

Unilateral facial pain in lung cancer

SIR,—Bindoff and Heseltine¹ reported unilateral facial pain in eight patients with lung cancer. I report an additional case.

A 41-year-old man had aching left facial pain which began as otalgia and retro-orbital discomfort; he had a remote history of left facial fracture that had needed surgical repair. A magnetic resonance imaging scan of the head and chest and radiography were normal, as were otolaryngological and neurological examinations. Nine months later, an aching, left thoracic pain developed; it worsened in the supine position, causing the patient to sleep upright. Results of a treadmill test and of endoscopy, as well as of upper gastrointestinal and repeated chest radiography, were uninformative; results of computed tomography of the chest, abdomen, and neck were normal apart from a vague density in the left aortopulmonary window. Left vocal cord paralysis developed one year after he was first evaluated. Thoracotomy showed poorly differentiated squamous carcinoma metastatic to local nodes; the patient had smoked two packs of cigarettes daily.

Features of our patient's condition were similar to those in some patients described previously.^{1,2} Our patient had chemotherapy before radiation, and many treatments for pain relief, none of which was effective until radiation therapy to the affected lung abolished both the facial and thoracic pain.

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Allergenic peanut oil in milk formulas

SIR,—Anaphylaxis induced by peanuts is a serious food allergy and fatal cases have been published.¹⁻³ Spontaneous recovery is unusual.⁴ Allergy to peanuts is one of the four most prominent childhood allergies,^{5,6} atopic dermatitis being a common feature. The increasing incidence of atopic dermatitis in infants led us to suspect a hidden peanut allergen in the nutritional intake.

A baby, now 6 months old, first had atopic dermatitis at 3 months. He had been breast-fed for 5 days and then given milk formula. He took babies' crackers (containing egg) infrequently. Prick tests were positive to peanut and egg extracts and a radioallergosorbent test (Rast Phadebas Cap-System) was positive at 19 KU/l to peanut. As a provocation test a drop of peanut butter was placed on the baby's lower lip and oedema and erythema extending to the chin were observed in less than 15 min. Oral provocation with 1 g peanut oil in stewed apple elicited a rash that spread to the abdomen; this began after 15 min and lasted for an hour.

Another baby who had had atopic dermatitis since the first month of life had been fed from the birth with the same milk formula as in the first case. At 4 months, spreading skin lesions motivated a search for food allergy. Prick tests were strongly positive to peanut proteins but negative to milk proteins. Specific IgE to peanut reached 1.7 KU/l. The labial provocation test was positive with peanut butter and with oil. An oral challenge with 1 ml peanut oil brought on a generalised rash in 15 min. Ordinary cow's milk was substituted for the formula and the eczematous lesions decreased impressively after a month. The parents then accepted a challenge with milk formula and in less than 36 h the baby's atopic dermatitis was worse.

We learned that the vegetable lipid content of the milk formula was 10%, 80% of which was peanut oil. The average intake was estimated at 7 g per day.

The residual allergenicity of peanut oil has been considered negligible: Taylor and colleagues⁷ oral challenges with oil in ten peanut-allergic adults revealed no reactivity and Keating et al⁸ did not detect even trace amounts of peanut allergen in virgin oil.

However, the labial and the oral provocation tests were obviously positive with oil in both these children, and withdrawal and then

reintroduction of milk formula confirmed its responsibility for symptoms of food allergy. This shows that allergenic proteins are present in the oil. Moreover, in the first case the parents did not ingest any peanut product, and breast-milk-induced sensitisation seems unlikely.

An inquiry about the nature of vegetable lipids in 45 milk formulae points to the presence of peanut oil in 11. The seriousness of anaphylaxis to peanuts makes the exclusion of peanut oil from milk formulae highly advisable.

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Screening for *Helicobacter pylori*

SIR,—We disagree with Dr Bateson's (Aug 10, p 393) conclusions that serum IgM measurement is the only reliable test for *Helicobacter pylori* infection. Although we did show a declining incidence of IgM antibodies to *H pylori* with increasing age,¹ which presumably reflects recent infection with the organism, previous work by us² and others³ has shown that IgM antibodies correlate poorly with active gastritis. Indeed in none of the 1018 blood donors we studied had endoscopy been done or *H pylori* looked for.¹

In prospective studies of serological tests for IgG, IgA, and IgM in patients of various ages with either non-ulcer dyspepsia or ulceration, we have consistently shown that IgG correlates best with active gastritis. IgM values have no relation to gastritis in any of the studies. Presumably after colonisation by *H pylori* and an immunological response there is a time lapse before development of gastritis. This could account for the lack of correlation between IgM values and active gastritis. Moreover, we have shown the value of IgG antibodies in a prospective study in healthy subjects with high concentrations only⁴ and in patients with low and high concentrations of IgG antibodies to *H pylori*⁵ (sensitivity and specificity about 100%).

Although we agree with Bateson's statement about further improvement of serological tests, we believe that measurement of serum IgG is the only reliable test as an indicator of active gastritis associated with *H pylori* infection.

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