Maternal (Oral) Hydration Increases Amniotic Fluid Volume

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Objective: To determine the effect of maternal (oral) hydration on amniotic fluid volume in patients with third trimester oligohydramnios. Design: Interventional study. Place and Duration of Study: Department of Obs & Gynae Unit III, Sir Ganga Ram Hospital, Lahore from May 2002 to October 2002. Patients and Methods: Twenty five women with third trimester oligohydramnios (AFI ≤5.0cm) and twenty five controls with normal amniotic fluid volume (AFI 8-24 cm) were prospectively recruited for this study. Maternal urine specific gravity and amniotic fluid index were determined before and after maternal hydration by asking them to drink 2 L of water in 2-4 hours before repeat amniotic fluid index and recorded on printed proformas. Results: Hydration increased amniotic fluid volume in women with oligohydramnios (mean change in amniotic fluid index 4.3 cm, 95% confidence interval 4.02 to 5.06; P value <0.001); as well as in women with normal fluid volume (mean change in Amniotic fluid index 2.7 cm, 95% confidence interval 2.23 to 3.21; P value <0.01). However, percentage increase in mean AFI was 58.6% in the oligohydramnios group, which was significantly greater (P value <0.05) than the percentage increase of 28.4% in control group. Hydration was associated with decrease in urine specific gravity in both groups. Conclusion: Maternal (oral) hydration increases AFV in women with oligohydramnios as well as in women with normal AFV and may be beneficial in the management of oligohydramnios.

Key words: Maternal hydration, oligohydramnios

Oligohydramnios has long been recognized as a predictor of adverse perinatal outcomes and has been correlated with increased rates of perinatal mortality, caesarean delivery for fetal distress, meconium passage, low APGAR scores and neonatal resuscitation or neonatal intensive care unit admission.

Oligohydramnios in the presence of intact membranes is a common obstetric complication, occurring in 3% to 5% of pregnancies at term. Such pregnancies are at increased risk of fetal distress and are associated with a high rate of operative delivery and meconium aspiration.

Randomized clinical trials have shown that correction of oligohydramnios by intrapartum amnioinfusion reduces the incidence of these complications. Although, this technique has increasingly been incorporated into the management of pregnancies with oligohydramnios, amnioinfusion has several limitations. It can only be instituted during labour in the presence of ruptured membranes, it requires an indwelling catheter and frequent monitoring, and, as with any invasive procedure, it is not without risk.

An alternative, noninvasive option to increase amniotic fluid volume is maternal (oral and intravenous) hydration. In recent studies Hofmeyr GJ, Magann EF and Malhotra B, Deka D reported that maternal intake of 2 L of water in a 2-hour period increased amniotic fluid index in pregnancies with oligohydramnios and in those with a normal amniotic fluid volume.

The purpose of this study was to confirm the observation that maternal hydration would increase amniotic fluid volume in women with decreased amniotic fluid levels. We chose to use the amniotic fluid index as the dependent variable because it is reliable, easy to measure, and normal ranges have been published. The normal range, though it varies slightly with gestational age, is approximately 8–24 cm. We chose an amniotic fluid index of 5.0 cm or lower as indicative of decreased amniotic fluid.

Aims and objectives:
To determine the effect of maternal (oral) hydration on amniotic fluid volume in patients with third trimester oligohydramnios.

Patients and methods:
This study was carried-out in the Department of Obstetrics and Gynaecology, Sir Ganga Ram Hospital Lahore during six months period from May 2002 to October 2002.

The study group consisted of twenty five women with third trimester oligohydramnios defined as an AFI ≤ 5.0 cm. Twenty five women with third trimester pregnancies and normal AFV, defined as an AFI between 8.0 to 24.0 cm, were recruited as controls. Women with oligohydramnios were recruited from the maternal fetal assessment unit; women with normal amniotic volume were recruited randomly from the low-risk population attending the ante-natal clinic. Patient's demographic characteristics and distribution of high-risk patients are summarized in Table I & II respectively.

Inclusion and exclusion criteria for both groups were as follows:

Inclusion Criteria:
- Patients with singleton pregnancy with well established dates at 28-42 weeks gestational age.
- Fetus with no congenital anomalies.
- Determination of AFV with technique of Phelan et al.
- Criteria for oligohydramnios is AFI ≤ 5 cm.
- Criteria for normal AFV is AFI 8 to 24 cm.
Exclusion Criteria:
- Women at risk of fluid overload such as those with cardiac disease, renal impairment, moderate or severe preeclampsia or hypertension and diabetes.
- Ruptured membranes.
- Multiple pregnancy.
- Women receiving prostaglandin synthetase inhibitors.

One of the fetuses in the study group was small for gestational age, defined as an estimated fetal weight < 5th percentile, but none in the control group were small for gestational age. Study variables, including urine specific gravity and AFI were performed before and after hydration by the same operator so that each woman acted as her own control. Each study took place at the same time of day (i.e. between 1 and 6 PM) and the hydration period was from 2 to 4 PM.

