BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 22, Issue 4

DOI:10.58240/1829006X-2026.22.4-111

REVIEW ARTICLE

ETIOLOGICAL ASPECTS AND EPIDEMIOLOGICAL PATTERNS OF KERATOACANTHOMA: NARRATIVE REVIEW

Khachik Khachikyan[1], Hovhannes Hovhannisyan[2], Roza Mesropyan[3]
[1]MD, PhD, DMSci, Professor; Head of Department of Dermatology and Venereology, Yerevan State Medical University after M. Heratsi, Yerevan, Armenia
[2]Head of National Center of burnes and dermatology of MOH RA, Yerevan, Armenia
[3]Assistant of Department of Dermatology and Venereology, Yerevan State Medical University after M. Heratsi, Yerevan, Armenia
Corresponding author: Professor Khachik Khachikyan,Head of Department of Dermatology and ,Yerevan State Medical University after M. Heratsi, Yerevan, Armenia
Received: May 4. 2026; **Accepted:** Jun 15. 2026; **Published:** Jun 20. 2026

ABSTRACT

Keratoacanthoma (KA) is a rapidly developing keratinizing epithelial neoplasm whose classification remains controversial because of its close relationship to cutaneous squamous cell carcinoma (cSCC). Despite its frequent occurrence in dermatological practice, the epidemiology and pathogenesis of KA remain incompletely understood. This narrative review summarizes current evidence regarding the etiological factors and epidemiological characteristics of KA. Available data indicate marked geographic variation in incidence, largely reflecting differences in ultraviolet (UV) exposure, environmental conditions, and population skin phototypes. The tumor occurs predominantly in fair-skinned individuals and is most commonly diagnosed in older adults with a history of chronic sun exposure.

Current evidence supports a multifactorial origin of KA. Ultraviolet radiation is considered the principal environmental risk factor and is associated with characteristic molecular alterations, including mutations in TP53 and HRAS. Additional contributors include human papillomavirus infection, particularly cutaneous beta-papillomavirus types; immunological dysregulation related to immunosuppressive therapy and immune-modulating drugs; chronic inflammatory stimuli following trauma, surgical procedures, or tattooing; exposure to chemical carcinogens such as arsenic and tar-derived compounds; and inherited cancer-predisposition syndromes, including Muir–Torre and Ferguson–Smith syndromes. Increasing evidence suggests that interactions between genetic susceptibility, environmental exposures, and local immune responses play a central role in tumor development. In conclusion, keratoacanthoma represents a multifactorial neoplastic process arising from the combined effects of environmental, genetic, viral, and immunological factors. Further epidemiological and molecular studies are needed to clarify disease mechanisms and improve risk stratification.

Keywords: keratoacanthoma, etiology of keratoacanthoma, epidemiology of keratoacanthoma.

INTRODUCTION

Keratoacanthoma (KA) is a distinctive keratinizing epithelial neoplasm of the skin characterized by rapid growth,a typical crateriform architecture, and a a variable biological behavior. It most frequently occurs on chronically sun-exposed areas, particularly in elderly patients, and usually presents as a rapidly developing dome-shaped lesion with a central keratin-filled depression. The clinical appearance of KA often resembles cutaneous squamous cell carcinoma (cSCC), creating significant diagnostic difficulties in daily dermatological and histopathological practice.

Although KA has historically been described as a lesion capable of spontaneous regression, its potential relationship with cSCC remains one of the most debated issues in cutaneous oncology [1–3].

For many years, KA was considered a separate benign or borderline epithelial proliferation with a characteristic evolution consisting of rapid enlargement, stabilization, and possible involution. However, increasing evidence from morphological, immunohistochemical, and molecular studies has demonstrated substantial similarities between KA and well-differentiated cSCC. These similarities include overlapping histological features, common genetic alterations, and comparable responses to environmental carcinogenic factors. As a result, the previous concept of KA as an entirely independent benign lesion has gradually changed, and its classification has become a subject of continuous discussion among dermatologists, pathologists, and oncologists [1,3,24].

