Natalia Gołuchowska[1], Aldona Ząber[1], Agata Będzichowska[1], Jakub Jagiełło[2], Karolina Agnieszka Iwanicka[3], Bolesław Kalicki[1,4], Agnieszka Rustecka[1]

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Melkersson–Rosenthal syndrome in a child – a rare diagnostic challenge. Case report

Zespół Melkerssona–Rosenthala u dziecka – rzadkie wyzwanie diagnostyczne. Opis przypadku

[1] Department of Paediatrics, Paediatric Nephrology and Allergology, Military Institute of Medicine – National Research Institute, Warsaw, Poland
[2] Military Institute of Medicine – National Research Institute, Warsaw, Poland
[3] Independent Public Complex of Health Care Facilities in Pruszków, Pruszków, Poland
[4] Faculty of Medicine, University of Warsaw, Warsaw, Poland
Correspondence: Natalia Gołuchowska, Department of Paediatrics, Paediatric Nephrology and Allergology, Military Institute of Medicine – National Research Institute, Szaserów 128, 04-141 Warsaw, Poland, e-mail: goluchowskan@gmail.com

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ORCID iDs
1. Natalia Gołuchowska https://orcid.org/0000-0002-1928-175X
2. Aldona Ząber https://orcid.org/0009-0004-3577-3203
3. Agata Będzichowska https://orcid.org/0000-0002-1756-7316
4. Jakub Krzysztof Jagiełło https://orcid.org/0009-0000-6660-8519
5. Karolina Agnieszka Iwanicka https://orcid.org/0000-0002-8803-332X
6. Bolesław Kalicki https://orcid.org/0000-0003-1606-5100
7. Agnieszka Rustecka https://orcid.org/0000-0003-3012-6942

Abstract

Facial nerve palsy has a heterogeneous aetiology. One of the rare causes of recurrent seventh cranial nerve palsy is Melkersson–Rosenthal syndrome, which is diagnosed primarily on the basis of its characteristic clinical presentation. This paper presents the case of a 17-year-old female patient hospitalised with right-sided peripheral facial nerve palsy. Physical examination, in addition to features of seventh cranial nerve palsy, revealed characteristic fissuring of the tongue. The medical history further indicated a previous episode of contralateral facial nerve palsy and recurrent self-limiting episodes of facial oedema. Extended laboratory and imaging diagnostics excluded infectious, neoplastic, autoimmune, and traumatic aetiologies. Based on the fulfilment of the diagnostic criteria, Melkersson–Rosenthal syndrome was diagnosed. Systemic glucocorticosteroid therapy, supportive treatment, and intensive rehabilitation were initiated, resulting in partial improvement in neurological status.

Keywords: facial nerve paralysis, Melkersson–Rosenthal syndrome, facial nerve paralysis in children, Melkersson–Rosenthal syndrome treatment

Streszczenie

Porażenie nerwu twarzowego ma zróżnicowaną etiologię. Jedną z rzadkich przyczyn nawracających porażeń nerwu VII jest zespół Melkerssona–Rosenthala, rozpoznawany głównie na podstawie charakterystycznego obrazu klinicznego. W niniejszej pracy przedstawiono przypadek 17-letniej pacjentki hospitalizowanej z powodu prawostronnego obwodowego porażenia nerwu twarzowego. W badaniu przedmiotowym poza cechami porażenia VII nerwu czaszkowego stwierdzono charakterystyczne bruzdowacenie na języku. Z wywiadu wynikało ponadto, że w przeszłości pacjentka przebyła epizod porażenia nerwu VII po stronie przeciwnej oraz doświadczała napadowych, samoistnie ustępujących obrzęków twarzy. Poszerzona diagnostyka laboratoryjna i obrazowa wykluczyła podłoże infekcyjne, nowotworowe, autoimmunologiczne oraz urazowe. W oparciu o spełnienie kryteriów rozpoznano zespół Melkerssona–Rosenthala. Włączono ogólnoustrojowo glikokortykosteroidoterapię, leczenie wspomagające oraz intensywną rehabilitację, uzyskując częściową poprawę stanu neurologicznego.

