but in both the bone age is often advanced and in Beckwith's syndrome the large size is associated with exomphalos or umbilical hernia, hypoglycaemia, and pronounced visceromegaly. This patient did not have these clinical features but appeared to have a new kind of diencephalic syndrome.

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Congenital abnormalities and maternal herpes zoster

Since Laforet and Lynch¹ in 1947 described a baby with congenital abnormalities after maternal varicella infection in early pregnancy, only two cases² associated with maternal zoster infection have been reported. This report describes a third.

Case report

AB was born at term, weighing 3350 g. The previous child was quite normal and there was no family history of abnormality. At ten weeks' gestation the mother had developed a typical herpes zoster rash at the level of T8 on the left side. This had been confirmed by the general practitioner when she attended for a pregnancy test at twelve weeks' gestation. No drugs apart from calamine lotion were used in the treatment. She had had chicken-pox as a child. The rest of her pregnancy had been uneventful.

At birth the baby was obviously microcephalic (head circumference 30 cm), had bilateral microphthalmia with dense corneal opacities, and neovascularisation. There was no evidence of cutaneous scarring or limb wasting reported in some previous cases. At the age of 3 months the head circumference was 33.5 cm, diverging further from the third percentile. There already was definite developmental delay.

At birth the haemoglobin was 18 g/dl; white cell count $8 \times 10^9/l$; platelets $161 \times 10^9/l$. X-ray films of the long bones were normal and skull x-ray film showed no evidence of intracranial calcification. The results of studies of chromosomes, urine amino-acids, organic acids, and mucopolysaccharides were normal. Serological studies showed no evidence of infection by toxoplasmosis, cytomegalovirus, rubella, herpes simplex, or syphilis. Maternal varicella zoster titre declined from 1/640 at 13 weeks' gestation to 1/160 at term and 1/40 three months later. The baby's titre was 1/10 at birth and at two months. The cord IgM concentration was not estimated. Specific varicella zoster IgM at two months was less than 1/8 but IgG was 1/256 and IgA 1/8 confirming the passage of maternal antibody to the baby.

Comment

The two reported cases of congenital abnormality associated with herpes zoster infection² presented at a few months of age with cataracts and microphthalmia. One child was also mentally retarded but had no microcephaly. From the data from ten other case reports, it seems that the varicella virus can be teratogenic. The eyes are particularly susceptible to damage, as 10 of the 12 reported infants have had ophthalmic lesions. Cataract and microphthalmia are the most frequent abnormalities but three cases of chorioretinitis have also been noted. This is the first report describing corneal opacity and neovascularisation.

The brain is also affected. Four children had cortical atrophy with

seizures, two had "gross mental retardation," and one was microcephalic. This baby's head is growing very slowly, which may represent cerebral damage resulting from encephalitis. A further example of the sensitivity of the nervous system in these cases is shown by seven babies with cutaneous scarring. Five babies had hypoplasia of the scarred limb and a sixth developed facial palsy, deafness, and scars corresponding to branches of the superficial cervical plexus. On the only occasion that electromyographic and nerve conduction studies have been performed the results suggested that the hypoplasia was secondary to denervation.³

It is difficult to know how frequent herpes zoster infection is during the first trimester, and therefore impossible to assess the incidence of congenital abnormality. In a cohort study of major congenital malformations associated with various maternal virus diseases⁴ the numbers implicating varicella zoster in early pregnancy were too small to be significant. At present the only evidence suggesting that the virus may be teratogenic has come from case reports of babies with similar abnormalities. All but two cases^{3,5} had no serological proof of varicella infection in the baby. A prospective study is needed to determine the incidence of infection and abnormalities.

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Tetanus and inhalation of a foreign body

Tetanus may be a complication of a contaminated wound or skin ulcer. We report a case of tetanus after the inhalation of a foreign body into a bronchus.

Case history

An 11-month-old boy was admitted to a peripheral hospital on the 4 October 1975 with a six-day history of a cold. He was febrile, dyspnoeic, and had bilateral bronchospasm. A chest x-ray film appeared normal. He was treated with ampicillin, bronchodilators, and steroids. By day 4 there was no improvement and a second chest x-ray film showed hyperinflation of the left lung with mediastinal shift to the right. A foreign body in the left bronchus was suspected and the patient transferred to the university hospital for endoscopy. At bronchoscopy on the same day a 15-mm long piece of the shell of the leg of a crab was removed from the right main bronchus. During the procedure this had been coughed out of the left bronchus and into the right. On review of the chest x-ray films the very faint outline of the shell could be seen on the left side. As the shell had a lumen, the lung had not collapsed.

On day 5 the patient had a cardiorespiratory arrest, from which he was resuscitated rapidly and transferred to the intensive care unit. On admission there he was afebrile and conscious, but had back and neck stiffness and trismus. Respiration was normal. A positive Kernig's sign was elicited, but lumbar puncture showed normal cerebrospinal fluid. Soon after, he started to have generalised tonic spasms, and a clinical diagnosis of tetanus was made. It was thought then that the cardiorespiratory arrest had occurred during a severe tetanic spasm. No recent skin wound could be found and no history of immunisation could be obtained from the mother. He was given antitetanus serum and started on phenobarbitone, chlorpromazine, diazepam, and penicillin. His spasms became controlled, but tracheostomy was required for chest toilet. The succeeding course of the illness was unremarkable. He was discharged home on day 49 and was followed up for seven months showing no apparent sequelae.

