The Sonographic Evaluation of Fetal Anomalies in Oligohydramnios Between 16 and 30 Weeks Gestation

The sonograms of all patients with oligohydramnios between 16 and 30 weeks gestation seen over a 4-year period were reviewed to determine (1) whether sonographically detectable fetal anomalies were present, and (2) when these anomalies were present, how this information was used in maternal fetal management. Cases of ruptured membranes and fetal demise were excluded from the study. Sixteen patients with severe oligohydramnios were identified. On postmortem examination, nine had urinary tract anomalies, one had evidence of a chronic intrauterine infection, and four had no anomalies. There were only two neonatal survivors: one had no anomalies while the other had posterior urethral valves. These findings confirm that second trimester oligohydramnios has a poor prognosis and is often associated with anomalies of the urinary tract. Sonography aids in the clinical management of such patients.

Oligohydramnios has been found in association with ruptured membranes, intrauterine growth retardation, preeclampsia, postmaturity, fetal demise, and renal anomalies [1]. Reports of these cases were based on sonographic findings not limited to a specific time period during pregnancy. We could find only one report in the literature that examined fetal outcome in cases of second-trimester oligohydramnios [2]. The present report describes the results of a retrospective study of fetal outcome in patients with oligohydramnios between 16 and 30 weeks gestation, roughly the period of the second trimester. We feel that diagnosis at this early stage can be very helpful in guiding clinical management.

Materials and Methods

A retrospective analysis was made of all patients who underwent obstetric sonography at The George Washington University Hospital between December 1981 and November 1985. Sonographic data, medical records, and autopsy/surgical pathology records of all patients with oligohydramnios identified for the first time between 16 and 30 weeks were reviewed. Oligohydramnios was diagnosed sonographically on the basis of subjective criteria previously described by Phillipson [3]. These criteria were (1) obvious lack of amniotic fluid, (2) poor fluid/fetal interface, and (3) marked crowding of fetal limbs. The degree of oligohydramnios was subjectively graded into mild, moderate, or severe, and measurements of the largest pocket of amniotic fluid were obtained. All scans were performed with real-time sector scanners using 3.5-, 5.0-, and/or 7.5-MHz transducer of appropriate focal length. The scans were performed in a routine fashion based on our department's protocol, and all scans were checked by a physician. An obstetric record was kept on every patient showing fetal measurements (biparietal diameter, head and abdominal circumference, and femur length), fetal position, placental grade and position, amount of amniotic fluid, presence or absence of fetal heart motion, and assessment of fetal anatomy. Multiple images on hard copy were obtained in every case. Patients with ruptured membranes or fetal demise at the time of sonographic evaluation were excluded from the study.
Results

Sixteen patients with severe oligohydramnios met the criteria for the study. They were scanned one or more times. Three patients had prior sonograms with normal amniotic fluid and had oligohydramnios on follow-up examinations (Fig. 1).

In six patients, a fetal urinary tract anomaly was noted on sonographic evaluation. These included two cases of infantile polycystic kidney disease (polycystic kidney disease type I), including one with Meckel’s syndrome, one case of marked cystic dysplasia of both kidneys (cystic kidney type II), two cases of bilateral hydronephrosis, and one of unilateral hydronephrosis. In four of these patients, a small fetal urinary bladder was identified sonographically. Serial examinations were made over a 2-hr period in two, and no bladder filling or emptying was observed (Fig. 2). In three patients the pregnancy was terminated on the basis of sonographic findings, and in one, termination was necessary because of severe chronic maternal hypertension. There were two liveborn infants, one of whom died in the first 12 hr of life. In all six, the postmortem or postnatal findings were consistent with the sonographic findings.

In six patients, the fetal kidneys and bladder were not identified sonographically. Termination of pregnancy was influenced by the sonographic findings in one. In two additional patients, termination was due to chronic maternal hypertension or preeclampsia. One patient had a spontaneous abortion and another a fetal demise. On postmortem examination, no urinary tract anomalies were demonstrated in three. One had bilateral renal agenesis, another had a horseshoe kidney, and another had marked cystic dysplasia of one kidney (cystic kidney type II) and absence of the contralateral kidney.

