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Review

Gaucher Disease and Dental Approaches

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ABSTRACT

Gaucher disease (GD) is a lysosomal storage disease that results in glucocerebroside accumulation in the lysosomes due to deficiency of the enzyme glucocerebrosidase (GBA). GD, inherited as an autosomal recessive disorder, is a panethnic disease. However, it is most common in Ashkenazi Jews with a rate of 1/13. GD typically presents organomegaly and multiple organ involvement, in which the bone marrow is infiltrated by lipid-laden macrophages. Three clinical types have been identified based on whether neurological involvement of GD is observed. Type I GD is non-neuronopathic and the most common clinical type. The diagnosis of GD is determined by detecting low levels of GBA enzyme in peripheral blood leukocytes. Mutation analysis should be performed in patients with low enzyme levels. Specific treatment of GD includes enzyme replacement therapy (ERT) as a first step and substrate reduction therapy that can be tried in patients who cannot tolerate ERT. Bisphosphonates can be used as supportive therapy in patients with osteoporosis when there is no response to specific therapy. It is extremely important for dentists to be familiar with the maxillofacial abnormalities such as generalized osteopenia, enlarged bone marrow spaces, pseudocystic lesions, cortical thinning, and mental demineralization observed in the patients with GD, as well as multiple organ involvement, and hematological and skeletal involvement of GD. The aim of this review is to comprehensively point out the general involvement of GD and to illuminate the dentomaxillofacial findings of this disease, leading dentists on possible oral and dental complications that may develop in dental and surgical procedures of the patients with GD.

Keywords: Gaucher disease, maxillofacial abnormalities, dental approaches

INTRODUCTION

Gaucher disease (GD) is a lysosomal storage disease that results in glucocerebroside accumulation in the lysosomes due to deficiency of the enzyme glucocerebrosidase (GBA).[1] Lipid-laden macrophages, called Gaucher cells, present a typical morphology as large size cells with eccentric nucleus, condensed chromatin, and heterogeneous cytoplasm under the light microscope. The fine lines created by the accumulated substances in the cytoplasm are called "crumpled tissue paper appearance." Although these cells can be seen wherever macrophages are normally found, they are mostly found in the liver, spleen, bone marrow, and lymph nodes.[2]

GD was first described in 1882 by the French physician Phillippe Ernest Gaucher.[3] The disease was named "Gaucher Disease" by N. E. Brill, the medical doctor who made the first premortem diagnosis of the patient with this pathology in 1905.[4] GD is an autosomal recessive inherited disease that can be seen in many ethnicities. It is seen as one in 40,00-50,000 live births. The most common form is Type I without neurological involvement. It constitutes approximately 90% of the patients. It is common in Ashkenazi Jews, and the carrier rate is known as 1/13.[5] Although there is no study about the prevalence of GD in Turkey, it is estimated that it is above the general average due to the prevalence of consanguineous marriage. Neuronopathic GD (Type II and Type III GD) is less common. It is known that less than 1% of 1,698 patients reported to the Gaucher registry unit, which has the largest Gaucher patient database in the world, were Type II GD and 5% were Type III GD.[6]

The most effective and reliable way to diagnose GD is to measure the level of GBA enzyme in peripheral blood leukocytes. Although there is usually 10-15% enzyme activity in Type I GD patients, there is almost no enzyme activity in Types II and III GD patients. Those whose enzyme level is lower than 4.2 IU mL^{-1} are considered GD. It is thought that people with an enzyme level greater than 10 IU mL^{-1} do not have GD. Individuals in the intermediate group may have difficulty in diagnosis. Besides, genetic tests can be used in the differential diagnosis of groups with an enzyme level between 4.2 and 10 IU mL^{-1}. The diagnosis is confirmed in patients with biallelic pathological mutations. The presence of Gaucher cells in bone marrow

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biopsy is a key finding for diagnosis but may not be necessary for most patients who have characteristic clinical and biochemical features.[7] Mutation analysis can also be useful in terms of giving genetic counseling to the family of the affected person and family screening, as well as obtaining information about the phenotypic characteristics for specific cases whose mutations have been studied and determining the severity of the disease.

