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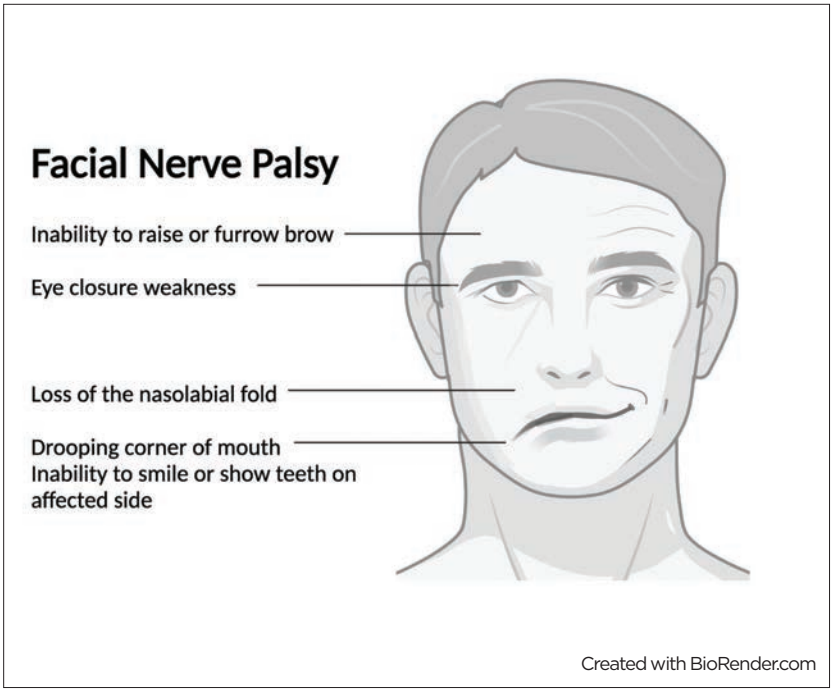


FIGURE 1
Clinical features of a lower motor neurone facial nerve palsy

How should patients be examined?

What are the differential diagnoses?

Which patients should be referred?

BELL'S PALSY IS THE MOST COMMON CAUSE OF FACIAL PALSY AND PRESENTS AS A UNILATERAL LOWER MOTOR neurone facial weakness in association with sensory and parasympathetic dysfunction.[1] The annual incidence of Bell's palsy is around 38 per 100,000 in the UK.[2,3]

The Scottish anatomist Sir Charles Bell first demonstrated in animal dissection studies during the 1800s that cutting the seventh cranial nerve resulted in an ipsilateral facial palsy.[4]

Although the severity of facial weakness in Bell's palsy varies, maximal facial weakness occurs within 72 hours and most cases recover over the following few weeks.[5,6]

Incomplete recovery, although uncommon, can result in permanent facial paralysis or abnormal reinnervation with considerable functional and aesthetic sequelae requiring long-term multidisciplinary team (MDT) support.

Bell's palsy is still considered to be idiopathic, in contrast to other facial nerve palsies with known infective, malignant or autoimmune causes. Most cases of Bell's palsy can be diagnosed and managed in primary care.

RISK FACTORS

Bell's palsy affects men and women equally and the incidence increases with age particularly around midlife.[6] The frequency of left- and right-sided weakness is similar.[7] Risk factors include: pregnancy, acute respiratory infection, immunocompromise, diabetes mellitus and hypertension.[3,8] Bell's palsy exhibits seasonal variation, increasing in colder months around the world.[7,9,10]

Risk factor reporting varies across studies, adverse prognostic factors include: decreased lacrimation, complete paralysis, advanced age, hypertension, diabetes mellitus and pain in areas other than the ear (including mastoid and facial pain).[3,4,7]

AETIOLOGY

The underlying pathophysiology of Bell's palsy is not fully understood and there are several causative theories relating to the basic mechanism of facial nerve inflammation, oedema and compression. The facial nerve is particularly vulnerable to compression and symptomatic ischaemia as it passes through the temporal bone in the narrow facial canal.[4,11,12]

The identification of herpes viral reactivation in a proportion of cases, particularly herpes simplex virus 1 (HSV-1) and varicella zoster virus (VZV), has led to the hypothesis that these are causative agents.[5,13,14] However, viral detection results vary across studies and notably in one study the presence of herpes viral DNA did not appear to influence clinical recovery outcomes.[5,15]

The first gene variant associated with Bell's palsy was recently identified in a meta-analysis, however the biological mechanisms underlying the increased associated risk are yet to be defined.[16]

DIFFERENTIAL DIAGNOSES

It is essential to consider alternative causes of acute peripheral facial palsy including: infection, malignancy and autoimmune disease (see table 1, p18).

