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Bell's palsy: symptoms and their treatment

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Abstract:-

A common cranial neuropathy that causes facial muscular paresis or total paralysis, usually on one side, is known as Bell's palsy, sometimes known as "acute facial palsy of unknown cause." Bell's palsy can develop suddenly and may increase over the course of 48 hours. It happens when the 7th cranial nerve, the facial nerve, or any of its branches are injured or inflamed while travelling along the course of the nerve, particularly in the bony canal.Although no age is immune and both sexes are equally affected, the incidence increases with age. Patients with diabetes, hypertension, pregnancy, obesity, and upper respiratory tract infections are at particularly high risk. It is diagnosed by ruling out other causes and is primarily thought to be idiopathic. Bell's palsy can have an adverse effect on patients and their loved ones and lead to both physical and psychological issues.Therefore, swift cause identification and early diagnosis play crucial roles in effective treatment. However, Bell's palsy's precise origin is uncertain, which has an impact on how it is treated. However, identifying likely risk and causal variables is essential for using a tailored therapy approach and necessitates a thorough examination and detailed history. Even if they are not treated, the majority of patients recover on their own in less than three weeks.However, there is always a chance of having residual paresis following therapy or recovery, which may call for medical attention. This review focuses on the anatomy, aetiology, clinical characteristics, diagnosis, clinical outcomes, and preferred therapeutic options in order to provide the most comprehensive understanding possible of Bell's palsy.

Categories: Neurology, Otolaryngology.

Keywords: electromyography, nerve excitability test, acyclovir, corticosteroids, lower motor neuron palsy, facial nerve, bell's palsy.

Introduction:-

The face is a highly significant component of an individual's identity and distinctiveness. Any problem with facial muscle function, in addition to physical handicap, causes social and psychological suffering because facial expressions are vital for expressing emotions and in social interactions . One of the most common causes of lower motor neuron facial paralysis, Bell's palsy is an acute-onset peripheral facial neuropathy. In 60 to 75 percent of all occurrences of facial paralysis, Bell's palsy is to blame. 7–40 cases per 100,000 persons occur

annually, with equal prevalence rates for both sexes.The aetiology is still unknown, however it is strongly linked to a few viral infections that produce nerve inflammation, localised edoema, demyelination, and ischemia. A person is more likely to develop palsy as a result of specific risk factors, such as elevated blood sugar uncontrolled blood pressure, severe pre-eclampsia , migraine and radiation exposure .help the pathogenic processes and increase a person's susceptibility to palsy. The numbness, slight pain, increased auditory sensitivity, and taste alteration may accompany the weakening, which may be total or partial. The diagnosis is made mostly by physical examination and is one of exclusion.The distinction between a central and a peripheral lesion can be made with the use of a basic understanding of the neuroanatomy of the nerve. This distinction is important since different etiologies require different management strategies. Antivirals are primarily advised in conjunction with corticosteroids due to their dubious utility.These antivirals are typically advised in conjunction with corticosteroids. The sooner the healing process starts, the lower the risk of complications and lingering paraesthesias. Patients who show improvement within the first three weeks of the onset of symptoms have a higher chance of making a full recovery. 36 percent of patients have palsy on the same side, and 4 to 14 percent of patients may experience recurrence.

History of Bell's palsy

Sir Charles Bell was a Scottish surgeon, anatomist, physiologist, neurologist, artist, and philosophical theologian. He is noted for discovering the difference between sensory nerves and motor nerves in the spinal cord. He is also noted for describing Bell's palsy.

It is believed that Sir Charles Bell (1774–1842) had idiopathic peripheral neuropathy. facial palsy, but because Bell understood that peripheral facial palsy was caused by involvement of the seventh cranial nerve (which he

referred to as the respiratory nerve), not because he was the first to observe or report this finding since depictions of facial palsy can be found in ancient art and texts . The muscles of face expression were under the direction of the seventh cranial nerve, as Bell showed through a series of clinical and experimental observations:

On cutting the respiratory nerve on one side of the face of aThe highly odd activity of his features on that side completely stopped being a monkey. When he flashed his teeth, his lips were drawn to the other side, mimicking the paralysis of an alcoholic, and his cautious movements of his eyelids and brows were lost. the inevitable conclusion is that the fifth pair of nerves have nothing to do with the movements of the lips, nose, eyelids, and forehead in expression. I A suppuration anterior to the ear, through which the nerve went on its way to the face, wounded a man's respiratory nerve of the face in the trunk. It was noted that his mouth was pulled when he smiled and laughed.in a very amazing way to the other side. When he tried to whistle, his lips twisted absurdly. When he sneezed, however, the side where the suppuration had affected the nerve remained calm while the other side displayed the typical distortion.

