

KELOID IN A PATIENT WITH ALBINISM

Jonel May Breytenbach, FC Plast Surg
Kenneth Segwapa, FC Plast Surg

SUMMARY

Keloids are limited to humans and although the exact prevalence is unknown, they have been reported in 5%-16% of Africans. Individuals with darker pigmentation are more susceptible to keloid formation, especially Africans and Asians. According to the literature, Caucasians are least effected and there has been only a few reports of albino patients with keloids. The author identified a patient with albinism who presented with a keloid to his right ear after trauma at the Dr George Mukhari Academic Hospital in Garankuwa, South Africa. The keloid was resected and sent for pathological evaluation to determine differences in collagen formation. The specimen was compared with tissue from a similar African patient with a keloid due to trauma on the left ear. Both keloids were noticed six months after the sustained trauma.

Keywords: Keloid; Albinism; Histology

INTRODUCTION

Keloids are collagenous lesions that extend beyond the original area of injury and are the result of abnormal wound healing.¹ The etiology of keloid formation is unknown,² but it is believed to be from aberrant wound healing caused by excessive fibroblast participation and collagen deposition.³ The highest incidence of keloids has been reported to occur between the first and third decade of life⁴ and the African population is most affected, with an incidence of 6%-16%.³ Many articles report that patients with albinism are spared from developing keloids^{3,5-9} and plastic surgery text books generally assert that keloids in patients with albinism have not been reported. Only one epidemiologic publication¹⁰ has reported a keloid prevalence rate of 7.8% in people

with albinism in Kenya and Tanzania. To my knowledge, there is only one reported case of keloidalis nuchae in a patient with albinism who is from Mali.¹¹

Here we report the first case of an earlobe keloid in a patient with albinism and discuss routine histologic findings compared with an earlobe keloid from an African non-albino patient.

CASE REPORT

A 26-year-old patient with albinism presented to the plastic surgery clinic at the Dr George Mukhari Academic Hospital in Garankuwa, South Africa, with an earlobe keloid due to a traumatic injury to the ear; the patient had been stabbed with a bottle six months earlier (Figure 1). He presented with a firm, hard, non-fluctuant, nodular mass on his right ear (1.8 cm × 1.2 cm × 1.2 cm). The lesion, which extended beyond the borders of the original injury, was excised under local anesthetic. The specimen was sent for pathological examination and the patient received radiotherapy. The histological specimen was then compared with an 18-year-old African patient who developed a firm, hard, non-fluctuant, nodular keloid mass on the left ear that stemmed from an ear-piercing six months earlier (2 cm × 2 cm × 0.7 cm). As both histological specimens were diagnosed as keloids, we decided to compare histologic findings to see whether the patient with albinism had a different composition of collagen.

Both parents of the patient with albinism are black of South African descent. He has a pigmented beard and brown eyes and therefore falls under the oculo-cutaneous albinism type 3 with a defect in the TYRP1 gene, usually affecting dark-skinned people and common in black South Africans, who exhibit features of reddish-brown skin, reddish hair, and hazel or brown eyes.

Written consent was obtained from the patients to publish this case report.



FIGURE 1: Earlobe keloid on patient with oculocutaneous albinism type 3.

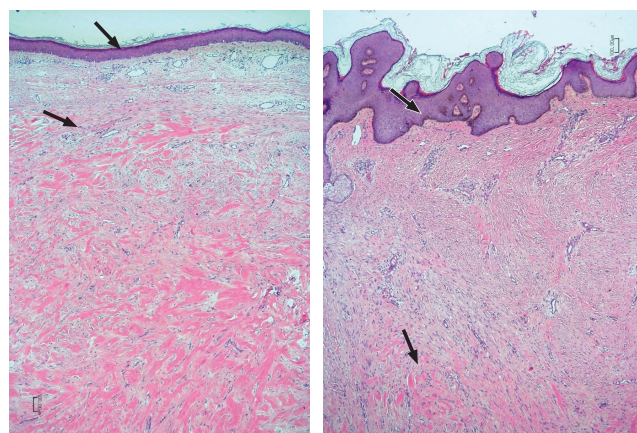
RESULTS

The results of the comparison of the histological specimens are shown in Table 1. The albino patient was Fitzpatrick skin type I and the non-albino patient was Fitzpatrick skin type VI.

