SEVERE ANÆMIAS OF PREGNANCY AND THE POSTPARTUM STATE*

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IN 1919 the late Sir William Osler published his "Observation on the severe anæmias of pregnancy and the postpartum states". He divided such cases into the four following groups: (1) Anæmia from postpartum hæmorrhage: (*a*) profuse and rapidly fatal hæmorrhage; (*b*) anæmia following repeated small hæmorrhages. (2) The severe anæmia of pregnancy: chloro-anæmia of pregnancy, which might pass on to a grave and fatal form. (3) The acute anæmia of postpartum sepsis. (4) Postpartum anæmia: a common form in which, following uneventful delivery without undue loss of blood, the patient becomes increasingly pale with a rapid fall in blood values. While clinically identical with Addison's anæmia, a large percentage of such postpartum cases recover spontaneously without any tendency to recurrence of the condition.

Sir William stressed the continuous septic-like fever which was often present and warned against being misled by it, citing a case which had been erroneously diagnosed as malaria fever. Cabot observed that not uncommonly such cases were diagnosed as typhoid fever.

Osler at that time made no division into temporary pernicious anæmia and Lederer's hæmolytic anæmia, but pointed out the variations seen in the blood studies of these cases, some with reticulation as high as 20 and 25% and marked evidence of hæmolysis, and others with leukopenia, thrombocytopenia and aplasia. Many cases showed the characteristic Addisonian picture, without, however, neurological changes.

Such a case was recently admitted to the gynæcological service of the Montreal General Hospital.

The patient was a 22-year old French Canadian para ii. Her first pregnancy was normal throughout and terminated with the spontaneous delivery of a healthy male infant. There was no anæmia before or following the labour. A second pregnancy occurred one year later. No prenatal care was received and the patient stated that she remained in good health until the last month of her pregnancy, when she became aware of easy fatigue, dyspnœa on exertion, and pallor. Delivery took place in her home, was easy, spontaneous and without gross hæmorrhage. The child, a male, was healthy and weighed seven pounds at birth.

Within the first 24 hours following delivery, it was observed by the patient's relatives that she was extremely weak and pale and appeared to be definitely febrile. These symptoms became progressively more alarming until on the eleventh day postpartum she was admitted by ambulance to the gynæcological ward of the Montreal General Hospital.

On admission, the patient was extremely pale and had a temperature of 103°. Dyspnœa was present on the slightest exertion, but not noticeably so when lying flat in bed. There were no petechiæ or ecchymosis, and the tongue was not smooth and apparently normal. The spleen was palpable at the costal margin. Pulse was 120, regular and good volume. Blood pressure was 110/60. There was no evidence of cardiac failure. On pelvic examination, the vagina was clean, lochia scant, normal in appearance, without odour. The cervix was lacerated, but clean. The uterus was well involuted and deep for eleven days postpartum, and mobile. No tenderness was elicited on deep palpation over the broad ligaments. The appendages were normal. The patient's temperature from time of admission was definitely septic in character. (See Chart 1).

The hæmoglobin was 18%, with a red blood count of 730,000, and white blood count of 4,800. A ten-

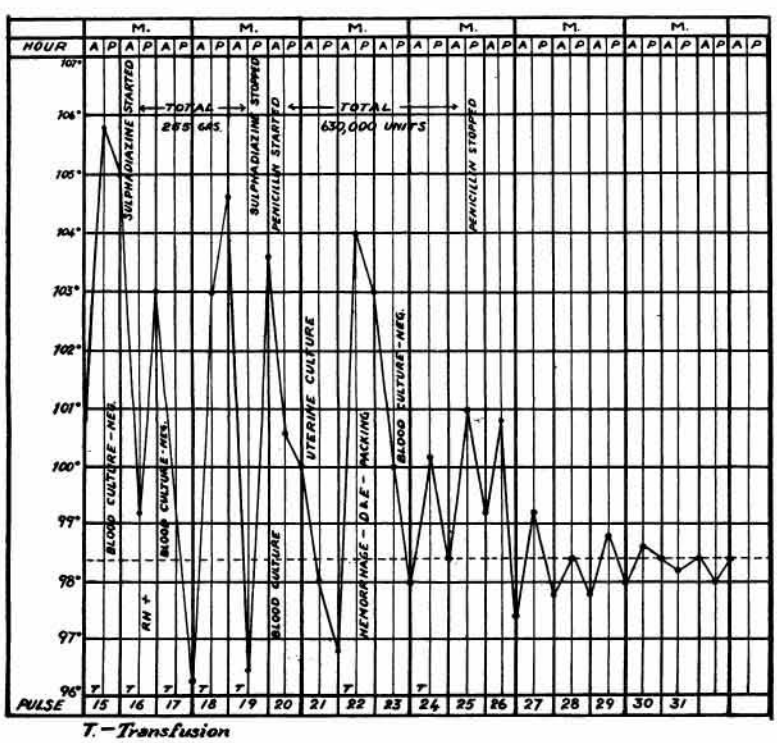

Chart 1.

