

and then cystectomy. He survived, but evidence of secondary deposits in the lung were later found.

Both men worked throughout their lives on the production line of a major car manufacturer. They never worked in the dye, rubber, tanning, or aluminium industries. Both were heavy cigarette smokers.

Comment

Epidemiological evidence for an association between bladder cancer and exposure to β -naphthylamine and benzidine, which are used in the manufacture of dyes, was shown conclusively by Case in 1954; recently it has been shown that workers in the manufacture of dyes who also smoke are at the greatest risk.⁵ Little industrial evidence exists, however, to suggest that exposure to finished dyes is a cancer risk, perhaps because they are used carefully and in dilute aqueous solutions.⁵ Chrysoidine has been manufactured and used for 100 years, and there is no evidence to suggest that its industrial use has led to an increased incidence of bladder cancer.⁴ The two men reported on here, however, had been exposed to high concentrations of chrysoidine dye for a long period (24 years) under conditions that would be quite unacceptable in industry.

That these two men were brothers as well as anglers might suggest a genetic predisposition to bladder cancer, but I am unaware of any evidence for an increased incidence in siblings. I must conclude, therefore, that smoking and prolonged heavy exposure to chrysoidine dye are the two factors most likely to have led to the development of transitional cell tumours of the bladder in these two related cases.

I thank Mr D Farrar and Mr J M O'Brien for allowing me to report on their patients.

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Acquired platelet dysfunction with eosinophilia

A bleeding disorder comprising non-thrombocytopenic purpura and eosinophilia occurs commonly in children in south east Asia¹ but has not been reported in other geographical areas or in non-indigenous children visiting south east Asia. We describe a child who developed the condition on returning to the United Kingdom after living in Malaysia for two and a half years.

Case report

A girl aged 8½ presented to her general practitioner with a three week history of painless bruising, which had first occurred two weeks after her return to the United Kingdom from Malaysia. Apart from having widespread small ecchymoses she was well. A routine blood count showed normal numbers of platelets ($212 \times 10^9/l$) associated with eosinophilia. She was referred to the haematology clinic for further investigation. There was no history of ingestion of drugs, allergy, rashes, or respiratory symptoms. She was habitually constipated and had not suffered from any gastrointestinal disturbance while abroad. A sister aged 10 and a brother aged 3½ were well and asymptomatic.

Examination showed a fit child with scattered old bruises up to 2 cm in diameter on the legs, arms, and back. There were no other abnormal physical findings. Initial investigations showed haemoglobin concentration 12.1 g/dl ; white cell count $7.2 \times 10^9/l$ (absolute differential neutrophils $2.66 \times 10^9/l$, lymphocytes $2.80 \times 10^9/l$, monocytes $0.08 \times 10^9/l$, and eosinophils $1.66 \times 10^9/l$); and erythrocyte sedimentation rate 4 mm in the first hour. A capillary resistance (Hess) test gave negative results, a chest radiogram was normal, and

three stools were negative for parasites, as were stools of her siblings. One week later she had a new bruise 2.5 cm diameter on the forehead. Routine coagulation tests, including prothrombin time, activated partial thromboplastin time, thrombin time, and factor VIII assay, gave normal results. A template (Simplat) bleeding time was 20 minutes. A normal platelet aggregation pattern was found with adenosine diphosphate, ristocetin, and adrenaline, but the platelets did not react with even high concentrations of collagen.

Three further stools were examined for parasites after a purge with sodium picosulphate. These tests, together with a Sellotape test and serological examination for filariasis, schistosomiasis, and toxocariasis gave negative results. Immunofluorescence tests for antibodies to cell nuclei, mitochondria, smooth muscle, parietal cells, and thyroid microsomes gave negative results, but immune complex concentrations were 62.7% (normal 0-24%). Serum IgG, IgA, and IgM concentrations were normal. A further episode of bruising occurred seven weeks after presentation, but three months later she was completely asymptomatic, though unwilling to provide further blood samples for analysis.

Comment

Acquired platelet dysfunction associated with eosinophilia is most common in children¹⁻⁴ and rare in young adults.³ The condition has a benign course lasting two to 12 months. Investigation usually shows a prolonged bleeding time but negative Hess test results. The most consistent defect of platelet aggregation is impaired endogenous release of adenosine diphosphate with collagen,^{1,3} but abnormalities of adrenaline and adenosine diphosphate aggregation may also occur.^{1,2,4} Parasitic infection has been reported in up to 60% of patients.¹⁻⁴ The parasites most commonly identified are *Ascaris*, *Enterobius*, and *Ankylostoma*.

Our patient showed the salient features of a syndrome that has hitherto been regarded as peculiar to south east Asian children. Evidence of parasitic infection could not be found at presentation or subsequently. The cause of the condition is unknown, but finding an increased concentration of immune complexes suggests a possible mechanism. It is known that immune complexes may trigger the platelet release reaction.⁵ This could produce a "storage pool defect," the magnitude of which may be related to an increasing concentration of immune complexes. This syndrome should be considered in children with obscure bruising who have recently returned from south east Asia.

We thank Dr P B A Kernoff of the haemophilia centre and haemostasis unit, Royal Free Hospital, who recognised the relevance of our findings.

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¹ Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;294:687-90.

² Osler AG. *Complement: mechanisms and functions*. Englewood Cliffs: Prentice-Hall, 1976.

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¹ International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1982;284:1766-70.