Case Report

Maternal Placentophagy as a Possible Cause of Breast Budding and Vaginal Bleeding in a Breast-Fed 3-Month-Old Infant

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A B S T R A C T

Background: Placentophagy, or the practice of placental consumption, has grown in popularity over the past decade. Although advocates endorse prevention of postpartum depression, increased breast milk production, reduction in postpartum bleeding, and provision of nutrients postpartum, scientific studies have failed to show benefit. No studies have explored the effect of placental hormone consumption on the hypothalamic-pituitary-ovarian axis of the offspring.
Case: We present a case of vaginal bleeding and breast budding in a 3-month-old infant whose mother was exclusively breastfeeding. Maternal history was notable for placentophagy. Upon discontinuation of consuming encapsulated placenta, the infant's vaginal bleeding resolved.
Summary and Conclusion: Our case raises concerns regarding placentophagy and infant endocrine function. More research is needed to assess maternal and infant exogenous estrogen exposure with maternal placental consumption.
Key Words: Placentophagy, Pre-pubertal vaginal bleeding, Pre-pubertal breast budding

Introduction

Interest in the practice of placentophagy, or consumption of the placenta after birth, by women in the postpartum period has been increasing in frequency in the past decade.[1] Advocates of postpartum placental consumption assert the practice is associated with preventing postpartum depression, reducing postpartum bleeding, improving lactation and providing nutrients, specifically iron, needed in the postpartum period. However no scientific studies have shown evidence to support these claims.[2] To our knowledge, no studies have explored the effects of maternal placenta consumption on the offspring.

Although the placenta can be eaten raw, cooked, roasted, or dehydrated, the most common method of placentophagy is encapsulation.[3] No standard techniques for encapsulation exist, but 2 general approaches have been advocated. In the traditional Chinese method, the placenta is cleaned, sliced, and then steamed, after which it is dehydrated at between 115°F and 160°F. The placenta is then powdered and encapsulated in gelatin capsules. In the raw foods method, the placenta is cleaned, sliced, and then dehydrated at 118°F or below, after which it is similarly powdered and encapsulated. Proponents of the raw foods method note the increased number of capsules that can be made after dehydration at lower temperatures because of the loss of placental volume when dehydrated at higher temperatures.[4] However, because of the variation in the temperature to which the placenta is heated and the duration of dehydration, concerns exist regarding the persistence of infectious bacteria in consumed placenta.[3]

Research has shown the loss of iron content from the processing of the placenta to levels below that which is recommended for daily supplementation; however, findings suggest that some hormones are retained in the placenta after encapsulation at theoretically physiologic concentrations.[5,6] Specifically, levels of estradiol, progesterone, and allopregnanolone have been found in encapsulated placenta at concentrations that could have physiologic effects depending on the maximum intake of encapsulated placenta daily. Placental estradiol and progesterone has been cited as the etiology for prevention of postpartum depression and increase in breast milk production.[5] To date, research on concentrations of hormones that are subsequently bioavailable to the neonate in breast milk are lacking.

We present a case of vaginal bleeding and breast budding in a 3-month-old infant whose mother was exclusively breastfeeding. Maternal history was notable for consumption of encapsulated placenta. Upon discontinuation of consuming encapsulated placenta, the infant's vaginal bleeding resolved.

Case

The patient was born full-term via uncomplicated vaginal delivery to a nulliparous Caucasian woman at a birthing center. Prenatal care was uncomplicated with normal antenatal ultrasound examinations. On day of life 1, the neonate was taken to her pediatrician, where examination revealed ambiguous genitalia. Subsequently the

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neonate was admitted to the hospital, and evaluation was notable for the diagnosis of salt-wasting 21 hydroxylase deficiency congenital adrenal hyperplasia. Hydrocortisone, fludrocortisone, and salt replacement treatment was started. At the time of her diagnosis, examination was remarkable for Tanner 1 breasts and Prader IV external genitalia. The phallic structure measured 14 mm (width) × 25 mm (length), with a single perineal opening noted posterior to and at the base of the phallic structure. Labial-scrotal folds were fused in the midline with rugae. She had a normal perineal body and patent anus. Karyotype was 46, XX karyotype, sex-determining region Y protein-negative, with a copy number loss within chromosome band 12q21.31 spanning approximately 0.098 Mb, including part of the Otogelin like gene.

The patient was subsequently followed in a multidisciplinary clinic for gender medicine. At 1 month of life she was seen in clinic without complaints with a plan to return in 8 months to discuss initial steps in addressing possible reconstructive surgery regarding her ambiguous genitalia. However, at 3 months of age, her mother noted a 2-week history of intermittent vaginal bleeding. Examination revealed Tanner 2 breast budding. Her urine culture was negative. Her androstenedione and renin were well controlled, and therefore the vaginal bleeding was not thought to be secondary to her congenital adrenal hyperplasia. A thorough history revealed the infant was exclusively breastfed and that the mother had been consuming encapsulated placenta. Therefore, the breast development and vaginal bleeding were thought to be secondary to an exogenous maternal estrogen effect. Because of issues regarding the cost of additional testing to evaluate for other etiologies of prepubertal vaginal bleeding, including hormonal evaluation and pelvic ultrasound examination, the parents declined additional workup. With discontinuation of consumption of the encapsulated placenta, the vaginal bleeding resolved, and breast development regressed.

Summary and Conclusion

The increasing practice of placental consumption in the postpartum period raises significant concerns for the mother and infant. The Centers for Disease Control and Prevention recommends against ingestion of placental capsules due to a reported case of late onset group B streptococcus agalactiae bacteremia.[7] Because of the variability in forms of placenta consumption and the lack of standardization in the temperature to which the placenta is dehydrated and duration of heat exposure in placental encapsulation, the placenta has the potential for significant infectious morbidity in the mother and the infant.

Furthermore, although most recommendations regarding placental consumption have focused on infection risk, our case raises concerns regarding the endocrine effect of placentophagy on the mother and the infant. No data exist on the level of exogenous estrogen exposure of the breast-fed infant whose mother is consuming encapsulated placenta. However, research supports the presence of physiologic levels of estradiol in the encapsulated placenta.[5] Our patient did not have previous evidence of significant maternal estrogen effect at birth because of the absence of breast budding in the neonatal period. Although vaginal bleeding in the neonatal period is usually attributable to withdrawal of maternal estrogen, the delayed onset of our patient's bleeding with findings of breast budding suggest either endogenous or exogenous estrogen. The resolution in bleeding with cessation of consumption of encapsulated placenta suggests a possible causal effect.

Because of the prompt resolution in vaginal bleeding and concerns regarding cost, we did not pursue further evaluation for vaginal bleeding in our patient. However, measurement of estradiol, luteinizing hormone, and follicle-stimulating hormone in addition to a pelvic ultrasound examination to assess for uterine shape, size, and endometrial stripe, would be helpful adjuvants in assessing estrogen effect before and after discontinuation of encapsulated placenta. Distinct from our patient's presentation is prepubertal vaginal bleeding seen in older girls. The etiology for premature menarche is still unclear.

As placentophagy gains popularity among postpartum mothers, further research is needed regarding the full hormonal implications of maternal placenta consumption on the neonatal hypothalamic-pituitary-ovarian axis. Our case highlights that maternal ingestion of encapsulated placenta is potentially a source of exogenous estrogen exposure in the neonatal and infant period, which could be clinically significant. Mothers who wish to consume their placenta in the postpartum period should be counseled about the infectious and endocrine implications of their choice and providers should be aware of the potential exogenous hormonal effect of consumed placenta.

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