Melkersson-Rosenthal syndrome

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Introduction

This article includes discussion of Melkersson-Rosenthal syndrome, Melkersson syndrome, Rossolimo-Melkersson-Rosenthal syndrome, Melkersson-Rosenthal-Scheuermann syndrome. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

Recurrent facial palsy with orofacial swelling and fissuring of the tongue is known as the Melkersson-Rosenthal syndrome. This chapter provides a review of the clinical features, hypothesized pathophysiology, and current management of this syndrome.

Key points

- Melkersson-Rosenthal syndrome should be suspected in cases of recurrent facial palsy associated with swelling of orofacial structures and/or a furrowed tongue.
- Biopsy of suggestive lesions typically reveals noncaseating granulomas with perivascular and lymphatic inflammatory cell infiltration.
- The pathophysiology involves an immunologically mediated inflammatory response.
- Treatment consists of various immune-suppressing and modulating agents, along with surgical correction of disfiguring lesions as necessary.

Historical note and terminology

In 1928, Ernst Melkersson (1883-1930), a Swedish physician, described a 35-year-old woman with facial edema and paralysis. Shortly after, in 1931, Curt Rosenthal (1892-1937), a German neurologist, described 3 patients who had fissured or plicated tongues in addition to orofacial swelling and facial palsies, and he also proposed a link between the triad of symptoms. Subsequently, the triad of clinical findings came to be known as the Melkersson-Rosenthal syndrome (Rosenthal 1931).

When the characteristic facial swelling is confined to the lips and occurs in a monosymptomatic or oligosymptomatic form (ie, in isolation or in conjunction with either a furrowed tongue or facial palsy), it is referred to as Miescher cheilitis or cheilitis granulomatosa (Greene and Rogers 1989; Sciluba and Said-Al-Naief 2003). If granulomatous swelling is localized mainly to the eyelids, the disorder is referred to as granulomatous blepharitis and considered a monosymptomatic form of the Melkersson-Rosenthal syndrome (Yeatts and White 1997).

Clinical manifestations

Presentation and course

Melkersson-Rosenthal syndrome is characterized by the triad of recurrent facial palsy, orofacial swelling, and a furrowed tongue. The full triad of symptoms is uncommon and present in only 8% to 25% of patients, making the correct diagnosis difficult at times (Hornstein 1987; Greene and Rogers 1989; Elias et al 2013; Bohra et al 2015; Lin et al 2016). If only 1 or 2 components of the triad are present, the disorder is considered, respectively, a monosymptomatic or oligosymptomatic form of the Melkersson-Rosenthal syndrome. In Hornstein's series of 73 patients and in Greene and Rogers' series of 36 patients, 18% and 28%, respectively, were recognized as having monosymptomatic lip swelling (ie, cheilitis granulomatosa) (Hornstein 1987; Greene and Rogers 1989). In El-Hakim and Camacho-Alonso's combined series of 11 patients, 8 patients manifested only cheilitis granulomatosa (Camacho-Alonso et al 2004; El-Hakim and Chauvin 2004). And in Greene and Rogers' series of 36 patients, half of them had oligosymptomatic Melkersson-Rosenthal syndrome.

Orofacial swelling is the most consistent and dominant feature of the Melkersson-Rosenthal syndrome (Lin et al 2016). It is a localized, painless, nonpruritic, and nonpitting form of lymphedema (Chen et al 2015). In a review of 200
patients diagnosed with Melkersson-Rosenthal syndrome, orofacial swelling was the most frequent initial presenting sign (Zimmer et al 1992). The lips are most commonly affected, with the swelling sometimes confined only to the lips. The upper lips are involved more frequently than the bottom lips. The swelling can be asymmetric or unilateral and is usually nontender, but it can be associated with pain or erythema; it may also involve various intraoral structures such as the gingiva, palate, tongue, pharynx, and larynx (Greene and Rogers 1989; Zimmer et al 1992). During the first few episodes, the facial swelling is usually short-lived, but with repeated episodes, the swelling may fail to clear, with residual swelling becoming firm and indurated with granulomatous inflammation (Rogers 1996). Less commonly, the swelling can involve the eyelid and orbital tissues, presenting as a granulomatous blepharitis (Mignogna et al 2003; Shapiro et al 2003; Cocuroccia et al 2005; Kondratiev et al 2010; Rawlings et al 2012). In some cases, the orofacial swelling can become prominent, causing significant cosmetic disfigurement.

