Short reports

Poliomyelitis-like illness associated with asthma

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SUMMARY A 10-year-old girl with a combination of paralytic disease, resembling poliomyelitis, and asthma is described. The girl developed neurological symptoms 5 days after a severe attack of asthma. No aetiology to the flaccid paralysis could be demonstrated although Cocksackie B5 virus was isolated from a stool. A similar poliomyelitis-like illness associated with asthma has previously been reported in 13 cases from Australia and the UK.

Poliomyelitis, even of sporadic distribution, has become rare in Europe. However, patients with a paralytic illness resembling poliomyelitis but not due to poliovirus are occasionally seen. Types of enteroviruses other than poliovirus, including Cocksackie virus types A7¹ and B5,² have been isolated from some of these patients. Hopkins³ described 10 patients from Australia with an unusual combination of a poliomyelitis-like illness and acute asthma. Adenovirus type 9 and echovirus 18 were isolated in 2 of them. Later, 3 more patients with a combination of flaccid paralysis and asthma were described from England.⁴⁻⁵

We describe the clinical features of a case of poliomyelitis-like illness and asthma in a Swedish child, and from whom Cocksackie virus type B5 was isolated.

Case report

A girl born 1964. She had an elder brother and sister who had had asthma since childhood. There was no neurological disease in the family. The girl had been delivered normally at term, and had remained well until the onset of asthma at age 4½ years. The following immunisations were administered without complications: BCG at 5 days, smallpox vaccine at 6 weeks, a course of 3 injections for diphtheria, tetanus, and pertussis at 3, 4, and 6 months, and poliomyelitis (Salk vaccine) subcutaneously at 9, 10, and 18 months, with a booster dose at 8 years.

From age 5 she had coughed and wheezed several times each month and had suffered attacks of acute

bronchospasm as often as every 3 months. Percutaneous and intradermal tests followed by bronchial provocation tests gave positive reactions only to moulds. Since 1971 she had been treated with hyposensitisation with Allpyral for 3 years in addition to conventional asthma therapy—such as salbutamol, ephedrine, and aminophylline orally. During this time her symptoms improved and she had been admitted only once for acute asthma and then recovered rapidly.

In September 1974 she was admitted for acute asthma of 12 hours' duration. Five days earlier she had received 500 PNU mould extract subcutaneously from the same batch as previously. The dose was similar to that she had received at monthly intervals for 3 years. She was given her usual asthma treatment but her condition deteriorated. Chest x-ray showed right-sided pneumonia. 10 hours after admission she was very tired, PCO₂ was raised to 7.5 kPa (56 mmHg), and she was intubated and put on a respirator. Her condition then gradually improved and 10 hours later she was extubated.

During the first 4 days in hospital she was treated with humidified oxygen, glucose infusions, and the following drugs: epinephrine, salbutamol, hydrocortisone, glyceryl guaiacolate, phenoxymethylpenicillin, diazepam, and succinylcholine chloride in recommended doses.

Five days after admission she complained of pain and weakness in her arms and legs. The pain was at no time severe and it disappeared without treatment in a few days. However, she continued to have a feeling of weakness in the left leg and the day after onset of pain she developed a flaccid paralysis of that leg. Some weakness in the knee and ankle jerks in the right leg were also discernible, but these disappeared after one day.

The tentative diagnosis was polyradiculoneuropathy, Guillain-Barré type, and she was given cortisone orally for one month. There was only slight clinical improvement during the first weeks of treatment and now, 5 years later, she still has a pronounced flaccid paralysis and muscular atrophy of the left leg, with 1.5 cm shortening.

Laboratory investigations

The following laboratory studies were done within 5 days of admission and gave normal results: Hb, white blood cell and differential counts, ESR, serum IgE, renal concentration capacity, and urinary sediment.

Lumbar punctures were done 5 and 12 days after admission, the CSF showing 2 and $10 \times 10^6/l$ polymorphs, and $59 \times 10^6/l$ lymphocytes and none respectively. Protein was 0.3 and 0.57 g/l (30 and 57 mg/100 ml), and electrophoresis of CSF showed markedly raised α_2 and γ -globulin fractions.

Serum samples were obtained 10 and 14 days after admission and tested for presence of cold agglutinins and complement-fixing antibodies against influenza A and B and *Mycoplasma pneumoniae*. There was no rise in titre between the two samples. Coxsackie virus type B5 was isolated from a stool specimen. No virus was isolated from the CSF.

Ten days after the initial motor symptoms from the left leg the motor conduction velocity (mcv) of the left peroneal nerve was reduced to 39 m/second whereas a normal value (51 m/second) was found in the right leg. EMG was not performed at that time. Four months later the EMG showed a total denervation of extensor digitorum brevis, tibialis anterior, gastrocnemius, and quadriceps on the left side—that is increased insertion activity, a moderate number of denervation potentials, and a total absence of motor units at both least and greatest efforts to activate the muscles. The mcvs were not significantly changed (36 and 51 m/second respectively). The sensory conduction velocity in nervus suralis at that time was 30 m/second on the left side and 36 m/second on the right. The patient refused examination with needle electrode in the right leg.

Discussion

The clinical and laboratory findings indicate injury to anterior horn cells and to the peripheral neurone. On admission the patient was moderately hypoxic, however, at no time was she unconscious and the neurological symptoms did not suggest anoxic injury.

