



Overview of Facial Nerve Paralysis in Saudi Arabia

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ABSTRACT

Background: Facial paralysis is a weakness of the seventh cranial nerve (CN VII), which causes a dysfunction in the facial muscles of expression mainly due to lower motor neuron lesion in the nerve. The most common causes of facial paralysis are Bell's palsy (60-75% idiopathic), infections, neuropathies, neoplasia, trauma, and congenital conditions. **Methodology:** This study is a simple review that accounted for the secondary data gathered from different sources, including databases, specialist organizations' reports (records of hospitals), articles, and earlier research done in Saudi Arabia and particularly in the healthcare facilities. The study aimed to review and identify the relevant literature that reported the prevalence of facial paralysis among their population with the possible prognostic factors. **Conclusion:** Our study indicated that the incidence rate of facial paralysis in Saudi Arabia is relatively rare and it gets lower rates among children. It also reported that most of the cases were exposed to cold air currents or experienced hearing loss, while the cases among children were mostly post-stroke facial paralysis or vascular ischemia. It is worth mentioning that there is a significant lack in the studies that assess the psychological state of facial paralysis patients and evaluate the outcomes and possible complications.

Key Words: Saudi Arabia, Facial Nerve Paralysis, CN VII.

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INTRODUCTION

Facial paralysis is a dysfunction of facial muscles of expression due to a lower motor neuron lesion that affects the CN VII (facial nerve). The lesion can be transient unilateral, or devastating permanent bilateral [1]. Temporal bone fractures, acoustic neuroma, tumor, or suppuration of the middle ear and parotid gland disorders mostly cause the localized lesions of the facial nerve [2-4]. The commonest cause of facial paralysis is a case called Bell's palsy (represents 73% of the cases), which can occur suddenly or arise gradually within months to develop head, and neck tumor, the less common causes include; infectious agents, neurologic conditions, neoplasia, trauma and congenital cases [5, 6].

The anatomy of the facial nerve makes it highly affected with various diseases, mainly because of the long intracranial, intra-temporal and extra-temporal courses of the

nerve that include three bends. The facial nerve includes motor, sensory and parasympathetic pathways; it is accountable for the voluntary and mimetic muscle movement of the face, taste sensation in the anterior 2/3 of the tongue, and to command the salivary and lacrimal glands' secretions [7].

The most prevalent causes of the sudden onset of the unilateral facial malady are stroke and Bell's palsy [8]. There are two ways to determine whether the facial palsy is peripheral or central, detecting the patient's history and neural examination. In case the weakness of the nerve is central, magnetic resonance imaging of the brain is essential to assess the patient for ischemia and for inflammatory diseases and infection. Otherwise, the facial weakness is peripheral with no obvious causes; then it is the case of Bell's palsy; approximately 60%-75% of Bell's palsy cases are idiopathic [9]. The annual incidence of Bell's palsy is quite rare, 30 cases/100,000 population, and

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it is responsible for about 70% of unilateral facial paralysis cases [7]. Bell's palsy affects both sexes. It has a median age of onset of 40 years, and it has lower rates of incidence among children [10]. The prognosis of Bell's palsy case is good, as the total recovery represents about 80% of the cases. There is a permanent nerve damage chance of 15%, and 5% of the patients undergo severe complications [11]. Considering that most of the cases of Bell's palsy are idiopathic, there are some possible pathomechanisms that may explain the peripheral neural lesion include Herpes simplex virus-1 infection, vascular ischemia, viral infection, or autoimmune disease. Bell's palsy irregularly affects pregnant women, diabetic patients, patients infected with influenza viruses or respiratory viruses, and patients who had a tooth root extracted [12].