The facial palsy is manifested as a peripheral VIIth nerve dysfunction, clinically identical to that of Bell palsy. It was one of the initial symptoms in 31% of patients diagnosed with the Melkersson-Rosenthal syndrome in a review of 117 patients reported in the literature between 1965 and 1990 (Zimmer et al 1992). In another series of 36 patients with the Melkersson-Rosenthal syndrome, a comparable 39% reported developing a facial paresis as the initial presenting symptom (Greene and Rogers 1989). The facial palsy can vary in severity from mild to complete and is typically recurrent and unilateral but can be bilateral or alternating. Some two thirds who initially present with unilateral facial paresis subsequently develop alternating facial paresis (metachronous contralateral paresis), whereas only about 5% have simultaneous facial diparesis (Rivera-Serrano et al 2014). Episodes of facial palsy usually come on acutely and typically last from days to months. Initial episodes are normally short-lived, but with repeated episodes the duration of facial paresis typically becomes prolonged or permanent. The facial paralysis usually occurs concomitantly on the side of facial swelling, but may occur months to years before or after the onset of orofacial swelling (Zimmer et al 1992; Rogers 1996; Shapiro et al 2003).

In addition to its hallmark facial nerve involvement, there have been a few case reports of Melkersson-Rosenthal syndrome presenting with dysfunction of other cranial nerves, such as the vagus and glossopharyngeal nerves (Khandpur et al 2006), the auditory nerve (Stein et al 2014), or the occulomotor nerve (Aluclu et al 2008; Parr et al 2012). Such presentations are rare.

The fissured tongue is present in 30% to 77% of patients with Melkersson-Rosenthal syndrome (Wadlington 1984). In a review of 42 patients, it was present in about 50% (Zimmer et al 1992), and it was present in 47% in another series of 72 patients (Elias et al 2013). Characteristically, there may be 1 or many longitudinal furrows with smaller fissures that radiate out laterally, giving rise to the appearance of a “plicated” or “scrotal tongue.” The fissuring on the tongue is usually congenital and completely asymptomatic in the majority of patients. Rarely, the deep furrows have been associated with local bacterial and fungal colonization and overgrowth but are otherwise an asymptomatic finding. Many authors have reported the presence of plicated tongues among relatives (Greene and Rogers 1989; Rogers 1996; Altoğlu et al 2000), suggesting a possible genetic etiology. However, in the vast majority of patients there is no family history of the disorder, and its presence in more than 1 family member may merely reflect a fairly common baseline prevalence of approximately 5% in the general population (Halperin et al 1953).

Prognosis and complications

The prognosis in Melkersson-Rosenthal syndrome is variable. The majority of patients experience a chronic relapsing-remitting course of orofacial swelling and facial paralysis often separated by months to years, but some may experience persistent, progressive, or permanent symptoms with each successive episode (Rogers 1996). The interventional use of various steroids and antibiotics is anecdotally reported to improve outcome with longer periods of symptomatic remission (Camacho et al 2001).

Compared with recurrent Bell palsy, the prognosis for recovery with recurrent facial paresis in Melkersson-Rosenthal syndrome is much worse; in patients with recurrent facial palsy, only 14% of patients with Melkersson-Rosenthal syndrome recover to House-Brackmann grade 1 by 1 year when compared to 71% of patients with recurrent Bell palsy (Wang et al 2015).

Complications from Melkersson-Rosenthal syndrome-associated facial palsy parallel those from typical Bell palsy. In the acute phase of facial nerve paralysis, inability to properly close and lubricate the affected eyelid can give rise to corneal and scleral injury. In addition, reinnervation of the facial nerve fibers may be incomplete or aberrant, leading to variable outcomes of sensory, motor, or parasympathetic nerve-fiber deficits.
Clinical vignette

A 38-year-old woman with other medical issues had a history of recurrent alternating facial nerve paresis, with episodes at ages 5, 18, 30, and 32 (Jasinska and Boczon 2015). Following bilateral facial palsy, she was unable to smile and had a relatively smooth left nasolabial furrow. She also had lingua plicata (fissured tongue) as did her 14-year-old daughter. MRI of the head, CSF studies, and basic laboratory tests were normal.

Biological basis

Etiology and pathogenesis

The etiology is unknown. There have been many proposed theories, including possible genetic, allergic, autoimmune, and infectious etiologies. However, none have unequivocally been shown to be causative (Greene and Rogers 1989; Rogers 1996; van der Waal et al 2001; D’Amore et al 2010).

