ORIGINAL ARTICLE

Facial nerve palsy: Analysis of cases reported in children in a suburban hospital in Nigeria

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Abstract

Aim: The study describes the epidemiology, treatment, and treatment outcomes of the 10 cases of facial nerve palsy seen in children managed at the Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife over a 10 year period. It also compares findings with report from developed countries.

Methodology: This was a retrospective cohort review of pediatric cases of facial nerve palsy encountered in all the clinics run by specialists in the above named hospital. A diagnosis of facial palsy was based on International Classification of Diseases, Ninth Revision, Clinical Modification codes. Information retrieved from the case note included sex, age, number of days with lesion prior to presentation in the clinic, diagnosis, treatment, treatment outcome, and referral clinic.

Findings: Only 10 cases of facial nerve palsy were diagnosed in the institution during the study period. Prevalence of facial nerve palsy in this hospital was 0.01%. The lesion more commonly affected males and the right side of the face. All cases were associated with infections: Mainly mumps (70% of cases). Case management include the use of steroids and eye pads for cases that presented within 7 days; and steroids, eye pad, and physical therapy for cases that presented later. All cases of facial nerve palsy associated with mumps and malaria infection fully recovered. The two cases of facial nerve palsy associated with otitis media only partially recovered.

Conclusion: Facial nerve palsy in pediatric patients is more commonly associated with mumps in the study environment. Successes are recorded with steroid therapy.

Key words: Children, facial nerve, malaria, mumps, Nigeria, palsy

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Introduction

Paralysis of the facial nerve (commonly referred to as Bell's palsy) is a well-recognized phenomenon. Roob *et al*.[1] reports that it occurs most commonly in children and young adults. Although sometimes idiopathic in nature, facial nerve palsy may result from an infectious or traumatic cause. Known infective cause include: Lyme disease, varicella, primary gingivostomatitis, herpes zoster oticus (Ramsay Hunt syndrome), coxsackievirus, trauma, otitis media, human immunodeficiency virus (HIV) infection, diseases causing tumors or demyelinations, compressions, and possibly Epstein-Barr virus.

Siwula and Mathieu[2] noted that in the past in the USA, facial nerve palsy was more often caused by bacteria infection of the middle ear. However, more recent studies show that Lyme disease has surpassed otitis media as a cause of the lesion. There were little or no observed gender variability in the presentation of facial nerve palsy in this region with about 85% of patients recovering within 2 months.[2-5]

The paralysis of the facial nerve disturbs motor function to the muscles of facial expression and results in a flaccid appearance of the face (unilateral or bilateral). This includes loss of the ability to smile, frown, blink, raise eyebrows, and talk normally. Long-term sequelae, seen in about 4-16%

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of cases, result in emotional and physical effects for the child. Such effects include negative impact on the child's self-esteem, and childhood and adolescent socialization.[2]

Viral infection or inflammation of the facial nerve as it enters the auditory meatus and/or parotid gland can affect the ability of the nerve to perform its normal functions.[1]

While multiple etiological factors have been reported to be associated with facial nerve palsy in children, there is very little known and understood about factors associated with facial nerve palsy in children in Africa, Nigeria inclusive. This is a report of 10 cases of facial nerve palsy managed in a teaching hospital (a tertiary health institution) in Nigeria over a period of 10 years. The report will describe the epidemiology, treatment and treatment outcomes of the 10 cases seen during this study period.

Methodology

Study design

This was a retrospective cohort review of pediatric cases of facial nerve palsy encountered in all the clinics run by specialists in the Obafemi Awolowo University Teaching Hospitals, Ile-Ife. These include patients seen and referred by the pediatric out-patient clinics, pediatric dentistry and oral medicine clinics, and Ear, Nose and Throat (ENT) clinics. Data were captured from these clinics as all pediatric cases would either be referred to these clinics form the emergency unit of the hospital, or referrals cases from external hospitals would be managed by either of these clinics. All case notes of pediatric patients (0-15 years) seen over a 10 years period (June 1st, 2002 to May 30th, 2012).

Study setting

The Obafemi Awolowo University Teaching Hospital sees on the average 72,415 new patients per annum with 4.0% being children. During the study period, 92,867 children were seen. All patients recruited for this study were diagnosed and managed by residents and trained specialists in the various units.

