of pulmonary involvement. The skin, the eyes, the lymph nodes, the liver, the spleen, the heart, the nervous system, and the salivary glands are often involved.[2] The main targets in the head and neck region are the lymph nodes, the skin, the nose, the oral mucosa, the palatine tonsils, the larynx, the trachea, the cranial nerves, and the salivary glands.[8]

The diagnosis of sarcoidosis is based on the finding of noncaseating granulomas, concordant clinicoradiologic findings, and exclusion of other granulomatous disorders.[2] There is no laboratory diagnostic test for sarcoidosis. Results of a laboratory evaluation may reveal an elevated erythrocyte sedimentation rate or other acute reactants. Anemia, leukopenia, and eosinophilia are commonly seen in blood counts. Hypercalcemia and/or hypercalciuria may be found.[8] Serum ACE is commonly elevated in sarcoidosis. The diagnostic value of ACE is debated because its elevation is both nonspecific and insensitive. However, increased ACE is considered useful in monitoring the course of disease.[1] Histologically, the nonnecrotizing inflammatory process with formation of epithelioid cell granulomas characterized of sarcoidosis can be differentiated from the histologic changes occurring in tuberculosis, Hodgkin disease, non–Hodgkin lymphoma, and especially in the Sjögren syndrome.[5]

The differential diagnosis for bilateral parotid swelling includes masseteric hypertrophy, sialoadenosis, bulimia, acute suppurative parotitis, mumps, human immunodeficiency virus, recurrent parotitis, the Sjögren syndrome, Wegener granulomatosis, sarcoidosis, the Kimura disease, polycystic parotid disease, pneumoparotid, papillary cystadenoma lymphomatosum, MALT lymphoma, and radioactive iodine.[9] In the head and the neck, sarcoidosis is commonly involved with the cervical lymph nodes (40%), the globe (50%), the parotid (7%), and the larynx (6%).[10] Salivary gland involvement is the most frequent in the parotids (macroscopically) and the accessory salivary glands (microscopically), in 5% and 50% of all cases, respectively.[8] Enlarged parotid glands involved by sarcoidosis are bilateral, nontender, and nodular.

The parotid glands are typically enlarged, with high signal intensity on T2-weighted images and enhancement on contrast-enhanced images. Parotid disease may appear as multiple, benign-appearing, noncavitating masses with a "foamy" appearance that is often associated with cervical adenopathy.[10] The glands tend to be firm, only slightly painful, and they do not fluctuate in size when eating. Because the sarcoidal granulomas replace the glandular parenchyma, a moderate decrease in salivary production results.[9] If a patient has bilateral parotid enlargement with uveitis and facial paralysis, the condition is referred to as the Heerfordt syndrome. This patient had bilateral parotid swelling; however, there was no facial palsy to suggest a Heerfordt syndrome.

Treatment of sarcoidosis may range from observation in asymptomatic or mild disease to systemic corticosteroid therapy in more severe cases. In the treatment of parotid gland sarcoidosis, pharmacotherapy is mostly applied. Some alternative agents such azathioprine, chloroquine, hydroxychloroquine, methotrexate, chlorambucil, pentoxifylline, cyclophosphamide, tetracycline derivatives, and infliximab may be used in refractory disease or as corticosteroid-sparing agents. Some patients, especially those with isolated disease, do not require pharmacotherapy because the disease may remit spontaneously. Isolated changes in the parotid glands can also be surgically removed, and when there are no other signs of the disease, pharmacotherapy is not required.[4,7,9] Our patient refused therapy; thus, routine check was suggested.

CONCLUSIONS

In summary, as mentioned previously, sarcoidosis is a multisystemic disease, with head and neck involvement. In rare cases, salivary gland involvement may not be accompanied by systemic symptoms. Bilateral parotid mass without systemic symptoms should be questioned for relevance of sarcoidosis by otorhinolaryngologists.

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Long-Term Complication After Rhinoplasty Using Porous Polyethylene Implant: Cutaneous Fistula of the Forehead

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Abstract: Nasal dorsum augmentation is one of the most frequently performed procedures during rhinoplasty, especially in Asians and reconstructive cases. One can use autogenous cartilage grafts or alloplastic implants for this purpose. However, the potential for permanent damage to the skin and soft tissues as well as complications such as infection and extrusion of the implant make autogenous tissue augmentation preferable to alloplastic implantation. Furthermore, there is scant literature information about long-term outcomes and complications related to these implants. This brief report aimed to

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describe a unique case of migration of an alloplastic implant from the nose to the forehead, simulating a frontal sinus fistula.
Level of Evidence: IV

Key Words: Rhinoplasty, Medpor, complications, alloplastics, implants

A 54-year-old healthy man presented with a small pustule in the lower central forehead that had progressed with intermittent discharge of unknown duration. Examination results showed a thickened subcutaneous orifice along the entire forehead as well as a friable and small erythematous orifice near the glabella (Fig. 1). His medical history was confusing, and data were limited in the preoperative consultations (occurring in year 2004). Computed tomographic imaging results were obtained and showed a blind-ended frontal sinus fistula (Fig. 2) without other positive findings. The clinical impression was a chronic infectious process evaluated through radiologic imaging and was confirmed as a cutaneous fistula of the forehead.