Materials and methods:-

This is an observational study of 30 Bell's palsy patients who submitted to the Department of ENT Hamdard Institute of Medical Sciences and Research, Jamia Hamdard, New Delhi since January 2018 by January 2019. The study included all patients who progressed unilateral idiopathic lower motor neuron palsy. The degree of paralysis wasrated using the House Brackmann rating system. Candidates with history of otitis media, neurological disease or any head trauma and neck were excluded from the study.

A thorough medical history including age and duration was taken paralysis and type of onset, whether sudden or gradual. other problems such as previous earache, any upper respiratory tract infection or rashes were also looked for. Along with that comes history for comorbidities such as diabetes mellitus, hypertension or others illness was also asked.Complete examination of the auricle, external auditory canal and tympanic membrane was performed. Proper examination of the cranial nerves was performed to rule out possible involvement of another nerve. Systemic an examination was also carried out. All patients were prescribed an oral dose of prednisolone 1mg/kg for 5 days followed by tapering over two weeks together with a five-day course of acyclovir 800 mg five times a day for three days. Facial massage and exercises were explained patients. Transelectrical nerve stimulation (TENS) has also been recommended.along with medication to speed recovery in all patients. These patients were monitored weekly for up to 6 weeks and recovery was analyzed using House-Brackmann scale.

Anatomical perspective:-

For a better understanding of the etiopathogenesis of Bell's palsy, basic knowledge about the course and innervation of the facial nerve is necessary. The facial nerve has three nuclei: motor, sensory and parasympathetic nuclei. The course of the facial nerve can be divided into six segments. The first segment is an intracranial segment that consists of the motor nucleus of the facial nerve located in the pons from where motor fibers arise, hook around the nucleus of the abducens nerve and are connected by the median nerve which carries sensory and parasympathetic components. Next, this mixed nerve passes through posterior cranial fossa and enters the bony facial canal (fallopian tube) through the anterior superior quadrant of the internal acoustic meatus. This is known as the meatal or canalicular segment. Inside inner ear, the facial nerve passes in the fallopian tube between the cochlea and the vestibule and then bends posteriorly at the

geniculate ganglion (first genus). This segment is the shortest and narrowest and is the most prone to inflammation and ischemia. It is known as the labyrinthine segment. Labyrinth segment extends to form the tympanic segment in the middle ear, takes another turn just distal to the pyramid eminence (second genus) and passes vertically downward as the mastoid segment. Bony fallopian tube in many cases it may be dehiscent in some areas and therefore more susceptible to damage. Mastoid segment it starts from the second genu, gives off its branches and ends at the forming stylomastoid foramen out-of-time segment. It also passes between the superficial and deep lobes of the parotid gland and it finally ends in five branches at the anterior border of the gland .The facial nerve provides the efferent motor supply to all the muscles of facial expression, the stapedius, the posterior belly of digastric muscles and parasympathetic and sensory fibers . These parasympathetic fibers supply the submandibular and lacrimal glands via the chorda tympani and the greater surface petrosal nerve, respectively . Therefore, all these fibers and structures are in the case of paralysis any damage to the facial nerve. Elongated and convoluted pathways and presence in a narrow bony canal making the 7th cranial nerve more susceptible to paralysis than any other nerve in the body .

