ABSTRACT:
Benign pigmented lesions on the oral mucosa are occasional findings that are physiologically or pathologically determined. Such lesions can occur in healthy individuals or as a manifestation of systemic disease. The pigmented lesions are presented as melanoplakia, melanocytic macule, melanocytic nevus or melanoacanthoma. Without appropriate treatment, they could transform into malignant melanoma. The purpose of the presentation at hand is to exhibit a case of focal oral pigmentation on the gingiva of the mandible of 17-years old patient without other comorbidities. The lesion subsequently is excised and pathohistologically examined in order to determine the diagnosis. Patient's recovery was uneventful, no complications are registered during the 6-months follow-up period.

Keywords: focal oral pigmentation, gingiva of mandible, melanoma

INTRODUCTION:
The term “Oral pigmentations” includes a large group of conditions represented by the accumulation of one or several types of pigments, thus causing color changes in the oral mucosa. [1] Oral pigmentations affect both males and females with no significant differences in the distribution [2]. The most commonly affected oral sites are the palate and maxillary gingiva. Mandibular involvement is rare. [3] Hyperpigmentations of the oral mucosa can be acquired or congenital, localized or diffuse, they can have exogenous or endogenous origin. [1] Exogenous oral pigments are most often attributed to foreign bodies implanted in the oral mucosa. Endogenous pigments include melanin, melanoid, oxyhemoglobin, reduced hemoglobin, carotene, bilirubin and iron. [4] Increased production of melanin can cause pigmented lesions of brown, blue, grey or black color, which is determined by the amount of melanin deposits and its distribution in the tissues. [5] The study of Akçiçek G et al. reveals that the most commonly detected mucosal lesions are Fordyce’s granules (20.1%), linea alba buccalis (16.9%), melanoplakia (15.9%), and frictional keratosis (2.5%). [6]
Fig. 2. Fourteen days following surgery

DISCUSSION:
Human oral mucosal epithelium is not uniformly colored, and several degrees of chromatic variegations may be observed in physiologic and pathologic conditions. Pigmentation of the oral mucosa is present in all races. Distribution and color intensity varies across individuals of different or same race, as well as different localizations in the same patient. Most of the oral pigmentation are physiological and, most likely, genetically determined. Well-defined areas of increased melanin deposits that are not associated with systemic conditions or syndromes are identified as ephelis, lentigo, melanoplakia, melanocytic macule and focal melanocytic melanosis. Melanin is a pigment produced in the melanocytes in the basal layer of the epithelium. It is then transported to keratinocytes through membrane-attached organelles called melanosomes. Melanocytes in the oral mucosa were isolated for the first time by Becker in 1927, however a few years later Laidlaw and Cahn isolated them for the first time in gingival samples. No evident difference is found in the melanocytic count in pale- and dark-skinned people, therefore it is assumed that variations in skin color are attributed to melanocytic activity.

Increased melanocytic pigmentation of oral mucosa can be evident for some systemic pathologies such as Peutz-Jeghers’ syndrome Addison’s disease. Albright’s syndrome, von Recklinghausen disease, hemochromatosis and acanthosis. Black pigmentation can be caused by systemic intoxication with heavy metals, such as mercury silver, lead. Some medications, such as antimarial drugs, some groups of antibiotics and chemotherapeutics can also lead to black pigmentation of the oral mucosa. Physiological pigmentation of the oral mucosa usually manifests in the first decade of life, and its color varies from light to dark brown. Such findings are localized bilaterally in the marginal region of the attached gingiva and resemble dark brown, garland-like band with well-defined borders. Physiological pigmentation are most often asymptomatic and do not require treatment.

Another form melanocytic hyperfunction can be presented as melanoma – one of the most malignant tumors in humans. There are no clinical differences between benign hyperpigmented lesions and melanoma of the oral mucosa. Most of the cases occur de novo, however, more than 30% follow benign localized pigmentation. The comprehensive etiology of oral melanoma is yet to be established, however, the presence of melanotic hyperpigmented areas is the single proven risk factor. This is why differential diagnosis between oral hyperpigmentation and melanoma malignum is of paramount significance.

CONCLUSION:
Oral pigmented lesions are postulated as benign hyperpigmentation of the oral mucosa with malignant potential. Therefore proper treatment includes radical excision; in each case of oral hyperpigmentation, histopathological evaluation is absolutely necessary for a diagnosis.

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