A major turning point in the understanding of KA occurred with the publication of the fourth edition of the World Health Organization (WHO) Classification of Skin Tumours in 2018. Based on accumulated clinical, histopathological, and molecular data indicating a close biological relationship between KA and cSCC, KA was incorporated into the spectrum of well-differentiated cutaneous squamous cell carcinomas and categorized as keratoacanthoma-type squamous cell carcinoma. This approach reflects the current view that KA and cSCC may represent different manifestations of closely related keratinocytic neoplastic processes rather than completely distinct diseases. According to the WHO classification, this entity is coded as ICD-O 8071/3 and is represented within ICD-10 and ICD-11 classification systems. Historically, KA has been described by several alternative terms, including molluscum sebaceum, keratinous molluscum, pseudocarcinomatous molluscum, and Gougerot's verruciform epithelioma [1–3,25].

The etiology and pathogenesis of KA are considered multifactorial, involving the interaction of genetic susceptibility, environmental influences, and alterations in epidermal homeostasis. Chronic ultraviolet (UV) radiation exposure is regarded as one of the most important contributing factors, particularly due to its ability to induce DNA damage and promote mutations affecting key regulatory pathways involved in keratinocyte proliferation and differentiation. Alterations in tumor suppressor mechanisms, including TP53 dysfunction, as well as changes in oncogenic signaling pathways such as RAS-related pathways, have been described in KA and may contribute to abnormal keratinocyte growth. In addition, immune dysregulation, impaired local immune surveillance, viral factors, trauma, and exposure to certain chemical agents have been suggested as possible triggers. The interaction of these mechanisms may explain the heterogeneous clinical course of KA, including both spontaneous regression and progression with morphological features resembling cSCC [12,15–17].

Despite extensive investigation, the exact biological nature of KA remains incompletely understood. The difficulty in predicting its behavior and the considerable overlap with cSCC continue to make KA an important diagnostic and scientific challenge. Further studies focusing on molecular mechanisms, genetic alterations, and clinicopathological correlations are necessary to better define the relationship between KA and cSCC and to improve the accuracy of diagnosis and classification [1,3,24].

The present narrative review aims to summarize and critically discuss current evidence regarding the epidemiology, etiological factors, and biological behavior of keratoacanthoma. Particular attention is given to the environmental, viral, genetic, and immunological mechanisms that may contribute to tumor development and influence its clinical course. The review is based on published clinical, epidemiological, and experimental studies, as well as contemporary international classification systems that have shaped current understanding of this unique and still controversial cutaneous neoplasm.

EPIDEMIOLOGY

Although keratoacanthoma (KA) is considered a relatively common cutaneous neoplasm, its epidemiology remains incompletely characterized [3]. Most of the available evidence originates from single-center studies or retrospective case series involving relatively small patient cohorts, which limits the ability to draw definitive conclusions regarding the global burden of the disease. Consequently, large population-based investigations remain particularly valuable for understanding the incidence and demographic characteristics of KA.

One of the most important epidemiological studies of keratoacanthoma is the Queensland Skin Cancer Cohort (QSkin) study conducted in Queensland, Australia, between 2010 and 2014[7,8]. This prospective population-based cohort study provided the first large-scale assessment of KA incidence and associated risk factors.

The analysis included 40,438 individuals aged 40–69 years selected from a random sample of 193,344 Queensland residents. Among these participants, 584 patients were diagnosed with keratoacanthoma.

The findings demonstrated an age-standardized incidence rate of 409 cases per 100,000 person-years based on the Australian standard population. Men were affected slightly more frequently than women, accounting for 58% of all diagnosed cases. The majority of patients were aged 60 years or older (76%), while fair-skinned individuals represented approximately 80% of the affected population. Notably, a history of actinic keratosis or cutaneous squamous cell carcinoma was documented in 89% of patients, emphasizing the close epidemiological relationship between these lesions and chronic ultraviolet-induced skin damage [7,8].

