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Oral squamous cell carcinoma in the paediatric patient: a literature review

ABSTRACT

Aim *Paediatric Oral Squamous Cell Carcinoma (OSCC) is rare, but its incidence is increasing, bringing forward the issue of the common pathogenic factors. The aim of this study is to verify the actual incidence of oral carcinoma reported in paediatric patients up to the age of 15 by thoroughly reviewing the available literature. Setting this cut-off age has allowed us to emphasise possible risk factors other than those always associated with the onset of this neoplasia, which are not present in this age bracket yet.*

Methods *In the first stage of the research, generic key words concerning OSCC in childhood were entered into two search engines. In the second stage, terms related to predisposing diseases connected to childhood oral carcinoma and those initially found were searched.*

Results *The literature review consisted of 55 documented cases from 1894 to 2011, of which 15 were part of complete published case reports.*

Conclusion *Paediatric OSCC, though uncommon, is not rare. The review has strongly highlighted the need to carry out an objective, thorough and standardised examination of the child's oral cavity, especially when systemic predisposing diseases, such as Epidermolysis bullosa, Xeroderma pigmentosum, Juvenile papillomatosis and Fanconi's anaemia, are present.*

Keywords *Oral Squamous Cell Carcinoma; Xeroderma pigmentosum; Squamous Cell Carcinoma; Epidermolysis bullosa; Papilloma virus.*

Introduction

Oral Squamous Cell Carcinoma (OSCC) is a very rare pathology in paediatric patients; since it is age-related its highest incidence is in male subjects in their sixties and seventies; in patients younger than 40 years it approximately accounts for 4% of all neoplastic pathologies; only in few cases this neoplasia occurs in paediatric subjects, i.e. younger than 18 years old; the percentage dramatically drops in the first decade of life [Llewellyn et al.,2001; Stolk-Liefferlink et al., 2008].

While carrying out our study we noticed that there is very little literature in this field; nevertheless, this pathology should not be underestimated as in various countries the incidence of OSCC has been increasing in younger segments of the population, bringing forward the issue of the chief usual risk factors in adulthood (tobacco and alcohol use, betel-nut chewing, etc.). In fact such a recrudescence would appear to be based upon a predisposition to genetic instability [Stolk-Liefferlink et al., 2008], but it is even more important to point up that when affecting paediatric patients OSCC is often associated with predisposing conditions, such as: Epidermolysis bullosa, Fanconi's anaemia, Xeroderma pigmentosum, Juvenile papillomatosis) [Scott Earl et al., 1988]. Besides, in underdeveloped countries the incidence of squamous cell carcinoma of the oropharynx and upper airways is high, and there have also been cases of this pathology affecting the same region and associated to Plummer-Vinson syndrome [Ndjyal et al., 2009]. On the other hand cases of OSCC not linked to any particular predisposing condition have been reported in industrialised countries [Socks et al., 1985].

OSCC in the paediatric age affects males and females almost with the same incidence and its onset in the oral cavity mainly occurs on the lips, tongue and gingivae.

OSCC in younger individuals is believed by researches to be distinct from the adult form as it often turns out to be difficult to diagnose and locally more aggressive [Llewellyn et al., 2001]. In literature it is highly recommended to include OSCC in the differential diagnosis of oral cavity lesions in the paediatric patient, in order to distinguish it from highly proliferative inflammatory lesions (e.g. Pseudoepitheliomatous Hyperplasia) [Ribeiro et al., 2011; Bill et al., 2001; Amichetti et al.,1989].

The aim of this study is to verify the actual incidence of oral carcinoma reported in paediatric patients up to the age of 15 by thoroughly reviewing the available literature. Setting this cut-off age has allowed us to emphasise possible risk factors other than those always associated with the onset of this neoplasia, which are not present in this age bracket yet.

