A Case of Unusual Obstetric Coagulopathy

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The pathogenesis of defibrination and hemorrhagic tendency in premature separation of the placenta is now more familiar to most obstetricians. In case of bleeding tendency subsequent to premature separation of the placenta Dieckmann in 1936 made the basic observation that fibrinogen was lacking not only in blood that passed from the uterus but also in all the circulating blood. It was thus a question of a general coagulation deficiency. Casuistic and even statistic reports have contributed to the to-days concept of the clinical picture of abruptio placentae. Our present understanding of the bleeding syndrome in premature separation of the placenta is partly based on the theory of intravasal disseminated fibrination as advocated by Schneider. The knowledge of the phenomena of fibrinolysis and fibrinogenolysis complete the theoretical understanding of the hemorrhagic diathesis in abruptio placentae.

Towards the end of a normal pregnancy the amounts of two of the main coagulation factors increase considerably. The concentration of fibrinogen in the blood raises successively with the continuation of the pregnancy and reaches near term a value of about twice that in non pregnants. The second coagulation factor to be found in increasing amounts in normal pregnancy is the thromboplastin. Placenta and decidua are especially rich in thromboplastic substances (Seegers, Schneider). In case of a premature separation of the placenta, in particular if the separation is central, an autoextraction of retroplacentar debris occurs (Schneider). In the area of the separation ruptured arteries continue to pump blood which passes through the hematom and the damaged uterine wall and returns through opened veins of the maternal circulation. The mobilized thromboplastic potential immediately causes a state of hypercoagulability of the mother's blood (Jürgens). An intravasal disseminated coagulation or fibrination consumes the fibrinogen of the mother with a hypofibrinogenic bleeding tendency as a consequence. Symptoms of organic lesions, in particular the lower nephron nephrosis syndrome, have been correlated to fibrin deposits observed in arterioles and venoles of the kidneys and in other organs.

Except the defibrination through autoextraction of thromboplastic matter from the placenta a fibrinogenic hemorrhagic tendency may be the result of fibrinolytic and/or fibrinogenolytic activity. The uterine muscle is rich in activators of the fibrinolytic systems of the mother (Lewis and Ferguson;

T a g n o n and S o u l i e r) and some degree of fibrinolysis is commonly observed in obstetric coagulopathy. Not only mechanical trauma may deliberate the activators of the fibrinolytic system. A more or less unknown regulation mechanism in the central nervous system is believed to exist, which may be the cause of fibrinolysis seen in non traumatic chock.

The fibrinolysis seen in cases of macerated fetuses ought to be due to the liberation of proteolytic enzymes from a macerated fetus and an autolytic degenerated placenta.

The obstetric coagulopathy to be reported here is particular in that no premature separation of the placenta occured nor was it the question of a macerated fetus. The mechanical trauma, subsequent large hematomas and possibly a mental chock, which a mother near term obtained after a fall down from three meters hight on a cement floor formed the basis of a severe hemorrhagic tendency during the delivery. The case was as follows.

The patient a II gravida aged 26 delivered normally in 1953; the weight of the infant was 4260 g. According to the last menstrual period and first movement of the fetus the expected date of birth this time was the 9th of January 1957. The patient was in perfect condition throughout the pregnancy; no signs of late toxemia, nor of secundary anemia; bloodgroup 0 Rh +.

Early in the morning of the 10th of December 1956 she accidentally fell down from 3 meters hight on the cementfloor of a cellar. She lost the consciousness for some minutes and experienced pain in various parts of the body, when she waked up. At noon the same day she was brought to the hospital. Irregular uterine contractions then started. The patient said on admission that she obviously had an internal bleeding because the urine shortly before she went to the hospital had been bright red.

