Bone Graft and Implants in a Patient with Systemic Mastocytosis

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ABSTRACT

Background: Systemic mastocytosis (mast-cell proliferation in various organs, including the skeleton) is a rare disease. Reports on mastocytosis that affects facial bones are few. The bone lesions may be osteolytic or sclerotic.

Purpose: To describe (for the first time) bone grafting followed by dental implant treatment yielding a good result in a patient with systemic mastocytosis.

Materials and Methods: A bone graft was performed on a 60-year-old woman with systemic mastocytosis. Dental implant treatment was performed 13 weeks after sclerotic bone of the iliac crest was grafted to the maxillary sinus bilaterally. A microimplant was installed simultaneously with the dental implants and was removed 6 months later for histomorphometric evaluation. Bone biopsy specimens from the donor site of the sclerotic iliac crest and later from the remodeled maxillary bone graft were histologically analyzed. A clinical examination of the patient with regard to her mastocytosis was performed by a dermatologist. The patient was followed up after 3 years.

Results: Bone grafting and dental implant treatment were successful, and the patient's clinical and radiologic situation was stable after 3 years. Histologic examination of the bone grafted from the iliac crest showed sclerotic lesions in the bone and a dense infiltration of mast cells. The bone graft seemed to remodel initially in a normal way in the maxillary sinus. However, computed tomography 3 years later showed regions of sclerosis in the remodeled maxillary bone. These lesions now had a pattern similar to the adjacent facial bone. Both the microimplant and the dental implants integrated well. Bone-implant contact measured on the microimplants was 20% higher in this actual case, compared to that of patients previously treated and grafted with the same technique.

Conclusions: There are many clinical implications to be considered when treating this group of patients. Bone grafting, remodeling of the bone, and dental implant installation were successful in this patient with systemic mastocytosis and signs of osteosclerosis. Installation of microimplants in patients with pathologic bone conditions may allow successful dental implant treatment.

KEY WORDS: bone graft, dental implants, histologic evaluation, mastocytosis, pathologic condition

Mastocytosis is a rare disease characterized by pathologic mast cell proliferation in various organs. The mast cell derives from bone marrow and is a mobile cell of great immunologic importance. The human mast cell is a secretory cell that releases histamines, proteoglycans (heparin, chondroitin sulfate), neutral proteases, acid hydrolases, lipid mediators (leukotrienes B_4, C_4, and D; platelet-activating factors; and prostaglandin D_2), chemokines, and cytokines (tumor necrosis factor-α, eosinophil leukocyte chemotactic factor, and interleukin-8).[1]

The signs and symptoms of mastocytosis are due to the release of these products and the localization of mast cells in different organs. The skin is often involved in all forms of mastocytosis. Urticaria pigmentosa (UP) is the most common skin manifestation in adults. The skin symptoms are usually those of flushing and red-brown pigmented pruritic lesions.

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Among patients with mastocytosis 11 to 33% have extracutaneous symptoms and mast-cell proliferation in bone marrow or other organs (ie, skeleton, gastrointestinal tract, liver, spleen, lymphatic glands, cardiopulmonary system, and nervous system).[2] After the skin, the skeleton is the most common organ involved. In a large study of systemic mastocytosis patients, 57% had radiologic evidence of diffuse bone involvement, and 9% had focal lesions. Diffuse demineralization, osteosclerosis, fractures, and a focal pattern were seen.[3] Medina and colleagues reported a case of mastocytosis in which an osteolytic lesion was found only in the mandible.[4] Bone marrow findings (eg, increased eosinophils, lymphocytes, and cellular aggregates of spindle-shaped mast cells) occur in approximately 80% of adults with systemic mastocytosis.

More than 35% of the patients have gastrointestinal involvement such as diarrhea, esophagitis, peptic ulcer, pain, or gastrointestinal bleeding.[2] Headache, bone pain, hypotension, palpitations, vascular collapse, syncope, and dyspnea are other symptoms observed in some patients with systemic mastocytosis.[1] The severity of the systemic disease appears to correlate with a later age of onset and extensive cutaneous lesions.[5]

Mast cells are normally found in all types of tissue in the oral cavity and play an important role in inflammatory processes.[6] In several oral conditions such as peripheral inflammation, pulpitis, gingivitis, and lichen ruber planus, mast cells release TNF-α, a cytokine that has the ability to promote leukocyte infiltration in the oral tissues.[6] Proteases secreted from mast cells may also disrupt the balance in basement membranes, allowing lymphocyte migration into the epithelium and thus causing cytotoxic action.[6]

