

Idiopathic gingival hyperplasia: A case report

Gingival hyperplasia may be described as an increase in the size of the gingiva (10). Idiopathic gingival hyperplasia may be observed as fibrotic hyperplasia of gingival tissue, which leads to esthetic and functional problems. Factors that lead to gingival hyperplasia include inflammatory causes, hormone-related causes, leukemia, vitamin C deficiency, non-specific causes, neoplasia and drugs (9,8). The drugs that may be responsible include anti-convulsive drugs, immune-suppressive drugs, anti-epileptic drugs and calcium channel blockers (5). Gingival hyperplasia with unknown causes, not included in any groups, may also be seen.

Case report

A 28-year-old female patient was admitted to the Oral – Maxillofacial Diseases and Surgery Clinic of Dentistry Faculty, Istanbul University on 15.02.2016. She was admitted to the Internal Medicine Outpatient Clinic with the complaints of gingival swelling and bleeding persisting for approximately 2 weeks and she was referred to our clinic as her routine blood tests and clinical examination findings were normal. Widespread hemorrhagic hyperplasia involving the free gingiva and almost all teeth both in the vestibular and lingual sides were observed on the intra-oral examination. The size of hyperplasia covered almost all of the vestibular surfaces of the teeth in the anterior, elevated up to the occlusal layer in the posterior side, particularly in the wisdom tooth. No abnormal findings were encountered on the radiological investigations. Gingival hyperplasia in the vestibular region of the right upper jaw was excised using diode laser one week later as her complete blood count was within the normal ranges. Prophylactic antibiotic (amoxicillin clavulanate) was prescribed and the treatment was supported with chlorhexidine gluconate. The pathological specimen was examined in the Pathology Department of Istanbul University with the pre-diagnosis of leukemia and reported as plasma cells on a background of ulcer and intense inflammatory infiltration rich with eosinophils, and hyperplasia in the neighboring mucosa (neoplastic cells were not seen). All inflammatory tissues were excised in 3 sessions (with laser and lancet surgery). However, the patient underwent bone marrow aspiration biopsy in order to exclude leukemia when she underwent the consultation of the Hematology Department and the result was found to be negative. She experienced concurrent influenza infection during her excisions and she was examined in the Pulmonology Department, and the computed tomography of the thorax performed with intravenous contrast medium injection was suggestive of lymphoma, metastasis, sarcoidosis, pleural tumor, and the biopsy was reported to be consistent with hypersensitivity pneumonia, although the findings supported benign inflammatory processes. The report included statements such as “The patient should be evaluated together with

clinical findings for hypersensitivity pneumonia, connective tissue disorders and drug toxicity under the light of histopathological findings". However, no diagnosis could be made according to the result of the clinical examinations. The patient was referred to the Rheumatology Clinic due to the presence of nodular lesions in the lung, and Wegener granulomatosis was suggested. However, it was excluded as p-ANCA was found to be negative. Minor salivary gland biopsy was obtained due to the pre-diagnosis of sarcoidosis and granuloma was not encountered. The Vitamin C level was analyzed as vitamin C deficiency may be a cause of gingival hyperplasia; however, it was found to be normal. Her fillings were removed due to the possibility of allergic reaction as the hyperplasia was reported to develop after fillings, and thereafter, the patch test was performed in the Allergy Outpatient Clinic of Department of Internal Medicine and the result was determined to be negative. Her intra-oral findings were completely resolved and no recurrence was observed.



Figure 1: Patient initial photo



Figure 2: Patient initial left side photo



Figure 3: After first operation of right side , 3 days later



Figure 4: Patient final photo (7 months later)



Figure 5: Patient final left side photo (7 months later)



figure 6: Patient final right side photo (7 months later)

Discussion

Gingival hyperplasia may lead to esthetic and functional losses (1). The pathogenesis may include systemic drug use, vitamin deficiency, neoplastic /non-neoplastic hematological disorders.

Omori et al. reported vitamin C deficiency and periodontal disease in their case report. While vitamin C deficiency may lead to irregular gingival hyperplasia and hemorrhagic fields as in our case, it is not

encountered frequently in modern communities today. The Vitamin C level was normal in our patient (7).

Widespread gingival hyperplasia and hemorrhage were seen together in a patient with acute myelodysplastic leukemia, which was reported by Güzeldemir et al. in 2006. Although these findings were seen together in our patient too, leukemia was excluded through blood tests and biopsies (4).

Baltacıoğlu et al. (2012) reported gingival hyperplasia in 3 patients who used cyclosporine and its treatment. However, our patient did not have the history of drug use causing gingival hyperplasia like immunosuppressive agents, anticonvulsants and calcium channel blockers (2).

Gingival hyperplasia was reported to completely cover the vestibular sides of the teeth in the anterior, deep sacs were observed in a patient with gingival fibromatosis reported by Güngör et al.. However, hemorrhage was not reported. Our patient had generalized hemorrhage, although similar gingival hyperplasia was observed and the biopsy result was not consistent with fibromatosis (3).

Nuyen et al. reported gingival metastasis in a patient with adeno-carcinoma of the lung in their case report in 2016. Although no metastasis –related gingival pain was felt, ulcer fields and hyperplasia were observed. However, our patient did not have a medical history or family history of malignancy (6).

Painless gingival hyperplasia lasting for one month in the posterosuperior region of the gingiva was reported in a 42-year-old male patient by Tripathi et al (2014). The patient was diagnosed with sarcoidosis and he was treated (11). Minor salivary gland biopsy was performed with the pre-diagnosis of sarcoidosis in our patient too. However, the diagnosis of sarcoidosis was excluded as granuloma was not encountered.

Conclusion

Gingival hyperplasia is among the most common gingival diseases. It is categorized according to its localization and size (1). Management and recurrence depend on its being able to be diagnosed correctly. Our patient was treated successfully, although a definite diagnosis could not be made and no recurrence was observed in the 7-month follow up. However, we could not make a definitive diagnosis unfortunately. All possible diagnoses were excluded through statements of the patient, tests and biopsies. However, we faced to a case which we had overlooked or not encountered before.

We will be glad if our valuable colleagues who have an opinion about the diagnosis provide feedback.

Sources:

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