Discussion

Tetanus toxin normally gains entrance to the body through

contaminated wounds or ulcers of the skin. Nevertheless, it has been postulated that the toxin could gain entrance via the respiratory or gastrointestinal tracts.¹ Many patients at this hospital, and at others, have shown no apparent injury to the skin.²⁻⁴

Tetanus after the inhalation of a foreign body should be very unlikely for several reasons. Firstly, *Clostridium tetani* is an anaerobic organism; secondly, bronchial foreign bodies are usually food or small household articles, unlikely to be carrying the spores of tetanus; thirdly, because of the early appearance of signs and symptom, the objects inhaled are usually removed without delay. In this case, however, the object was almost certainly picked up from the earth floor of a back yard. It did not produce the usual symptoms because it had a lumen, thus delaying removal and probably allowing the formation of microabscesses around it, giving rise to localised anaerobic conditions. Tetanus was diagnosed on clinical grounds. Bacteriological confirmation was not obtained, which is common in our experience and that of others.⁵

In conclusion, then, probably a foreign body in the bronchus was the most likely cause of tetanus in this child.

We are grateful to Dr S Sivapragasam for advice in the preparation of this article, and to Professor C Miller and Dr R McNeil for permission to report this case.

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Colonic diverticulosis in a child

In a 9-year-old boy with diverticulitis a colovesical fistula developed. We believe that this is the youngest patient in which this complication has been described.

Case report

A boy born on 11 February 1965 at full term after a normal delivery, weighing 8 lb (3.6 kg), developed osteomyelitis of the upper left humerus and a urinary tract infection at 6 weeks of age; he was treated with antibiotics. Thereafter he had intermittent urinary tract infections and at 13 months an intravenous pyelogram and micturating cystogram showed bilateral hydronephrosis, and hydroureters with gross reflux on the left side and a lesser degree on the right. At 15 months of age with a blood urea concentration of 14.6 mmol/l (88 mg/100 ml) both ureters were reimplanted. He remained generally unwell: the blood urea concentration rose to 16.9 mmol/l (102 mg/100 ml) and the pyelogram showed no improvement. At 2 years of age the ureters were again reimplanted and after this procedure he was healthy but continued to have intermittent urinary tract infections.

In September 1970 he was admitted for further investigation. The urine culture grew coliform organisms, blood urea was 7.6 mmol/l (46 mg/100 ml), a pyelogram showed scarring of both kidneys, and a micturating cystogram showed persistent bilateral reflux. To control his urinary tract infections, he was treated with long-term antibiotics. In September 1974 it was thought that he might have passed urine per rectum, but cystoscopy showed no evidence of a fistula. Nevertheless, in December 1975 (when he was aged 9 years and 9 months) after left-sided abdominal pain he passed faecal stained urine. He was tender in both loins and there was faecal material at the urethral orifice. As expected, a urine culture grew a mixed faecal organism. A micturating cystogram showed definite reflux up the left ureter with two small elongated diverticula of the bladder but no evidence of a fistula. Cystoscopy showed an oedematous area on the right posterior wall with slough; histological examination of a biopsy specimen of this area showed an inflammatory exudate with vegetable fibres, and hence established the presence of a colovesical fistula. A barium enema showed diverticular disease of the type seen in adults.

In March 1976 he was again admitted because of two days' abdominal pain, anorexia, and difficulty in micturition. It was decided that a defunc-

tioning colostomy was necessary in the management of the colovesical fistula. After this his urinary symptoms improved and with antibiotics his urine became sterile. When he had completely recovered in January 1977, a repeat barium enema (see figure) showed an increased severity of the diverticular disease. At present he is awaiting a sigmoid colectomy with closure of the fistula.

Diverticular disease of the sigmoid colon in a 9-year-old boy.

Comment

Diverticular disease is recognised as a common disease seen more usually with increasing age. Though the current vogue is to incriminate the increasingly refined modern diet in its aetiology, there have been sporadic reports of diverticulosis in young people.^{1 2} Some of these cases have shown features unlike diverticular disease in adults—for example, solitary diverticulum or diverticula associated with infectious disease.³ Nevertheless, true diverticular disease in young patients can hardly be attributed to the prolonged effect of our bland diet on a congenitally normal colon.

Intrinsic weakness of the colonic musculature as seen in diverticulosis in Marfan's syndrome⁴ and Ehler's Danlos syndrome, may cause diverticula to develop at a younger age. Possibly our patient has some intrinsic abnormality, whether neurogenic or muscular, as he also has abnormalities in his urinary system. Another feature here was that this boy developed a colovesical fistula secondary to his diverticulitis. This complication of diverticulitis is said to occur in 6-23% of adult cases.⁵ We were unable to find this complication of diverticulitis described in such a young patient.

We thank Mr D B E Foster and Mr R A Mogg for allowing us to describe this patient.

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