SHORT REPORTS

Human parvovirus aplasia: case due to cross infection in a ward

Infection with human parvovirus is an important cause of aplastic crises in patients with sickle cell anaemia.¹ Recently the virus has been implicated in aplastic crises in hereditary spherocytosis² and pyruvate kinase deficiency.³ Epidemiological studies suggest that the virus causes an illness with an incubation period of about nine days,⁴ resulting in suppression of erythropoiesis for five to 10 days.

We report two cases of human parvovirus aplasia presenting in North London in which the infection was probably transmitted within a hospital ward and examine the implications of this finding.

Case reports

Case 1—A 4 year old girl was admitted on 9 July 1983 with a history of headache, vomiting, and fever for 24 hours. She suffered from hereditary spherocytosis and her haemoglobin concentration had been stable at around 10.5 g/dl. On examination she was seen to be pale and her spleen was palpable 3 cm below the left costal margin. Haemoglobin concentration was 7.8 g/dl, white cell count $7.9 \times 10^9/l$, and reticulocyte count 8%. After a blood transfusion her symptoms disappeared. She was discharged two days later with a haemoglobin concentration of 10.0 g/dl and a reticulocyte count of 0.2%. On 20 July she was readmitted; she did not have symptoms but her haemoglobin concentration was 6.8 g/dl and reticulocyte count 4.5% and she was given a second transfusion. Serum tests 12 days after presentation confirmed recent infection with human parvovirus: antihuman parvovirus IgM 56 radioimmunoassay units, antihuman parvovirus IgG 40 units. She subsequently required no more blood.

Case 2—A 12 year old girl with homozygous sickle cell anaemia was admitted on 8 July 1983 with an infarctive sickle crisis. On admission she had a haemoglobin concentration of 9.4 g/dl and a reticulocytosis of 8%. She was treated with fluids, antibiotics, and analgesics and discharged with a haemoglobin concentration of 8.9 g/dl and a reticulocytosis of 12.6%. On 20 July she began to have headache and fever and seven days later she was readmitted. Haemoglobin concentration was 3.3 g/dl, white cell count $10.9 \times 10^9/l$, and platelet count $340 \times 10^9/l$. She had complete reticulocytopenia. Her clinical condition improved after blood transfusion and she was discharged after four days, when reticulocytes were again detectable. Serum assays during the first admission showed no evidence of current or previous infection with human parvovirus. During her second admission high levels of human parvovirus specific IgM antibody were detected, indicating recent infection (table).

Haematological and virological data in case 2. (Contact with case 1 during 9-11 July 1983)

	Date			
	8/7/83	13/7/83	27/7/83	10/8/83
Haemoglobin concentration (g/dl)	9.4	8.7	3.3	9.8
Reticulocytes (%)	8.0	12.0	0	2.4
Antihuman parvovirus	{ IgM	<1.0	>100.0	>100.0
(radioimmunoassay units)				
	{ IgG	<0.3	12.5	64.0

Comment

Our interest in these two cases lies in the possibility of cross infection with human parvovirus in a hospital ward. The patient with hereditary spherocytosis was admitted with a feverish illness. She subsequently developed a low reticulocyte count and required transfusion on two occasions for the first time in her life. The patient with sickle cell anaemia was in hospital with an infarctive crisis when she was exposed to the patient with hereditary spherocytosis. She developed new symptoms between nine and 11 days after contact and was admitted one week later severely anaemic and with absent reticulocytes. The spread of aplastic crisis among patients with haemolytic anaemia in hospital has not been noted before, but the timing of contact and subsequent clinical course in our patients strongly suggest that the second was infected with human parvovirus by the first and developed an aplastic crisis as a result.

The epidemic nature of aplastic crisis in sickle cell anaemia is well recognised¹ and outbreaks in families with hereditary spherocytosis have been repeatedly reported. Human parvovirus appears to be the predominant cause.⁴

Infection with human parvovirus is prevalent wherever it has been

looked for. Some 30-40% of blood donors in Britain and Australia have antibody against the virus.² In normal people infection with the virus may be subclinical or may cause a mild feverish illness with vomiting, hypochondrial pain, and myalgia. It also appears to be the cause of erythema infectiosum (fifth disease),⁵ an acute exanthematous disease of children. Most infections are mild, but in patients whose red cells have an appreciably reduced life span erythroid suppression due to the virus results in a rapid and life threatening fall in haemoglobin concentration, as seen in our second patient.