The purpose of the treatment of GD is to reduce the symptoms and complications of the disease and to increase the quality of life. It is aimed to improve organomegaly and symptomatic cytopenia in visceral and hematological involvements, to prevent bone pain, bone crises, and osteonecrosis in patients with skeletal involvement, and to increase the mineral density of trabecular bone. Specific treatment of GD includes enzyme replacement therapy (ERT) as a first step and substrate reduction therapy (SRT) that can be tried in patients who cannot tolerate ERT. Bisphosphonates can be used as supportive therapy in patients with osteoporosis when there is no response to specific therapy.[7]

The purpose of this review is to discuss the general involvement of GD comprehensively and to guide dentists about the issues that should be taken into consideration in the dental treatments of GD patients by shedding light on the dentomaxillofacial findings of this disease.

CLINICAL AND RESEARCH CONSEQUENCES

Clinical Sub-Types of Gaucher Disease

Three clinical types of GD have been defined according to the presence of neurological findings and the progression rate of these neurological findings.

Type I Gaucher Disease

Type I GD is the most common clinical type, which neurological findings are not observed. The clinical manifestation can vary significantly. The disease is classically characterized by splenomegaly as a result of glucosylceramide accumulation in splenic macrophages. Apart from the spleen, lipid-rich macrophages called "Gaucher cells" can be observed in the liver and bone marrow. For this reason, damage in GD is mostly observed in organs that are rich in macrophages.[8] Cytopenia, splenomegaly, and hepatomegaly are common in patients due to spleen, liver, and bone marrow involvement. In addition, orthopedic complications and other hematological changes can be observed.

Although it is generally thought that late-onset Type I GD has a slow course and shows little progression in the long term, recent studies have shown that this group of patients may have significant visceral, hematologic, and skeletal system disorders in the long-term follow-up, and also, late-onset patients may have an increased risk of malignancy.[9] According to the Gaucher report data of the International Gaucher Group (ICGG: International Collaborate Gaucher Group), the life expectancy in late-onset Type 1 Gaucher patients was found to be decreased by approximately 8.9 years compared to the population.

Type II Gaucher Disease

Type II GD is the clinical type characterized by the onset of symptoms in early infancy and the rapid progression of neurological symptoms. Typically, patients die within the first 3 years of life. There are hepatosplenomegaly, anemia, thrombocytopenia, and significant brainstem involvement findings. Pyramidal involvement and cognitive disorders may accompany. Brainstem manifestations include dysphagia, stridor pyramidal signs, spasticity, retroflection of the neck, trismus, and opisthotonus.[10]

Type III Gaucher Disease

Type III GD is called the chronic progressive neuronopathic type and may present in infancy, childhood, adolescence, or adulthood. Type III GD exhibits a more heterogeneous clinic than Type II. This clinical type was previously thought to be limited to people living in the Norbotten and Vasterbotten regions of Northern Sweden.[11] However, it was later understood that it was more common than thought and was seen in all ethnic groups.

Tissue and Organ Systems Affected by Gaucher Disease

GD is a disease with a wide clinical variety. Symptoms of the disease may differ even in patients with the same genetic mutation and enzyme level. Symptoms of the disease usually occur due to local effects of accumulated substances, organomegaly, and organ dysfunction. The main symptoms of the disease are hepatosplenomegaly, osteopenia, bone pain, cytopenias, bleeding, fatigue, easy bruising, and oculomotor apraxia. Table 1 reveals the clinical conditions and symptoms frequently detected in GD subtypes. Splenomegaly is seen in approximately 90-95% of GD patients. It plays a key role in diagnosis. Hepatomegaly is seen in approximately 75-80% of all patients. This rate is higher in patients who have undergone splenectomy.[6]

The majority of patients have clinical or radiological bone involvement. These involvements can vary from asymptomatic osteopenia to osteonecrosis. There may be acute and chronic bone pain, pathological fractures, and degenerative disorders due to subchondral resorption. Some patients may have painful bone crises, usually involving a single limb or joint. During a

Main Points

- The main symptoms of Gaucher disease are hepatosplenomegaly, osteopenia, bone pain, and cytopenias.
- Radiographic findings of dentomaxillofacial involvement in Gaucher patients include generalized rarefaction, loss of trabecular structure, enlarged marrow spaces, cortical thinning, osteosclerosis, pseudocystic radiolucent lesions, and loss of the anatomical structures.
- The radiographic appearance of the jawbones in Gaucher disease may be confused with other jaw lesions, and incorrect surgical procedures may be applied.
- The increase in osteopenia, which is present in almost all Gaucher patients, may cause pathological fractures and osteomyelitis in the jaws.

