A Systemic Approach to Facial Nerve Paralysis

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Abstract

Purpose. To present systemic approach and recognition of red flags in facial nerve paralysis.

Background. Patients with facial nerve paralysis commonly present themselves to the primary care physicians. Misdiagnosis of other causes of facial nerve paralysis as Bell’s palsy is not uncommon. Occult malignancy can present with acute facial paralysis resembling Bell’s palsy; with no other clinical findings and normal imaging findings. A systematic diagnostic approach and recognition of red flags is important.

Conclusion. A careful search for the etiology of facial paralysis will avoid misdiagnosis and medicolegal complications. Bell’s palsy should always remain a diagnosis of exclusion.

Introduction

Facial nerve is the seventh cranial nerve. From its origin in the lower pons, the facial nerve runs a complex course which consists of pontine, subarachnoid, intratemporal and extratemporal portions. Patients with facial nerve paralysis commonly present themselves to the primary care physicians with unilateral facial drooping. The commonest cause of acute facial paralysis is Bell’s palsy. Although Herpes Simplex virus type-1 is postulated to be the cause, Bell’s palsy remains a diagnosis of exclusion. A large number of Bell’s palsy patients are managed at the primary health care level. Bell’s palsy is considered by many physicians as a straightforward diagnosis that is easy to manage. However, misdiagnosis of other causes of facial nerve paralysis as Bell’s palsy is not uncommon. Morris et al found that as high as 28% of patients had symptoms not attributable to facial nerve paralysis in patients diagnosed with Bell’s palsy. These include limb paresis, limb paraesthesia and clumsiness. On the other hand, Boahene et al and Quesnel et al reported that occult malignancy can present with acute facial paralysis resembling Bell’s palsy; with no other clinical findings and normal imaging findings. The neuroanatomy of the facial nerve is complex but well characterized. Collateral damage to the many structures along its course provides important clues for the localization of pathological processes. A systematic diagnostic approach and recognition of red flags is important in the approach of facial nerve paralysis.

Methods

CLINICAL EVALUATION OF FACIAL PARALYSIS

The standard order of management includes history, clinical examination, relevant investigations and treatment. An overview management flowchart is helpful (Illustration 1).

The differential diagnoses of facial nerve palsy are numerous. An extensive review of medical literature from 1900 to 1990 by May and Klein found more than 50 causes of facial paralysis. Upper motor neuron facial paralysis (UMNL) is caused by diffuse or localized intracranial pathologies. The causes of lower motor neuron facial paralysis (LMNL) can be similarly divided into systemic and local pathologies (Illustration 2).

Clinical examinations should include a complete neurological and systemic examination, otoscopy of the external auditory canal, the regional skin, lymph nodes and the parotid gland. The diagnosis of Bell’s palsy should be considered last after excluding all other possible causes. Nine red flags which guard against the diagnosis of Bell’s palsy are the presence of 1) facial pain, 2) limb weakness, 3) paresthesia of the face of limbs, 4) cerebellar signs, 5) involvement of other cranial nerves, 6) gradual onset of facial weakness, 7) previous history of facial weakness, 8) previous history of regional skin cancer, and 9) prolonged facial paralysis beyond 6 months.

CLINICAL EXAMINATION FOR FACIAL MUSCLE WEAKNESS

The motor root of the facial nerve innervates striated muscles of the second pharyngeal arch, which include the muscles of facial expression (Illustration 3). The five terminal branches of the facial nerve are the temporal, zygomatic, buccal, marginal mandibular and cervical. The temporal branch innervates the forehead muscles and the superior part of the orbicularis oculi. The zygomatic branch innervates the muscles of the nasolabial fold. The buccal branch innervates the buccinator and the orbicularis oculi. The marginal mandibular branch innervates the depressors of the mouth and the cervical branch innervates the platysma muscle.

The forehead is controlled by the frontalis, corrugators...
superciliii and procerus muscles. Clinically, the frontalis muscle is tested by asking the patient to wrinkle the forehead. Asymmetry or weakness of the forehead wrinkles points towards LMNL, whereas UMNLP spares the forehead muscles (Illustration 4).

A weakened orbicularis oculi will cause lagophthalmos and in severe cases, paralytic ectropion. The power of the orbicularis oculi is examined clinically by asking the patient to close his or her eyes tightly. Inspect for incomplete closure and incomplete buring of eyelashes. The eyes are forced open for muscle tone asymmetry.