The surgery was performed under general anesthesia using a direct (median) approach to explore the fistula. An alloplastic material (porous polyethylene implant) (Medpor; Porex Surgical, College Park, GA) was visualized above the frontal bone (Fig. 3), which was removed. No gross purulent material was present during the removal. Skin closure was performed, and no dressing was used after the surgery. The wound had healed completely in a couple of weeks.

DISCUSSION

This study describes a patient who developed a forehead cutaneous fistula caused by migration of an alloplastic implant. Lack of appropriate data collection was caused by a confused patient, which played an important role in this outcome. After the surgery, the medical history was collected again and the patient confirmed that he had a rhinoplasty 10 years prior; however, he did not know that the surgeon used an implant for nasal dorsum augmentation. The unusual localization of the fistula also contributed to the mistaken diagnosis because, in previous series, most of the extrusions secondary to nasal implants cited are related to ulceration of the nose itself. Another important contributing factor was that radiologic images obtained through a non–multislice computed tomography could have induced a wrong diagnosis. Ozturk et al[1] reported a Medpor fracture (nasal dorsum implant) confirmed through multislice computed tomography, suggesting the good accuracy of the method in the proper visualization of the Medpor implant material.

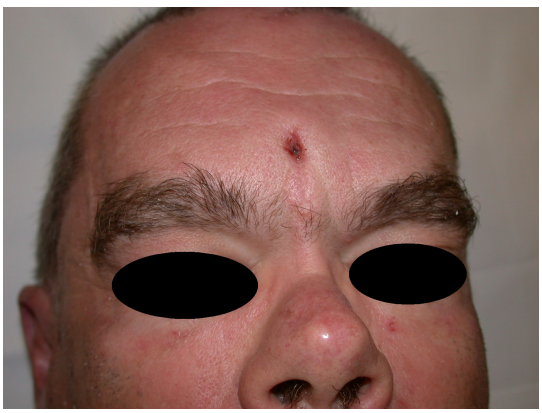

FIGURE 1. Frontal sinus fistula near the glabella.

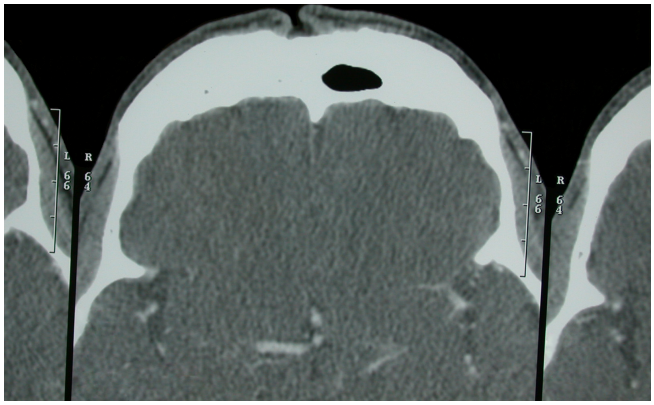

FIGURE 2. Computed tomographic image showing a blind-ended frontal sinus fistula.

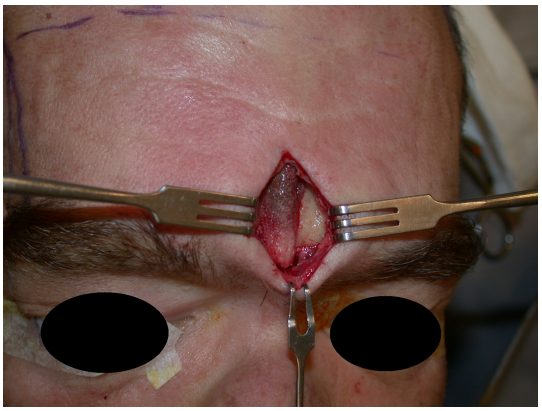

FIGURE 3. Alloplastic material visualized above the frontal bone intraoperatively.

One of the main advantages of using alloplastic implants in the nose is the ease of use. Alloplasts require much less time in preparation of the pocket and sculpting when compared with autogenous tissue.[2] The 3 most commonly used implants in rhinoplasty are Medpor, silicone, and expanded polytetrafluoroethylene (Gore-Tex).[3] Medpor has been used in a variety of facial reconstructions since the 1980s. Others authors have suggested that Medpor is a much more versatile implant because it is easy to shape, flexible, remarkably stable, and exhibits rapid soft-tissue ingrowth.[4] Moreover, dislocation and migration have not yet been reported using this material. Nevertheless, a literature review showed extrusion and infection rates ranging from 2.8% to 7.4%, with the need for Medpor removal in this series.[3,5] Nasal cyst formation and implant migration have been reported after silicone augmentation;[6] whereas, to our knowledge, there is no report in the literature of such complications with Medpor, including migration of a nasal dorsum Medpor implant to the forehead 10 years after a rhinoplasty, presenting as a local fistula. Therefore, we believe that complication rates of such a procedure may be higher in a long-term evaluation than that initially reported.