Study protocol

All case notes of those diagnosed with facial palsy were reviewed. A diagnosis of facial palsy was based on International Classification of Diseases, Ninth Revision, Clinical Modification codes were enrolled in this study: 767.5 (facial nerve injury with facial paralysis), 951.4 (injury to facial nerve), 998.2 (accidental iatrogenic injury), 351.0 (Bell palsy), 351.8 (other facial nerve disorders), and 351.9 (other facial nerve disorders, not otherwise specified). Patients were excluded if there were discrepancies in episode diagnosis and International Classification of Diseases, Ninth Revision, Clinical Modification code.[6] Information retrieved from the case note included sex, age, number of days with lesion prior to presentation in the clinic, diagnosis, treatment, treatment outcome, and referral clinic.

Results

Ten patients with ages ranging from 6 years to 15 years (mean age of 9.6 years ± 3.1 years) were diagnosed with facial palsy. For the study population, the prevalence of facial nerve palsy was therefore 0.01%. Nine of these patients were male.

All patients presented with unilateral weak partial closure of eye on the affected side, inability to move the eyebrow, wrinkle the nose, smile, whistle, and inability to puff the cheek. The lips deviated to the contralateral unaffected side on smiling. Masticatory function was intact. Eye movement in the horizontal and vertical planes was normal. Eight of the 10 patients lost the ability to wrinkle the forehead (cases 1 and 6 retained the ability to wrinkle the face). All other motor functions appeared to be within normal limits. There was no case associated with hyperacausis or altered taste. All other extraoral and intraoral examinations (for the cases seen in the oral medicine clinic) showed no other oral health problems. Table 1 shows the profile of the 10 cases seen. Seven of the 10 cases had unilateral paralysis on the right side of the face.

Laboratory investigations reveal that 3 of the 10 patients tested positive for malaria. For the test, venous blood was taken from patients by pricking a finger and blood collected in sequestrine-EDTA-anticoagulant coated tubes. Blood films were made and stained with 5% Giemsa and examined at × 100 of oil immersion using light microscopy. The density of parasites was then quantified.[7] Parasites in 1 μl of blood was estimated by counting the number of parasites present until 200 white blood cells (WBC) have been seen and then multiplying the parasites counted by 40. The standard value of WBC count of 8000 WBC per μl was used. In estimating the intensity of parasite infection, only parasites in thick-fields containing no more than 20 WBC per × 100 oil immersion fields were counted.[8]

Six cases (cases 1, 2, 3, 5, 6, and 7) were diagnosed with mumps based on clinical evaluation. The clinical symptom was swelling of the parotid glands starting from one parotid gland, followed 1-5 days later by enlargement of the contralateral gland. The patient also complains of pain and tenderness in the area of the gland. The swelling then starts to subside 5-7 days later [Table 1].

Two of the cases (cases 4 and 8) presented with a history of trauma: One patient was slapped on the face by one his peer in the school 1 month preceding the onset of the lesion. The patient was also febrile on presentation resulting in the need to evaluate for malaria infection. The outcome of the malaria investigation came out positive. Clinicians felt the history of trauma was unrelated to the clinical diagnosis due

Table 1: Age, sex, etiology, and clinical features of patients diagnosed with facial palsy

Cases	Age	Sex	Identified etiology	Duration at presentation (days)	Side of lesion	Clinical features: Ability to:								
						Eye closure	Wrinkling of forehead	Cheek	Wide smile	Whistling	Blowing	Hyperacausis	Altered taste	Deviation of lip to contralateral side
1	12	M	Mumps	1	Right	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Present
2	8	M	Mumps	14	Left	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
3	6	M	Mumps and malaria infection	9	Right	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
4	8	M	Trauma and malaria infection	6	Right	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
5	12	M	Mumps	1	Right	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Present
6	8	M	Mumps	14	Left	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
7	6	M	Mumps and malaria infection	9	Right	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
8	8	M	Malaria and trauma	6	Right	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
9	13	F	*CSOM and meningitis	1	Right	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present
10	15	M	Acute otitis media	4	Left	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present

*CSOM=Chronic suppurative otitis media, M=Male, F=Female

to the long history of trauma preceding the lesion. A positive history of mumps (acute or sub-acute lesion) was ruled out during the clerking [Table 1].

Two cases also presented with otitis media: One was suppurative (case 9) while one case presented with meningitis [Table 1].

