Lesson of the Week

Missed jaundice in black infants: a hazard?

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Although it seems self evident that severe neonatal jaundice may be missed or underestimated in dark-skinned infants we have been unable to find published reports about this hazard. There are no figures to show how many of such infants suffer avoidable morbidity. We report on two babies, one West Indian and one West African, whose neonatal jaundice was diagnosed and treated late. Both required exchange transfusions. One of them was premature and had underlying glucose 6 phosphate dehydrogenase deficiency and developed kernicterus.

Jaundice should be suspected in non-white infants because of the high incidence of glucose 6 phosphate dehydrogenase deficiency

Case reports

Case 1—A black boy weighing 2180 g was born at 33 weeks' gestation to West Indian parents. He was admitted to a special care nursery and was initially fed through a nasogastric tube. His progress was uneventful until the eighth day when a doctor noticed that his sclerae were yellow. His mother had noticed this on the third day but had not wanted to trouble the staff by asking about it. The total serum bilirubin concentration was 550 μmol/l (32 mg/100 ml) with a conjugated fraction of 30 μmol/l (1·8 mg/100 ml). His blood group was A rhesus negative. His direct Coombs test was negative, the haemoglobin concentration was 12·7 g/dl, and his blood film showed target cells and schistocytes with poikilocytosis. His haemoglobin electrophoresis was normal for age and his urine sterile and free from reducing substances. His red cell glucose 6 phosphate dehydrogenase activity was 30 mU/10^9 red cells (normal 100-160 mU/10^9 red cells), confirming glucose 6 phosphate dehydrogenase deficiency.

Phototherapy was begun immediately the bilirubin concentration was known. He received two exchange transfusions of fresh whole blood after which the jaundice resolved satisfactorily. Shortly before the first exchange transfusion he developed neck retraction followed by frank opisthotonos with intermittent extensor spasms of the arms, general irritability, and a shrill cry.

At a chronological age of 15 months he had generally increased muscle tone that was greater in the arms than the legs, asymmetrical tonic neck reflex, and athetoid-like movements. At 12 months he had a motor developmental score of 9½ months assessed on the Griffiths scale.[1] It has not yet been possible to do a formal audiometric examination, but no clinical impairment of hearing has been detected.

Case 2—A brown-skinned girl was born at 38 weeks' gestation weighing 3360 g to Nigerian parents. She was breast fed from birth and nursed in hospital. On the fourth day she was thought to be mildly jaundiced. The total serum bilirubin concentration was 475 μmol/l (28 mg/100 ml) with a conjugated fraction of 40 μmol/l (2·4 mg/100 ml). She had lost 15% of her birth weight. On admission to the special care nursery her plasma sodium concentration was 138 mmol(mEq)/l, urea concentration 9·1 mmol/l (55 mg/100 ml), haemoglobin concentration 16·6 g/dl, and there was no evidence of haemolysis on the blood film. The reticulocyte count was 2%. Her blood group was B rhesus positive, and the direct Coombs test was negative. Her mother's blood group was O rhesus positive with anti B agglutinins present in maternal serum at a titre of 1 in 64. The urine was sterile and contained no reducing substances. Her red cell glucose 6 phosphate dehydrogenase activity was normal. A diagnosis of physiological jaundice exacerbated by dehydration was made. The baby was rehydrated intravenously and phototherapy was begun. After an exchange transfusion the jaundice resolved quickly. There were no abnormalities on examination when she was discharged or when reviewed at 6 weeks of age.

Comment

These two cases illustrate that severe jaundice may easily be missed or underestimated in black or brown-skinned newborn infants. At age 15 months the baby in case 1 had signs of athetoid cerebral palsy due to kernicterus. Had his jaundice been detected earlier than the eighth day of life this may have been averted. Had phototherapy been started earlier in the baby in case 2 an exchange transfusion may have been avoided. Careful assessment, particularly of the sclerae and also of nose, gums, and tongue, is important in non-white infants if jaundice is to be detected early.

Neonatal jaundice should be expected in non-white infants because the incidence of glucose 6 phosphate dehydrogenase deficiency is high. This has been reported to be greater than 10% among healthy newborn infants in many countries of Africa, the West Indies, the Middle and Far East, and Asia.[2] The results of a study from Jamaica estimated that 9·4% of healthy newborn infants were deficient in glucose 6 phosphate dehydrogenase, and in severely jaundiced babies the proportion was 70%.[3] Among Nigerian infants with serum bilirubin concentrations above 340 μmol/l (20 mg/100 ml) 51% had glucose 6 phosphate dehydrogenase deficiency.[4] In Cape Town kernicterus occurred in 2·2% of infants with glucose 6 phosphate dehydrogenase deficiency.[5] Although haemolytic jaundice in such deficient infants may be precipitated by infections, hypoglycaemia, or drugs there is often no apparent cause and often no evidence of