The presence of flaccid asymmetric paralysis suggested poliomyelitis. The girl had been fully immunised against all 3 polio serotypes and at the time of onset of symptoms there had been no paralytic case of polio in Sweden for many years.

The symptoms were similar to those previously reported in 13 patients with a combination of poliomyelitis-like illness and asthma (Table). Typical features of these children's illnesses included neurological symptoms in previously polioimmunised

Table Poliomyelitis-like illness after acute asthma: summary of reported cases

	Reported cases (n = 13)	Present case
Sex M/F	8/5	F
Age at onset (years)	2-8	10
Previous immunisation against polio	13/13	+
Days between onset of wheezing and paralysis	4-11	5
Meningismus	3/13	-
Muscle pain	9/13	+
Neurological findings in acute phase	Paralysis in arm or leg	Paralysis in leg
CSF		
Polymorphs ($\times 10^6/l$)	0-75	2
Lymphocytes ($\times 10^6/l$)	7-495	59
Protein (g/l)	0.16-0.55	0.30
Reduced motor conduction velocity	2/3	+
Denervation signs in electro-myography	2/2	+
Residual weakness	13/13	+
Virus isolated in faecal specimen	Adeno 9 (1) Echo 18 (1)	Coxsackie B5

children 4 to 11 days after an acute attack of asthma. In 2 of the patients electromyography and nerve conduction velocity were recorded,⁴⁻⁵ and these were consistent with anterior horn cell or axonal damage. The children had been treated with various drugs, they showed no meningismus, and they complained of muscular pain with rapid onset of a persistent flaccid paralysis in an arm or leg. The unusual combination of symptoms and the similar clinical findings argue against a chance association between asthma and paralysis in these children.

Toxic reaction to one of the drugs is possible but unlikely. The doses given were all within recommended limits and adverse reactions of this kind have not been reported. Each of the 14 children received a different treatment.

Viral infection has been suggested although not proved.⁵ In our patient Coxsackie B5 virus was isolated from a stool specimen, but this did not prove the virus to be aetiologically related, since no rise in antibodies against this virus was sought. Other viruses were isolated in previous reports.³ Infections with enterovirus, other than poliovirus, may occasionally result in an illness very similar to that caused by poliovirus¹⁻²—that is persistent flaccid asymmetric paralysis. Possibly, an infection with a neutropic virus, like other acute viral and bacterial infections, may occasionally trigger acute status asthmaticus in a sensitive subject.

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An explanation for failure of impedance apnoea alarm systems

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SUMMARY 24-hour recordings of the ECG and respiration, the latter from an impedance technique, have shown a phenomenon which could account for hitherto unexplained failures of impedance apnoea alarm systems. Whenever apnoea is accompanied by bradycardia there is a pronounced increase in the amplitude of the cardiac impulse on the respiration carrier. This imitates the respiration signal and prevents the alarm from sounding. Conversely, apnoea unaccompanied by bradycardia does not present this problem and is detected by the alarm. If impedance alarm systems are to be used to detect apnoea they must be accompanied by a heart rate (ECG) detector.

Impedance alarm systems are widely used for the detection of apnoea in the newborn infant. They are often used in the routine care of preterm infants, 25 to 84% of whom are likely to have apnoeic attacks.¹⁻² They have also been advocated for the protection of those infants at special risk of sudden infant death.³⁻⁴ Failures of impedance apnoea alarm systems, however, are known to occur and some have been reported.^{3,5} These failures have occurred with no detectable electrical fault in the monitoring system, and with effective electrode contact. We are currently using combined 24-hour electrocardiogram and respiratory tape recordings to study infants with apnoeic attacks. During this study a phenomenon was observed which could explain failures in impedance apnoea alarm systems.

Methods

Combined 24-hour tape recordings of ECG and respiration* were made from 4 pregelled electrodes. The respiration signal, from a separate portable impedance unit, was recorded from 2 electrodes

* Oxford Medical Systems.

placed 10 cm apart on one side of the chest wall, and the ECG signal from 2 electrodes positioned over the heart on the opposite side of the chest. The tapes were analysed by a newly developed system* programmed to detect both variations in heart rate and rhythm from preset R-R interval limits on the ECG and apnoea of greater than 10 seconds' duration from the respiration channel.

Patients and results

Combined recordings of ECG and respiration were performed on 3 infants with apnoeic attacks. A phenomenon was consistently documented in these infants, and in many others, which probably explains some of the failures of impedance apnoea alarm systems.

Case 1. This baby boy was admitted to hospital at age 3 weeks with episodes of pallor, cyanosis, and apnoea. He was term and weighed 2.36 kg at birth. He was monitored on an impedance alarm system (Hewlett Packard 78202B) set to trigger after more than 15 seconds of apnoea. A combined 24-hour ECG and respiration recording showed four episodes of apnoea (10-40 seconds in duration) with bradycardia of down to 66/min. The apnoea alarm failed to sound with any of these episodes and the 24-hour recording showed that during all episodes the size of the cardiac impulse during bradycardia increased, resembling the respiration signal (Fig. 1).

Case 2. This baby girl, who weighed 1.60 kg and was born at 31 weeks' gestation, presented at age 5 weeks with apnoeic episodes. She was monitored on a combined heart rate (from ECG) and apnoea (from impedance pneumograph) alarm system (Hewlett Packard 78213D cardiorespirograph). The alarms were set to trigger for heart rates below 100/min