behind the eyeball. The slits allow CSF to escape to relieve pressure.

Shunt implantation: Lumbo-Peritoneal Shunt to drain extra CSF from lumar subarachnoid space to peritoneum where it can be absorbed.

(2) Specific measures: directed to treat the cause of raised ICP.

(3) Barbiturate Coma: Induction of coma with short-acting barbiturates is the last resort in the management of rise ICP when all other measures fail. The most commonly used drug is thiopental. It decrease ICP by inhibit cerebral metabolism and reduce cerebral blood flow.

Head Trauma

Pain generated from pain sensative structure in the Scalp, Skull and Brain after head trauma. Diffuse brain lesions result from acceleration-deceleration injuries to the brain, and they are of two types;

(a) Concussion It is condition characterized clinically by transient traumatic lose of consciousness for less than 6 hours associated with some degrees of post-traumatic amnesia (amnesia for events related to the injury and afterward). The inertial force causes deeper structures within the brain to deform resulting in wide spread disruption of brain function but most of the strain is insufficient to cause structural damage.

(b) Diffuse axonal injury It is condition characterized clinically by traumatic lose of consciousness from the time of injury that continues beyond 6 hours. There are microscopic damage scattered throughout the brain including focal axonal changes that lead to focal impairment of axoplasmic transport and disconnection.

Post-concussion syndrome (PCS) is a set of symptoms that may continue for weeks, months, or a year or more after a concussion. The condition is associated with a wide range of non-specific symptoms such as headache, dizziness, difficulty concentrating, sensitivity to light or noise, depression and irritability. The headache is typically a steady ache affecting both sides of the head and occurring daily or almost everyday. Most often treatment of this type of headache consists of such medications as the tricyclic antidepressants (for example amitriptyline). These agents not only diminish depression but also decrease pain.

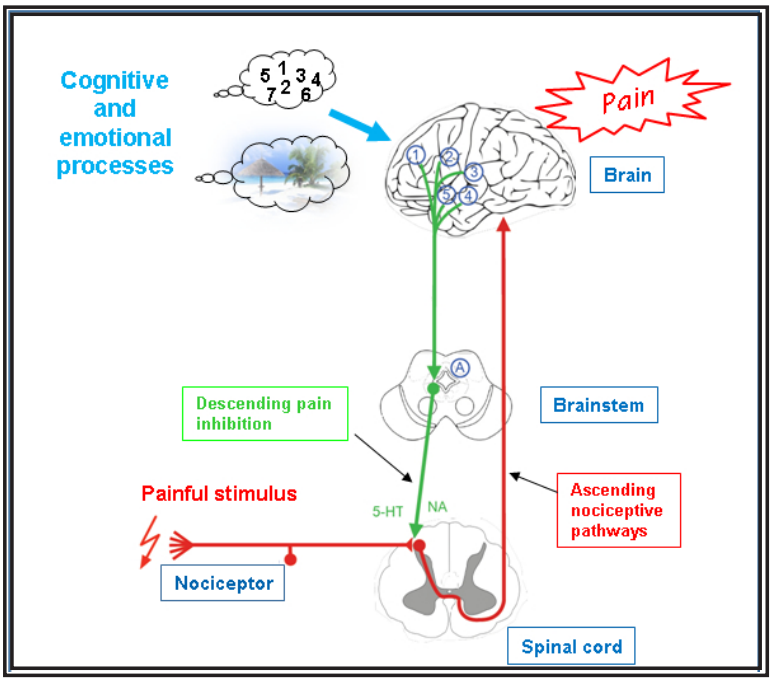
Pain related to the Head

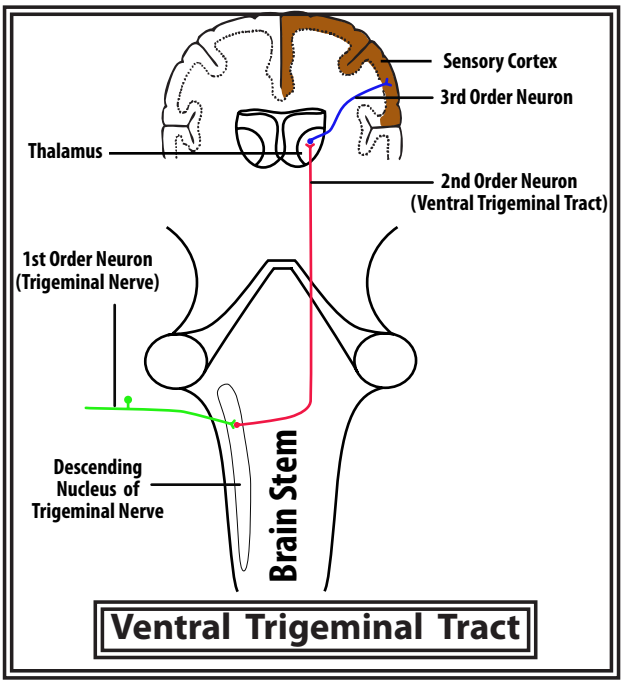
Pain is unpleasant sensory and emotional experience associated with actual or potential tissue damage.