The nasolabial folds are maintained by the zygomaticus major, zygomaticus minor, levator labii superioris and levator labii superioris alaeque nasi muscle. These muscles are tested as a group by asking the patient to smile, show his or her teeth or pull back the corners of the mouth. The buccinators, by blowing the cheek. The orbicularis oris, by puckering the lips. Depressing the corners of the mouth tests the group of depressor anguli oris, depressor labii inferiorioris and the mentalis muscles. The platysma is usually activated while depressing the corners of the mouth.

Subtle weakness of the muscles of facial expression is noted in the asymmetry of the nasolabial folds and the mouth while the eyes are closed tightly. The most subtle signs of facial weakness are the blink reflex and incomplete lid closure. The blink reflex is observed during conversation, or tap gently on the glabella. In cases with strong suspicion of facial muscle weakness, the patient is asked to lie supine, with head slide-off the examining table so the head is below the body. This forces the eyelids to work against gravity. The patient is asked to close both eyes and inspect for incomplete closure. Gentle glabella tap will elicit asymmetry in blinking.

MEASUREMENT OF FACIAL NERVE FUNCTION 7
Measurement of facial nerve function is important for baseline documentation, monitoring of progression or recovery of facial paralysis and for the comparison of different treatment modalities. The House-Brackmann grading system has been accepted by the American Academy of Otolaryngology-Head and Neck Surgery as the standard used in reporting facial nerve function. Although disfiguring facial asymmetry is alarming to most patients, the most devastating complication is exposure keratopathy which may result in corneal ulceration and blindness. Patients with grade 1 – 3 are able to complete close their eyes (Illustration 5).

OCULAR PROTECTIVE MECHANISMS AND COMPLICATIONS
This is best performed by the ophthalmologists. Five important ocular protective mechanisms are eyelid motility and closure, corneal sensation, blink reflex, Bell’s phenomenon and tear production. Patients with grade 3 are only able to achieve complete eyelid closure with maximal effort, hence are at risk of exposure keratopathy especially in the absence of Bell’s phenomenon.

NEUROANATOMICAL LOCALIZATION
The diagnostic flowchart for syndromes of UMNLP and LMNL facial paralysis are presented (Illustration 6 and 7). The neuroanatomical details are beyond the scope of this manuscript. Readers are advised to refer to standard neuroanatomy texts for more complete references. Further information is provided by the excellent works of Nicolai et al 8, Terao et al 9 and Kim J 10.

In the temporal bone, the facial nerve takes a serpentine course. In progressive order, the facial nerve gives off parasympathetic fibers, motor branch to the stapedius muscle, receiving taste fibers from the anterior two-third of the tongue (via chorda tympani) and general sensory fibers. Historically, topognostic tests were use to pin point the location of intratemporal lesions. The principle is that lesion distal to the site of a particular branch of the facial nerve will spare the function of that branch. Recent evidence showed that topognostic tests are of limited clinical value due to marked discrepancies. Inflammation and demyelination may involve multiple sites. The parasympathetic fibers and chorda tympani nerves are damaged more easily in trauma despite intact facial nerve. Transmission of nerve impulses occur through the tumor mass itself until late in the disease with different areas of the nerve being affected at different times.11

BELL’S PALSY- A DIAGNOSIS OF EXCLUSION
Bell’s palsy is an idiopathic condition characterized by acute, isolated, unilateral LMNL facial nerve palsy involving all the branches of the facial nerve. The weakness is maximal within 48 hours after onset. The Copenhagen Facial Nerve Study showed 85% of patients had functional recovery within 3 weeks and in the remaining 15% after 3 to 5 months.12 Any deviation from the above characteristics warrants further investigations for an underlying cause. Boahene et al reported 15 cases of occult neoplasm presented with acute facial paralysis.3 Eleven patients were misdiagnosed as Bell’s palsy.

Pain is a common feature in Bell’s palsy. About 70% of patients report pain in or around the ear.2 Pain becomes suspicious if it occurred elsewhere or persistent beyond facial paralysis. Malignancy of the parotid gland is highly suggested by the triad of ear pain, facial paralysis and sensory loss in the second and third divisions of the trigeminal nerve.
Neuroimaging may fail to detect occult malignancy because of the predominant perineural, intraneural, and perivascular spread of cancer, failure to scan the entire course of the facial nerve and failure to identify small parotid gland tumor located in the deep lobe or close to the stylomastoid foramen. Surgical exploration of the facial nerve is indicated if there is a progressive and prolonged pattern of paralysis without recovery, pain, involvement of other cranial nerves, and a history of regional skin cancer.