* From the Gynæcological Service of the Montreal General Hospital.

tative diagnosis of puerperal sepsis with anæmia was made, and while awaiting the results of a blood culture, multiple blood transfusions were given. Satisfactory increase in hæmoglobin and erythrocytes occurred, but a progressive leukopenia and thrombocytopenia continued. (See Chart 2). There was a macrocytosis and marked variation in the red cells.

By the fifth hospital day, the platelet count had fallen to 35,000 and there were only 1,200 leucocytes per c.mm. The reticulocytes remained below 1%. Spontaneous uterine hæmorrhage occurred at this time, necessitating packing. The usual therapy, including sulfadiazine and penicillin, failed to cause any improvement in the septic picture. Repeated blood cultures showed no organisms, and it became obvious that the case was an acute postpartum anæmia, closely resembling that described by Sir William Osler.

Repeated transfusions, totalling 6,000 c.c. augmented by general supportive measures was followed by an uneventful recovery. Since discharge on the 29th day, the patient has remained in good health, without any suggestion of anæmia, and a normal blood picture has been maintained for eight months without further treatment.

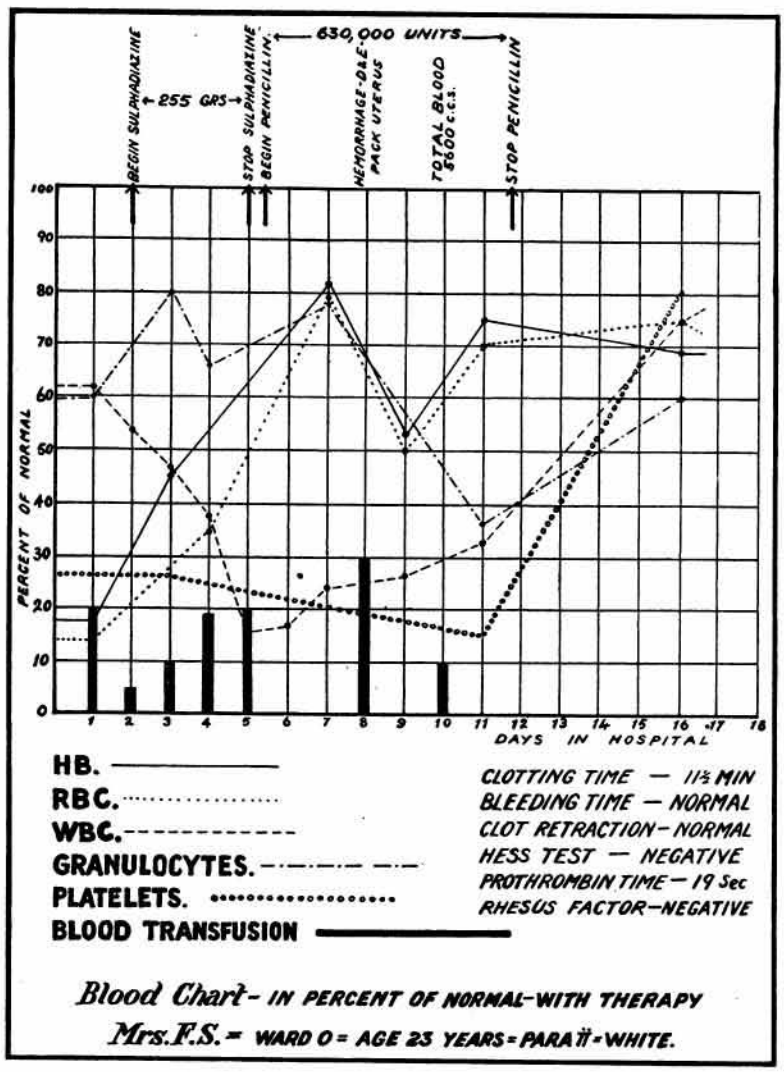

Chart 2.

