BLOOD DYSCRASIA COMPLICATING PREGNANCY AND PUER-PERIUM, WITH A REPORT OF TWO CASES*

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CASE 1.—A woman of nineteen, born in Armenia; in United States five years. Ten years ago patient was sick for one month with fever, during which time she was exceedingly pale. A diagnosis was made of "pale sickness" which, in Armenia, is the popular term for malaria. She apparently made a complete recovery from that ailment. Menstrual history normal. Gravida 2, para i, miscarried at 3 months one year ago. Soon after she became pregnant, she was very pale and weak. These symptoms, however, cleared up following a spontaneous abortion and she regained her normal health within a month.

Present Illness.—Last menstrual period began Dec. 25, 1924, labor expected Oct. 1, 1925. Patient felt well throughout pregnancy except for slight nausea and vomiting in the first three months. About three weeks before admission to the hospital patient stated that she, as well as her friends, noticed that she was growing pale. Except for a slight feeling of weakness she had no other complaints until she entered the hospital. July 28, 1925, examination revealed a systolic murmur at the right base and a blood pressure of 130 over 40. She was seen once more on Aug 18, 1925, at which time she felt well. No mention is made on the chart of any pallor. Urine negative. On Aug. 30, 1925, patient was admitted to the hospital in labor. She was delivered of a normal male baby, breech presentation, seven hours after admission.

When patient was first seen at the hospital she presented the appearance of one who had just suffered a severe hemorrhage. Her face was extremely pale with a

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somewhat yellowish tinge, her lips were almost white, yet, curiously enough, she complained of hardly any weakness or indeed of any other discomfort except her labor pains. Blood pressure was 150 over 80, no bleeding from anywhere. Spleen was not palpable; slight swelling of the ankles. Blood examination (before delivery), R. B. C., 1,350,000; Hgb., 28; W. B. C., 2,400; polymorphonuclear leucocytes, 32; lymphocytes, 68. There was anisocytosis, polychromatophilia, poikilocytosis and a few normoblasts. Urine showed albumin one-plus, acetone two-plus.

The following day the hemoglobin dropped to 15 with less than 1,000,000 red cells, her temperature rose to 101.5°, pulse 90, respiration 30. Five hundred c.c. of citrated blood were given intravenously Aug. 31, 1925. The day after the transfusion her temperature rose to 103°, then dropped sharply to 99° and from then on until her exitus it varied from 101° to 106°. On several occasions preceding a sharp rise in temperature patient had a chill that lasted a half hour.

Following the transfusion her hemoglobin rose to 25, and within two days dropped back to 15. Two days after the first transfusion a blood examination showed bleeding time forty minutes; clotting time fourteen minutes; bilirubin dilution 1 to 150; cells, one-fourth volume of total blood; no clot retraction after two and one-half hours; platelets 64,000; Van den Bergh test positive for indirect reaction. The smear presented the same characteristics as on the previous examination.

September 6, 1925, a second transfusion of 500 c.c. of citrated blood was given. Again there was a slight rise in hemoglobin which dropped back to 15 in two days. During this time her spleen became palpable, patient was losing ground perceptibly, uterine bleeding increased, pulse grew weak and she became moribund. Four days later a splenectomy was performed preceded and followed by transfusions of whole blood. Soon after the operation uterine bleeding increased, hemoptysis and epistaxis set in, pulse grew weak, and respiration became labored. She expired within forty-eight hours after the operation.

The necropsy showed acute degenerative cardiac changes and emboli with petechial spots scattered over the endocardium. The lungs presented pulmonary edema and a small infarct. The liver showed acute degenerative changes, the kidneys an acute nephritis. The uterus showed an acute, septic endometritis. The spleen weighed 260 gm., consistency was normal and microscopic examination showed evidence of sepsis. Uterine and splenic cultures revealed Streptococcus hemolyticus.

The baby was discharged in normal condition.

Discussion.—The patient remained in the hospital long enough to allow a thorough investigation of the case, yet the various antemortem examinations together with the advantages of a partial autopsy did not help to establish an exact diagnosis. The postmortem findings together with the mode of death clearly suggest sepsis. woman came to our notice with a blood condition well advanced towards an unfavorable outcome, and if that blood condition was not directly responsible for the actual death, it was certainly the determining factor in the production of complications. The history of the case with all the findings is by no means indicative of any sharply defined blood disease. The onset, particularly its occurrence during pregnancy, would suggest an acute pernicious anemia. The blood picture, however, does not bear out this diagnosis. For pernicious anemia is essentially a megaloblastic anemia, and no megaloblasts were found in any of the smears, nor was the hemoglobin index as high as is commonly found in pernicious anemia. The next disease

that would suggest itself is purpura hemorrhagica. Here the blood findings are more consistent, particularly the fact that the platelets were found so low in number, as well as the prolonged bleeding time and absence of clot retraction. The one serious objection to this diagnosis is the absence of purpura. It must also be remembered that the patient did not bleed from any part of the body until after delivery when it was noticed that the uterine bleeding was excessive. Hemorrhage from other mucous membranes did not set in until shortly before death. A diagnosis of purpura hemorrhagica in the absence of the two most outstanding clinical features of this disease is unwarranted.