Once an aplastic crisis is established management is supportive. It may, however, be possible to prevent aplastic crises with normal human immunoglobulin which has a high titre of antibody against human parvovirus. Its use should be considered in family and hospital contacts of human parvovirus infection who have haemolytic anaemia as soon as the risk is recognised.

This is the first report of probable spread of the disease in hospital and it emphasises the danger of contact between patients with possible parvovirus infection and known cases of haemolytic anaemia. In the management of haemolytic anaemias the threat of infection with human parvovirus, the potential protective effect of passive antibody, and the availability of specific tests for susceptibility and for current infection are all important considerations.

¹ Sergeant GR, Topley JM, Mason K, *et al.* Outbreak of aplastic crises in sickle cell anaemia associated with parvovirus-like agent. *Lancet* 1981;iii:595.

² Kelleher MD, Naomi LC, Luban MD, Mortimer PP, Kamimura T. Human serum "parvovirus." A specific cause of aplastic crises in children with hereditary spherocytosis. *J Pediatr* 1983;103:720-2.

³ Duncan JR, Potter CG, Cappellini MD, Kurtz JB, Anderson RJ, Weatherall DJ. Aplastic crisis due to parvovirus infection in pyruvate kinase deficiency. *Lancet* 1983;ii:14-6.

⁴ Mortimer PP. Hypothesis: the aplastic crisis of hereditary spherocytosis is due to a single transmissible agent. *J Clin Pathol* 1983;36:445-8.

⁵ Anderson MJ, Jones SE, Fisher-Hoch SP. Human parvovirus, the cause of erythema infectiosum (fifth disease)? *Lancet* 1983;ii:1378.

(Accepted 25 November 1983)

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Unusual dislocations associated with epileptic fits

Dislocation of a joint is a possible complication of epileptic fits. I report two cases of unusual joint dislocations resulting from epileptic fits that do not appear to have been described previously.

Case reports

Case 1—A 32 year old housewife with a history of three epileptic fits since childhood was noted by her husband to be walking around in a daze and not responding to questions. He then observed her sit down and start twitching in her arms and legs; her eyes rolled up, and she frothed at the mouth. She repeatedly flung her arms across her chest and finally upwards into the air, at which stage she became unrousable. On arrival at the accident and emergency department she was conscious but drowsy and disorientated. There was no evidence of incontinence, tongue biting, or other neurological signs. Further examination showed a deformity of both shoulders, which proved on x ray examination to be bilateral anterior dislocation of the shoulder joints plus a fractured right greater tuberosity. An electroencephalogram obtained at a later date was consistent with epilepsy. The dislocations were reduced easily under sedation with intravenous diazepam, and the arms were rested in

bilateral slings. After three weeks physiotherapy was started with good return of shoulder movements. She had not been receiving treatment before the incident but was subsequently given sodium valproate in an appropriate dosage with no further recurrence of her epileptic fits.

Case 2—A 26 year old woman attended the accident and emergency department having been unable to close her mouth since waking. Clinically her jaw was dislocated and required reduction. The following year she presented on seven further occasions over two months with an identical history. On each occasion the dislocation was reduced successfully under sedation with intravenous diazepam. On the last occasion her mother remarked that she had often heard noises from her daughter's bedroom and on entering would find her jerking her arms and legs, each episode lasting about one minute before she fell asleep again. No evidence of incontinence or tongue biting was noticed, but she would be unable to close her mouth on regaining consciousness. An electroencephalogram was consistent with a diagnosis of epilepsy. Her convulsive seizures and associated dislocations of the jaw disappeared once she had acquired a therapeutic blood concentration of phenytoin.

One other patient with epilepsy, who was mentally handicapped, presented to the accident and emergency department with dislocations similar to those seen in case 1. She was found to have long standing unreduced bilateral anterior dislocations of the shoulder joints, which may well have been related to her fits.

Comment

Posterior dislocation of the shoulder joint is the most common type of dislocation associated with epileptic seizures; it may be bilateral and associated with a fracture.¹⁻⁴ I have been able to find only one reference (a letter)⁵ to anterior dislocation of the shoulder joint, either unilateral or bilateral, occurring during epileptic fits. I could not find any reference to dislocations of the temporomandibular joints complicating epileptic fits. Clinicians should be aware that these complications may occur or, indeed, be the presenting features of epilepsy itself.

I thank Dr R G Daniels for his help in formulating this report and Dr R D Marshall for his helpful review.

¹ Honner R. Bilateral posterior dislocation of shoulders. *Aust NZ J Surg* 1969;38:269-72.

² Shaw JL. Bilateral posterior fracture—dislocation of the shoulder and other trauma caused by convulsive seizures. *J Bone Joint Surg [Am]* 1971;53:1437-40.

