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From Paediatric Unit, Kandang Kerbau Hospital for Women.
Moderator: DOCTOR GWEN SMITH.

Haemorrhagic Disease of the Newborn

3 Cases presenting with haemorrhage under the scalp

Case Reports

Presented by Doctor Tay Kah Seng.

FIRST CASE: Regd. No. 23752. Chinese female baby.

Birth Weight: 6 pounds 8 ounces.

The mother had been admitted into hospital for pre-eclamptic toxaemia and anaemia (Hb. 40%) and abruptio placentae. She had received 4 pints of blood. Delivery was by Kielland's forceps, rotation for persistent occipito posterior position.

Baby was well until the third day when pallor and enlargement of the head was noted.

Examination showed the baby to be in a state of peripheral circulatory collapse. The face was puffy and slightly jaundiced. The scalp was swollen especially in the temporal regions. A fluctuant haematoma was present beneath the whole of the scalp, limited laterally by the zygomatic arches, anteriorly by the attachment of the aponeurosis and posteriorly by the nuchal lines. The fronto-occipital circumference of her head was 15¾ inches. The spleen and liver were not palpable. Hb. 49%.

Treatment: Vit. K 2.5 mgm. I/M. and transfusion of 100 c.c. of blood. The condition improved. Hb. the next day 108%. She took feeds well, gained weight, and was discharged well on the 11th day.

SECOND CASE: Regd. No. 119. Chinese female baby.

Birth Weight: 7 pounds 8 ounces.

Delivered by Kielland's forceps. Indication: severe pre-eclamptic toxaemia and persistent occipito-posterior position. Both mother and baby were well after delivery.

On the third day, the baby vomited altered blood. Examination showed severe pallor and fresh bleeding from the umbilicus. There was a huge fluctuant haematoma under his scalp similar to that of Case 1. The fronto-occipital circumference of the head was 15 1/8 inches. The lower limbs were spastic. The spleen and liver were not palpable.

Investigations: Hb. 45%. Bleeding time — 1 minute. Clotting time — 4½ minutes. Prothrombin time (Quick's method) — 1 minute 20 seconds. Platelet count 170,000.

TREATMENT: Vitamin K. 2.5 mgm. I/M followed by blood transfusion of 120 c.c. and Syrup Chloral gr. 1 x 6 hourly were given.

The next day Hb. 90%. There was no recurrence of bleeding. One week later the fronto-occipital circumference was still 15 inches and the sagittal suture was very wide. The lower limbs were still spastic. Prednisolone 5 mgm. daily was prescribed to prevent fibrosis of intracranial blood clot. Bilateral subdural taps excluded subdural haematomata. The baby's condition gradually improved. Prednisolone was discontinued after 7 days. The circumference of the head was then 14½ inches. The baby was discharged well the 20th day. Hb. 95%.

THIRD CASE: Malay male baby. Birth Weight: 7 pounds 4 ounces.

Delivered by Barnes-Neville's forceps after prolonged labour. Head was in mid-transverse arrest. The second stage of labour was 3 hours. Manual rotation of the head before forceps were applied. Both mother and baby were well after delivery.

On the third day, the baby was reported to be feeding badly. Examination

showed pallor and a haematoma under the scalp similar to that of cases 1 and 2. Fronto-occipital circumference 15 inches. The liver was just palpable and the spleen was not palpable.

INVESTIGATION: Hb. 40%. Bleeding time — 6½ minutes. Clotting time — 21 minutes, Prothrombin time (Quick's method)—7 minutes 19 seconds. and Platelet count 160,000 per cub. m.m.

TREATMENT: Vit. K. 5 mgm. was given I/M. The baby developed acute circulatory failure. Blood transfusion 140 c.c' improved his condition and Hb. the next day was 70%. Improvement continued for 24 hours. and then gross haematuria, melaena. haematemesis and abdominal distension developed and he died. Consent for post-mostem was refused.

Discussion

Doctor Field, in opening the discussion. said that these 3 cases were particularly interesting because she had not seen this form of haemorrhage before. Two of the cases presented with pallor. On examination, enlargement of the head was found: due to haemorrhage under the scalp. A diagnosis of haemorrhagic disease of the newborn was considered and in two cases was probably correct. The third case was interesting because the haemorrhage did not stop after Vit. K therapy and a blood transfusion. The important clinical point is that, if sudden pallor occurs on or about the third day, concealed haemorrhage should be looked for. The site of the bleeding might be the gastro-intestinal tract, the brain, lungs or bladder. This was the first time that she had seen extensive haemorrhage occurring under the scalp.

