required when the meter reading differs from $P_{aO_2}$ in a blood sample by more than $\pm 11.5$ mmHg (95% confidence).

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References

Haemorrhage responsive to vitamin K in a 6-week-old infant

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SUMMARY

A 6-week-old breast-fed infant presented with vomiting, jaundice, and irritability. Haemorrhage occurred after lumbar puncture, and a coagulation abnormality which responded to vitamin K was found. It would seem prudent to estimate the prothrombin time before invasive procedures in breast-fed infants of this age, or to give vitamin K to such infants when doubt exists about previous vitamin K administration.

Case report

A term baby boy weighing 3450 g was delivered after spontaneous onset of labour to a primagravid mother. Apgar scores were 3 at one minute and 5 at five minutes. He was resuscitated with oxygen delivered by face mask. Mild, presumed physiological jaundice developed on day 3 but otherwise the neonatal period was uneventful. Neither mother nor baby received vitamin K. He was breast fed. Slight jaundice persisted after his discharge on day 5 but he remained well until admission at 6 weeks with a 2-day history of nonprojectile vomiting after feeds without haemorrhage. For 5 days there had been slight irritability on handling, but sleep, feeding, and bowel function were all normal. The icterus had intensified. There was no history of maternal or infant drug ingestion, but both parents had developed upper respiratory tract infections 5 days previously.

On admission the baby was afebrile, slightly jaundiced, mildly dehydrated, and irritable on handling. Weight was on the 50th centile with length and head circumference on the 90th centile for age. The anterior fontanelle was full but not tense. Systematic and neurological examination, including fundoscopy, were normal. No clinical evidence of pyloric stenosis was detected. Initial investigations included a haemoglobin of 9.1 g/dl, a white blood count (WBC) of $9.2 \times 10^6/\text{l}$ (9200/mm$^3$) (neutrophils 35%, lymphocytes 65%), and a platelet count of $360 \times 10^6/\text{l}$ (360 000/mm$^3$). The film contained a few atypical lymphocytes. Serum electrolytes were within normal limits. Serum total bilirubin was $90 \mu\text{mol/l}$; $5-3 \text{ mg/100ml}$ (direct $39 \mu\text{mol/l}$; $2-3 \text{ mg/100ml}$). Full infection screen, including CSF analysis, showed no evidence of a bacterial infection.
Within 8 hours he became pale and lethargic. An extensive haematoma at the lumbar puncture site and oozing from the venepuncture site were noted, but no petechiae or ecchymosis. He remained afebrile. His haemoglobin had fallen to 7·4 g/dl, the WBC had risen to 13·9 × 10⁹/1; 13 900/mm³ (neutrophils 53 %, lymphocytes 38 %), and the platelets were 450 × 10⁹/l (450 000/mm³). His blood film showed no evidence of red cell fragmentation nor of toxic neutrophil changes. Coagulation screen showed a prothrombin time of 300 seconds (control 13 s), a PTTK of 120 seconds (control 37 s) and serum fibrinogen of 1·7 g/l; (170 mg/100ml). His serum electrolytes were unchanged. The direct Coombs's test was negative and the serum bilirubin was unchanged. Hepatic enzymes showed no increase with an ALT of 49 units/l and an AST of 52 units/l (normal values for each 5–120 units/l). Serum alkaline phosphatase was 599 new units/l. Serum T4 was 150 nmol/l (11·65 μg/100ml). Urine contained no reducing substances and there was no evidence of bacterial or viral pathogens from cultures of urine, stools, blood, or throat and nose swabs. There was no alteration in viral titres to CMV, herpes simplex, rubella, and toxoplasma. He was Australia antigen negative. Maternal prothrombin time was 15 seconds. Treatment included transfusion with packed red cells (80 ml), 2 mg IV vitamin K, and gentamicin, cloxacillin, and IV fluids. Within 12 hours the prothrombin time was corrected to 15 seconds (control 15 s), the PTTK to 34 seconds (control 35 s), and the haemoglobin raised to 10·7 g/dl. There was no other alteration in the coagulation screen. Fibrin degradation products were normal throughout. No further bleeding occurred. Mild fever persisted for some 3 days but without emesis. Weight gain continued with breast feeding and he showed no further clinical or laboratory disturbance of coagulation during the ensuing week. One week after admission serum bilirubin had fallen to 19 μmol/l (1·1 mg/100ml).

Two months later he was thriving with no further haemorrhagic sequelae.

Discussion

This infant presented with vomiting and an increasing jaundice at 6 weeks. No evidence of bacterial infection was found and, although no viruses were isolated, there had been a presumed viral infection in the family and, in addition, atypical lymphocytes were seen on the blood film.

The prolonged neonatal jaundice may have been related to breast feeding or liver immaturity but normal liver enzymes excluded gross hepatic disease. There was no evidence of congenital infection, galactosaemia, or hypothyroidism. The jaundice may have been exacerbated by decreased fluid intake, and emesis with the infection.

We presume that the bleeding after lumbar puncture was related to vitamin K deficiency in view of the prolonged prothrombin and partial thromboplastin times and their rapid correction with vitamin K.

Normal platelet counts, serum fibrinogen, fibrin degradation products, and blood film excluded a consumptive coagulopathy. There was no maternal history of any drug ingestion.

In classical haemorrhagic disease of the newborn, bleeding occurs as a result of deficiency of vitamin K-dependent clotting factors and is virtually confined to breast-fed babies. It is preventable if vitamin K is given at birth. A haemorrhagic syndrome with a similar coagulation abnormality occurs beyond the neonatal period, usually between 4 and 12 weeks.

Factors which have been previously implicated are: (1) decreased vitamin K intake due to breast feeding; (2) prolonged diarrhoea; (3) antibiotics causing an alteration in gut flora. Nammacher et al. (1970) described 4 such infants aged between 5 and 8 weeks. None had been given vitamin K at birth and 3 were breast fed; 2 had intracranial bleeding and the coagulation abnormality was corrected with vitamin K in all 4. Other cases have been similarly described in association with diarrhoea and antibiotics (Goldman and Deposito, 1966).

In this case, the baby had not received vitamin K at birth and was entirely breast fed with a presumed defective vitamin K intake. Breast milk contains 15 μg/l vitamin K whereas cows' milk has a higher level of 60 μg/l. He had not been given antibiotics nor had he had diarrhoea. It would appear that breast feeding alone was responsible for his vitamin K deficiency state, and perhaps the coagulation abnormality would not have come to light had he not developed an infection necessitating lumbar puncture and venepuncture.

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