The most common infective causes of acute peripheral facial palsy include herpes zoster (Ramsay Hunt syndrome) and Lyme neuroborreliosis.[17]

The diagnostic clue in Ramsay Hunt syndrome is herpetic lesions in the external ear canal, however these may appear after the facial palsy or not at all in the case of zoster sine herpete.[13,17]

Other distinguishing features of Ramsay Hunt syndrome include severe ear/postauricular pain and audiovestibular nerve involvement.[18] »

Lyme neuroborreliosis should always be considered for patients in an endemic area or who have characteristic features, so it is important to ask about recent travel, tick bites, rashes etc.

Multiple cranial palsies are suggestive of sinister causes such as compressive or infiltrative neoplastic lesions.

Systemic features may suggest basal meningitis caused by infective, malignant or inflammatory diseases.[17,18]

PRESENTATION

Bell's palsy is a clinical diagnosis. Patients typically present with an acute onset (ranging from a few hours to 72 hours) unilateral lower motor facial weakness (see box 1, below). The degree of weakness varies significantly and may be preceded or accompanied by: postauricular pain, altered taste, dry eye and hyperlacrimation.[21] These associated non-motor symptoms reflect the involvement of sensory and parasympathetic facial nerve pathways.[4] In some cases, onset may be so acute as to mimic stroke. Patients often complain of sensory symptoms affecting the face, although the facial nerve is purely motor as regards the face.

A clinical diagnosis in primary care is acceptable if the above features are confirmed but it is important to identify any unusual features, including red flags which require referral for further investigation (see table 2, p19).

FOCUSED HISTORY

The following key areas should be addressed when taking the history:

Timing

- Was the onset more than three days ago? Bell's palsy never presents insidiously.
- How has the weakness evolved over this time? Maximal weakness in Bell's palsy occurs within 3 days, deviation from this course is atypical.[1]

The frequency of recurrence ranges from 6.5 to 19.4% and recurrent episodes are associated with lower recovery rates.[22] Recurrence of symptoms within weeks or months should prompt further investigation.

Severity

- Is there incomplete eye closure?

All patients with eye closure weakness require an eye care regimen consisting of lubricating daytime eye drops with optional patching to protect against injury and eye ointment with taped closure at night. Inability to close the eye completely leaves the patient at risk of corneal ulceration and advice from an ophthalmologist may be required.[19]

Eating, communication and aesthetics

Patients may experience difficulty eating, drinking, or speaking. The psychopathology associated with facial disfigurement, even temporary, should not be underestimated and reassuring patients that a good prognosis is likely is important.

Additional associated symptoms

Accompanying symptoms include: postauricular pain in around half of patients, altered taste due to chorda tympani involvement in around a third, eye dryness in around a third and dry mouth in around a fifth.

Additional symptoms may include: facial pain, hyperlacrimation, aural fullness and loud noise intolerance (hyperacusis).[21]

Box 1

The clinical features of Bell's palsy: diagnostic criteria[18,20]

- Acute progressive (not spontaneous) onset over hours and up to 3 days
- Unilateral lower motor facial weakness affecting upper, mid and lower facial zones
- Maximal facial paralysis at 72 hours before improvement within 4 weeks
- The absence of any symptoms and signs to suggest an alternative cause

Table 1

Alternative causes of acute unilateral lower motor neurone facial palsy[1,17-19]

Infective	Neoplastic	Inflammatory	Autoimmune	Metabolic	Trauma	Other*
Ramsay-Hunt syndrome	Facial nerve schwannoma	Sarcoidosis	Guillain–Barré syndrome	Diabetic mononeuritis	Temporal bone fracture	Melkersson-Rosenthal syndrome
Zoster sine herpete	Head/neck cancer and facial nerve infiltration e.g. parotid carcinoma	Vasculitis	Multiple sclerosis		Post-operative	Cholesteatoma
Lyme neuroborreliosis	Cerebello-pontine angle tumours e.g. acoustic neuroma	Basal meningitis	Miller Fisher syndrome			
HIV, EBV, Zika	Cerebral or leptomeningeal metastatic spread					
TB, polio, syphilis						
Otitis media/ malignant otitis externa/ mastoiditis						
Basal meningitis						

