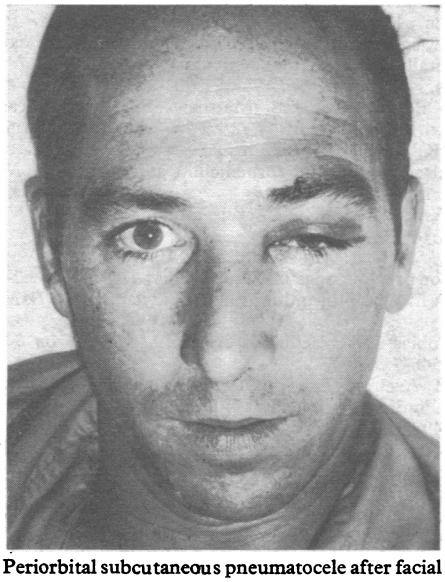
SHORT REPORTS

Subcutaneous pneumatocele after facial trauma

Two patients recently presented complaining of periorbital swelling on blowing their nose after minor facial trauma. This curious complication of fracture of the frontal or ethmoid sinus has apparently not been reported.

Case reports

(1) A 32-year-old healthy labourer presented with swelling around his left eye. Two hours before he had been sitting by his car adjusting the brake when the point of the screwdriver slipped and struck him above the left eyebrow. It did not appear to penetrate deeply and he easily controlled the bleeding, bandaged the wound, and expected no further difficulty. An hour later he tried to blow his nose but stopped when he realised that the periorbital tissues were swelling. He blew again a few minutes later, with his wife watching. The eye closed almost completely. The contents of the handkerchief were not examined. When seen in the casualty department he was afebrile and not acutely distressed. A slightly tender, 4-mm, X-shaped wound was visible above his left eyebrow. The left eyelid was discoloured and swollen almost shut (figure). The mass was soft, not tender, and did not crepitate. Further physical examination and past medical history added nothing. In particular, he had no history of recent rhinorrhoea or sinusitis. Radiographs showed a small fracture of the roof of the left orbit and a small flake of bone detached at one end was impinging on the cavity of a fairly extensive frontal sinus.



Periorbital subcutaneous pneumatocele after facial trauma.

The patient was given co-trimoxazole 480 mg twice daily by mouth and admitted for observation. Against instructions, he blew his nose the next morning and was rewarded by even more swelling around the eye. He remained afebrile without headache or meningimus. The periorbital swelling subsided over four days without signs of inflammation developing. On the seventh day the antibiotic was discontinued and he was discharged. Four weeks later he was well and radiographs of the frontal sinus showed no other abnormality.

(2) A 12-year-old boy was brought to the casualty department half an hour after falling 6 feet (2 metres) from a tree, landing on his right hand and forehead on a level surface of turf. When shortly after he blew his nose his right eyelid swelled shut. There was no history of rhinorrhoea or sinusitis. The only apparent injury was an area (1 x 2 cm) of bruising above the right orbit similar to that seen in case 1. In this case, however, palpation disclosed surgical emphysema. Radiographs of the skull and facial bones failed to show the presumed fracture. He was admitted to hospital, treated with erythromycin 500 mg six-hourly, and advised not to blow his nose. He remained afebrile and the periorbital swelling disappeared by 48 hours. One week later he was allowed tentatively to blow his nose. There was no ill result. When seen four weeks after his injury he was well.

Comment

In 1974 Jarvis reported an atraumatic subcutaneous pneumatocele in the region of the orbits occurring in conjunction with an ethmoid mucocele and dilated sphenoidal sinuses.[1] Pneumatocele may also result from air escaping from the middle ear or mastoid air cells after trauma or surgery.[2] Although no serious consequences arose in our cases the management of penetrating and non-penetrating injuries in the vicinity of the paranasal sinuses should be cautious,[3 4] especially when the wall of the sinus is breached. Injuries may penetrate the dura, even the brain. Turner[3] has recently reviewed 342 midface and upper facial fractures, none of which resulted in abnormal collections of air. In each case the collection was intracranial (pneumocephalus). Severe fractures of the anterior wall of the frontal sinus may require surgical elevation of depressed bone fragments. Fracture of the posterior wall may result in a potentially serious dural tear.[4]