ROLE OF PRIMARY CARE PHYSICIANS

Primary care physicians are in the front line of management. A careful search for the underlying etiology will avoid misdiagnosis and possible future medicolegal complications. Ocular protective therapy should be initiated as soon as possible with preservative-free artificial tear during daytime and ointment during sleep. Referral to the respective specialty (neurologist, ophthalmologist, otolaryngorhinologist, physiotherapist) are advocated even in Bell's palsy, as electrophysiology tests performed at early stage of the disease are important in prognostication. Surgical decompression of the facial nerve may be considered in total facial paralysis by some otolaryngorhinologist. In the case of Bell's palsy, oral prednisolone 60 mg daily should be initiated as soon as the diagnosis is made (if there is no contraindications). Axelsson et al. found that treatment with prednisolone within 48 hours achieve higher complete recovery rate in patients above the age of 40 and less synkinesis in those younger than 40 year old.13 The oral prednisolone dosing regime was 60 mg daily for the first 5 days, then reduce 10 mg daily for the next 5 days, with a total treatment time of 10 days. Acyclovir and valacyclovir was not proven to be effective.14,15 Physiotherapists play an important role in the early facial rehabilitation to prevent facial synkinesis and contracture. Synkinesis is due to permanent aberrant regeneration circuits in the peripheral facial nerves. Synkinesis causes face distortion and asymmetry at any attempted movement. Contracture is due to fixed muscle-shortening which cause facial deformity even during resting conditions. Incorrect rehabilitative technique will aggravate abnormal movement pattern and reinforce aberrant circuits of synkinesis with eventual contracture.16 Patients with mild or moderate Bell's palsy managed by the primary care level should be followed up for improvement in facial nerve function. Prolonged facial paralysis beyond 6 months warrants further investigations for an underlying cause. Incomplete recovery of facial function can be treated with various facial reanimation surgeries.

Conclusion(s)

A careful search for the etiology of facial paralysis will avoid misdiagnosis and medicolegal complications. Bell’s palsy remains a diagnosis of exclusion and its management is multidisciplinary. The nine red flags which guard against the diagnosis of Bell’s palsy are the presence of 1) facial pain, 2) limb weakness, 3) paresthesia of the face of limbs, 4) cerebellar signs, 5) involvement of other cranial nerves, 6) gradual onset of facial weakness, 7) previous history of facial weakness, 8) previous history of regional skin cancer, and 9) prolonged facial paralysis beyond 6 months.

Abbreviation(s)

UMNL: Upper motor neuron lesion
LMNL: Lower motor neuron lesion

Authors Contribution(s)

Tan Aik Kah & Faridah Hanom Annuar: equal contribution in the preparation of the manuscript.

References


Illustrations

Illustration 1

Overview management of facial paralysis

- Facial Paralysis
- Diagnosis: History, Clinical examination, Investigations
- House-Brackmann grading of facial nerve function
- Ocular protective mechanisms & complications: UMNL - low risk, LMNL - high risk
- Treatment: Underlying cause & Ocular management
- Follow-up: Facial rehabilitation

Illustration 2

Causes of facial paralysis

- Causes of Facial Nerve Paralysis
- UMNL
  - Diffuse process: Meningitis, Encephalitis
  - Local pathologies: Space occupying lesion, Cerebrovascular accident, Brainstem vascular accident, Trauma
- LMNL
  - Systemic: After EXCLUSION of all pathologies
  - Local pathologies
  - Bell’s palsy: Acute onset, Unilateral, Isolated facial nerve LMNL

**RED FLAGS**
- Pain
- Paralysis of limbs
- Paresthesia of face or limbs
- Taste alteration
- Progression: gradual onset, involvement of other cranial nerves
- Previous facial paralysis
- Previous regional skin cancer
- Persistent facial paralysis beyond 4 months
Illustration 3

Muscles of facial expression with their respective innervations

Illustration 4

Bilateral supranuclear innervation to muscles of forehead and eyes
Illustration 5

Simplified House-Brackmann grading system for facial nerve function

Illustration 6

Diagnostic flowchart for syndromes of UMN facial paralysis
Illustration 7

Diagnostic flowchart for syndromes of LMNL facial paralysis
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