Etiopathogenesis:-

Although the exact pathogenesis of Bell's palsy is unknown and is considered to be idiopathic, specific immune,ischemic and hereditary factors are strongly correlated with its etiology. Based on recent reports of reactivation the migration of dormant herpes virus in the geniculate ganglion and its migration to the facial nerve were considered to be vital in causation . Herpes zoster virus (HZV) and herpes simplex virus (HSV) are human neurotropic alpha herpes viruses and are most often involved .These can remain latent throughout life ganglia .The HZV virus is considered more aggressive because it spreads through the nerve via satellite cells.Usually, herpes simplex is involved in cold sores and genital herpes, while herpes zoster is causative agent of chicken pox and shingles. An infection is said to be latent when no virus is active replication, but in the presence of antibodies or immunodeficient states, nerve damage and inflammation may occur to the facial nerve, resulting in its further compression due to its fact in a narrow bony canal. other viruses known to be involved in causing Bell's palsy are Epstein Barr viruses causing infectious mononucleosis, cytomegalovirus, adenovirus, mumps virus, influenza B, etc. Vascular ischemia can be primary, secondary or tertiary. Primary ischemic neuropathy that causes inflammation of the affected nerve, is more likely to occur under specific clinical circumstances, such as diabetes mellitus .It is usually triggered by cold or emotional stress. Although he has a facial nerve good vascularity and stiff epineurium, vasospasms can cause decreased blood flow and acute inflammation that leads to primary ischemic neuritis, which is uncommon . It can follow secondary ischemia, which further exacerbates nerve damage by causing increased capillary permeability which leads to fluid accumulation, edema and thus nerve compression. In about 4-14% of individuals hereditary predisposition narrows the fallopian tube. This genetic component is mostly autosomal dominant and exposes the nerve to an additional risk of early compression even with the slightest edema .

