Squamous Cell Carcinoma

Essentially squamous cell carcinoma is caused by sun damage in genetically susceptible people. There are however, a range of other conditions which may result in the development of multiple squamous cell carcinomata, including genetic conditions such as scleroderma pigmentosa (which is a DNA breakage), arsenic poisoning, radiation, chronic burns scars and other rarer conditions. Squamous cell carcinoma develops in the squamous cell layer of the epidermis, but its progression is more subtle and is often unrecognized and illogically classified in the current environment. As Bernard Ackerman from the USA very aptly points out, solar keratosis is an in situ squamous cell carcinoma. As with all cancers, the body is constantly sending immune cells throughout the tissues to hunt down and eradicate any mutancy, and as a result, many early carcinomas are eradicated before they become problematic. Solar keratosis falls into this category and may resolve spontaneously in a percentage of patients.

Nevertheless, the histology of this condition shows bizarre shaped basal cells which contain many nuclei (this is abnormal), bizarre shaped squamous cells, a recognizable prickly cell layer and clear cell layer, and a keratinocyte layer that contains bizarre and sick-looking cells, and instead of the skin being sealed bya layer of dead keratinocytes, this skin of solar keratosis is piled high with sick keratinocytes that have visible nuclei in them and refuse to fall off. The pathology is contained entirely within the epidermis, and the cell layers are recognizable.

The next progression towards squamous cell carcinoma is called Bowenoid keratosis. This is one step further on from solar keratosis, and all of the cell layers are bizarre and unrecognizable and the bizarre cells extend down along the hair follicles, sebaceous glands and sweat glands into the dermis, although they are still separated from the dermis by the [peri-potential basal cell layer] which normally sits under the basal cells. There is a [ramina] membrane thus separating the epidermis from the dermis.

The next progression of squamous cell carcinoma is when the tumour transgresses this barrier, and penetrates into the dermis. This is called early invasive squamous cell carcinoma. Clinically, the appearance of these tumours change. Both solar keratosis and Bowenoid keratosis appear as a roughened, scaly area of skin, which may occasionally bleed when scratched. The early invasive squamous cell carcinoma takes on a slightly warty or crusty appearance and feels thicker. As the progression into the dermis continues, and invasive squamous cell carcinoma becomes more established, the tumour becomes very thick, may contain a central core of dead tissue and paradoxically may appear localized, albeit over a large area.

If left untreated, the squamous cell carcinoma can become enormously thickened, crusty, wart-like, with or without tissue necrosis. Squamous epithelium is not only found in the epidermis of the skin, but it is also found covering the mucosal surface of the lips, inside the mouth, the tongue and areas of the aerodigestive tract, including sinuses within the skull (which are there to reduce the density of the bone to lighten the skull and therefore lighten the weight of the head, which needs to be supported by the neck musculature).

Squamous cell carcinoma can also involve these tissues, and can therefore be found in the mouth, throat and facial sinuses.

The clinical behaviour of squamous cell carcinoma depends on the site of the tumour and the thickness of the tumour. Tumours arising from the tongue, aerodigestive tract and sinuses tend to be much more aggressive and should really be treated in a specialist unit with a multi-disciplinary team approach that involves not only an appropriate surgeon, but also an oncologist and radiotherapist, along with their appropriate support teams. To this end, this kind of tumour presenting in a private setting is usually referred on to a multi-disciplinary team. Squamous cell carcinoma of the skin however, tends to be fairly localized in most cases and is eminently treatable in the private clinic setting. Only in extremely advanced cases, is there a very small percentage chance of metastatic disease.

There are three forms of treatment for squamous cell carcinoma. Surgery remains the mainstay, with the highest cure rates, and is the most definitive form of therapy. Laser therapy has a role to play in some instances, and chemotherapy has a role to play [n some instances, but both the latter modalities have a lower cure rate and are often not definitive. Laser therapy may be undertaken either using a standard CO2 laser, a pulsed dye laser or new PDT laser which involves photosensitization of tissues, with a chemotherapeutic agent prior to applying the laser beam.

The chemotherapeutic treatment of squamous cell carcinoma involves applying creams to the tumour that will either kill the cancer cells, or stimulate an immune response to the cancer cells. Efudex (5-fluoro-uracil) is a chemotherapeutic agent that produces very aggressive blistering which erodes away the tumor. The wound subsequently heals with minimal scarring.

Imiquimod (Aldara cream) is another agent which is currently being trialled for the treatment of squamous cell carcinoma. This is an immune stimulating drug, and again produces blistering over the tumour when applied to the affected area. When the blistering heals, the skin tumour is eradicated. This is successful in about 80% of people who are treated for superficial forms of squamous cell carcinoma. This treatment has PBS authority only for the treatment of genital warts, and if it is being used for the treatment of squamous cell carcinoma, it is not subsidized by the government, and a typical treatment will cost in excess of $500 purely for the cream alone. In addition, treatment with Imiquimod will take up to six to 12 weeks, depending on the regime used. (Different regimes are prescribed by different practitioners and as it is still in the trial phase, no recommended preferred approach has been decided upon, although from my reading of the literature, the most successful regime to date is to apply the cream twice a day for three consecutive days for 6 consecutive weeks). The only disadvantage of this therapy is that patient compliance is not so good, as the blistering can be quite severe.

It is not recommended that any skin cancer be treated conservatively without first obtaining a tissue biopsy of the area in order to make an accurate diagnosis, and once treatment has been undertaken it is sometimes worthwhile repeating the biopsy to prove cure.

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