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haemolysis.[6] The higher incidence of neonatal jaundice in babies with this deficiency may be due to a coexistent reduction in liver conjugation.[7] Some controversy exists over whether phototherapy itself initiates haemolysis in infants with such deficiency[8] since normal red cells in vitro become glucose 6 phosphate dehydrogenase deficient on exposure to phototherapy, perhaps because photo-oxidation of riboflavin may result in decreased production of nicotinamide-adenine dinucleotide phosphate.[9] But the clinical evidence of reduced concentrations of riboflavin in the blood[10] and increased haemolysis[11] due to phototherapy in glucose 6 phosphate dehydrogenase deficient infants is inconclusive.

In countries where the incidence of glucose 6 phosphate dehydrogenase deficiency is high, such as Singapore,[12] cord blood is sometimes routinely screened for this. There is at present no evidence that screening cord or peripheral blood of affected ethnic groups in Britain would be justified. There is in any case no substitute for a careful examination for jaundice and awareness of its relevance in non-white infants.

The icterometer[13 14] may help to make a diagnosis (Thomas Ingram, Santon Works, PO Box 305, Solo Hill, Birmingham B19 1BB). It is useful as a screening test to assess jaundice. It consists of a perspex bar on which is printed a series of yellow strips of increasing intensity. These are compared with the yellowness of the underlying skin on which it is pressed. The icterometer consistently underestimates jaundice in dark-skinned babies.[15] Better results may perhaps be obtained if it is used on the gums of such infants.[14 16] A spectrophotometric transcutaneous bilirubinometer has been described as unreliable on pigmented skin,[17] and both of these devices are inaccurate after phototherapy or exchange transfusion has been done.

We think that parents should be encouraged to report jaundice in their babies. Some description of jaundice in the newborn could be included in antenatal classes and in booklets, stressing its harmlessness in most babies but explaining the need for careful examination, particularly in dark-skinned babies. In a survey of the latest editions of 46 textbooks only five mentioned that it was difficult to estimate jaundice clinically in non-white infants.[14 18-21] We wonder how commonly this hazard is encountered and should be interested to hear of the experience of others.

We thank Dr C L Newman, Royal Berkshire Hospital, Reading, for permission to report on the patient in case 1 and Dr E E Jones, Hospital of St Cross, Rugby, for follow-up information on this child.

We are grateful to Professor Sir Peter Tizard, for his helpful criticism, and to Professor D Morley for advice. A tape slide set on prevention of newborn kernicterus in developing countries is available from the Foundation for Teaching Aids at Low Cost, Institute of Child Health, 30 Guilford St, London WC1N 1EH.

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(*Accepted 12 November 1982*)

Clinical curio: Beethoven—cause and cure of postcardiac bypass blues

Depression after surgery has been well documented, and a large number of contributing factors have been cited. I would like to relate one factor which has yet to be mentioned in the literature.

My father recently underwent quadruple bypass surgery. While in the surgical coronary intensive care unit, and just after being extubated he indicated that he would like to hear the slow movements from the Beethoven quartets. I subsequently recorded these movements from all the Beethoven quartets for him. During the next three days he made rapid physical improvement but he became very depressed. He insisted that the slow movements of the Beethoven quartets were the only music he could tolerate listening to as the music was soothing. On the fifth day after surgery, and still very depressed, my father stated emphatically "these quartets are killing me." He related to me that he was continually hearing in his mind the opening bars from the adagio molto e mesto (very slow and sad) from the quartet in F, opus 59, No 1 ("Rasoumoffsky"). It is interesting to note what Beethoven wrote in the margin of the manuscript for this movement: "... a weeping willow or an acacia tree over my brother's grave," in an apparent reference to his baby brother who had died.

From that point on we altered my father's musical diet—first with Beethoven's ninth symphony and then with Rossini overtures. Within hours of this change he commented that his spirits were uplifted. Possibly, one indication that he is cured will be when he is able to listen to a complete performance of the *Rite of Spring* by Stravinsky. Obviously there is a need for well controlled studies in this regard.—MARC A STRASSBURG, Los Angeles, USA.

Does electrolysis, as carried out in hairdressers' shops, carry a risk of spreading hepatitis B?

Any insertion of a needle through the skin may result in contamination with blood and therefore carries a potential risk of spreading hepatitis B virus. This is unlikely in electrolysis if the operator is skilled, but the best method of avoiding the risk of spread would be to subject the electrodes to a temperature of 100°C for two minutes after each customer. This would inactivate any virus particles.—J B SAUNDERS, lecturer in liver diseases, London.