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Lung scar cancer—a reappraisal

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SUMMARY A retrospective clinicopathological study of 100 necropsy cases of lung carcinoma revealed three scar cancers. The scarring in a further 11 probably occurred secondary to the tumour. The premise that lung scars initiate malignancy is questioned.

Peripheral lung tumours occasionally arise in association with focal, but more usually diffuse scarring. A causal relation was suggested by the finding during routine necropsies of unsuspected early malignancy adjacent to scars.[1] Although the pathogenesis is unclear, possible mechanisms include uncontrolled epithelial hyperplasia in relation to bland fibrosis or the concentration of carcinogens by scars.[2] Carcinogenic properties of some fibrogenic agents such as asbestos or of scar components, namely elastic[3] and cholesterol[4] are also incriminated. Scarring is seen in up to 40% of surgical resections and 10% of necropsies.[5] Forty-five per cent of adenocarcinomas in men[6] and 25% of all lung tumours[7] are scar-related. Significantly scar cancers are becoming more common in America[8] while smoking-induced tumours there are expected to decline.[9]

This paper records the frequency of scar cancer in a population generally free of fibrogenic hazards though prone to one form of lung scarring, tuberculosis.

Material and methods

One hundred consecutive necropsies involving primary lung carcinoma were reviewed. The patients' hospital records, necropsy protocols and all of the tissue sections were studied. Clinical data, particularly residence, occupation, smoking habit and previous respiratory illness was noted. The pathological features included tumour location and the presence of other diseases such as chronic bronchitis, emphysema and scarring. In addition to haematoxylin and eosin (H & E) stains, special stains were performed where appropriate, especially for elastic and fibrous tissue.

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Results

SCAR CANCERS

Two adenocarcinomas (Fig. 1) and one squamous carcinoma were accepted as true scar cancers. They had arisen at the lung periphery adjacent to macroscopically obvious scars. In two cases tumour was discovered only after histological examination. Two of the scars were tuberculous and one non-specific. No pathological evidence of chronic obstructive airways disease was seen. One patient had smoked cigarettes but ceased 20 years previously. All were urban dwellers, free of occupational exposure. None had a history or clinical features of any other respiratory complaint.

CANCER WITH SCARRING

Eleven cases formed this group. Each had grossly obvious carcinoma. Four tumours were peripheral and contained small areas of macroscopic scarring. Seven (five central, two peripheral) had scar tissue observed only after histological evaluation. Within each tumour there was fibrosis and elastosis and also foci of dense hyaline scarring (Fig. 2). Each showed elastosis in blood vessels and alveoli within and adjacent to tumour. Where these structures were being enveloped and distorted by tumour the impression of stromal elastosis was enhanced (Fig. 3). Anthracotic pigment was usually perivascular but after tissue distortion appeared to lie in tumour stroma closely applied to elastosis and fibrosis (Fig. 4). These areas were frequently so compact that it was only after careful study of sections stained for elastic that the components of the scar could be identified (Fig. 5). We also frequently observed small areas of interstitial lung fibrosis immediately adjacent to tumour. After compression and collapse this resembled "bronchiolisation" and "adenomatosis."

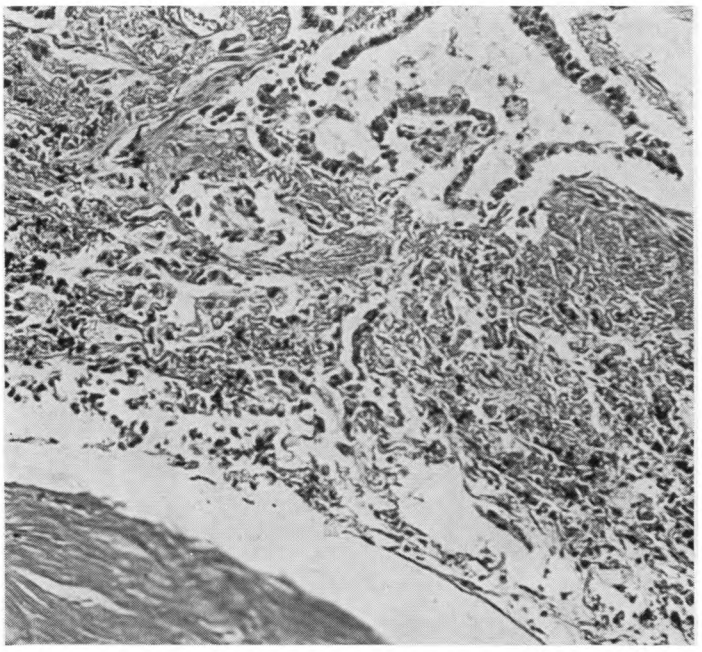


Fig. 1 *True scar cancer: adenocarcinoma arising at the edge of a healed and fibrotic tuberculous lesion. Haematoxylin and eosin × 100.*

