

Case Report

Diagnosis Procedure In Case Of a Gingival Hyperplasia: Presentation of One Case

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Abstract: **Introduction:** Gingival hyperplasias are abnormal increases in gingival volume, due to exaggerated proliferation of its cells. There are a number of causes for this condition. The cause can be local but also be the expression of certain hormonal status, of general pathologies and/or their therapies. **Observation:** A 12 year old patient, suffering from anemia, consulted for a painful gingival overgrowth evolving for more than a year. The diagnosis procedure led us to an inflammatory gingival hyperplasia of infectious origin. **Discussion:** In the presence of gingival hyperplasias the case history and the clinical examination orientate the diagnosis. Further examinations permit to retain the cause and exclude other possible etiologies. This procedure can turn out difficult. **Conclusion:** Before the multiple etiologies possible for a gingival hyperplasia in a child, it is important to carry out complementary examinations in order to avoid bad surprises.

Keywords: Diagnostic approach, gingival hyperplasia, clinical case.

INTRODUCTION

Gingival hyperplasia is an overgrowth of gum tissue as a result of abnormal (excessive) proliferation of its cells. There are a number of causes for this condition. They can be inflammation causes, hormonal (puberty, pregnancy, hypothyroidism), systematic causes (vitamin deficiency), tumorous (benign tumour of the buccal mucosa, proliferative hemopathy) or be drug-induced as side effect of prescribed medications (calcium inhibitors, anticonvulsants, immunosuppressors) (Persson, R.E. *et al.*, 2003; Dongari-baqtzoglou, A. 2004; Laine, M.A. 2002).

In case of gingival hyperplasia in a child, the case history and the clinical examination give different diagnosis orientations. Further examinations permit, after exclusion of all other etiologies, to retain a diagnosis.

We present here by, the diagnosis procedure in case of a gingival hyperplasia.

Clinical Observation

A 12 year old patient addressed to the stomatology service of the general hospital of Grand Yoff at Dakar-Sénégal by his treating dental surgeon for painful gingival over swelling located at the mandibular at the mandibular. According to the patient, the lesion has been evolving for more than a year and had increased in size with time. The patient described major pain on teeth contact and/or during feeding, associated with weight loss. Six months earlier, she consulted her treating medical doctor for asthenia and anorexia. The biological examination revealed a severe anaemia, a blood transfusion was done after patient hospitalisation. No past medical history was retained.

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Figure 1: Right mandibular gingival hyperplasia.

At the exobuccal examination, we noted the presence of soft, submandibular bilateral adenopathies, mobiles, unpainful on palpation. Endobuccal clinical examination revealed the presence of a gingival over swelling going from the 31 to 47. The lesion with erythematous aspect occupied the vestibular and lingual borders of the mentioned teeth with partial colonization of the occlusal surfaces. The lesion was soft, bleeding on contact (**fig. 1**). The mucosa covering the buccal cavity was pale colored. The presence of tartar was noticed at the first and fourth quadrant, testifying of a preferential left unilateral mastication. At the end of the first consultation, several diagnosis hypotheses were formulated: inflmatory gingival hyperplasia, epulis, proliferative hemopathia. A radiological examination (orthopantomogram) and biological examinations (complete blood count, coagulation factors) were requested.

Table I : Results of blood count. Values followed by a * are anormal.

	Results	Unit	References
Erythrocytes	4,00	T/L	4,2 - 5,2
Haemoglobine	11,4*	G/dL	12 – 16
Plaquettes	497*	G/L	150 – 450
Procalcitonin	0,50*	mg/L	0,1 - 0,4
PN neutrophiles	2,63	G/L	4,2 - 5,2
PN basophiles	0,14*	G/L	< 0,10
Lymphocytes	2,35	G/L	1 – 4
Monocytes	0,71	G/L	0,2 – 1
VS	40*	mm.1h	<20



Figure 2: Initial Orthopantomogram

The orthopantomogram revealed a discreet print of the mower dental canal, a mesioversion of the 45 with a slight bone lysis between 45 and 46 (**fig. 2**). The complete blood count put in evidence an anaemia (11, 4 g/dl), a thrombocytosis (497 giga/L), a basophilia (0, 14 giga/L) and an increase in procalcitonine (0, 50%). The sédimentation speed was of 40 mm within him first hour (**Tab. I**).most of these tests was in favor of an infectious process. However the recurrent anaemia and the basophilia made us fear an atypical proliferative hemopathia. Before this clinical picture, a biopsy was realized during this consultation, this with objective the exclusion hypothesis of an atypical proliferative hemopathia. The histological examination of the biopsy revealed Malpighian epithelia presenting a slight acanthosis, keratotic without cytopathy nor atypical cytonuclear.



