a patient who developed this second form of spurious polycythaemia over a six-month period.

Case report

A 46-year-old man, previously healthy, developed a hemiparesis in April 1976. He smoked 40 cigarettes daily and drank alcohol heavily. He had a dense left hemiparesis, slight hepatomegaly, and was normotensive. Computerised axial tomography showed no intracranial abnormality. A mild initial liver dysfunction returned rapidly to normal. Cerebral thrombosis and alcohol-induced liver dysfunction were diagnosed. After 11 days he developed calf vein thrombosis (confirmed by venography) and a small pulmonary embolism (on lung scan). He was treated with heparin and warfarin, and was discharged on warfarin after six weeks. Thereafter he smoked 15 cigarettes daily and took little alcohol.

When in hospital his blood picture was repeatedly normal (table). The next blood count, five months later, showed a strikingly raised packed cell volume, haemoglobin, and red cell count, and the abnormalities have persisted for over one year (table). Red cell indices, white cell count, serum urate and sodium concentrations, and blood pressure remained constant.

Mean values for haematology, biochemistry, and blood pressure measurements during first admission (April-May 1976), and during 11 months (November 1976 to October 1977) after packed cell volume had risen

	April-May 1976 Mean ± SD (n)	Nov 1976-Oct 1977 Mean ± SD (n)
Haemoglobin (g/dl)	13·3 ± 0·8 (6)	19·5* ± 0·9 (11)
RBC (10^{-12}/l)	4·17 ± 0·32 (6)	6·31* ± 0·26 (11)
Packed cell vol (%)	38·3 ± 2·1 (6)	56·7* ± 2·8 (11)
MCV (fl)	92·0 ± 3·0 (6)	91·1 ± 2·7 (11)
MCH (pg)	31·7 ± 1·2 (6)	31·5 ± 1·0 (11)
MCHC (g/dl)	34·3 ± 0·9 (6)	34·4 ± 0·8 (11)
WBC (10^{-9}/l) ..	8·43 ± 0·79 (6)	9·58 ± 1·39 (11)
Platelets (10^{-9}/l) ..	— (0)	193·6 ± 19·8 (11)
Serum albumin (g/l) ..	30·2 ± 3·9 (4)	39·7† ± 3·1 (4)
Serum protein (g/l)	60·0 ± 1·8 (4)	70·7* ± 0·4 (3)
Serum urate (mmol/l)	0·22 (4)	0·21 (2)
Serum sodium (mmol/l)	137·4 ± 2·6 (8)	137·9 ± 1·4 (3)
Blood pressure (mm Hg)	138 ± 15 (22)	137 ± 7 (12)
	87 ± 11 (22)	85 ± 10 (12)

*P < 0·001, †P < 0·01 } versus results for April-May 1976, Student's unpaired *t* test.

Conversion: SI to traditional units—Urate: 1·0 mmol/l ≈ 20 mg/100 ml. Sodium: 1 mmol/l = 1 mEq/l.

Serum albumin and protein concentrations also increased. Platelets were normal. In August 1977 investigations showed: red cell volume (^{51}Cr-RBC) 35·3 ml/kg, predicted 27·7 ml/kg; plasma volume (^{131}I-albumin) 30·9 ml/kg, predicted 42·9 ml/kg; marrow appearances normal; splenomegaly absent by palpation, ultrasound, and scintiscan; leucocyte alkaline phosphatase raised; respiratory function (FVC, FEV_1, FEV_1/FVC, diffusing capacity) normal; arterial Po_2 96 mm Hg. Renal disease was excluded by intravenous pyelogram and ultrasound; hepatic disease by scan, ultrasound, and α-fetoprotein; and chronic dehydration by 24-h urine volume (1450 ml) and sodium (173 mmol). The results of chest *x*-ray examination, and concentrations of lipoproteins, plasma cortisols, and urine normetadrenaline were normal.

Comment

Spurious polycythaemia was diagnosed by definition (raised packed cell volume with red cell mass < 36 ml/kg[1,2]) and also by excluding polycythaemia vera and recognised causes of secondary polycythaemia. Raised leucocyte alkaline phosphatase has been reported in spurious polycythaemia.[3] There was no evidence that a high packed cell volume had been masked in April-May 1976—for example, by iron deficiency (red cell indices were unaltered) or haemodilution (serum sodium was unaltered). The reason for the rise in packed cell volume is unknown. The concomitant rise in plasma albumin could indicate contraction of the plasma volume but more probably reflected improved liver function, for which there was other evidence. The rise in packed cell volume was in any event disproportionate to the rise in albumin. Tissue hypoxia resulting from the pulmonary embolism might have increased the red cell mass, but this seems improbable in view of the normal arterial Po_2 and pulmonary function. Heavy smoking, heavy alcohol intake,[4] and hypertension[5] apparently played no part in raising the packed cell volume.