Doctor Tay revised the anatomy of the scalp. He said that the scalp was made up of five parts: the skin, superficial fascia, muscular aponeurotic layer, loose areolar tissue and pericranium. The skin was thin and studded by hair follicles. The superficial fascia was dense, tough and fibrous, adherent to the skin above. and to the epicranial aponeurosis below, except over muscles. The third layer consisted of the frontal and occipital muscles and their aponeurosis. The frontalis muscle had no bony attachments. It merged with the aponeurosis behind. In front it was inserted into the skin of the lower part of the forehead, merged with the orbicularis oculi, and a few fibres attached to the skin of the nose. The occipital muscles were inserted into the superior nuchal line. The aponeurosis was a tendinous sheet continuous with the two muscles described At the sides, it was adherent to the temporal lines. It also sent a strong attachment to the zygomatic arches and the pinna of the ears. The fourth layer of the scalp was made up of loose areolar tissue. Its looseness allowed the first three layers to move over the cranium. Effusion of blood in this layer would therefore raise the scalp from the bone, but blood could not pass readily into either the face, temple, or the back of the neck. The last layer was the pericranium. Bleeding under this last layer was limited by the suture lines, and formed the classical cephalhaematoma.

The scalp was richly supplied with blood vessels, which entered it from the periphery. On each side they were the superficial temporal artery, (one of the 2 terminal branches of the external carotid), the posterior auricular, the occipital, the supra-orbital and the supra-trochlear and in the temporal region, the middle and deep temporal vessels.

In a forceps delivery, when the blades were correctly applied it was easy to realise that vessels in the temporal region could be traumatised.

He next discussed the mechanism of blood coagulation. The clotting process according to the classical theory of Morawitz (1905) occurred in two stages:

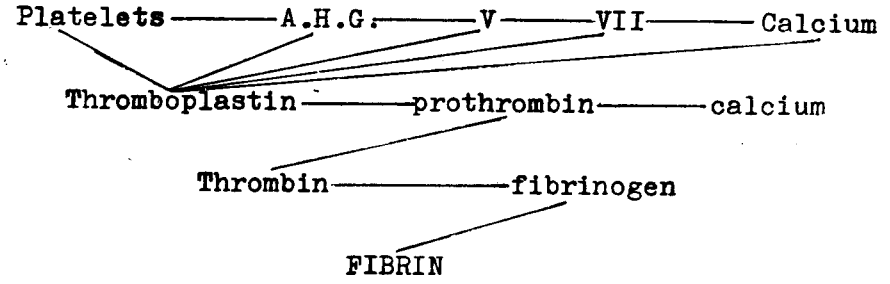
(i) Thromboplastin + prothrombin + calcium ⟶ thrombin.

(ii) Thrombin + fibrinogen ⟶ fibrin

Plasma proteins were formed exclusively in the liver. In the newborn the plasma proteins were low and this may be exaggerated by poor maternal diet. Owren (1947) discovered factor V. and since then a large number of new clotting factors have been described.

FACTORS	SYNONYMS
V	Proaccelerin; labile factor; accelerator globulin.
VI	Accelerin (somewhat hypothetical).
VII	Proconvertin; serum prothrombin conversion accelerator or s.p.c.a.; co-thromboplastin.
VIII	Antihaemophilic globulin or A.H.G.; Antihaemophilic factor A.
IX	Christmas factor; plasma thromboplastin component or P.T.C.; antihaemophilic factor B.
X	Koller's factor (somewhat hypothetical).
XI	Plasma thrmboplastin antecendent or P.T.A.

According to the theory of Biggs & Macfarlane (Wilson 1956), platelets, antihaemophilic globulin and factors V, VII and IX (Christmas) combine to form blood (intrinsic) thromboplastin. It is thus represented:



Doctor Smith discussed these factors in relation to haemorrhagic diseases. Fibrinogenopenia could occur in liver diseases, nutritional deficiency disorders, toxaemias of pregnancy, and as a sequel of antepartum haemorrhages. The production of prothrombin by the liver was dependent on the catalytic action of Vitamin K. which was a fat soluble naphthoquinone derivative. Vitamin K. was formed by coliform organisms in the gut. Its absorption depended on the presence of bile salts. Hence haemorrhagic disease due to Vitamin K. deficiency was seen only in the first few days of life when the gut was sterile, and in cases of biliary obstruction. Thromboplastin was derived from disintegrated platelets, and was absent in thrombocytopenic purpura.

Referring to the first case, she regretted that no laboratory data were available before blood transfusion. It was unlikely that the stored blood given would have affected the platelet count. The mother had anaemia and toxaemia. This case probably fell into the low prothrombin group due to Vitamin K. deficiency, but it was not possible to exclude deficiency of other factors. There had been no bleeding disorders in the family of the 3rd baby to suggest haemophilia. The first transfusion corrected the anaemia but did not stop the bleeding, and had he been re-transfused, his life might have been saved.

In the second and third cases, the prothrombin times were definitely prolonged, being 1 minute 20 seconds and 7 minutes 19 seconds respectively.

She added that haemorrhagic disease of the newborn was said to occur once in two hundred cases (Stone 1945). Dam & Schnheyder (1935) attributed the disease

to a deficiency of Vitamin K The value of routine administration of prophylactic Vitamin K. before delivery has been disputed. Potter (1945) reported no difference in the incidence but, Smith and others (1953) claimed a reduction in incidence from 6 per 1,000 to 0.24 per 1,000 following prophylactic administration of Vitamin K, and improvements in diagnostic criteria.