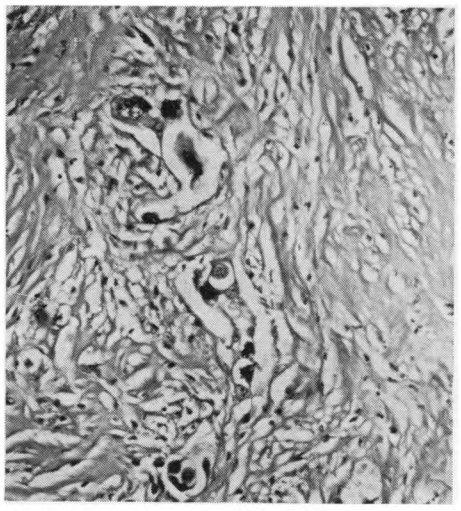


Fig. 2 *Malignant tumour within dense fibrous tissue: not true scar cancer. Haematoxylin and eosin × 100.*

Seven patients in this group were smokers. Ten were judged clinically or pathologically (or by both) to have chronic airways disease. Six tumours were squamous carcinomas, three were oat cell carcinomas and two were adenocarcinomas.

Discussion

The higher incidence of lung scar cancer reported by others[3 5–8] reflects differences in the interpretation of histological appearances. The assumption that fibrosis, elastosis, and anthracosis represents pre-existing scarring is frequently erroneous. Similar features were seen in 11% of our cases including central tumours and may occur as a secondary phenomenon. In these instances we suggest the sequence of events to be:

(a) Invasive tumour and associated inflammatory response causes interstitial fibrosis in adjacent lung parenchyma; desmoplasia occurs as in any other tumour; compaction results in focal hyaline scars;

(b) Stromal desmoplasia may proceed to elastosis but tumour-induced vascular and alveolar elastosis, after compression, contributes significantly to the elastic content;

(c) Anthracotic pigment in alveoli and lymphatics is trapped in the fibrosed, elastotic areas.

Observer differences also exist in the evaluation of malignancy arising at the margin of lung infarcts. Fifty-six per cent of scar cancers are deemed to occur in this way.[8] Where we observed viable tumour surrounding a necrotic area, we concluded it was the result of simple ischaemia within the tumour.

Our views on the evolution and role of scarring, especially elastosis, contrasts with those who con-

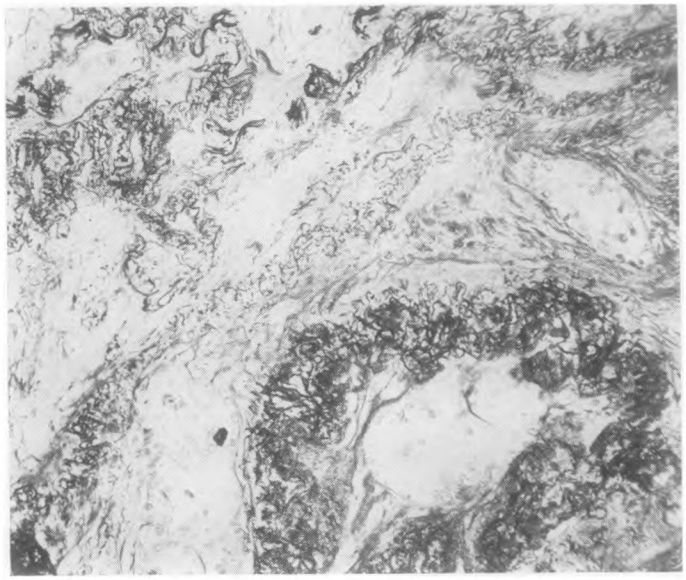


Fig. 3 *Secondary scarring in an adenocarcinoma. Vascular and "stromal" elastic is conspicuous. Verhoeff* × *100.*