To my knowledge, spurious polycythaemia developing during observation has not been reported. A high-normal red cell mass (< 36 ml/kg) and low-normal plasma volume (> 30 ml/kg), as seen in this patient, typify the more common form of spurious polycythaemia, considered to represent the extreme of the normal range.[2,3] This view of the condition is difficult to sustain in this case, where spurious polycythaemia was observed to develop over a six-month period.

I thank Drs A Hutcheon, N Lucie, and G Young for helpful discussion, and Dr A Hutcheon for measurements of red cell and plasma volumes.

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Oesophageal perforation due to paraquat

The severe and often fatal effects of paraquat on the lungs and kidneys are well recognised.[1] The corrosive effect on the upper alimentary tract mucosa[2] has received less prominence. We report two cases of paraquat ingestion causing oesophageal ulceration and death from mediastinitis.

Case 1

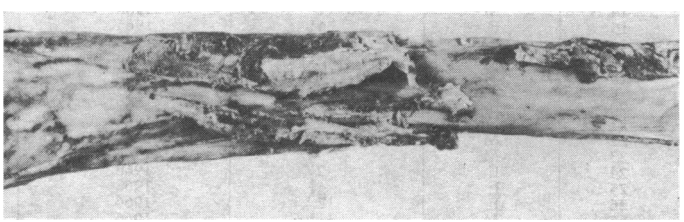
A 59-year-old man was admitted in April 1976 having ingested 50–100 ml Gramoxone three days earlier. The previous day he had consulted his doctor about a sore mouth and throat. Paraquat was present in the urine. On admission he had severe stomatitis and coarse basal crepitations in the chest Blood urea was 26·2 mmol/l (178 mg/100 ml) creatinine 0·66 mmol/l (7·3 mg/100 ml), and plasma paraquat 0·55 mg/l. Haemodialysis was instituted. Two days after admission he had severe central chest pain; radiography showed mediastinal surgical emphysema. He died 10 hours later.

Necropsy showed surgical emphysema in the supraclavicular fossae, mediastinum, and hila of both lungs. Ulceration of the mucosa of the tongue and oesophagus was present, though no distinct perforation was seen. The stomach was not ulcerated. The lungs were collapsed and showed oedema, congestion, and hyaline membranes around some alveolar walls. Paraquat concentrations in lung and kidney were 1·5 μg/g and 2·6 μg/g respectively.

Case 2

An 18-year-old man was admitted three hours after ingesting 50–75 ml of Gramoxone. After gastric lavage he was given one litre 30% Fuller's Earth. Charcoal haemoperfusion was started six hours after ingestion and continued for 54 hours, including six hours in tandem with a haemodialyser. Fuller's Earth and magnesium sulphate by mouth and a forced diuresis were maintained throughout. Vomiting started after the first dose of Fuller's Earth and continued for 24 hours. Seven hours after vomiting ceased he complained of sudden severe chest pain; chest radiography showed mediastinal emphysema. Oesophagoscopy (Mr Dark) showed generalised necrosis of the oesophageal mucosa. He died 70 hours after ingestion of poison. The initial plasma paraquat concentration was 2·7 μg/l.

At necropsy there was surgical emphysema in the mediastinum and bilateral pulmonary collapse with bronchopneumonia. The oesophagus was ulcerated throughout its length (figure) with complete loss of the epithelium



Photograph of oesophagus showing ulceration of mucosa.

and a mononuclear reaction on the surface. The stomach was normal. Paraquat concentrations in kidney and lung were 0·5 μg/g and 1·0 μg/g respectively.

Comment

The striking feature of these cases was the total ulceration of the oesophageal mucosa whereas the stomach was spared. Whether the lack of gastric ulceration was due to the type of epithelium, the presence of acid, mucus, or other local factors is unknown. Ulceration of the mouth and pharynx is almost universal after paraquat ingestion, and pain on swallowing and severe retrosternal discomfort have been reported,[2 3] but oesophageal perforation is uncommon and has not been specifically cited as causing death. Its possible development should be remembered during management. Although paraquat should be removed from the stomach quickly, the immediate use of emetics causing further exposure of the oesophagus to paraquat may be contraindicated. Likewise early control of vomiting produced either by paraquat or Fuller's Earth may be important. Stomach washouts should be done with care because of the danger of oesophageal perforation.[4] In neither of our cases was a definite perforation found, but there is little doubt that a small leak in the oesophagus led to mediastinitis and death.

We thank Dr Rose of ICI Limited, for the paraquat estimations, and Mr Summerfield, the late Manchester coroner, who gave us permission to report these cases.

