Case 3: A Man with a Facial Pigmented Patch

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CASE SUMMARY

History

A 74-year-old Caucasian male presented with a several years history of a pigmented patch on the left cheek. He noticed a slow increase in size of the lesion for one and a half years. The patch was asymptomatic, with no itching or bleeding. His past health was unremarkable. There was no family history of skin cancer.

Physical examination

There was a 2 x 4 cm asymmetrical patch at left cheek, with irregular edges and variegated pigmentation (Figure 1). No papular component was present. The lesion was on a background of actinic damage. No cervical lymphadenopathy was detected.

Differential diagnoses

The differential diagnoses included lentigo maligna, solar lentigo and flat seborrheic keratosis.

Skin biopsy

An incisional biopsy was performed. The biopsy showed marked contiguous melanocytic proliferation at the dermo-epidermal junction. A thin epidermis was found. Marked contiguous melanocytic proliferation was noted in the dermo-epidermal junction. The melanocytes exhibited cytological atypia with prominent nucleoli. Focal pagetoid proliferation was present although neither stromal invasion nor mitosis was noted in the dermis. There was a moderate amount of mononuclear inflammatory cells in the superficial dermis. Solar elastosis was also present.

Management

The absence of papular component clinically and absence of dermal invasion histologically suggest a diagnosis of lentigo maligna. The patient was referred to the plastic surgeons for excision of the skin lesion. The specimen obtained could then be examined pathologically to rule out an invasive component.

REVIEW ON LENTIGO MALIGNA

Lentigo maligna was first described by Hutchinson in 1890 as 'Hutchinson's melanotic freckle'. Its annual

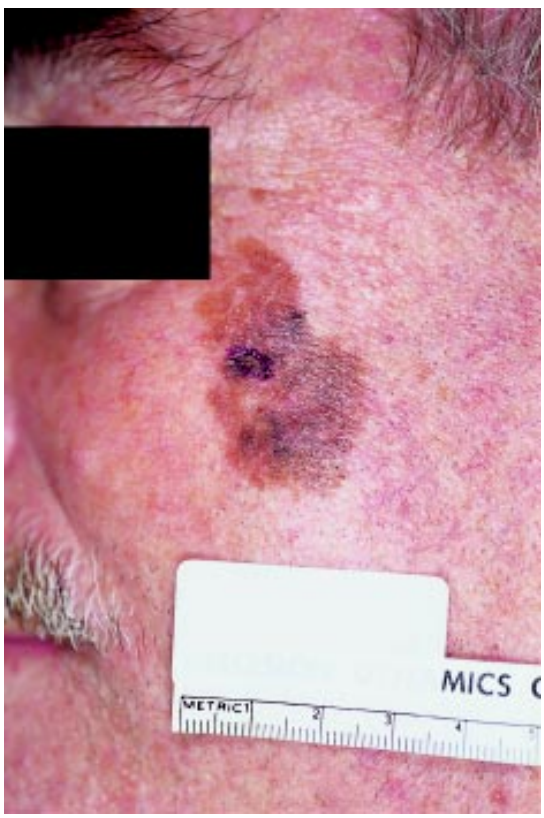

Figure 1: Variegated pigmented patch on left cheek

incidence is 1.3:100,000 in Australia. According to the literature, the lentigo maligna subtype represents 4 to 15% of all malignant melanomas. Long-term cumulative ultraviolet radiation remains the most widely accepted risk factor.[1]

Clinical features

Lentigo maligna predominantly affects Caucasian persons. Most patients are older than 40 years of age, with a peak onset in the 7th to 8th decades of life. It occurs mainly on sun-exposed areas of the head and neck, especially on the cheeks. It usually presents as an ill-defined pigmented patch with variation in colour. The patient often gives a history of slow, gradual enlargement of the lesion. It usually occurs on a background of actinic damage and solar lentigines.[1]

However about 21 cases of amelanotic lentigo maligna have been reported, and 11 affect the face.[2] They usually appear as depigmented or erythematous lesions and are difficult to diagnose clinically. Differential diagnoses include Paget's disease, superficial basal cell carcinoma, Bowen's disease and actinic keratosis. Histological examination is therefore essential. Agenesis of melanosomes or abnormal melanogenesis in melanocytes are possible aetiologies.[2]

Histopathology

Typical histological features[1] include:

1. epidermal atrophy and effacement of the rete ridges
2. atypical melanocytes along the basal layer, in solitary units and small nests
3. periadnexal extension of atypical melanocytes
4. multinucleated melanocytes
5. dermal infiltrate of lymphocytes and melanophages
6. solar elastosis

Immunohistochemical techniques are sometimes helpful. HMB-45 monoclonal antibody can distinguish lentigo maligna from pigmented actinic keratosis in difficult cases. HMB-45 stain delineates the boundary of lentigo maligna histopathologically, enabling the pathologist to decide if lesion has been completely excised with clear margins. Also it can highlight an invasive dermal component which indicates vertical growth, signifying lentigo maligna melanoma. HMB-45 is quite specific for malignant melanoma although not 100% certain. It can also stain some junctional nevi, junctional components of compound nevi, most dysplastic and Spitz nevi. On the other hand it does not stain desmoplastic melanoma.[1]

