

Pseudocarcinoma: A Case Report and Review

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ABSTRACT

Pseudocarcinoma is another name for keratoacanthoma, which is a self-limiting benign epithelial proliferative lesion that originates from the pilosebaceous glands. Currently, Keratoacanthoma is considered as a low grade variant of squamous cell carcinoma (SCC), due to the clinical and histological resemblance to well differentiated SCC. Various etiological factors have been implicated in pathogenesis of keratoacanthoma. Most keratoacanthomas occur in sun exposed areas of skin and 8% of all the cases occur in vermilion border of the lips. The present article discusses and reviews a case of keratoacanthoma in mucosal surface of lip commissure which is rare when compared to the cutaneous counterpart.

Keywords: Keratoacanthoma, Pseudo-carcinoma, Self healing carcinoma.

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INTRODUCTION

Keratoacanthoma is a relatively common low grade tumor composed of keratinized squamous cells that originates from the pilosebaceous glands and closely resembles squamous cell carcinoma (SCC). Keratoacanthoma involving mucosal surface is less frequent than its cutaneous counterpart.¹ The occurrence of keratoacanthoma in mucosal surfaces suggests its possible origin from the surface epithelium or ectopic sebaceous glands.² The clinical and histopathological features of keratoacanthoma are similar to SCC, and thus represent a diagnostic challenge to the dental professional.

CASE REPORT

A 72 years old female patient reported to the outpatient department with chief complaint of a growth on the

upper lip of 3 years duration. History revealed that the growth started as a small nodule and attained the present size. The patient had a history of betel quid chewing for the past 30 years, but quit the habit 3 years ago. The patient also had a habit of lip biting. Her medical history revealed that she was a known cardiac patient currently under medication.

Clinically, a well defined solitary growth was present on the lip commissure at the left side with the margins of the lesion approximating the vermilion border of the upper and lower lips (Fig. 1). The lesion was sessile, measured approximately 0.5 × 0.5 cm, and was ulcerated. On palpation, the lesion was tender and not fixed to underlying musculature. The patient did not mention any other lesion in the body. Based on the clinical findings, the provisional clinical diagnosis keratoacanthoma was made. Excisional biopsy was done for histopathological examination (Fig. 2). The patient was recalled after 7 days and complete regression of the lesion was observed (Fig. 3).

The histopathological evaluation of hematoxylin and eosin (H&E) stained tissue section revealed a hyper parakeratinized stratified squamous epithelium which showed hyperplasia, individual cell keratinization, minimal cellular atypia and central keratin filled crater. Connective tissue showed hair follicle and associated sebaceous glands (Figs 4 and 5). The connective tissue stroma was moderately collagenous with dense chronic inflammatory cell infiltration. Correlating the clinical and histopathological features, a final diagnosis of keratoacanthoma was confirmed.



Fig. 1: Well-defined nodular growth with ulceration

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Fig. 2: Gross specimen



Fig. 3: Postoperative healing without scarring

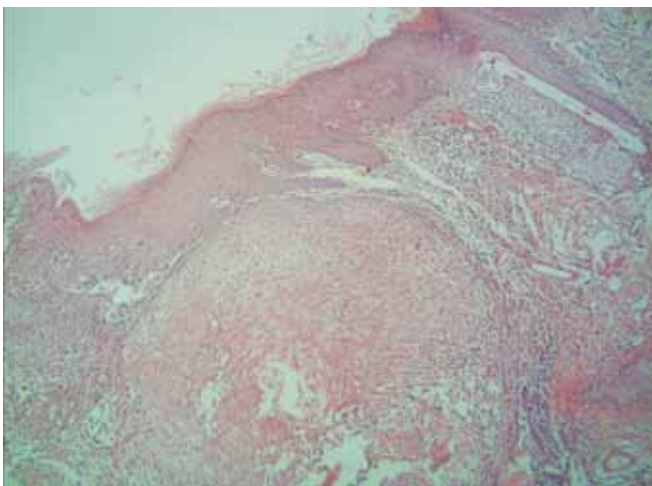


Fig. 4: Histopathology showing hyperparakeratinized hyperplastic stratified squamous epithelium, crater formation, sebaceous glands with associated hair follicle (H&E staining, 400x)

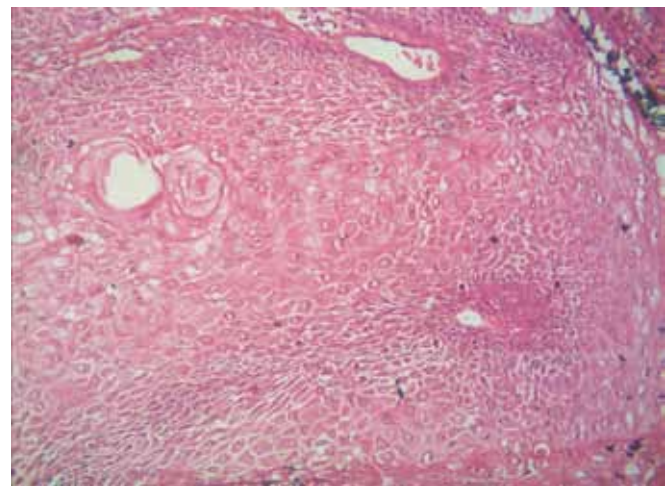


Fig. 5: Histopathology showing individual cell keratinization with minimal cellular atypia (H&E staining, 1000x)