Materials and methods

The bibliographical research was done by entering a

query into two search engines: PubMed and Google Scholar. The following search terms were typed in: Oral cancer in children, Oral squamous cell carcinoma in children, Oral SCC in children, Oral cancer in childhood, Oral squamous cell carcinoma in childhood, Oral SCC in childhood; they were entered one by one into PubMed and indexed for all databases. Then each result in the database was individually searched for along with each quotation connected with each article. The same search terms were entered in Google Scholar. The articles already found in PubMed were ruled out from the search in Google Scholar. At the first stage of our research we found a group of 34 articles which were analysed and provided further terms useful for our query: these key words were chiefly related to those predisposing pathologies linked to childhood oral carcinoma: "Cancer in children", "Head and neck cancer in children", "Papilloma virus and oral cancer in children", "Visceral cancer in children", "Squamous cell carcinoma in children", "Fanconi's anaemia and oral cancer in children", "Fanconi's anaemia in children", "Xeroderma pigmentosum in children", "Xeroderma pigmentosum and oral cavity in children", "Xeroderma pigmentosum and squamous cell carcinoma in children", "Epidermolysis bullosa in children, "Epidermolysis bullosa and oral cancer". The same procedure was performed during the following stage of our research; 18 articles were found. The outcomes of our bibliographical research were then processed by a spreadsheet and statistically analysed.

Results

Our literature review allowed us to find 55 documented cases from 1894 to 2011, of which 15 were part of complete published case reports (Tab. 1). The earliest case report in literature dated back to 1958 and dealt with a 13-year-old male patient affected by squamous cell carcinoma on the tongue, whose prognosis proved fatal after two years [Moore, 1958]. This article showed the data of a careful literature review on visceral carcinomas from 1871: only 12 cases were found and all restricted to the oral cavity. The latest articles always show, in addition to case reports, the updated review results [Sidell et al., 2009; Woo et al., 2009]. In an article dated February 2011, 55 cases of paediatric SCC were reported; 28 of these were male and 22 were female; the remaining 5 cases did not provide data about the patients' sex [Ribeiro et al., 2011] (Fig. 1). There was clear evidence of an age-related rise in the onset of this pathology; the outcome was supported by the calculation of the average age of the subjects, which was 9 years (standard deviation 2.83; standard error ±0.94) (Fig. 2). As to the data about relapse and survival this case showed a lack of exhaustive documentation (37 cases out of 55 were incomplete), a drawback common to most of the reports found. Yet, of the 18 well-documented cases 13 patients did not show sign of recurrence after 1 year, whereas 5 died two years after first diagnosis.

Discussion

If in adults in their 60s and 70s OSCC is the most common neoplasia of the head and neck region, it is less frequent in subjects younger than 40 and very rare during the paediatric age [Atula et al., 1996; Newman et al., 1983]. Notwithstanding this, an increase in its occurrence has been reported both in the most industrialised countries and in the less developed ones [Woo et al., 2009]. The aetiopathogenesis of the juvenile form has been discussed for a long time and it is still controversial. The common risk factors, such as tobacco and alcohol, involved in the genesis of carcinoma in adulthood are aetiologically of little significance in paediatric patients and in adolescents too; this is due to the fact that a very short-term exposure is not enough to induce neoplastic degeneration [Patel, 1999; Son et al., 1985; Bill et al., 2001]. In accordance with the acknowledged postulate that carcinogenesis may not only depend on the type

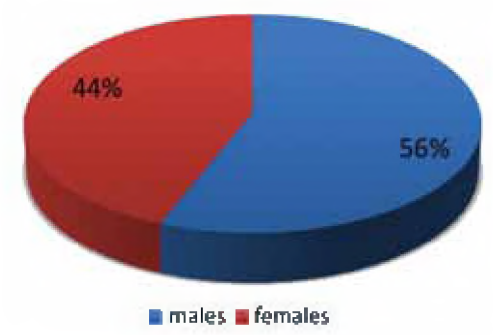

FIG. 1 Percentages of male and female subjects related to all cases reviewed in which this datum was available.

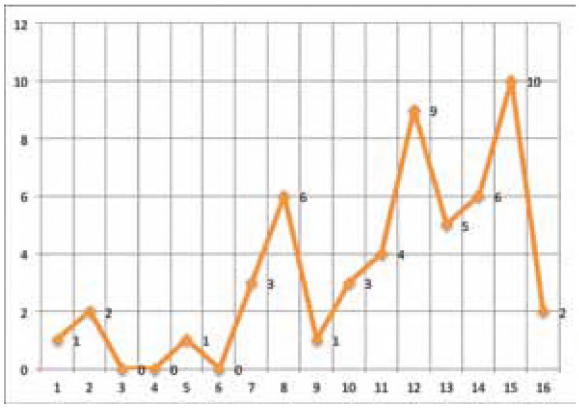

FIG. 2 Linear distribution of the cases of paediatric oral carcinoma on the basis of age brackets.