Another diagnosis considered was acute lymphatic leucemia. Here both clinical and laboratory findings are highly suggestive since on repeated examinations the lymphocytic count was high. But we also had smears that showed a lymphocytic count of not more than 30 and such findings were not discovered shortly before death as is sometimes the case with leucemia. Banti's disease, we believe, can be easily ruled out for the reason that neither the clinical nor the pathologic features pointed to this. It must be remembered that Banti's disease is essentially a chronic ailment with enlargement of the spleen preceding the anemia and liver changes and ascites late in it course.

We are forced to conclude that we are here dealing with a toxemia of pregnancy which manifested itself chiefly in the form of a blood dyscrasia. The signs of sepsis which complicated the picture in the puerperium must be looked upon as secondary to a blood condition which was dangerously approaching a state of incompatibility with life.

Tice describes a condition which he calls "hemolytic anemia of pregnancy." This resembles our case in many respects. For want of a more plausible or more certain diagnosis we conclude that ours is a case of this type.

Case 2.—A woman of twenty-four, nativity Russian, eleven years in United States. Admitted to hospital June 1, 1925, in labor.

Her last regular menstrual period began Aug. 7, 1924; labor expected May 14, 1925. Patient felt weak throughout pregnancy and suffered with frequent headaches which she attributed to some pills taken in the second month to bring on menstruation. Menstruation began at thirteen, periods irregular, coming on at three-to five-week intervals, flow lasting five days, dysmenorrhea the first two days.

Patient stated she was always prone to bleed profusely from the slightest cause, particularly so after her last confinement.

On April 16, 1925, swelling of feet was noticed and petechial spots scattered over the body. Blood pressure was 150 over 80. During her later visits to the prenatal clinic she complained of headache, weakness, ecchymotic spots over the body and bleeding from the gums. Urine showed a faint trace of albumin and a few red cells. Patient went into labor June 1, 1925, and was delivered the same day of a normal male child.

On admission to the hospital, patient had a yellowish pallor, body and extremities

were covered with petechial and ecchymotic spots ranging in diameter from 1/4 to 1/4 cm. Mucous membranes were pale. Heart sounds were normal except for an accentuated second pulmonic. There were occasional crepitant râles over both bases.

Blood examination showed: R. B. C. 2,400,000; Hgb. 30; W. B. C. 15,000; polymorphonuclear leucocytes 80; lymphocytes 20. Smear showed anisocytosis, poikilocytosis and normoblasts. Up to June 27 blood examination varied: R. B. C. from 1,500,000 to 3,000,000; Hgb. from 30 to 50; bleeding time from 3.5 to 32 minutes; elotting time from 3 to 10 minutes; platelets from 40,000 to 50,000. There was always a moderate leucocytosis with an increased polymorphonuclear count. These variations were probably influenced by the blood transfusions which she received at frequent intervals.

After June 27 we observed a steady decrease in red blood cells, a lowered hemoglobin, increased bleeding time, decreased number of platelets and poor clot retraction. Patient was bleeding from the gums and blood was found in the urine and stool. As the disease progressed uterine bleeding increased.

Temperature on admission was normal, soon after delivery it rose to 101°; on third day postpartum it rose sharply to 105°; from then on it was irregular ranging from 100° to 103°.

Between June 6 and July 8 patient received six transfusions with only transitory improvement in general condition and blood picture. She was also given horse serum and human blood intramuscularly, calcium lactate orally, x-ray over the spleen and tonic and supportive treatment. None of these therapeutic measures were of any avail; new crops of petechiae continually appeared. The second week in July the patient began to lose ground rapidly; uterine bleeding increased, heart action weakened and on July 12 patient expired. Baby was discharged from the hospital in normal condition.

This is a case of purpura hemorrhagica complicating pregnancy and puerperium.

Discussion.—A review of the literature shows that purpura hemorrhagica is an exceedingly rare complication of pregnancy, only 39 cases having been reported up to 1923. In a report of a similar case G. C. Mosher reviews eleven others reported in the literature. It appears that the complication occurs most often in multiparae; the attack usually becomes manifest between the sixth and seventh months of gestation with a purpuric rash and bleeding from various parts of the body. The outcome, particularly if the patient goes to full term is generally fatal, death occurring from within a few days to a few weeks postpartum. The baby as a rule is normal.

A comparative study of the two cases of this report reveals points of unusual interest. While the predominating feature of both cases is the severe grade of anemia and while the gestation in both instances probably bears an etiologic relation to the anemias, there is nevertheless a decided difference between the two. Whereas the first case entered the hospital in her eighth month without any history of loss of blood, the second patient showed the main features of a purpura hemorhagica two months before she was delivered. It is fair to assume that the anemia of the second case was at least in part caused by the loss of blood from the gums and into the skin. The first patient, on the other hand, reached an alarmingly severe type of anemia

without having lost any blood. Evidently in this case direct insult was suffered by all the formed elements of the blood giving rise to a picture not unlike that of an aplastic anemia. Another point corroborative of the secondary nature of the anemia in the hemorrhagic purpura case, is the constantly high leucocytic count as contrasted with the decided leucopenia of the first case.

Practically all authorities agree that in the severer grades of anemia incident to pregnancy, transfusions yield prompt and satisfactory results but where the blood dyscrasia assumes the character of an aplastic anemia or purpura hemorrhagica, the outlook is very gloomy and in most instances hopeless.

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