The patient treated in the present study had systemic mastocytosis, osteosclerosis, and periodontitis and underwent a bone grafting procedure from the iliac crest to the maxilla, followed by installation of dental implants and a microimplant. Bone graft procedures followed by dental implant treatment in patients with mastocytosis have not previously been reported. The microimplant was later removed at the abutment operation. Histologic analysis was done on biopsy specimens of the bone graft from the iliac crest, the healed bone in the maxilla, and the removed microimplant. Results from these analyses and from the 3-year follow-up with computed tomography (CT) of the maxilla are also reported.

MATERIALS AND METHODS

A 60-year-old female with periodontal disease in the right upper jaw was initially referred to the Department of Oral and Maxillofacial Surgery, Stockholm Söder Hospital.

Her medical history revealed an onset of UP in 1981. The skin lesions had progressed since then. Progression of the skin lesions was noted at the follow-up in 2003; lesions affected the trunk, face, arms, and legs (but not the soles and palms) (Figures 1 and 2). The lesions, reddish brown maculae appearing as scattered single or confluent efflorescences, were symmetrically located. When rubbed the lesions exhibited a characteristic urticarial response. The main symptom of these lesions was itching. A histopathologic examination of a skin biopsy specimen shortly after the start of symptoms confirmed the diagnosis. In 1989 and 1991 the patient was treated symptomatically with psoralen and ultraviolet A.

In March 2002 the patient had a short period of meteorism, belching, and melena after ingesting 1,000 mg of acetylsalicylic acid and 100 mg of caffeine. Results of investigation of the gastrointestinal tract with rectoscopy and radiology of the colon were normal. Gastroscopy showed a superficial gastropathy in the corpus and antrum, petechial lesions of the mucosa of the duodenum, but no ulcers. A biopsy specimen from the pylorus showed a normal amount of mast cells.

At a follow-up visit in May 2003, the patient presented with an almost generalized UP of the skin and

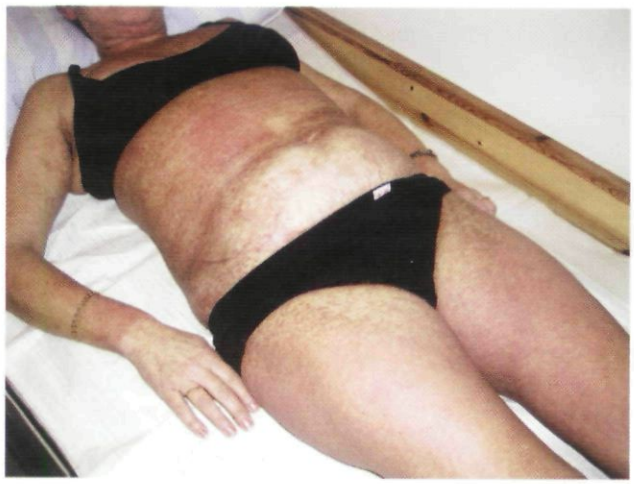

Figure 1 Multiple red-brown macules of urticaria pigmentosa (mastocytosis) seen in the patient at the follow-up examination in 2003.

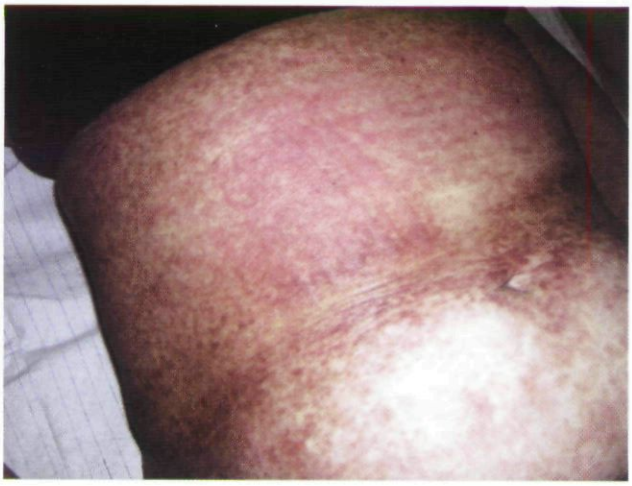

Figure 2 Urticated papules induced by rubbing (Darier's sign) in the patient with urticaria pigmentosa at the follow-up examination in 2003.