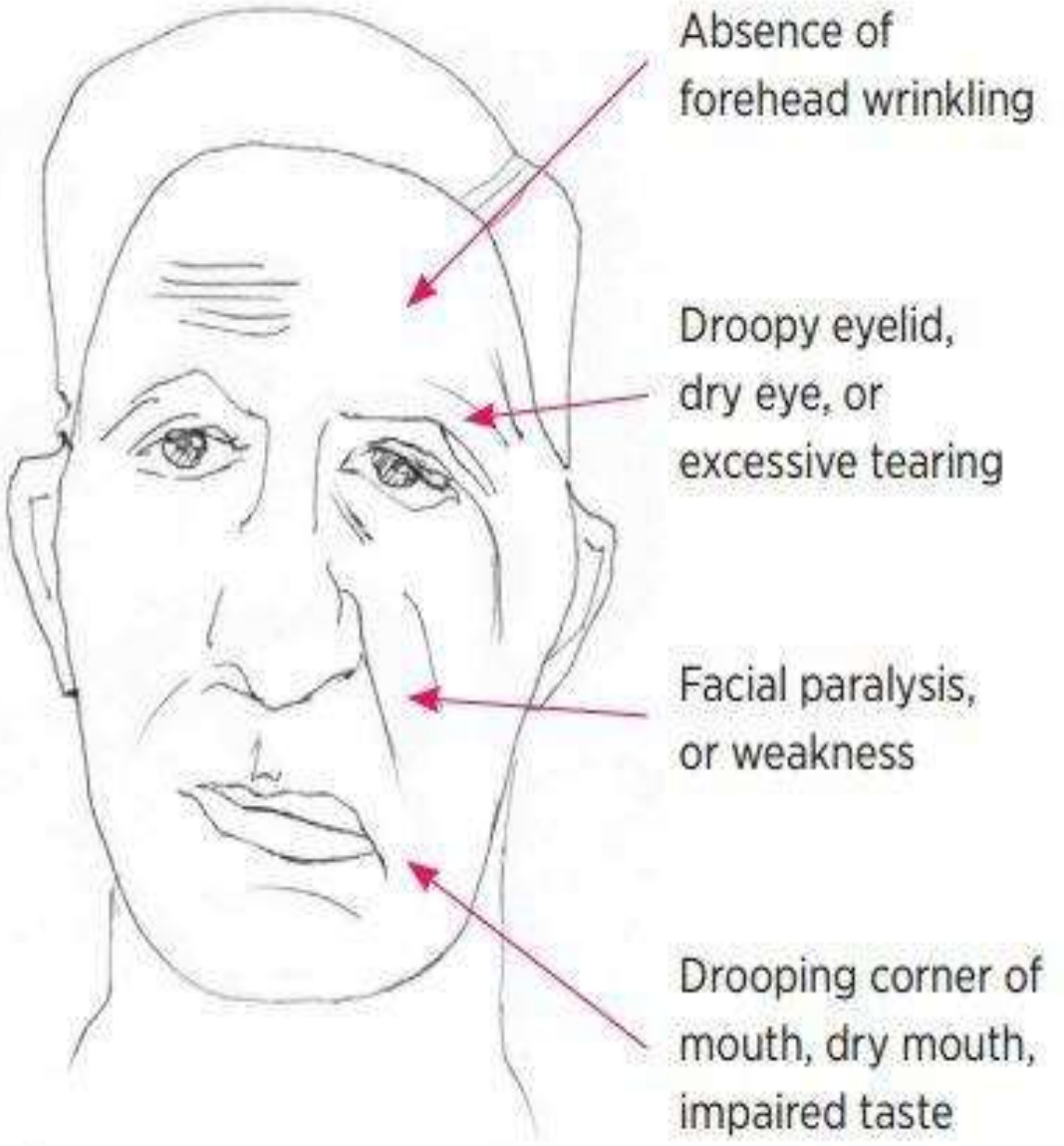


Fig:-1 symptoms of bell's palsy

Approach to the clinical examination of Bell's palsy:-

- **Watch for asymmetry during the conversation:-** Be careful on blinking, nasolabial folds and corners of the mouth.
- **General examinatio**:- otoscopy, palpation for masses near the neck and face and skin examination.
- **Assess motor function and ask the patient to**: Raise both eyebrows, Close both eyes tightly ,Smile,Inflate the cheeks,Pucker your lips,Show upper and lower teeth (grimaces).
- **Assess special sensory function if clinically indicated:**-Face and ear sensation,Taste perception of the front two thirds of the tongue,
- **Assess reflexes**
- **Orbicularis reflex:** tap the glabella and observe the asymmetry in the blink pattern.
- **Bell phenomenon:** observe the upward movement of the eyes with forced closing of the eyes.

EVALUATION OF BELL'S PASSPORT:-

Usually patients with Bell's palsy develop facial weakness over 1 to 2 days. They can find that toothpaste liquids or food leakage from the affected side of the mouth that eyelid does not close, or that there is more hard to talk, which leads many patients to the emergency room for fear of a stroke.A key first step in assessment the patient is to determine whether facial weakness is peripheral or central thrall It is worth noting that while almost all of patients with hemiparesis from strokes have facial weakness, it is rare symptom and is often noticed by others rather than others patient .As discussed in part on anatomy with central facial palsy, sparing upper-a third of the contralateral face occurs.With peripheral facial palsy, weak-all the muscles of facial expression is running out. The wake is lost from eyebrows and the patient cannot raise himself eyebrows (i.e. in response to he commanded: ``Raise your forehead like you're surprised''), eye furrow-it is certainly wider, the nasolabial fold is flattened, face cannot be inflated out and noses don't burn with a heavy inspiration. The patient is unable whistle, and when you smile or point-ing teeth, the mouth is drawn to intact side. Have the patient test his or her ability to whistle is a useful way document recovery.Although most patients with Bell's polio does not notice dry eye (remember that the lacrimal gland is innervated by cranial nerve VII), however, as discussed in the treatment section,proper lubrication and eye care is necessary, especially when they are serious orbicularis oculi weakness curse to the extent that the upper a the lower lids cannot be zoomed.Paradoxically, some patients can before sent with tears streaming down his cheeks,probably due to weakness lower part of the orbicularis oculi, preventing the formation of tears directed to the tear duct, possibly in combination with eye irritation. If the stapedius is a muscle hyperacusis may occur, e.g contraction of stapedia functions to dampen the bones. Despite innervation of the minor salivary facial nerve various glands, dry mouth is usually absent experienced. Connecting chords tympani causes loss of taste in ipsilateral anterior two-thirds language; this may be a first ever a symptom noticed by the patient, a impaired taste can portend worse prognosis of recovery. Other factors reported to be associated with worse results in complete facial paralysis, older age, diabetes mellitus, etc mentioned in the following section, pain outside the ears.18Y20 This is not unusual for patients with Bell's palsy usually report pain around the ear, mastoid and faceVand pain outside the ears was associated with worse prognosis. So are patients may report facial "numbness" (ie important to ask what he means numbness) but sensory testing is usual normal ally in Bell's palsy. Still some the authors suggested that patients with Bell's palsy, they actually have a skull bone polyneuropathy. Adour11 found evi- cranial nerve damage V, VIII, IX and X in patients with Bell's disease paralysis.34 In a series of 51 patients diagnosed nose with Bell's palsy, Benatar a colleagues35 found that 8% had evi- at least one of them other cranial nerve, including trigeminal geminal, glossopharyngeal and hypo- shiny. These findings raise several questions: is bell's palsy really cranial polyneuropathy simply dominates facial nerve injury? Or,would have signs of involvement by others cranial nerves to "rule out" Bell's palsy?It is important that in anan otherwise "typical" case of Bell's palsy,patients may have other subtle cranial nervous symptoms and signs but their presence should be a red flag that the condition may not be Bell's palsy and close monitoring is warranted.