Maternal Intravenous Hydration: Once basal measurements (amniotic fluid index and urine specific gravity) were taken, women were given a 1L plastic container marked in milliter and asked to drink 2 litre of water in 2-4 hours period. Throughout the study these women were supervised directly by the midwife in the antenatal assessment unit to ensure compliance, and they had free access to toilet facilities.

Urine Specific Gravity Studies: Baseline maternal urine sample was taken immediately before the hydration for direct measurement, which was performed by standard laboratory techniques. At the end of the hydration period, patient was instructed to micturate again and then the next sample was collected for analysis.

Amniotic Fluid Index: The AFI was measured by technique of Phelan et al by dividing the uterus into four quadrants, measuring the deepest pool in each and calculating it as the sum of the four measurements. Equipment used in this study included Acuson model machine which was equipped with 3.5 and 5.0-MHz curvilinear transducers. All measurements were made with medium transducer pressure to reduce intraobserver variability. For each AFI the mean of two measurements was used for analysis.

Statistical Analysis: The data analysis was computer based using statistical package SPSS version 10.0 and sample independent "t" test was used to compare the mean AFI and urine specific gravity before and after treatment, and the posttreatment AFI - pretreatment AFI (Delta AFI).

Results:
All women compiled with the protocol and no maternal complications were found. Baseline urine specific gravity (USG) was comparable in the two groups, there was no significant difference in USG between the groups.

Table III summarizes the results. In women with oligohydramnios, the mean AFI increased from 3.5 cm to 7.8 cm (mean Δ AFI 4.3cm, 95% confidence interval 4.02 to 5.06; P value < 0.001), displayed in Fig. I. Significant change in AFI was observed in women with normal AVF; mean AFI increased from 8.4cm to 11.0 cm (mean Δ AFI 7.6 cm, 95% confidence interval 12.23 to 3.21; P value < 0.001), displayed in Fig. II. However, the percentage increase in mean AFI was 58.6% in the oligohydramnios group, which was significantly greater (P value < 0.05) than the percentage increase of 28.4% in control group.

USG did not show any significant difference between the 2 groups after treatment.

<table>
<thead>
<tr>
<th>Table I: Patient demographic characteristics</th>
<th>Oligohydramnios Group (n=25)</th>
<th>Control Group (n=25)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr, mean ± SD)</td>
<td>22.6 ± 3.6</td>
<td>22.3 ± 3.4</td>
<td>NS</td>
</tr>
<tr>
<td>Parity (mean ± SD)</td>
<td>0.5 ± 0.6</td>
<td>0.4 ± 0.7</td>
<td>NS</td>
</tr>
<tr>
<td>No. of nulliparous women</td>
<td>17 (68%)</td>
<td>15 (60%)</td>
<td>NS</td>
</tr>
<tr>
<td>No. of multiparous women</td>
<td>8 (32%)</td>
<td>10 (40%)</td>
<td>NS</td>
</tr>
<tr>
<td>Estimated gestational age (days, mean ± SD)</td>
<td>272 ± 6.0</td>
<td>273 ± 5.0</td>
<td>NS</td>
</tr>
<tr>
<td>No. of patients with postdates pregnancies</td>
<td>5 (20%)</td>
<td>4 (16%)</td>
<td>NS</td>
</tr>
<tr>
<td>(&lt;287 days)</td>
<td></td>
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<tr>
<td>Amniotic fluid index</td>
<td>3.2 ± 0.79</td>
<td>8.4 ± 1.8</td>
<td>S</td>
</tr>
<tr>
<td>NS= Non-significant, S= Significant</td>
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<table>
<thead>
<tr>
<th>Table II: Distribution of high risk patients</th>
<th>Oligohydramnios Group(n=25)</th>
<th>Control Group (n=25)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension pregnancy induced hypertension</td>
<td>13 (52%)</td>
<td>5 (20%)</td>
<td>S</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Previous caesarean delivery</td>
<td>6 (24%)</td>
<td>5 (20%)</td>
<td>NS</td>
</tr>
<tr>
<td>Postdates pregnancies (&gt;287 days)</td>
<td>5 (20%)</td>
<td>4 (16%)</td>
<td>NS</td>
</tr>
<tr>
<td>IUGR</td>
<td>1 (1%)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>0</td>
<td>1 (1%)</td>
<td>NS</td>
</tr>
<tr>
<td>History of pyelonephritis</td>
<td>1 (1%)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>NS=Non-significant, S=Significant</td>
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Table III: Results of maternal hydration

