Case Report

Management of Aplastic Anemia in Pregnancy

Manajemen Anemia Aplastic pada Kehamilan

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Abstract

Objective: To illustrate the management of anemia aplastic in pregnancy.

Methods: Case report.

Case: A 29 years old women came to Dr. Cipto Mangunkusumo due to aplastic anemia in 24 weeks of gestational age. Laboratory result showed anemia normocytic normochromic and thrombocytopenia. During the next visit, the patient had hypertension with controlled blood pressure and IUGR. Therefore, the pregnancy should be terminated and the mode of delivery was a cesarean section.

Results: The baby was asymmetric IUGR indicating hypoxic environment due to anemia in pregnancy.

Conclusions: Anemia aplastic was a rare case during pregnancy and close monitoring was imperative to detect early complication such as intrauterine growth retardation.

Keywords: aplastic anemia, pancytopenia, pregnancy

Abstrak

Tujuan: *Untuk menggambarkan manajemen anemia aplastik pada kehamilan.*

Metode: *Laporan kasus.*

Kasus: *Seorang perempuan 29 tahun datang ke RSCM karena kehamilan 24 minggu. Hasil laboratorium menunjukan anemia normositik normokrom dan trombositopenia. Pada kunjungan berikutnya pasien memiliki tekanan darah tinggi terkontrol, dan PJT. Maka dari itu, kehamilan harus diakhiri dan cara persalinan adalah seksio sesarea.*

Hasil: *Bayi PJT asimetris menandakan keadaan hipoksia karena anemia selama kehamilan.*

Kesimpulan: *Anemia aplastik pada kehamilan adalah kasus yang jarang dan pengawasan ketat penting dalam mendeteksi komplikasi seperti PJT.*

Kata kunci: *anemia aplastik, kehamilan, pansitopenia*

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INTRODUCTION

Acquired aplastic anemia is an uncommon disorder characterized by progressive pancytopenia caused by altered bone-marrow function.[1] Incidence is estimated to be one to two cases per million per year.[1] Given the complexity of anemia aplastic and the limited experience by most providers, new guidelines by the British Society for Standards in Hamatology on the diagnosis and management of adult aplastic anemia were recently published.[2] Pathogenic mechanisms underlying this disease are likely to be immune-mediated and include the overproduction of bone marrow inhibiting cytokines elicited by abnormal T-cell response in a genetically predisposed individual.[3] Pregnancy in association with aplastic anemia is a rare but serious condition that poses serious maternal and fetal risks. Unfortunately, most of the current literature has been limited to case reports, with few studies exploring risk factors and perinatal complications.[4]

Pathophysiological mechanisms underlying the association between aplastic anemia and pregnancy have not been clearly elucidated.[4] It is known that estrogens increase plasma volume in pregnancy more than red-blood-cell production, resulting in anemia of pregnancy. It has been postulated that hormonal influences may contribute to worsening of blood counts in pregnant patients with aplastic anemia, but the exact mechanism and causes are still unclear.[5] Animal models have provided some insight into mechanisms for altered maturation and

proliferation of blood cells in pregnancy. In a murine model, injection of 17-estradiol inhibits the development of developing thymocytes.[6] Demonstrated enhanced proliferative activity of erythroid precursors in bone marrow that was increased by concomitant administration of iron.[7] According to one theory, a population of primitive CD34 progenitors responsible for cellular proliferation and regeneration is produced in maternal bone marrow in response to interaction with umbilical cord blood cells via immunologic signals.[8] Pregnancy is also sometimes accompanied by gestational thrombocytopenia and relative leukocytosis. The factors responsible for the observed thrombocytopenia in pregnant patients with aplastic anemia are yet to be definitively elucidated.[9]

CASE

A 29 years old woman, G1 24 weeks of gestational age was referred due to bicytopenia which was anemia and thrombocytopenia to Dr. Cipto Mangunkusumo Hospital. There was negative sign for fatigue, headaches, weakness, and pale. Her LMP and ultrasound in second trimester equivalent to 24 weeks. Her blood count (table 1) revealed anemia macrocytic hyperchromic (Hemoglobin level 9 g/dl, MCV 101.9 fl and MCH 34.1 pg), neutrophil (3,096 /µL) and thrombocytopenia (53,000/µL). The result of Bone Marrow Punction 5 years ago at Abdul Moeloek Hospital, Lampung was hypocellular suggesting aplastic anemia. She was assessed as G1 24 weeks gestational age with anemia aplastic and treated with methylprednisolone orally.

During 3rd visit, her blood pressure was 150/100 mmHg, on Nifedipine 10 mg with proteinuria +1 (qualitatively) and 1 gram/24 hours (quantitatively). General status was normal. Complete blood count showed haemoglobin, leukocyte and thrombocyte were 9.2 g/dl, 5,450/µl, 14,000/µl, respectively. Other tests including liver function, renal function, and total bilirubin/direct/indirect revealed normal value. Immunology test such as ANA and Anti DS-DNA tests gave negative results. Ferritin was high 706.444 ng/mL. Coombs test was not performed due to antibody tests were negative. Ultrasound examination revealed an estimated fetal weight of 1500 g in 31 weeks gestation. She was assessed as G1 31 weeks gestational age with anemia aplastic, hypertension in pregnancy, IUGR.