and degree of exposure to carcinogenic agents, our study indicates that paediatric SCC is nearly always associated with conditions like Epidermolysis bullosa, Xeroderma pigmentosum, Juvenile papillomatosis, which imply skin involvement [Yagi et al., 1981]. Lately more and more cases of patients affected by Fanconi's anaemia have been reported [Oskuszoglu et al., 2002]. The complex issue concerning the incidence of this neoplasia has been discussed since the 1950s when Moore, reviewing the literature about visceral carcinoma, reported a case of tongue carcinoma in a 13-year-old subject. He suggested a hypothesis for its aetiology related to irritation induced by the spur of a nonviable broken tooth [Moore et al., 1958]. The possibility that constant irritation or a parafunction may cause carcinoma is also claimed by Amichetti in the case report of a 14-years-old girl used to sucking and chewing a plastic ballpen always in the same position, while studying [Amichetti, 1989]. In literature there is also a case of oral carcinoma in a 14-years-old boy due to dental appliance [Newman et al., 1983] (Fig. 3). The chance that it may occur as a malignant degeneration of Pseudoepitheliomatous Hyperplasia (PEH) or other chronic inflammatory conditions has been highlighted as well. In an article dated 2011, Ribeiro et al. reported the case of a 7-year-old patient affected by well differentiated gingival carcinoma, in wich histological evaluation revealed a chronic inflammatory substrate attributable to a form of PEH [Ribeiro et al., 2011]; this is the only case report in which such condition is referred to as direct aetiological factor and not as a cofactor of the neoplastic pathology. However, in our study, though we found cases of subjects in good health affected by this neoplasia, most of paediatric patients, in addition to a family history positive for carcinomatous pathologies, show very clear predisposing conditions [Krolls et al., 1976; Chow et al., 2007].

Nowadays great attention is drawn to Fanconi's anaemia, an autosomal recessive, or in rare case X-linked, disease characterised by severe cytopenia and constitutional abnormalities. The phenotype severity is partly due to the group of specific complementation and more significantly to the kind of genetic mutation [Murayama et al., 1990; Somers et al., 1995; Oskuszoglu et al., 2002]. Patients affected by Fanconi's anaemia tend to develop haematologic malignant disorders, but they are also at high risk of falling ill with solid tumours like SCCs. On this point all mainstream medical opinion indicates as a possible cause poor DNA repair and inadequate detoxification from oxygen free radicals, with the consequent inability to keep the genome integrity, which leads to a high degree of chromosomal instability. The role played by the mutations of gene p53 (the preeminent oncosuppressor gene localised on chromosome 17) and its protein, very frequently detected in SCC, is the subject of a study by Kutler et al. [2003]. The authors found out a high incidence of this mutation in oral carcinomas in the young population affected by Fanconi's anaemia associated with Human Papilloma virus (HPV) infection. It is thought that this aetiological mechanism is at the root of the other predisposing conditions.

Like in Fanconi's anaemia, in Xeroderma pigmentosum [Yagi et al., 1981] there are different groups of complementation; the subjects are more likely to develop carcinoma in the oral cavity, especially on the tongue surface and its onset is between 6 and 9 years [Reihnard et al., 2007]. Very similarly, also in Epidermolysis bullosa, a disease which causes epithelial fragility and that can be classified into 3 groups according to the degree of epithelial cleavage (simple, junctional and dystrophic), OSCC (hard palate) seems to be a frequent complication, especially in the forms related to the junctional subtype. Particular attention is drawn to HPV, of which more than 100 types have been identified. The genome of this virus consists of 6 genes with rapid expression and 2 with late expression; genes E6 and E7 have been studied more thoroughly because of their interaction with oncogenic p53 and Rb, a feature that helps HPV to mediate oncogenesis, especially in regard to lining epithelia. Virus type 16 is present in most cases of HPV-related carcinoma [Woo et al., 2009]. In the paediatric age infection is mostly connected with mother-to-child transmission, generally via the delivery duct, but it can also occur through contact with hands, feet and mouth. In adolescents infection commonly occurs through sexual transmission (Fig. 4).

