ROLE OF COTRTICOSTEROIDS IN THE TREATMENT OF BELLS PALSY

''META-ANALYSIS STUDY ''

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بسم الله الرحمن الرحيم

قُلْ إِنَّ صَلَاتِي وَنُسُكِي وَمَحْيَايَ وَمَمَاتِي لِلَّهِ رَبِّ الْعَالَمِينَ

{162} لَا شَرِيكَ لَهُ وَبِذَٰلِكَ أُمِرْتُ وَأَنَا أَوَّلُ

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List of abbreviations

ACTH: Adrenocorticotrophic hormone

B.P: Bell`s palsy

Chi²: Pearson Chi-square

CRF: chronic renal failure

CT: Computed tomography

df: degree of freedom

D.M: diabetes mellitus

EMG: Electromyography

ENoG: Electroneuronography

FPRP: facial paralysis recovery profile

H&B scale: House and Brackmann scale

HSV-1: herpes simplex virus type 1

HTN: hypertension

IAC: internal auditory canal

I²: useful statistic for quantifying inconsistency

IFNP: Idiopathic facial nerve paralysis

LMN: lower motor neuron

MEV: maximal excitability values

M-H:Mantel-Haenszel

MRI: magnetic resonance imaging

MST: maximal stimulation test

P values: illustrate level of significance for different outcome results, and measuring the heterogeneity

RCTs: randomized, controlled trials

RevMan: Review Manager Programme

RR: relative risk

Z: standard score

95% CI: confidence interval

INTRODUCTION

Idiopathic facial nerve paralysis, namely, Bell's palsy, is a non-life-threatening disorder that causes important functional, esthetic, and psychosocial disturbances to the patient **(Hughes, 1990)**. It has an acute onset and is almost unilateral (mononeuritis) with limited duration without detectable causes (**Diego, 1998**) and considered the most common cause of facial nerve paralysis. According to **Adour(1976),Grose(1973), Charous and Saxe (1962)and Bastian(1972)**, Bell's palsy is a part of cranial polyneuritis, often involving the trigeminal, glossopharyngeal, audiovestibular and the contralateral (clinically unaffected) facial nerves.

The natural history of Bell's palsy has been examined in an article by **Peiterson (1982)** who evaluated the outcomes of 1011 patients with Bell's palsy who were not medically or surgically treated. It was found that this condition occurs in every decade of life with a mean age between 40-44 years. It is less common before the age of 15 and after the age of 60 years. The incidence in men and women is similar. Approximately 6-9% develops recurrent Bell's palsy. Facial paresis alone occurred in 31% of patients, while the remainder had unilateral complete paralysis. Of those experiencing only a paresis, over 95% recover without sequelae. Of the remaining 69% with complete paralysis, some return of facial function is evident within 3 weeks in 85% and an estimated 71% of patients

achieve a House-Brackmann grade 1 and 13% a House-Brackmann grade 2. The remaining 16% in this complete paralysis group have a fair to poor recovery (House-Brackmann grades 3-5). This subset of patients is the controversial group of patients that may benefit the most from medical or surgical intervention.

Thus, of all patients with either a complete or partial facial paralysis, approximately 85% recover to normal within one year without treatment. It was noted that in patients that experienced delayed recovery over 3 months, all developed sequelae such as diminished function or contracture with associated movements. Return of at least some facial function was noted in all patients. Bell's palsy disproportionally attacks pregnant women, patients who have diabetes, influenza, a cold, some other respiratory alignment, or have undergone tooth root extraction **(Slavkin, 1999)**. Some patients report exposure to an air-condition outlet, or an open window before the attack **(Slavkin, 1999)**. Familial occurrence has been reported also **(Zaidi, 2005).**