The study also provided valuable information regarding lesion distribution. Keratoacanthomas were most commonly located on the lower extremities, including the legs and feet (48%), whereas involvement of the head and neck region was relatively uncommon (7%). An additional noteworthy observation was the occurrence of multiple lesions. Although 584 patients were included in the analysis, a total of 738 keratoacanthomas were diagnosed, indicating that approximately 18% of affected individuals developed multiple tumors [8].

Earlier population-based data were obtained from a five-year epidemiological study conducted on the island of Kauai, Hawaii, between 1983 and 1987 [9,10]. During this period, 53 incident cases of keratoacanthoma were identified, including 36 men and 17 women. The average annual incidence was calculated at 106 cases per 100,000 inhabitants. Interestingly, the incidence of keratoacanthoma was found to be comparable to that of cutaneous squamous cell carcinoma, challenging the previously accepted assumption that KA occurred approximately three times less frequently than cSCC. Subsequent investigations performed in different geographic regions demonstrated substantial variability in the KA-to-cSCC ratio, ranging from 1:0.6 to 1:5, suggesting that environmental and population-specific factors may significantly influence disease occurrence [9–11].

The Hawaiian study further demonstrated that the hands and shoulders were the most common anatomical sites affected by keratoacanthoma, whereas lesions of the trunk and other body regions occurred less frequently. The investigators also observed a lower incidence among individuals with darker skin pigmentation, supporting the widely accepted view that KA occurs most frequently in individuals with Fitzpatrick skin phototypes I–III. Men were affected approximately twice as often as women, with a male-to-female ratio of 2:1 [9–11].

Although keratoacanthoma has been reported across virtually all age groups, its incidence increases steadily with advancing age. The highest incidence rates are observed in individuals aged 70 years and older, whereas the disease is distinctly uncommon in patients younger than 20 years of age [2,9,11].

Taken together, currently available epidemiological evidence indicates that keratoacanthoma predominantly affects fair-skinned older adults, particularly men, and occurs most frequently in populations exposed to high levels of ultraviolet radiation. Nevertheless, significant geographic variation exists, reflecting differences in environmental exposure, genetic susceptibility, skin phototype distribution, and diagnostic practices. Additional large-scale population studies are required to better define the global epidemiological patterns of this unique and still controversial cutaneous neoplasm.

Table 1. Epidemiological characteristics of keratoacanthoma

Parameter	Current evidence
Typical age group	Older adults (>60–70 years)
Sex predominance	Male predominance
Skin type	Fitzpatrick I–III
Main environmental factor	Chronic UV exposure
Common locations	Sun-exposed skin, extremities
Multiple lesions	Reported in a subset of patients
Association	Actinic keratosis and cSCC history

ETIOLOGY

Despite decades of research, no single unifying hypothesis has been established to explain the etiology and pathogenesis of keratoacanthoma (KA). The tumor is generally believed to originate from the hair follicle epithelium, while its development appears to result from the interaction of multiple environmental, genetic,

viral, and immunological factors. Among the most frequently implicated risk factors are chronic ultraviolet (UV) radiation exposure, chemical carcinogens, immunosuppression, certain medications, genetic susceptibility, human papillomavirus (HPV) infection, and skin trauma or surgical interventions [2–5,11–14]. Notably, most etiological factors associated with KA closely resemble those recognized for invasive cutaneous squamous cell carcinoma (cSCC).

Ultraviolet Radiation and Photocarcinogenesis

Chronic exposure to ultraviolet radiation is widely considered one of the most important risk factors for keratoacanthoma development. This association is supported by the characteristic distribution of lesions, which occur predominantly on chronically sun-exposed areas of the body, including the face, ears, scalp, and dorsal surfaces of the hands. These anatomical locations account for approximately 80–85% of all reported cases [6].

