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Moebius-Poland Syndrome: A Case Report from West Africa

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Abstract

Background: Moebius syndrome is a rare, nonprogressive, neurological congenital defect characterised by unilateral or bilateral congenital 7th and 6th cranial nerve paralysis and, less commonly, abnormalities of other cranial nerves (CN) III, IV, and IX–XII. It is frequently characterised by a lack of facial expressions, inability to make facial mimics, speech disorders, paralysis of the oculomotor nerve, eyelid ptosis, strabismus, swallowing and chewing disturbances, and tongue muscle atrophy. Poland syndrome presents with chest wall aplasia and ipsilateral upper extremity anomalies. This syndrome is characterised by hypoplasia of the forearm or breast, rib cage deformities, bilateral epicanthus and talipes equinovarus, and agenesis of the nipple. The management of Moebius-Poland syndrome is through a multidisciplinary supportive approach aimed at addressing the various symptoms associated with the condition. **Aim:** This report examines the clinical presentation of the case and includes a literature review on the incidence, diagnosis, associated features, and management of Moebius-Poland syndrome. **Case Report:** A male child of 16 years presented to us for the first time with a lack of facial expression, which was first noticed at 12 years of age. There were delayed developmental milestones, but the child is doing well academically. A lack of facial expression is notable, with the mouth open constantly and occasional drooling. Abnormalities in cranial nerves VI, VII, and XII were also noticed. **Conclusion:** Moebius and Poland syndrome can occur as separate syndromes or together. Poland features may be present in a patient with Moebius syndrome with very subtle signs, which can be missed without a thorough evaluation and knowledge of the possible co-existence of both syndromes. The overlap of these syndromes

highlights their complexity and the need for thorough evaluation and multidisciplinary management.

Keywords

Moebius Syndrome, Poland Syndrome, Abducens Nerve, Eye Movements, Pectoralis Major Muscle, Congenital Facial Palsy

1. Introduction

Moebius syndrome was named by Paul Julius Moebius in 1888, but was originally described by von Graefe and Saemisch in 1880, when they grouped certain patients with rare congenital disorders of the facial region that were nonprogressive. Alfred Poland first described Poland syndrome in 1841 when he documented a patient at autopsy with aplastic left pectoralis muscles and ipsilateral symbrachydactyly [1] [2]. Poland and Moebius syndromes are rare congenital disorders of musculoskeletal and nervous system anatomy, respectively, which may be associated with one another. While some researchers believe the combination to be independent of one another, others believe them to be variations of a similar cause [1] [3].

Poland syndrome presents with chest wall aplasia and ipsilateral upper extremity anomalies. This syndrome is associated with anomalies such as hypoplasia of the forearm or breast, rib cage deformities, bilateral epicanthus, talipes equinovarus, and agenesis of the nipple [3].

Moebius syndrome is a neurological disorder also known as congenital facial paralysis, congenital ocular-facial paralysis, or nuclear aplasia, whose cause is not yet demonstrated [4] [5]. It is classically described as combined congenital bilateral abducens and facial nerve palsies [6], and its clinical feature is peripheral facial paralysis [7]. Facial diplegia is the most noticeable symptom, including gaze palsy, dysphagia, and breathing difficulties, which are usually the presenting problems. This may be observed soon after birth, with incomplete eyelid closure during sleep, drooling, and difficulty sucking.

The prevalence of Moebius syndrome is estimated to be 0.0002% - 0.002% of live births [8], with equal incidence in both sexes [5]. While most cases are sporadic, familial cases, representing about 2% of all affected individuals, have also been reported [9]. Associated syndromes include Poland syndrome, Pierre Robin sequence, Carey-Fineman-Ziter, and Klippel-Feil anomaly. The estimated prevalence of Mobius-Poland syndrome is 1:500,000 and tends to affect more males than females (3:1), as well as the right side more than the left [2] [3].

2. Case Presentation

A male child of 16 years presented to us for the first time with facial weakness. As a baby, the lack of facial expression was noticed by the parents but was not diag-

nosed early due to a lack of access to specialist care in the town they resided in. The signs were subtle, and the medical facilities in their town lacked the necessary medical personnel to make a diagnosis of such a rare syndrome. A diagnosis of Moebius syndrome was made at 12 years of age when his uncle brought him to a medical screening mission in the capital, where he was assessed by a team of plastic surgeons. He was subsequently seen at the Plastic Surgery Unit of the Korle-Bu Teaching Hospital, Accra, Ghana, where a thorough evaluation was done together with the Radiology unit and a final diagnosis of Moebius-Poland syndrome was made.