Table 1. Clinical Sub-Types and Symptoms of Gaucher Disease

Sub-Types	Primary CNS Involvement	Bone Disease	Others
Type I GD	Absent	Present	• Splenomegaly • Hepatomegaly • Cytopenia • Pulmonary disease
Type II GD (acute or infantile)	• Bulbar signs • Pyramidal signs • Cognitive impairment	Absent	• Splenomegaly • Hepatomegaly • Cytopenia • Pulmonary disease • Dermatological changes
Type III GD (subacute, juvenile)	• Oculomotor apraxia • Attack • Progressive myoclonic epilepsy	Present	• Splenomegaly • Hepatomegaly • Cytopenia • Pulmonary disease

GD, Gaucher disease; CNS, central nervous system.

bone crisis, there may be tenderness, heat increase, and edema in the joint. Fever and leukocytosis are also observed. Clinical, radiological, and laboratory findings can be confused as osteomyelitis or septic arthritis. Therefore, unnecessary surgeries can be performed.[12]

Among the main findings of hematological involvement of GD, anemia, thrombocytopenia, neutropenia, and coagulopathy are the most common findings.[13] Hematological involvement usually manifests itself with symptoms such as weakness, fatigue, epistaxis, and easy bruising. Some patients may present with anemia severe enough to be transfusion dependent. Leukopenia is less common. In GD, thrombocytopenia is prominent in the early period, but pancytopenia can be seen in progressive cases.[12]

Dentomaxillofacial Findings of Gaucher Disease

The underlying pathology of bone involvement in GD is associated with the accumulation of Gaucher cells that infiltrate the bone marrow compartment and directly/indirectly lead to local bone defects including cortical thinning, osteonecrosis, and lytic lesions.[14] Maxillofacial bone involvement is less commonly seen, while long bone involvement is common in GD. However, the mandible is considered to be a long bone that, unlike the maxilla, appears to be potential reservoir areas for Gaucher cells. Therefore, the mandible is also likely to be a focus of bone involvement.[15] Jawbone involvement is generally asymptomatic[15,16] and is detected as incidental findings in routine dental radiographs.[17] These radiographic findings include generalized rarefaction of the bone (osteopenia), loss of trabecular structure, enlarged marrow spaces, cortical thinning, osteosclerosis, pseudocystic radiolucent lesions, demineralization of the mental region, thinning of the lamina dura, displacement of the mandibular canal, and the root resorption in the teeth adjacent to the lesions.[18–21] There are only a few case reports reporting maxillary sinus involvement.[20,22,23] Several case reports have also reported that the mandible is a nidus of Gaucher cell infiltration and/or bone crisis.[22,24–27] In Table 2, studies reporting maxillofacial involvement of GD published from 1938 to 2020 are summarized.[28–34]

Dentist's Approach in Gaucher Disease

There are studies in the literature reporting that bone regeneration is observed after tooth extraction in patients with GD, and normal trabecular appearance is retrieved.[20,35,36] Besides, it is very important for dentists to know the jaw bone changes such as generalized osteopenia, enlarged marrow cavities, pseudocystic lesions, cortical thinning, and mental demineralization. These findings are of critical clinical importance in surgical procedures such as implant placement, bone graft removal, and orthognathic surgery.[37] Also, the increase in osteopenia, which is present in almost all GD patients, is associated with an increased risk of bone fractures in both adult and pediatric patients.[38] A soft diet should be recommended to patients to avoid the possibility of pathological fractures in the jaws.

In GD, osteomyelitis may occur in the mandible as a result of trabecular bone loss in the jaws, and owing to GD, the susceptibility of the bone to infection may increase.[39] The importance of oral hygiene to prevent odontogenic infections and secondary osteomyelitis should be emphasized if any findings related to GD in the jawbones are detected on panoramic radiographs or cone-beam computed tomography images. Clinical and radiological examinations of patients in terms of mandibular involvement are recommended to be performed periodically.[40,41]

Diffuse sclerotic radiopaque appearance seen in patients with GD may resemble Paget's disease or fibrous dysplasia.[20] Radiographic appearance may be confused for plasma cell myeloma or cancer metastasis, especially when accompanied by acute bone pain.[35] According to the radiological features associated with jaw lesions, the differential diagnosis of gnathic changes in GD is considerably extensive and includes bone marrow defects,[26] thalassemia, and sickle cell anemia[20,35] for generalized osteopenia. The "dome phenomenon" due to