Prognosis

There is no longitudinal, prospective study on progression to lentigo maligna melanoma from lentigo maligna. The estimated lifetime risk of lentigo maligna melanoma (LMM) is 5%. If nodule formation occurs, the lesion is likely to have entered the invasive phase. If lentigo maligna melanoma is diagnosed, the prognosis is the same as other types of melanoma i.e. dependent on tumour thickness.[1]

Management

A) Surgical Excision

1) Conventional Surgery

Complete excision offers the greatest likelihood of cure. A 91% cure rate and a recurrence rate of 9% are estimated for conventional surgery.[1] The margin rule [0.5cm for melanoma-in-situ, 1cm for thin melanoma <1mm][3] cannot be easily applied to lentigo maligna although Wood's light can be used to highlight the clinical border.

2) Mohs Micrographic Surgery

The potential benefits of tissue conservation and higher cure rate of Mohs surgery need to be balanced against the difficulty in interpreting frozen sections. One modification is to perform serial en-face frozen sections till negative margins are seen, followed by rush permanent sections to confirm, plus adjunctive immunoperoxidase staining HMB-45.[4] Cohen reported a 97% cure rate in 45 patients with a median follow-up of 58 months.[5]

Another modification known as the 'Square' procedure was proposed by Johnson.[6] A surgical margin of 0.5-1 cm in geometric configuration with angled corners of the lines, in the form of a square or rectangle is marked (1 of figure 2). A peripheral strip of tissue (2-4mm wide) is removed by a two-bladed knife down to fat tissue, for vertical permanent section (2 of figure 2). Then further excision of margin(s) is made till all margins are clear of tumour cells (3, 4 of figure 2). The central island of skin is excised and reconstruction of the defect undertaken.[6]

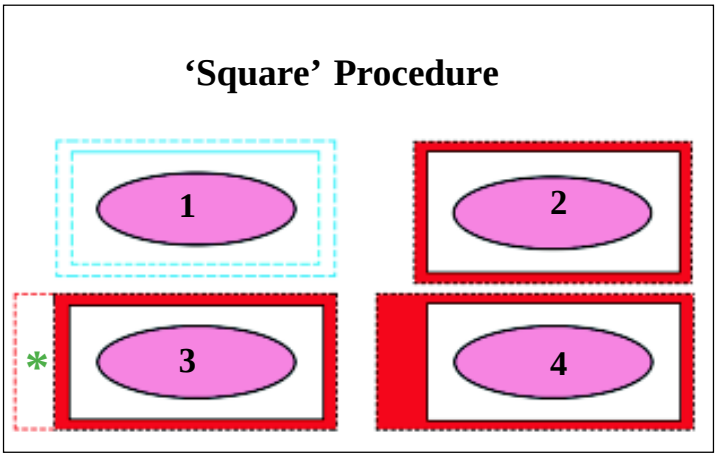

Figure 2: The 'Square' procedure for lentigo maligna[6]

B) Destructive Methods

While complete excision offers the greatest hope of cure, destructive forms of treatment are often considered for lesions or patients not suitable for surgery. Their disadvantages include the following: failure to treat deep periadnexal melanocytes, inability to detect atypical melanocytes beyond the clinical margin and the lack of a specimen to detect invasive melanoma.[1] The use of cryosurgery, radiotherapy and laser are discussed below:

1) Cryosurgery

The use of cryosurgery depends on the selective destructive of melanocytes at -4°C to -7°C and the resistance of squamous cells up to -20°C. The recurrence rate varies from 0 to 36%, and it is more effective in mucosal lesions.[1] A double freeze-thaw cycles with a 5mm margin, each of 30 sec, plus the use of thermocouples (temperature -20 to -50°C) is often adopted. If pigmentation occurs at a previously treated site, a biopsy to differentiate between recurrence from reactive lentiginous pigmentation is often necessary.[1]

2) Radiotherapy

Conventional fractionated radiation therapy with superficial X-rays is now used in order to treat deeper atypical melanocytes. Cure rates of 86-91% have been reported.[1] Focal lentigo maligna melanoma may be missed.

3) Laser

The efficacy of laser treatment of melanocytic lesions with malignant potential has not been evaluated. Case reports of failure with Argon and Q-switched ruby laser have been reported.[7,8] It has been postulated that atypical melanocytes may be resistant to the standard fluences used for cosmetic procedures.[8]

Conclusion

Lentigo maligna should be regarded as a melanoma-in-situ and complete surgical excision remains the treatment of choice. Tissue conservation and margin control procedures are being investigated but require pathological expertise. Patients should be advised to return if pigmentation recurs regardless of the treatment modality used.

Learning points:

Patient with lentigo maligna should be advised to return if pigmentation recurs regardless of the treatment modality given.

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