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An Unforeseen Complication Arising From Inferior Alveolar Nerve Block: Is Anemia Possible?

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Abstract: Complications after administration of local anesthesia for dental procedures are well recognized. We present here 2 cases of patients with anemic areas on their faces resulting from inferior alveolar nerve block (IANB). The precise cause of this complication is unknown; however, it may be derived from anastomosis of the maxillary artery, rapid injection of local anesthetic solution, misdirection of the needle, and spread of the solution to the upper region of the mandible. Although neurologic occurrences resulting from IANB are rare, dentists should keep in mind that certain dental procedures such as administering IANB could cause anemic areas on the face. Henceforth, dentists should consider the possibility of anemia after administration of IANB and pay attention to avoid complications during the procedure.

Key Words: Inferior alveolar nerve block, complication, anemia

Intraoral administration of local anesthetics is one of the most common dental procedures.[1] The inferior alveolar nerve, also known as the mandibular nerve, is the third and most inferior division of the trigeminal, or fifth, cranial nerve. This nerve innervates the teeth in the half of mandible with the lower lip, skin, and mucosa of chin in the ipsilateral.[2] Inferior alveolar nerve block (IANB) are administered in dentistry for years, and the most common neurological complication following IANB is a facial nerve palsy.[3]

Anemia develops from decreased blood flow to the tissues as a result of blood vessels narrowed by adrenaline. This is a rare complication arising from a posterior superior alveolar nerve block because of the maxillary artery.

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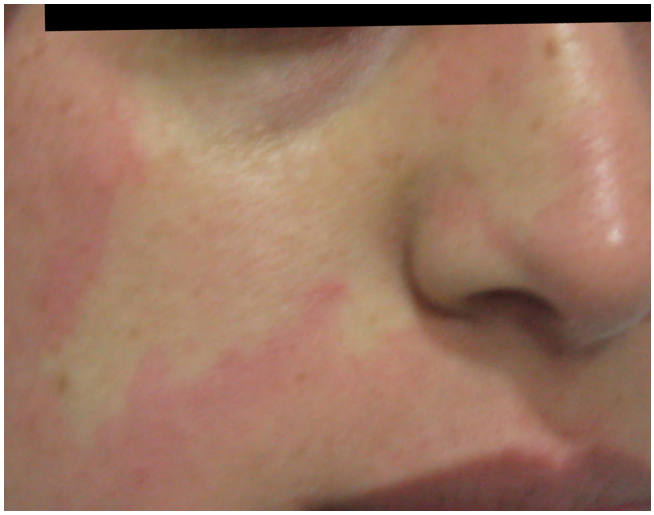

FIGURE 1. The clinical view of patient 1.

CLINICAL REPORT

Two female (aged 29 and 32 years) patients were referred to the Oral and Maxillofacial Surgery Department of our faculty complaining of painful mandibular third molars. To extract these teeth, an IANB and infiltrating anesthesia of the buccal nerve trajectory were used. The patients were normally healthy, were taking no medication at that time, and had no known allergic or toxic reaction to any local anesthetic agent. We used a 24-mm-long disposable 3-mL syringe and 27-gauge-size needles for a conventional IANB via the direct intraoral approach. The local anesthetic agent—Ultracain D-S forte (Sanofi-Aventis)—contained articaine HCl (40 mg/mL) and epinephrine (adrenaline, 0.012 mg/mL). The solution was administered 1–1.5 mL to the IANB and 0.5 mL to anesthetize the lingual nerve. No positive aspirations were encountered during the administration of the IANB. Afterwards, an anemic area on the face covered the lateral nasal wall, inferior orbital ridge, and 1 cheek (Figs. 1, 2). In patient 2, this anemic area also covered half of her superior lip. After 20–30 minutes, all areas returned to normal.

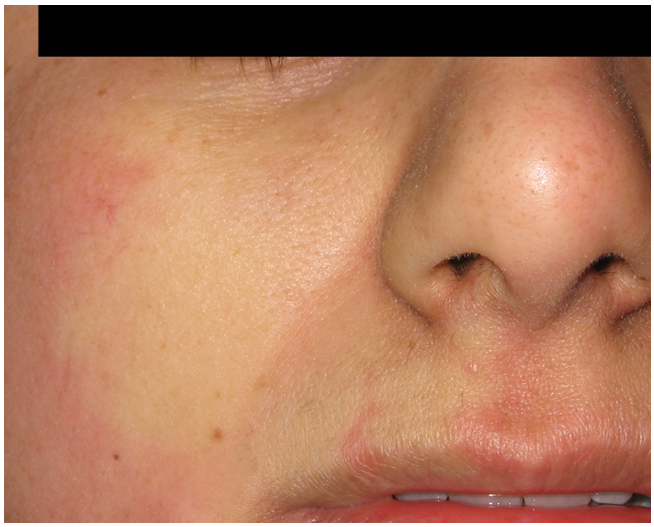

FIGURE 2. The clinical view of patient 2.

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