Are patients with Bell’s palsy receiving the right treatment?

The treatment of Bell’s palsy has been debated for decades. There is now good scientific evidence that early prednisolone increases the number of patients who recover, and reduces the number with lasting sequelae. Despite this, almost half of patients still receive no treatment whatsoever.

Bell’s palsy is an acute, unilateral peripheral facial nerve palsy of unknown aetiology. The disease is characterised by sudden unilateral paresis or paralysis of the facial muscles, which causes the face to droop. Patients have difficulty moving the mouth and closing the eye on the affected side. Other common symptoms include pain around the ear and reduced sense of taste on the palsied side.

The incidence of Bell’s palsy is approximately 30–40 per 100 000 person-years (1), which corresponds to 1 500–2 000 patients annually in Norway. The disease affects all age groups, but incidence increases with age (1). Approximately 70% of patients regain normal nerve function without any treatment over the course of six months, while the remaining 30% develop sequelae with permanent paresis of the facial muscles, muscle contractures and/or synkinesis (2).

Bell’s palsy accounts for around 70% of cases of peripheral facial nerve palsy (2), and is a diagnosis of exclusion made when other causes of acute peripheral facial nerve palsy have been ruled out. The most common known causes of peripheral facial nerve palsy are herpes zoster infection, neuroborreliosis, acute and chronic otitis media, trauma to the nerve and malignant tumours of the parotid gland. A peripheral facial nerve palsy affects all nerve branches on the palsied side, whereas in a central facial palsy, nerve function in the forehead remains intact.

There are several theories regarding the cause of Bell’s palsy. Reactivation of herpes simplex virus in connection with the facial nerve has been proposed (3), while an autoimmune origin has also been discussed. Another theory is that there is swelling of the nerve caused by an inflammatory reaction with secondary ischaemia. This is consistent with MRI studies which have shown oedema of the facial nerve within the temporal bone (4).

Prednisolone is effective

In an effort to shorten recovery time and increase the number of patients who regain nerve function, a number of therapeutic options have been investigated. Antiviral agents have been tried – based on the theory that the herpes simplex virus is the cause of the palsy – while several studies have administered cortisone to reduce the presumed inflammatory reaction and thus limit nerve damage. Up until the early 2000s, numerous small studies were carried out with both types of drugs, in the absence of any evidence that either had an effect (5, 6).

Clinical practice has varied as a consequence with some countries giving no treatment and others using cortisone and/or antiviral agents.

In the 2000s, however, several large randomised studies were conducted to examine the efficacy of cortisone and antiviral agents in adults with Bell’s palsy. There is now grade A evidence that treatment with prednisolone shortens recovery time and increases the proportion of patients who recover completely. Several meta-analyses have been published, from the Cochrane Group among others, which recommend prednisolone within 72 hours for adult patients (7, 8). There is currently insufficient evidence available, however, regarding the treatment of paediatric populations. The last few years have also seen the publication of review articles with guidelines on diagnosis and treatment, which again recommend early prednisolone (9–11).

These meta-analyses and reviews are based largely on one Scottish and one Scandinavian study. The Scottish study is a randomised, placebo-controlled, double-blind trial that compared the efficacy of prednisolone, aciclovir and combined prednisolone and aciclovir versus placebo in 551 patients (12). Prednisolone was administered at 50 mg per day and aciclovir at 2 000 mg per day for ten days. The results were published in 2007 and showed recovery in a significantly greater number of patients in the prednisolone group after three and nine months. Aciclovir had no effect, either as monotherapy or in combination with prednisolone (12).

In parallel with the Scottish study, a similar trial was conducted in Sweden and Finland (13). A total of 839 patients were randomised to receive placebo, prednisolone, aciclovir or combination therapy with prednisolone and valaciclovir. Prednisolone was given at 60 mg per day for five days and then tapered by 10 mg per day for a further five days. Valaciclovir was given at a dose of 1 gram three times daily for seven days. The results were published in 2008 and showed a significantly shorter recovery time and greater proportion of patients with normal nerve function after 12 months in the prednisolone group. Antiviral treatment with valaciclovir had no significant effect on recovery compared to placebo (13).

In both studies, treatment was initiated within 72 hours of the onset of the facial nerve palsy. Subgroup analyses from the Scandinavian study suggest that prednisolone is effective irrespective of palsy severity and in all age groups over 18 years. The results also suggest that prednisolone is more effective if given within 48 hours (14, 15).

Correct treatment?

To examine the impact of these studies on the management of Bell’s palsy, a recent study reviewed electronic medical records from 640 general practitioners in the UK for the period 2001 to 2012. The study showed an increase in the prescription of prednisolone for patients with Bell’s palsy after 2007, but also that 44% of patients still received no treatment whatsoever in 2012 (1). Some of these patients probably had other disorders that precluded the use of prednisolone. Prednisolone should be used with caution with stomach ulcers, psychoses, diabetes mellitus, kidney and heart failure and certain infections, for example. However, these relative contraindications can probably explain only a small proportion of the 44% who received no treatment (1). In agreement with Morales and co-workers (1), our explanation for the fact that many patients do not receive effective treatment is that the results of clinical trials have not reached health care practitioners. This demonstrates the importance of disseminating the results of international studies at a national level too in order to ensure that patients receive effective treatment.

There are no equivalent data from Norway, but we believe there are probably many patients who do not receive correct treatment here too, and who are thus at increased risk of permanently reduced nerve function with
the functional and psychosocial problems that entails.

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Received 24 February 2015, first revision submitted 4 May 2015, accepted 10 May 2015. Editor: Hanne Støre Valeur.