In one patient, sonographic examination showed a small urinary bladder but no kidneys. A prenatal cytogenetic diagnosis of full trisomy 22 was made. The fetus died, and postmortem examination showed multiple congenital malformations including agenesis of the left kidney and an ectopic right pelvic kidney.

In three patients there were no sonographic abnormalities of the fetal urinary tract. Of these, two died, but no urinary tract anomalies were noted on postmortem examination; the other was a liveborn infant with no postnatal evidence of urinary tract anomalies.

Discussion

Amniotic fluid in early pregnancy is derived from maternal and fetal extracellular fluid by dialysis across tissue layers largely impermeable to protein [1]. After midpregnancy, diffusion across the fetal skin is impaired by cornification. Fetal urine begins to play an increasingly important role in the maintenance of amniotic fluid volume, and excretion of hypotonic fetal urine has been noted as early as 12 weeks
gestation [4]. At 18 weeks gestation, fetal urine production has been estimated to be 7–17 mI/day, and it increases steadily throughout pregnancy. At term, it is approximately 800 mI/day, which equals the total amniotic fluid volume at this stage [4]. Amniotic fluid in normal pregnancy increases by a maximum of 10 mI/day [4]. Approximately half the daily urine produced is removed by fetal swallowing. Recent work indicates that most of the remainder may be reabsorbed in the fetal lungs [4]. Amniotic fluid is felt by some to play a crucial role in fetal lung development [5]. With early and severe oligohydramnios, thoracic compression may additionally contribute to pulmonary hypoplasia [5]. With less severe oligohydramnios, sufficient pulmonary growth and development may occur allowing for survival with varying degrees or even absence of pulmonary dysfunction. Hypoplastic lungs associated with oligohydramnios show histologic changes consistent with arrested maturation [5].

The period of time selected for our study (16–30 weeks) is of interest for two important reasons. First, the role of fetal urine excretion in the maintenance of amniotic-fluid volume is probably not significant before this time. One patient had a sonogram at 12 weeks and two at 17 weeks gestation with normal amniotic fluid. On subsequent scans at 29, 23, and 21 weeks gestation, respectively, marked oligohydramnios was present (Fig. 1). This time period is also important because any gross, life-threatening anomalies should be recognized before 20 weeks if a therapeutic abortion is to be performed.

The sonographic diagnosis of oligohydramnios in our patients was based on subjective criteria (Fig. 1). Quantitative measurements of the largest pocket of amniotic fluid were also obtained in all of the patients. Several investigators have advocated quantitative techniques in the assessment of oligohydramnios [6–8]. We felt that over a wide range of gestation ages, numeric values did not provide a consistent assessment of oligohydramnios.

A number of conditions have been associated with a decreased volume of amniotic fluid. These include ruptured membranes, fetal demise, intrauterine growth retardation, and renal anomalies [1]. The explanations in cases of ruptured membranes or fetal demise are obvious, as there is leakage of amniotic fluid or cessation in production, respectively. The etiology in intrauterine growth retardation is less clear. One mechanism that has been proposed is a decreased production of fetal urine and lung liquid as a result of redistribution of cardiac output induced by hypoxemia. Experimental work in fetal lambs and monkeys has demonstrated that chronic uteroplacental insufficiency is associated with an increased incidence of intrauterine growth retardation and fetal hypoxemia [9–10]. Experimental hypoxemia in fetal lambs results in redistribution of blood flow from “low priority organs” (lung and kidneys) to “high priority organs” (brain and heart) [11]. The degree of redistribution is felt to be related to the severity of the insult.

Chronic fetal hypoxemia may have contributed to diminished or absent renal function in at least six of our patients. In three, marked intrauterine growth retardation was suspected from sequential clinical and sonographic evaluations. One additional patient had a probable chronic intrauterine infection, a condition previously associated with intrauterine growth retardation. In three additional patients, the pregnancy was complicated by chronic maternal hypertension or preeclampsia, and although fetal growth was felt to be consistent with dates on sequential evaluations, chronic uteroplacental insufficiency may have been present.