At the teaching hospital, a thorough history and examination were taken, and investigations were carried out. He had delayed developmental milestones growing up. He did not suckle well and often drooled. He walked and talked at 2 years compared with his 2 elder siblings, who walked and talked at least 1.5 years. He is the third of three children. He is doing well academically and is sociable but lacks facial expressions. He was treated at an orthopaedic facility with prefabricated shoes for his right foot, likely clubfoot.

The child was born at the government hospital. The mother was an antenatal clinic attendant and took only routine drugs. The antenatal and pregnancy periods were uneventful. There was no history of alcohol or tobacco use, as well as misoprostol use during pregnancy. The child was delivered via spontaneous vaginal delivery.

On examination, a well-looking male child with no facial expression was observed. The mouth was constantly open and drooled occasionally. He is unable to shut his eyes entirely (**Figure 1(a)**), and his forehead does not wrinkle when looking up, as seen in **Figure 1(b)** (CN VII). He was unable to follow objects laterally (CN VI). There was no corneal erosion or strabismus. Protrusion of the tongue showed deviation to the left as seen in **Figure 1(c)** (CN XII). There was no lip retraction and no difficulty in breathing. The right chest is slightly underdeveloped compared to the left (**Figure 1(d)**), but has no missing toes or fingers. A provisional diagnosis of Moebius-Poland syndrome was made, with congenital facial palsy and isolated Moebius syndrome as possible differential diagnoses. Further investigations were done, which confirmed Moebius-Poland syndrome as the final diagnosis.

Limited grayscale ultrasonography of the pectoralis muscles and their insertional tendons was performed. The right and left pectoralis muscles were of comparable size, measuring 1.65 cm and 1.68 cm, respectively, in the sagittal plane. However, the tendons of the right and left pectoralis major on the humeral head displayed a discrepancy in their thickness, measuring an average of 1.1 mm and 3.5 mm, respectively (**Figure 2**) in the sagittal planes.

An unenhanced MRI of the brain was carried out, which revealed hypoplasia of both abducens nerves (CN VI) and absence of both facial nerves (CN VII) and the facial colliculi (**Figure 3** and **Figure 4**). The other cranial nerves were unremarkable. No structural abnormalities were noted in the cerebral parenchyma. Moderate mucosal thickening of the maxillary sinuses bilaterally and the

ethmoid sinuses, with a minimal air-fluid level in the right maxillary sinus, was noted.

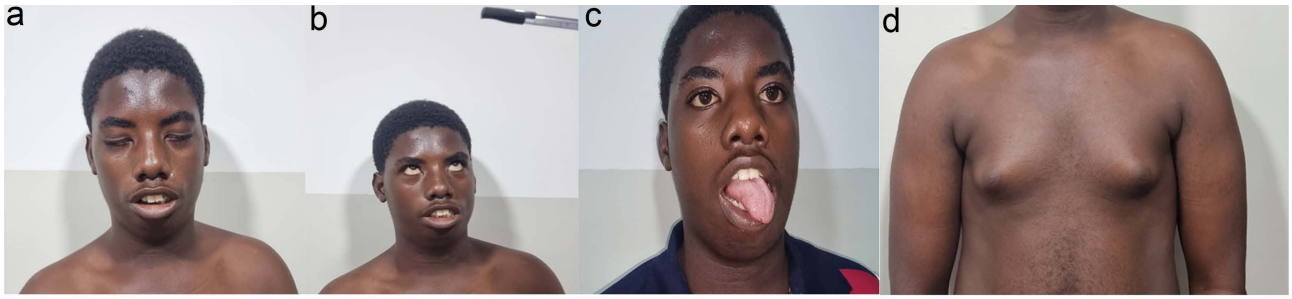

Figure 1. Patient noted to have (a) an inability to close eyes completely, (b) loss of forehead wrinkling, and (c) tongue deviation to the left, (d) Patient was noted to have a drooping of the right chest due to a hypoplastic right pectoralis major tendon.

