LETTERS TO THE EDITOR

Long term survival after pulmonary resection for small cell carcinoma of the lung

In the retrospective review by Drs U S Prasad and others (October 1989;44:784–7) 97 patients underwent a surgical resection for small cell lung cancer (SCLC). Staging procedure consisted of clinical examination and histological examination. The cumulative five year survival was 17% for all small cell lung cancer, and 35% and 23% for stage I and II small cell lung cancer respectively. Limited stage disease was treated by surgery alone. For these patients distant metastases accounted for relapses. The authors concluded that surgery offers survival benefit in limited stage disease. Many phase II studies of surgical management in small cell lung cancer have shown similar results (for review see ref 1).

Recent studies suggest that metastatic disease is underevaluated in small cell lung cancer. By means of magnetic resonance imaging it has been shown that half of all cases of so called limited stage disease in small cell lung cancer according to routine staging procedures have bone marrow disease.[2] In our institution we have observed in many instances early metastatic relapses in patients with small cell lung cancer after surgery alone (figure). Thus chemotherapy remains the first line treatment for small cell lung cancer because of early metastatic spread and we prefer to avoid lung cancer surgery before histological classification.

In phase II studies of surgery for limited stage disease long term survival may be due only to a selection bias of patients with good prognosis. To assess the survival benefit obtained by surgery phase III trials comparing chemotherapy with and without surgery are needed.[1] Moreover, neoadjuvant chemotherapy in small cell lung cancer may treat the microscopic metastatic disease. Surgery following neoadjuvant chemotherapy may resect chemoresistant tumour remainders and it offers the opportunity to study the biology of small cell lung cancer.[3]

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1 Klastersky J. Therapy of small cell lung cancer: anything new? *Eur J Cancer Clin Oncol* 1988; 24:107–12.
2 Carney DN, Redmond O, Harford P, Stack J, Ennis J. Bone marrow involvement by small cell lung cancer using magnetic resonance imaging. In: Joss RA, Brunner KW, eds. *Proceedings of the Fifth World Conference on Lung Cancer*. Interlaken: International Association for the Study of Lung Cancer, 1988:27.
3 Pujol JL, Simony J, Laurent JC, *et al*. Phenotypic heterogeneity studied by immunohistochemistry and aneuploidy in non-small cell lung cancers. *Cancer Res* 1989;49:2797–802.

Pulmonary haemorrhage in Henoch-Schönlein purpura

Drs H S Markus and J V Clark (June 1989;44: 525–6) describe a case of Henoch-Schönlein purpura complicated by fatal pulmonary haemorrhage. The clinical features, histological findings, and absence of immune deposits in the glomerular lesions are more in keeping with a diagnosis of microscopic polyarteritis. Immunoglobulins with or without complement C3 are invariably present in the mesangium in Henoch-Schönlein purpura.[1] Pulmonary haemorrhage is a well recognised complication of microscopic polyarteritis, occurring in 29% of patients in the original series.[2]

The classification of the systemic vasculitides is confusing. Many patients with cutaneous small vessel vasculitis and deep organ lesions are classified as having Henoch-Schönlein purpura. Cream's original series of Henoch-Schönlein purpura in adults contains many patients who would now be classified as having microscopic polyarteritis.[3] The distinction between the two is of practical importance. There is growing evidence that substantial recovery of renal function can be obtained in patients with microscopic polyarteritis with the use of aggressive immunosuppressive treatment.[4] The discovery of antineutrophil cytoplasmic antibodies (ANCA) in virtually all patients with microscopic polyarteritis means that for the first time there is a non-invasive sensitive test with a high specificity capable of distinguishing between these two disorders.[5] More widespread use of ANCA in patients with cutaneous vasculitis and deep organ lesions, we may hope, will assist physicians in the diagnosis and management of these conditions in future.