Urinary tract anomalies associated with decreased or absent fetal urine production are well recognized causes of oligohydramnios. These include bilateral obstructive uropathy, bilateral renal agenesis, and bilateral cystic renal disease [2, 8, 12, 13]. Unilateral fetal urinary tract disease (i.e., unilateral obstructive uropathy) has also been associated with oligohydramnios [12]. The fetal urinary bladder should be consistently identifiable by 18 weeks gestation, and persistent failure of visualization strongly suggests nonfunction of the kidneys (Fig. 3) [5]. Normal fetal kidneys may also be identified at 18
weeks gestation, although early visualization is not as easy as with the bladder.

Infantile polycystic kidneys (polycystic kidney disease type I) and cystic dysplastic kidneys (cystic kidneys type II) are irreversible conditions, and sonographic differentiation from potentially reversible fetal hydronephrosis may be clinically useful. Fetal interventional procedures consisting of in utero decompression by suprapubic and transurethral catheter drainage of fetal urine into the amniotic sac have been used in a limited number of fetuses with bilateral hydronephrosis [5]. Preterm delivery, particularly after 32 weeks and extraterine decompression, may be an option [5]. Early hydronephrosis results in dilatation of the infundibulum and calices and has the sonographic appearance of branching, fluid-filled structures of uniform size deployed centrally (Fig. 4) [1]. With progression, thinning of the renal parenchyma occurs, ultimately resulting in a large, solitary, cystic structure. Arger et al. [14] have suggested that the combination of a renal pelvis measuring 10 mm or more in anteroposterior diameter and a maximal transverse pelvic diameter exceeding 50% of the renal diameter at the same level can be used to identify significant fetal hydronephrosis. Cystic dysplastic kidneys appear sonographically as multiple cysts of various sizes (Fig. 5) [1]. The cystic structures cannot always be identified and, when seen, are randomly distributed. Infantile polycystic kidneys are sonographically large and more hyperechoic than normal [1]. Cystic structures are rarely identified (Fig. 2).

Ten of our sixteen patients had a urinary tract anomaly and
seven of these were anomalies that have been associated with oligohydramnios. Sonography identified a renal anomaly in five of the seven. Furthermore, sonography was able to more specifically characterize the urinary tract anomaly as hydronephrosis or cystic renal disease. In a sixth patient who had bilateral renal agenesis on postmortem examination, no kidneys could be identified sonographically. The fetal urinary bladder may be visualized sonographically in the absence of adequate urine output. In four of these six patients, a small bladder was visualized. The important observation is whether there is bladder filling and emptying on serial examinations. In two cases where this was done, no bladder filling or emptying was seen (Fig. 2). In the seventh patient, no fetal kidneys were identified sonographically, and a single cystic dysplastic kidney was noted on postmortem examination. The failure to visualize a fetal bladder suggested nonfunction of the kidneys. The sonographic findings played a role in electively terminating four of the six pregnancies, and all four had findings on postmortem examination that were incompatible with extraterine viability (i.e., bilateral renal agenesis, cystic renal disease, either bilateral or with agenesis of the contralateral kidney, or severe hydronephrosis with pulmonary hypoplasia).

Two patients had unilateral urinary tract disease. Sonography was able to identify the anomaly in one of these. In the other, the solitary pelvic kidney was not identified, possibly because of its ectopic location. As previously discussed, fetal hypoxemia probably contributed to the development of oligohydramnios in these two patients. One patient had a horseshoe kidney, a urinary tract anomaly not causally related to oligohydramnios. The fetal kidney was not identified, again, possibly because of its ectopic location.

In three of six patients without urinary tract anomalies on postmortem examination or postnatally, no fetal kidneys or bladder were identified sonographically. The poor fetal/fluid interface as a result of severe oligohydramnios most likely contributed to the nonvisualization of the kidneys. The bladder was not identified presumably because of the lack of fetal urine production.

In conclusion, sonographic detection of oligohydramnios between 16 and 30 weeks is associated with a poor prognosis and a high frequency of urinary tract anomalies. Because of this, a careful examination of the fetus should be performed with special attention to the fetal kidneys and bladder along with dynamic sonographic assessment of fetal urine production, if indicated. This information can then be used in developing a rational maternal and fetal management strategy.

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REFERENCES