Some controversy exists with regard to the uncanny similarities present in the clinical and histopathologic manifestations of the oral pathology between Melkersson-Rosenthal syndrome and Crohn disease. Some consider macrocheilitis, a granulomatous affliction of the mouth and an oligosymptomatic manifestation of the Melkersson-Rosenthal syndrome, to represent a regional manifestation of Crohn disease (Lloyd et al 1994; Sciubba and Said-A-Naief 2003). This is substantiated by reports of a few patients who were initially diagnosed with granulomatous cheilitis but were subsequently also diagnosed with Crohn disease (Sciubba and Said-Al-Naief 2003; Tonkovic-Capin et al 2006). Interestingly, in a series of 72 patients evaluated at the Mayo Clinic in Rochester, Minnesota, over a period of 30 years, about 8% of patients were also diagnosed as having Crohn disease as a comorbidity (Elias et al 2013). However, as the majority of patients who develop the Melkersson-Rosenthal syndrome do not have manifestations of Crohn disease, this is a point of continued controversy.

An immune-mediated inflammatory reaction appears to be central to the development of the orofacial symptoms. This is supported by the fact that many of the successful treatments for this disorder are immune-suppressing agents (see Management). There have also been reports of various autoimmune-mediated diseases, such as multiple sclerosis (Cabrera-Gomez et al 2005), Hashimoto thyroiditis (Scagliusi et al 2008; D’Amore et al 2010; Lee et al 2014), Crohn disease (Santos et al 2001), and mixed connective tissue disease (Jasinska and Boczon 2015), occurring in association with Melkersson Rosenthal syndrome.

Early in the course of inflammation, there is mild epithelial hyperplasia characterized by dilated lymphatics, with perivascular congregations of histiocytes, lymphocytes, and plasma cells in a background of nonspecific edema. As the lesions become established over weeks of persistent swelling, biopsy often reveals areas of noncaseating granulomas with perivascular lymphocytic infiltration in the lamina propria or dermal layers (Greene and Rogers 1989; Glickman et al 1992; Zimmer et al 1992; Rogers 1996; Kondratiev et al 2010; Rawlings et al 2012). Noncaseating intralymphatic granulomas have also been visualized and are postulated to cause lymphatic obstruction and associated facial tissue edema (Gonzalez-Garcia et al 2011). Similar pathologic findings of lymphatic flow obstruction were demonstrated with abnormal lymphoscintigraphy studies in 3 of 4 patients diagnosed with Melkersson-Rosenthal syndrome (Nittner-Marszalska et al 2010).

Postulated mechanisms for the disorder include reactive immune-mediated responses directed toward some nonspecific offending stimulus that result in autonomic vasomotor disturbances of the vasa nervorum and small penetrating arterioles of the facial nerve and subcutaneous tissue. Those processes are subsequently postulated to allow for greater vascular permeability, antigenic penetration, and tissue reactivity, followed by the characteristic granulomatous changes (Streeto 1964; Hornstein 1973).

Serum IgG levels were elevated in 5 patients with Melkersson-Rosenthal syndrome (Yetiser et al 2002). Two of the 5 patients also demonstrated elevated CSF IgG and albumin levels, but none of them demonstrated increased IgG indexes or oligoclonal bands in the CSF. Yetiser and colleagues argued that these findings support the idea that there is a sustained immunologic response from an as yet unidentified stimulus in patients with recurrent facial palsies and Melkersson-Rosenthal syndrome (Yetiser et al 2002).

Kaminagakura and Jorge reported more T and B cells within and surrounding granulomatous lesions in a patient
manifesting the complete triad of symptoms, compared to primarily T cells with very few B cells in the biopsy of a patient with the monosymptomatic form (Kaminagakura and Jorge 2011). They suspect different and greater humorally mediated host responses for the more complete form of disease presentation.

A 52-year-old woman with Melkersson-Rosenthal syndrome in conjunction with isolated IgE hypogammaglobulinemia has been reported (Nakane et al 2007). From this case, the authors concluded that an IgE-mediated hypersensitivity reaction is unlikely to account for the development of the Melkersson-Rosenthal syndrome.