Examination under microscopy revealed few differences in appearance. The keloid from the albino patient displayed an atrophic epidermis with irregular, thick, homogenous collagen fibres that was acellular and the keloidal features started mid-dermis (Figure 2A). The non-albino African control patient had normal epidermal thickness and the keloidal features of irregularly oriented collagen whorls started lower in the dermis (Figure 2B).

TABLE 1. Histological results comparing keloid specimens of the albino and non-albino patients

Fitzpatrick I Albino patient	Fitzpatrick VI African patient
Non-pigmented, atrophic epidermis	Epidermis with adnexal structures (hair follicles and sebaceous glands); epithelium of normal thickness
Broad, irregular, thick, homogenous masses of collagen that are acellular, broad coiled bundles of collagen	Proliferation of connective tissue extends in whorls and bundles up to the deepest excision margin
Focal chronic inflammatory infiltrate	Focal chronic inflammatory infiltrate
Keloidal features starting mid-dermis	Keloidal features starting lower down in the dermis



A. Thin epidermis
Keloid starting mid-dermis
B. Normal epidermis
Keloid starting lower in dermis

FIGURE 2: H&E staining of keloid from albino patient (A) and African patient (B). Magnification 4x

Due to the observed differences of the dermal structure, the specimens were stained to investigate possible variabilities in collagen types. Masson's trichrome staining selectively stains for muscle, collagen fibers, fibrin, and erythrocytes and was used here to identify collagen fibers, which stain blue (Figures 3A and B).

Picrosirius stain (Merck, Garankuwa, SA) is a connective tissue stain that is used to visualize type I and type III collagen in skin. Collagen can be visualized under normal microscopy light or under polarized light, which results in birefringence of the collagen fibers that helps to distinguish type I and type III collagen. Type I collagen shows thicker bundles with yellow-orange birefringence and type III shows green birefringence (Fig. 4).

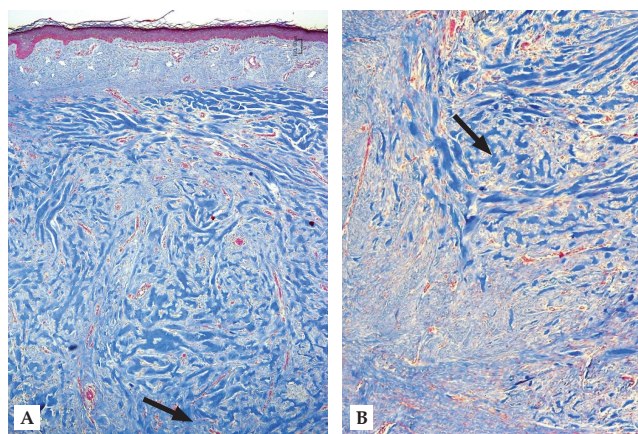


FIGURE 3: Masson's trichrome stains positive for collagen in albino patient (A) and African non-albino control patient (B). Magnification 4x.