Infections are the second common cause of facial paralysis. The infectious agents include varicella-zoster virus (VZV), which is commonly correlated with Ramsay Hunt syndrome and is characterized by peripheral facial nerve paralysis. It is also correlated with zoster vesicles on the ear [13]. Otitis media (OM) complications can cause facial paralysis. The hazard of neural lesion exists in acute supportive OM, malignant OM, chronic cases of OM, and OM with effusion. The mechanism of the facial paralysis caused by OM is still unspecified, but studies mentioned that it might occur because of inflammatory edema, toxins, and infrequently result from compression of the nerve, e.g., cholesteatoma [13]. The bacterium *Borrelia burgdorferi* is the causative organism of Lyme disease. Humans usually get the infection by the bite of infected black-legged ticks. The complications can take place if left untreated, which include spread to the joints, heart, and, more importantly, to the nervous system. Lyme disease-associated facial paralysis, specifically in children, has been reported [2, 14]. Peripheral neuropathies can also occur due to Human immunodeficiency virus (HIV) infection; typically, facial paralysis occurs in the early stages of the infection. The mechanism of the neuropathy becomes clear in the late stages, as the greater the immunodeficiency, the more the incidence of facial paralysis due to secondary opportunistic infections. The infections also include; Tuberculosis, Mumps virus, Rubella virus, influenza virus, and infectious mononucleosis [13].

Incidence of trauma can result in facial paralysis, and it includes; blunt, penetrating, and iatrogenic types. Traumatic accidents to the CN VII are mostly due to fractures of the temporal bone, about 5% of these fractures comprise the otic capsule, and half of those lead to facial nerve injury [13].

The importance of asking for the patient's history exists in that some of them have a neoplastic history that may cause facial paralysis. The most prevalent tumor causing facial nerve dysfunction is the acoustic neuroma or vestibular schwannoma (VS). Furthermore, the facial nerve itself is

mostly affected by facial schwannoma [15]. Tumors that originate from the cerebellopontine angle (CPA) or the internal auditory canal are frequently correlated with loss of hearing and tinnitus. If the tumor arises from the middle ear, it steadily evolves facial paralysis and may cause conductive hearing loss [16].

Facial paralysis in newborns is mainly because of traumatic or congenital causes. Oftentimes, congenital facial paralysis affects the lower lip with unilateral weakness. The presence of unilateral or bilateral paralyzes of the facial nerve is mainly due to a congenital syndrome called Möbius syndrome. The other congenital palsies involve Goldenhar syndrome, CHARGE syndrome, DiGeorge syndrome, and muscular dystrophy [13, 17].

METHODOLOGY

This study is a simple review based on the secondary data assembled from various sources, including databases, specialist organizations' reports (hospital records), articles, and earlier research done in Saudi Arabia and particularly in the healthcare reforms. This study aims to review and identify the relevant literature in Saudi Arabia that reported the prevalence of facial paralysis among their population with the possible prognostic factors.

Epidemiology in Saudi Arabia

Most of the studies that discussed the epidemiology of facial palsy conducted comparisons of its incidence among adults and children [1], both sexes, or considering the age of the patients [18].

In a study conducted in Arar, Northern Saudi Arabia, a total of 156 patients were included, of which 41 (26.3%) were positive for facial palsy. The mean age of the affected patients was 33.65 ± 11.71 , and it was more prevalent in females (61%) than males (49%). The study also indicated that facial palsy had sudden onset in 75.6% of the cases, the incidence in (92.7%) of the patients was due to exposure to cold currents, (34.1%) of the cases were related to hearing loss, and (24.4%) were associated with OM [19]. Another hospital-based prospective study detected the incidence of facial nerve paralysis among the population of the Asir region, Saudi Arabia, and found that the frequency of Bell's palsy to develop is 5.53/100,000 population annually, and the incidence increases with age [20]. In 1989, a study in Thugbah, Eastern Saudi Arabia, screened a population of 23,227, 12 cases, and reported Bell's palsy with a rate of 0.53 per 1,000 [21].