The risk of developing KA is particularly elevated among individuals with Fitzpatrick skin phototypes I and II. Numerous studies have demonstrated that cumulative lifetime sun exposure contributes significantly to disease risk regardless of the pattern of exposure, while a history of previous sunburns represents an additional predisposing factor [2,12].

At the molecular level, UV-induced skin carcinogenesis is associated with mutations affecting tumor suppressor genes and signaling pathways involved in cellular proliferation and apoptosis. Similar to cutaneous squamous cell carcinoma, keratoacanthomas frequently exhibit alterations involving TP53, HRAS, and other genes that enable damaged keratinocytes to evade programmed cell death and continue proliferating [12,15,16].

Human Papillomavirus Infection

Human papillomavirus infection has long been considered a potential contributor to keratoacanthoma development. Particular attention has been focused on HPV types 9, 11, 13, 16, 18, 24, 25, 33, 37, and 57. Nevertheless, despite the growing number of studies demonstrating an association between HPV and KA, the precise role of the virus in tumor initiation and progression remains uncertain [11,17].

The hypothesis of viral involvement was first proposed in the 1960s following the identification of virus-like particles within tumor cells using electron microscopy. Subsequent molecular investigations confirmed the presence of HPV DNA in keratoacanthoma specimens. Current evidence suggests that cutaneous beta-HPV types may be of particular importance in the pathogenesis of the disease [11,17].

Interestingly, studies evaluating viral load during different stages of tumor evolution have revealed dynamic changes in HPV DNA concentration. Viral load tends to increase during the growth and stabilization phases of keratoacanthoma but decreases dramatically during spontaneous regression. In some regressing lesions, HPV DNA becomes undetectable altogether, suggesting a possible role for viral infection in tumor maintenance rather than initiation alone [11,17].

The potential contribution of viral and immune-mediated mechanisms has also been highlighted by reports of keratoacanthomas developing at sites of COVID-19 vaccination. Both solitary and eruptive forms have been described, with lesions occurring directly within the vaccination area and demonstrating a clear temporal association with vaccine administration [18,19].

Drug-Induced Keratoacanthoma and Immunological Factors

The spontaneous development of multiple keratoacanthomas has been reported in association with several pharmacological agents. Among the most frequently implicated medications are targeted therapies used in the treatment of advanced melanoma, particularly inhibitors of the MAPK signaling pathway (RAS–RAF–MEK–ERK). Vemurafenib and sorafenib are the drugs most commonly associated with the development of eruptive keratoacanthomas and non-melanoma skin cancers [20,21].

Dabrafenib has also been linked to an increased risk of keratinocytic neoplasms, although the incidence appears to be considerably lower than that observed with vemurafenib (approximately 14% versus 26%) [21]. The underlying mechanism is thought to involve paradoxical activation of proliferative signaling pathways and alterations in immune regulation. Inhibition of PI3K-, MAP kinase-, and NF-κB–related pathways may reduce cytokine release from Langerhans cells and impair T-lymphocyte-mediated surveillance against newly emerging tumor cells [15,20]. An increased risk of keratoacanthoma has also been reported in patients receiving immune checkpoint inhibitors targeting programmed cell death protein 1 (PD-1), including pembrolizumab and sintilimab.

Furthermore, cases have been described in individuals undergoing immunosuppressive therapy with leflunomide for active rheumatoid arthritis, further supporting the role of immune dysregulation in tumor development [12]. Skin Trauma and Tattoo-Associated Keratoacanthoma Traumatic injury is recognized as another important triggering factor for keratoacanthoma. Surgical procedures, burns, chronic irritation, and other forms of skin damage have all been associated with subsequent tumor development. In recent years, particular attention has been directed toward keratoacanthomas arising within tattooed skin. A systematic review demonstrated that approximately 79.2% of tattoo-associated keratoacanthomas occurred within red-pigmented areas of tattoos, suggesting a possible role for specific pigment components in tumor induction [22,23].