Figure 3: Healing of the gingiva six months later.

The clinical and complementary examinations permitted an exclusion of other hypothesis. The only plausible diagnosis amongst the ones evoked was that of inflammatory gingival hyperplasia of infectious origin. A medical prescription (amoxicilline + clavulanic acid 2g, 2x/day ; paracetamol 3g, 3x/day) and a mouthwash made of Chlorhexidine were given. A scaling and polishing followed of a resection of the hyperplasia with a cold bistoury blade; then a cautérisation with the electrical bistoury under local anaesthesia were realised on the dental chair. Control was done at day 7, day 14, day 30, day 60, day 120 and day 180 (**Fig. 3**). The patient was sent back to the haematolgy service for specified examinations and the handling of her anaemia.

DISCUSSION

We report here a case of inflammatory gingival hyperplasia lasting over a year on a young girl suffering from anaemia with no past history of medication known.

Clinically, inflammatory gingival hyperplasia presents an oedematous, shining red purplish soft gingival that easily bleeds. The voluminous gingival mostly covers and important part of the crowns, leading to an increase in the sulcus depth, favoring sticking of food debris and accumulation of bacteria (kinane, D.F. 2001).

Inflammatory gingival hyperplasia is mostly of local cause, provoked by bacterial toxins inducing biofilm through its enzymes and toxins inducing an inflammatory response of the gingiva. Nevertheless, certain cases of inflammatory gingival hyperplasia due to buccal respiration have been reported in literature (Laine, M.A. 2002; Kinane, D.F. 2001).

Histologically, gingival hyperplasia is composed of, an excess of fibrous connective tissue covered by a squamous stratified epithelium of normal thickness or hyperacanthotic (thickening of the mucosal body of malpighi) (Kaquelier JC et Le May O 1998).

The positive diagnosis must be done, excluding all other causes of gingival hyperplasia. In our case, epulis and proliferative hemopathy were cited as differentials.

Gingival epulis is amongst the gingival hyperplastic pseudo-tumours. The most frequent causes cited are traumatic and hormonal. We find them at the level of the neck of one or two contiguous teeth. It can be sessile pediculated or ligamented, staying classically on the incisivo-canin vestibular section (Ch. FAYE N'DIAYE, B. *et al.*, 1996; AKAZANE, A., & BADREDINE, H. (2014). In our case, gingival hyperplasia occupied the vestibular and lingual sectors going from 31 to 47.

Proliferative hemopathy are the most frequent malignant pathologies found in children less than 15 years old (Sepúlveda, E. *et al.*, 2012). In certain aspects, this case resembled a proliferative hemopathy:

- The first, frequently seen gingival hyperplasia that usually results from the infiltration of oral tissues by malignant cells ;
- The second is the presence of anaemia ;
- The third corresponds to the increase in the amount of polynuclear basophiles circulating which is observed in cases of proliferative hemopathy, chronic haemolytic anaemia (Sepúlveda, E. *et al.*, 2012; Rerhrhaye, M. *et al.*, 2010).

However, there was no unexplained dental pain at the clinical exam and the blood count returned without neutropenia, nor thrombopenia. Elements frequently seen in cases of proliferative hemopathy (Sepúlveda, E. *et al.*, 2012; Zhou, Y. *et al.*, 2011). Several stages of evolution and of gravity exist, from the most discreet to the most evident. Moreover, certain cases of atypia have been described in literature, making the diagnosis more difficult (Lafon, A. *et al.*, 2010). The coagulation factors came without any particular informations. We noted an increase in the

procalcitonin concentration. Procalcitonin is a prohormone which blood concentration can be measured specifically. Its increase in blood is revelatory of an inflammatory process, precisely bacterial, this, even in the absence of neutropenia (Sager, R. *et al.*, 2017).

Gingival hyperplasia is less frequent in children. Granulomatous diseases (Crohn's disease, Wegener granulomatosis), malignant tumours and hereditary must be taken into consideration in the differential diagnosis of gingival hyperplasia in children (Krishna, K. B. *et al.*, 2014; Olczak-Kowalczyk, D. *et al.*, 2011). In our case, the diagnosis retained after a synthesis of all collected elements was that of inflammatory gingival hyperplasia of infectious origin.