*Rarely a focal pontine lesion can result in isolated lower motor neurone facial weakness, the spontaneous onset would differentiate this from other causes[18]

Table 2

Red flags suggesting an alternative cause requiring urgent referral [20]

History red flags
- Insidious onset (weeks/months)
- Prolonged evolution of symptoms/lack of improvement/relapses
- Recurrence within weeks/months
- Audiovestibular (vestibulocochlear nerve) symptoms; hearing loss, tinnitus, vertigo
- Systemic symptoms (infective/immune) e.g. rash, fever, weight loss
- History of malignancy, stroke or trauma
- HIV risk factors
- Lyme disease risk factors (exposure in endemic area and symptoms/signs)

Examination red flags
- Any other cranial nerve deficits
- Any new or altered preexisting neurological signs
- Abnormal systemic examination findings; potentially malignant skin lesions, vesicular rashes, parotid gland masses

Isolated facial nerve deficits
In particular the absence of diplopia, dysphagia or any audiovestibular symptoms such as hearing loss, tinnitus, vertigo. Patients may complain of altered sensation which appears to be related to the weakness.

Systemic symptoms
Enquire about, and exclude, features of systemic disease (e.g. weight loss, fever, night sweats, general unwellness etc).

Past medical history
Malignancy, particularly cutaneous malignancy affecting the head and neck, a history of trauma, previous stroke or other vascular risk factors should be noted.

EXAMINATION

A diagnosis of Bell's palsy can only be made in the absence of other neurological or systemic signs suggestive of an alternative cause.

General head and neck
It is important to inspect the skin for lesions, check the anterior two thirds of the tongue and soft palate for herpetic lesions, palpate to check for a parotid gland mass and enlarged lymph nodes.[18] The patient should be assessed for otitis media or vesicles in the external auditory canal.

Cranial nerve examination
This should be carried out at rest. The patient should be assessed for loss of tone evidenced by drooping of the eyebrow, loss of forehead wrinkles, loss of the nasolabial fold, and drooping of the corner of the mouth (see figure 1, p17).

Facial asymmetry and unilateral lower motor neurone weakness across the upper, middle and lower facial zones should be looked for. The House-Brackmann grading system has been widely used as a study outcome measure to grade facial weakness (I-normal facial function to VI-total paralysis) and is a useful reference during examination (see below).[23]

The House-Brackmann grading system[23]
I Normal symmetrical function in all areas
II Slight weakness on close inspection, able to close eye
III Obvious but not disfiguring weakness
IV Obvious disfiguring weakness and unable to close eye
V Barely perceptible movement
VI No movement

The patient should be assessed at rest and also when the face is expressive. Comparison with previous photographs may be helpful.

Assessment of facial expression
- Inability to raise or furrow the brow on the affected side (frontalis weakness). Note that forehead sparing is generally seen in upper motor neurone/central facial weakness due to the bicortical representation of the forehead. In milder cases lower facial weakness may be more apparent than forehead weakness
- Ask the patient to smile, show their teeth, whistle, puff their cheeks out. Orbicularis oris weakness causes the mouth to be drawn to the unaffected side
- Eye closure weakness. Orbicularis oculi weakness may also cause excess lacrimation as tears cannot be directed across the eye surface.[19]

The results of the rest of the neurological examination should be normal.

CONFIRMING DIAGNOSIS

In most cases of new onset facial palsy no further laboratory, electrodiagnostic or imaging investigations are required although blood glucose to exclude diabetes is a reasonable precaution to take.[12]

Patients should be referred in the following situations:
- Urgent referral to neurology or ENT, depending on local referral pathways, if there is uncertainty about the diagnosis or there are atypical features[20]

In moderate to severe cases of Bell's palsy:
- Urgent referral to an ophthalmologist if there is concern about eye safety with incomplete eye closure
- Psychological support should be considered
- Referral to a plastic surgical facial reanimation service should be considered if patients have persisting facial weakness several months following symptom onset.[24]

MANAGEMENT

Current NICE guidance recommends early treatment with a short course of high-dose prednisolone. Treatment should be started within the first 72 hr of presentation or as soon as possible within the first 2 weeks at a dose of 50 mg daily for 10 days or 60 mg daily for five days followed by a daily reduction in dose of 10 mg (for a total treatment time of 10 days).[20]

While corticosteroids alone have been shown to reduce the rate of incomplete recovery compared with antivirals alone, the evidence to support dual therapy, i.e. prednisolone plus an antiviral is unconvincing.[25,26]

The latest Cochrane meta-analysis reported, with a moderate level of confidence, that combined therapy reduced the number of long-term effects such as facial movement abnormalities compared with steroid monotherapy.[27] This meta-analysis was significantly limited following the exclusion of small studies at high risk of bias.