However, the pathogenesis of tattoo-related KA remains incompletely understood. Current evidence suggests that several mechanisms may contribute simultaneously, including local tissue trauma caused by tattooing, chronic inflammatory responses, ultraviolet radiation exposure, genetic susceptibility, and the biological effects of pigment constituents. The carcinogenic potential of tattoo pigments remains insufficiently investigated, although mercury sulfide, commonly used in red inks, has been proposed as a possible carcinogenic agent. Other substances identified in red tattoo pigments, including iron, zinc, and titanium oxides, have also attracted attention because of their potential biological activity [22,23].

Genetic Predisposition

Several hereditary disorders are associated with an increased risk of keratoacanthoma development, providing evidence for an underlying genetic susceptibility. These include Muir–Torre syndrome, Ferguson–Smith syndrome, Witten and Zak familial multiple keratoacanthoma, xeroderma pigmentosum, and generalized eruptive keratoacanthoma of Grzybowski, among others [12,24]. The occurrence of KA within these syndromes supports the concept that inherited abnormalities affecting DNA repair, tumor suppression, or immune regulation may facilitate tumor formation.

Chemical Carcinogens

Long-term exposure to chemical carcinogens has been implicated in the development of both invasive cutaneous squamous cell carcinoma and keratoacanthoma. Potentially hazardous substances include coal tar, pitch, arsenic, soot, paraffin, tar products, unrefined kerosene, creosote, and mineral oils [5,13,25].

Occupational exposure to such agents has been documented in approximately 42% of patients with keratoacanthoma in some clinical series [25].

Chronic contact with chemical carcinogens promotes cutaneous inflammation, epithelial hyperplasia, cellular atypia, and tissue remodeling. Over time, these alterations may lead to the formation of focal proliferative lesions that can subsequently evolve into malignant epithelial tumors [5,13].

Table 2. Etiological factors and proposed mechanisms in KA development

Risk factor	Proposed mechanism
UV radiation	TP53 mutations, keratinocyte damage
HPV infection	Viral persistence and immune modulation
Immunosuppression	Reduced tumor immune surveillance
MAPK inhibitors	Paradoxical pathway activation
Trauma/tattoos	Chronic inflammation
Genetic syndromes	DNA repair defects
Chemical carcinogens	Chronic epithelial stimulation

Although the exact mechanisms underlying keratoacanthoma development remain incompletely understood, current evidence suggests that several etiological factors may interact through complex molecular pathways. Ultraviolet radiation, genetic alterations, immune disturbances, viral factors, and external triggers may promote abnormal keratinocyte proliferation and contribute to the formation of the characteristic keratoacanthoma lesion.

The balance between proliferative signals and immune-mediated regression may influence the clinical course of keratoacanthoma, including spontaneous regression or progression toward a squamous cell carcinoma-like phenotype (Figure 1).

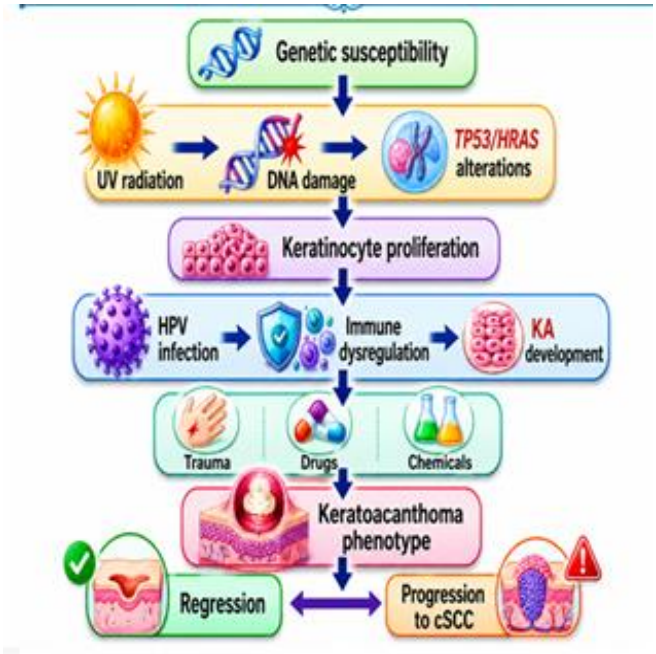

Figure 1. Multifactorial etiological factors and proposed mechanisms involved in keratoacanthoma development.

