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Case report

Facial nerve palsy due to a parotid abscess: Two case reports and a review of literature

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ABSTRACT

Introduction and importance: Parotid gland swelling with facial nerve palsy is highly suggestive of a malignancy. Facial nerve palsy is however rarely caused by a parotid abscess. We hereby present two cases, propose treatment and present a review of the literature.

Case presentation and clinical discussion: One 75-year-old female and one 81-year-old female presented with a facial nerve paralysis, both caused by a parotid gland abscess. Broad-spectrum antibiotics and incision and drainage was commenced in both cases. Both patients showed good clinical improvement, however, without facial nerve improvement. Magnetic resonance imaging (MRI) scans showed no malignancies at presentation nor during follow-up after one year.

Conclusion: Facial nerve palsy is rarely caused by a parotid abscess. Incision and drainage in combination with antibiotic treatment is recommended. Chances of facial nerve recovery seem somewhat higher in patients with facial nerve paresis than those with a paralysis.

1. Introduction

Facial nerve palsy, combined with a pre-auricular mass, is usually highly suggestive of a primary salivary gland malignancy or metastasis in the parotid gland. Parotid abscesses are a very rare cause of facial nerve palsy or paralysis, with only 15 reported cases in the literature to date [1–13].

Here, we present two cases of facial nerve palsy and paralysis due to a parotid abscess and provide a literature review on facial nerve dysfunction due to benign parotid gland lesions. Subsequently, we provide insights into the diagnostic process, therapeutic options, and facial nerve recovery. This case report has been reported in line with the SCARE 2020 criteria [14].

2. Case one

A 75-year-old female presented at the outpatient clinic of a peripheral hospital with a painful, rapidly growing infra-auricular swelling extending into the neck at the left side, which had been present for approximately two weeks. Her medical history revealed that she was an active smoker, and she consumed alcohol on a frequent basis. Also, she was known to have hypertension and atrial fibrillation, for which she used anticoagulant therapy. The patient received vaccinations for the mumps, measles, and rubella through the Dutch civil vaccination program.

There were no signs of fever, dysphagia, dyspnea, or weight loss at presentation, nor did she have a history of parotid disease or skin cancer, or cutaneous lesions suspect for skin cancer of the head and neck. She did, however, experience xerostomia and odynophagia. Physical examination revealed an infra-auricular erythematous swelling on the left side, and intraoral examination showed pus discharge from the orifice of the left Stensen's duct, which was released while palpating the infra-auricular mass. A routine flexible nasopharyngolaryngoscopy showed a slight asymmetry at the base of the tongue but no signs of airway compromise. Swabs from the discharge were taken for microbiological examination. A thorough facial nerve examination showed no signs of facial palsy at this time.

Additional laboratory tests revealed a CRP of 191 mg/L (normal values <10 mg/L) and leukocytosis of 15.1 * 10^9/L (normal values 4,0–11,0 * 10^9/L). Computed tomography (CT) scan on the day of the presentation showed a heterogeneous structure at the left parotid gland of 6.2 × 5.9 × 6.7 cm with a discernible wall, suspected of a parotid gland abscess. A fine-needle aspiration cytopathological (FNAC) evaluation was performed, and broad-spectrum intravenous antibiotics

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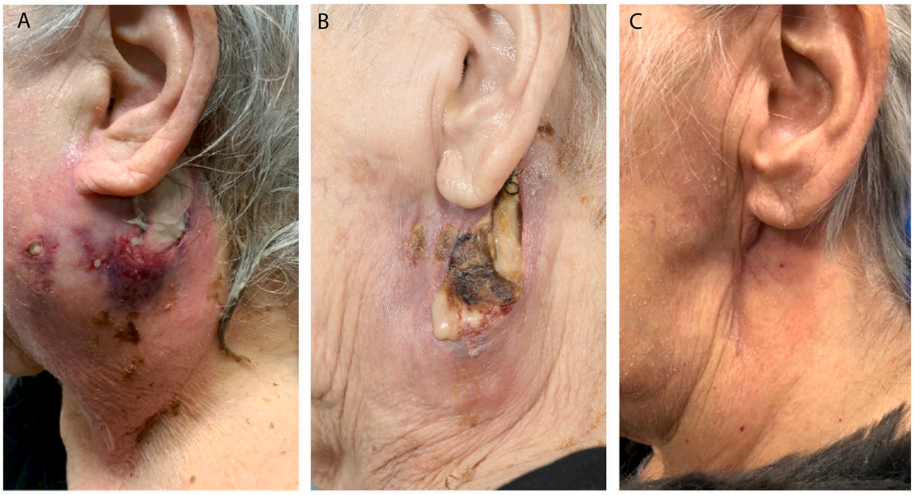