Presumably the underlying mechanism for the facial palsy in Melkersson-Rosenthal syndrome is related to increased tissue swelling in the facial nerve sheath; thus, decompression of the facial nerve in its intratemporal course may prevent recurrent episodes of nerve compression within a tight and unyielding bony canal. During decompression of the facial nerve intraoperatively in a patient with the Melkersson-Rosenthal syndrome, Dutt and colleagues described a pathological “large oedematosus facial nerve sheath,” providing some support for this purported pathophysiology of facial palsy (Dutt et al 2000).

**Epidemiology**

The disorder is rare and the true incidence is not clearly known. The symptoms of Melkersson-Rosenthal syndrome usually occur during the second decade, but have been reported to affect patients across a wide range of ages from 2 to 81 years (Greene and Rogers 1989; Zimmer et al 1992; Shapiro et al 2003). In a series of 36 patients reviewed by Greene and Rogers, the mean age of initial symptom onset was 33 years. Another systematic review, pooling data from published cases, suggested that females are more often affected than males (56% vs. 44%) (Zimmer et al 1992).

There are several reports of patients with Melkersson-Rosenthal syndrome who have family members with components of the triad of symptoms, but the complete triad is rarely reported. In 1 review of 27 patients, 3 patients had first-degree relatives who had plicated tongues, and only 1 patient reported all 3 symptoms a family member (i.e., a sister), but this was not substantiated by the authors (Camacho et al 2001). Another series of 42 patients studied at the Mayo Clinic revealed no family history for the complete triad of symptoms, but 2 patients had 3 family members with plicated tongues, 2 patients had 3 family members who had Bell palsy, and another 2 patients had 4 family members with orofacial swelling (Zimmer et al 1992). In another series of 15 patients, a family history was positive in 5 of 16 patients (31%) with facial paresis in Melkersson-Rosenthal syndrome (Sun et al 2015). The significance of such findings remains unclear but may possibly suggest a familial predisposition.

**Prevention**

There is no known prevention.

**Differential diagnosis**

The differential diagnosis of Melkersson-Rosenthal syndrome includes a variety of disorders that can mimic the histopathologic and clinical findings (Zimmer et al 1992; Rogers 1996; Camacho et al 2001). Entities that can cause orofacial swelling and granulomatosis include facial trauma, insect bites, cellulitis, lymphatic obstruction, eyelid lymphedema, eosinophilic facial swelling, periodontal and skull-based infections, silica-induced granulomatous cheilitis, contact dermatitis, acute leukemia, leprosy, syphilis, tuberculosis, sarcoidosis, and Crohn disease. The differential diagnosis for disorders that can produce peripheral facial nerve palsies include Bell palsy, skull trauma, Ramsey-Hunt syndrome, herpes simplex virus-1, HIV, Lyme disease, sarcoidosis, tuberculosis, syphilis, vasculitis, acute demyelinating neuropathies, and neoplastic lesions.

**Diagnostic workup**

As there are no pathognomonic laboratory findings for the Melkersson-Rosenthal syndrome, diagnosis depends on clinicopathologic correlation (Worsaae et al 1982). Thus, considering all the entities in the differential diagnosis by clinical findings, laboratory tests, imaging studies, and histology is essential to correctly establish the diagnosis. Sometimes the correct diagnosis may only be reached based on the exclusion of other causes of orofacial granulomatosis (Rogers 1996).

Biopsy of the swollen orofacial tissue should be sought to document the presence of noncaseating epithelioid-cell granulomas (Bohra et al 2015). In rare cases, swelling and noncaseating epithelioid-cell granulomas may involve the
Infliximab (Chu et al 2016). Often, the biopsy may only reveal nonspecific inflammatory changes or fibrosis (Greene and Rogers 1989; Zimmer et al 1992; Rogers 1996). The typical granulomas may be absent on biopsy, but this should not exclude the diagnosis of Melkersson-Rosenthal syndrome in the presence of typical symptoms, as individual granulomas reportedly form and resolve within days to weeks and do not necessarily parallel the clinical course of swelling (Hornstein 1973). Routine examination of tissue for mycobacterial infection (with Ziehl-Neelsen staining) and foreign bodies should also be performed because these entities may produce characteristic granulomatous changes.