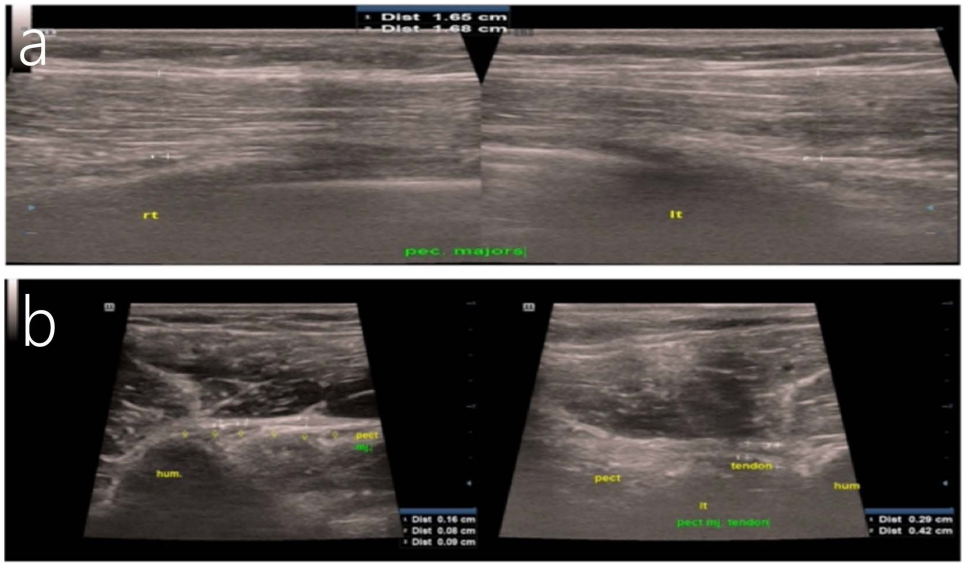

Figure 2. (a) Grayscale ultrasound of the pectoralis major muscles in the parasagittal plane demonstrating pectoralis major muscle thickness. (b) Measurements of the right and left pectoralis muscle tendons at their insertional points on the greater tubercle of the humerus.

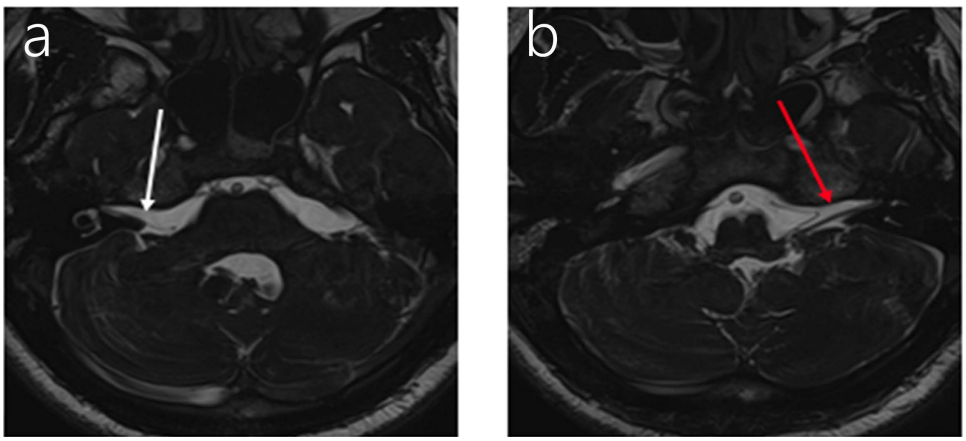

Figure 3. Axial MRI T2-weighted FIESTA images of the brain showing absence of the right (a) and left (b) facial nerve.

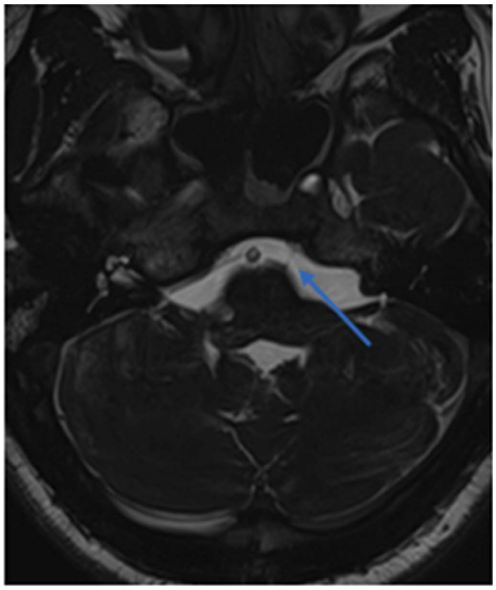

Figure 4. Axial MRI T2-weighted FIESTA showing hypoplasia of the left abducens nerve (CN VI).