A retrospective study detected the medical records of 83,067 infants born in King Abdulaziz Medical City, Riyadh, Saudi Arabia, in the period between 1994 and 2005; It reported 29 infants diagnosed with traumatic facial weakness or paralysis with a rate of (0.03%) [22]. A study conducted at King Khalid University Hospital, Riyadh, KSA, investigated 104 Saudi children with stroke and

reported that 2 of them had unilateral facial nerve palsy, and one of them had temporoparietal ischemia and difficulty in speech [23]. A study in Northern Saudi Arabia investigated the recurrence of OM among 64 children and reported only one child with facial paralysis with a percentage of (1.6%) [24]. A very rare congenital condition of a 20-day-old Saudi girl referred to the Pediatric Otolaryngology service at King Abdulaziz Medical City was reported that Möbius syndrome had affected the facial nerve and caused facial paralysis [25].

Outcomes and complications

There are many different complications of facial nerve palsy. Keratitis, corneal dryness with possible ulceration due to the inability to close the eye of the paralyzed side, and scarring are the most common ophthalmic complications, which can be avoided with profound eye care. Patients should be informed on how to avoid corneal irritation by rubbing and being exposed to fans, winds, and cold air currents. In addition, they should be aware of the corneal irritation signs, including itching, redness, blurred vision, and foreign body sensation [26].

The other hyperkinetic complications include hemifacial spasm, which manifests unilaterally in one half of the face, and it is secondary to the axonal degeneration of the facial nerve [27]. Facial asymmetry constitutes psychological distress on the patient. Synkinesis is a voluntary muscle movement of the face followed by involuntary movements. Ocular-oral Synkinesis is represented by involuntary movement of the mouth accompanied by eye closure [28]. In line with outcomes, facial paralysis was found to have psychological effects on patients such as attentional distraction, contemptible social perception with patients who look less attractive, and in result, having a lower quality of life. Moreover, facial paralysis makes patients lose their self-esteem, be more anxious, have higher rates of depression, and decrease quality of life, particularly in female patients [29].

Management

Facial nerve paralysis needs optimizing health care for the patients. The management team should include otolaryngologists, ophthalmologists, and psychologists. Traditionally, the acute phase of facial paralysis requires removing any harmful agents, as in the case of acute OM. The first line of treatment for facial paralysis is corticosteroids, and this is due to the edema, which is a primary cause of facial paralysis in Bell's palsy. Many studies and meta-analyses supported the fact that steroids may increase the recovery rates of the motor function of the nerve. Patients with corneal exposure need conservative care, which includes artificial tears, lubricants, and closed eyes taping to prevent corneal ulceration [30].

To defeat the viral infection of Ramsay Hunt syndrome, a combination of steroids therapy and daily acyclovir (about 800 mg) is required. Meta-analyses of prospective randomized control studies supported that antiviral treatment and acyclovir cause an improvement of the symptoms within the first 72 hours. Whereas acute OM and bacterial infections require intravenous antibiotic therapy [31]. The management of Lyme disease depends mainly on the patient's age and the severity of the disease. When the disease is localized and affects individuals over the age of eight, daily doxycycline (200 mg) is applied for ten days. In case it affects patients younger than eight years, amoxicillin, or cefuroxime course is required to prevent possible tooth staining with the use of tetracycline [32]. Following the neurologic management of long-standing facial paralysis, surgical management is required. The most common uses of surgical management include facial nerve decompression, facial nerve repair techniques, nerve grafting options, and muscle transfer techniques, mostly with chronic facial paralysis patients [33].

CONCLUSION

The reported studies indicated that the rate of incidence of facial paralysis in Saudi Arabia is relatively rare, and it decreases among children. It also showed that most of the cases were exposed to cold air currents or experienced hearing loss, while the cases among children were mostly post-stroke facial paralysis or vascular ischemia. There is a significant lack of studies that assess the psychological state of facial paralysis patients and evaluate the outcomes and possible complications.

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