The distinctive feature of our cases was the appearance of subcutaneous air on blowing the nose by a process analogous to a tension pneumothorax. Relatively minor trauma was sufficient to fracture the thin plate of bone between orbit and frontal or ethmoid sinuses. In each case there was no evidence of underlying sinus or nasal disease. Appropriate management of these cases appears to require active observation in hospital, awareness of the possibility of intracranial injury, and antibiotic treatment against upper respiratory tract pathogens.

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[3] Turner JS. Pneumocephalus with facial fractures. *Laryngoscope* 1968;**78**:713-27.
[4] Birrell JF, ed. *Turner's diseases of the nose, throat, and ear.* Bristol: J Wright, 1977:84-5.

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Convulsions associated with campylobacter enteritis

Campylobacter enteritis tends to occur in epidemics and is associated with fever, malaise, abdominal pain, and diarrhoea that is often bloody and profuse. These symptoms may last from a few days to a week. With improved techniques, campylobacter has become the commonest identifiable cause of diarrhoea.[1] We report here several cases in which convulsions were a presenting feature of campylobacter enteritis.

Patients, methods, and results

During a recent large epidemic,[2] which will be reported elsewhere, 14 children with campylobacter enteritis were admitted to the children's ward of our hospital. All the children had diarrhoea, seven with blood and mucus in their stools. Ten had abdominal pain and eight had vomiting. Eleven of these children had a temperature of over 38°C on admission. Nine were admitted after a generalised grand-mal convulsion, and one of them had another convulsion after admission. Seven of these children had had a febrile convulsion before. *Campylobacter jejuni-coli* was isolated from the stools of all 14 children. They received symptomatic treatment only, and all recovered within three or four days of admission. The mean age of the nine children presenting with convulsion was 5 years and 8 months (range: 3 years 3 months to 7 years 7 months), and five were aged over 5½ years. These five underwent electroencephalography, and the results were normal in four children and slightly abnormal in one.

A retrospective study was made of 40 consecutive children admitted to the children's ward with febrile convulsion not associated with campylobacter enteritis. The mean age of these children was 22 months (range: 5 months to 4 years 2 months).

Comment

Convulsion as a manifestation of campylobacter enteritis has not been reported. The nine children in our series who had convulsions had the unusually high mean age of 5 years 8 months, which is outside the range normally associated with febrile convulsions. Rutter and Smales[3] reported a mean age for febrile convulsions of about 22 months, which is similar to that in our own small series.

Convulsion is known to occur with shigellosis. The incidence is about 10%, and convulsions may occur in children with only slightly increased or normal body temperatures.[4]

There are several explanations that may partly account for this unusual incidence of convulsion and high mean age: firstly, the outbreak largely affected schoolchildren and not younger children; secondly, the outbreak was large with 223 proved cases in children aged 10 years or less, of whom only 14 were admitted to hospital and only nine had convulsions; and thirdly, campylobacter, like shigella, may predispose children to convulsions.

[1] Skirrow MB. Campylobacter enteritis: a "new" disease. *Br Med J* 1977;ii:9-11.
[2] Communicable Disease Surveillance Centre. Explosive outbreak of campylobacter enteritis. *Communicable Disease Report* 1979;No. 13.
[3] Rutter N, Smales ORC. Role of routine investigations in children presenting with their first febrile convulsion. *Arch Dis Child* 1977;**52**:188-91.
[4] Smith DH. Shigellosis. In: *Nelson textbook of pediatrics*. Philadelphia: W B Saunders, 1975:602-6.