Most patients with Bell's disease polio will not recur, but a recurrence occurs in about 7%, e.g. there on the same or opposite side.36Y38 Repetition should lead to caution look for an alternative cause, e.g such as sarcoidosis39,40 or other inflammation macular or infiltrative disorders. The Melkersson-Rosenthal syndrome is other consideration for recurring pe- reefer facial palsy; it is characteristic with a cracked tongue and a periodical lip or facial swelling,but many patients do not having the whole triad; control language can be an important clue. By following Bell's definition paralysis as spontaneous, acute, unilateral eral, isolated

peripheral facial palsy and if no red flags indicate an alternative the cause, then not even a laboratory examination no need for imaging,unfortunately, most patients will undergo imaging.MRI of patients with Bell's palsy may show improvement in tracanalicular and labyrinthine segments

facial nerve (Case 5-2),23,44 a doesn't necessarily imply that alternative diagnostics. As he pointed out Sartoretti-Schefer and colleagues,the normal facial nerve may show en- hancement geniculate ganglion and the tympanic-mastoid segment. Electrodiagnostic studies have few role in the management of Bell's palsy.According to the clinical practice manual - line from the American Academy Otorhinolaryngology, head and neck circumference gery Foundation, based at the C level evidence, electrodiagnostic studies are not recommended for patients with incomplete facial paralysis, most who will have a good recovery but the patient may be offered s complete paralysis for prognostic pur-poses, though that probably won't change management.

DIFFERENTIAL DIAGNOSIS:-The diagnosis of Bell's palsy is made by exclusion other causes of unilateral facial paralysis,and 30% to 60% of facial palsy cases are caused by an underlying disorder that mimics ics Bell's palsy, including the central nervous system dark lesion (e.g. stroke, demyelinating disease), tumor of the parotid gland, Lyme disease, Ramsay Hunt's syndrome, granulomatous disease, otitis media, cholesteatoma, diabetes, trauma and Guillain-Barré syndrome . Many these conditions have associated features which help differentiate them from Bell's palsy. Facial paralysis that does not improve after 3 weeks should expedite referral to a neurologist.Brain lesions It is uncommon to have isolated facial palsy with a cortical or subcortical brain lesion,from corticobulbar and corticospinal tracts move in close proximity. Cortical signs such as hemiparesis, hemisensory loss, neglect,and dysarthria indicate brain damage bark. In addition, it spares the front muscles is expected in supranuclear lesions.Brainstem lesions can manifest in multiple waysipsilateral cranial nerve palsies and contractions lateral limb weakness. Sarcoidosis and lepto meningeal carcinomatosis tends to involve skull base and present with multiple cranial neuropathy.

They have brain tumors or parotid glands insidious onset and can cause systemic symptoms such as fever, chills and weight loss. It is indicated by headaches, seizures and hearing loss intracranial lesions. Palpable flesh nearby ear, neck, or parotid gland requires imaging face and were looking for that parotid gland plague.

TABLE 2

Differential diagnosis of Bell palsy

Differential diagnosis	Cause	Distinguishing characteristics
Central nervous system lesion	Stroke, space-occupying lesion	Forehead sparing, headache, limb weakness, multiple neurologic signs
Autoimmune diseases	Guillain-Barré syndrome	Ascending weakness, absent reflexes
	Multiple sclerosis	Upper motor neuron signs, abnormal cerebrospinal fluid
Metabolic diseases	Diabetes	Elevated blood glucose
Infectious diseases		
Meningitis, encephalitis	Viral, bacterial, fungal pathogen	Headache, fever, meningeal signs, abnormal cerebrospinal fluid
Herpes simplex	Reactivation of herpes simplex virus type 1 from geniculate ganglion	Fever, malaise
Lyme disease	*Borrelia burgdorferi*	Rash, arthralgia, malaise, bilateral facial palsy
Ramsay Hunt syndrome	Varicella zoster	Pain, vesicular eruption
Granulomatous disease	Sarcoidosis	Bilateral facial palsy, elevated angiotensin-converting enzyme
Neoplasm	Parotid tumor, facial nerve tumor, metastasis	Insidious onset, palpable mass, partial involvement of facial nerve branches