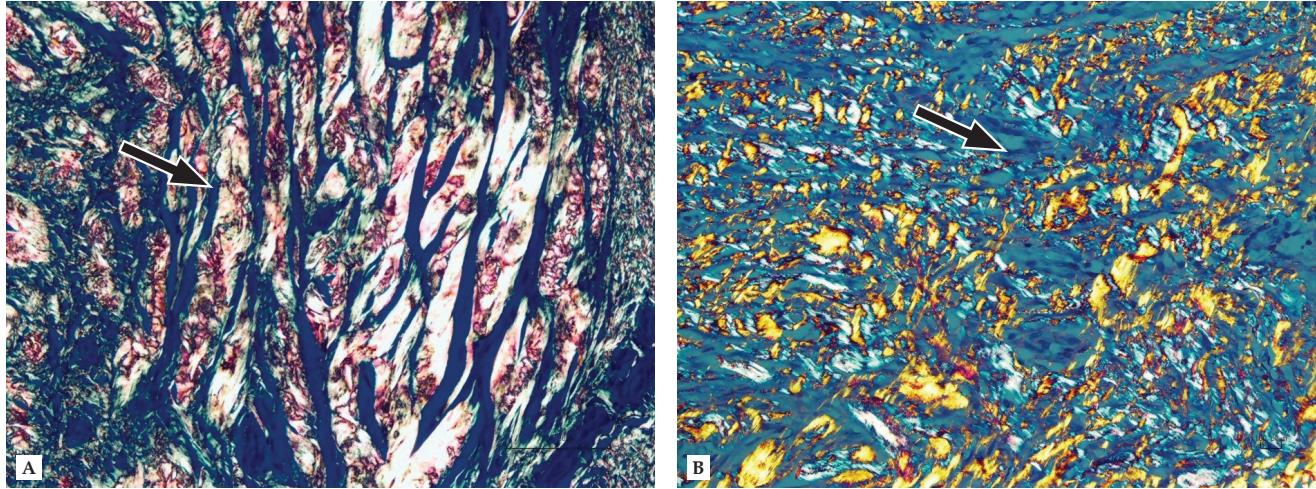


FIGURE 4: Picrosirius stain for albino patient (A) stains positive for type I (yellow-orange) and III (green) collagen, with collagen type I being more prominent. The non-albino African control patient (B) also stains positive for type I and III collagen, with somewhat lesser type III collagen. Magnification 40x.

Using H&E, Masson's trichrome (Merck, Garankuwa, SA), and picrosirius red staining techniques, no significant distinguishing features between the specimen from the patient with albinism and from the control patient were found.

DISCUSSION

This paper reports on the occurrence of an earlobe keloid in a patient with albinism and may be the first such reported case from South Africa. In a research letter, Kiprono et al. (2015)¹⁰ presented epidemiologic data of keloid patients with albinism from Kenya and Tanzania, with a keloid prevalence of 7.8% compared with 8.3% in non-albinism patients with normal skin pigment. Unfortunately, that report did not further elaborate on keloids in patients with albinism and went largely unnoticed in the current keloid literature, as did Frikh and Boui's case report.¹¹ Both descriptions failed to classify the albino type of their patients.

In this study, we had the opportunity to compare the histology of two earlobe keloids from two patients of similar age, one of whom had oculo-cutaneous albinism. There were no significant differences between the two, although the patient with albinism showed a more advanced and mature lesion: The collagen fibers were thicker and the epidermis atrophic, suggesting a more aggressive keloid growth. However, this difference could be attributed to the severity of the trauma, which could have evoked a more pronounced inflammatory response.

CONCLUSION

This report confirms that keloids do exist in patients with albinism. However, the histological appearance was not different from a similar lesion in a patient of comparable age with normal skin pigmentation. As more patients with albinism and keloids are identified, histological, genetic, and molecular analyses should be performed to study whether there are differences in keloid formation in this population.

DISCLOSURE

The author has no financial interests to declare.

GRANT INFORMATION

The author declares that no grants were involved in supporting this work.

REFERENCES

1. Andrews JP, Marttala J, Macarak E, Rosenbloom J, Uitto J. Keloids: the paradigm of skin fibrosis – pathomechanisms and treatment. *Matrix Biol.* 2016; 51:37-46.
2. Gauglitz GG, Korting HC, Pavicic T, Ruzicka T, Jeschke M. Hypertrophic scarring and keloids: pathomechanisms and current and emerging treatment strategies. *Mol Med.* 2011; 17:113-25.
3. Huang C, Murphy GF, Akaishi S, Ogawa R. Keloids and hypertrophic scars: update and future directions. *Plast Reconstr Surg Glob Open.* 2013;1:e25.

4. Bran GM, Goessler UR, Hoffman K, Riedel F, Sadick H. Keloids: current concepts of pathogenesis (review). *Int J Mol Med.* 2009; 24:283-93.
5. Edriss AS, Mesták J. Management of keloid and hypertrophic scars. *Ann Burns Fire Disasters.* 2005; 18:202-10.
6. Chike-Obi CJ, Cole PD, Brissett AE. Keloids: pathogenesis, clinical features, and management. *Semin Plast Surg.* 2009; 23:178-84.
7. Kelly PA. Update on the management of keloids. *Seminars in Cutaneous Medicine and Surgery.* 2009; 28:71-6.
8. Viera MH, Vivas AC, Berman B. Update on keloid management: clinical and basic science advances. *Adv Wound Care (New Rochelle).* 2012; 1:200-6.
9. Song C. Hypertrophic scars and keloids in surgery: current concepts. *Ann Plast Surg.* 2014;73 Suppl 1:S108-18.
10. Kiprono SK, Chaula BM, Masenga JE, Muchunu JW, Mavura DR, Moehrle M. Epidemiology of keloids in normally pigmented Africans and African people with albinism: population-based cross-sectional survey. *Br J Dermatol.* 2015; 173:852-4.
11. Frikh R, Boui M. Albinisme et folliculite fibrosante de la nuque chez un homme d'ascendance africaine. *Annals de dermatologie et de venerologie.* 2015; 142:690-691.