Słowa kluczowe: porażenie nerwu twarzowego, zespół Melkerssona–Rosenthala, porażenie nerwu twarzowego u dzieci, leczenie zespołu Melkerssona–Rosenthala

INTRODUCTION

Facial nerve palsy in children is relatively rare. In adults, its incidence is approximately 30 cases per 100,000 individuals per year, while in children under 10 years old it is about 2.7 cases per 100,000, and in children over 10 years old approximately 10.1 cases per 100,000 per year[1]. Nevertheless, it remains the most common cranial nerve neuropathy in both paediatric and adult populations[2].

The facial nerve (the seventh cranial nerve) contains motor, autonomic, and sensory fibres originating from the facial motor nucleus and the superior salivatory nucleus in the pons[3]. Its primary function is the control of facial expression, and thus emotional expression, by innervating muscles responsible, among others, for the position of the nostrils and eyebrows, as well as eyelid closure. It also plays a crucial role in maintaining proper lip position. Damage to the facial nerve results in speech disturbances, difficulties in maintaining oral hygiene, and drooling[1,3,4]. In addition, the facial nerve participates in sensory conduction from the auricle and the external auditory canal. It is also responsible for attenuation of excessively loud sounds, perception of taste from the anterior two-thirds of the tongue, and regulation of secretion from the salivary, lacrimal, and nasal glands[1].

Each case of facial nerve palsy requires thorough clinical evaluation and careful differential diagnosis. Possible causes include congenital, traumatic, idiopathic, and iatrogenic factors[5] (Tab. 1).

Congenital	Acquired		
Birth trauma	Idiopathic causes – Bell's palsy		
Möbius syndrome	Traumatic	Iatrogenic	After dental anaesthesia
Syringobulbia			After mandibular distraction osteogenesis surgery
		Trauma	
Goldenhar–Gorlin syndrome	Infectious	Varicella-zoster virus infection (Ramsay Hunt syndrome)	
		Epstein–Barr virus infection	
Hemifacial microsomia		*Borrelia burgdorferi* infection	
		Cytomegalovirus infection	
CHARGE syndrome		Mumps virus infection	
		Coxsackie virus infection	
Otic capsule cavitation		Herpes simplex virus (HSV) infection	
	Neoplastic	Parotid gland tumour	
Arnold–Chiari malformation		Schwannoma	
		Rhabdomyosarcoma	
	Systemic diseases	Guillain–Barré syndrome	
		Melkersson–Rosenthal syndrome	
		Kawasaki disease	
		IgA-associated vasculitis	
		Arterial hypertension	

Tab. 1. The most common causes of facial nerve palsy in children

Among the most common causes of facial nerve palsy in children is Bell's palsy, an idiopathic form of facial nerve palsy. It occurs less frequently in the paediatric population than in adults, with an estimated incidence of 6.1 cases per 100,000 children aged 1–15, compared with 20–30 cases per 100,000 in adults. Left- and right-sided involvement occurs with similar frequency, and the condition affects both sexes equally[1,6]. Similarly frequent in children is facial nerve palsy occurring during or following infections, including those caused by *Borrelia burgdorferi*, herpes simplex virus, Coxsackie virus, mumps, and varicella-zoster virus[1,7,8]. Moreover, seventh nerve palsy may represent a complication of mastoiditis secondary to acute otitis media; however, in the era of widespread antibiotic use, this complication has become increasingly rare[1,7,8]. After idiopathic and infectious aetiologies, trauma represents another important cause of facial nerve palsy in children[1]. Among less common causes, neoplastic lesions should also be considered. In paediatric patients, these most often include schwannomas, haemangiomas, rhabdomyosarcomas, and tumours of the parotid gland[1].

Primary tumours of the facial nerve are exceptionally rare. Facial nerve schwannomas, which are the most common primary tumours of this nerve, account for approximately 5% of all cases of facial nerve palsy[9]. Other potential causes of seventh nerve palsy include IgA-associated vasculitis, Kawasaki disease, stroke, and multiple sclerosis[1]. Iatrogenic cases should also be considered, for example following dental anaesthesia or mandibular distraction osteogenesis procedures[10,11]. Among the rare causes of facial nerve palsy, Melkersson–Rosenthal syndrome (MRS) should also be mentioned[12].

This article presents the case of a 17-year-old female patient with facial nerve palsy who was ultimately diagnosed with MRS.

CASE REPORT

A 17-year-old girl was admitted to the Department of Paediatrics, Paediatric Nephrology and Allergology, Military Institute of Medicine – National Research Institute due to symptoms of right-sided facial nerve palsy. From the day of admission, the patient reported headache localised to the temporal region, the area of the mandibular angle, and the right ear, as well as tingling of the tongue and excessive lacrimation of the right eye. In addition, she experienced reduced taste perception during food intake. On admission, the patient was in good general condition.