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Paget's disease of bone in two American cities

Paget's disease is rarely found among African blacks.[1] Although it is known to occur among blacks in the USA the prevalence has not been documented. We carried out a radiological survey of the prevalence of the disease in hospital patients in two American cities to compare prevalences in blacks and whites. One of the cities selected was in the north and the other in the south to investigate further a suggestion that prevalence is lower in the south.[2]

Methods and results

The cities selected were New York and Atlanta. In each city a survey was made in two hospitals, one privately and one publicly funded, serving mainly white and black patients respectively. Samples of about 1000 abdominal radiographs of people aged 55 and over were taken from the files of stored films in each hospital. The sampling procedure was the same as that used in earlier surveys.[3,4] Only patients whose films showed the pelvis, sacrum, femoral heads, and all lumbar vertebrae were included. Diagnosis was by one observer (PBG) using the same standardised criteria used in the previous surveys.

The medical records were consulted to determine the patients' ethnic group. At each hospital medical records were available for all patients with Paget's disease and for more than 80% of those without the disease. The ethnic distribution of the remaining patients was assumed to be identical with that of the patients whose records were available. In Atlanta all but one of the patients whose notes were consulted were found to be classified as either black or white; but in New York about 5% were of other ethnic groups and were excluded from the survey.

The table shows the prevalence of Paget's disease by sex and ethnic group in New York and Atlanta. Each prevalence was standardised[5] for age against the aggregate population of a large British survey,[3] so that the results could be compared with those already reported for Britain[3] and Australia.[4] In each city the prevalence in blacks was similar to that in whites. In addition the blacks showed a higher prevalence among men, similar to the well-documented male preponderance of the disease in European whites. The higher prevalence in New York than in Atlanta was shown by both sexes and both ethnic groups. Previous surveys have shown that the radiological prevalence of Paget's disease is higher in radiographs taken specifically to show the skeleton than in other radiographs of the pelvis. The proportions of skeletal radiographs were, however, similar in the two cities and ethnic groups (blacks in New York 12%, in Atlanta 12%; whites in New York 13%, in Atlanta 15%.)

Prevalence of Paget's disease by ethnic group. Numbers of cases are given in parentheses

City	Ethnic group	No of patients	Age-standardised prevalence (%)		
			Men	Women	Both sexes
New York	Black	950	3·3 (12)	2·0 (12)	2·6 (24)
New York	White	1082	5·2 (30)	2·5 (13)	3·9 (43)
Atlanta	Black	1111	1·9 (11)	0·6 (3)	1·2 (14)
Atlanta	White	1563	0·9 (7)	0·8 (6)	0·9 (13)

Comment

In contrast to the position in black Africans, in whom Paget's disease is rare, the condition is similarly prevalent in blacks and whites in the USA. This finding provides further evidence of the influence of environment in the aetiology of the disease. The difference between the northern and southern cities, found in both sexes and in both ethnic groups, indicates a pronounced variation in prevalence within the USA. The figure for the whites in New York is comparable with the lowest values found in a survey of 14 towns in Britain,[3] and similar to the prevalence in Perth, Australia (3·6%).[4]

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[3] Barker DJP, Clough PWL, Guyer PB, Gardner MJ. Paget's disease of bone in 14 British towns. *Br Med J* 1977;i:1181-3.
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Eclampsia in multipara

Three consecutive eclamptic patients seen by me at the Ikedife Hospital within one week were multiparous. Routine history taking revealed changes in paternity in the first two cases. This led me to make confidential inquiries about the actual paternity in the pregnancies of other eclamptic multiparous patients.

Patients, methods, and results

Forty-six eclamptic multiparous patients with singleton pregnancies were seen between August 1968 and July 1978 in a rural practice centred on Nnewi, Anambra State, Nigeria. The Ikedife Hospital serves as an unofficial reference centre for 22 small maternity hospitals within a radius of 35 km. I have excluded in this series three multiparous patients brought in dead after eclamptic fits, two cases of epilepsy, two other patients whose blood pressure was still over 160/90 mm Hg six weeks postpartum, and one uncontrolled diabetic multiparous patient who had fits during the last week of pregnancy. The 46 patients were confidentially interviewed by me