Treatment

All the patients presented within 1-14 days of the lesion. Those who were seen within the first 7 days of presentation were treated with prednisolone and encouraged to use an eye pad at night. Eye pad was used to reduce the risk of corneal abrasions since the eye is unable to close completely or tear. All patients that were seen within 2 weeks of presentation were prescribed prednisolone along with some form of physical therapy. Physical therapy includes jaw exercise in the form of blowing balloons, and chewing sugar free chewing gums as often as possible each day prior to discharge. The two patients with otitis media had antibiotics added to their treatment regimen. Patients were reviewed every 1 week until lesion completely resolved. All patients were managed as out-patients. Table 2 gives a summary of the treatment and treatment outcome for all patients.

Prednisolone was dispensed as 20 mg in divided doses of 10 mg twice daily. For the patient that recovered completely at the 1st week of review, prednisolone was discontinued. For the patients that recovered after 1 week, prednisolone was tailed off during the week when there was evidence of significant recovery of facial muscle function with only telltale signs left. Prednisolone was tailed off as 5 mg 8 hourly for 2 days, then 5 mg 12 hourly for 2 days, and finally, 5 mg daily for 2 days. Patients were no longer on steroid at the time of discharge.

The three cases diagnosed with malaria were treated with Quantem^{R} in line with the National Regulations and Guidelines on the management of malaria in children in Nigeria.

Treatment outcome

The lesion had completely resolved in 8 of the 10 cases by the 3rd week of review: The shorter the time of presentation and initiation of therapy, the faster the resolution of the lesion. None of these patients had any observable residual lesion at the time of discharge the two patients with partial resolution were children diagnosed with otitis media. These patients continue to be monitored at the ENT clinic [Table 2]. The mean recovery time for the eight patients who fully recovered was 2.25 weeks ± 0.89 weeks.

Discussion

Facial nerve, also known as the seventh cranial nerve, exits the skull from the internal acoustic meatus. Motor components enter the stylomastoid foramen, penetrate through the parotid gland and supply the muscles of facial expression.[9] Thus, pathologies that affect the parotid gland has a huge tendency to cause compression of the nerve resulting in facial nerve palsy.

Table 2: Diagnosis, treatment, and treatment outcome of patients diagnosed with facial palsy

Cases	Diagnosis	Treatment	Number of review visits (number of weeks reviewed)	Treatment outcome	Clinic where managed
1	Upper motor neurone paralysis	Prednisolone+eye pad	One	Complete resolution	Oral medicine clinic
2	Lower motor neurone paralysis	Prednisolone+eye pad+physical therapy	Three	Complete resolution	Oral medicine clinic
3	Lower motor neurone paralysis	Prednisolone+eye pad+physical therapy+antimalaria	Three	Complete resolution	Oral medicine clinic
4	Lower motor neurone paralysis	Prednisolone+eye pad+antimalaria	Two	Complete resolution	Oral medicine clinic
5	Upper motor neurone paralysis	Prednisolone+eye pad	One	Complete resolution	ENT clinic
6	Lower motor neurone	Prednisolone+eye pad+physical therapy	Three	Complete resolution	ENT clinic
7	Lower motor neurone	Prednisolone+eye pad+physical therapy	Three	Complete resolution	ENT clinic
8	Lower motor neurone	Prednisolone+eye pad	Two	Complete resolution	ENT clinic
9	Lower motor neurone	Antibiotics+physiotherapy+eye pad+eye ointment at night	Seven	Incomplete resolution at last review	ENT clinic
10	Lower motor neurone	Antibiotics+physiotherapy+eye pad+eye ointment+predisolone	Eight	Incomplete resolution at last review	ENT clinic

ENT=Ear nose and throat

Fibers also join with the lingual nerve to form the chorda tympani while another portion of the nerve gives sympathetic fibers to the lacrimal gland and other mucous membranes.[9] Facial nerve palsy can therefore, result in hyperacousia and dryness of the cornea.

The facial nerve also innervates smaller accessory muscles. Portions of the facial nerve join with the trigeminal and supply taste to the anterior two-third of the tongue. Taste fibers also travel to the palate.[9] in view of this, facial nerve palsy could be associated with altered taste sensation.

The nerve is most likely to be damaged at the entrance to the narrowest portion of the facial canal.[10] Depending on the location of the inflammation or demyelination, lacrimal function,[1] taste, and hearing can also be disturbed.

