Klinik ve Deneysel Araştırmalar Dergisi /
Journal of Clinical and Experimental Investigations

Cilt/Vol 1, No 3, 226-227

CASE REPORT / OLGU SUNUMU

Up to which limit can one survive with iron deficiency anemia?

Demir eksikliği anemisi ile hangi sınıra kadar yaşanabilir?

Mehmet Vural[1], Hakan Büyükhatipoğlu[2]

[1]Department of Obstetrics and Gynecology, School of Medicine, Harran University, Şanlıurfa, Turkey
[2]Department of Internal Medicine, School of Medicine, Harran University, Şanlıurfa, Turkey

ABSTRACT

Iron deficiency anemia is an important public health problem worldwide. In this case report we presented an unusual case of iron deficiency anemia manifesting with extremely low hemoglobin (Hb) level of 1.7 g/dl. This case was challenging because, the cause of anemia in this patient was insufficient intake, which is an unusual cause of iron deficiency anemia. Secondly, the degree of Hb level that the patient had might not be compatible with life. On the other hand, by means of this case we wanted to present the adaptive capability of human body to chronic long-standing anemia. Therefore, the question of when and how much transfusion will be given should be queried. *J Clin Exp Invest 2010; 1(3): 226-227*

Key words: Iron deficiency anemia, lower limit, hemoglobin, survival

ÖZET

Demir eksikliği anemisi dünya çapında önemli bir sağlık sorunudur. Bu vaka sunumunda 1.7 g/dl gibi çok düşük hemoglobin (Hb) seviyesine sahip bir hasta rapor edilmiştir. Bu vaka ilgi çekici idi çünkü anemi nedeni nadir olmasına rağmen yetersiz oral alım idi. İkinci olarak, Hb seviyesi yaşamla bağdaşmayacak kadar düşük idi. Ayrıca, uzun süreli kronik bir anemiye insan vücudunun uyum sağlama kapasitesini irdelemeyi amaçladık. Dolayısıyla, ne zaman ve ne kadar kan transfüzyonu yapılması gerektiğini sorguladık. *Klin Den Ar Derg 2010; 1(3): 226-227*

Anahtar kelimeler: Demir eksikliği anemisi, alt sınır, hemoglobin, sağ kalım

INTRODUCTION

Severe iron deficiency is relatively low as compared to mild-to moderate iron deficiency anemia. In this paper we want to share a very severe iron deficiency anemia with interesting aspects.

CASE

A 17-year-old female was referred to our hospital because of lethargy, fatigue, loss of appetite and severe debilitation, which was worsened during the last three months. The patient was moderately mentally retarded. She had tachycardia (130 beats per minute) and was pale in appearance. She described primary amenorrhea that was probably due to hypophyseal impairment and malnutrition. She was 41 kg in weight with a body mass index of 15 kg/m^2. Laboratory values were as follows: white blood cell counts (WBC) 4900 cells/mm^3, hemoglobin 1.7 g/dl, hematocrit 6.36%, mean corpuscular volume (MCV) 48, platelets 124.000/mm^3, ferritin 1, total iron binding capacity 470 and red cell distribution width (RDW) 75. Certain serum parameters are given in Table 1.

Serum Vitamin B12 and folate levels were within the normal ranges. On peripheral blood smear anisocytosis, significant hypochromia, and microcytosis were present. She was immediately transfused with two units of red blood cells and started on parenteral (intra-venous) iron-sucrose therapy (Venofer®). On the following days she was given two more units of erythrocyte suspension transfusions. Before discharge she was put on oral iron for six months. On the following visits the patient's symptoms including tachycardia, fatigue, and respiratory distress resolved at discharge (at 1 week). On a follow-up visit at 4 weeks she was doing well with normalized hemoglobin (12.1 g/dl) and ferri-

Yazışma Adresi /Correspondence: *Assist. Prof. Dr. Mehmet Vural, Department of Obstetrics and Gynecology, School of Medicine, Harran University, Sanliurfa, Turkey* **Email:** drmvural@yahoo.com
Geliş Tarihi / Received: 10.07.2010, Kabul Tarihi / Accepted: 28.10.2010

tin levels (45 ng/ml) with no complaints. She goes on to come follow-up examinations. Because of all abnormalities were corrected by iron supplementation, no further work-up was performed.

Table 1. Certain serum parameters of our patient.

Parameter	Result (reference values*)
Thyroid stimulating hormone (ng/dL)	1.82 (0.27-4.20)
Free thyroxine (ng/dL)	1.34 (0.90-1.70)
Follicle stimulating hormone (mIU/L)	3.34 (3.5-12.5)
Luteinizing hormone (mIU/mL)	< 0.1 (2.4-12.6)
Estradiol (pg/mL)	< 5.00 (12.5-166.0)
Prolactin (ng/mL)	6.20 (6.00-29.90)
Progesterone (ng/mL)	0.10 (0.20-1.50)
Testosterone (ng/mL)	< 0.02 (0.06-0.82)
Vitamin B12 (pg/mL)	318.60 (200-900)
Folic acid (ng/mL)	6.03 (4.20-19.90)

*Reference values are for the follicular phase of the menstrual cycle.

DISCUSSION

The main reason of reporting this patient was the unexpected severity of anemia. Unfortunately, mental retardation and malnutrition accompanied by the low socioeconomic circumstances of the family seem to have led to this situation. Because iron deficiency anemia is described as most common nutrition defect worldwide[1], our case might represent an extremely severe malnutrition characterized by a hemoglobin measurement below 2 g/dl. In a recent study Lin et al. have showed that degree of disability has positive and body mass index has negative correlation with anemia in the population of the 937 children with intellectual disabilities.[2] For this reason, mental retardation may be the main reason for chronic iron deficiency anemia in our patient. On the other hand the compensatory mechanism of the human body worth to be mentioned. Even in such low degrees of anemia, one can survive for months.

Ghosh et. al have revealed non hematological effects of the iron deficiency anemia and stated that iron deficiency can cause poor cortical arousal, diminished attention span, reduced scholastic performance in schools and reduced mental alertness.[3] Most probably iron deficiency and mental retardation are in a vicious cycle. Our case also may be in such a condition.

Blood transfusions might have some risks. Here, we have to emphasize the significance of avoiding unnecessary blood transfusions even in the setting of severe iron deficiency. Our case is practically is an exception and might illustrate the strength of compliance and compensatory mechanisms against severe anemia human body can compensate.

This severe anemia case should be a message for health authorities and Non-governmental organizations for public and social awareness. Since almost 25% of the worlds' population is estimated to be affected with iron deficiency[4], preventive interventions should be kept in mind widespread not only by clinicians also other healthcare workers as well.

REFERENCES

1. Clark SF. Iron deficiency anemia. Nutr Clin Pract 2008;23:128-41.

2. Lin JD, Lin PY, Lin LP, et al. Prevalence and associated risk factors of anemia in children and adolescents with intellectual disabilities. Res Dev Disabil 2010;31:25-32.

3. Ghosh K. Non haematological effects of iron deficiency -a perspective. Indian J Med Sci 2006;60:30-7.

4. Caballero B. Global patterns of child health: the role of nutrition. Forum Nutr 2003;56:249-50.