Table 2. Compilation of Published Studies and Case Reports on the Dentomaxillofacial Manifestations of GD

Author, Year	Age/ Gender	Region	RG	Findings	Treatment
Bender, 1938[24]	13/F	md and mx	Periapical and extraoral X-ray	Cyst-like radiolucency, loss of trabecular structure in the premolar-molar region, generalized osteoporosis in the mandibular region	Extraction of the lower first molar tooth
Bender, 1959 (follow-up)[28]				Different RL areas—further loss of trabecular structure, endosteal bone degeneration and reduction in thickness, episodes of spontaneous bleeding in the hard palate region	Extraction of the lower first molar tooth
Bender et al., 1996 (follow-up)[35]				Significant improvement in radiolucency with regeneration of trabeculae	Uncomplicated extraction of posterior teeth, ERT since 1991
Moch et al., 1953[33]	39/F	md	Extraoral X-ray	Pseudocystic RL areas on both sides of the mandible, alveolar abscess of the right upper second premolar	Abscessed tooth extraction and curettage of apical area (penicillin + 500 cc complete blood)
Spiegel, 1957[34]	19/F	md and mx	Periapical and extraoral X-ray	Radiolucency in the premolar and molar regions, and loss of trabecular structure, osteolysis in the left maxillary premolar region, prolonged bleeding after minor surgeries	Removal of the tissue flap on the right lower third molar
Michanowicz et al., 1967[36]	21/M	md	Periapical X-ray	Small and large carious lesions in most mandibular and maxillary teeth, radiolucency in the right third molar and first molar, generalized osteoporosis, and cavities in the mandible	Extraction of right lower third molar, left lower first molar and left upper second molar teeth
Weigler et al., 1967[48]	28/M	md	Periapical X-ray	A large RL area from the first premolar to the third molar, enlargement of bone marrow spaces, loss of lamina dura at the lower-left second premolar and first molar root	Extraction and curettage of the lower left first molar
Bildman, 1972[17]	16/F	md	Periapical x-ray	Bilateral radiolucent areas in the body of the mandible	The extraction of the upper and lower first molars
Sela, 1972[25]	67/F	md	Autopsy examination	Histologically, tumor-like accumulation of Gaucher cells replaced by the bone marrow in the mandible, nodular reddish and yellowish tumor-like masses	-
Browne, 1977[46]	39/F	md	Periapical X-ray	Poor oral hygiene, yellow pigmentation of the oral mucosa, root resorption at the apical of the mandibular molars on radiographs, generalized trabeculation loss	Periodontal treatment and uncomplicated tooth extraction
Hall et al., 1985[39]	47/M	md	Panoramic X-ray	A large RL lesion with secondary infection in the left mandible	Debridement of the left mandibular corpus and ramus, lower second premolar, first and second molar extraction, IV and oral penicillin

Table 2. Compilation of Published Studies and Case Reports on the Dentomaxillofacial Manifestations of GD (Continued)

