Prolonged erythrocyte T-polyagglutination in two children with bowel disorders

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SUMMARY *In vivo* erythrocyte polyagglutination of microbial origin is usually a transient condition. In two children with bowel disorders, erythrocyte T-polyagglutination persisted for 12 months in one case and for seven months in the other. Both children required blood transfusions to support surgery. Washed red cell concentrates were transfused instead of whole blood to prevent dangerous destruction of T-transformed erythrocytes by anti-T antibodies normally present in the plasma of blood donors.

Interest in erythrocyte polyagglutination, a source of error in blood grouping, has greatly increased in recent years. There are various forms of erythrocyte polyagglutination (Table 1): some are due to the action of bacterial or viral enzymes on the erythrocyte surface. In T-polyagglutination, the enzyme responsible is neuraminidase, which is produced by some viruses and many bacteria (Gottschalk, 1957; Müller, 1974): Tk-polyagglutination is caused by an unidentified enzyme produced by some strains of *Bacteroides fragilis* (Inglis *et al.*, 1975). For *in vivo* polyagglutination to occur, bacteria need not be in the blood stream; their enzymes may enter the blood stream by absorption from either an infected focus or a disordered gut (or lung) (Bird, 1977): more enzyme than is required to neutralise enzyme-inhibitors normally present in human plasma must enter the blood stream.

T- and Tk-polyagglutination are similar but not identical. In both forms, the red cells are agglutinated by a lectin obtained from peanuts, *Arachis hypogaea* (Bird, 1964; Bird and Wingham, 1972). However, T- and Tk-polyagglutination can be distinguished by application of the scheme given in Table 2.

Children with erythrocyte polyagglutination associated with bowel disorders (Bird and Stephenson, 1973) or respiratory infections (Rickard *et al.*, 1969) have been reported. The child described by Bird and Stephenson (1973) had intestinal obstruction and transient erythrocyte polyagglutination which was stated to be due to T-transformation; no mention was made, however, of the exclusion of Tk. Since it

Table 1 *Classification of erythrocyte polyagglutination*

Microbial	Acquired forms of erythrocyte polyagglutination
	i Passive adsorption of bacterial products by red cells
Non-microbial	ii Enzymic—T, Tk, acquired B, VA
	Tn
Inherited forms of erythrocyte polyagglutination:	
Strongest form of Cad (Super Sd ^a)	
HEMPAS	

Table 2 *Differences between T- and Tk-polyagglutination*

	T	Tk
Sialic acid level	Reduced	Normal
Aggregation by polybrene	—	+
Agglutination by peanut lectin	+	+
Agglutination by soya bean lectin	+	—
Agglutination by BS II lectin*	—	+
AB serum after absorption of anti-T	—	+
AB serum after absorption of anti-Tk	+	—
Effect of ficin on receptor	Damaged	Enhanced

* A second lectin isolated from the seeds of *Bandeiraea simplicifolia* (Judd *et al.*, 1975): test in albumin at 4°.

has been observed by two of us (GWGB and JW) that a prolonged form of T-polyagglutination is sometimes seen in children with bowel disorders, we thought it might be useful to describe two recent examples.

Case reports

CASE 1

A 22-day-old boy was admitted to hospital because of failure to thrive. On the day after birth he had

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abdominal distension and vomiting, and he had passed no faeces since delivery. A simple abdominal x-ray confirmed the diagnosis of intestinal obstruction. An enema released a small plug of meconium. He remained constipated until he was admitted to hospital. Rectal biopsy established a diagnosis of Hirschsprung's disease with non-specific colitis. Blood grouping showed that he was group AB, Rhesus positive, whereas his mother's group was O, Rhesus positive. Investigation of this anomaly showed that the baby's cells were polyagglutinable and that they were strongly agglutinated by peanut and soya-bean extracts and not aggregated by polybrene. More elaborate tests (absorption, sialic acid estimation, etc) confirmed that they were T- and not Tk- polyagglutinable. There was no evidence of haemolytic anaemia. Blood culture was sterile. Urine examination showed the presence of staphylococci—1000/ml; *Escherichia coli*—1000/ml; and *Streptococcus faecalis*—10 000/ml: these numbers are probably of no significance.

Since his Hb was 7 g/dl he was transfused with 100 ml of group O, Rhesus positive, washed red cell concentrate before a left iliac colostomy was done. Washed red cell concentrate was given instead of whole blood because most human adult plasma contains anti-T.

CASE 2

A 16-day-old boy was admitted to hospital because of continual vomiting. On the sixth day after birth he vomited bile, and on the 10th day there was melaena. He continued to vomit bile and had 'offensive' and bloody stools. On admission a simple abdominal x-ray suggested malrotation of the mid-gut. The baby (group A, Rhesus positive) was transfused with 30 ml of group A, Rhesus positive red cell concentrate. At laparotomy volvulus neonatorum was found and corrected. There was some doubt about the viability of the small bowel but no resection was done at this stage. On the fourth day after operation the baby's condition deteriorated, and a second laparotomy was performed. Most of the small intestine was found to be infarcted and was resected. *E. coli* 099 was isolated from the peritoneal fluid. Blood culture was sterile. A week later a second blood transfusion was needed. The blood group at this stage appeared to be AB, Rhesus positive. This anomaly was investigated and found to be caused by T-polyagglutination by the methods used for case 1. The baby was transfused with 30 ml of group A, Rhesus positive washed red cell concentrate without any ill-effects.

Discussion

Since each patient had a gut disorder, it is reasonable to assume that the inflamed bowel permitted the

transfer of enough bacterial neuraminidase to the blood stream to cause T-polyagglutination. There was no haemolytic anaemia in either case, which is not surprising since anti-T is not present in the sera of young children. Neither patient suffered any ill-effects from transfusion of red cell concentrate, whereas the infant reported by Van Loghem *et al.* (1955) had a fatal reaction, and the child described by Bird and Stephenson (1973) a severe reaction, after the transfusion of whole blood, the plasma of which contained anti-T antibodies.

Polyagglutination disappeared after 12 months in case 1 and after seven months in case 2. Microbial polyagglutination is usually transient; however, the two patients described in this report showed that T-polyagglutination associated with bowel disorders in children may persist for months.

In conclusion, we must emphasise that all blood group AB findings in very young babies should be carefully checked to exclude erythrocyte polyagglutination as a source of error. The necessity for this precaution in blood grouping in connexion with blood transfusion or paternity testing should be obvious.

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