A- Ascending Pathways: ***Ventral Trigeminal Tract*** (carry painful stimuli from the face). Painful stimuli activate the free nerve endings (pain receptors). The free nerve endings are the peripheral process of unipolar ***First order neurons*** whose bodies are located in the trigeminal ganglia (Trigeminal nerve). The Central process of the trigeminal ganglia enter the Descending nucleus of trigeminal nerve.

The ***Second Order Neurons*** cross to the other side and form: *Ventral Trigeminal Tract* (Brainstem level). These tracts ends in thalamic nuclei.

The ***Third order neurons*** in the thalamus reach the cerebral cortex and end into: (a) *Primary Sensory Cortex* (for estimation of stimulus intensity and fine localization of the stimulated zone, (b) *Secondary Somatosensory area* (for spatially directed attention toward noxious stimuli, (c) *Anterior Cingulate Cortex* (As a part of the Limbic System; this cortex play a role in emotional aspect of pain.





B- Descending Pathways: These form the descending control system of the ascending pathways. Inputs from frontal cortex and hypothalamus activate cells in the midbrain (*periaqueductal gray mater*) that control pain-transmission cells (ascending pathways) via cells in the medulla. Each of the component structures of the pathway contains *Opioid receptors* and is sensitive to the direct application of opioid drugs. Stimulation of the descending pathways result in various degrees of pain control (stimulation of periaqueductal gray mater in Deep Brain Stimulation Procedure).

Classifications of Pain

A- According to the tissue healing time:

1- Acute pain: signals acute tissue injury. It is generated by activation of nociceptors in the tissue and resolve as the injured tissue heal.

2- Chronic pain: is that what outlasts the typical period required for healing of acute injury [some definition of acute pain are based on the duration of pain (e.g. pain that lasts longer than 3 or 6 months); this is not always an accurate distinction, because different types of acute injuries require different healing times]. The persistently stimulation of spinal dorsal horn (or brainstem equivalents) nocicepetive transmission pathways, will induce changes within them lead to re-organisation within the nociceptive pathways and lower the threshold for pain transmission and may lead these neurones to generate their own electrical activity in the absence of any external stimuli.

B- According to the level of tissue injury:

1- Nociceptive pain *(at innervated tissue level)*: is generated by activation of receptors subsequent to injury or disease (cancer pain) that lead to normal peripheral and central neuronal activity. It is typically constant (sometimes with paroxysmal),and aching. Patient usually use description like throbbing, aching or dull. It is usually best treated with opioid medications.

2- Neuropathic pain *(at nerve tissue level)*: is the result of pathological process (injury or disease) affecting the peripheral or central nervous system that involve aberrant regeneration or conduction which leads to abnormal neuronal excitability and spontaneous discharges. Patient used descriptions like burring, shooting, tingling, itching, crawling or shock-like (electrical). Neuropathic pain may be either:

(a) Paroxysmal: commonly seen in neuralgias; it is thought to represent abnormal sensitisation of the nerve, which lead to attacks of lancinating (electric-like) pain in the distribution of the affected nerve. It is best treated with anticonvulsants.

(b) Constant: is thought to occur in the setting of damage to the nerve or central nervous system which lead to abnormal balance of inputs in neuromodulatory circuits (frequently described as itching, burning or crawling). It is best treated with Psychotropic medications (e.g. Tricyclic antidepressants).

Pain Management

A- Medical: *Acute pain* reflect acute tissue injury and treatment should be aimed at promoting tissue healing. Treatment might include; rest, immobilization, analgesics and passive physical therapy modalities. Many individuals with *Chronic pain* rehabilitation and often dictates a treatment programs in addition to treatment of the pathological elements. Medical therapy should always proceed the surgical intervention for *Nociceptive pain*; beginning typically with non-opioid analgesics (Non Steroidal Anti Inflammatory Drugs), if adequate pain control is not obtained with non-opioid medications mild opiod analgesics might be required followed by strong opiod analgesic if necessary. *Neuropathic pain* frequently require treatment with non-opioid analgesics. Non-pharmacologic adjuvants for nociceptive and neuropathic pains include; psychological support, relaxation therapies and passive physical therapy modalities (massage, heat or cold).

B- Surgical: In general surgical treatment of pain is appropriate for individuals in whom;

1- medical treatment have not provided adequate pain relief.

2- other treatments are associated with unacceptable side effects (e.g. medication side effects).

3- direct treatment of the underlying cause of pain is not possible or practical (e.g. Cancer pain in patient with severe coronary heart disease).

4- no medical contraindications to surgery.

Surgical Procedures are performed either through percutaneous approaches or through Open surgical approaches. They are include;

(a) Anatomic: nerve decompression (Microvascular decompression).