A multidisciplinary team consisting of obstetricians, anesthesiologists, hematologists, and neonatologists planned on cesarean section under general anaesthesia. Cesarean section was performed at 33 weeks of gestation born baby boy of 1,600 g with Apgar scores of 7 and 8, at 1 and 5 min, respectively. The head circumference, abdominal circumference, and ratio Head to Abdominal circumference were 30 cm, 24 cm, and 1.25 equivalent to asymmetric IUGR.

Table 1. Laboratory Examination

Parameter	2nd Visit	3rd Visit
Hb/Ht/Leukocyte/Thrombocyte	9/26.9/5.470/53.000/	9.2/27.1/5.450/14.000/
MCV/MCH/MCHC	101.9/34.1/33.5	92.2/31.3/33.9
Basophil/Eosinophil/Neutrophil/ Lymphocyte/Monocyte (%)	0.4/0.2/56.6/34.9/7.9	0.2/0.2/49.4/44.2/7
Reticulocyte Absolute (/µl)	96600	
RET-HE	36.2	

Table 2. Ultrasound Examination

Parameter	24 weeks	26 weeks	31 weeks
BPD/HC/AC/FL (mm)	58/218/190/38	64/234/203/44	80/285/247/58
EFW (g)	569g	750g	1500g
AFI/SDAU	12/4.9	15.5/5.5	12.8/3.8

DISCUSSION

Finding Hemoglobin level, MCV, MCH was 9 g/dl, 101 fl and MCH 34.1 pg equivalent to anemia macrocytic hypochromic. The differential diagnosis for it was deficiency B12 and aplastic anemia. The diagnosis of anemia aplastic was established by finding 2 out 3 of anemia, neutropenia, and thrombocytopenia In this patient anemia (Hemoglobin level 9 g/dl) and thrombocytopenia (53,000/µL) occurred.

The cause of anemia aplastic remained unknown. However, it was related to the pharmacologic agent, infection (particularly hepatitis), or hereditary forms with late-onset manifestation (telomeropathies). The severity of the disease was based on the level of neutrophils, platelets and reticulocyte. The level of neutrophil, platelets, and reticulocytes were 3.096 /µL, 24,000/µl, 96600/µl. Therefore, the classification was non-severe.[4,5]

Pregnancy in association with aplastic anemia was a rare but serious condition that poses serious maternal and fetal risks. Unfortunately, most of the current literature had been limited to case reports, with few studies exploring risk factors and perinatal complications.[6,7]

Aplastic anemia was known to increase antenatal complications. The risk of preterm birth was 12.1 %, intrauterine fetal death was 16.7 %, stillbirth was 15.1 %, and spontaneous miscarriage was 16.7 % among pregnant women with the diagnosis of aplastic anemia. Although previously cited complications are commonly encountered in cases of aplastic anemia, no such complications accompanied our case. Hemorrhage at the time of delivery/abortion is another danger. Postpartum hemorrhage is an important complication among patients with the diagnosis of aplastic anemia due to decreased platelet count. However, we've been managed this possibility by given platelets pre-operatively. Intrauterine growth retardation complicated as one of our cases.[4,9-11]

Fetal growth surveillance should be performed by 28 weeks of gestation, and antenatal testing should also be offered by 30-32 weeks, due to the high prevalence of growth restriction. In this case, growth restriction was identified after close monitoring and established by abdominal circumference and estimated fetal weight in 31 weeks gestational age correspond to 1500 g. The asymmetrical IUGR finding from the ratio head and abdominal circumference (>1.2) indicated new-onset hypoxia. This was caused by maternal factors which was aplastic anemia.[10,11]

Cesarean in our case was indicated due to fetal IUGR although in cases of aplastic anemia, vaginal birth is preferred. As recommended to this patient we make sure that the platelet count was >20x103/µL which acceptable for vaginal delivery and 50x103/µL for cesarean delivery.[10,11]

CONCLUSION

Aplastic anemia is a complex disorder that warrants a comprehensive multidisciplinary-team approach, in order to devise an obstetric, hematological, anesthetic, and neonatal plan and anticipate complications during the peripartum period. Conservative transfusion strategies are necessary to avoid complications related to alloimmunization. Anesthetic management has to be individualized and should include considerations related to the degree of blood cell line compromise, as well as possible complications that have an impact on the anesthetic technique. An absolute number of circulating platelet count necessary to perform a safe neuraxial block cannot be recommended at this time, and the choice of the anesthetic technique depends largely on thorough clinical evaluation leading to a judicious balance of risks and benefits on a case-by-case basis. Despite good supportive care, we had a case of fetal growth restriction, but the fetal outcome was good.

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