Fig. 1. Clinical images of case 1 at a) initial presentation in our academic hospital, b) discharge from our hospital, 15 days after admission, and c) 4 months follow-up.

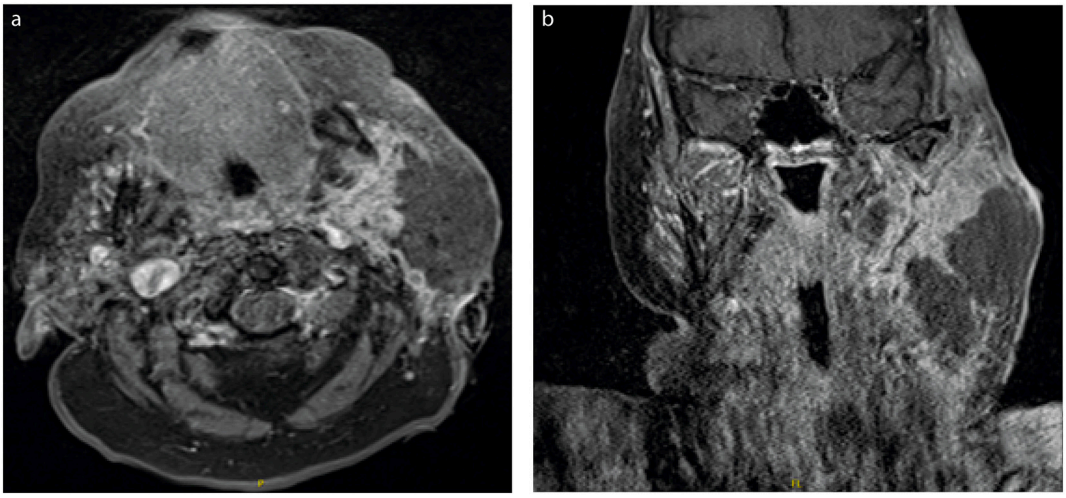

Fig. 2. MRI head and neck T1 coupes with fat suppression of case 1, at initial presentation in our academic hospital; a) transverse image, b) coronal image.

(amoxicillin/clavulanic acid 1.2 g 4 times daily) were started. During treatment, the FNAC provided the diagnosis of purulent infection, without signs of malignancy.