DISCUSSION

Keratoacanthoma remains one of the most debated entities in dermatopathology because its biological behavior is not yet completely understood. Historically, it was considered a benign, self-limiting epithelial tumor; however, accumulating clinical, histopathological, and molecular data indicate a close biological relationship with cutaneous squamous cell carcinoma. This overlap remains an important challenge in diagnosis and clinical decision-making [1–3,12]. The findings summarized in this review support the concept that keratoacanthoma is a multifactorial disease resulting from complex interactions between environmental exposures, genetic susceptibility, viral factors, and immune regulation. Chronic ultraviolet radiation appears to play a central role in tumor development, both through cumulative photodamage and through the induction of molecular alterations involving genes such as TP53 and HRAS [12,15,16]. The predominance of lesions on chronically sun-exposed skin and the increased incidence among fair-skinned individuals further support the importance of ultraviolet radiation in disease pathogenesis [7–12].

The contribution of human papillomavirus remains controversial. Although HPV DNA has been detected in numerous keratoacanthoma specimens and viral load appears to correlate with certain stages of tumor evolution, current evidence is insufficient to establish a direct causal relationship [11,17]. Similar uncertainty exists regarding the role of trauma, tattoo pigments, and vaccine-associated cases, which may represent triggering factors in predisposed individuals rather than independent causes of tumor formation [18,19,22,23].

Another important observation is the increasing number of reports describing keratoacanthomas associated with targeted anticancer therapies and immune-modulating agents. These findings suggest that immune surveillance and signaling pathways involved in keratinocyte proliferation may play a critical role in maintaining cutaneous homeostasis. Disruption of these mechanisms may facilitate the emergence of keratoacanthoma in susceptible individuals [12,20,21].

Despite significant advances in molecular pathology, a major challenge remains the absence of reliable biomarkers capable of consistently distinguishing keratoacanthoma from invasive cutaneous squamous cell carcinoma. This diagnostic uncertainty continues to influence clinical decision-making and largely explains why complete surgical excision remains the preferred therapeutic approach in most cases [1–3].

Overall, the available evidence suggests that keratoacanthoma should not be viewed as a single disease entity with a uniform pathogenesis. Instead, it is more likely to represent a biologically heterogeneous group of lesions sharing common clinical and histopathological features while arising through multiple overlapping pathogenic pathways.

LIMITATIONS AND FUTURE RESEARCH DIRECTIONS

Several limitations should be considered when interpreting the current evidence regarding keratoacanthoma. First, much of the available literature consists of retrospective studies, case series, and isolated case reports, while large prospective investigations remain relatively scarce. Second, significant heterogeneity exists among published studies with respect to diagnostic criteria, patient selection, and classification systems, which complicates direct comparisons between studies. Third, the ongoing debate regarding the biological nature of keratoacanthoma itself may have influenced epidemiological estimates and clinical reporting, particularly in studies conducted before the adoption of contemporary WHO classifications.

Further research is required to clarify the molecular mechanisms underlying keratoacanthoma development,

progression, and spontaneous regression. Particular attention should be directed toward the identification of diagnostic biomarkers capable of reliably differentiating keratoacanthoma from invasive cutaneous squamous cell carcinoma. Additional large-scale epidemiological studies are also needed to better define geographic variations in disease incidence and to evaluate the relative contributions of ultraviolet radiation, viral infection, genetic predisposition, and immune dysregulation. Advances in molecular profiling and translational research may ultimately facilitate more individualized diagnostic and therapeutic approaches for patients with keratoacanthoma.