<table>
<thead>
<tr>
<th></th>
<th>Oligohydramnios Group (n=25)</th>
<th>Control Group (n = 25)</th>
</tr>
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<tbody>
<tr>
<td><strong>Pre-treatment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AFI (cm)</td>
<td>3.5 ± 0.79</td>
<td>8.4 ± 1.53</td>
</tr>
<tr>
<td>USG</td>
<td>1.011 ± 0.007</td>
<td>1.012 ± 0.009</td>
</tr>
<tr>
<td><strong>Post-treatment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AFI (cm)</td>
<td>7.8 ± 1.35</td>
<td>11.0 ± 1.68</td>
</tr>
<tr>
<td>USG</td>
<td>1.005 ± 0.008</td>
<td>1.006 ± 0.007</td>
</tr>
<tr>
<td>Delta AFI (cm)</td>
<td>4.3 ± 1.25</td>
<td>2.7 ± 1.22</td>
</tr>
</tbody>
</table>

AFI= Amniotic fluid index. USG= Urine specific gravity. Delta AFI = Post treatment AFI - Pre-treatment AFI.
Data are presented as mean ± standard deviation, \( P < 0.05 \)

However, there are several possible confounding variables that should be discussed. We do not know for sure that the patient's background hydration status was the same in each group because no attempt was made to assess descriptively the amount of liquid each woman usually drinks. This was believed to be very cumbersome and not particularly reliable. However, urine specific gravity is considered an estimate of hydration state, as it correlates with urine osmolality, and was not significantly different between the groups before treatment suggesting a similar hydration state. Thus, it seems unlikely that a difference in initial hydration state between the groups could account for the difference in delta AF index.

Although maternal hydration increased the AFI, neither the mechanism of this change in AFV nor the length of time the increase would persist is clear. This study was not designed to answer these questions, but to investigate in a noninvasive fashion whether AFI could be increased with maternal hydration. Although it is not known why AF decreases in certain pregnancy states, it is well accepted that fetal urine output is a major contributor to AFV in the second and third trimesters of pregnancy. Thus a reasonable initial approach to investigate a mechanism of AFV control is to evaluate how maternal, placental, or fetal factors could affect fetal urine output, while accepting that there may be other regulating factors as well.

Two factors known to affect urine output in adults are intravascular volume and osmolality. There are clinical data, albeit sparse, suggesting that the fetus can respond to a maternal change in either intravascular volume or osmolality. Goodlin et al.\(^1\) reported a correlation between measured maternal intravascular volume and AFV after patients with diabetes or fetal anomalies were excluded. Intravenous hydration with 6500 mL of an isotonic solution increased AFV in a markedly dehydrated woman\(^4\). In addition, Battaglia et al.\(^5\) demonstrated that when maternal osmolality was increased or decreased, fetal osmolality changed in a parallel fashion. Whether this alteration in fetal osmolality would affect either fetal urine output or AFV was not addressed in that paper. Experimental data, primarily in sheep, do show that the fetal urine output changes with alterations in maternal osmolality. Water deprivation or IV mannitol in the ewe resulted in a decrease in fetal urine flow, implicating maternal osmolality as an important variable\(^6\).
However, it is also clear that changes in fetal volume or osmolality can alter fetal urine output, AFV, and, to a lesser extent, fetal intravascular volume. In our study because we did not measure maternal osmolality or intravascular volume, we cannot know with confidence whether either changed. However, it seems more likely that 2 L of a hypotonic solution would decrease osmolality.

Only 11 of 25 women in both groups had a difference in AFI greater than the mean change in AFI. That the AFI did not significantly increase in all patients with maternal hydration raises several intriguing questions. Methodologic problems could account for this. In addition, perhaps relative maternal dehydration is a prerequisite for maternal hydration to increase the AFV. This assumes that the decrease in fluid is at least a partial result of maternal dehydration. However, our data do not support this if one accepts maternal urine specific gravity as a reasonable estimate of hydration state. We did not find a significant correlation between the initial maternal urine specific gravity and the delta AFI.

More interesting, because we do not know the etiology of oligohydramnios, the different indications for antenatal testing may affect how AF responds to maternal hydration. For example, decreased fluid in a post-date parturient may occur by a different mechanism than that in a patient with IUGR. Unfortunately, because this study did not set out to analyze the effect of the antenatal testing indication on hydration effect, there are insufficient subjects in each testing indication to address the question meaningfully.

A further theoretical explanation for the lack of uniformity in the response is that adequate uterine/placental perfusion and, if fetal urine output is involved, adequate fetal renal response must exist for any augmentation of AFV to occur. This likely relates to the indication for fetal testing, or more specifically, to the cause of the initial oligohydramnios. One might propose, in a severely growth-retarded fetus with chronic oligohydramnios and compromise, that maternal hydration would be insufficient to alter the AFV. In contrast, a fetus with a more acute cause for decreased fluid may, respond to maternal fluid challenge with an increase in fluid. Support for this hypothesis might be garnered by comparing delivery outcome between hydrated women whose AFV does increase with hydrated women showing no AF improvement.

**Conclusion:**

In conclusion, our study demonstrates that amniotic fluid volume is markedly influenced by acute changes in maternal plasma osmolality (maternal fluid volume) and suggests a potential role for maternal hydration in the treatment of oligohydramnios.

**References:**