no sign of hepatosplenomegaly or lymphadenopathy at the physical examination. Laboratory investigations gave the following values: erythrocyte sedimentation rate, 4 mm/Hg; hemoglobin, 152 g/L; white blood cell count, 17.3 $\times$ 10^9/L (8.3% neutrophils, 5.8% lymphocytes, 2.2% eosinophils, 1% monocytes, and 0.1% basophils). Serum concentrations of albumin, potassium, and sodium were normal, as were liver and renal function test results. Excretion of the main metabolite of histamine, methylimidazoleacetic acid (MeImAA), in the urine was increased to 23.9 mmol per mole of creatinine (reference value, 0.4–2.4 mmol of creatinine).

A crest bone marrow specimen showed an increased number of eosinophils and a few focal accumulations of mast cells. Measurement of bone mineral density by dual energy x-ray absorptiometry showed an increased bone density (hip: 1.44 g/cm^2, *t*-value 3.7; lumbar spine: 1.45 g/cm^2, *t*-value 1.6; total body: 1.25 g/cm^2, *t*-value 1.6). (*t*-Value expresses the number of standard deviation differences from healthy adult controls.)

The patient had been smoking for many years, and her current consumption was five cigarettes daily. Her only medication at that time was substitution therapy with estrogen.

The patient had previously lost several teeth because of periodontitis in both jaws. The remaining teeth in the upper jaw were the first right central incisor to the first left premolar. In the lower jaw the cuspid and first premolar on both sides were connected by a metal-ceramic bridge.

Bone Grafting Procedure and Oral Rehabilitation

On the basis of preoperative clinical and radiologic findings of <5 mm residual bone under the maxillary sinuses, a sinus lift bone grafting procedure[7] with particulate corticocancellous bone from the iliac crest was performed in September 1999. Four months later a test microimplant was installed; it was taken out after an additional 4 months following the treatment plan of the dental implant procedure.[8,9]

The procedure was approved by the regional ethical committee. The patient was informed and gave her written consent.

Iliac-Crest Bone Graft Harvesting and Anesthetic Procedure

Harvesting of a corticocancellous iliac-crest bone graft was performed with the patient under general anesthesia. An incision was made through a rather thick dermal and subdermal layer of tissue, down through muscle to the periosteum. The bone graft was taken from the inner table of the right iliac crest with a reciprocating saw. Noticeably hard sclerotic bone (especially in the cancellous part) was taken out as a block. Hemostasis at the donor site area was achieved with bovine collagen (Lyostypt®, B. Braun Surgical GmbH, Melsungen, Germany). The incision was closed in layers, with resorbable sutures. An activated drain was used postoperatively, through the following day.

The bone block was milled twice with a bone mill (Leibinger®, Howmedica Leibinger GmbH & Co. KG, Freiburg, Germany) into small particles of approximately 2 $\times$ 2 mm.

Maxillary Sinus Augmentation Procedure

Following a crest incision a posterior mucoperiosteal flap was raised bilaterally in the edentulous parts of the maxilla and was reflected laterally, exposing the lateral walls of the maxillary sinuses. A thin rectangular osteotomy of about 1.0 $\times$ 1.5 cm (made with a round bur, almost completely through the bone) was outlined in the lateral wall of the maxillary sinuses. The thin bone piece was carefully fractured, loosened, and then pushed into the sinus, still attached to the schneiderian membrane. The membrane was freed carefully

and elevated from the bottom of the maxillary sinus bilaterally to avoid rupturing the thin membrane.

The particulate bone graft was mixed with autologous platelet gel derived from platelet-rich plasma (PRP).[10,11] To initiate clot formation, the following ratio of components was used: 4 mL of PRP mixed with 1 mL of autologous thrombin in a 10 mL syringe, with 1 mL of air for mixing the components in the syringe.[10,11] After approximately 2 minutes the syringe contained a gel that was mixed together with the bone.

The particulate bone graft and platelet gel mix was packed bilaterally into the prepared subsinus membrane spaces. Additional gel mixes were added as the spaces were filled with about 2.5 cc of bone. The incisions were closed with resorbable sutures.

