Asthma knowledge attitudes and quality of life in adolescents

EDITOR,—Gibson et al report a low level of knowledge about asthma in adolescents with and without asthma and their teachers, and propose improved training in schools.1 As part of a recent survey of knowledge about common childhood disorders, we administered the following five true/false/don’t know multiple choice questions concerning asthma to 120 unselected parents attending our paediatric casualty department:

(1) Children with asthma often get attacks when they have colds and infections (true).

(2) Smoking about the house is no worse for asthma than the traffic fumes on a normal street (false).

(3) Children who cough a lot at night are more likely to have asthma than children who bring up lots of sputum (phlegm) with coughing in the morning (true).

(4) Children with very bad asthma always make loud breathing noises (false).

(5) Antibiotics are often useful in children to help cure bad attacks of wheezing (for example, due to asthma) (false).

The response rate was 91% (109/120); 77 parents had a family member or close relative with asthma, 24 did not, eight were unsure. Our results also demonstrate fairly low levels of knowledge (% correct = 1: 79%; 2: 67%; 3: 28%; 4: 34%; 5: 58%). Although our questions were not comprehensive, they covered a wide area including precipitating factors, diagnostic features, assessment of attack severity and treatment issues. The proportion correct in the two subgroups (asthma in family or not) was closely similar for all questions except question 4 (35% and 8% correct respectively, p<0.001, χ² analysis).

Perhaps, in addition, knowledge and understanding of asthma could and should be better enhanced during the innumerable contacts that health services have with young asthma sufferers and their parents before they reach adolescence.

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Cutaneous polyarteritis nodosa

EDITOR,—Cutaneous polyarteritis nodosa (PAN) was first described in 1931 and more than 100 cases have been reported since then.1,2,3 Cutaneous PAN involves the skin, skeletal muscle, and peripheral nerves without any evidence of visceral involvement and typically has a benign but often relapsing course. It is a distinct entity from the common systemic PAN although progression to visceral involvement has been reported.4,5 One of us (JLV) described the first childhood case in this journal in 19801 and we now report this patient’s subsequent clinical course.

In June 1976 a girl aged 5-5 years presented acutely with malaise, painful swollen limbs, and a 12 month history of an asymptomatic rash. On examination she was febrile and had a swollen face, forearms, wrists, left hand, right knee, and a large, raised, eczematous rash on the left calf. There was a widespread, patchy maculopapular, erythematous eruption. There was neither organomegaly nor hypertension.

Investigations on admission showed an erythrocyte sedimentation rate of 100 mm/hour, antistreptolysin O titre (ASOT) 900 (normal <200) Todd units. Thorax swabs produced no growth. Hepatitis B serology was negative. A skin biopsy specimen showed necrotic arteritis at the junction of the deeper dermis with subcutaneous tissue. Muscle biopsy showed normal striated muscle. Treatment comprised aspirin and bed rest and the illness subsided over the ensuing three weeks, during which time the skin showed more obvious nodular and keloidal lichenification. The patient experienced milder exacerbations in 1980, 1983, 1984, 1986, and 1995. In July 1995 she had her most severe flare since 1976 with painful nodules, necrotic vasculitic areas over shoulders, buttocks and right hip, myalgia, and arthralgia. This followed an episode of streptococcal sore throat was accompanied by an ASOT of 333 Todd units and a throat swab which grew group G β-haemolytic streptococci. Treatment was started with prednisolone 60 mg daily, penicillin V 2 g daily for a week, and naproxen 500 mg daily. The latter two drugs have been continued to the present time, the penicillin dosage having been reduced to 500 mg daily as prophylaxis. As the prednisolone dosage was reduced, the drug was continued for a period of approximately three months.7 Prednisolone was discontinued in March 1995.

Current clinical findings include widespread livedo reticularis and a few small erythematous nodules over the lower limbs in addition to areas of markedly hyperpigmented postnecrotic scar tissue on the left shoulder and right hip area.

During her most recent exacerbation the blood pressure remained normal but a creatinine clearance of 47 ml/min and a 24 hour urinary protein excretion of 0.58 g (normal 15 g/year) were recorded in 1993. Her most recent creatinine clearance was, however, 103 ml/min and there is no persistent proteinuria or other evidence of visceral involvement.

This case illustrates several important features of cutaneous PAN. The original presentation is typical in that the nodules of cutaneous PAN show histological features of PAN, whereas PAN in patients with systemic PAN are usually non-specific and include urticaria, purpura, and livedo reticularis.2 Nodulation is particularly characteristic of cutaneous PAN. There have been repeated exacerbations over a 19 year period, at least some of which have been associated with streptococcal infection.

Published series have documented a strong association between streptococcal infection and flares in disease activity and long term penicillin prophylaxis has been recommended.8,9 In the present case, her chronic relapsing course our patient shows no definite evidence of systemic disease, although continued follow up is clearly advisable in view of isolated reports of such progression.

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Blood pressure and smoking: observations on a national cohort

EDITOR,—Charlton and White state that the reasons why adult smokers have lower blood pressure than non smokers have not been determined.1 They report that prospectively those children who had lower diastolic blood pressures at age 10 years were more likely to smoke at age 16. Although self reported smoking history was obtained at age 16, there was no record of smoking history at 10 years of age when the blood pressure and pulse were measured.2 The most likely explanation for this finding is that those children with the lowest recorded diastolic blood pressures and pulse were already smoking at the age of 10.

An attractive hypothesis to account for the lower diastolic blood pressure in smokers would be vasodilatation due to regular inhaled nitric oxide exposure. Inhaled nitric oxide is a potent pulmonary vasodilator at concentrations as low as 6 parts per million (ppm).3 The concentration of nitric oxide in cigarette smoke can be as high as 1000 ppm.3 In adults smoking has significant effects on endothelial nitric oxide production, and on nitric oxide levels in the lung. Compared with non-smokers, smokers have significantly decreased exhaled endogenous nitric oxide production,4 possibly as a result of down regulation of receptors secondary to repeated exogenous exposure. It is possible that regular exposure to high levels of nitric oxide in children who smoke cigarettes results in a persistent vasodilatation, with resultant decrease in diastolic blood pressure.

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