Clinical appearance of oral carcinoma is extremely variable, very often asymptomatic until diagnosis (Fig. 5). Cases of tooth mobility in oral carcinoma on the gingiva mainly in the lower jaw have been reported. Generally lesions have a warty appearance and are indurated with a rolled border, often ulcerated, but they may resemble fungal infection, since they are covered with white spots, or herpes-like lesions (Fig. 6). Sometimes lesions are histologically referable to a malignant degeneration of inflammatory lesions of reactive nature. They can be painful on palpation and during mastication. Their

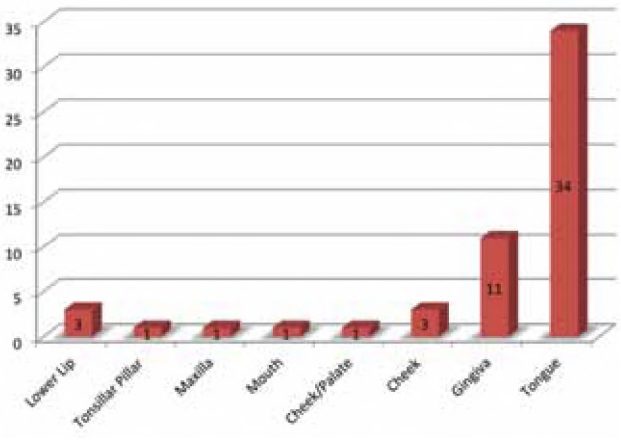

FIG. 3 Specific-site distribution of the cases of oral carcinoma.

YEAR	AUTHORS	ASSOCIATED CONDITIONS	SURVIVAL	TNM
1894	Authors	N.A.	N.A.	N.A.
1936	Variot	N.A.	Absence of relapse after 24 months	N.A.
1940	Frank et al.	N.A.	N.A.	N.A.
1940	New & Hertz	N.A.	N.A.	N.A.
1949	Pack & LeFevre	N.A.	N.A.	N.A.
1955	De Noronha	N.A.	N.A.	N.A.
N.A.	Merrifield et al.	N.A.	Absence of relapse after 1 year	N.A.
N.A.	Kapiloff	N.A.	N.A.	N.A.
N.A.	Warthin	N.A.	N.A.	N.A.
N.A.	Pack & LeFevre	N.A.	N.A.	N.A.
N.A.	Pack & LeFevre	N.A.	N.A.	N.A.
N.A.	Pack & LeFevre	N.A.	Absence of relapse after 3 years	N.A.
N.A.	Pack & LeFevre	N.A.	Death after 8 months	N.A.
N.A.	Friedman & Rubenfeld	N.A.	Death after 1 year	N.A.
N.A.	Saleeby	N.A.	Absence of relapse after 1 year	N.A.
N.A.	De Nicola	N.A.	Death after 15 months	N.A.
1958	Callum	Ionising radiation	Absence of relapse after 2 years	N.A.
1966	Moore	N.A.	Death after 8 months	N.A.
1969	Kaeriae et al.	Chronic histiocytosis	N.A.	N.A.
1970	Lancaster e Fournet	N.A.	Absence of relapse after 15 years	N.A.
1974	Kaeriae et al.	N.A.	N.A.	N.A.
1976	Turner & Snitzer	N.A.	N.A.	N.A.
1976	Patel & Dave	N.A.	N.A.	N.A.
1976	Patel & Dave	N.A.	N.A.	N.A.
1976	Krolls & Hoffmann	N.A.	N.A.	N.A.
1976	Krolls & Hoffmann	N.A.	N.A.	N.A.
1979	Krolls & Hoffmann	N.A.	Death after 1 year	N0
1981	Kaeriae et al.	Xeroderma pigmentosum	N.A.	N.A.
1982	Yagi et al.	N.A.	N.A.	N.A.
1982	Nuutinen et al.	N.A.	N.A.	N.A.
1983	Soni & Chatter	N.A.	N.A.	N.A.
1985	Newmann et al.	Francosi's anaemia	N.A.	N.A.
1985	Kaplan et al.	Xeroderma pigmentosum	N.A.	N.A.
1985	Wade & Plotnick	Nothing	Absence of relapse after 2 years	N0M0
1987	Sacks et al.	Xeroderma pigmentosum	N.A.	N.A.
1987	Usenius et al.	N.A.	N.A.	N.A.
1988	Usenius et al.	Parafunctions	Death after 5 months	T3N1M0
1989	Amichetti	Nothing	N.A.	N.A.
1989	Earle et al.	N.A.	N.A.	N.A.
1989	Keukens et al.	Francosi's anaemia	N.A.	N.A.
1995	Murayama et al.	Franconi's anaemia, BMT, radiation	N.A.	N.A.
1999	Somers et al.	Xeroderma pigmentosum	N.A.	N.A.
2001	Witold	Orthodontic appliance	Absence of relapse after 3 years	N0M0
2001	Bill et al.	Franconi's anaemia, BMT, radiation	N.A.	N.A.
2007	Mondovits et al.	Francosi's anaemia	Absence of relapse after 6 months	N0M0
2007	Reinhard	Franconi's anaemia, BMT, radiation	N.A.	N.A.
2007	Chow et al.	Ewing's sarcoma	N.A.	N.A.
2007	Chow et al.	Franconi's anaemia, BMT, radiation	N.A.	N.A.
2007	Chow et al.	Nothing	N.A.	T4aN0M0
2007	Stolk-Lifferink et al.	Nothing	N.A.	T1N0M0
2008	Binhamed et al.	Nothing	Absence of relapse after 2 years	pT4N0M0
2009	Mehanna	Nothing	N.A.	N.A.
2009	Sidell et al.	Nothing	Absence of relapse after 6 months	N.A.
2009	Woo et al.	Nothing	Absence of relapse after 24 months	N.A.
2011	Woo et al.	PEH	N.A.	T3N0M0