Author, Year	Age/ Gender	Region	RG	Findings	Treatment
Schwartz et al., 1988[22]	46/M	maxillary sinus and sphenoid sinus	Extraoral sinus X-ray and CT	Radiographic opacification of the maxillary sinus and sphenoid sinus, thinning of the posterolateral walls of the sinuses and mild enlargement of the maxillary antrum	Irrigation of the bilateral maxillary sinuses and medical treatment for the sinusitis-like condition
Heasman, 1991[19]	40/F	md	Periapical and panoramic X-ray	Hemorrhagic lesions on the face and lips; Bilateral RO areas seen in the premolar and molar regions of the mandible, generalized edematous gingivitis	-
Lustmann et al., 1991[26]	50/M	md and mx	Periapical and panoramic X-ray	Moderate periodontal disease and poor oral hygiene, four RL lesions characterized by reduced trabeculation and uncertain borders, two in the body of the mandible and two in the maxillary canine-premolar region, advanced apical resorption in canines, premolar and molars on both sides	-
Regenye et al., 1992[47]	23/M	mandibular trauma	Periapical, occlusal, panoramic X-ray and CT	There is mandibular fracture as a result of a motor vehicle accident, postoperative infection, no excessive bleeding during surgical procedures, a decrease in bone remodeling was detected	Stabilization of mandibular fractures, treatment of postoperative infection with clindamycin
Karabulut et al., 1997[23]	14/F	md, mx and sphenoid bones	Posteroanterior lateral radiography and CT	Diffuse osteopenia, trabecular loss in both mandible and maxilla, opacification of the sphenoid and maxillary sinuses due to widening of the medullary spaces	-
Carter et al., 1998[18]	28 patients	md and mx	Panoramic X-ray	Twenty-five of 28 patients have radiographic evidence of jaw involvement, the most common findings: enlargement of the bone marrow spaces, cyst-like lesions, cortical thinning, root resorption, displacement of the mandibular canal, and delayed permanent tooth eruption	-
Wasserstein et al., 1999[41]	12/F	md	Panoramic X-ray and CT	Large multilocular RL lesions extending to the lower cortical borders of the left and right mandible and invading between the teeth from above, the symmetric large RL lesion with ill-defined borders in the mandibular incisor region	A soft diet recommendation to reduce the risk of pathological fractures
Fischman et al., 2003[29]	87 patients and 31 carriers	-	Clinical examination only (DMFS index and GI)	Despite the prevalence of thrombocytopenia, there is no gingival bleeding, although there is radiological evidence of bone involvement, no tooth loss or mobility was found	-

Table 2. Compilation of Published Studies and Case Reports on the Dentomaxillofacial Manifestations of GD (Continued)

Author, Year	Age/ Gender	Region	RG	Findings	Treatment
Horwitz et al., 2007[20]	47/F	md	Periapical and panoramic X-ray	On clinical examination, petechiae, large plaque, and calculus deposits on the right buccal mucosa, acute generalized gingivitis, spontaneous bleeding and swelling, cauliflower-like papillae, bilateral cyst-like lesions on RG examination, severe root resorption in the premolar molar region, enlargement of the bone marrow cavities, loss of cortical margins of the mandibular canal	Detertrage and curettage, open flap surgery, extraction of three teeth on the right maxilla
Lisboa et al., 2011[32]	24/F	-	Periapical X-ray	History of excessive bleeding in previous extractions of the patient, deep carious lesion affecting the pulp of left maxillary first molar, and excessive crown destruction	Extraction of the left maxillary third molar, postoperative bleeding, pre- and postoperative prophylactic antibiotic and antifibrinolytic drug protocol due to the risk of infection
Givol et al., 2011[30]	Seven patients	-	-	A study investigating thrombocytopenia and bleeding in dental procedures, all patients were thrombocytopenic and coagulation tests were normal for most patients, the need for hematological replacement therapy during the procedures according to the total bleeding risk score and hematological (platelet count, dysfunction, coagulopathies, prolonged PT and PTT, and decreased fibrinogen level) risk score of the dental procedure	Detertrage, crown lengthening, surgical tooth extraction, root canal treatment, and cyst enucleation
Kumar et al., 2012[31]	47/M	mx	CT	Whitish necrotic bone not covered with mucoperiosteum from the right canine tooth to the third molar, bone destruction of the entire hard palate containing the right maxillary alveolus, and erosion of the right maxillary sinus floor	Surgical removal of necrotic bone including right-sided alveoli, maxillary sinus floor, and hard palate
Nobre et al., 2012[37]	Ten patients and 20 control	md and mx	Panoramic X-ray and CBCT	In CBCT images, mandibular involvement in all cases, both mandible and maxilla involvement in six, generalized rarefaction and enlarged marrow spaces in all patients, cortical thinning and osteosclerosis in five, pseudocystic RL lesions in nine, mental demineralization in seven, flattening of the condyle head in one, loss of the anatomical structures in eight, and thickening of the maxillary sinus mucosa in three patients, no pathological fracture, root resorption or delay in tooth eruption	-

Table 2. Compilation of Published Studies and Case Reports on the Dentomaxillofacial Manifestations of GD (Continued)