Physical examination revealed the following significant abnormalities: psoriatic lesions on the scalp, signs of right-sided peripheral facial nerve palsy (positive Bell's phenomenon with incomplete closure of the right palpebral fissure, facial asymmetry during grimacing, drooping of the right corner of the mouth, flattening of the nasolabial fold, and smoothing of the forehead on the right side), and deep longitudinal fissures of the tongue (Figs. 1–4).

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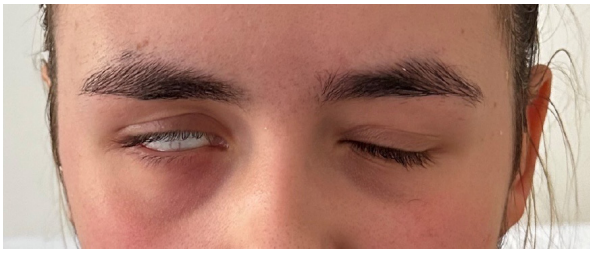

Fig. 1. Bell's sign in facial nerve paralysis – inability of the right palpebral fissure to close

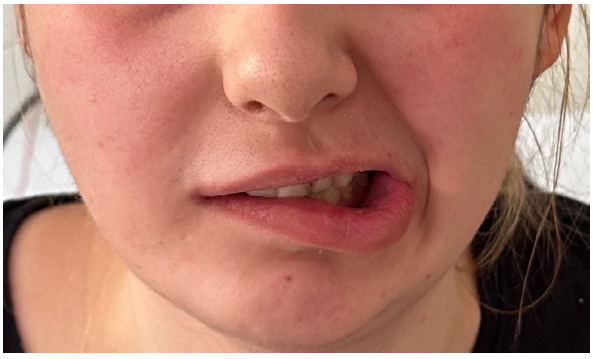

Fig. 2. Facial nerve palsy – noticeable facial asymmetry during attempted grimacing

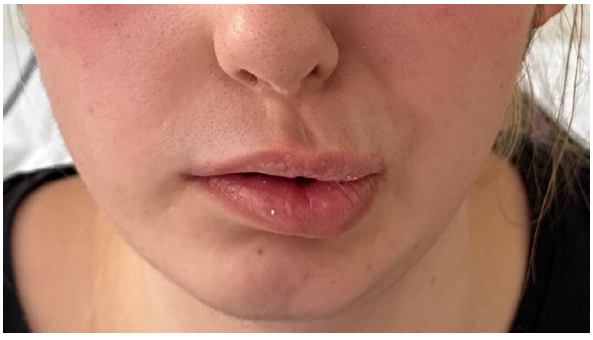

Fig. 3. Facial nerve palsy – noticeable facial asymmetry when attempting to whistle

Fig. 4. Fissuring of the tongue

Laboratory investigations showed no significant abnormalities in the complete blood count; inflammatory markers were not elevated (erythrocyte sedimentation rate 19 mm/h, reference value <20 mm/h; C-reactive protein 0.1 mg/dL, reference value <0.5 mg/dL), and electrolyte levels were within normal limits. Peripheral blood gas analysis revealed no significant acid-base disturbances. Recent infection with cytomegalovirus and *Borrelia burgdorferi* was excluded. Autoimmune diseases were also excluded (no antibodies against Ro and La antigens, antinuclear antibodies, antineutrophil cytoplasmic antibodies, anticardiolipin antibodies, autoantibodies against double-stranded deoxyribonucleic acid, antibodies against cyclic citrullinated peptide, autoantibodies against myeloperoxidase, or rheumatoid factor were detected). Polymerase chain reaction testing of a nasopharyngeal swab did not detect any of the tested pathogens.

During hospitalisation, ophthalmological, neurological, and otolaryngological consultations were performed. No focal neurological deficits or signs suggestive of central nervous system involvement were identified, and idiopathic facial nerve palsy was confirmed. Hearing assessment using pure-tone and impedance audiometry was conducted; pure-tone audiometry revealed mild conductive hearing loss at 500 Hz in the right ear. Computed tomography of the head showed no abnormalities.