TAB. 1 Cases analysed in the literature review.

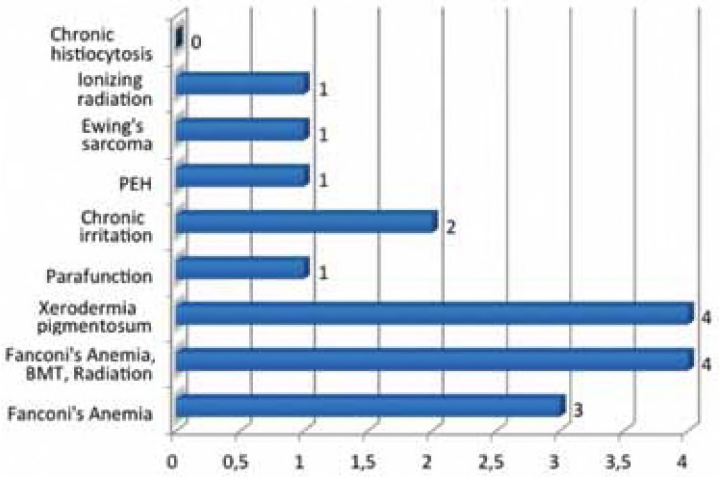

FIG. 4 Distribution of the pathologies regarded as paediatric OSCC-related found out in literature.

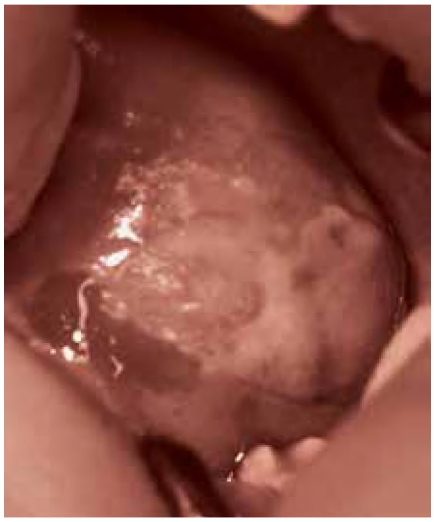

FIG. 6 Squamous cell carcinoma on the tongue border in a 13-year-old girl affected by Fanconi's anaemia. The leukoplakia-like clinical appearance of the lesion resembling a fungal infection should be noticed; courtesy by Dr Graf, Department of Paediatric Oncology and Haematology, Hamburg University General Hospital, Germany.

histological appearance can vary, even if they tend to keratinise more than in the adult manifestation (Fig. 7). The case reports in literature are usually related to well-differentiated carcinomas.

