SUPERFICIAL SPREADING MALIGNANT MELANOMA: COMPLETE REMISSION AFTER SURGICAL EXCISION

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Summary. Superficial spreading type of malignant melanoma (SSM) is usually characterized as the most common form of cutaneous melanoma in Caucasians. The average age at diagnosis is in the fifth decade, and it tends to occur on sun-exposed areas of the skin, especially on the backs of males. Surgery to remove melanoma is the standard initial treatment. However, the surgical approach to every melanoma should be customized based on the specific details of the patient and the melanoma, location of the primary tumor, and how the resultant wound could be finally closed. A simple excision of a melanoma can usually be accomplished by sewing the skin together, resulting in a linear scar, as in our patient. We present a 72-year-old patient with SSM on his back. Eliptical excision with 2 cm margins and very good cosmetic results was performed. The examination of the slides revealed clear margins. The patient remained disease-free at 6 months follow-up.

Key words: *SSM, surgery, sun exposure, Melan A, S-100*

INTRODUCTION

Superficial spreading type of malignant melanoma (SSM) is usually characterized as the most common form of cutaneous melanoma in Caucasians. The average age at diagnosis is in the fifth decade, and it tends to occur on sun-exposed areas of the skin, especially on the backs of males. As the risk of spreading varies with tumor thickness, early SSM is more frequently cured than late nodular type of melanoma.

Melanoma is a malignant tumor of the melanocytes. Melanocytes are cells that produce the dark pigment called melanin, which is responsible for the dark color of the skin. They predominantly occur in the skin, but are also found in other parts of the body, including the bowel and the eye. Although melanoma represents only 4% of all skin cancers, it causes 77% of deaths related to skin cancer. Surgery to remove melanoma is the standard initial treatment. It is necessary to remove not only the tumor but also some additional normal tissue around it, with the purpose to reduce the risk that any cancer remains. The width and depth of the surrounding skin to be removed depends on the thickness of the primary melanoma and how deeply it has invaded the skin. When the tumor tissue is very thin, the biopsy may remove all the cancerous tissue and no additional surgery may be necessary. In some other clinical cases, following the initial biopsy, every melanoma undergoes possible re-excision to remove the skin around the original site of the tumor in order to ensure complete removal and reduce the risk of recurrence of the tumor at that site. The re-excision margin depends on the thickness of the primary melanoma, with increasing margins for increasing categories of thickness, from 5 to 9 millimeters on either side of the scar for melanoma in situ to potentially two centimeters on either side for melanoma exceeding two millimeters in thickness.

However, it is important to note that the surgical approach to every melanoma should be customized based on the specific details of the patient and the melanoma, location of the primary tumor, and how the resultant wound could be finally closed. A simple excision of a melanoma can usually be accomplished by sewing the skin together, resulting in a linear scar, as in our patient. In most instances, wide excision alone can be performed under local anesthesia in the physician's office.

We present a 72-year-old patient with SSM on his back. Eliptical excision with 2 cm margins and very good cosmetic results was performed. The examination of the slides revealed clear margins. The patient remains disease-free at 6 months follow-up.

ANAMNESIS

A 72-year-old patient presented to the Dermatology Polyclinic of University Hospital Lozenetz, "Saint Kliment Ohridski University" for evaluation of a dark macule with duration of several months (Fig. 1a). The lesion occurred soon after extreme unprotected sun exposure for a week during his regular summer holidays. Physical examination revealed a dark macule with irregular borders and pigmentation on the back, with signs of regression on one side and a small ulceration (Fig. 1a). The routine laboratory investigations were within the normal range. Abdominal ultrasound and pulmonary X-ray did not reveal any abnormalities. No enlarged lymph nodes were detected. Eliptical excision with 2 cm margins was performed (Figs. 1b-1c) and the examination of the slides revealed clear margins. The patient remained disease-free at 6 months follow-up.

Histopathology of the lesion (Figs. 2a-2b) was performed and provided the diagnosis of SSM, Clark level 3, tumor thickness 0.6 mm, pT1a. Melan-A and S100 were positive in the cells both in the junctional component and in the superficial dermis, confirming the melanocytic nature of the lesion. Ki-67 was difficult to interpret due to the presence of inflammatory cells and pigment in the dermis, but apparently shows a moderate proliferation rate.