Infection:-

A number of infections can cause an acute face paralysis. The most common is herpes simplex virus and the next most common is chicken pox zoster. Herpes simplex virus, Ramsay Hunt syndrome and Lyme disease may be related aching pain and skin changes. Erythema tympanic membrane suggests otitis media, e.g. especially with earaches and hearing loss.Ramsay Hunt syndrome is caused by re-activation of the herpes zoster virus from geniculate ganglion, affecting the facial nerve.Careful examination of the ear canal a oropharynx may show vesicles.In Lyme disease, facial palsy is the most common common cranial neuropathy, seen in up to 50% ,63% of patients with Borrelia burgdorferi men-ingitis. In people with a history of rash,arthralgia, tick bite or travel to endemic area, Lyme titers should be checked earlier starting treatment with corticosteroids. Bilateral facial palsy is rare and occurs in less than 1% of patients. It was reported in patients with Lyme disease, Guillain-Barré syndrome, sarcoidosis, diabetes mellitus, virus infection and pontine glioma.

Diagnostic evaluation:-

serological examination, electrodiagnostic studies,and imaging are not normally necessary diagnose Bell's palsy. However, the reference to appropriate specialist (neurologist, otorhinolaryngologist gologist, optometrist, ophthalmologist) see if the patient's forehead is spared muscle, multiple cranial neuropathies, signs infection or persistent weakness without symptoms significant improvement after 3 weeks.

Laboratory testing:-

Complete blood count with differential may indicate infection or lymphoproliferation disorder. When indicated, screening for diabetes mellitus with fasting blood glucose or hemoglobin A1c may be helpful. In Lyme - in endemic areas, patients should undergo an enzyme immunosorbent assay or in- direct fluorescent antibody test for screening disease. If positive, diagnosis of Lyme the disease should be confirmed by Western blotting.If vesicles are present on examination, check serum antibodies to herpes zoster. In the app - appropriate clinical setting, angiotensin-converting enzyme, human immunodeficiency virus and inflammatory markers can be tested.Cerebrospinal fluid analysis is general it will not help diagnose Bell's palsy, but it can distinguish it from Guillain-Barré syndrome,leptomeningeal carcinomatosis and infection involving the central nervous system.

Display

Viewing is not recommended at the beginning assessment of Bell's palsy if there are no symptoms and examinations are atypical. From 5% to 7% of cases of facial palsy is caused by a tumor (e.g. facial neuroma, cholesteatoma, heman-gioma, meningioma), whether benign or ma-lignant.Therefore, in patients with insidise onset of symptoms that do not improve about 3 weeks, calculated with increased contrast tomography or gadolinium magnetic resonance imaging of the inner ear channel and face is guaranteed.

Electrodiagnostic studies

Electrodiagnostic testing is usually not included assessment of acute Bell's palsy, but in it can help patients with complete paralysis assess the degree of nerve damage and chances of recovery, especially from patients with complete paralysis have a higher risk incomplete recovery.Electrodiagnostic studie should be performed at least 1 week after onset of symptoms to avoid false negative results.

TABLE 3

Bell palsy treatment recommendations for adults presenting within 72 hours

Class of medication	Recommendation grade	Examples
Corticosteroids	A (established as effective)	Prednisone 50 mg orally daily for 5 days, followed by 10 mg less each day for 5 days
		Prednisolone 50 mg orally daily for 10 days
Antivirals[a]	C (possibly effective)	Valcyclovir 1 g three times daily for 7 days[b]
		Acyclovir 400 mg five times daily for 7 days[b]

TREATMENT :-The treatment of Bell palsy focuses on maximizing recovery and minimizing associated complications.Protect the eyes Patients who cannot completely close their eyes should be given instructions on ocular protective care to prevent exposure keratopathy. Frequent application of lubricant eyedrops with artificial tears during the day or ophthalmic ointment at bedtime is recommended. The physician should also recommend protective eyewear such as sunglasses during the day. Eye patching or taping at night may be useful but could be harmful if applied too loosely or too tightly. Patients with vision loss or eye irritation should be referred to an ophthalmologist.Corticosteroids are recommended in the first 72 hours In two randomized clinical trials (conducted by Sullivan et al20 in 511 patients and Eng-ström et al21 in 829 patients), prednisolone was found to be beneficial if started within 72 hours of symptom onset. In a double-blind, randomized, placebo-controlled study of prednisone in 58 patients,those who received the drug recovered faster, although long-term outcomes in these patients were not significantly different than those in the control group.22 The American Academy of Neurology23 rated this study as class II, ie, not meeting all of its criteria for the highest level of evidence, class I. Nevertheless, al-though prednisone lacks class I evidence, its use is recommended because it is a precursor to its active metabolite, prednisolone, which has been studied extensively. The current guidelines of the AmericanAcademy of Neurology, updated in 2012, state, “For patients with new-onset Bell palsy, steroids are highly likely to be effective and should be offered to increase the probability of recovery of facial nerve function”23 (leveEvidence, i.e. proven to be effective). They also concluded that the adverse effects of corticosteroids were generally small and temporary.Similarly, the directive of the Amer Academy of Otolaryngology - Head and Neck Surgery, published in 2013, recommend oral corticosteroids within 72 hours of onset symptoms of Bell's palsy in patients aged 16 a older.The

