Unusual Etiology for a Common Problem

Muhammad Rashid DO PGY-2, Ahmed Abdelmonem, Ashraf Mohamed MD

CENTER FOR HEALTH SCIENCES

INTRODUCTION

Diagnosing the etiology of iron deficiency anemia can be very challenging

Hypothesis

Etiology of iron deficiency anemia is not always straightforward

Abbreviation Key

CXR = Chest X-Ray
CRP = C- Reactive Protein
ESR = Erythrocyte Sedimentation Rate
WBC = White Blood Cells
MCV = Mean Corpuscular Volume
Hgb = Hemoglobin
PICU = Pediatric Intensive Care Unit
SLE = Systemic Lupus Erythematosis
GI = Gastrointestinal
TIBC = Total Iron Binding Capacity
IV = Intravenous

Case 1

2-year-old female presented with fever, cough, pallor for 4 days. CXR showed diffuse pulmonary opacities. CRP 3.45, ESR 21, WBC 6, platelet of 361, Hgb of 4 gm/dl, MCV 54, developed hemoptysis and was transferred to the PICU with respiratory failure. Bronchiolar lavage and lung biopsy showed extensive hemosiderin-laden macrophages. Iron deficiency anemia, progressive cough, dyspnea, and infiltrates on CXR was consistent with idiopathic pulmonary hemosiderosis. Pneumocystis, HIV, histoplasma, SLE, vasculitides were negative. Patient responded to a burst of steroid with normalization of breathing and Hgb level during follow up.

Idiopathic Pulmonary Hemosiderosis

A rare disease in which there are recurrent episodes of diffuse alveolar hemorrhage. First other causes of repeat hemorrhage must be ruled out, then diagnoses with idiopathic pulmonary hemosiderosis can be considered. Recurrent hemorrhage can cause free iron in the pulmonary tissue leading to pulmonary fibrosis. Symptoms include recurrent cough and dyspnea which can progress to hemoptysis of increasing severity. The patient will eventually develop moderate to severe anemia. Diagnostic testing includes bronchoalveolar lavage and cytologic analysis showing hemosiderin laden macrophages. Long term treatment includes immunosuppressive therapy such as corticosteroids.

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Case 2

16 y/o female marathon runner with iron deficiency anemia refractory to iron supplementation, Hgb 7.4gm/dl over 6 month period despite taking iron supplements. MVC was 68, ferritin of 2, transferrin/TIBC levels were elevated. Hemoccult stool negative. Combination of intense physical exercise, refractory iron deficiency anemia, and lack of GI blood loss led to consideration of march hemoglobinuria. Urinalysis positive for blood, confirming the diagnosis. With IV iron and reduction of intensity of running, Hgb was up to 14.1.

March Hemoglobinurea

Non-immune intravascular hemolysis. This occurs with red cell damage due to mechanical trauma in the vessels of the plantar surface of the feet. The increase in free hemoglobin in the blood due to RBC breakdown leads to the presence of reddish discoloration of urine, which may be mistaken for hematuria. In actuality it is hemoglobinuria that may appear. The increased breakdown of RBC can in some cases cause iron deficiency due to the continued repetitive trauma and breakdown. This process is slow and ongoing and difficult to detect usually until the development of significant symptoms such as fatigue, pallor, decreased exercise tolerance, and dysregulation of physiologic processes such as oligomenorrhea. Treatment includes cessation of offending activity and iron supplementation.

CONCLUSION

These two cases of uncommon causes of blood loss highlight the importance of considering rare causes for iron deficiency anemia especially when it is not responding to iron supplement. Without treatment of underlying cause, anemia would persist

REFERENCES

Kliegman, Robert., et al. *Nelson Textbook of Pediatrics*. Edition 20. Phialdelphia, PA: Elsevier, 2016.

Millman MD. Idiopathic Pulmonary Hemosiderosis. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. https://www.uptodate.com(Accessed on September 07, 2018.)

Schrier MD. Extracorpuscular non-immune hemolytic anemia:fragmentation hemolysis and hyperslenism. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. https://www.uptodate.com(Accessed on September 07, 2018.)

ACKNOWLEDGEMENTS

Dr. Mohamed, Dr. Fugate, OSU PEDIATRICS