key points

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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.