³ McLaughlin HL. Posterior dislocation of shoulder. *J Bone Joint Surg [Am]* 1952;34:584-90.

⁴ Prillaman HA, Thompson RC Jr. Bilateral posterior fracture-dislocation of the shoulder. A case report. *J Bone Joint Surg [Am]* 1969;51:1627-30.

⁵ Campbell AMG. Bilateral posterior dislocation of shoulders. *Br Med J* 1969;ii:822.

(Accepted 21 November 1983)

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Accidental childhood poisoning with household products

Accidental poisoning in childhood remains an important reason for admission to hospital and an occasional cause of death. The introduction of child resistant containers for medicines has led to a reduction in such poisonings,¹ and there are currently moves to extend the use of child resistant containers to household products.² It would clearly be unwise to package every household product in this way, and to define which substances would be the best candidates for such safety packaging we surveyed children admitted to hospital.

Patients, methods, and results

We studied the notes of all children aged under 5 admitted to hospital in Newcastle and South Glamorgan from 1974 to 1982 with a diagnosis of poisoning with a household product. The population of this age in the Newcastle catchment area was 29 100 and in South Glamorgan 27 000. Some children were also admitted to hospital in South Glamorgan from

outside the area. A total of 893 children were identified as having been poisoned, 439 from Newcastle and 380 from South Glamorgan with an additional 74 from elsewhere in south Wales. Thus one child in 597 in Newcastle and one in 639 in South Glamorgan were admitted to hospital with poisoning each year.

The table shows the major substances ingested and the proportion of cases with severe symptoms. These were defined as any symptoms other than transient ones such as coughing and spluttering or spontaneous vomiting. Of 89 children who were poisoned with bleach, none had more than transient perioral erythema or slight blistering. The duration of admission was noted in Newcastle: 380 children stayed in hospital for one day or less and only 14 stayed for four or more days. Of these 14, seven were detained in hospital for reasons other than the poisoning episode such as other medical problems or severe social problems highlighted by the admission.

Substances ingested that caused severe symptoms

	No of cases	No (%) with serious complications
Turpentine, white spirit, or turpentine substitute	195	12 (6)
Paraffin	45	5 (11)
Alcohol	34	7 (21)
Paint remover	12	3 (25)
Sodium hydroxide (caustic soda)	10	8 (80)
Camphor oil	10	1 (10)
Resin hardener	3	1 (33)
Strong acids	2	1 (50)
Antifreeze	1	1 (100)
Soldering flux	2	2 (100)
Total	314	41 (13)

Substances that did not cause serious symptoms: bleach (89 cases), rat or mouse poison (53), disinfectant (43), perfume or after shave (43), insecticide (33), cleaning fluids (28), nail varnish remover (22), window cleaner (21), shampoo (17), slug pellets (malaldehyde) (13), paint (13), and petrol (12).

Comment

In nine years in two major cities only 41 children had an episode of poisoning that caused serious symptoms, this being only 4.6% of the total number admitted to hospital poisoned with household products. These products fell into 10 different categories, one of which was alcohol. The remaining 95.4% of children did not develop serious symptoms, which confirms the report of Goulding *et al.*³ There are two groups of substances that children ingest: those that are commonly taken but rarely cause harm, and those that are rarely ingested but produce serious symptoms. Of the first group, the only one for which we consider that child resistant containers should be used is turpentine (including turpentine substitute and white spirit). We believe that there is insufficient evidence of toxicity from ingestion of bleach to justify using child resistant containers for bleach. Paraffin is rarely ingested from a bottle, and child resistant containers would not therefore prevent many cases of poisoning. Of the less common substances taken, the strongly caustic materials—such as paint remover, sodium hydroxide (caustic soda), strong acids, and possibly soldering flux, resin hardener, antifreeze, and camphor oil—should be in child resistant containers. The solution to the problem of ingestion of alcohol among young children probably lies in education rather than child resistant containers. Interestingly, the duration of stay in hospital, which has often been taken to indicate the severity of the poisoning episode, may be misleading in half the children staying for four days or more.

¹ Lawson GR, Craft AW, Jackson RH. Changing pattern of poisoning in children in Newcastle, 1974-81. *Br Med J* 1983;287:15-7.

² House of Commons Official Report (*Hansard*). *Dangerous household products (child safety) packaging bill*. 1982 January 29;16:1158-66. (No 46.)

³ Goulding R, Ashforth EK, Jenkins H. Household products and poisoning. *Br Med J* 1978;ii:286-7.

(Accepted 10 November 1983)

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