Other laboratory studies may be useful depending on the index of clinical suspicion and include serum angiotensin-converting enzyme levels, gallium scan, chest x-ray, and purified protein derivative placement to rule out sarcoidosis and tuberculosis. HIV, Epstein-Barr virus, cytomegalovirus, and herpes simplex virus-1 viral titers, serum Lyme titers, and venereal disease research laboratory test (VDRL) may be useful to exclude infections. CSF cell count, protein, and cytology examination to check for the presence of acute inflammatory demyelinating polyradiculoneuropathy, multiple sclerosis, meningeal carcinomatosis, and various meningitides should also be considered in the diagnostic workup.

Panorex periodontal films and skull CT may be indicated to evaluate for local infection and abscesses. For recurrent facial paresis, especially when associated with a progressive course and accompanied by other cranial nerve dysfunction, an MRI of the brain with gadolinium contrast may be useful to exclude mass or infiltrating lesions involving the facial nerve.

If oral lesions associated with cobblestoning or skin tags accompanied by characteristic gastrointestinal complaints are present, a referral to the gastrointestinal specialist for endoscopy may be indicated to exclude Crohn disease.

Management

The current treatment for Melkersson-Rosenthal syndrome is primarily aimed at reducing the orofacial swelling and inflammation of the facial nerve and preventing recurrences. Effective treatments currently include systemic or intralesional corticosteroids, antibiotics, nonsteroidal anti-inflammatories, cosmetic surgery, and more recently, monoclonal antibodies.

Corticosteroids are an effective therapy (Zimmer et al 1992; Liu and Yu 2013), although not all cases respond (Saini et al 2016). Topical steroids have been used for mild cases of orofacial swelling with fair results (van der Waal et al 2002). Intralesional corticosteroid (triamcinolone) injections are commonly used and may decrease facial swelling in more severe cases, often for prolonged periods of remission (Glickman et al 1992; Zimmer et al 1992; Stein and Mancini 1999; Camacho et al 2001; van der Waal et al 2002; Mignogna et al 2003; Shapiro et al 2003; El-Hakim and Chauvin 2004; Perez-Calderon et al 2004; Cocuroccia et al 2005). Intravenous or oral methylprednisolone has also been associated with temporary improvements in orofacial swelling and facial paresis in several patients (Kesler et al 1998; Glickman et al 1992; Alioglu et al 2000; Ziem et al 2000; Mignogna et al 2003). Following steroid treatment, symptoms may improve or remit for weeks to months, but may also recur, necessitating maintenance therapy or repeated high-dose bolus administrations. Early treatment of facial swelling with steroids is advocated because the longer the delay in treatment, the less effective and incomplete the recovery (Mignogna et al 2003).

Antibiotics such as tetracycline, clofazimine, dapsone, and metronidazole have also been used empirically, not only for their antibactercial qualities but also for their reported properties in preventing the formation of granulomas (Kano et al 1992; Emiroglu et al 2016). Clofazimine was used as sole therapy in 18 patients and decreased the frequency and intensity of facial edema in 94%. However, only 62% of these patients were relapse free during a follow-up period of up to 3 years (Tausch and Sonnichsen 1992). Antibiotics are commonly used in combination with steroids and provide better clinical outcomes than either therapy alone (Fisher 1990; Camacho et al 2001). Clofazimine in conjunction with systemic or intralesional corticosteroids produced satisfactory results in 3 patients with Miescher cheilitis (Camacho-Alonso et al 2004). The combination of minocycline (tetracycline derivative) and prednisone produced favorable results with cessation of disease progression in 2 children who were 10 and 12 years of age; however, 1 patient required continued maintenance doses of minocycline with occasional intralesional steroid injections (Stein and Mancini 1999).

Traniast (N-[3,4-demethoxyxannamoyl]-anthranilic acid) is an oral cytokine inhibitor and mast cell membrane stabilizer, which has been reported to improve swelling of the eyelid in granulomatous blepharitis, a likely monosymptomatic variant of Melkersson-Rosenthal syndrome (Kato and Tagami 1986; Iwao et al 2003).

Infliximab is a chimeric, monoclonal antibody that binds soluble bioactive tumor necrosis factor alpha and neutralizes
its proinflammatory properties. An overproduction of tumor necrosis factor alpha contributes to the inflammatory mucosal and dermal damage in Melkersson-Rosenthal syndrome. Infliximab produced dramatic reduction of lip swelling in a 24-year-old woman with granulomatous cheilitis resistant to initial treatments with minocycline, erythromycin, and oral prednisolone (Barry et al 2005). Infliximab at 3 mg/kg administered at 0, 2, 6, and 9 weeks resulted in noticeable reduction in the swelling as early as the second infusion. The maintenance dose was increased to infusions of 5 mg/kg every 8 weeks, and the patient remained stable with restored anatomy of her lips. Another patient presenting with granulomatous blepharitis was successfully treated with infliximab until she developed side effects, at which time she was switched over to adalimumab (another monoclonal antibody directed against tumor necrosis factor alpha) with a good clinical response (Kakimoto et al 2007). Another patient with an atypical form of Melkersson-Rosenthal syndrome achieved total remission with adalimumab (Stein et al 2014).