The patient received intravenous penicillin (Bensylpenicillin®, Astra, Södertälje, Sweden) perioperatively and received phenoxymethyl penicillin (Kåvepenin®, AstraZeneca, Södertälje, Sweden) and metronidazole (Flagyl®, Aventis Pharma, Stockholm, Sweden) orally for 10 days post operation.

The patient was then monitored weekly during the first month and thereafter every month until fixture installation. The patient recovered well after surgery and had no extraordinary problems from the donor site. There were no complications of anesthesia during the operation.

Dental Implants

Four months after grafting, dental implants were installed. In the right upper jaw, four TiOblast™ (Astra Tech AB, Mölndal, Sweden) 3.5 mm implants (two 13 mm in length and two 11 mm in length) were installed; in the left upper jaw two implants (15 mm in length) of the same type were installed. Two trephine biopsy specimens (3 mm) were taken from the right side; at the same time, a 2 × 5 mm test microimplant (TiOblast surface) was installed in a horizontal direction in the grafted bone area (Figure 3). Healing of the implants was uneventful. Abutments were installed after 4 months and 1 week; at the same time, the microimplant was taken out with a 3 mm–wide trephine. The microimplant with the attached bone sample was sent to an oral pathologist for histologic examination.

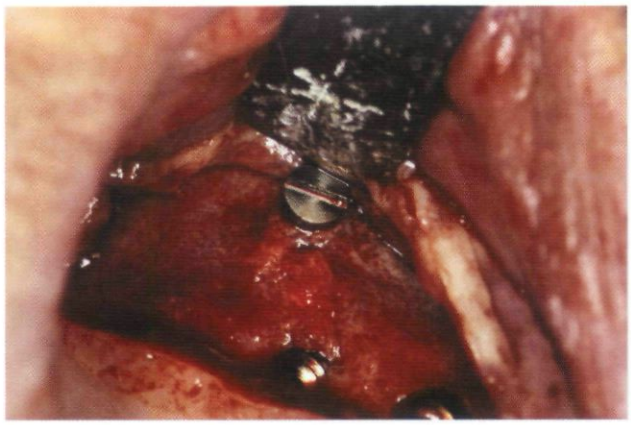

Figure 3 Test microimplant in the area of the sinus bone graft.

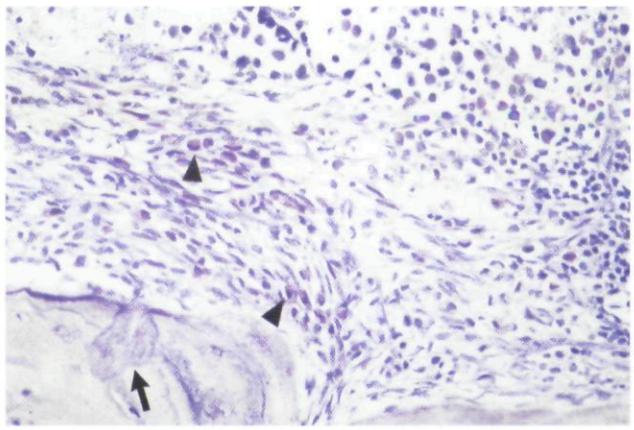

Figure 4 Biopsy specimen from the iliac crest at the time of bone grafting shows mast-cell infiltrates (*arrowheads*) in the marrow spaces of the corticocancellous bone at the site of the bone graft sample. Also note signs of ongoing bone remodeling (*arrow*) (×100 original magnification; stained with Giemsa).

The macroscopic appearance of the bone was normal at the time of implant installation as well as at abutment connection 6 months later. The bone quality was very hard upon preparation as well as when the two samples were taken.

The prosthetic reconstruction was done in a standard fashion, with metal-ceramic restorations.

Histologic Examinations

Biopsy Specimens from the Bone Graft and the Remodeled Bone Graft in the Maxillary Sinus Lift Region. The samples from the donor bone graft and the healed site of the bone-grafted area at the time of implant installation were fixed by formalin, decalcified, and embedded in paraffin. Sections were 3 to 4 μm thick and were stained with hematoxylin-eosin stain.

Evaluation of the bone graft sample from the iliac crest by light microscopy showed dense masses of infiltrating mast cells in the marrow spaces. The trabecular bone showed evidence of remodeling activity with sclerosis (Figure 4).

