332 Letters to Editor

represent the idea that bilirubin, a breakdown product of hemoglobin, causes indirect bilirubin level increase by accessing the fetus through the placenta and that this causes fetal gallbladder stone formation^[4].

It has been asserted that smoking during pregnancy, hematologic diseases, blood incompatibilities between mother and fetus, and structural anomalies like choledochal cysts may cause formation of fetal gallbladder stones^[5].

On the other hand, Brown and colleagues proposed that high estrogen levels increase cholesterol secretion and decrease bile acid production and that this mechanism might cause pigment stones in gallbladder [3].

Flaxseed (Linum usitatissimum L., Linaceae) is a vegetable product, which contains high quantity of alpha linolenic acid that can be converted into omega-3. Phytoestrogens are polyphenolic nonsteroidal vegetable origin compounds which structurally and functionally resemble β-estradiol found in mammals^[6]. Phytoestrogens have both agonistic and antagonistic effects on estrogen receptors. These behave like endogen estrogens as agonists and trigger estrogenic effects. Flaxseed contains high level phytoestrogen which is in lignan structure (8 mg/g secoisolariciresinol dry weight) which mimics the structure of 17 βestradiol and synthetic estrogen "diethylstilbestrol". Breakdown products of lignans also have estrogenic activities[6].

This is more likely due to high omega-3 content, flaxseed is generally preferred during pregnancy. But there is controversy in consuming flaxseed during pregnancy and lactation. It has been proved that flaxseed would pass from mother to baby during pregnancy and from breast milk following birth. However it is believed that its phytoestrogen content may increase rates of breast, prostate and endometrium cancer, as well as cause truncal obesity, hypertension, anabolic activity increase and stone formation in gallbladder due to its estrogenic activity^[7].

For the patient we mentioned, high consumption of flaxseed may have caused stone formation in the gallbladder because of its phytogenic lignan structure which increases estrogenic activity.

Key words: Gallbladder Stone; Hyperbilirubinemia; Infant

References

- Beretski I, Lankin DH. Diagnosis of fetal cholelithiasis using real time high resolution imaging employing digital detection. J Ultrasound Med 1983, 2(8):381-3.
- Agnifili A, Verzaro R, Carducci G, et al. Fetal cholelithiasis: a prospective study of incidence, predisposing factors, and ultrasonographic and clinical features. *Clin Pediatr (Phila)* 1999; 38(6): 371-3.
- 3. Brown DL, Telle DI, Doubilet PM, et al. Echogenic material in the fetal gall bladder; sonographic and clinical observation. *Radiology* 1992; 182(1):73-6.
- Fanaroff AA, Martin RJ, Miler MJ. Identification and management of high-risk problems in the neonate.
 In: Creasy RK, Resnik R, eds. Maternal-Fetal Medicine, Principles and Practice, 2nd ed. Philadelphia: Saunders, 1989; Pp. 1176-9.
- 5. Abbitt LP, Mc Ilhenuy J. Prenatal detection of gallstones. *J Clin Ultrasound* 1990; 18(3):202-4.
- Ososki AL, Kennelly EJ. Phytoestrogens:a review of the present state research. *Phytother Res 2003*; 17(8):845-69.
- Jarrell J, Foster WG, Kinniburgh DW. Phytoestrogens in human pregnancy. *Obstet Gynecol Int* 2012; 2012:850313.

Leukoerythroblastosis Mimicking Leukemia: A case report

Aylin Canbolat Ayhan*, Cetin Timur, Yusuf Ayhan, Gulsen Kes

Department of Hematology-Oncology, Education and Research Hospital, Pediatrics, Istanbul Medeniyet University Goztepe, Turkey

Received: Oct 23, 2013; Accepted: Feb 23, 2014; First Online Available: Mar 16, 2014

To the Editor,

Leukoerythroblastosis due to infections can resemble leukemia, differential diagnosis can be difficult. Bone marrow examination is essential for differential diagnosis. Herein we describe a patient with leukoerythroblastosis and hepatosplenomegaly associated with inguinal abscess which was difficult to distinguish from juvenile myelomonocytic leukemia.