For disfiguring swelling resistant to usual medical treatment, surgical resection of swollen tissue from the affected lips (chiloplasty) and eyelids (blepharoplasty) has been advocated. These surgical procedures may provide helpful restorative cosmetic outcomes (Zimmer et al 1992; Ellitsgaard et al 1993; Camacho et al 2001; Kruse-Losler et al 2005; Tan et al 2006), but not uncommonly, even after surgery, orofacial swelling may recur, necessitating repeat surgical procedures (Oliver and Scott 2002). Chiloplasty in combination with intradermal steroids (triamcinolone) and postoperative tetracycline may provide superior outcomes with longer periods of remission than surgery alone (Camacho et al 2001). Some authors recommend surgery only after a stable, relapse-free period of 8 to 12 months to avoid stimulation and aggravation of the granulomatous inflammatory process by the surgical procedure itself (Kruse-Losler et al 2005).

For recurrent or intractable facial palsies, some authors have recommended complete surgical decompression of the facial nerve (Graham and Kartush 1989; Dutt et al 2000; Dai et al 2014; Tan et al 2015). Dutt and colleagues reported diminished frequency and severity of facial palsies following middle fossa decompression of the facial nerve in a 27-year-old woman with Melkersson-Rosenthal and recurrent facial palsies (Dutt et al 2000). Complete cessation of recurrent facial palsy for greater than 8 years despite recurrent facial edema has also been reported in a patient with Melkersson-Rosenthal syndrome after facial nerve decompression (Graham and Kartush 1989).

As the usual course of the facial palsy in Melkersson-Rosenthal syndrome is one characterized by spontaneous remissions and relapses (Greene and Rogers 1989; Glickman et al 1992; Zimmer et al 1992; Rogers 1996), published reports of improvement in function after a particular interventional treatment should be carefully scrutinized with the natural course of the symptom in mind.

**References cited**


Fisher AA. Chronic lip edema with particular reference to the Melkersson-Rosenthal syndrome (MRS). Cutis 1990;45(3):144-6. PMID 2311430


Worsaae N, Christensen KC, Schiodt M, Reibel J. Melkersson-Rosenthal syndrome and cheilitis granulomatosa. A


Yetiser S, Satar B, Kazkayasi M. Immunologic abnormalities and surgical experiences in recurrent facial nerve paralysis. Otol Neurotol 2002;23(5):772-8; discussion 778. PMID 12218633


**References especially recommended by the author or editor for general reading.

**Former authors**

Lawrence Y Kim MD

**ICD and OMIM codes**

**ICD codes**

ICD-9:
Melkersson syndrome: 351.8

ICD-10:
Melkersson syndrome: G51.2

**Profile**

**Age range of presentation**

02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

**Sex preponderance**

female>male, >1:1

**Family history**

family history may be obtained

**Heredity**

heredity may be a factor

**Population groups selectively affected**

none

**Occupation groups selectively affected**
Differential diagnosis list

facial trauma
insect bites
cellulitis
lymphatic obstruction
eyelid lymphedema
eosinophilic facial swelling
periodontal and skull-based infections
silica-induced granulomatous cheilitis
contact dermatitis
acute leukemia
leprosy
syphilis
tuberculoid
sarcoidosis
Crohn disease
Bell palsy
skull trauma
Ramsey-Hunt syndrome
Herpes simplex virus-1
HIV
Lyme disease
sarcoidosis
tuberculosis
syphilis
vasculitis
acute demyelinating neuropathies
neoplastic lesions

Associated disorders

Bell palsy
Cheilitis granulomatosa
Crohn disease
Granulomatous blepharitis
Miescher cheilitis
Monosymptomatic Melkersson-Rosenthal syndrome
Oligosymptomatic Melkersson-Rosenthal syndrome

Other topics to consider

Bell palsy
Neurologic manifestations of Crohn disease
Ramsay Hunt syndrome