British Journal of Plastic Surgery (1993), **46**, 156–157

BRITISH JOURNAL OF PLASTIC SURGERY

Perineural infiltration in basal cell carcinomas

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SUMMARY. **4376 basal and squamous cell carcinomas were dealt with over a period of 100 months. 76.6% of these were basal cell carcinomas. Perineural spread was noted in 0.178% of basal cell lesions and in all examples was found in recurrent tumours, which were predominantly in the preauricular and malar areas in this series.**

Review of cases

From January 1981 to April 30, 1989, 4376 basal and squamous cell carcinomas were treated, with an average incidence of 43.76 per month. 76.6% of these were basal cell carcinomas. Out of the 3355 basal cell lesions 6 showed perineural spread, an incidence of 0.178%. Of these six, five were in female patients and the age range was 54–73 with an average of 65.4. All the tumours with perineural spread were recurrent, with four of the six having previously been treated by radiotherapy. The Table shows the sites of the lesions. Three patients had complained of burning or stinging facial pain. Four patients had petrosectomy with excision of the tumour and the defects reconstructed with local or distant flaps. There have been two deaths, both 3 years after petrosectomy and flap repair.

Discussion

It has been observed that some skin cancers have invisible and impalpable extensions which can invade deep tissues including dermis, fascial planes, periosteum, perichondrium, embryologic fusion planes, nerve sheaths, lymphatic vessels and blood vessels[1].

Cruveilheir first reported intraneural spread of tumour cells in 1835. Such spread in skin cancers of the head and neck area was extensively studied by Ballantyne *et al.*[2]. They reported a series of eighty cases of intraneural spread, most of which were with squamous cell carcinomas, although there were four examples of basal cell carcinomas[2]. Subsequently 16 other basal cell carcinomas with dissemination in nerves have been reported[3–6].

The mechanism of intraneural spread has been the subject of some debate. Shattock claimed that spread was via perineural lymphatics[7], a view supported by Ballantyne *et al.*[2], but an experimental study by Larson *et al.*[8] was unable to demonstrate a perineural lymphatic network using a dye injection technique. They concluded from clinical and animal observations that tumour cells grew along tissue planes of the perineurium[8].

Previous reports of perineural invasion have stressed that patients present with symptoms of pain variously described as burning, stinging or shooting[2] and like ants crawling in the skin[9]. All investigators report a latent period of up to six years between the onset of the nerve symptoms and frank recurrence, suggesting a slow rate of spread by tumour cells along perineural tissue planes. The tumour spreads proximally along the nerve sheath, with axial extension exceeding concentric growth. Continued concentric growth results in enlargement of the nerve, focal areas of pressure, and degeneration of the nerve fibre. This growth can cause bony erosion in confined areas such as foramina and canals and also results in the pain becoming more severe and unremitting[6].

An aid to diagnosing intraneural spread radiologically is detection of changes in the small foramina through which the cranial nerves pass[3]. Changes were noted in 44% of cases using selective views and tomography. Since then computerised tomography has been introduced, which may further improve the diagnosis of cranial nerve invasion.

Treatment of perineural extension of tumours should, when practical, involve resection of all invaded tissue. Serial frozen section histology of the nerve will help to determine the limits of invasion but will fail to recognise skip lesions. A compromise is surgical excision carried out to practical limits imposed by the anatomy, followed by immediate full dose, postoperative radiotherapy to the whole distribution of the nerve[9].

In our series, the initial clinical and histological examination did not suggest the subsequent aggressive nature of the tumour. Our incidence of perineural spread is much lower than the 1% (17 of 1686 cases) quoted by Mohs and Lathrop[1]. This may be because of our larger number of cases or the fact that conventional pathology uses vertical sections, whereas the Mohs' technique involves examination of horizontally cut sections. Small areas of perineural invasion are more likely to be identified in the latter. Whereas Hanke *et al.*[6] found that perineural spread with basal cell carcinomas was more common in men, in our series five out of the six patients were women.

In conclusion, perineural infiltration with basal cell carcinomas is rare and is more commonly picked up retrospectively on microscopic examination of usually recurrent tumours (16 of 17 in Mohs', 9 of 10 in Hanke's and 4 of 4 in Ballantyne's series were recurrent

Table Characteristics of patients with recurrent basal cell carcinomas and perineural invasion

No.	*Born*	*Primary*	*First diagnosis*	*Radiotherapy*	*Petrosectomy*	*Perineural*	*Reconstruction*
1	1935	Cheek	1955	1979	1982	1982	Scalp flap
2*	1921	Preauricular	1968	1974	1981	1981	TFL flap
3*	1924	Malar	1948	1979	1983	1982	Scalp flap
4	1922	Ear	1977	No	1985	1985	Radial free flap
5	1930	Postauricular	1982	No	No	1987	Scalp flap
6	1916	Temple	1974	74, 82, 84	No	1989	Graft

* Deceased

tumours). It may present with paraesthesia or motor impairment and symptoms may precede the clinical recognition of the recurrence by many months. The preauricular and cheek areas are the most common locations[6]. Treatment is difficult, as in many the diagnosis is delayed. Mohs' approach has been an effective method for early recognition and treatment[10] but in more advanced cases, major surgical resection and reconstruction followed by radiotherapy can give a successful outcome. In cases of intracranial involvement, radiotherapy alone may be indicated but the prognosis is usually poor. It is apparent that the chances of cure are enhanced by an early diagnosis and therefore a high index of suspicion is required when reviewing patients with recurrent basal cell carcinomas.

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Paper received 21 April 1992.
Accepted 25 September 1992, after revision.