recommendation is for 10 days a course of corticosteroids lasting at least 5 days high dose (prednisolone 50 mg orally daily for 10 days or prednisone 60 mg orally daily for 5 days followed by a 5-day taper). The benefit of corticosteroids after 72 hours is not clear.

Although the guidelines recommend corticosteroids, the decision to use them should diabetics and pregnant women be individualized. Discretion is advised because no all patients with Bell's palsy must be treated.Most recover spontaneously, especially those with mild symptoms.

Antiviral therapy may offer modest benefit:-

Antiviral therapy has not been proven beneficial in Bell's palsy and current guidelines do not recommend an oral antiviral alone. However, the antiviral in combination with corticosteroids may offer modest benefit if initiated within 72 hours of onset of symptoms (level C evidence, i.e. possibly effective).patients starting antiviral therapy should it stands that its benefit has not been proven.Surgical decompression remains controversial A 2011 Cochrane systematic review found insufficient evidence of safety and effectiveness of surgical intervention in Bell's palsy.Surgery should only be considered in patients

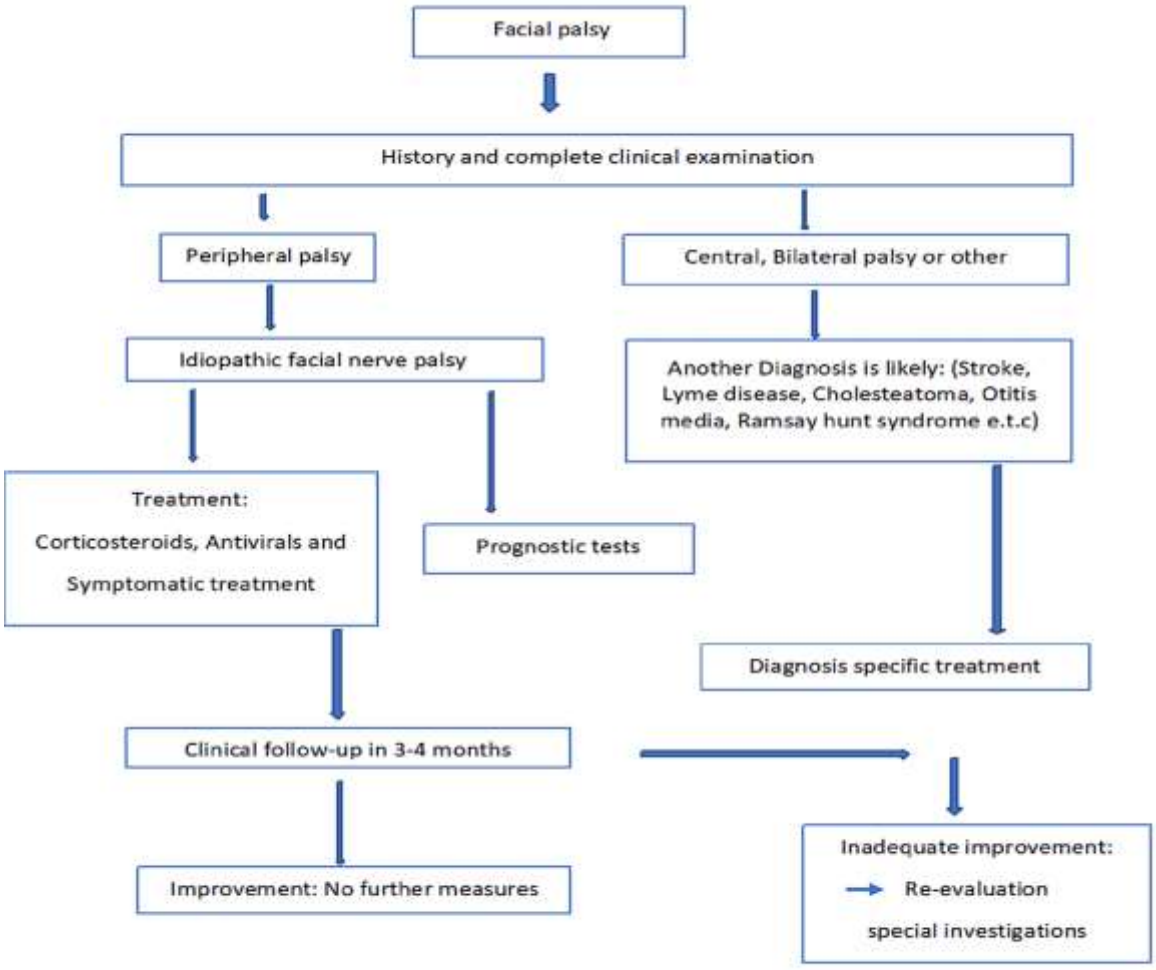
with complete paralysis with greater than 90% reduction in motor amplitude on the nerve conduction studies compared to non affected side and absence of free activity on needle examination. Acupuncture: No recommendation There is currently no recommendation for acupuncture in the treatment of Bell's palsy.A recent randomized clinical trial suggests effect of acupuncture in combination with corticosteroids, but high-quality studies to support its use is missing. Physical therapy: Insufficient evidence There is not enough evidence to prove this Does physical therapy benefit—or harm—vs Bell's palsy. However, some poor quality studies indicated that facial exercises and facial expression may improve function in patients with central paralysis.