On admission December 10th the patient was fully conscious, bloodpressure 120/70, pulsrate 72/min. She had several large subcutaneous hematomas in the back and on the right thigh. Moderate pain over the XIth right rib but no fractures. Bloodcounts: Hgb 12.3 g. Leucocytes 12 800. Differentiation of white count normal. Thrombocytes 64 600. Sedimentationrate 16 mm/l hr. Bleeding- and coagulationtime whithin normal range. Bilirubin in serum not estimated because of a slight red colour of the serum. Blood nitrogen 35 mg%. Urine bright red coloured but transparent. No protein nor reducing sugar. Urine sediment: Leucocytes 1—0; Erythrocytes 0—2; Two hyaline and several granulated casts; no bacteria. The obstetrical status normal. No tenderness of the uterine wall, a fetus of normal size in occiput position. Fetal heart-tones normal, rate 140/min. Slight discharge of blood contained mucus. Irregular uterine pains.

The red colouring substance in the urine was considered to be myoglobin obviously from crushed muscles. As known the renal threshold for myoglobin is much lower than for hemoglobin (S p a e t , R o s e n t h a l and D a m e -

s h e r), which is in good correlation with the observation in this case, when a bright red coloured urine occurred simultaneously with only a pink coloured plasma. Test for hemolysis was within normal limits, too. The urine cleared up rapidly and the red colour disappeared entirely the day after admission under normal diuresis. No casts then more in the sediment and a slight proteinuria disappeared after two days. The patient did well all the time except for some pain from the areas with the hematomas. The fetal heartbeats were normal. With small doses of opiates the irregular uterine pains disappeared. On the third day after admission, December 12th, regular contractions started and after 8 hours of labour a live infant of 3360 g was delivered normally; no perineotomy. The amniotic fluid, which passed when the membranes ruptured 15 minutes before delivery had the same bright red colour as had the urine of the patient two days earlier. No sign of rupture of marginal sinuses nor of premature separation of the placenta, which was expelled spontaneously. Bleeding 500 g during stage III.

As the bleeding went on after the expulsion of the placenta exploration of the vagina and uterine cavity was performed. A slight athony was corrected with "Methergin". Diffuse bleeding from minor superficial scratches in the vagina and from the uterus continued and totaled 1700 g. There was no clotting of the blood, that passed from uterus and vagina. In the meantime the patient went into chock. Infusion of 500 g dried plasma did not stop the bleeding. The fibrinogenconcentration in plasma was determined to 85 mg%. It was observed that a loose clot which formed in a glasstube dissolved quickly and then no more clotting occured in the testtube. When it was apparent that a hypofibrinogenic condition caused the bleeding 2 g of pure fibrinogen was administered intravenously. Bleeding stopped entirely within 30 minutes and under ordinary therapy of the chock (1200 g blood) the condition of the patient rapidly improved. Twelwe hours later, on December 13th, fibrinogen was 290 mg%, Hgb 8.2 g, prothrombin 90% of normal, plateletcount 58 300. The patient did quite well and despite an attack of a lower nephron nephrosis syndrome her condition improved all the time from now. Urinary output decreased and increased again from December 12th, to December 21th: 400, 100, 80, 30, 25, 128, 88, 225, 445, 700, 1200 g. The specific gravity fell to 1004 and bloodnitrogen reached a maximum of 190 mg% on December 20th but fell steadly to normal within two weeks. Fibrinogen, white- and redcount were then normal. An increase in plasma potassium concentration to 34 mg caused a change of the ecg which disappeared when the fluid balance was corrected.

The infant a girl of 3360 g showed signs of asphyxia immediately at birth but got the normal colour of the skin and started to breathe normally under adequate oxygen supply. A melaena persisted for two days. The meconium had an unusual bright red colour. The urine was normal. Blood counts were all nor-

mal. Of the coagulation tests prothrombin time increased from a low value of 70 seconds to normal 35 seconds within 4 days. The condition of the baby was good all the time.

The case described here shows that a transitoric hemorrhagic diathesis under obstetric conditions must not always be the result of a hypofibrinogenemia due to an abruptio placentae or a macerated fetus and an autolytically destroyed placenta. From the case described it is quite obvious that at least near the term of an otherwise normal pregnancy the result of extensive traumatizing of tissues may be a hypofibrinogenic hemmorrhagic condition. Also if the therapeutic management of the patient was clear it had been of interest to know whether the hypofibrinogenemia was caused by an fibrinogenolytic mechanism or if a defibrination on the basis of liberated thromboplastins from the traumatized areas resulted in hypofibrinogenemia. Unfortunately no specific tests in this sense were performed but the rapidity with which the clot disappeared from the glasstube containing a bloodsample from the cubital vein indicated the presence of a remarkable fibrinogenolytic component. Possibly the substance, presumably myoglobin, which coloured the urine and the amniotic fluid red, activated the fibrinogenolytic system in the blood of the patient. The heavy mental chock, which the patient contracted when she fell down on the cement floor may too have contributed towards an activation of the proteolytic system.