COMMENT

The temporary pernicious anæmia of pregnancy and the puerperium is clinically identical with Addison's anæmia. It occurs most frequently in multipara, in the middle or later years of reproductive life. The early signs are often insidious, appearing during the last trimester, but may develop, as in our case, suddenly or even dramatically, soon after delivery. There is a macrocytosis, with a positive colour index. Reticulocytes remain less than 1%, thrombocytopenia is common. Improvement is rapid and sustained with liver therapy. On the other hand, in the acute hæmolytic anæmia, the reticulocytes invariably become elevated without treatment while improvement seldom follows liver therapy.

From time to time one encounters cases which present certain features because of which classification is difficult. In Lescher's series of 17 cases of grave anæmia, associated with pregnancy, nine were diagnosed temporary pernicious anæmia, and eight hæmolytic anæmia. All but three of the latter, however, showed macrocytosis, some had low or absent gastric HCl., and in one, excellent improvement followed liver therapy alone. It is very difficult to prove clinical hæmolysis by hæmoglobinæmia or abnormal blood cell fragility, as these are seldom increased. Evidence of moderate hæmolysis occurs in pernicious anæmia, as evidenced by an increased icterus index, indirect Van den Bergh reaction, and changes in skin colour. The reticulocytosis, normal platelet count, and failure of liver therapy are the outstanding diagnostic aids in Lederer's anæmia, while thrombocytopenia, normal reticulocyte count, and the spectacular response to liver therapy, constitute the diagnostic features of temporary pernicious anæmia.

The constant thrombocytopenia mentioned above, may occur before a diminution of hæmoglobin, or red cells.

Another case, para iii, routinely examined at 3½ months, showed an abnormal platelet count of 83,000. The hæmoglobin was 80%, red cells 4,120,000, white cells 7,650. Over a short period,

platelets fell to 43,000 and were not improved by iron or fresh blood transfusions. In the hope of arresting this rapid deterioration, the patient was admitted to the gynæcological service of the Montreal General Hospital and the uterus evacuated by abdominal hysterotomy. Within the next two weeks, the platelets rapidly increased to a level of 179,000 on discharge. This woman had severe hæmorrhages with each of her previous confinements. With her first baby the bleeding occurred eight days postpartum, for no apparent reason.

We have seen two cases, more recently, in whom spontaneous abortion occurred, with similar thrombocytopenia, which rapidly returned to normal following termination of pregnancy.

In view of the foregoing, further investigation on the significance of blood platelet counts in the anæmias associated with pregnancy, is being carried out. It may well be that an early fall in platelets may be the forerunner of a progressive anæmia, for which iron is of little or no value. The judicious use of liver extract instituted as a prophylactic measure may forestall the grave anæmia described by Osler.

The etiology of these anæmias is unknown. The fact that in the majority of reported cases there has been inadequate prenatal supervision, suggests an extrinsic factor, and emphasizes the need for proper diet, fortified, if necessary, by food supplements. Repeated blood examinations during this period are essential.

Osler suggested that some hæmolytic agent or toxin was responsible, but was unable to determine its nature and as yet no further progress has been made. Reynolds in the "Clinical Significance of the Rh Factor", suggested that incompatibilities between maternal and fetal bloods resulted in hæmolysis of the maternal blood, and profound anæmia. This was brought about, he felt, when an Rh + mother, gave birth to an Rh – baby, and antibodies formed in the fetus entered the maternal circulation.

In case 1, the mother was Rh –, and both her babies Rh +, with no evidence of erythroblastosis fetalis. No antibodies have been demonstrated in either mother or her babies.

Rh incompatibility may be responsible for the hæmolysis of Lederer's anæmia, but, as yet, we have been unable to prove this thesis. It would appear in the light of existing evidence, that the maternal anæmia is probably not produced by antibodies formed in her fetus.

SUMMARY

1. A case of acute postpartum anæmia, with thrombocytopenia and leukopenia is presented.

2. Grave anæmias of pregnancy and the postpartum are discussed.

3. Thrombocytopenia may be a forerunner of the severe anæmias of pregnancy and the postpartum states, described by Osler. Platelet counts therefore should be a routine in prenatal blood studies.

We are indebted to Dr. L. K. Diamond of Boston for verifying Rh factors and for his constructive suggestions.

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