Following the initial five days of intravenous antibiotic treatment, the patient developed an ipsilateral facial nerve palsy. Also, there were no improvements in her infectious parameters, and a repeat CT-scan showed progression of the mass and further subcutaneous infiltration. Therefore, the clinicians decided on referral to our academic hospital. Physical examination in our center confirmed the infra-auricular mass that extended retro-auricular as well as down the neck (Fig. 1a). The swelling was non-fluctuating, painful on palpation, and there were three ulcerations on the overlying skin. The facial nerve paresis that was previously detected only affected the marginal mandibular branch. It was graded as House-Brackmann (HB) [15] grade II and Sunnybrook (SB) [16] score 79. Otomicroscopic evaluation of her left ear showed destruction of the canal floor, from which pus evacuated upon chewing. Intra-oral examination revealed the presence of dental prosthesis and no other foci of infection. The other previously mentioned examination findings were confirmed. Due to the observed facial nerve palsy and bone destruction, a malignancy of the parotid gland was suspected, and therefore a Magnetic Resonance Imaging (MRI) was performed. The MRI showed a large cystic/necrotic mass of 5.8 × 4.8 × 7.3 cm extending from the superficial lobe of the left parotid gland to the external ear canal, cutis, temporomandibular joint, and lower neck, which confirmed the earlier suspected parotid abscess on CT (Fig. 2). However, metastatic disease or a primary malignancy could not be ruled out. Subsequently, ultrasound-guided FNAC was repeated, and a superficial histopathological biopsy was taken from the parotid bed through one of the skin ulcerations. After one day, both these results came back negative for malignancy. After consultation with the radiologist and pathologist, we decided on deeper biopsies from the parotid gland to further rule out underlying malignant disease. The retro-auricular skin defect revealed the tense capsule overlying the swollen parotid gland. In local anaesthetics, this capsule was incised to help release the pressure, and with blunt dissection, a large quantity of pus was released from several pockets. New swabs were taken, and additionally, we performed deeper tissue biopsies. A Penrose drain was left at the surgical site for daily flushing with sodium chloride.

Swab results showed a *Staphylococcus aureus*, and intravenous antibiotic treatment was switched to intravenous flucloxacillin (1000 mg six times daily). Four days after the initial procedure, incision and drainage were repeated to drain pus from a deeper pocket. Histopathological examination of the deeper biopsies also showed necrotic infection without signs of malignancy nor a benign parotid tumor.

After these procedures, the patient's condition drastically improved. The infectious parameters declined, and the parotid swelling decreased.

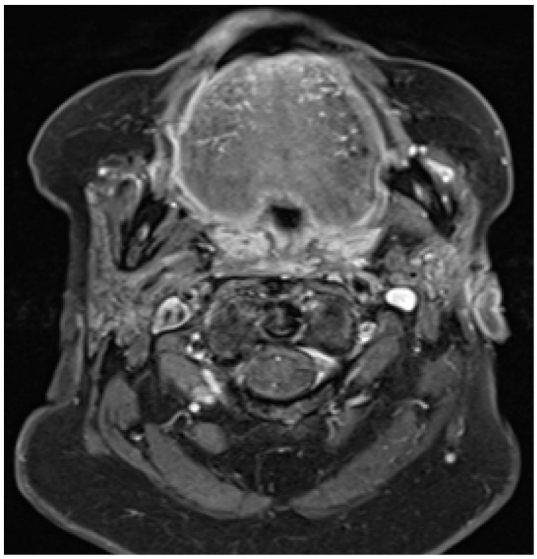

Fig. 3. MRI head and neck transverse T1 coupes with fat suppression of case 1 after 4 months.

The patient was eventually discharged fifteen days after initial admission and twelve days after admission to our academic hospital (Fig. 1b). Oral clindamycin (600 mg three times daily) was continued up to five days after discharge, and wound dressings were changed daily at home. However, upon discharge, the facial nerve function had not improved (HB grade II).

After two and four months, MRI scans showed a healed cutaneous tissue defect, parotid gland atrophy, and no signs of malignancy (Fig. 3). At six months follow-up, the wound had healed (Fig. 1c), but the facial nerve palsy (HB grade II) was not improved. Follow-up after one year also showed no improvement of the facial nerve palsy.

3. Case two

An 81-year-old female patient was first seen at the outpatient clinic of a peripheral hospital with a painful, pre-auricular swelling on the right side. Her medical history revealed hypertension, diabetes mellitus type II, atrial fibrillation for which she used anticoagulant therapy, mild renal impairment, and status after Wertheim-Megs surgery for a carcinosarcoma of the uterus ten years before the consultation. She had no history of parotid disease or skin cancer, nor did she currently have cutaneous lesions suspect for skin cancer. This patient also received all her vaccinations through the Dutch civil vaccination program.