3. Literature Review and Discussion

Moebius syndrome and Poland syndrome are rare congenital conditions affecting the muscles of the face and chest wall, respectively, and their association possibly results from a common aetiology [2].

Moebius syndrome is a rare, non-progressive, neurological congenital defect characterised by unilateral or bilateral congenital 7th and 6th cranial nerve paralysis and, less commonly, abnormalities of other cranial nerves (CN) III, IV, and IX-XII, as well as craniofacial, odontological, ophthalmological, and orthopaedic anomalies. It results in an absence of mimicry and strabismus.

Although the pathogenesis of Moebius syndrome remains unclear [10] and controversial, foetal toxic exposure, genetically determined vascular rhombencephalic disturbances in development, or an acquired ischemic event occurring after the fifth week of pregnancy have been named as determinants [9]. Statistical evidence supports the argument for a generalised midbrain-hindbrain and a complex patterning disorder of the brainstem as etiological factors [11]. The PLXND1 and REV3L genes [12], and the homeobox family of genes, particularly HOXA1, HOXB1, and SOX14 [5], have been implicated in the pathogenesis. Even though the majority of Moebius syndrome cases are sporadic, various familial trends, such as autosomal dominant, recessive, and X-linked recessive inheritance, have also been reported [5] [13].

The aetiology of Poland syndrome remains unclear, although a vascular injury of the subclavian artery has been hypothesised [14]. Vascular disruption theory remains the most accepted pathogenic mechanism. It may be due to the disruption of the development of the proximal subclavian artery and its branches that supply the pectoral muscles around the 6th week of gestation [15]. Bavinck & Weaver [16] introduced a pathogenic hypothesis called "the subclavian artery

supply disruption sequence" (SASDS) to describe the disruption of embryonic vessels, suggesting a common pathogenesis in PS, Klippel-Feil, and Moebius syndrome.

The characteristic clinical phenotype of Moebius Syndrome has been observed in various features. These include the paralysis of CN VII (96%), which can be entire, partial, unilateral, or bilateral, and of CN VI (85%), which is currently exhibiting a wide range of recently identified symptoms [9]. Its presentation is highly variable, with involvement of other cranial nerves and other cranial and extracranial dysmorphisms [11]. Our patient was noted to have cranial VI, VII, and XII abnormalities, characterised by inability to follow objects laterally with the eyes, loss of forehead wrinkling, and tongue deviation to the left (**Figure 1**).

From birth, Moebius syndrome is clinically marked, frequently by a variety of in-dicators: a) lack of facial expressions, the impossibility of making facial mimics, and various types of speech disorders; b) paralysis of the oculomotor nerve, eye-lid ptosis, strabismus; c) if it affects the glossopharyngeal nerve, it may be associ-ated with swallowing and chewing disturbances and tongue muscle atrophy [4]. The significant feature of our patient noted at birth and in infancy was poor suck and drooling. Other features may not have been noticed due to a lack of knowledge or minimal attention to identifying them. As he approached puberty, the expressionless face became apparent, hence identified at age 12 years. This corresponds to what Singham and colleagues (2004) noted, that with growth, a typical emotionless "mask-like facies"—unable to make a facial expression or smile—becomes apparent. These are responsible for major relational disorders [10] [17].

In addition to ocular motility and neurovisual function, ophthalmologic manifestations identified include delayed onset of lacrimation (between 4 - 6 months). Patients may later experience the crocodile tear phenomenon and dry eyes, and epiphora [5]. Other prevalent conditions include bilateral epicanthus and unilateral amblyopia, abnormal binocular vision, stereopsis, suppressive scotoma, incomplete or defective closure of the eyelids (lagophthalmos), photophobia [5] [9], and esotropia, the most common form of strabismus [6].

Infants with Moebius syndrome may have oral-maxillofacial malformations such as dental and palatal problems as well as micrognathia, microstomia, tongue malformation, high-arched palate, cleft lip or cleft palate, bifid uvula, and dental malocclusion[18] [19]. Furthermore, malocclusion, periodontal disease, inadequate alveolar bone growth, impaired chewing skills, slurred speech, and other issues can be caused by missing teeth [18].