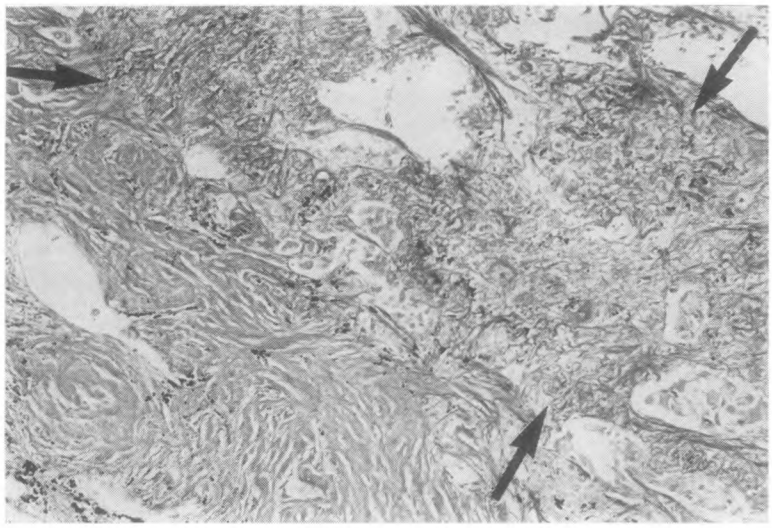


Fig. 4 *Compressed masses of elastic (between arrows) closely apposed to hyaline fibrous tissue and anthracotic pigment. Interpreted as secondary scar within an adenocarcinoma. Gomori's aldehyde fuschin* × *100.*

sider it premalignant. Similar focal scars are not present or observed in all lung tumours, but this cannot be offered as proof of a cause and effect relation in a particular instance. Although most tumours exhibiting scarring are adenocarcinomas, this does not mean the scarring preceded their development. Mesenteric deposits of ileal carcinoids cause such severe vascular elastosis that bowel infarction results.[10] Focal and diffuse elastosis with collapse occurs in most breast carcinomas as a tissue response to the infiltrative lesion.[11] It is likely that any tumour but especially adenocarcinomas cause scarring in a non-specific way.

We believe most of the tumours we studied were caused by agents other than scars. Eighty-seven per cent of our patients had chronic obstructive airways disease, and 62% smoked cigarettes until their deaths. Air pollution in Dublin is similar to that of industrialised England.[12] Moreover, the view that malignancy occurs in pre-existing lung scars irrespective of smoking habit[13] is unacceptable.

Lung scar cancer cannot be diagnosed merely

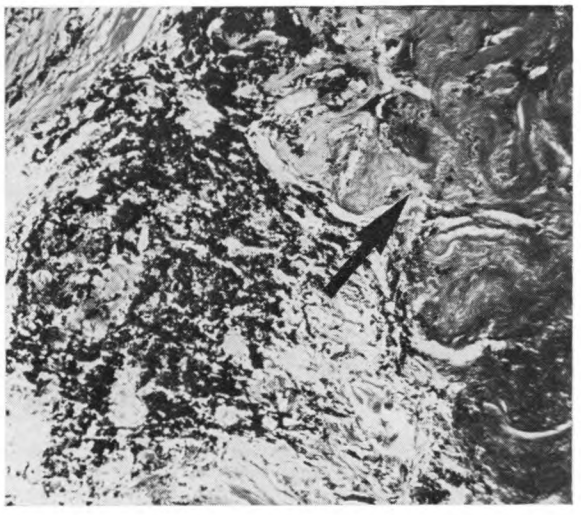


Fig. 5 *Secondary scarring within a tumour; although the components are intimately related, the elastic is seen to be derived from a sclerosed vessel (arrowed). Verhoeff × 100.*

because microscopic scarring is seen within a tumour. Carcinoma originating at the edge of a scar, especially one large enough to be seen naked eye, is likely to represent this entity. As more patients survive serious chest diseases, notably infections, it is probable that pulmonary scarring and subsequent malignancy will increase. Our figure of 3% represents a more accurate assessment of the incidence now, if criteria are critically applied.

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