Author, Year	Age/ Gender	Region	RG	Findings	Treatment
Zeevi et al., 2013[16]	30/M	md	Panoramic X-ray and CT	Large, multilocular, well-defined asymptomatic bilateral RL lesion with scallop-like cortical margins in panoramic radiography, reduced trabecular structure, bone expansion and buccal bone perforation on axial CT images, loss of bilateral mandibular canal borders, tooth displacement, and extensive postoperative bleeding complication	Biopsy taken from the area of the lesion
	37/F	md and mx	Panoramic X-ray and CT	Rarefaction of bone trabeculae in the mandible and maxilla, asymptomatic unilocular RL lesion in the left posterior mandible, bilateral loss of mandibular canal margins and obliteration of maxillary sinuses, obliterated maxillary sinus on axial CT, enlarged bone marrow cavities in the maxilla, rarefaction of the mandibular bone and well-defined lesion of the left posterior mandible	Biopsy taken from the area of the lesion
Ahmadieh et al., 2014[21]	46/F	md	Periapical and panoramic X-ray and CT	Severe and persistent throbbing pain in the posterior left mandible, relatively well-circumscribed pseudocystic multilocular RL lesions in many regions of the mandible in the panoramic image, decreased trabeculation, effacement of the mandibular canal, and mild opacification of the maxillary sinus, a large multilocular lytic lesion and enlarged bone marrow cavities in the left mandible with the patient's complaint in CBCT images, thinning of periodontal ligament space with loss of the lamina dura in the affected molars	Use of oral bisphosphonates, a surgical biopsy to exclude other pathologies in the area of pain, surgical removal of pathological tissue
Mohamed et al., 2020[14]	42 pediatric patients (16 Type I GD and 26 Type III GD)	md	Panoramic X-ray	Delayed permanent tooth eruption in five patients in intraoral examination. The most common findings: thinning in the inferior cortex of the mandible, localized and generalized rarefaction, generalized rarefaction with a similar frequency in Type 1 and Type III GH, enlargement of the bone marrow spaces is more common in Type I GD, pseudocystic RL lesions, cortical thinning, anodontia, and dental anomalies are more common in Type III GD, no difference was found between dental age and chronological age	Bisphosphonate and enzyme replacement therapy

GD, Gaucher disease; RG, radiography; F, female; M, male; Md, mandible; Mx, maxilla; CT, computed tomography; CBCT, cone-beam computed tomography; RL, radiolucent; RO, radiopaque; PT, prothrombin time; PTT, partial thromboplastin time.

preprosthetic maxillary sinus augmentation may also resemble sinus devastation associated with GD.[16] Well-defined radiolucent osteolytic lesions, such as a keratocystic odontogenic tumor, central giant cell granuloma, aneurysmal bone cysts,[42] and even traumatic bone cysts,[16] which are thought to be more similar, should also be considered in the differential diagnosis. Gaucher lesions may also resemble alveolar bone loss associated with periodontal disease.[43] When GD is suspected, the least invasive method for diagnosis is reduced b-GBA enzyme activity in blood samples.[43] Jaw biopsy is not recommended unless another condition such as malignancy is suspected. To diagnose GD, an enzymatic evaluation is required, not a biopsy or a bone marrow sample.

Some studies have reported the use of bisphosphonates in osteopenia/osteonecrosis therapy as a supportive treatment for ERT in GD.[14,44] It has been reported that bisphosphonates preserve bone density by proving an antiresorptive effect; however, prolonged IV bisphosphonate treatment rarely causes osteonecrosis of the jaw that is triggered by exposed bone after tooth extraction and subsequently bacterial contamination.[45] Therefore, the use and duration of bisphosphonates should be investigated in Gaucher patients before tooth extraction or dental surgeries.

There is a possibility of complications like excessive bleeding due to thrombocytopenia during surgical procedures, especially in patients who do not undergo splenectomy.[35,46,47] Horwitz et al.[20] reported increased bleeding during tooth extraction and its local control in the case report they presented. However, in most of the other cases reported in the literature, it was stated that there was no abnormal bleeding, and the recovery was normal.[17,35,36,46–48] Nevertheless, in patients with GD, it is recommended to consult the patient's physician before invasive procedures and to check complete blood count including PT, PTT, bleeding time, and platelet count at the first examination. Local hemostasis should be applied according to the patient and procedure.

CONCLUSION

As a result, dental radiographs can play a role in the early diagnosis of GD, especially in the absence of clinical symptoms. The roadmap to be followed for the diagnosis of Gaucher lesions in the jawbones is a comprehensive medical history, and clinical and radiological examinations. Dentists should be familiar with the dentomaxillofacial findings of GD and be aware of possible oral and dental complications that may develop. Furthermore, when GD is suspected in undiagnosed patients, patients should be able to be referred to the necessary departments.

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