Fig. 1. (a, b, c). 1a. A dark macule with irregular borders and pigmentation on the back, with signs of regression on one side and a small ulceration. 1b. Eliptical excision of the lesion. 1c. A linear scar after excision of the melanoma

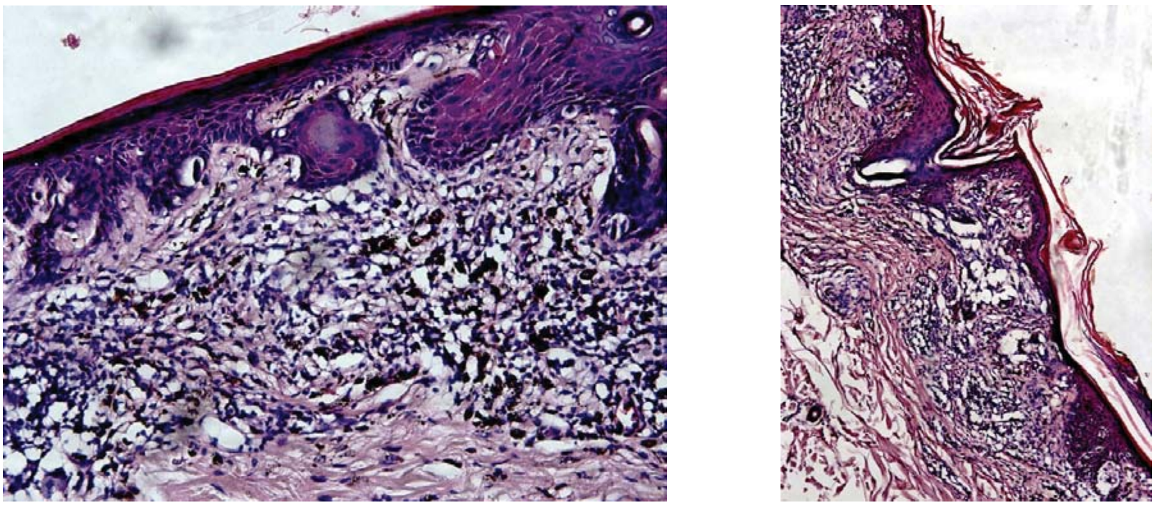

Fig. 2. (a, b). Histopathology of the lesion

DISCUSSION

The incidence of cutaneous melanoma is increasing worldwide. The higher rates are considered to be due to the increased detection of early lesions resulting from improved awareness, but also from increased acute sun exposure and use of sunbeds [1]. Epidemiological data provides a strong link of intermittent sunlight exposure to the development of melanoma [2]. However, besides the scientific and clinical advances, many aspects of melanoma pathophysiology and etiology remain unknown.

SSM is the most common type of cutaneous melanoma with an estimate of around 70% of all patients with melanomas. It is more common in older men and on the sun-exposed areas of the trunk [3]. Multiple studies found a strong association of SSM development with acute sun exposure in a dose-dependent manner, sunburns and the number of nevi [3]. Wide excision is the treatment of choice for patients with stage II and III of the disease, with high curative rates and good prognosis [4].

The variation in risk factors for different types of melanomas could be explained by the hypothesis that melanomas arise through distinct causal pathways, supported by recent research [5]. One is the pathway induced by chronic sun exposure and the other is the pathway dependent on the nevus-prone phenotype of the host with the presence of contiguous neval remnants. Histologically, these divergent origins are presented as dermal elastosis in adjacent skin in lentigo maligna melanoma (LMM) and neval remnants contiguous with the tumor in SSM, respectively.

Ultraviolet (UV) light, particularly UVB, is believed to be the predominant carcinogen in sunlight related to melanoma development [6]. UVB reacts with DNA of melanocytes and keratinocytes leading to the formation of individual DNA photoproducts and distortion of the DNA helix at dipyrimidine sites. These UV-induced lesions are usually removed and repaired by the nucleotide excision repair (NER) pathway, thus restoring the integrity of the genome. When left unrepaired, the distorted helix leads to mutations during DNA replication. The known mutation spectrum in melanoma demonstrates that the somatic mutations are mostly fingerprint mutations from UV-induced DNA damage [7].

When compared to other cell types, melanocytes have a lower NER capacity [8]. Additionally, keratinocytes are removed by apoptosis when severely damaged by UV radiation, while melanocytes with similar damages are retained with a potential for further mutations [2]. Wang et al. showed that melanin in melanocytes binds to DNA impeding damage recognition and repair by NER enzymes [8]. These factors lead to the accumulation of UV-induced DNA mutations that may result in avoidance of cell death and melanomagenesis after exceeding a set mutational threshold. The biological mechanism of transition from dysplastic cells to melanoma cells and the mutational threshold are yet to be elucidated.

Based on the current knowledge of SSM and melanoma mechanisms in general, we suggest that the ‘acute’ presentation of SSM in our and in other similar cases most probably results from exceeding the threshold of DNA repair capacity of the melanocytes in the neval remnants due to the intermittent recreational sun exposure.

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