Magnetic resonance imaging of the head revealed a small vascular malformation in the left frontal lobe, as well as slight contrast enhancement of the right facial nerve at the fundus of the internal auditory canal. Abdominal ultrasonography and chest radiography showed no abnormalities. The medical history revealed that several years prior to admission, during COVID-19 infection, the patient had developed left-sided peripheral facial nerve palsy. At that time, head magnetic resonance imaging and computed tomography showed no abnormalities. The patient underwent intensive rehabilitation, with incomplete resolution of symptoms. Following more detailed history-taking, the patient reported recurrent episodes of facial oedema that resolved spontaneously.

Based on the overall clinical picture, recurrent idiopathic facial nerve palsy consistent with MRS was diagnosed. Treatment included systemic glucocorticosteroids (50 mg of prednisone per day, followed by gradual dose reduction), artificial tear preparations, galantamine (initially 2.5 mg, with dose increases of 2.5 mg every three days to a maximum daily dose of 20 mg, administered for approximately one month), B vitamins, and rehabilitation (massage, galvanisation, laser therapy, infrared radiation), resulting in partial improvement.

The patient was discharged home with recommendations to continue outpatient rehabilitation.

DISCUSSION

MRS is a rare disease entity with an incompletely understood aetiology. In the pathogenesis of the syndrome,

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Main symptoms	Neurological symptoms	Gastrointestinal symptoms	Ocular symptoms	Psychiatric symptoms
Recurrent, spontaneously resolving swelling of the face and lips	Headache and dizziness	Dysphagia	Visual disturbances	Depression
	Migraine attacks	Recurrent aphthous ulcers	Excessive tearing	
Recurrent facial nerve palsy	Facial paraesthesia	Diverticulitis	Conjunctival hyperaemia	Personality disorders
	Tinnitus			
Grooves/fissures of the tongue	Impaired taste sensation	Ulcerative colitis	Uveitis	Anxiety episodes
	Decreased/increased facial skin sweating			

Tab. 2. Symptoms occurring in MRS (main diagnostic symptoms and additional neurological, gastrointestinal, ocular, and psychiatric manifestations)

the contribution of infectious factors has been considered, including infections caused by *Borrelia burgdorferi*, *Toxoplasma gondii*, *Treponema pallidum*, *Mycobacterium* spp., and HSV[13]. The literature has also described the coexistence of MRS with autoimmune and inflammatory diseases such as thyroiditis, multiple sclerosis, granulomatosis with polyangiitis, sarcoidosis, keratitis, diabetes mellitus, allergic diseases, and ulcerative colitis[13]. Attention has also been drawn to a possible association between MRS and hereditary granulomatous diseases, Down syndrome, and psoriasis[13–15]. Reports of familial occurrence of the syndrome suggest a significant role of genetic factors in its development[15].

The first symptoms of MRS most commonly appear between 25 and 40 years of age, with a predominance in women (female-to-male ratio of approximately 2:1)[13]. The diagnosis of MRS is based on the clinical presentation. Establishing the diagnosis requires the presence of at least two of the three features of the classic triad: recurrent facial and/or labial oedema, facial nerve palsy, and fissured tongue (*lingua plicata*). *Lingua plicata* is defined as the presence of grooves on the dorsal surface of the tongue measuring at least 15 mm in length and ≥2 mm in depth[13,15]. In 40–70% of patients, the first manifestation of the disease consists of recurrent, often alternating facial nerve (VII) palsy, which may precede the onset of orofacial oedema by months or even years[12].

In addition to the cardinal symptoms, patients with MRS may experience numerous nonspecific complaints, including headaches and dizziness, migraine attacks, facial paraesthesias, dysphagia, tinnitus, taste disturbances, recurrent aphthous ulcers, conjunctival hyperaemia, excessive lacrimation, visual disturbances, and disorders of facial sweating. Involvement of other cranial nerves has also been reported, including the olfactory, auditory, trigeminal, glossopharyngeal, and hypoglossal nerves[13]. The course of the syndrome may also include extra-neurological manifestations, such as diverticulitis, ulcerative colitis, uveitis, as well as psychiatric disorders, including anxiety episodes, depression, and personality disorders[13] (Tab. 2).