Follow:-

Patients should be instructed to call after 2 weeks report progression of symptoms and beevaluated within 1 month or within 1 month, with deadline tendency to facial weakness and eye irritation.If occurred, further evaluation is required no improvement if symptoms worsen or if new symptoms have developed. The psychosocial impact of Bell's palsy cannot be discounted as disfigurement can have negative consequences for self-esteem and social relations. Suitable recommendations for an ophthalmologist, neurologist, otolaryngologist essence, social worker or plastic surgeon necessarily.

■ COMPLICATIONS AND PROGNOSIS:-

Most patients with Bell's palsy recover completely, but up to 30% have residual symptoms at 6 months. Furthermore, although Bell polio usually has a monophasic course, up to 7%12% of patients have a recurrence. Long-term complications may include repeated sidual facial weakness, facial synkinesis, facial contracture and spasm. Incomplete closure of the eye may benefit from surgery (tarsor-raffia or gold weight implantation) ventilate corneal ulceration. Facial synkinesis is as a result of aberrant regeneration of nerves and occures in 15% to 20% of patients after recovery from Bell's palsy.Patients may describe lacrimation while chewing ("crocodile tears"), involuntary movement of the corners of the mouth blinking or ipsilateral eye closure when the jaw opens ("jaw wink"). facial contracture,facial synkinesis and facial spasms can be treateded by botulinum toxin injection.



Clinical decision-making and Management of Facial palsy

Conclusion:- Bell's palsy is an ipsilateral, idiopathic, and acute lower motor neuron paralysis of the seventh cranial nerve which causes weakening of the platysma and facial muscles and significantly affects the patient's appearance and living standards and psychosocial well-being. Symptoms begin with mild facial weakness muscles without any neurological abnormalities and peaks in the first week and then gradually declines three weeks to three months even without any medical care, but it can lead to various complications.and leave the patient with varying degrees of residual paralysis if early diagnosis and intervention are not made accepted. It can affect any age and affects both sexes equally, although its incidence peaks in the 40s often occurs in people with diabetes.The diagnosis is one of exclusion and requires careful anamnesis and a thorough clinical examination. If established history or risk factors may suggest testing for Lyme disease and diabetes. Incomplete closure eyelid with resultant dry eye, dysphagia, and slurred speech are common short-term complications. An less frequent long-term complications are contractures and permanent weakening of the facial muscles. Although most patients undergo spontaneous recovery, treatment with a short course of valaciclovir or acyclovir and tapering dose of prednisone, started within three days after the appearance of symptoms, is considered to reduce the time and chances of a full recovery.

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