The syndrome of lower nephron nephrosis which started to develop immediately subsequently to the delivery and the hemorrhage may have been the result of a damage to the kidneys caused by myoglobin released from the crushed muscles. The transitoric occurence of protein, hyaline and granulated casts in the sediment typical in myoglobinuria indicates the possibility of such a mechanism for the damage of the kidneys. The rapid disappearance of the red colour, the protein and casts from the urine and the very good urinary outpout the days following the trauma speak, however, in favour of an other mechanism of the ethiology of the lower nephron nephrosis. The symptoms of the tubular lesions appeared as a consequence to the severe hemorrhage after the delivery just in the same way as the symptoms from the kidneys use to develop in case of hemorrhage due to premature separation of the placenta. It is thus most likely that the tubular damage in this case developed on the basis of defibrination and dissemination of fibrin deposits in capillaries of the kidneys. A component of vasospasm may have contributed to the impaired function of the renal system.

It is thus too most likely that the major part of the hemorrhagic tendency in the case described developed as a consequence of a defibrination started by thromboplastic substances released through the severe trauma to the tissue.

Summary

A case of unusual obstetric coagulopathy is described.

A woman near term of a normal pregnancy contracted severe damage to muscles and other tissues. The infant, placenta and uterine function remained unimpaired.

Subsequently to the trauma the patient developed transitoric myoglobinuria and three days later after an otherwise normal delivery a severe hemorrhage occured, which was caused by hypofibrinogenemia. A possible fibrinogenolytic part in this condition is discussed.

A lower nephron nephrosis syndrome developed, which was similar to the syndrome in abruptio placentae. It is considered that the hypofibrinogenemia in this case developed on the basis of a defibrination and dissemination of fibrin in the sense of S c h n e i d e r .

Résumé

Un cas de coagulopathie obstetrique est décrit.

Une femme près du terme d'une grossesse normale souffre de lésions des muscles et d'autres tissues à la suite d'une chute. L'enfant, le placenta et la fonction utérine restent normaux.

A la suite du traumatisme la patiente développe une myoglobulinurie passagère. Trois jours plus tard, après un accouchement normal la patiente fait une hémorragie causée geno par une hypofibrinogenémie. La possibilité d'un processus fibrinolytique est discutée.

Un syndrome néphrotique du néphrone inférieur s'est développé qui est très semblable au syndrome observé dans la rupture du placenta. L'auteur considère que l'hypofibrinogènémie, dans ce cas, est due à une défibrination avec dépôt disséminé de fibrine comme l'a décrit S c h n e i d e r .

Zusammenfassung

Es wird eine Patientin mit einer ungewöhnlichen Gerinnungsstörung im Anschluß an eine Entbindung beschrieben. Eine Frau erlitt knapp vor dem Ende der Schwangerschaft eine schwere Schädigung von Muskeln und anderem Gewebe; Kind, Plazenta und Uterusfunktion blieben ungeschädigt.

Im Anschluß an das Trauma entwickelte die Patientin eine vorübergehende Myoglobinurie. Drei Tage später erfolgte eine normale Entbindung. Anschließend daran kam es zu einer schweren Blutung, welche durch eine Hypofibrinogenämie verursacht war. Die mögliche Rolle einer Fibrinogenolyse für die Entstehung des Krankheitsbildes wird diskutiert.

Es entwickelte sich ein lower-nephron-Syndrom, ähnlich dem bei vorzeitiger Plazentalösung. Es wird erwogen, daß in diesem Fall die Hypofibrinogenämie auf der Basis einer Defibrinierung und Ausscheidung von Fibrin im Sinne Schneiders entstand.

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