It was generally agreed that Vitamin K should be given in small doses to all infants following difficult or traumatic deliveries, as it was likely for intracranial haemorrhage or bleeding elsewhere to occur. Certainly prophylaxis should be given to babies subjected to operation in the first week of life. Vitamin K in large doses was a liver poison. Large doses given to premature infants exaggerated the physiological jaundice to such a degree as to produce kernicterus. Dose of 1 mgm. to premature babies, and 2.5 mgm. to the bigger ones was enough. It was not possible to quote the incidence of this disease in Kandang Kerbau Hospital owing to lack of a diagnostic index system. It was probably not as high as 1 in 200.

Professor Sheares said that his impression of haemorrhagic disease of the newborn was of a haemorrhagic diathesis which manifested itself without provocation on the 3rd or 4th day of life (rarely 2nd or 5th day) when presumably the prothrombin level was physiologically low and the prothrombin time therefore prolonged. At the most, trauma which might be a precipitating cause was minimal or very slight. However, in the case of all the 3 babies under review, mid-forceps had been applied, and haemorrhage had occurred under the scalp, indicating that more than just minimal trauma was a factor in the bleeding manifestation. He wished to remind the house that bleeding in the newborn might occur from other causes, e.g., syphillis, erythroblastosis foetalis, sepsis, leukaemia, thrombocytopenia and haemophilia. The most dangerous location of the bleeding is the brain and meninges; other common sites are the umbilical cord, the bowel and stomach. The baby might present signs of shock due to internal bleeding. The usual laboratory data obtained in cases of haemorrhagic disease of the newborn are prolonged prothrombin time and coagulation time. The platelet count, bleeding time and clot-retraction time are usually normal. There is also anaemia and leucocytosis. He said that four factors were blamed for the deficiency in prothrombin in the newborn:

(1) Poor diet of the mother.

(2) Sterility of the infant's intestinal tract in the first 3 days of life.

(3) Insufficient nourishment given to the newborn baby and therefore insufficient food in its intestines for synthesis of Vitamin K.

(4) Functional immaturity of the liver which therefore cannot utilise Vitamin K for production of prothrombin.

It has been stated that less than 5 out of 10,000 newborn babies exhibit a degree of prothrombin deficiency which would result in haemorrhage. According to Stanford and Potter the incidence is 1 in 3,000 babies. Vitamin K is usually prescribed for haemorrhagic disease of the newborn, but there is a lack of complete correlation between the prothrombin level, the disease and the therapeutic response to Vitamin K. Usually there is a spontaneous halt of the bleeding and uneventful recovery. However, regardless of the cause in his opinion, fresh blood 10 ml. per body weight, was the most reliable agent to arrest the bleeding in the stubborn case. He used to resort to this treatment in the days before the advent of Vitamin K.

Doctor Sinha agreed with Professor Sheares that forceps delivery was a factor to be considered in etiology of the 3 cases under discussion, especially as Kielland's forceps had been used as in the first and second cases to rotate the foetal head from the occipito-posterior to the occipito-anterior position.

Doctor Smith agreed that forceps delivery was probably a factor in determining the site of haemorrhage. All three babies showed evidence of bleeding until the 3rd day. This was probably related to the prothrombin level in the blood which then allowed extensive bleeding to follow minor trauma. The second baby's prothrombin time was 1 minute and 19 seconds. She stressed the importance of giving prophylactic Vitamin K to infants born of toxaemic mothers after prolonged labour, or by instrumental delivery.

Doctor Field believed that it was worthwhile giving Vitamin K prophylactically where the risk of birth trauma was

high or when the mothers had toxaemia. She agreed with Doctor Smith that this disease was commonly seen here. She suggested a relationship between the disease and the mother's diet or toxaemia where the plasma proteins were low. She estimated that the incidence was about 1 in 500 cases.

She believed that a single injection of Vitamin K. would stop the bleeding. It was unnecessary to transfuse every case of haemorrhagic disease of the newborn. Blood transfusion was necessary when the baby was really anaemic. In these 3 cases the Hb. levels were all below 50%. There were a few cases of haemorrhagic diseases that were not due to a Vitamin K deficiency. Factors V and VII were probably implicated. These cases did not respond to Vitamin K. therapy. Referring to the 3rd case. she doubted whether a second transfusion would have saved the baby. She had seen several cases of persistent bleeding where the factor involved was unknown. In all the three cases presented. she thought that aetiology was both Vitamin K deficiency and trauma. Trauma precipitated and determined the site of the bleeding.

Professor Sheares suggested that much devolved on the definition of haemorrhagic disease of the newborn. and asked for elucidation.

Doctor Field defined it as a condition occurring in newborn babies. where there was a low prothrombin level in the blood. The bleeding could occur anywhere. more commonly in sites liable to trauma.

Doctor Smith suggested defining it as haemorrhage occurring in the first week of life, which was disproportionate to the degree of trauma. She advised house-doctors to give Vitamin K 2.5 to 5 mgm. at once when bleeding first occurred, to determine the blood-group of the infant and cross-match some blood to be held in reserve. If a further haemorrhage occurred this would then be ready immediately as a life-saving measure.

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