The previous findings of mast cells could not be seen in the sample taken from the healed bone graft in the sinus lift region. Marrow spaces were fibrotic or slightly edematous, with only a few inflammatory cells. Bony trabeculae were similar to normal remodeled grafted bone; there was nonviable bone with empty osteocyte lacunae, and there was also evidence of newly formed bone (Figure 5).

Biopsy Specimen with Microimplant. The microimplant and surrounding tissues were removed en bloc with a trephine bur, immersed in 4% neutral buffered formaldehyde, and processed to be embedded in light-curing resin (Technovit® 7200 VLC, Heraeus Kulzer GmbH & Co, Wehrheim, Germany). Undecalcified cut and ground sections were prepared with the EXAKT® sawing machine and grinding equipment (EXAKT Apparatebau GmbH & Co., Norderstedt, Germany).[12,13] At a thickness of 10 to 15 μm, the sections were stained with toluidine blue mixed with pyronin G.

By light microscopy the survey picture of the cut and ground sections revealed the microimplant surrounded by cancellous bone (Figure 6). Bone trabeculae could be observed outside the threaded area of the implant, and the threads were partly filled with bone as the bone tissue appeared to grow along the microimplant surface.

At higher magnification mostly newly formed bone was observed within the threads of the microimplant. However, areas of older bone were also visible (Figure 7). The bone appeared to be similar to the normal bone of other patients whose grafts were performed with the same technique. Demarcation lines (cement lines) could be observed between the old bone and the young bone, both in the threaded region and around the implant. In general, few areas of ongoing bone formation were observed; however, there was no marked resorptive activity either. Inflammatory cells (mainly lymphocytes) were observed in the marrow compartment between the bone trabeculae.

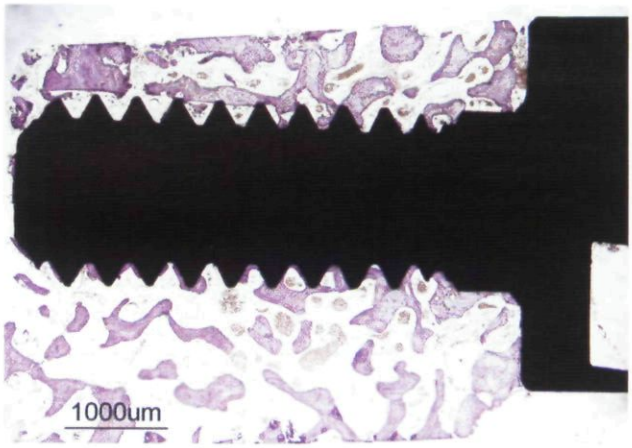

Figure 6 An overview of the biopsy specimen with the microimplant (13 weeks after installation) and surrounding bone (×1.6 original magnification).

Microimplant. The percentages of bone-to-metal contact and bone area in all threads and in the three best consecutive threads were calculated. "Mirror image" measurements of the percentage of bone in the

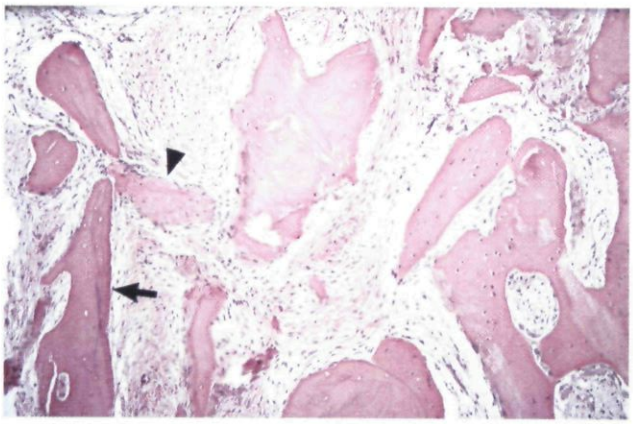

Figure 5 Biopsy specimen of a healed graft in the sinus lift region 13 weeks after grafting. Islands and spicules of grafted bone show reversal lines, indicating remodeling associated with bone with empty osteocyte lacunae (*arrow*) as well as evidence of ongoing bone formation (*arrowhead*). Fibrous tissue in marrow spaces shows little inflammation (×25 original magnification; stained with hematoxylin-eosin).

