



Fava Beans

Revised: June 15, 2024.

Drug Levels and Effects

Summary of Use during Lactation

Fava beans (*Vicia faba*) contain the compounds vicine and convicine. These chemicals are metabolized to divicine and isouramil, which are potent oxidizing agents. In persons with glucose-6-phosphate dehydrogenase (G6PD) deficiency, these compounds cause hemolysis by disrupting the red cell wall. Many cases of hemolysis, and subsequent hyperbilirubinemia have been reported in breastfed infants after maternal fava bean intake. Most of the cases have been reported from around the Mediterranean and Middle East or in infants whose heritage was from this region. The prevalence of G6PD deficiency is relatively high in this geographic area, where perhaps more susceptible variants occur—at least 14 variants of G6PD deficiency are known. Most reports are of male infants, but some female infants have been affected. Favism via breastmilk can be quite severe. One breastfed infant developed renal cortical necrosis following maternal fava bean ingestion. The infant died of renal failure in the hospital 10 days after maternal fava bean ingestion. Mothers nursing a G6PD deficient infant should not consume fava beans.

Drug Levels

Maternal Levels. Relevant published information was not found as of the revision date.

Infant Levels. Relevant published information was not found as of the revision date.

Effects in Breastfed Infants

In a review of 67 cases of favism over a 3-year period by a physician in Cyprus, 2 of the cases were breastfed infants, aged 5 and 9 months. Their mothers reported eating fava beans, but not feeding any beans to their infants.[1]

A 6-month-old Yemenite infant who was primarily breastfed with supplements of wheat flower became ill 2 days after his mother ate fava beans. He became pale, vomited and passed red urine. Urine output then ceased. On the day after admission to the hospital, he became semi-comatose with a BUN of 276 mg/dL and the next day he became comatose and died. Bilateral renal cortical necrosis was determined to be the cause of the infant's death. [2]

Disclaimer: Information presented in this database is not meant as a substitute for professional judgment. You should consult your healthcare provider for breastfeeding advice related to your particular situation. The U.S. government does not warrant or assume any liability or responsibility for the accuracy or completeness of the information on this Site.

Attribution Statement: LactMed is a registered trademark of the U.S. Department of Health and Human Services.

A 4-month-old Israeli infant who was breastfed with supplementary cereals became ill after her mother ate fava beans. A day after fava bean ingestion, the infant's skin turned yellow. On admission, the infant's urine was dark and had elevated urobilinogen content. Reduced red cell glutathione was also found. After two transfusions, the infant recovered and six weeks later was shown to have G6PD deficiency. Both parents were probable carriers of the gene defect, but neither were affected.[3]

Five hundred six children under 15 years of age were admitted to a Greek hospital over an 11-year period with signs of favism. Twenty-eight patients were less than 12 months of age. Of these, 18 were breastfed and developed symptoms 2 to 6 days after maternal ingestion of fava beans. Overall, the male to female ratio of the patients was 6.2 and the highest frequency of attacks was in May when fava beans ripened.[4]

A 22-year-old Greek woman had a hemolytic event after eating in a Mexican restaurant in Nuremburg, Germany. Laboratory examination found that she was heterozygous for G6PD deficiency (3.1 units/gram of hemoglobin). Her father, mother and sister were also found to be heterozygous for G6PD deficiency. As a neonate, she reportedly suffered hemolysis and kidney failure while breastfeeding after her mother ate fava beans.[5]

Four exclusively breastfed male Arab infants developed favism after their mothers ingested fava beans. They ranged in age from 2 to 4 months and developed excessive pallor 2 to 3 days after maternal ingestion of fava beans. Their urine was deep orange in color, severe acute hemolytic anemia was diagnosed and all were shown to be G6PD deficient.[6]

Two exclusively breastfed Israeli infants, one male and one female, developed phototherapy-resistant jaundice after maternal ingestion of fava beans. Both infants were the children of Sephardic Jewish parents. The girl developed the condition at 2 days of age and the mother admitted eating fava beans on several occasions on the days leading up to delivery. The male infant was found to have elevated bilirubin at 36 hours of age. The infant's mother had consumed fava beans for several days before delivery, despite knowing that she was G6PD deficient. Passage of the hemolytic agent via the placenta cannot be ruled out.[7]

Effects on Lactation and Breastmilk

Relevant published information was not found as of the revision date.

References

1. Joannides CC. Favism in Cyprus: Analysis of 67 cases admitted to Nicosia General Hospital during the last 3 years. *Cyprus Med J* 1952;5:795-9. PubMed PMID: 14926361.
2. Casper J, Shulman J. Bilateral cortical necrosis of the kidneys in an infant with favism. *Am J Clin Pathol* 1956;26:42-7. PubMed PMID: 13292383.
3. Emanuel B, Schoenfeld A. Favism in a nursing infant. *J Pediatr* 1961;58:263-6. PubMed PMID: 13726584.
4. Kattamis CA, Kyriazakou M, Chaidas S. Favism: Clinical and biochemical data. *J Med Genet* 1969;6:34-41. PubMed PMID: 5771221.
5. Kautek L, Solem E, Böhler H. Hämolytische krise nach stillen! *der kinderarzt Der Kinderarzt* 1988;19:808.
6. Taj-Eldin S. Favism in breast-fed infants. *Arch Dis Child* 1971;46:121-3. PubMed PMID: 5555486.
7. Kaplan M, Vreman HJ, Hammerman C, et al. Favism by proxy in nursing glucose-6-phosphate dehydrogenase-deficient neonates. *J Perinatol* 1998;18 (6 Pt 1):477-9. PubMed PMID: 9848766.

Substance Identification

Substance Name

Fava Beans

Scientific Name

Vicia faba

Drug Class

Breast Feeding

Lactation

Milk, Human

Foodborne Diseases

Poisoning

Anemia, Hemolytic

Glucosephosphate Dehydrogenase Deficiency