A 3-month-old boy was admitted to hospital with complaints of fever, vomiting and abdominal distension. He was febrile (38 °C). Liver was palpable 3 cm, spleen 4 cm below the costal

^{*} Corresponding Author; Address: Tuğlacıbaşı mh. Gedikli SK No:12/13 Feneryolu, İstanbul, Turkey E-mail: canbolataylin@hotmail.com

margins. Enlargement of bilateral inguinal lymph nodes and a mass lesion with fluctuation in the left inguinal region were observed. Laboratory findings: Hemoglobin 3.3 gr/dl, WBC 75000/mm³, platelets 253000/mm³, serum lactate dehydrogenase (LDH): 108 1U/L, uric acid: 6.4 mg/dl, other biochemical tests were normal. Creactive protein (CRP) 6.61mg/L. Direct and indirect Coombs tests were negative. Peripheral examination revealed segmented neutrophils 53%, band neutrophils 7% lymphocytes 18%, monocytes 13%, promyelocytes 1% myelocytes 2%, metamyelocytes 4%, eosinophils 2%. Left shift was accompanied by normoblasts but erythrocyte morphology was not compatible with hemolytic anemia and there were no blasts. During his follow-up his thrombocyte count decreased to 45000/mm³. Abdominal ultrasound revealed hepatosplenomegaly, bilateral inguinal lymphadenomegaly (left: 34×18 mm, right: 31×11 mm) and a mass lesion (35×20 mm) in the left inguinal region. Pathological evaluation of needle aspiration of the lesion demonstrated abscess formation but we could not identify any microorganism from drainage specimen. Systemic antibiotic therapy was started. Because of the elevated leucocyte extremely count monocytosis, circulating immature myeloid cells and nucleated red blood cells (RBCs) chronic juvenile myelomonocytic leukemia (JMML) was and bone marrow suspected aspiration performed. It did not demonstrate any blasts or monoclonality. Flow cytometric immunophenotyping excluded leukemia. Philadelphia chromosome and BCR/ABL fusion were negative. Leukocyte alkaline phosphatase (LAP) score was 54. Karyotyping was normal. All these results excluded leukemia. Bone radiographs were normal so osteopetrosis was not considered. He did not have immunodeficiency. His serum immunoglobulin G, A, M levels were normal. Analysis of lymphocyte subsets was normal. Cytomegalovirus (CMV) immunoglobulin M (IgM), EBV IgM, Parvovirus IgM, Toxoplasma gondii IgM, Rubella IgM, Rubeola IgM and Varicella IgM were negative; CMV and Parvovirus DNA PCR were also negative. Based on these findings our diagnosis was leukoerythroblastosis due to inguinal abscess. On the 10th day of antibiotics, White blood cells (WBC) decreased to 30000/mm³, on the 27th day to 20000/mm³, Platelet 154000/mm³. At discharge liver, spleen and lymphadenopathies were not palpable anymore. WBC was 13000 /mm³. Peripheral WBC count higher than 50000/mm³ with significant increase in early myeloid precursors is called leukemoid reaction. In leukoerythro-blastosis left shift is accompanied by RBCs^[1,2]. Differential diagnosis of leukemoid reactions should be made with leukemias and other causes such as infections, hemorrhage, drugs, hypersensivity syndrome, myeloid growth factors, malignancy and splenectomy^[1-3]. In 35% of patients with WBC >50000/mm³, leucocytosis was caused by leukemoid reaction[2]. In leukemoid reaction WBC count returns to normal when the predisposing factor is treated. Leukoerythroblastic reaction and presence of monocytosis is usually seen in JMML, chronic myeloid or acute monoblastic leukemias[1]. CMV infection could mimic JMML^[4]. Some infections can lead to serious depression of complete blood count and cause hepatosplenomegaly. In leukemoid reaction anemia and thrombocytopenia are usually not expected. Hepatosplenomegaly, lymphadenopathy, contribution of anemia and thrombocytopenia are frequently the features of leukemia^[5].

leukoerythroblastosis can be associated with infections but bone marrow examination is essential for differential diagnosis with leukemia.

Key words: Leukoerythroblastosis; Leukemoid reaction; Leukemia; Abscess

References

- George TI. Malignant or benign leucocytosis. Hematology Am Soc Hematol Educ Program 2012. 2012; Pp:1475-84.
- Halkes CJ, Dijstelbloem HM, Elkman Rooda SJ, et al. Extreme leucocytosis: not always leukaemia. Neth J Med 2007;65(7):248-51.
- 3. Zeng Q, Wu Y, Zhan Y, et al. Leukemoid reaction secondary to hypersensitivity syndrome to phenobarbital: a case report. *Int J Clin Exp Pathol* 2013;6(1):100-4.
- Moritake H, Ikeda T, Manabe A, et al. Cytomegalovirus infection mimicking juvenile myelomonocytic leukemia showing hypersensitivity to granulocyte-macrophage colony stimulating factor. *Pediatr Blood Cancer* 2009;53(7):1324-6.
- 5. Lanzkowsky P. Manuel of Pediatric Hematology and Oncology. 5th ed. Elsevier 2011; Pp: 272-320.