Associated feeding abnormalities include poor neonatal sucking and swallowing, need for nasogastric tubes and gastrostomy, nutritional problems (Picciolini *et al*., 2016), and failure to thrive [11]. These oral disorders may be responsible for dysarthria [11] and defective speech [7] [9]. Failure of the typical mother-child attachment bond can also result from a lack of facial expressiveness and trouble making speech sounds that negatively impact the parent-child feedback interaction's reward [9].

Musculoskeletal abnormalities can also be associated with Moebius syndrome.

The most frequent symptom is Talipes [11], but there are other characteristics as well, such as ectrodactyly, brachydactyly, syndactyly, acheiria, arthrogryposis, kyphosis, scoliosis, kypholordosis, absent or deformed fingers or toes, and rib abnormalities [5] [6] [9]. Bell *et al*. (2019) reported the presence of hypotonia in younger children and abnormal motor coordination. Goldenhar anomaly (absent serratus anterior muscle), Klippel-Feil anomaly (fusion of the cervical vertebrae into a single short bone), and hypoglossia-hypodactyly may also be linked to Moebius syndrome [6].

Poland's anomaly is characterised by the congenital absence or hypoplasia of the pectoral head with ipsilateral hand deformity [11]. Other associated skeletal abnormalities may also be present, such as underdevelopment or absence of upper ribs, elevation of the shoulder blade (Sprengel deformity), and shortening of the arm with underdevelopment of the forearm bones (*i.e.*, ulna and radius) [20]. Our patient was noted to have hypoplasia of the right pectoralis major muscle.

Endocrine disorders such as Kallmann's syndrome (hypogonadotropic hypogonadism and anosmia) and premature thelarche have also been described in conjunction with MS [5] [6]. Dextrocardia, ventricular septal defects, and transposition of the great vessels have also been reported [5] [6]

In recent studies, diagnostic and imaging modalities are being employed in the diagnosis of Moebius syndrome [5]. Computed tomography (CT) and Magnetic Resonance Imaging (MRI) may depict brainstem hypoplasia with straightening of the floor of the fourth ventricle, indicating the absence of the facial colliculus; calcification in the pons in the region of the abducens nuclei; absence of the hypoglossal eminence at the medulla; and cerebellar hypoplasia, extraocular muscle hypoplasia, and other associated abnormalities [21]. The bilateral absence of the facial and abducens nerves can be considered a characteristic feature on MRI [21].

The management of Moebius and Poland syndromes is through a multidisciplinary supportive approach aimed at addressing the various symptoms associated with the condition [8]. The multidisciplinary team typically includes neurologists, ophthalmologists, plastic surgeons, otolaryngologists, and speech pathologists. Early involvement from clinical dietitians, consistent, careful monitoring of postnatal weight growth, and assessment for swallowing are essential. In extreme circumstances, feeding tubes may be necessary to provide additional nutritional support [8] [22]. Early audiology and speech therapy assessment and rehabilitation lead to optimal conditions for speech and language development later in life [8] [22]. It is recommended that patients undergo early ophthalmological evaluation to diagnose and treat gaze palsies, as well as to prevent corneal ulcers [8]. Our patient has been referred to see the speech therapist and ophthalmologist for further management of his speech and ocular deficiencies. He will need a long period of speech therapy to improve his speech.

Surgical treatment of Moebius patients often focuses on correction of limb and ocular deficiencies as well as restoration of a degree of volitional facial movement [10]. The restoration of facial movement in terms of verbal and nonverbal com-

munication can be appreciated [10]. To establish commissure retraction and increase cheek muscle tone, free transplantation of the masseter, temporalis, and platysma muscles has become more and more common [10].

The repair of chest wall defects in Poland syndrome aims to rectify cosmetic and functional capacity, depending on the anatomical type, gender, and patient preference [23]. Treatment options include autologous fat injection, pedicled latissimus dorsi muscle transfer, transverse rectus abdominis musculocutaneous flap, custom-made chest wall prosthesis, nipple-areola complex repositioning, mammary prosthesis, and sternal/rib reconstruction, or a combination of these techniques [20].

4. Conclusion

Moebius syndrome and Poland syndrome are rare congenital conditions affecting the muscles of the face and chest wall, respectively. These syndromes may rarely occur concurrently because of the subclavian artery supply disruption sequence hypothesis. This case highlights the importance of thorough evaluation for overlapping congenital syndromes, especially in resource-limited settings.

Informed Consent

Informed consent was obtained from the patient's guardian to publish this case report.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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