Prognosis in paediatric subjects affected by OSCC can vary and does not necessarily depend on the local extension of neoplasia [Son et al., 1985]. Metastasisation is local or involves the omolateral lymph nodes, but this is not a standard feature, since the lesion growth is mainly exophytic, more often with extensive involvement of the underlying alveolar bone [Mehanna et al., 2009; Binhamed et al., 2007] (Fig. 8). Survival rate is variable; prognosis is favourable after 2 years without signs of relapse. Deaths are often linked to the recrudescence

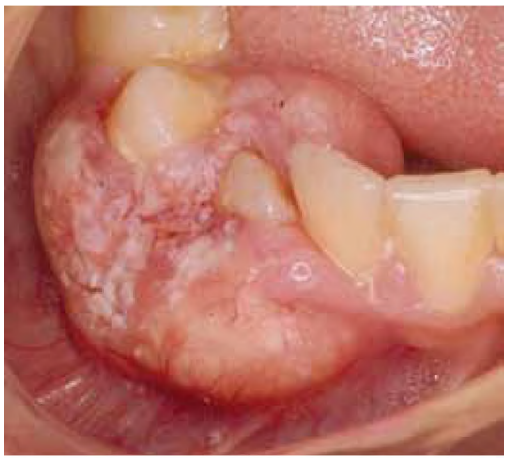

FIG. 5 Mandibular squamous cell carcinoma in a 10-year-old child. Reprinted from: Mehanna P, Patel PJ, Bailey BMW. Mandibular SCC in a 10-year-old child: a clinical rarity. British Journal of Oral and Maxillofacial Surgery 2009;47(2):148-150 (with permission from Elsevier).

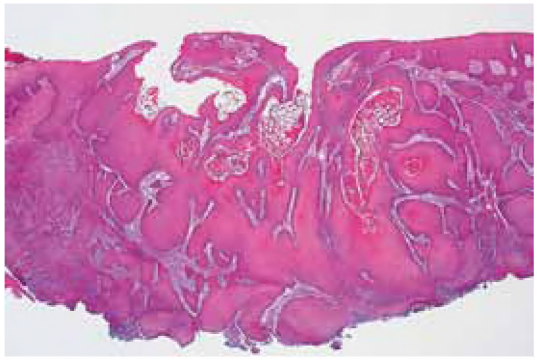

FIG. 7 Hematoxylin-eosin stained histological section of a well-differentiated carcinoma with keratoacanthoma-like features, among which central invagination replete with keratin. Courtesy of Dr Victoria Woo, Associate Professor, Department of Biomedical Sciences, School of Dental Medicine, Las Vegas University, USA.

of the concomitant pathology (e.g. Fanconi's anaemia). In this connection it is important to point out how the patients who suffer from Fanconi's anaemia and have undergone bone marrow transplantation develop earlier and more severe OSCC than non-transplanted subjects [Reinhard et al., 2007].

The protocol of treatment of OSCC in paediatric patients does not differ from that in adults, as it is chiefly based on radical resection and reconstruction. However, reconstruction will have to take into account the patient's residual growth. Unlike adults, the combination with radiation therapy, if possible, is always avoided because the collateral effects, which often arise as a consequence, may turn out to be even more harmful than those brought about by the primary lesion, particularly in those patients with a condition predisposing to genetic instability [Bill et al., 2001].

FIG. 8 Intraoral x-ray showing a vertical and horizontal loss greater than 50% of the basal bone. Courtesy of Dr John Fantasia, Chief of the Department of Oral and Maxillofacial Pathology, North Shore Long Island Jewish Medical Center, USA.

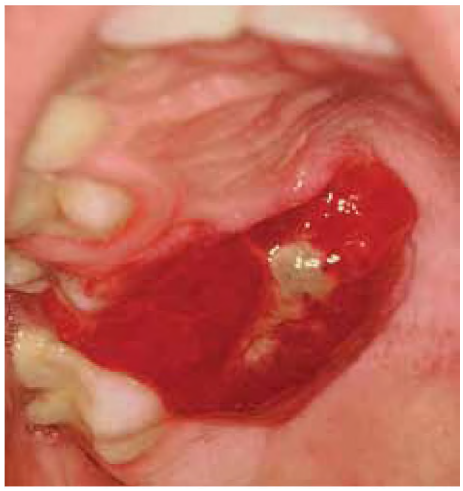

FIG. 9 Bright red, large esophytic lesion spreading from the palate to the gingival mucosa in a 12-year-old boy. Suspected OSCC. Histological evaluation yielded an unexpected diagnosis of nodular fasciitis. Courtesy of Prof Verrina, Galliera Hospital, Genoa, Italy.

Conclusion

Paediatric OSCC, though uncommon, is not rare (Fig. 9). Our review has strongly highlighted the need to carry out an objective thorough standardised examination of the child's oral cavity, especially when systemic predisposing pathologies such as Epidermolysis bullosa, Xeroderma pigmentosum, Juvenile papillomatosis and Fanconi's anaemia are present.

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