Case Report

Amniotic fluid volume changes in response to frusemide induced maternal fluid shifts

K. Manikandan, S. Raghavan

Department of Obstetrics and Gynaecology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India

Received: 17-03-2013 **Revised: 29-05-2013** **Accepted: 16-06-2013**

ABSTRACT

Frusemide use in pregnancy is generally restricted to patients with cardiac decompensation. In the past, maternal administration of oral frusemide had been tried to identify non-functioning fetal kidneys by utilizing the diuretic effect of frusemide on the fetal kidneys. We report a case of primigravida with severe mitral stenosis presenting in pulmonary edema that received high dose of frusemide. This was associated with the development of transient oligohydramnios. We discuss the clinical significance of this observation and the possible explanations.

Key words: Frusemide, oligohydramnios, pregnancy

INTRODUCTION

Maternal use of frusemide had been tried to induce fetal diuresis to help differentiating fetuses with non-functioning kidneys from those suffering from severe placental insufficiency. Maternal frusemide causes a transient increase in the fetal urine production and consequently a small increase in the amniotic fluid volume (AFV). We report an unusual case of frusemide induced transient oligohydramnios.

CASE REPORT

A 26-year-old unbooked primigravida, weighing 50 kg with a pre-pregnancy diagnosis of severe rheumatic mitral stenosis (mitral valve area 1 cm^2) and pulmonary artery hypertension, presented to us at 34 weeks of gestation with acute pulmonary edema. She was managed with intravenous morphine, intravenous frusemide, digoxin, fluid restriction, oxygen by mask. Her cardio respiratory status improved over the next 48 h. During this period, she had received 80 mg frusemide intravenous loading dose and then was on 40 mg frusemide every 8 h. She had passed a total of 2000 ml of urine per day during these 2 days. On the 4^{th} day of her admission, obstetric ultrasound was performed based on clinical suspicion of reduced liquor. The amniotic fluid index (AFI) was 4 cm. The biophysical profile was normal and hence it was decided to review the fetal status after 1 week. Meanwhile, her general condition improved and she had been switched over to 40 mg oral frusemide once daily. The AFI repeated after 1 week and 2 weeks from the first scan were 9 cm and 11 cm, respectively. She continued the pregnancy till 38 weeks and had an outlet forceps delivery to cut short the second stage of labour. Post-natal period was complicated by episiotomy wound dehiscence, which required secondary suturing. She was discharged on the post-natal day 10.

Access this article online	
Quick Response Code:	Website: www.jpharmacol.com
	DOI: 10.4103/0976-500X.130075

Address for correspondence:
K. Manikandan, Department of Obstetrics and Gynaecology, Jawaharlal Institute of Postgraduate Medical Education and Research Quarters, Dhanvantari Nagar, Puducherry - 605 006, India. E-mail: manikandan.k@jipmer.edu.in

DISCUSSION

The bulk of amniotic fluid inflow is contributed by fetal urine production in the second half of pregnancy. When placental perfusion is optimum, the functioning fetal kidneys produce urine at a rate of about 5 ml/h at 20 weeks increasing to about 51 ml/h by the third trimester.[1] Placental perfusion is influenced by several factors, which include but not limited to maternal hydration status and intravascular volume, perfusion pressure in the uterine arteries and functional placental surface area. Maternal hydration therapy, orally as well as intravenously has been tried in cases of oligohydramnios to improve the liquor status, with some degree of success.[2] The increase in the AFI is limited to parenteral hypotonic fluid infusions,[3] suggesting a mechanism beyond simple expansion of intravascular volume. It has been suggested that the maternal fluid compartment contributes significantly to the AFV in the last trimester,[4] possibly through the so-called transmembranous pathway.[5,6]

Maternal administration of frusemide had been shown to induce fetal diuresis in both normal and growth restricted fetuses.[7] This effect has also been studied to differentiate fetal non-functioning kidneys from severe placental insufficiency in pregnancies with anhydramnios.[8] In addition to bladder filling, a transient small increase in the AFV is expected due to fetal diuresis. In our case, due to the prolonged administration of high dose loop diuretic and consequent maternal diuresis, maternal intravascular volume contraction transiently reduced the placental perfusion. In addition, an increase in amniotic fluid outflow through the trans-membranous pathway in response to fall in the hydrostatic pressure on the maternal side is also possible. The placental function was normal in this pregnancy as seen by the return of AFI to normalcy with time, during which time the fluid shifts between extravascular and intravascular compartments would have been equilibrated. The sequence of events suggests that the amniotic fluid space also behaves as an extension of maternal fluid spaces.

This case emphasizes the importance of taking the maternal status and potential effect of frusemide in interpreting the AFI. Otherwise, the fetus would have been delivered prematurely on the grounds of placental insufficiency.

REFERENCES

1. Rabinowitz R, Peters MT, Vyas S, Campbell S, Nicolaides KH. Measurement of fetal urine production in normal pregnancy by real-time ultrasonography. Am J Obstet Gynecol 1989;161:1264-6.
2. Flack NJ, Sepulveda W, Bower S, Fisk NM. Acute maternal hydration in third-trimester oligohydramnios: Effects on amniotic fluid volume, uteroplacental perfusion, and fetal blood flow and urine output. Am J Obstet Gynecol 1995;173:1186-91.
3. Hofmeyr GJ, Gülmezoglu AM. Maternal hydration for increasing amniotic fluid volume in oligohydramnios and normal amniotic fluid volume. Cochrane Database Syst Rev 2002;1:CD000134.
4. Modena AB, Fieni S. Amniotic fluid dynamics. Acta Biomed 2004;75 Suppl 1:11-3.
5. Gilbert WM, Brace RA. The missing link in amniotic fluid volume regulation: Intramembranous absorption. Obstet Gynecol 1989;74:748-54.
6. Gilbert WM, Brace RA. Amniotic fluid volume and normal flows to and from the amniotic cavity. Semin Perinatol 1993;17:150-7.
7. Wladimiroff JW. Effect of frusemide on fetal urine production. Br J Obstet Gynaecol 1975;82:221-4.
8. Raghavendra BN, Young BK, Greco MA, Lustig-Gillman I, Horii SC, Hirsch MA, *et al*. Use of furosemide in pregnancies complicated by oligohydramnios. Radiology 1987;165:455-8.

How to cite this article: Manikandan K, Raghavan S. Amniotic fluid volume changes in response to frusemide induced maternal fluid shifts. J Pharmacol Pharmacother 2014;5:153-4.

Source of Support: Nil, **Conflict of Interest:** None declared.