ANATOMY

Knowledge of the anatomy of the facial nerve is important in understanding the pathophysiology and management of acute facial paralysis of Bell's type. The intracranial segment of the facial nerve and the nervus intermedius exit the brainstem adjacent to the pons, cross the cerebellopontine angle, and enter the internal auditory canal. The meatal segment of the facial nerve and the nervus intermedius remain in the anterior-superior quadrant of the IAC and enter the fallopian canal at the meatal foramen, superior to the transverse crest and anterior to the vertical crest (Bill's bar). **Ge** and **Spector** have determined that the narrowest portion of the fallopian canal is located at the meatal foramen (mean diameter .68 mm) **(Esslen, 1977)**. Thus, the size of the foramen coupled with a tight arachnoid band located at this segment contributes to the constriction of the facial nerve at this point in Bell's palsy. The labyrinthine segment of the nerve, encased within the narrowest portion of the fallopian canal, courses 2-4 mm to the geniculate ganglion, where the nerve takes an acute turn at the external genu to enter the middle ear. The tympanic segment (horizontal segment) courses a total of 11 mm slightly above the cochleariform process and oval window and turns into the second turn (pyramidal turn) inferior to the lateral semicircular canal. The mastoid segment (vertical portion) then descends 13 mm to the stylomastoid foramen **(Coker, 2001).**

Pathogenesis, etiology

Fortunately, the etiology of acute facial paralysis due to Bell's palsy is becoming clearer. Historically, there have been two prevailing theories: (1) vascular congestion with secondary ischemia to the nerve and (2) viral polycranioneuropathy. (**Hilger, 1949** and **Blunt, 1956).**The facial nerve travels through a narrow bony canal in the skull beneath the ear to the muscles on each side of the face. Acute inflammation and edema of the facial nerve are thought to lead to entrapment of the nerve in the bony canal, which leads to compression ischemia. An observation that supports the hypothesis of an inflammatory etiology is that the facial nerve shows enhancement on magnetic resonance imaging (MRI) in patients with acute Bell palsy **(Anwar, 2005).**

McGovern (1955) postulated autonomic vascular instability with spasm of the nutrient arterioles. This vasospasm would lead to ischemia, nerve edema, and secondary compression within the fallopian canal. The mechanisms responsible for such insults included cold temperatures or psychosomatic causes.

Antoni in 1919 proposed the term acute infectious polyneuritis cerebralis acusticofacialis**. McCormick (1971**) subsequently postulated the etiologic agent to be the herpes simplex virus. Recent studies by **Murakami (1996)** strongly suggest that herpes simplex virus type 1 (HSV-1) is active in

cases of Bell's palsy. In his study, endoneural fluid from 11 of 14 patients undergoing transmastoid decompression during the acute phase of the disease displayed DNA fragments of HSV-1 via the polymerase chain reaction. None from this surgical group displayed evidence of herpes zoster or Epstein Barr virus in the obtained samples. None of the affected controls undergoing surgery for temporal bone trauma or tumors displayed either HSV-1 or herpes zoster in their fluid samples either. Of the patients undergoing decompression for Ramsay Hunt syndrome, none exhibited DNA fragments of HSV-1 in their samples while all had herpes zoster. Indeed, **Schirm** and **Mulkins (1997)** stated that the Murakami study was so well controlled that "it provides conclusive evidence that reactivation of HSV genomes from the geniculate ganglia is the most important cause of Bell's palsy."

Even more compelling was the finding of HSV-1 DNA in a temporal bone section of a patient dying six days after developing Bell's palsy **(Burgess, 1994)**.An animal model for Bell's palsy has been developed in which animals inoculated with HSV-1 demonstrate transient facial paresis **(Sugita, 1995)**, thus further supporting the idea that Bell's palsy is the result of a viral inflammatory response that induces edema within the facial nerve.

A review of 12 autopsy cases of patients with Bell palsy was summarized in **Peter Dyck's Peripheral Neuropathy (2005)**, which stated that most cases showed inflammatory changes around the mastoid cells and walls of the arteries. The most common site of involvement was the geniculate ganglion. Surgical findings described constriction of the nerve at the stylomastoid foramen with swelling of the nerve itself. Microscopic findings showed an inflammatory reaction with infiltration of macrophages on the nerve.

In discussing the occurrence of the viral infection, the herpes virus have access to the body via the mucous membrane, and then travels up the axons of sensory nerves to take residence in the geniculate ganglion, this explains the numbness of the face and posterior pharynx and aberrant taste that often precede the Bell's palsy . With reactivation ; by a variety of stresses such as physical fatigue, psychological stress ,common cold, exposure to cold and dental treatment, the virus replicates within the ganglion cells ,then travels down the axons to cause edema in the myelin sheath of the nerve endings. As a result of this edema and compression of the nerve in the narrow fallopian canal, the nerve action potential can't be generated with demyelinization of the nerve and neurapraxia, but ultimately Wallerian degeneration **(Rosenberg, 1974)**