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Severity of notified measles

The introduction in 1968 of routine measles vaccination in the UK was followed by a drop in measles notifications to one-fifth of the previous figures. Recently, however, less than half the children eligible have been vaccinated and annual measles notifications continue to exceed 55 000.[1] To assess current risks from the disease the severity and complications of notified cases were studied by sending an inquiry form to the notifying doctor.

Methods and results

Forms were returned for 8978 cases (82% of the forms sent). The cases were notified in the last quarter of 1976 from 92 area health authorities in England and Wales. They represented one-sixth of the total notifications to the Office of Population Censuses and Statistics in 1976,[2] with a similar age distribution: 27% of patients were under 3 years, 67% between 3 and 9, and only 0·6% under 6 months. The doctor considered that 4% of cases were severe, 44% moderately severe, and 50% mild. There was little variation with age, although 72% of the few patients under 6 months had mild infections, presumably owing to the persistence of maternal antibody. This was in contrast to the findings in a study on the severity of whooping cough,[3] in which the illness was most severe under the age of 3 months.

There were two deaths (both in handicapped children) in the cases studied (0·22/1000), compared with 14 deaths in the 55 361 total notifications (0·25/1000). A total of 934 cases (10%) had one or more complication (table), and 125 (1·4%) were admitted to hospital, with a mean stay of 5·8 days. The 62 neurological complications included two cases of encephalitis confirmed by EEG (aged 2 and 5), one of meningitis (aged 5), and two of meningism; the first three developed a week after an unremarkable attack of measles at home. Of the 42 children with convulsions, one-third had a history in themselves or their siblings; in over half the convulsion was the reason for admission to hospital. Behaviour changes included persistent screaming, excessive irritability, head banging, and excessive drowsiness. The 403 respiratory complications included 113 cases of bronchopneumonia and 12 of bronchiolitis, the remainder being unusually severe bronchitis. Two critical cases of pneumonia occurred in 2-year-old boys, who became increasingly dyspnoeic, cyanosed, and dehydrated a week after the attack of measles. Other complications included: dehydration from vomiting (5), severe epistaxis (3), and conjunctivitis (47). A 25-year-old woman suffered deterioration of her eyesight with partial recovery, and Henoch-Schönlein purpura occurred in a 2-year-old. A 4-year-old developed diabetes and there were two cases of punctate keratitis, one in an 18-year-old, the other in a 1-year-old.

Information about measles vaccine was frequently unobtainable but measles occurred in 4% of children vaccinated, two-thirds had a mild attack; there were no neurological complications. The incidence did not increase with the interval since vaccination.

Comment

The last survey of the complications of measles in the UK was in 1963, when 53 000 cases were studied,[4] compared with the 9000 in this survey, in 1976. Nevertheless, the rates for deaths (0·2/1000 cases), admissions to hospital (1%), and respiratory complications (4%) were the same in 1963 and 1976. More neurological complications and otitis media were reported in 1976—0·6% and 5%, respectively—compared with 1963, when the rates were 0·4% and 2·5%.

Any study of notified cases is necessarily incomplete, and this one is no exception. Cases which escape notification may include more which are mild and uncomplicated and if this were so the complication rate would be lower. Nevertheless, it is striking that the rates for deaths, hospital admissions, and respiratory and neurological complications have not decreased in 13 years. The only factor which has changed is the total number of cases; the potential dangers from the disease appear to be the same.

I thank all the participating general practitioners, paediatricians, district community physicians and their staffs, and Mrs A Allchin and Mrs G Smith, of the Epidemiological Research Laboratory.

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Numbers (and percentages) of cases with complications, and individual complications according to age. (Cases with more than one complication are included under each heading)

Age group	No of cases	Total No (%) of cases with complications	CNS complications	Behaviour changes	Convulsions	Respiratory	Otitis media	Other
<6 months	54	4 (7)				3 (6)		1 (2)
6-11 months	425	46 (11)	1 (0·2)	1 (0·2)	2 (0·5)	22 (5)	18 (4)	6 (1)
1-2 years	1941	228 (12)	1 (0·1)	8 (0·4)	20 (1)	92 (5)	118 (6)	18 (0·9)
3-4 years	2368	245 (10)		2 (0·1)	13 (0·5)	107 (5)	123 (5)	17 (0·7)
5-9 years	3664	361 (10)	3 (0·1)	3 (0·1)	6 (0·2)	159 (4)	178 (5)	31 (0·8)
10-14 years	350	37 (11)		1 (0·3)	1 (0·3)	16 (5)	19 (5)	1 (0·3)
15+ years	101	9 (9)				3 (3)	2 (2)	4 (4)
Not stated	75	4 (5)				1 (1)	3 (4)	
Total	8978	934 (10)	5 (0·1)	15 (0·2)	42 (0·5)	403 (4)	461 (5)	78 (0·9)