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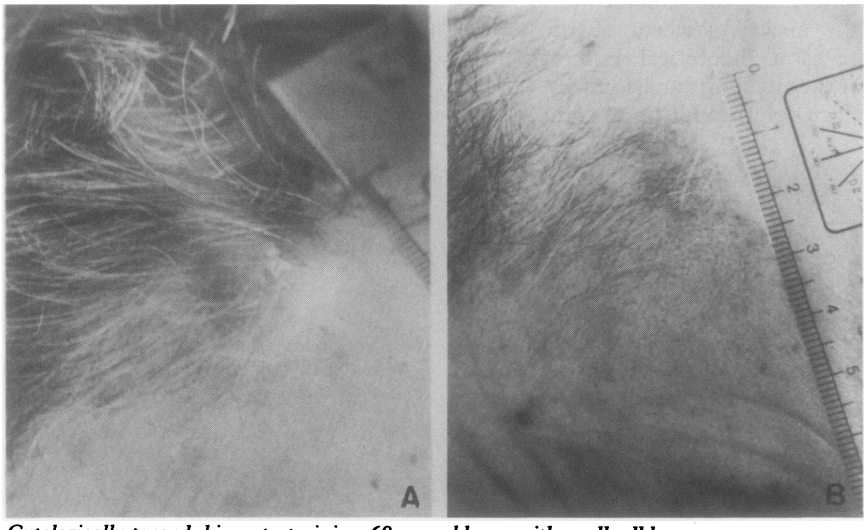

Cytologically proved skin metastasis in a 69 year old man with small cell lung cancer. (A) Appearance 21 days after a complete surgical resection for wrongly diagnosed limited stage disease; (B) clinical response 15 days after adjuvant chemotherapy consisting of doxorubicin, vincristine, cyclophosphamide, etoposide, and cisplatin.

1 Pusey CD, Venning MC, Peters DK. Immunopathology of glomerular and interstitial disease. In: Schreier DW, Gotschalk LW, eds. *Diseases of the kidney*. Boston: Little, Brown, 1988.
2 Savage COS, Winearls CG, Evans DJ. Microscopic polyarteritis: presentation, pathology and prognosis. *Q J Med* 1985;56:467–83.
3 Cream JJ, Gumgil JM, Peaching RDE. Henoch-Schönlein purpura in the adult. *Q J Med* 1970;NS 39:461–84.
4 Hind CRK, Lockwood CM, Peters DK, *et al*. Prognosis after immunosuppression of patients with crescentic nephritis requiring dialysis. *Lancet* 1983;i:263–6.
5 Savage COS, Winearls CG, Jones S, Marshall PD, Lockwood CM. Prospective study of radioimmunoassay for antibody against neutrophil cytoplasm in diagnosis of systemic vasculitis. *Lancet* 1987;i:1389–93.

AUTHOR'S REPLY Dr Veale and his colleagues raise two points: firstly, the diagnostic label that should be attached to our patient's disease and, secondly, the clinical use of the antineutrophil cytoplasmic antibody (ANCA) test.

The clinical features of our case are those described in classical Henoch-Schönlein purpura: namely, arthralgia, gastrointestinal haemorrhage, glomerulonephritis, and the characteristic rash affecting the buttocks and extensor surfaces of the lower limbs. Since reading their letter we have restained sections of the original renal tissue, using improved immunofluorescence techniques; the glomeruli show no IgG but IgA and IgM are present in the glomerular tufts. There is also, however, some intraepithelial tubular staining, suggesting that at least some of the glomerular staining may be due to a prefixation diffusion artefact; immunoglobulins may enter cells by passive diffusion if fixation is not prompt. The necropsy in this case was carried out on the fifth day after death. Thus we are reluctant to attach too much significance to the immunofluorescence findings.

The ANCA test does offer exciting possibilities in the non-invasive diagnosis and classification of the arthritides, though our patient presented in 1984 before it was available. A recent study, however, has suggested that it may not be quite as specific or sensitive for microscopic polyarteritis as suggested by Savage *et al*.[1] Using an indirect immunofluorescence assay, Harrison *et al* found the granular pattern of cytoplasmc staining to be highly specific for Wegener's granulomatosis[2]; but the diffuse pattern of staining usually reported in microscopic polyarteritis was less specific or sensitive for microscopic polyarteritis: seven of 12 cases showed weak or insignificant staining, whereas strongly positive diffuse staining was seen in cases of polymyalgia rheumatica and Paget's disease. Savage *et al* did use a combination of an immunofluorescence assay and radioimmunoassay, but the very high sensitivity and specificity they reported in microscopic polyarteritis needs confirmation; if it is confirmed both assays need to be available for firm clinical decisions to be based on the result.

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1 Savage COS, Winearls CG, Jones S, Marshall PD, Lockwood CM. Prospective study of radioimmunoassay for antibody against neutrophil cytoplasm in diagnosis of systemic vasculitis. *Lancet* 1987;i:1389–93.
2 Harrison DJ, Simpson R, Kharbanda R, Abernethy VE, Nimmo G. Antibodies to neutrophil cytoplasmic antigens in Wegener's granulomatosis and other conditions. *Thorax* 1989;44:373–7.