The lack of demonstrable combined therapy efficacy may also reflect disease heterogeneity, with viral reactivation including zoster sine herpete representing only a subset of cases. Antiviral therapy does not appear to be associated with adverse effects and so it may be reasonable to treat patients empirically with the features of zoster sine herpete such as more severe facial weakness and pain with dual therapy.[18,26]

Surgical decompression of the facial nerve as an alternative acute »

key points

SELECTED BY

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Bell's palsy is the most common cause of facial palsy and presents as a unilateral lower motor neurone facial weakness in association with sensory and parasympathetic dysfunction. Maximal facial weakness occurs within 72 hours and most cases recover over the following few weeks. In some cases, onset may be so acute as to mimic stroke. The degree of weakness varies significantly and may be preceded or accompanied by: postauricular pain, altered taste, dry eye, and hyperlacrimation.

Bell's palsy affects men and women equally and the incidence increases with age particularly around midlife. The frequency of left- and right-sided weakness is similar. Risk factors include: pregnancy, acute respiratory infection, immunocompromise, diabetes mellitus and hypertension.

It is essential to consider alternative causes of acute peripheral facial palsy including: infection, malignancy and autoimmune disease. It is important to identify any unusual features, including red flags which require referral for further investigation. Urgent referral to neurology or ENT, depending on local referral pathways, is warranted if there is uncertainty about the diagnosis or there are atypical features.

All patients with eye closure weakness require a protective eye care regimen. In moderate to severe cases of Bell's palsy an urgent referral to an ophthalmologist should be made if there is concern about eye safety with incomplete eye closure. The psychopathology associated with facial disfigurement, even temporary, should not be underestimated and reassuring patients that a good prognosis is likely is important.

NICE guidance recommends early treatment with a short course of high-dose prednisolone. Treatment should be started within the first 72 hr of presentation or as soon as possible within the first 2 weeks. Antiviral therapy does not appear to be associated with adverse effects so it may be reasonable to treat patients with the features of zoster sine herpete such as more severe facial weakness and pain with dual steroid and antiviral therapy.

Adverse prognostic factors include: decreased lacrimation, complete paralysis, advanced age, hypertension, diabetes mellitus and pain in areas other than the ear (including mastoid and facial pain). Between 20 to 30% of patients are left with residual weakness and in around half of these cases the deficit is moderate to severe. Chronic facial muscle weakness can result in the development of spasm and contractures and long-term disability.

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intervention is not recommended.[12]

Between 20 to 30% of patients are left with residual weakness and in around half of these cases the deficit is moderate to severe.[24,28] Aberrant reinnervation may occur 5 months or more after symptom onset resulting in motor or autonomic synkinesis, for example jaw-winking or salivation induced facial sweating.[4,19] Chronic facial muscle weakness can result in the development of spasm and contractures and long-term disability.

MDT management involves a range of interventions including physiotherapy, which although not clearly evidence based, may be beneficial for patients with long-term complications.[12,19] Chemodenervation using botulinum toxin can be used for troublesome aberrant reinnervation.[19,29] The role of nerve grafting procedures remains uncertain.[24]

CONCLUSIONS

Bell's palsy is a clinical diagnosis and most cases can be managed with acute high dose steroid therapy, an eye care regimen and reassurance in the primary care setting. A short course of oral steroids improves recovery rates. Dual steroid and antiviral therapy may be beneficial however it is not possible to draw firm conclusions from the current evidence base.

The majority of patients will recover completely within a few weeks. However, complicated cases of Bell's palsy may need early specialist referral for MDT management.

Recurrence may occur in up to 20% of cases and should be managed in the same way as the primary episode.

Competing interests: None

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Useful information

Facial Palsy UK
www.facialpalsy.org.uk

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