Common etiological factors associated with facial palsy include, primary herpetic gingivostomatits, herpes zoster, varicella, rubella, acute otitis media, HIV, meningitis, Guillain-Barre syndrome, coxsackievirus, sarcoidosis, Melkersson-Rosenthal syndrome, tumors or other compressions, trauma and/or post-surgical complications, and diseases that cause nerve demyelination.[1,11] Idiopathic Bell's palsy is also a common cause of facial palsy. This may be due to genetic, vascular, metabolic and/or autoimmune reactions and unknown infectious agents.[11,12]

Within this study population, facial nerve palsy in children seems to be rare. This is in line with prior observations made by Wang *et al.*[13] A low annual incidence of 6.6 per 100,000 individuals had been earlier reported in children in the UK[14] and about 2.7 per 100,000 individuals under the age of 10 in India.[15] This report gives a much lower incidence though the incidence reported in UK and India. Patients with facial nerve palsy in the study environment may report in various other clinics or hospitals like the pediatric outpatient clinic, ENT clinic, and other private clinics in town. Efforts were made to track records on facial nerve palsy cases seen in children in these other clinics.

This lesion also occurred predominately in males, was mainly caused by mumps, and occurred on the right side of the face. Prior observation had also noted that infective causes of facial nerve palsy was more common in children[16] and the lesion occurred more in the right side of the face.[13] However, unlike past reports where otitis media had been the main etiological infective cause of facial nerve palsy in children, this study only observed 2 (20%) cases of facial nerve palsy associated with otitis media. Rather, a large number of cases with mumps as the suspected cause of lesion were observed. Prior to date, there have been few reports of facial nerve palsy associated with mumps.[15]

Wang *et al.*[13] noted that facial nerve palsy arising from otitis media was often recorded in toddlers age 2 years or less. Increased cases of otitis media in children age 2 years or less is believed to be due to an inferior immunological response.[17,18] Contrary to these reports, the two cases of facial nerve palsy associated with otitis media in this study were recorded in adolescents. While otitis media is not a rare diagnosis made in adolescents living in Nigeria,[19] the low incidence of facial nerve palsy associated with otitis media in this study population varies significantly from reports from developing countries. The reason for this observation cannot be readily adduced.

The possible role of malaria as a possible cause of facial nerve palsy has not been reported prior to now. The three cases (case 4 suspected to be wrongly diagnosed as mumps and the diagnosis of active malaria in the other two cases) make it important to explore the possible role of malaria infection in the etiology of facial nerve palsy (FNP). It is possible to find a causal relationship between malaria infection and middle ear disorders as an online survey showed that 0.56% of 2,835 persons who had malaria and participated in the survey reported inner ear disorders associated with their malaria infection.[20] All persons who experienced inner ear disorders in the study were males, the same observation made in this study: All those with facial nerve palsy with a history of malaria infection were all males. The online survey however, did not rule out if the problem was associated with malaria infection *per se* or drug used in the management of malaria. Also, the survey did not describe the specific inner ear problems. While the pathophysiology of malaria infection causing facial nerve palsy is not quite clear, this should not rule out the need for further studies to explore possible causal relationship between malaria infection and facial nerve palsy.

Steroid therapy in pediatric FNP is controversial and variable.[21-24] There are however no standard global protocol on how best to manage facial nerve palsy in children. The rapid recovery of this lesion in the case series reported here gives credence to the use of steroid in the management of facial nerve palsy in children. Furthermore, the rapid recovery of patients with facial nerve palsy resulting from mumps infection following the use of steroid is better than the report of Incecik *et al*.[15] who recorded a recovery period of 8-10 weeks in the patients with facial nerve palsy resulting from mumps they managed.

Limitation of the study

The present study was subject to the limitations of all retrospective reviews. Furthermore, the absence of serological tests makes the definite diagnosis debatable. For example, the case 4 could actually have been a case of sub-acute mumps infection without associated parotid gland enlargement. The associated fever makes this a possibility. The co-malaria infection is a possibility since the country lies in a malaria endemic belt.

Conclusion

Facial nerve palsy in children is a rare event in Nigeria. Majority of cases with facial nerve palsy have mumps infection as the causative factor. Recovery was rapid and complete for all cases of facial nerve palsy associated with mumps infection. Recovery was incomplete for the cases of facial nerve palsy associated with otitis media. The possible role of malaria infection in the etiology of facial nerve palsy should be further investigated as the feasibility of this cannot be completely ruled out based on this case report series.

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