Table 3. Keratoacanthoma and cutaneous SCC: similarities and differences

Feature	KA	cSCC
Growth	Rapid	Variable
Histology	Similar	Similar
Regression	Possible	Rare
Metastatic risk	Very low	Present

CONCLUSION

Keratoacanthoma is a unique keratinizing epithelial neoplasm characterized by complex and still incompletely understood biological behavior. Current evidence supports a multifactorial pathogenesis involving chronic ultraviolet radiation, genetic alterations, viral factors, immune dysregulation, chemical carcinogens, pharmacological triggers, and traumatic skin injury. Epidemiological studies consistently demonstrate a predominance among fair-skinned older individuals and populations exposed to high levels of solar radiation.

Although keratoacanthoma has traditionally been regarded as a self-limiting lesion, accumulating clinical and molecular evidence indicates that some cases may share biological features with cutaneous squamous cell carcinoma. However, important questions regarding its origin, progression, and potential for regression remain unresolved.

Future investigations focusing on molecular biomarkers, tumor microenvironment interactions, and mechanisms of spontaneous regression may improve diagnostic accuracy and enable more personalized management strategies. A better understanding of these processes may ultimately reduce diagnostic uncertainty and contribute to more effective treatment approaches for affected patients.

DECLARATIONS

Competing Interests
The authors declare no conflict of interest.
Funding
None.

Ethical Approval
Ethical approval was not required as this is a narrative review of published literature.

REFERENCES

1. Bahmad HF, Stoyanov K, Mendez T, et al. Keratoacanthoma versus squamous cell carcinoma: histopathological features and molecular markers. *Dermatopathology*. 2024;11(4):272-285. doi:10.3390/dermatopathology11040029.
2. Zito PM, Scharf R. Keratoacanthoma. [Updated 2023 Aug 8]. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499931/
3. Tisack A, Fotouhi A, Fidai C, et al. A clinical and biological review of keratoacanthoma. *Br J Dermatol*. 2021;185(3):487-498. doi:10.1111/bjd.20389.
4. Efanova EN, Vasileva EA, Rusak YuE, Maksimova KYu. Giant keratoacanthoma of the skin in dermatovenerologist's practice. *Vestnik SurGU. Meditsina*. 2025;18(1):61-66. doi:10.35266/2949-3447-2025-1-7.
5. Yunusova EI, Yusupova LA, Garaeva ZSh, Mavlyutova GI. Keratoacanthoma: clinical presentation, diagnosis and therapy. *Far Eastern Medical Journal*. 2021;(2). Available from: https://cyberleninka.ru/article/n/keratoakantoma-klinika-diagnostika-terapiya.
6. Snarskaya ES, Shnakhova LM, Gomich DA, Vasileva KD. A clinical case of atypical keratoacanthoma. *Russian Journal of Skin and Venereal Diseases*. 2020;(6). Available from: https://cyberleninka.ru/article/n/klinicheskiy-sluchay-atipichnoy-keratoakantomy.
7. Claeson M, Pandeya N, Dusingize JC, et al. Assessment of incidence rate and risk factors for keratoacanthoma among residents of Queensland, Australia. *JAMA Dermatol*. 2020;156(12):1324-1332. doi:10.1001/jamadermatol.2020.4097.
8. Kolmodin A, Pandeya NP, Olsen CM, et al. Patient and tumour characteristics of keratoacanthoma in a

