Mid-trimester diagnosis of bladder neck obstruction by ultrasound and paracentesis

JOSEF SHALEV, ZION BEN-RAFAEL, BOLESLAV GOLDMAN*, I ENGELBERG†, AND SHLOMO MASHIACH

Department of Obstetrics and Gynecology, *Institute of Human Genetics, and †Department of Pathology, Chaim Sheba Medical Center, Sackler School of Medicine, Tel-Aviv University, Tel-Hashomer, Israel.

SUMMARY A bladder neck obstruction was suspected after ultrasound investigation at 16 weeks' gestation. Evaluation of protein content in the amniotic fluid, fetal ascites, and fluid from the overdistended bladder supported the diagnosis. Bladder outflow obstruction in the second trimester of pregnancy was not associated with raised alphafetoprotein levels in the amniotic fluid and maternal serum.

Ultrasonic diagnosis of fetal urinary tract malformations and in utero treatment of urinary tract obstruction has been previously reported.1-3 The identification of an intra-abdominal cystic structure such as the fetal bladder may be difficult, as retention of up to 3500 ml urine has been reported.4 In this report ultrasonic guided paracentesis and chemical analysis of the fetal and amniotic fluids were performed for antenatal diagnosis which enabled early termination of the pregnancy.

Case report

A 28-year-old woman, GI, PO, underwent routine ultrasonic examination at 16 weeks' gestation. A single fetus was observed with a BPD of 29 mm, appropriate for gestational age. The fetal abdomen was distended by a 11 cm × 7 cm cystic mass (fig 1) and the viscera were totally compressed against the chest. A lethal anomaly was suspected. After gaining the parents' informed consent, the pregnancy was terminated by intra-amniotic infusion of hypertonic saline. During this procedure amniotic fluid was tapped and the contents of the cystic structure partially aspirated. Thereafter, free intra-abdominal fluid (ascites) in the fetus was successfully aspirated.

**TABLE**

<table>
<thead>
<tr>
<th>Laboratory data</th>
<th>Amniotic fluid</th>
<th>Ascites</th>
<th>Cystic structure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mEq/l)</td>
<td>137</td>
<td>83</td>
<td>98</td>
</tr>
<tr>
<td>Potassium (mEq/l)</td>
<td>3.7</td>
<td>2.6</td>
<td>2.9</td>
</tr>
<tr>
<td>Chloride (mEq/l)</td>
<td>108</td>
<td>75</td>
<td>84</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>34</td>
<td>64</td>
<td>44</td>
</tr>
<tr>
<td>Total protein (mg/dl)</td>
<td>800</td>
<td>175</td>
<td>10</td>
</tr>
</tbody>
</table>

Fig 1  Real-time echograph 9 cm above the symphysis pubis demonstrating the grossly dilated fetal bladder (B). Fetal viscera are compressed against the fetal chest (ch) and the fetal abdomen is much larger than the fetal head (H).
The chemical analysis of the fluid samples obtained (amniotic fluid, fluid from the cystic structure, and fetal ascites) are given in the table. A clear difference in protein content was observed in the three fluids, with the lowest values being in the fluid aspirated from the cystic structure which represented the fetal bladder. The alphafetoprotein level in the amniotic fluid was found to be normal. Eighteen hours after amniocentesis a male fetus was aborted. Necropsy confirmed the presence of bladder neck obstruction (fig 2).

Discussion

The successful diagnosis of urinary tract malformation in an uneventful pregnancy stresses the importance of routine ultrasonic screening in the second trimester.

Differential diagnosis of a fetal intra-abdominal cystic mass may be difficult. Evaluation of its fluid content might help in reaching the diagnosis. The presence of bile acid in the amniotic fluid positively supported the diagnosis of intestinal obstruction. Nevin et al found raised alphafetoprotein levels in the amniotic fluid in cases of overdistended fetal bladder caused by absence of the urethra. Chemke et al reported Meckel’s syndrome associated with polycystic kidneys in which raised alphafetoprotein was found. Kjessler et al observed raised alphafetoprotein in congenital nephrosis. In our case the AFP level in the amniotic fluid was found to be normal. This was probably owing to secondary intestinal obstruction proximal to the duodenum, caused by the extremely overdistended bladder, which partially prevented regurgitation of bile into the amniotic fluid.

The decision as to whether to treat low urinary tract obstruction in utero or to terminate the pregnancy is based upon the degree of renal damage and the ability to recuperate. Dynamic ultrasound studies of renal function in cases of bladder neck obstruction or posterior urethral valve are of limited value, because they are based on the normal functioning of the urinary bladder. Abnormal alphafetoprotein levels in the amniotic fluid do not indicate the presence of urinary tract malformations. Further studies of the composition of the urine at various stages of pregnancy and ultrasonic evaluation of renal anatomy could be important future methods in determining the degree of renal damage in cases of bladder outflow obstruction.

References