At the first presentation on the outpatient clinic of a peripheral hospital, there were no other symptoms than pre-auricular pain. Physical examination revealed a pre-auricular swelling at the right side, and the intra-oral examination also showed purulent discharge from the orifice of Stensen's duct. There were no other abnormal signs on physical examination. In particular, the facial nerve examination showed no signs of facial nerve palsy. Subsequent swabs were taken from the purulent discharge for microbiological analysis, and clarithromycin antibiotic therapy (250 mg twice daily) was started. Five days after the initial consultation, the patient noticed that she was unable to shut her right eye. Initially, the swelling declined at the start of the antibiotic treatment, but it started to grow again after a short period of time. She did not experience any of the following: pain, trauma, dizziness, tick-bite, hearing loss, or purulent discharge from the ear. The swab examination revealed a *Methicillin-Resistant Staphylococcus aureus (MRSA)*. On further physical examination, there was a facial nerve paralysis (HB grade VI, SB score 10) present on the right side. Furthermore, a spontaneous rupture of the pre-auricular swelling was seen, from which purulent discharge evacuated. The patient was referred to our academic hospital for further diagnostic and therapeutic possibilities.

Physical examination at the academic hospital showed a pre-auricular mass of about 6 × 8 cm extending to the upper neck levels I, II, and III (Fig. 4a). The overlying skin was erythematous and had several ulcerations. The facial nerve paralysis was confirmed, and all other cranial nerves proved intact. Routine flexible nasopharyngolaryngoscopy showed parapharyngeal bulging on the right side, accompanied by a slight uvula-shift to the left. There were no abnormalities on otomicroscopy, and intra-oral examination showed good oral hygiene and no further infection foci.

Laboratory tests showed a CRP of 360 mg/L (normal values <10 mg/L), leukocytosis of 19.2 * 10^9/L (normal values 4,0–11,0 * 10^9/L) and a severe hyperglycemia of 38.3 mmol/L. CT scan showed a swollen right parotid gland with a hypodense collection of 9,5 × 4,5 × 6,6 cm in the parapharyngeal space expanding towards the cutis and the palatine tonsil, with tapering of the internal carotid artery and presence of subcutaneous infiltration suspect for a deep parotid abscess (Fig. 5).

The same night surgical drainage of the abscess was performed, various additional microbiological swabs were taken, and multiple deep pus pockets were released. These were located anteriorly of the sternocleidomastoid muscle in the neck, posterior to the submandibular gland, medial to the mandible towards the parapharyngeal space near the palatine tonsil, and superficially over the parotid gland capsule. Three Penrose drains were left at the surgical site for four times daily flushing with sodium chloride 0,9%. Antibiotic treatment was switched to intravenous teicoplanin (400 mg once daily) because of the MRSA

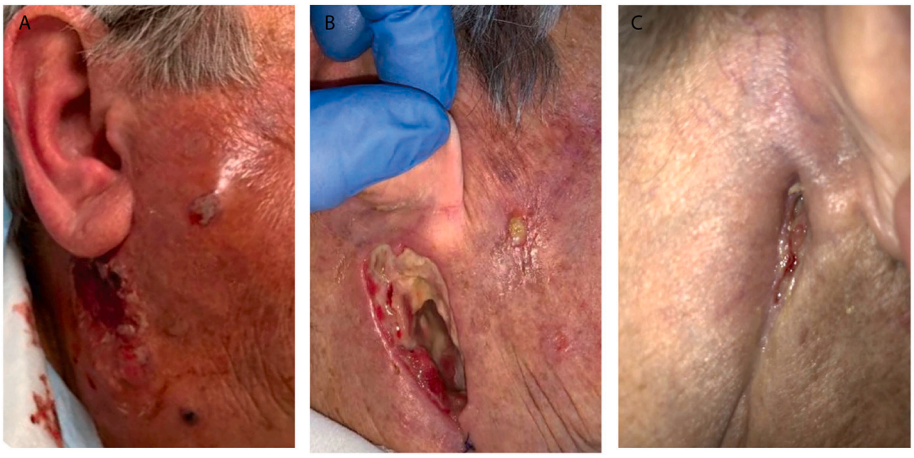

Fig. 4. Clinical images of case 2 at a) initial presentation in our academic hospital b) after removal of all drains, 11 days after admission and c) at 3 months follow-up.