large, community-based cohort study from Queensland, Australia. *Acta Derm Venereol*. 2021;101(6). doi:10.2340/00015555-3824.
9. Chuang T, Reizner GT, Elpern DJ, et al. Keratoacanthoma in Kauai, Hawaii: the first documented incidence in a defined population. *Arch Dermatol*. 1993;129(3):317-319. doi:10.1001/archderm.1993.01680240057005.
10. Chuang TY, Reizner GT, Elpern DJ, et al. Squamous cell carcinoma in Kauai, Hawaii. *Int J Dermatol*. 1995;34(6):393-397. doi:10.1111/j.1365-4362.1995.tb04439.x.
11. Hoffpauir LA, William DJ, editors. Keratoacanthoma. [Updated 2025 Feb 25]. In: *Medscape [Internet]*. Available from: https://emedicine.medscape.com/article/1100471-overview
12. Hyeraci M, Didona D, Abeni D, et al. Keratoacanthoma: new insight into pathogenesis and management of the disease. *Preprints*. 2025;2025082218. doi:10.20944/preprints202508.2218.v1.
13. Gantsev ShKh, Yusupov AS. Cutaneous squamous cell carcinoma. *Practical Oncology*. 2012;13(2):80-91.
14. Dzybova E, Vardanyan K, Vasilevskaya E. Squamous cell carcinoma of the skin: clinical presentation, diagnosis, treatment, and prevention. *Klinicheskaya Dermatologiya i Venerologiya*.2015;14(4). doi:10.17116/klinderma20151444-14.
15. Sustretov VA, Kutilin DS, Maksimov AYu, et al. Genetic and epigenetic abnormalities in malignant skin tumors: basal cell carcinoma and squamous cell carcinoma. *Modern Problems of Science and Education*. 2022;(4). doi:10.17513/spno.31793.
16. Brash DE. Roles of the transcription factor p53 in keratinocyte carcinomas. *Br J Dermatol*. 2006;154(Suppl 1):8-10. doi:10.1111/j.1365-2133.2006.07230.x.
17. Kladova AYu, Kuevda DA, Kuntsevich ZhS, et al. Association of keratoacanthoma with human papillomavirus infection. *Almanac of Clinical Medicine*. 2007;15. Available from: https://cyberleninka.ru/article/n/k-assotsiatsii-keratoakantom-s-virusom-papillomy-cheloveka
18. Yumeen S, Robinson-Bostom L, Firoz EF. Solitary eruptive keratoacanthoma developing at site of COVID-19 vaccine injection. *R I Med J (2013)*. 2023;106(11):49-51.
19. Ilaria P, Nevena S, Ersilia T, et al. Generalized eruptive keratoacanthoma after Pfizer mRNA-BNT162b2 COVID-19 vaccination successfully treated with cemiplimab. *Viruses*. 2024;16(8):1260. doi:10.3390/v16081260.
20. Alloo A, Garibyan L, LeBoeuf N, et al. Photodynamic therapy for multiple eruptive keratoacanthomas associated with vemurafenib treatment for metastatic melanoma. *Arch Dermatol*. 2012;148(3):363-366. doi:10.1001/archdermatol.2011.3080.
21. Mazurenko NN. Genetic heterogeneity of cutaneous melanoma: new targets for selective therapy. *Malignant Tumors*. 2015;4(Special Issue 2):3-8. doi:10.18027/2224-5057-2015-4s2-3-8.
22. Lebhar J, Jacobs J, Rundle C, et al. Skin cancers arising within tattoos: a systematic review. *JAAD Int*. 2024;16:133-143. doi:10.1016/j.jdin.2024.03.015.
23. Parker ER, Ahlers CG, Hicks AB. A report of a keratoacanthoma-type cutaneous squamous cell carcinoma arising within a multicolored ink tattoo. *J Clin Aesthet Dermatol*. 2024;17(4):33-36.
24. Kwiek B, Schwartz RA. Keratoacanthoma (KA): an update and review. *J Am Acad Dermatol*. 2016;74(6):1220-1233. doi:10.1016/j.jaad.2015.11.033.
25. Galil-Ogly G.A., Molochkov V.A., Sergeev Yu.V. Dermatooncology. Moscow: Medicine for everyone; 2005: 870 p.