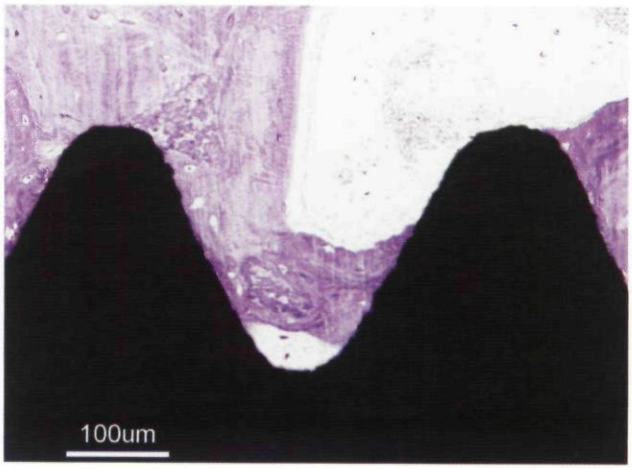

Figure 7 Bone in close contact with the titanium microimplant. Both older and more newly formed bone can be observed (×10 original magnification).

outfolded thread area were taken for the three best consecutive threads.[14] An objective of ×10 magnification and a zoom lens of ×2.5 magnification were used for these measurements. The percentage of total bone in a region of interest around the entire implant was measured with an objective of ×1.6 magnification.

Percentages of new bone and old bone in a region of interest at three selected positions around the implant were measured at all implants. A mean value for the entire implant was calculated; this measurement was performed with an objective of ×4.0 magnification.[15]

The quantitative evaluation of bone formation around the microimplant indicated a total of 28% bone in a region of interest around the implant; 21% was newly formed bone, and 20% consisted of older bone. The bone-to-metal contact was 40%, and the bone area within the threads was 41%, as compared to a bone area of 39% within the outfolded "mirror image" thread area.

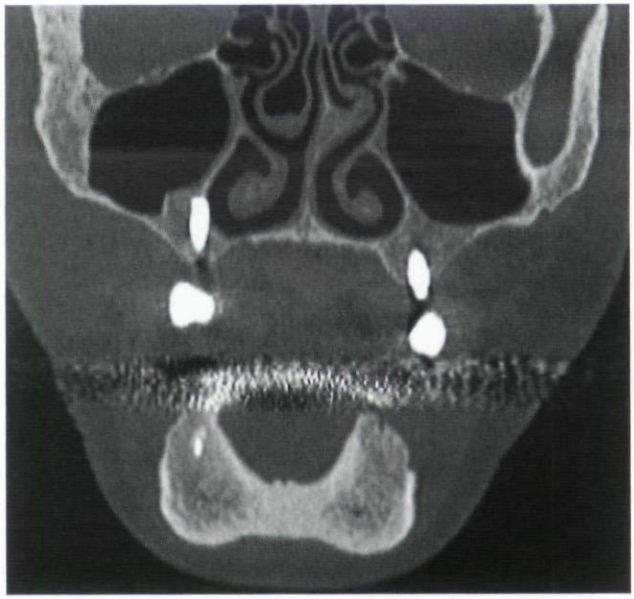

Figure 9 Computed tomography scan showing normal pneumatization in the maxillary sinuses at the 3-year follow-up examination.

RESULTS

A 3-year follow-up examination in 2003 showed a stable oral situation with a well-healed bone graft and integrated implants. The patient was then smoking 10 cigarettes per day.

The marginal bone level of the four dental implants was measured on intraoral dental films of the left side in the area where biopsy specimens had been taken. The level of the bone was seen at the implants from the third thread of the implant in one implant, from the second thread in two implants, from the first thread in one implant, and superior to the threads in two implants. CT of the jaws revealed the healed transplanted bone in the inferior part of both maxillary sinuses. CT showed sclerotic lesions in the remodeled maxillary sinus bone that were in a pattern similar to that of the adjacent facial bone. The bones of the skull and the jaws were described radiologically as being more sclerotic than normal and as having smaller marrow spaces than those normally seen (Figure 8). Normal pneumatization of both maxillary sinuses was also noted (Figure 9).