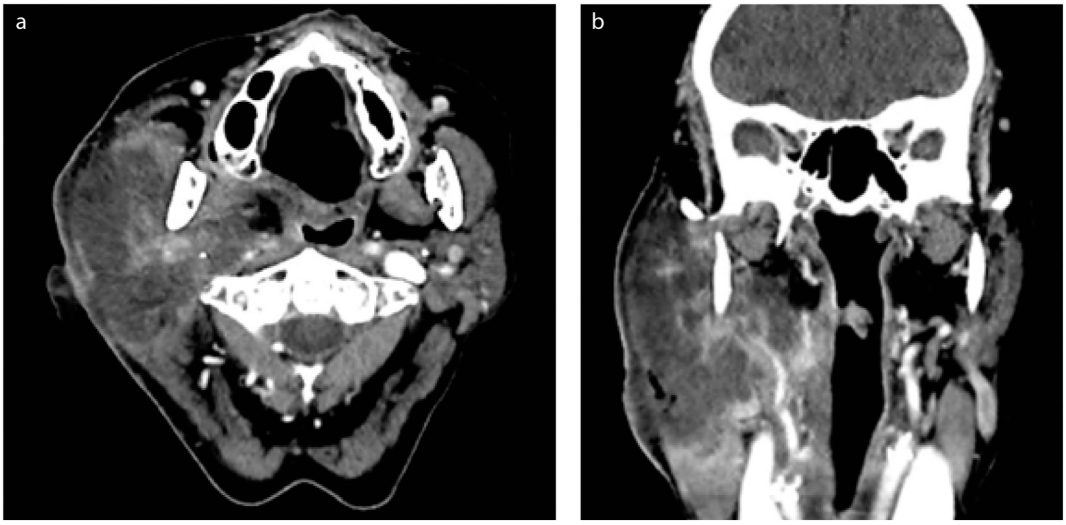

Fig. 5. CT head and neck with contrast of case 2, at initial presentation; a) transverse image, b) coronal image.

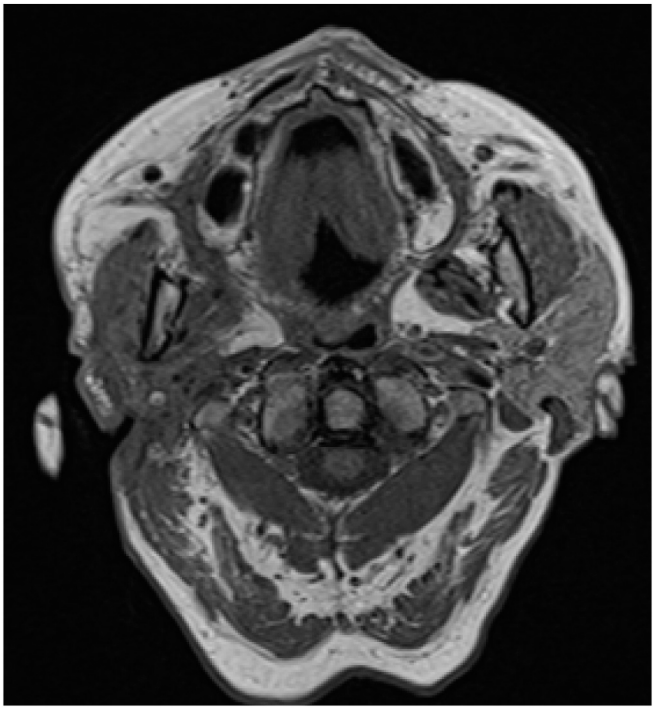

Fig. 6. MRI head and neck transverse T1 coupes of case 2 after 4 months.

colonisation. Internal medicine was consulted to regulate the hyperglycemia.