(b) Augmentative: they are generally preferred as initial surgical treatments because of their relative safety and reversability. They are directed to the descending pain pathways and include;

1- Stimulation (thalamus, motor cortex)

2- Direct drugs (e.g. opioids) infusion to the CNS (intraventricular)

(c) Ablative: directed to the ascending pain pathways like ***Trigeminal Tractotomy*** (Lesioning the ventral trigeminal tract at brainstem level)

Meningitis

Meningitis is an infection of the protective membranes that surround the brain and spinal cord (meninges). Early meningitis symptoms may mimic the flu (influenza). Symptoms may develop over several hours or over a few days.

Possible **signs and symptoms** include: Sudden high fever, Stiff neck, Severe headache with nausea or vomiting, Confusion, Seizures, Sleepiness, Sensitivity to light, No appetite or thirst & Skin rash. *Viral infections are the most common cause of meningitis*, followed by bacterial infections and, rarely, fungal and parasitic infections. Bacterial meningitis is serious and can be fatal within days without prompt antibiotic treatment. Delayed treatment increases the risk of permanent brain damage or death.

Meningitis complications includ: Hearing loss, Memory difficulty, Learning disabilities, Brain damage, Gait problems, Seizures, Renal failure, Shock, Brain abscess, or Death. With prompt treatment, even people with severe meningitis can have good recovery.

Normal composition of CSF

	Normal range
Color	Clear
Specific gravity/pH	1.006–1.007/7.4
Opening pressure	50–200 mm H_2O
RBCs count	Nil
WBC count	0–5 (upto 30 in neonates)
WBC types	Lymphocytes
CSF Proteins	15–40 mg/dL
CSF lactate	1–3 mmol/ L
CSF glucose	50–80 mg/dL (two thirds of blood glucose
Microbial examination	No microorganism

Initial **diagnosis** of meningitis can be made by clinical examination followed by a (Spinal tap - lumbar puncture). The bacteria can sometimes be seen in microscopic examinations of the spinal fluid (lumbar puncture). CSF analysis may help identify which bacterium caused the meningitis. If viral meningitis is suspected, a DNA-based test known as a **P**olymerase **C**hain **R**eaction (**PCR**) amplification or a test to check for antibodies against certain viruses is used to determine the specific cause and proper treatment. If herniation syndrome or signs of expanding posterior fossa lesion with brainstem compression are present, lower the intracranial pressure and obtain CT scan for diagnosis. Lumbar puncture is contraindicated.

If no evidence of herniation is detected; Brain CT scan should be obtained. If meningitis is suspected, and there is no mass lesion presence in the CT scan (especially in posterior fossa), then lumbar puncture is indicated for diagnosis. Treatment should be instituted immediately. If CT scan shows coexisting mass lesion in a patient in whom an infectious causes is strongly suspected (e.g. rupture abscess) especially in posterior fossa, CSF examination could be obtained by methods other than lumbar puncture; like ventricular tap, but antibiotic therapy should not be delayed.

The clues that the doctor uses are the levels of white cells, protein and glucose in the CSF. Typically in *bacterial meningitis* the white cell count is much higher than in *viral meningitis* (and is a different type of white cell), the protein is much higher and the glucose is much lower than in viral meningitis.

Temporal Arteritis

Temporal arteritis is a form of vasculitis (inflammation of the blood vessels). In temporal arteritis, also known as ***giant cell arteritis***, the temporal arteries which supply blood from the heart to the scalp, are inflamed (swollen) and constricted (narrowed). The vasculitis that causes temporal arteritis can involve other blood vessels, such as the posterior ciliary arteries (leading to blindness). If not diagnosed and treated quickly, temporal arteritis can cause damage to eyesight (including sudden blindness in one or both eyes) or damage to blood vessels, such as an aneurysm. The causes of temporal arteritis are poorly understood.

There is no well-established trigger or risk factors.

The most common symptom of temporal arteritis is a throbbing, continuous ***headache*** on one or both sides of the forehead. Other symptoms may include: ***Fatigue***, ***Fever***, ***Jaw pain*** (that may become worse after chewing), ***Tenderness*** (at the scalp or temples), ***Vision problems*** (such as double vision, blurry vision, or transient (brief) vision loss), ***Muscle aches*** (in the upper arms or shoulders, hips, upper thighs, lower back, and buttocks), ***Loss of appetite***.

Although the ESR and/or CRP are almost always high in Temporal arteritis, they are not specific for it. Their role is more important to rule out Temporal arteritis when they are normal rather than to confirm Temporal arteritis when they are elevated. The best way to confirm a diagnosis is by taking a small sample (biopsy) of the temporal artery.

The main **treatment** for giant cell arteritis consists of high doses of a ***corticosteroid*** drug such as prednisone. Because immediate treatment is necessary to prevent vision loss, your doctor is likely to start medication even before confirming the diagnosis with a biopsy.