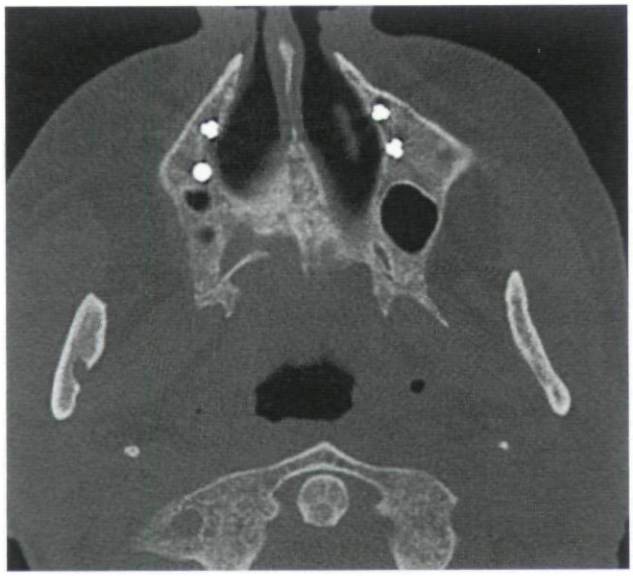

Figure 8 Radiographic appearance of bone with more sclerosis and showing smaller marrow spaces than normal at the 3-year control.

DISCUSSION

The clinical signs of systemic mastocytosis in this case were pruritic red-brown pigmented skin lesions, an increased number of mast cells in the skin and bone biopsy specimens, increased mineral bone density, osteosclerosis, focal accumulation of mast cells in the bone marrow, and increased excretion of MeImAA, the main metabolite of histamine. An increased excretion of MeImAA has been found to positively correlate to an increased number of mast cells in the bone marrow[16] and could also be an indicator of osteosclerosis in mastocytosis.[17]

The patient described in this article is a smoker, which could be one factor causing her periodontal disease. However, mast cells play a critical role in the induction of inflammation in oral mucosa and in dental pulp.[6] The release of cytokines and proteases from mast cells has been shown to promote gingival periapical inflammation and pulpitis.[6] It would therefore be tempting to speculate that the increased number of mast cells in this patient also could affect the process of periodontitis, resulting in a more severe course of events.

We have demonstrated that normal healing of a bone graft and integration of titanium implants are possible even if the bone grafted is affected by dense numbers of mast cells and shows sclerotic lesions. The use of the test microimplant in the two-stage implant sequence offers a well-functioning method of studying the integration of implants and the remodeling of bone.[18] The method offers a possibility of studying the integration of a test microimplant in a patient with a pathologic bone condition where implant treatment is considered. In the present case the amount of bone-implant contact was 20% higher when compared to a mean value from nine other normal patients treated by the same technique.[15] Bone-implant contact and remodeling of the graft are two examples of what can be studied with the test-microimplant technique. However, the result with this test microimplant cannot predict how dental implants will manage in the future when loaded by a prosthodontic construction.

The literature includes reports of several drugs (eg, codeine, acetylsalicylic acid, morphine, dextran, and muscle relaxants) that may act as specific mediator-releasing agents in mastocytosis triggering for severe systemic reactions and are thus hazardous in the operative management of mastocytosis patients.[1,19] Several case reports discuss this fact, and it is obvious that special care has to be taken in managing these patients during anesthesia and surgical interventions.[20–23] Mastocytosis should be considered in cases with recurrent anaphylaxis showing no other obvious etiology.[24]

There was no sign of adverse reactions to the general anesthetics (nitrous oxide, sevoflurane, ephedrine, fentanyl), the analgesic (rocuronium bromide), and the local anesthetic (prilocaine) used during the operations in this case. A case report by Auvray and colleagues showed that remifentanil and sevoflurane could be used for general anesthesia in patients with mastocytosis.[25] A report by Nelson and Savelli-Castillo discusses the measures to be taken in the dental treatment of a pediatric mastocytosis patient, including keeping the risk of life-threatening anaphylaxis from mast-cell degranulation in mind and avoiding histamine-releasing medications.[26]

CONCLUSIONS

Bone grafting and endosteal implant treatment are possible in patients with systemic mastocytosis and periodontal disease. The use of pathologic bone as a source of bone graft did not seem to interfere with the remodeling of bone and the installation of dental implants in this case. However, further studies with a larger population and extended follow-up are needed. It is tempting to speculate that the increased release of cytokines and proteases from the increased number of mast cells in mastocytosis may play a role in inflammatory conditions in the oral tissues (ie, periodontitis).

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