Over the course of seven days, all drains were independently removed. After ten days of antibiotic treatment, intravenous antibiotics were switched to oral linezolid (600 mg twice daily). The patient developed neutropenia on linezolid, which caused another switch of antibiotics to levofloxacin (250 mg twice daily). To facilitate wound healing, several debridements of the surgical site were performed in the following two weeks. The facial nerve paralysis did not improve. Eventually, the patient was discharged from the hospital 22 days after admission to our academic hospital.

An MRI scan performed two months after discharge showed volume loss of the parotid gland and no signs of malignancy in the course of the facial nerve (Fig. 6). Six months after treatment, no improvement of the facial nerve was seen. Together with the patient we decided on functional static reconstruction rather than a dynamic reconstruction considering the wish and age of the patient. Therefore, the patient was scheduled for the implantation of a gold-weighted implant in the upper eyelid, a treatment for persistent lagophthalmos caused by the facial nerve paralysis.

4. Discussion

Peripheral facial nerve palsy or paralysis of the extratemporal part of the facial nerve secondary to benign pathology is exceedingly rare. Malignancies of the parotid gland are more likely to cause facial nerve palsy due to compression or perineural tumor invasion of the facial nerve. Therefore, a malignancy should be ruled out in patients with a parotid mass and facial nerve dysfunction. There are, however, several case reports in the literature that have described benign lesions such as pleomorphic adenoma [17], (cystic) Warthin tumors [18,19], (lympho) epithelial cysts [20,21], epidermoid cysts [22], keratocystomas [23], oncocytomas [24] and intraparotid facial nerve schwannomas [25] to cause peripheral facial nerve palsy.

Some parotitis cases causing peripheral facial nerve paralysis have been reported in children and adults after mumps parotitis [26] or parotitis caused by the Epstein Barr virus [27]. Parotid abscesses are another benign yet extremely rare cause of facial nerve palsy or paralysis, with only 15 reported cases in the literature to date [1–13]..

Parotitis itself can have numerous causes. The most common is acute bacterial parotitis, which can develop following an ascending infection up the parotid duct (Stenson's duct), primary parenchymal involvement, or through peri- and intraparotideal lymph nodes. Also, *Mycobacterium tuberculosis* can cause parotitis. The *Paramyxovirus* is the most frequent cause of acute viral parotitis, otherwise known as the “mumps”. Sjögren syndrome can be an autoimmune cause of parotitis.

A parotid gland abscess is most frequently caused by acute bacterial parotitis. Predisposing factors may include diabetes mellitus, autoimmune diseases, immunosuppression, dehydration, and poor oral hygiene. A parotid abscess's clinical manifestations might include local pain, pre-auricular and infra-auricular swelling, erythema, dysgeusia, fever, trismus, and occasionally referred pain to the ear, jaw, or neck. Manual palpation of the parotid gland may result in suppurative discharge from Stensen's duct, and signs of systemic dehydration may be found during physical examination.

In all 17 patients described to date, including our two cases, parotid abscesses with facial dysfunction are more common in female patients than in male patients (12:3), occurring at all ages ranging from ten months to 87 years. Facial nerve palsy was seen in 13 patients, and facial

Table 1.
Literature review facial palsy by parotid abscess.