When the hyperplasia is minimal, a careful scaling of the teeth associated with a correct hygiene can be sufficient. For the important lesions, a rigorous surgical intervention is necessary (Cuest, A. *et al.*, 1998). In our case, the treatment associated a cleaning of the buccal cavity under antibiotic covering and a gingival resection under local anaesthesia.

Recidivism is the most common problem in the management of gingival hyperplasia. To avoid this recidivisms, it is recommended to program a gingival therapy of maintenance (Cuest, A. *et al.*, 1998). So we reviewed our patient for control sessions. Six months later the patient was doing great and the gingival mucosal looked stable.

CONCLUSION

The objective of this work was to demonstrate that for a gingival hyperplasia in a child we can have different diagnosis. Before the multiple etiologies possible gingival in a child, certain being able to involve the vital pronostic of the patient, it is important to do complementary examinations in order to avoid bad surprises.

Conflict of Interest

None.

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REFERENCES

1. Persson, R.E., Hollender, L.G., MacEntee, M.I., Wyatt, C.C., Kyak, H.A., & Persson, G.R. (2003). Assessment of periodontal conditioned and systemic disease in older subjects. *J Clin Periodontol*, 30(3), 207-213.
2. Dongari-baqtzoglou, A. (2004). Research, Science and Therapy Committee, American Academy of Periodontology. Drug-associated gingival enlargement. *J Periodontol*, 75(10), 1424-3.

3. Laine, M.A. (2002). Effect of pregnancy on periodontal in dental health. *Acta Odontol Scand*, 60 (5), 257-64.
4. Kinane, D.F. (2001). Marshall GJ, periodontal manifestations of systemic disease. *Aust Dent J*. 46 (1), 2-12.
5. Kaqueler JC et Le May O (1998). Bucco dental Anatomy and pathology.
6. Ch. FATY N'DIAYE, B., DIALLO, S., DIATINE, G., LARROQUE, H., & SZPIRGLAS. (1996). Institut De L'information Scientifique Et Technique. Titre périodique : AOS. Actualites Odonto-Stomatologiques, Les épulis au Sénégal : revue de 96 cas (1991-1994). N° 195, septembre. P475/481.
7. AKAZANE, A., & BADREDINE, H. (2014). Épulis à propos d'un cas. *Pan Afr Med J*, -19.
8. Sepúlveda, E., Brethauer, U., Fernández, E., Cortés, G., & Mardones, C. (2012). Oral manifestations as first clinical sign of acute myeloid leukemia: report of a case. *Pediatr Dent*, 34(5), 418-21.
9. Rerhrhaye, M., Abdellaoui, L., Bouziane, A., & Ennibi, O. (2010). Le bilan biologique en odontostomatologie: intérêt et interprétation. *Actualités Odonto-Stomatologiques*, (250), 117-135.
10. Zhou, Y., Qian, M., Liang, Y., Liu, Y., Yang, X., Jiang, T., & Wang, Y. (2011). Effects of leukemia inhibitory factor on proliferation and odontoblastic differentiation of human dental pulp cells. *Journal of endodontics*, 37(6), 819-824.
11. Lafon, A., Belangeon, T., Ahossi, V., Larras, P., & Perrin, D. (2010). Leucémie aiguë myéloïde : le tableau clinique est parfois trompeur. *Med Buccale Chir Buccale*, 16, 177-181.
12. Sager, R., Kutz, A., Mueller, B., & Schuetz, P. (2017). Procalcitonin-guided diagnosis and antibiotic stewardship revisited. *BMC medicine*, 15 (1), 15. Epub 2017/01/25.
13. Krishna, K. B., Raju, P. K., Chitturi, R. R., Smitha, G., Vijai, S., & Srinivas, B. V. V. (2014). Prevalence of gingival enlargement in Karnataka school going children. *Journal of international oral health: JIOH*, 6(1), 106..
14. Olczak-Kowalczyk, D., Krasuska-Sławińska, E., Rokicki, D., & Pronicki, M. (2011). Case report: Infantile systemic hyalinosis: a dental perspective. *European Archives of Paediatric Dentistry*, 12(4), 224-226.
15. Cuest, A., Carneo, R., & Bornancini, C. (1998). hereditary generalised gingival fibromatosis associated with hypertrichosis : Report of five cases in one family. *J oral Maxillo-fac Surg*, 46, 415-420.