DISCUSSION

Keratoacanthoma was first described as a crateriform ulcer of the face by Jonathan Hutchinson in 1889, but the term 'Keratoacanthoma' was coined by Freudenthal and later used by Rook and Whimster in 1950 in their respective case series. Keratoacanthoma has been reported in all age groups, but incidence is seen to increase with age. Incidence is estimated at 1 in 1,000 and peak incidence occurs in those aged over 60 years. The male to female ratio for keratoacanthoma is 2:1.^{1,2}

The cause of keratoacanthoma is still uncertain, but various etiological factors have been postulated, including trauma, sunlight, chemical carcinogens, human papilloma virus, genetic factors and immunocompromised status. Industrial workers exposed to pitch and tar have been well established as having a higher incidence of keratoacanthoma as well as SCC. At present, it has been identified that up to one-third of keratoacanthomas harbor chromosomal aberrations in some extent similar to SCC. Recurrent aberrations include gains on 8q, 1p,

9q with deletions on 3p, 9p, 19p, 19q are observed in keratoacanthoma.³

Keratoacanthomas are common in pale complexioned population who are chronically exposed to sun radiation.⁴ The most common sites of involvement in keratoacanthomas are face, neck, and dorsum of the upper extremities. In the face, lower lip is a common site of involvement. The tumor usually appears on sun-exposed areas in middle-aged or older patients, which suggests an etiological association with ultraviolet light exposure. Keratoacanthoma and conventional SCC share very similar epidemiological features, which suggest a possible common pathogenesis, such as actinic damage.^{3,4}

Keratoacanthoma is usually solitary and begin as firm, round, skin colored or reddish papules that rapidly progress to dome-shaped nodules with a smooth shiny surface. A central crater of ulceration with keratin plug usually projecting like a horn may develop. This is referred to as 'volcano'.⁵ The spontaneous regression after an initial growth is an important hallmark of

Table 1: Criteria by Ackerman et al (1992) and Cribier et al (1999)⁹ to differentiate keratoacanthoma and SCC

Criteria	Keratoacanthoma	SCC
Clinical	Exo-endophytic lesion	Predominantly endophytic lesion
	Central keratin-filled crater	No central keratin-filled crater
	Overhanging 'lips' of epithelium	No 'lips' of epithelium
	Rarely ulcerate	Commonly ulcerated
Histopathological	Minimal cellular atypia	Abundant cellular atypia
	Lack of anaplasia	Present
	Sharp outline between tumor nests and stroma	Indistinct
	Absence of stromal desmoplasia	Present
	Intraepithelial abscesses within the lesion	Rare
	Central keratin plug	Absent

keratoacanthoma; however, some keratoacanthoma do not fit this pattern.¹ In this present case report, the patient did not recall any regressive behavior and the histopathological diagnosis provided a clue for final diagnosis.

Clinically, two types of keratoacanthoma exist, solitary and multiple.⁶ Solitary keratoacanthoma may be divided into following types:

Types	Subtypes	Location
Solitary	Giant	Dorsa of the hands
	Centrifugum marginatum	Nose and eyelids
	Subungual	Nail apparatus
Multiple		Any region

Keratoacanthoma appearing as multiple lesions are generally associated with a few hereditary syndromes. It can be associated with a condition called Muir-Torre syndrome, which is described as a cancer-associated genodermatosis with multiple sebaceous neoplasms, keratoacanthomas and gastrointestinal malignancies.⁷

Histopathology is essential for establishing the diagnosis of keratoacanthoma. Keratoacanthomas are composed of squamous epithelium that shows only a mild degree of pleomorphism. Masses of keratin constitute the central core of keratoacanthoma. Pseudocarcinomatous infiltration in keratoacanthoma typically presents a smooth, regular, well-demarcated front that does not extend beyond the level of the sweat glands.⁸ Corbalan Velez et al indicated immunohistochemical staining for laminin-322 as useful marker for distinguishing between keratoacanthoma and SCC.⁶ Keratoacanthoma can be differentiated from SCC by certain specific criteria, as postulated by Ackerman and Cribier et al (Table 1).

The primary therapy for keratoacanthoma is surgical excision of the tumor. Excision of tumors with adequate margins (3–5 mm) is advisable. This would allow a proper histopathological diagnosis and exclusion of other neoplastic conditions. Because the biological behavior of an individual keratoacanthoma cannot be predicted, the surgical treatment of keratoacanthoma is to be equivalent to treatment for SCC.⁵ Mohs micrographic surgery may be indicated for large and recurrent keratoacanthomas

or those located in anatomic areas with cosmetic or functional considerations.¹⁰

Both laser therapy and cryotherapy have been used successfully in small keratoacanthomas, and also in keratoacanthomas found in difficult to treat locations. Intralesional methotrexate (MTX), fluorouracil, bleomycin and steroids have been used with success in patients as an adjunct to surgical removal.¹¹ Keratoacanthomas are radiosensitive and respond well to low doses of radiation (<10 Gy). Radiation therapy may be useful in selected patients with large lesion. Keratoacanthoma occasionally have tissue destructive and metastatic potential, with infiltrative growth. In such cases, the clinical diagnosis between keratoacanthoma and well-differentiated SCC is difficult. Thus, keratoacanthoma may also be considered a subtype of malignant lesion and should be treated accordingly.¹² In this present case, complete surgical excision was performed with regular follow-up.

CONCLUSION

Keratoacanthoma is a common benign epithelial proliferation. Due to its clinical and histopathological resemblance to SCC, a histological diagnosis in many cases is a challenge and cases are to be kept on follow-up for many years, although chances of recurrence are very rare.

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