SHORT REPORTS

Value of measuring cord blood bilirubin concentration in ABO incompatibility

Risemberg *et al*[1] suggested that "the risk to infants with ABO incompatibility and cord blood bilirubin levels greater than 68 μmol/l (4 mg/100 ml) is so consistent that frequent re-evaluation is mandatory." As this was not our impression we studied cord blood bilirubin concentrations in 2117 consecutive births at Hope Hospital.

Methods and results

A 2-ml sample of cord blood was obtained without milking the cord and stored in a heparinised plastic tube. When necessary the sample was kept overnight for not more than 10 hours in a refrigerator protected from light. The bilirubin concentrations were measured in the hospital biochemistry department by an American Optical Corporation No 10200 bilirubinometer, a direct reading bilirubinometer that correlates well with spectrophotometric methods.[2] The mother's blood was grouped on the following indications: (1) jaundice on the first day; (2) a cord blood bilirubin concentration of 68 μmol/l (4 mg/100 ml) or more; (3) severe hyperbilirubinaemia—that is, 273 μmol/l (16 mg/100 ml) at 12-36 hours of age. Blood was taken from the infant for haemoglobin and direct Coombs test at the same time.

Out of the 2117 infants 212 (10%) required treatment for jaundice. ABO incompatibility was defined as follows[3]: mother group O and infant group A, B, or AB; mother group A and infant group B, or vice versa; presence of anti-A or anti-B haemolysins.[4] Haemolysins were present in 48 (22·6%) jaundiced infants, all of whom had anti-A haemolysin. Other possible causes of jaundice were excluded as far as possible. None of the 212 infants had a cord blood bilirubin concentration above 68 μmol/l, range 2 μmol to 51 μmol/l, mean (±SD) 8 μmol/l±6·0 μmol/l (range 0·10 mg/100 ml to 3 mg/100 ml±0·35 mg/100 ml), but two had concentrations of 51 μmol/l (3 mg/100 ml) and 47 μmol/l (2·75 mg/100 ml) respectively. The infant with cord blood bilirubin of 51 μmol/l required one exchange transfusion when his serum bilirubin was 346 μmol/l (20·25 mg/100 ml). He was the only one who required an exchange transfusion in this group (table). The subsequent bilirubin concentrations in these 212 infants ranged from 150 μmol/l to 346 μmol/l (8·75 mg/100 ml to 20·25 mg/100 ml), mean 198·5 μmol/l± 46·4 μmol/l (11·50 mg/100 ml±2·75 mg/100 ml). The Coombs test was positive in 15 (31·2%) infants with ABO incompatibility.

Relationship between cord blood bilirubin concentrations in 48 infants and degree of hyperbilirubinaemia (insignificant, moderate, or severe) at 36-48 hours, Coombs test, and exchange transfusion

	Serum bilirubin concentrations (μmol/l), range and mean (±SD)		
	150-171 (163±6·4)	171-257 (203·7±23·1)	273 (328±25·2)
No with cord bilirubin concentration:			
<68 μmol/l	10	36	2
>68 μmol/l	—	—	—
No Coombs test positive	—	13	2
Exchange transfusion	—	—	1

Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 0·06 mg/100 ml.

Comment

There are no definite criteria of ABO haemolytic disease that could predict immediately after birth which infants may require treatment for hyperbilirubinaemia, yet increased pressure on hospital beds and early discharge policies make early diagnosis important. Risemberg *et al*[1] suggested that a cord blood bilirubin concentration of 68 μmol/l was significant. Robinson *et al*[5] thought that a concentration of 51 μmol/l was highly suggestive of ABO haemolytic disease. In our 48 cases no baby had a concentration over 68 μmol/l and only one infant had a concentration of 51 μmol/l. There were 38 (79·1%) infants who had concentrations under 51 μmol/l who required treatment, which we gave as phototherapy. In all infants who had insignificant bilirubinaemia—that is, 171 μmol/l (10 mg/100 ml) or less—the Coombs test was negative. We consider that the cord blood bilirubin level is unreliable for predicting hyperbilirubinaemia due to ABO incompatibility.

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Spontaneous pneumomediastinum complicating anorexia nervosa

We report two cases of apparently spontaneous pneumomediastinum occurring in girls with anorexia nervosa.

Case reports

(1) A 15-year-old girl presented as an emergency to the casualty department with weight loss and weakness. She weighed 52 lb (23·6 kg) and was 61 in (1·53 m) tall. Anorexia nervosa had been diagnosed six months previously at another hospital, but she had defaulted from outpatient follow-up after three months, when she weighed 70 lb (31·8 kg). She had many features of the condition—cachexia, scaphoid abdomen, preservation of the breasts, lanugo, erythema ab igne, bradycardia (pulse rate 44/min), hypotension (blood pressure 80/50 mm Hg), pretibial petechial rash, and denial of illness. The diagnosis was consistent with Feighner's criteria.[1]

Chest radiographs 24 hours after admission showed pneumomediastinum and pneumopericardium. Later surgical emphysema of the neck developed. Meanwhile, a nasogastric feeding tube had been passed without difficulty. There was no history of lung disease, hyperventilation, or self-induced vomiting and barium swallow and meal radiographs were normal. The nasogastric tube was left in situ, she remained asymptomatic, and within five days the surgical emphysema had resolved and the *x*-ray pictures were normal. She made a good recovery and weighed 84 lb (38 kg) when transferred to a psychiatric unit six weeks later. Within seven weeks normal menstruation had resumed.

(2) A 17-year-old girl was admitted to hospital with a four-month history of weight loss, amenorrhoea, anorexia, and vomiting and many of the classical features of anorexia nervosa. Her weight was 91 lb (41·4 kg) and her height 61 in (1·53 m). The diagnosis was in accordance with Feighner's criteria. Surgical emphysema of the neck was noted and a chest radiograph showed mediastinal emphysema. Barium swallow and meal radiographs and panendoscopy of the respiratory tract and oesophagus showed no abnormality. She recovered without further complication. The emphysema resolved both clinically and radiologically within seven days. She weighed 93 lb (43·8 kg) when discharged to psychiatric outpatients.

Comment

We conclude, in the absence of other causes, that in the first case pneumomediastinum developed spontaneously. Vomiting may have been a contributory factor in the second case. One case of emphysema, pneumomediastinum, and pneumoretroperitoneum in functional anorexia has been reported,[2] but, in an extensive search, we have not