	Author (year)	Sex	Age (years)	Underlying disease	Superficial/ deep abscess	Side	House-Brackmann at diagnosis	Bacterial culture	Treatment	Time from start palsy to surgical treatment	House-Brackmann at end follow-up	Follow-up
1	Perry (1985) [2]	Male	41	Acute bacterial endocarditis	ND	Left	HB IV	*Pseudomonas aeruginosa*	IV clindamycin 900 mg tds + gentamycin 100 mg tds, later IV tobramycin 100 mg tds + incision and drainage	10 days	ND	49 days
2	Shone et al. (1985) [1]	Female	77	Congestive heart failure, renal failure, dehydration	Superficial	Right	HB VI	*S. aureus*	IV antibiotics + partial gland excision under local anesthesia	ND	Deceased[a]	5 weeks
3	Smith et al. (1997) [3]	Female	68		ND	Left	HB VI	S. aureus	IV nafcillin and oral metronidazole + incision and drainage	ND	HB I	4 months
4	Marioni et al. (2003) [4]	Male	74	DM	ND	Right	HB IV	*Candida albicans*	Fluconazole 400 mg qd + 2× incision and drainage	Same day	HB I	3 months
5	Tan et al. (2007) [5]	ND	ND	DM	ND	ND	HB III	No growth	IV antibiotics + incision and drainage	<2 days	HB III	2 years
6	Tan et al. (2007) [5]	ND	ND	DM, septic	ND	ND	HB IV	Pseudomonas aeruginosa, S. aureus	IV antibiotics + incision and drainage	<2 days	Deceased[b]	
7	Orhan et al. (2008) [6]	Female	45		ND	Left	HB V	No growth	IM clindamycin 600 mg bd	NA	HB I	3 months
8	Athar et al. (2009) [7]	Female	72	DM	Deep	Right	HB VI	Klebsiella	IV amoxicillin + incision and drainage via modified Blair incision. One week later surgical debridement necrotic parotid tissue	ND	HB VI	6 months
9	Noorizan et al. (2009) [8]	Female	40	DM	Deep	Left	HB IV	No growth	IV augmentin 1200 mg tds and IV metronidazole 500 mg tds + incision and drainage	Same day	HB I	6 months
10	Kristensen et al. (2012) [9]	Female	22		Both	Left	HB IV	MRSA	IV benzylpenicillin, dicloxacillin and metronidazole. Surgery: acute tonsillectomy + US drainage superficial abscesses. Repeated US drainage multi- loculated abscess two days later. After MRSA swab oral clindamycin 4 weeks	Same day	HB IV	1 month
11	Kristensen et al. (2012) [9]	Female	46		Deep	Right	HB II	*Propionibacterium acnes*	IV ceftriaxon and metronidazole + acute tonsillectomy and deep lope incision of parotid gland	Same day	HB I	5 days
12	Hajioannou et al. (2013) [10]	Female	87	Dehydration	Deep	Left	HB V	No growth	IV empiric antibiotics + incision and drainage	ND	HB II	15 days
13	Anitha et al. (2014) [11]	Female	10 months		ND	Left	Facial nerve paresis	No growth	IV metrogyl, co-amoxyclav and ceftriaxone + incison and drainage	ND	HB I	10 days
14	Alam et al. (2016) [12]	Female	50		Deep	Left	HB IV	Fusobacterium, *Bacteroides fragilis*, S. aureus, Klebsiella	IV co-amoxiclav 1200 mg tds, amikacin 500 mg bd and metronidazole 500 mg tds + incision and drainage	ND	HB I	2 months
15	Kim et al. (2018) [13]	Male	7	Neutropenia	Superficial	Left	Facial nerve paresis	Pseudomonas aeruginosa	Iv broad-spectrum antibiotics +2× incision and drainage	ND	no improvement	3 years
16	This study (2020)	Female	75		Both	Left	HB II	S. aureus	IV co-amoxiclav 1200 mg qid + incision and drainage 2×, and capsule incision	3 days	HB II	12 months
17	This study (2020)	Female	81	DM	Both	Right	HB VI	MRSA	IV teicoplanin 400 mg qd + incision and drainage	1 day	HB VI	9 months

DM = Diabetes mellitus NA = Not applicable ND = Not described.
[a] Due to congestive heart failure.
[b] Due to septicaemia + aspiration pneumoniae after difficult intubation.

nerve paralysis in four patients with a parotid abscess. Both the palsies and paralyses gradually developed during the course of the parotid abscess. *Staphylococcus aureus* [1,3,5,12] and *Pseudomonas aeruginosa* [2,5,13] are the most common bacteria involved in parotid abscesses with facial nerve involvement. However, infections with *Klebsiella* [7,12], *Propionibacterium acnes* [9], *Candida albicans* [19], *Fusobacterium* [12], *Bacteroides fragilis* [12], and *MRSA* [9] have also been reported. In five case reports, culturing purulent material failed to yield any bacteria or fungi [5,6,8,10,11], possibly due to oral or intravenous antibiotics had been administered before swabs were taken. All details of the 17 cases are summarized in Table 1.