It should be emphasised that only a proportion of patients (20–75%) present with the complete triad of symptoms, which makes the diagnosis of MRS challenging and often delayed[13]. Due to the lack of specific biomarkers, MRS remains a diagnosis of exclusion and requires extensive differential diagnostics[14]. The diagnostic workup includes laboratory investigations such as complete blood count, glucose levels, assessment of renal, hepatic, and thyroid function, as well as testing for autoimmune diseases, including antinuclear antibodies and antineutrophil cytoplasmic antibodies.

In the differential diagnosis of facial nerve palsy, serological testing and cerebrospinal fluid analysis are important for excluding infections of the central nervous system, including cytomegalovirus, Epstein–Barr virus, HSV, varicella-zoster virus, *Borrelia burgdorferi*, and *Treponema pallidum*[14]. Imaging studies – computed tomography and magnetic resonance imaging of the head – also play a crucial role by allowing exclusion of trauma, iatrogenic complications, and neoplastic lesions[13,14]. The diagnostic evaluation should be complemented by an otolaryngological examination to exclude acute or chronic otitis media and mastoiditis[14]. Additional supportive investigations include brainstem auditory evoked potentials, facial nerve conduction studies, visual evoked potentials, and ophthalmological examination with slit-lamp evaluation for uveitis[13].

In cases of orofacial oedema, differential diagnosis should include allergic and hereditary angioedema, foreign body reaction, rosacea, Graves' disease, tuberculosis, and neoplastic conditions (including granulomatosis with polyangiitis and orbital tumours)[13,14]. In children, MRS additionally requires differentiation from congenital syndromes associated with facial nerve palsy, such as Arnold–Chiari malformation, Möbius syndrome, and Goldenhar syndrome, as well as consideration of perinatal trauma in the medical history[14].

Histopathological examination may constitute an important element of the diagnostic process. Biopsy of the mucosa obtained during an acute episode typically reveals perivascular mononuclear infiltrates or lymphoepithelioid granulomas[13,15]. In the present case, the patient already fulfilled the clinical criteria for MRS at the time of admission, presenting with facial nerve palsy and a fissured tongue. A history of recurrent, spontaneously resolving facial oedema further supported the diagnosis; therefore, given the typical clinical presentation and the results of differential diagnostics, biopsy was not performed.

To date, no definitive treatment guidelines for MRS have been established[13–15]. The cornerstone of therapy remains glucocorticosteroids administered systemically or intramuscularly. Prednisolone is most commonly used at an

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initial dose of 1–1.5 mg/kg body weight per day, followed by gradual dose tapering over several weeks, leading to clinical improvement in 50–80% of patients and a reduction in relapse frequency by 60–75%[14]. In the presented case, systemic glucocorticosteroids, galantamine, and artificial tear preparations were administered. For the treatment of orofacial oedema, first-line therapy consists of intramuscular administration of triamcinolone acetonide (1–1.5 mL of a 10–20 mg/mL solution) with the addition of lidocaine[13]. As no oedema occurred during hospitalisation, this approach was not implemented.

An essential component of management in patients with MRS and facial nerve palsy is intensive rehabilitation. In cases refractory to treatment or characterised by frequent relapses, immunosuppressive therapy (methotrexate or thalidomide) or surgical decompression of the facial nerve may be considered[13].

Due to improvement in the patient's general condition and partial resolution of symptoms, she was discharged home with recommendations to continue outpatient rehabilitation.

CONCLUSIONS

MRS is a rare but clinically significant cause of recurrent facial nerve palsy in children and adolescents, and should always be considered in the differential diagnosis of peripheral seventh cranial nerve neuropathy. The case of the 17-year-old patient highlights the crucial importance of a carefully obtained medical history and a thorough assessment of the clinical presentation. The presence of the characteristic symptom triad may allow diagnosis of MRS without the need for invasive procedures such as biopsy. The presented case also demonstrates that early diagnosis and timely initiation of treatment – primarily based on glucocorticosteroids and rehabilitation – may contribute to symptom alleviation and reduction of the risk of long-term neurological sequelae. Patients diagnosed with MRS should receive comprehensive multidisciplinary care (otolaryngological, ophthalmological, neurological, and rehabilitative) to ensure the best possible quality of life and minimise the risk of recurrence.

Conflict of interest

The authors declare no conflicts of interest.

Authors' contributions

Original concept of study: NG, AB. Collection, recording and/or compilation of data: NG. Analysis and interpretation of data; writing of manuscript: NG, AZ, JJ, KAI. Critical review of manuscript: NG, AB, BK, AR. Final approval of manuscript: BK, AR.

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