Several mechanisms of the pathogenesis of facial nerve dysfunction resulting from a parotid abscess have been proposed. Compression of the facial nerve, leading to ischemic neuropathy due to mechanical pressure of the abscess, is one of the suggested mechanisms that was histologically confirmed in a case of a Warthin tumor causing facial nerve palsy [28]. Another proposed factor contributing to ischemic neuropathy is the virulence of the offending organism in the abscess, releasing endotoxins and exotoxins, causing perineuritis [8,10]..

The presence of a parotid gland abscess in combination with facial nerve dysfunction warrants further detailed examination because of the possibility of underlying malignancy. Ultrasound-guided FNAC is the routine diagnostic measure for discriminating between benign and malignant tumors of the parotid gland. This may prove useful in the acute stage when a mass is seen or suspected. However, when no mass is found and considering the intermediate sensitivity of FNAC [29], it is also advised to perform an MRI scan after the acute stage of the infection as MRI is the most accurate imaging study to evaluate parotid gland masses.

When an abscess of the parotid gland is confirmed, infection parameters should be assessed, and swabs of purulent discharge should be taken, preferably before the start of antibiotic treatment. Ideally, the treatment, besides analgesics, consists of two main phases. First, it is advised to start broad-spectrum intravenous antibiotics, covering gram-positive, gram-negative, and anaerobes, while awaiting microbiological results. However, considering that antibiotics alone most often do not prove sufficient, as the antibiotic infiltration into the abscess is minimal, the second phase of treatment consists of surgical incision and abscess drainage. This is required to remove the purulent discharge and release the pressure from the facial nerve in an attempt to preserve the facial nerve and rehabilitate its function. Limited but adequate drainage seems advocated in small abscesses with limited parotid swelling, but in extensive or rapidly progressing abscesses, quick and repeated drainage seems warranted. Additionally, measures such as hydration, sialogogues, parotid gland massage, and good oral hygiene are endorsed.

Of the 13 patients with facial nerve palsy, one patient deceased short after hospital admission due to aspiration pneumonia after difficult intubation and an overwhelming septicaemia [5], and one patient lacked information on the follow-up of facial nerve performance [2]. At follow-up, varying from 5 days until three years, facial nerve function fully recovered in six out of the remaining 11 patients (55%) [4,6,8,9,11,12], one patient (9%) [10] showed some recovery, while no improvement was seen in four patients, including our patient (case 1) (36%) [5,9,13].

Of the four patients with facial paralysis, one patient deceased during hospitalization within five weeks of onset due to congestive heart- and renal failure [1]. Facial nerve function was normalized in one patient at four months follow-up (33%) [3], but remained paralyzed in two other patients (67%) [7], including one of our patients (case 2), at six months follow-up.

5. Conclusion

We described two unique cases of facial palsy and facial paralysis resulting from parotid abscesses. Facial nerve palsy or paralysis caused by benign parotid gland pathology is rare, especially in the case of a parotid abscess. If a swelling of the parotid gland is associated with facial nerve dysfunction, underlying malignancy should always be ruled out. As in all parotid abscesses, incision and drainage are recommended in patients with facial nerve dysfunction to avoid ischemic neuropathy by pressure, combined with targeted intravenous antibiotics. Based on the 17 described patients, the chances of complete facial nerve recovery seem somewhat higher in patients with facial nerve paresis than those with paralysis. Excluding the 3 cases without follow-up, facial nerve recovery was complete in 6 out of 11 patients (55%) with facial nerve palsy, and in 1 out of 3 patients (33%) with facial nerve paralysis.

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Ethical approval

Not applicable.

Informed consent

Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution

IP and SR wrote the paper, collected and analysed data. DW, CN and HS all critically reviewed the paper and contributed to the intellectual content.

Research registration (for case reports detailing a new surgical technique or new equipment/technology)

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Data statement

The deidentified individual participant data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Declaration of competing interest

None to declare.

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